CHAPTER - II

Literature Review
Every research contributes a drop to the vast Ocean of knowledge. Knowledge is therefore the sum of total of a multitude of researches conducted by different investigators over a vast period of time. Not only does research contribute to knowledge per se, but by clarifying and raising new issues, it provokes further research.

Therefore, it is essential to be familiar with developments which have taken place in the domain of our research. This will enable to benefit from vicarious experience, by selecting issues that are pertinent and avoiding the limitations and drawbacks which become clear only after the research has been conducted. In this chapter an attempt has been made to recapitulate the researches which have been conducted in the area. Since it is humanly impossible to list all the work that has been done, important milestone and major studies which provide perspective of the work are being put forward. Studies conducted during the last decades are being given special emphasis although important studies conducted earlier are also being referred to.

We will first examine studies relating to CHD and type-A behavior. The nomenclature of type-A and type-B has been used extensively in psychological literature. Type-A individuals, are described as extremely competitive and achievement oriented; they have a sense of time urgency, find it difficult to relax and become impatient and angry when confronted with delays or with people they view as incompetent.

Early studies provided compelling evidence of type-A behavior as a risk factor in CHD (Rosenman and others, 1975; Jenkins, Zyzanski & Rosenman and others, 1976; Friedman et al. 1973; Haynes and other, 1980; Byrne & Reinhart, 1989; Ghulam, Gupta, Bandyopadhyaya & Misra, 1990). Later on, when the concept of type-A behavior was more clearly understood and its specific components were identified, extremely interesting results were observed. It was found that not all components of
type-A behavior may be coronary prone, rather some like "engagement involvement" may even be protective (Keltikangas-Jervinen & Raikkenen, 1990). Global type-A behavior pattern was not found to be a risk factor for CHD whereas some specific components of type-A behavior like anger and hostility were found to be independent risk factors for CHD (Dembroski, MacDougall, Costa & Grandits, 1989).

Keltikangas-Jervinen & Raikkenen (1990) investigated type-A factors related to the level of somatic risk factors of coronary heart disease in 1,209 healthy adolescents and young adults. They have found that of the type-A factors, hard-driving had the strongest association with the somatic risk level. "Aggression competitiveness" was also related to the risk but not very strongly. Impatience was of no importance while "engagement-involvement" was likely to be protective factor.

In another study Weekes & Waterhouse (1991) compared 25 coronary heart disease (CHD) patients, 32 cancer patients, and 31 disease-free controls to determine if hostile attitudes (HAS) were unique features of CHD patients, and independent of the experience of serious illness. They found that HAS may have a subordinate causative role in the development of CHD and may contribute to coronary risk because of an association with more fundamental components of the coronary prone personality.

Dembroski, MacDougall, Costa & Grandits (1989) examined whether hostility (HOS) was associated with increased relative risk for coronary death and nonfatal myocardial infarction among 192 coronary heart disease (CHD) patients and 384 matched controls. Findings are consistent with evidence suggesting that HOS (especially an antagonistic, interactional style) but not global type-A behavior, is an independent risk factor for CHD. In another study Dembroski & Costa (1987) reviewed research into the relationship between type-A (coronary prone) behavior and its components and coronary heart disease (CHD). A relationship between potential for hostility (PH), a type-A component, and CHD has been suggested. However, PH also is likely to be a multidimensional construct.
Recent research elucidating factors in PH shows promise for further elucidating the nature of coronary prone behavior.

Pfiffner & Battig (1989) reviewed the literature on the psychophysiological aspects of the type-A behavior pattern (TABP) up to 1987. These studies, together with current state of epidemiological and coronary angiography studies, suggest that TABP is unsatisfactory in explaining the risk of coronary heart disease in coronary prone subjects. Hyperreactivity to challenges, whether of behavioral (e.g. anger-hostility, self-involvement, TABP) or constitutional origin (e.g. hypertension in parents) may be an indicator of pathogenic processes in the cardiovascular system.

