CHAPTER- III

REVIEW OF RELATED LITERATURE

In the recent years it has become increasingly popular to view health on a continuum ranging from illness to wellness. Psychological Stress and emotional adjustment are associated with changes in symptom complaints, disability and physiological indices of disease over the course of illness (Smith & Gallo, 1994). Considerable research has shown that personal or life-related stressors and emotional vital signs like anxiety, anger, depression have a damaging effect on health and well-being. Hypertension presents the largest risk for cardiovascular disease and has been a major focus of biomedical and psychosomatic research for decades.

3.1 Prevalence of Cardiovascular Disorders/Essential Hypertension

Coronary heart disease prevalence rates have more then doubled in the last thirty years in the developing Asian countries and the Urban population of India, who are being exposed to stress of acculturation and modernization (Gupta, 1997). Meta-analysis shows that as compared to 1960's the prevalence of coronary heart disease (CHD) has increased nine times in urban subjects.

Higher CHD incidence and mortality has been reported in migrant Indians to England, North America, Singapore and South Africa in comparison to local populations (McKeigue, 1992; Enas & Mehta, 1995). CHD prevalence in first generation emigrants to Britain is almost twice of native Europeans (Enas & Mehta, 1995; McKeigue,
Shah & Marmot, 1991), whereas among the second-generation emigrants Indians prevalence is similar to native Europeans (Williams, 1995).

Bhatnagar et al., (1995) compared the coronary risk factors in a group of Punjabi Asian migrants settled in West London with their siblings living in Punjab, India. Results state that Asians are genetically predisposed to atherothrombotic risk factors, which migration and westernization unmask and potentiate.

However, Dhawan and Bray (1994) report no significant differences in risk profiles or extent of coronary disease between Asian men, settled in Britain and an age matched population living in India. None-the-less, since prevalence rates of CHD in India vary greatly depending on location (Chadha et al., 1990), socioeconomic status (Gupta et al., 1994), comparison between British Asians with Indian Asians is difficult.

Epstein (1996) reported higher coronary heart disease prevalence in Chinese emigrants to South Asian Countries and Japanese emigrants to the US when compared to matched groups of native populations. Hughes et al., (1989) report that Asian men were significantly younger at the time of their infarcts, had more extensive atheroma and lower mean cholesterol concentrations than white men.

Balarajan et al., (1984) and Marmot (1992) state that mortality from CHD increased by 8% in Asian men and 14% in Asian women from 1970 to 1985 while overall mortality fell in all Western European countries. Beckles et al., (1986), in a prospective study of a community including both men and women aged 35-69 years living in Port-of Spain, Trinidad reported that by comparison with adults of African descent, age-adjusted relative risks of death from all causes and from
cardiovascular diseases well significantly increased in those of Indian origin (1.5 and 2.6 respectively) and reduced in those of mixed descent (0.5 & 0.3 respectively). Adults of European descent had an all-cause and cardiovascular mortality relative risk of 0.8 and 2.0 respectively. These ethnic differences, however, were accounted for by the high prevalence of diabetes in Indians (19%).

In an earlier study by Lipowski (1980), the magnitude of essential hypertension (EH) in various parts of the world and in different populations show marked differences. Higher prevalence rates were found in population undergoing rapid socio-cultural changes, urbanization and socio-cultural morbidity.

Essential hypertension in the US Black population is among the highest of any group in the world (Akinkugbe, 1985) and US Blacks have been found to experience correspondingly higher rates of hypertension related morbidity and mortality from coronary diseases (Myers, 1986; Dimsdale, Pierce, Schoenfeld, Brown, Zusman and Graham, 1986).

Prevalence studies in India from 1949 to 1995 report that in urban adult populations hypertension prevalence was 1.24 in Calcutta (1949), 4.24% in Kanpur (1954), 3.03% in Bombay (1959), 14.08% in Ludhiana (1985) and 10.99% in Jaipur (1995). In rural subjects hypertension prevalence increased from 1.99% (Delhi) and 0.52% (Bombay) in 1959 to 4.02% (Maharashtra in 1993 and 7.08% (Rajasthan) in 1995. Increase in prevalence rate has been higher in the Urban areas while it has not been as steep in rural areas (Gupta & Gupta, 1996).