In another study Helmers, Krantz, Howell, Klein and others (1993) assessed the relationships of hostility to the extent and severity of exercise induced cardiac ischemia and daily life ischemia in 63 male (mean age 62.4 years) and in 17 female (mean age 64.1 years) patients with coronary artery disease (CAD) who underwent exercise tomographic thallium testing to assess myocardial perfusion. A composite hostility score was positively correlated with severity of perfusion defects. This relationship was nonsignificant in the male subjects, but significant among women and among middle aged men. In an overlapping sample of 42 CAD patients with ischemia, after controlling for gender, both a hostility inventory and a composite hostility score were positively correlated with total minutes of ischemia. Results indicated that, in patients with CAD, hostility traits are significantly correlated with extent of daily life ischemia and with severity of exercise-induced myocardial ischemia.

Fukunishi, Hathori, Nakamura and Nakagawa (1995) examined the influence of narcissism and social desirability on hostility in 215 Japanese college students and 30 Japanese patients (aged 35-78 years) with myocardial infarction (MI). It was found that (1) MI patients had stronger cynical hostility and lower social desirability than college students; (2) hostility was related to narcissism before and after controlling for social desirability in both MI patients and college students; and (3) these
tendencies were stronger in MI patients than in college students, although there were no significant differences.

In another case control study Mendes de Leon (1992) examined the relationship between anger and impatience/irritability and acute coronary heart disease (CHD) in 40-65 years old white men (31 Ss with myocardial infarction (MI), 26 Ss with unstable angina pectoris (AP), and 26 hospital controls) of low socio-economic status (SES). MI was associated with anger-out and impatience/irritability, particularly in the subgroup of patients who did not have a previous MI. The same factors were associated with AP, but only when this acute ischemic event was not preceded by an MI. Results indicated that overt behavioral expression of anger is related to CHD in this particular group of patients.

In another study Krantz, Contrada, Hill and Friedler (1988) overviewed research on the behavioral antecedents of coronary heart disease. It has been found that stressful occupational settings characterized by high demands and low levels of control over the job are associated with increased coronary risk. They also reviewed the current status of the type-A behavior pattern with recent evidence suggesting that type-A may not be a potent risk factor in all populations. However, specific type-A components such as hostility as well as mode of anger expression (anger-in) have been related to coronary disease in recent studies. They have also concluded that physiologic responsiveness (reactivity) to emotional stress is the marker of processes in the development of cardiovascular disease. They have also found that stress, type-A components, and psychophysiological reactivity are promising candidates for research on clinical intervention. They have also realised, however, that further evidence is needed before stress and reactivity can be regarded as proven risk factors for coronary disease. In another study Wielgosz, Wielgosz, Biro, Nicholls and others (1988) found that acute myocardial infarction (AMI) subjects reported significantly lower levels of relaxation and income but higher levels of suppressed hostility and a higher incidence of hypercholesterolemia. Findings confirm the significance of hostility and
suppressed hostility as AMI risk factors and inadequate relaxation as an independent risk factor associated with AMI.

Matthews (1989) examined factors accounting for why women in most industrialized countries are protected from coronary heart disease (CHD). They had focused on the effects of female reproductive hormones (i.e. estrogens) on lipid and lipoprotein metabolism and blood pressure. Epidemiological studies that statistically adjust for sex difference in lipids, blood pressure, and smoking status cannot explain sex differences in CHD morbidity and mortality. Data also show elevated risk of myocardial infarction and stroke among women who use oral contraceptives. Men who are prescribed estrogens have elevated risk of CHD, and case control studies show that male CHD patients have elevated estradiol, compared to controls. Simple main-effect models of female protection from CHD are inadequate. Reproductive hormones are important determinants of protection from CHD, and behavioral characteristics can influence the effects of reproductive hormones on CHD risk factors.

A large number of studies have showed that CHD is the result of excessive stress. Studies related to stress and CHD are being presented.


Singh and Misra (1987) found that Myocardial infarction was significantly related to the number of stressful life events and suggest that myocardial infarction proneness involves poor coping skills.

Krantz & Raisen (1988) reviewed the literature on two areas of biobehavioral research: the effects of environmental stress and the role of psychophysiologic reactivity in the development of ischemic heart disease. They found the evidence that low socio-economic status (SES),
low social support and occupational settings characterized by high demands and low levels of control over the job are associated with increased coronary risk. On the basis of animal primate model of social stress, they explained the development of coronary atherosclerosis, and physiological responsiveness (reactivity) to emotional stress. In another literature review Pomerleau and Pomerleau (1989) found that psychological stressors (e.g. video games or mental arithmetic) can enhance adrenergic activity and sympathetic tone, setting into motion widespread physiological and biochemical perturbations, thus leading to CHD. They found the use of Nicotine in response to stress may exacerbate the potentially detrimental health consequences associated with chronic or excessive stimulation of the adrenomedullary system, particularly in susceptible or hyperactive person.

Siegrist, Peter, Junge & others (1990) developed a concept of work related socio-emotional distress which considers a mismatch between high workload and low control over occupational status (e.g. job insecurity, poor promotion prospects, status inconsistency) and related it to the occurrence of ischemic heart disease (IHD). Based on a 6.5 year prospective study of IHD incidence (n = 21) in 416 blue-collar men (25-55 years old), this concept was tested with logistic regression analysis. They found that status inconsistency, job insecurity, work pressure and “need for control” independently predict IHD occurrence after adjusting for major confounding somatic and behavioral coronary risk factors. Reviere & Eberstein (1992) based their study on longitudinal data from the US National Health and Nutrition Survey. Women who were unmarried, younger women and better educated women were at relatively low risk, whereas women who left the labor force and women who were homemakers were at relatively high risk for coronary heart conditions.

In a review of literature, Epstein & Perkins (1988) found that smoking is a major coronary heart disease risk factor that has proven to be very resistant to treatment efforts. For many smokers, environmental stressors, which has also been related to coronary heart disease risk, appear to
be strong determinants of smoking behavior and of relapse after cessation. This review focuses on the interrelation between stressors and smoking and on the potential impact of this interrelation on coronary heart disease risk beyond that which is due to stressors or to smoking alone. In another study Andrade Viera, Didier et al (1981) studied the link between life changes and the onset of illness in 63 male patients treated for their first heart attack. The life events covered by the scale (in the areas of living conditions, working conditions, marital situation, legal problems, psychosocial interactions, sex life, legal problems and crime) held different significance for Ss, depending on their psychosocial status. The onset of illness could not be attributed to any particular life change(s).

Eysenck (1991) argues that there is sufficient evidence to regard psychosocial variables (i.e. personality & stress) as important risk factors for cancer and coronary heart disease (CHD), equal in importance to smoking, heredity, cholesterol level, blood pressure, and other physical variables. Both types of factors act synergistically; that is, each by itself is relatively benign, but their effects multiply to produce high levels of disease. The evidence shows that psychological treatment can modify a person's reaction to stress, so that risk of cancer and CHD can be greatly diminished, and duration of survival significantly increased in those terminally ill with cancer. He says that psychological influences on physical diseases appear to be much greater than suspected in the past.

Dembroski and others (1984) described the development of research on type-A (coronary prone) behavior, and summarized his own research on predictors of heart disease. Investigations of the connections, among behavior, stress and heart disease have focussed on two main topics: finding the mechanisms that translate stress into heart disease, and identifying the behavioral traits that correlate with stress. Several preliminary studies suggest that physiological reactivity to challenge may be an important predictor of heart disease. Excessive physiological arousal (e.g. a sharp increase in blood pressure or in cardiac output) is known as "hot reactivity". Significantly, some individuals whose overt behavior identifies
them as type-A are not hot reactors, while other individuals who do not display type-A behavior are hot reactors.

Markowitz & Matthews (1991) reviewed clinical role of platelet activation in atherosclerosis and acute coronary events. Because epinephrine and possibly shear stress are clinically important activators of platelets, it is proposed that platelet reactivity to psychological stress may be a major mechanism in coronary events. The literature supports the hypothesis that platelet activity is increased by emotional stress.

Yeung, Ganz & Selwyn (1992-1993) reviewed studies to develop a new understanding of the pathogenic relationship between stress, coronary atherosclerosis (AR), and the clinical problem of the ischemia. It was found that myocardial ischemia caused by mental stress seem to involve disturbances of myocardial oxygen supply and demand. Mental stress produces increases in blood pressure and heart rate, thus raising the myocardial oxygen demand via the cardiac sympathetic system and the adrenal system. Stress also increases the total coronary vascular resistance in coronary artery disease patients. Investigators have discussed the mechanisms behind the decrease in blood supply in terms of dysfunction of vascular endothelium and biochemical changes in AR. They have also presented the contribution of mental stress to more pronounced vasoconstriction in AR and the effect of this response on endothelial vasodilator function.