However, several recent studies have reported a higher increase in rural populations. Kumar and Chaudhary (1991) found an overall prevalence of hypertension of 3.8% in a rural community of Western
Rajasthan. Khurana, Gulati, Prakash and Wander (1990), found 13.8% prevalence of hypertension in the rural community of Punjab. The most obvious reason being that most of the rural patients are left either undiagnosed or untreated. A prevalence rate of 5.4% in the rural community at Dayalpur (Haryana) was reported by Wasir, Ganai and Nath (1983) with higher prevalence in the females 7.5% as against 3.2% in males. A 5-year follow up among adults in the age group 25 to 64 years in a rural population of Gurgaon district (Haryana) by Gopinath et al., (1995) reports that the annual incidence of HT was 2.8/1000 (male : 3.8/1000 and female : 2.4/1000). Subjects also reported a family history of HT in 10.5% and obesity in 42.1%.

3.2 Sociodemographic factors in Cardiovascular disorders.

Sociodemographic factors play a significant role in the onset and exacerbation of cardiovascular disorders. Strachan, Leon and Dodgeon (1995) investigated the extent to which geographical variations in mortality from IHD and stroke were influenced by factors in early life or in adult-hood in Britain. Findings state that geographical variations in mortality from CVD in Britain may partly be determined by genetic factors, environmental exposure or life style acquired early in life.

Age has been found to be one of the non-modifiable risk-factors for CAD and the peak incidence is attained between 51-60 years. Recent findings report that at any level of risk factors, age could increase the incidence of cardiovascular events (Wenger, 1997). In a recent study, Krishnaswami (1998) hypothesized that certain risk factor combinations resulted in an acute coronary event at a fixed age pattern. In a subgroup analysis of 1210 patients with CAD who had selective coronary arteriography and had their first manifestation at acute MI, he
reported that when smoking and/or positive family history were risk factors, acute MI occurred at a mean age of $44 \pm 6$ years. While diabetes and/or hypertension led to acute MI at a mean age of $54 \pm 4$ years.

Rose, Sivik and Delimar (1996) in a study of gender, psychological well-being and somatic cardiovascular risk factors explored the associations between cardiovascular risk factors and subjective experiences of psychological well-being with special reference to gender related differences. In both men and women subjective experience of psycho physiological well-being was significantly correlated with cardiovascular risk factors. Estimation of general health, anxiety, self-control and vitality were significantly related with cardiovascular risk factors in men while more complex significant relationships between the sets of variables were found for women. Moreover the direction of the correlations differed between the genders. In India data on premature CAD is meagre and most of the study groups consist of small cohorts in different regions of India. (See Kaul et al., 1986; Garg et al., 1987; Vyas et al., 1994).

3.2.1 Sociodemographic factors and Coronary Heart Disease (CHD)

Zodpey, Kulkarni, Vasudeo and Chaubey (1994), in a pair-matched case-control study of 154 cases and 154 age and sex matched controls examined socio-economic status, physical activity, family history of CHD, TABP, cigarette smoking, alcohol consumption, body mass index, diabetes mellitus, hypertension, total serum cholesterol and oral contraceptive use (in women) for association with CHD. The additive risk scoring system based on the results of conditional multiple logistic regression identified socio-economic status, physical
activity, diabetes mellitus, hypertension and total serum cholesterol to be related with increased risk of CHD.

The effect of BP on cardiovascular mortality and morbidity has been well documented as has the interaction of BP and Cholesterol levels. The interaction of hypertension (HT) and blood lipids has also been suggested as potential explanation for the lack of continued decline in CHD mortality (Chobanian, 1988). A review of literature published (1980-1994) shows that a woman particularly at a risk for developing CHD hails from a low social class, has little educational attainment, indulges in smoking, little physical exercise and experiences increased stress. Women seem to be at higher risk of post MI, depression and psychosocial impairment than men in reference to adjustment to CHD (Brezinka and Kittel, 1996).