A substantial amount of work has been done on hypertension. Essential hypertension is currently viewed as a heterogeneous condition brought on by many possible disturbances in the various systems of the body that are responsible for regulating blood pressure. Blood pressure may be elevated by increased cardiac output, the amount of blood leaving the left ventricle of the heart per minute; by increased resistance to the passage of blood through the arteries, that is, by vasoconstriction; and by an increase in the body's volume of fluids. The combination of physiological mechanisms that contribute to regulation of blood pressure
is extremely complex. The sympathetic nervous system, hormones, and salt and water metabolism, as well as central nervous system mechanisms, are all involved (Weiner, 1977). Many of these controlling physiological mechanisms can be affected by psychological stress.

Various stressful conditions have been examined to determine their role in the etiology of essential hypertension. Stressful interviews, natural disasters, anger and anxiety have been found to produce short-term elevations in blood pressure (Innes, Millar and Valentine, 1959; Ruskin, Board and Schaffer, 1948; Ax, 1953). Kasl and Cobb (1970) have examined the stressful effects of the loss of employment and its effect on blood pressure. They studied a group of workers beginning two months before their jobs were to be terminated and for two years subsequent to loss of employment. A control group consisting of men in similar occupations who did not lose their jobs, was examined for the same twenty-six months period. Each participant in the study was visited at home several times by a nurse during six separate periods so the blood pressure could be measured. For the control subjects there were no overall changes in blood pressure. In the men who lost their jobs, however, elevated blood pressure was found with anticipation of job loss, after termination of employment, and during the initial probationary period of a new job. Those who had great difficulty finding stable employment suffered the longest periods of high blood pressure.

Livingston (1987) argued that social and socio-psychological factors hold greater explanatory power for all phases of hypertension, including its etiology, pathogenesis, control and prevention. In a study conducted on Black Americans, they found socio-psychological stress to be the most dominant and important of all life-style factors in elucidating the disproportionately higher incidence of arterial blood pressure.

In another study Schnall, Pieper, Schwartz and others (1990) tested the hypothesis that job strain is a risk factor for hypertension and for increased left ventricular mass. Results confirm the hypothesis that job
strain increased the likelihood of being classified as having hypertension.

Blumenthal, Thyrum and Siegel (1995) found that high job strain was associated with elevated systolic blood pressure among women but not among men. High status occupations men and women showed significantly higher blood pressures during daily-life and during laboratory mental stress testing. It was also found that men with high job status had higher systolic blood pressure than low job status men. For women marital status was found to be an important moderating variable, with married women having higher ambulatory blood pressure than single women. During mental stress testing, married persons had higher systolic blood pressures than unmarried individuals.

In another study Fredrikson & Mathews (1990) found that essential hypertensives had large BP responses during all stressors, but especially during passive stressors not requiring a behavioral response. Borderline hypertensives had moderately large and more reliable BP and HR responses primarily during active stressors (ASTs). Normotensive offspring of HTNs had moderately large and reliable BP and HR responses to ASTs. According to investigators, excessive sympathetic nervous system activity during behavioral stress seems to have a pathophysiologic role in the development of hypertension.

In another study of marital discord and high blood pressure Ewart, Taylor, Kraemer & Agras (1991) examined the impact of normal family arguments on 24 females and 19 males (aged 32-73 years) with essential hypertension. It was found that as patients and their partners discussed their problems, their BP increased. But the causal pathways differed by sex. In women, hostile interaction and marital dissatisfaction were associated with increased BP; supportive or neutral exchanges were unrelated to BP. Whereas, in men BP fluctuations were related only to the patient's speech rate.

In another study Theorell, de Faire, Johnson and others (1991)
examined the role of job stress in the pathogenesis of hypertension. Ambulatory 24 hour recordings of blood pressure (BP) were made for 161 men (aged 35-55 years) with borderline hypertension. It was found that physical demands were of importance to both systolic and diastolic blood pressure levels during the round of daily life. Job strain was also important, but only to diastolic blood pressure and only at night and during work, not during leisure. They also found that a measure of job strain derived from the occupational classification appears to be useful in predicting variations in Diastolic BP level for men with borderline hypertension. In another study of job strain and hypertension, Albright, Winkleby, Regland, Fisher and others (1992) found that lower levels of job demands and job strain were associated with a higher prevalence of hypertension. It was also found that after 12 confounding variables were controlled for, the association between occupational stress and hypertension became nonsignificant.

Taras & Sallis (1992) assessed the generalizability of blood pressure (BP) reactivity across various stressors in young children (aged 3-6 years) to identify a stress response that could be suitable as an early marker for cardiovascular disease and hypertension. It was found that systolic blood pressure reactivity level was highest after physical exertion followed by competitive task and cognitive task. The 2-week test-retest reliability was higher for physical stress, systolic BP reactivity level than for baseline systolic BP and the other two stressors. The reliability of the systolic BP change score was significant only for physical stressors. Correlations among the three stressors ranged from 75 to 79 for systolic BP reactivity level and from 37 to 50 for change in systolic BP. It was found that change in systolic BP after physical stress correlated with skin-fold thickness. There was evidence of generalizability across stressors.

Anderson, Williams, Lane & others (1987) found that type-A behavior was associated with systolic and diastolic blood pressure hyperresponsivity during a structured interview (SI) but not during mental arithmetic. Certain speech components of the type-A pattern, as well as features of the
potential for hostility component, were also related to cardiovascular responses during the SI. And family history of hypertensive did not influence the cardiovascular parameters. Results also suggest that many of the cardiovascular response characteristics of the type-A pattern that have been observed in predominantly white samples also hold true for blacks.

In another study McCann & Mathews (1988) found that subjects with a hypertensive parent had larger diastolic blood pressure responses during all the three behavioural stressor studies than did subjects without hypertensive parents; this effect was particularly pronounced among the type As. It was also found that boys exhibited larger systolic blood pressure responses to all the tasks than did girls. Investigators have concluded that early signs of hostility may be related to psychophysiological responses thought to be pathophysiological mechanisms in the etiology of cardiovascular diseases.

Some investigators have based their studies in part on psychoanalytic theory and have tried to determine whether blood pressure elevation is associated with the inhibition of aggression (Hokanson and Burgess, 1962; Hokanson, Burgess, and Cohen, 1963; Hokanson, Willers, and Koropsak, 1968). The results of this classic series of investigations indicate that harassment of course causes blood pressure to rise but that, for males, aggressing against a source of frustration then helps blood pressure to decrease. With no opportunity to aggress against the frustrator, blood pressure is significantly slower to decrease after frustration. Only aggression directed at a low-status frustrator (college student) proved helpful in decreasing blood pressure, however, not that directed towards a high-status frustrator (visiting professor). These findings did not hold for female subjects.

A study by Harburg and others (1973) extends Hokanson’s ideas to the natural environment and to the high incidence of hypertension among black Americans. They found that blood pressure was higher among the
black males than among the whites; and blacks living in the high-stress area had higher blood pressure than blacks living in the middle-class neighborhoods. Thus previous statistics revealing racial differences in blood pressure were substantiated, but with the important reservation that environmental stress is also a major factor. When responses on the test were related to blood pressure, the following pattern emerged: for all subjects except blacks in the middle-class neighborhood, holding anger in and feeling guilt were related to higher blood pressure levels.

Johnson, Spielberger, Worden & Jacobs (1987) examined the relationship between blood pressure (BP) and personality and traditional risk factors in 219 black and 270 white males (aged 15-17 years). Several personality and traditional risk factors significantly predicted elevated blood pressure for both groups, but suppressed anger and weight were the major independent predictors. It was also found that familial factors were independent predictors of systolic and diastolic BP only for the white subjects. Findings indicated that adolescent males who were at increased risk for elevated systolic and diastolic blood pressure could be identified by how often angry feelings were suppressed. In another study Boutelle, Epstein & Ruddy (1987) have found that hypertensives had significantly higher scores than normotensives on scales of autonomic arousal, proneness to anger, and guilt or avoidance of the expression of anger. The two groups were almost identical on scales of depressive feelings and defensiveness. The findings are consistent with the hypothesis that hypertension can result from the inhibition of feelings of anger.