Kop (1997), provides evidence that age-dependent associations between psychosocial factors and risk of cardiac events is at least in part mediated through the severity of underlying CHD. Importance of both chronic psycho-social factors (e.g. low socioeconomic status and/or high hostility) and episodic psychological distress syndromes, such as vital exhaustion and depression in the onset of acute coronary syndromes is also stated.

3.2.2 Sociodemographic factors and Essential Hypertension/ elevated Blood Pressure.

The pathogenesis of essential hypertension (EH) is determined by various psycho-social and demographical variables, Smith and Galo (1994) suggests that psychological stress and emotional adjustment are associated with changes in symptom complaints, disability and physiological indices of disease over the course of illness.
Black people have constantly shown a disproportionate risk for hypertension and the morbidity, mortality, and sequelae of this disease (Myers & McClure, 1993) because blacks are more likely to experience chronic socio-cultural stressors which are thought to interact with psychological risk factors (e.g. suppressed anger, anxiety, and depression). A combination of chronic socio-cultural stress and a psychological risk as suppressed anger may induce greater pathophysiologic activity related to EH than either factor alone.

Additionally, Brown (1997) showed (i) that EH is a polygenic disease (ii) provided evidence for different types of EH, and (iii) demonstrated that the families with multiple affected siblings are likely to have a single gene, conferring most of the risk of hypertension. Findings report that the pathogenesis of hypertension is programmed during the period of rapid growth in early life (Osmanand et al., 1994; & Fall et al., 1995). BP tracks from birth, but by the time hypertension is diagnosed clinically it is already established and its too late to prevent structural changes in the heart and blood vessels that are responsible for the complications of hypertension and for the irreversibility of hypertension itself.

In a study Carmelli, Dorit, Swan and Rosenman (1986) examined the association between a spouse’s characteristics and his/her partner’s BP and the combined effect of parental characteristics on the BP levels of offsprings in 520 subjects from 126 families recruited from the Western Collaborative Group Study (WCGS). Results indicate a differential pattern of cross-spouse and cross-family associations for parents and offsprings in their families. Higher scores on the Thurstone Temperament Schedule (TTS) were associated with increased levels of BP in males and decreased levels of BP in females.
The findings from the cross-family association analysis are contrasted with the separate patterns of familial correlation of BP and personality characteristics (see also Semenchuk & Larkin, 1993; Trieber et al., 1994).

The relationship between hypertension and various demographical factors have been reported by researchers in Indian settings. Wasir, Ganai and Nath (1983) reported a higher prevalence of hypertension in females, 7.5% against 3.2% in males. Rise in BP was directly proportionate to age, body weight, socio-economic status and smoking habits and inversely related to physical activity. Likewise, Baldwa (1984) stated that the prevalence of hypertension was higher in females below the age of 50 years while it was lesser after the age of 50 years as compared to males. Recently, Bharani, Joshi and Singh (1990) investigated the prevalence of hypertension amongst the employees of M.J. Hospital, Indore and their adult family members. They reported a higher prevalence amongst males (14.2% Vs 10.48%) and a rise with advancing age. A mild increase in DBP (95 to 104 mm Hg) in 66.6%, moderate (105-115 mm Hg) in 26.4% and severe (DBP < 116 mm Hg) in 7% and was detected first time in 66.7% subjects. Large majority of hypertensives had no symptoms (58%) or mild symptoms (42%). Hypertension was more prevalent in the upper socio-economic class and the associated diseases included IHD in 9%, diabetes mellitus in 4.4% and obesity in 30.8%. However, no significant differences were evident between hypertensives and normotensives with regard to smoking, tobacco, chewing and alcohol consumption.