Johnson, Schork & Spielberger (1987) examined the relationship between measures of the experience of anger and anxiety, traditional risk factors and blood pressure (BP) in 171 black and 279 white female 15-17 years old. Results showed that whereas a number of the personality and traditional risk factors were significantly correlated with BP, body mass was the most consistent predictor. It was also found that blood pressure was significantly higher for subjects who frequently harbored grudges and held in their angry feelings. In another study Johnson (1989) found that
emotional factors, particularly suppressed anger, were significantly related to elevated blood pressure for both black and white adolescents. Results also showed that black experienced anxiety more frequently and more intensely and experienced a greater intensity of state-anger reactions. Whereas for white adolescents traditional risk factors were stronger predictors of blood pressure.

Goldstein, Edelberg, Meier & Davis (1988) studied 21 male and 24 female nonmedicated subjects on anger, which they experienced at home and at work and extent to which others were aware of their anger and the extent to which anger had been expressed in their families. Subjects were then physiologically monitored during a 2-min relaxation period. It was found that expressed anger was inversely related to systolic blood pressure (SBP) and diastolic blood pressure (DBP), while family expressed anger was inversely related to SBP only. When subjects were divided into normotensives and hypertensives, normotensives showed significant associations between experienced anger and SBP, expressed anger and DBP, and family expressed anger and SBP. Both sexes showed a significant association between expressed anger and DBP; only females showed this relationship with SBP. In another study Sommers-Flanagan & Greenberg (1989) reviewed scientific literature between 1979 and 1986 concerning the link between psychosocial variables and elevations in blood pressure. Strong support is found for an association between hypertension and such psychosocial factors as the identification and expression of anger, the use of inhibiting defense mechanisms (denial and repression) and interpersonal anxiety. It is suggested that these findings cannot be attributed to artifacts. Such as limited measurement of blood pressure levels or patient’s awareness of their hypertensive status. The need for psychosocial interventions for hypertension is noted.

Desheilds, Jenkins & Tait (1989) compared 19 hypertensive patients to a chronic disease control group of 12 diabetics and 11 nonpatient controls on measures of anger expression, assertiveness and perceptions of health status. All subjects were 50-70 years old. The chronic patient
groups differed significantly from the nonpatient controls in reporting more anger in general, greater frequency of anger experiences, and a tendency to express anger more outwardly. The chronic patient group also reported greater severity of health problems. Comparison between the hypertensives and diabetics revealed a greater level of current anger among diabetics. Investigators are of the opinion that psychological distress may be a natural consequence of a disruptive medical condition rather than the cause of it.

Jorgensen & Houston (1988) found that diastolic blood pressure reactivity was associated with not overtly expressing hostility. For persons with a parental history of hypertension, systolic blood pressure (SBP) reactivity was associated with low scores on the covert hostility measure of irritability, while for persons without a parental history of hypertension, SBP reactivity was associated with high scores for irritability. In another study of anger-coping styles and blood pressure, Harburg, Gleiberanan, Russell & Cooper (1991) found that older black males with a high reflective mode of anger (constrain anger and try to solve the problem) had significantly lower BP than those with low scores on this mode. In contrast, older Black subjects with a higher anger-out mode (impulsive anger strongly expressed) had significantly higher BP than those low on this mode. This anger-out pattern was also found for older white men; but for younger whites only higher alcohol and lower education were significantly related to higher BP levels. An anger-in mode (impulsive anger not expressed) was not significantly related to BP.

Ekeberg, Kjeldsen, Eide & Leren (1990) conducted a study on 17 hypertensive and 18 normotensive 50 year old men. It was found that hypertensives had fewer siblings, fewer sons, less education, lower income and more type-A behavior patterns. It was also found that 11 hypertensives and 5 normotensives had experienced considerable traumas in childhood (e.g. death of a parent, psychotic parent, separation from one or both parents, or beating by an alcoholic father).
Cottington, Matthews, Talbott & Kuller (1986) found that HT was more strongly associated with self-reports of an uncertain job future and dissatisfaction with co-workers and promotions among subjects who suppressed their anger than among subjects who did not habitually suppress their anger. The interactions between suppressed anger and job stress significantly predicted HT status, controlling for age, body mass index, smoking, alcohol consumption and family history of HT.

In another study Rosenman (1986) examined evidence that relates the sympathetic nervous systems to hypertension and hostility/anger dimensions to the pathogenesis of hypertension, coronary disease (CHD), and its complications. The findings relate type-A (coronary prone) behavior to enhanced coronary artery disease and increased incidence of clinical CHD as a consequence of enhanced or central noradrenergic secretion. It is argued that aggression, hostility, anger dimensions are affected by evolutionary gender differences in limbic system anatomy, but socialization, conditioning and learning from infancy play an important role. It was also found that neurochemical pathology shows a chemistry of emotions that can be manipulated by opening or closing brain receptor binding sites. Investigators considered anger management as an appropriate therapeutic intervention in the treatment of hypertension and the prevention of CHD.

In a review of literature Niaura & Goldstein (1992) found the effect of socio-cultural and interpersonal factors on CAD and hypertension. Several studies have also demonstrated a connection between CAD, sudden death and acute disturbing life events.

In an extensive review of literature Feshbach (1986) found that suppressed anger was linked to elevation in blood pressure, while frequently expressed anger was linking to coronary malfunctioning. However, the absence of anger is seen as having negative psychological consequences. Anger in moderation appears to be the ideal psychosomatic resolution.
The researches cited above suggest the need for further studies in the area. There is strong evidence that a role is played by stress as well as anger in CHD and hypertension. But the nature of the influence is not very clear. Although it has been found in a majority of studies that anger is an important predictor of CHD in fact a much more important predictor than global type-A behavior, the evidence about role of expressed and suppressed anger is conflicting. Some studies link CHD with expressed anger while others link it with suppressed anger. About the role of anger in HT, majority of studies show that anger-in is associated with HT, although a few studies reveal the role of anger-out in HT. Undoubtedly, stress has found to play an important role in both diseases but it would be meaningful to find out whether the disease group differ in terms of stress and in terms of positive and negative events perceived by them. The investigator therefore proposes the following hypotheses:

1. Coronary heart disease group differs from HT group on (a) anger-total, (b) anger-out, (c) anger-in and (d) anger control dimensions.

2. Coronary heart disease group differs from CHD + HT group on (a) anger-total, (b) anger-out, (c) anger-in, and (d) anger control dimensions.

3. Coronary heart disease group differs from disease free group on (a) anger-total, (b) anger-out, (c) anger-in, and (d) anger-control dimensions.

4. HT group differs from CHD + HT groups on (a) anger-total, (b) anger-out, (c) anger-in, and (d) anger-control dimensions.

5. HT group differs from disease free group on (a) anger-total, (b) anger-out, (c) anger-in, and (d) anger-control dimensions.

6. CHD + HT group differs from disease free group on (a) anger-total, (b) anger-out, (c) anger-in, and (d) anger-control dimensions.
7. Coronary heart disease group differs from HT group on stress scores.

8. Coronary heart disease group differs from CHD + HT group on stress scores.

9. Coronary heart disease group differs from disease free group on stress scores.

10. HT group differs from CHD + HT group on stress scores.

11. HT group differs from disease free group on stress scores.

12. CHD + HT group differs from disease free group on stress scores.

13. Coronary heart disease group differs from HT group on number of positive stressful life events.

14. Coronary heart disease group differs from CHD + HT group on number of positive stressful life events.

15. Coronary heart disease group differs from disease free group on number of positive stressful life events.

16. HT group differs from CHD + HT group on number of positive stressful life events.

17. HT group differs from disease-free group on number of positive stressful life events.

18. CHD + HT group differs from disease free group on number of positive stressful life events.

19. Males of various disease groups differ from each other in number of positive events perceived, (a) CHD and HT, (b) CHD and combined
disease (CHD + HT), (c) CHD and disease free (d) HT and combined disease (e) HT and disease free, and (f) combined disease and disease free.

20. Females of various disease groups differ from each other in number of positive events perceived, (a) CHD and HT, (b) CHD and combined disease (CHD + HT), (c) CHD and disease free, (d) HT and combined disease, (e) HT and disease free (f) combined disease and disease free.

21. Males of various groups differ from females groups in positive events perceived in (a) CHD group, (b) HT group, (c) combined disease group, and (d) disease free.