The prevalence of hypertension has been reported to increase with age in a linear fashion (see Dash et al., 1986; Kumar & Chaudhary, 1991; Johnson, Reid & Vogt, 1994; and Toumisto, 1997).
Recently, Shashikala et al., (1996) studied the prevalence of HT among the elderly and the various factors having a bearing or influencing EH among 809 elderly subjects from four slums with a total population of 19,133. A structured pre-tested questionnaire was used and BP was measured by a mercury sphygmomanometer according to WHO (1978 & 1993) guidelines. HT prevalence reported was 27% and isolated systolic HT was 17%. The variables significantly associated with HT in univariate analysis were stress, anxiety, depression, obesity, light activity, unemployment, low income, spouseless persons and religion. Those with isolated systolic HT were gender (females), occupation, religion, obesity light physical activity, stress, anxiety, depression, smoking and chronic tobacco chewing. Gender (male/female) habits (smoking, alcoholism) and psycho-social variables (stress, anxiety & depression) were inter-related and grouped to form a "Factor" among hypertensives in a multivariate analysis (factor analysis technique). However, habits (smoking, alcoholism) alone formed a 'Factor' among isolated systolic hypertensives. HT is stated to be lifestyle disease and management of the same relies on physical, mental and social health. Earlier Gupta, Majumdar, Gupta and Gupta (1994) determine age-specific BP trends and prevalence of HT among 2112 subjects (1415 males and 797 females) from Jaipur aged more than 20 years. The US Fifth Joint National Committee (JNC-V) and WHO guidelines were used to determine the status of being a hypertensive. A significant rising trend of BP with each decade of age in both males (ANOVA : SBP = F = 42.4; P < 0.001; DBP = F = 9.7; P < 0.001) and females (SBP = F = 39.3, P < 0.001; DBP = F = 26.7, P < 0.001) was present. According to the JNC-V criteria (BP > 140/90 mm Hg or past diagnosis) the overall prevalence of HT was 29.5% in males and
33.5% in females. Using the WHO criteria (BP > 160/95 mm Hg, or past diagnosis) the prevalence was 11.3% in males and 12.2% in females. There was a significant age related increase in the prevalence of HT in both males.

Sharma, Arora, Bansal, Sagar and Khurana (1985) in a study of industrial workers (n = 3340), executives and professionals (n = 1008) of Ludhiana, India reported that the prevalence of hypertension showed an upward trend with increasing age, body weight and alcohol consumption. Also SBP and DBP correlated positively with increasing age, body weight and economic status.

A Survey in four areas of three different geographical regions of Nepal by Pandey (1984) reports highest prevalence rate of hypertension in an urban community of hill region (9.89%) followed by rural plains (8.11%) rural hill region (5.30%). The distribution of BP in each quartile was also higher in urban hill and plain regions as compared to the two other areas. Also prevalence of hypertension positively correlated with salt intake (more than 5 mg. per day), obesity sedentary habit and alcohol consumption.

Das Gupta (1984), reported that mean SBP and DBP as well as prevalence of HT were not significantly different from one another in rural areas at 13,000 ft., 10,000 ft., and 4,000 ft. altitude. A similarity with the North Indian Plains was also reported. BP and prevalence of HT at Shimla (7,000 ft., Urban) was significantly higher than the rural areas irrespective of the altitudes eliciting that it is the way of life further than the altitude which influences the BP. Similar findings were obtained by Kaushal (1994) reporting highest prevalence of HT at Shimla (11%) and an overall prevalence of 7.6% (7.3% in men and 7.9% in women). A weak to significant positive correlation
between BP and altitude were found. A positive correlation between age and HT was also observed.

Cross sectional surveys in Moradabad (N. India) and Trivandrum City (S. India) were also conducted on individuals randomly selected in the age group 25-64 years in both males and females. EH was significantly highest in South Indians, compared to North Indians respectively. Slightly higher prevalence in males than in females was found in the younger age groups, however, the prevalence rates and BP levels increased with age in both North and South Indians irrespective of gender. Multivariate analysis revealed strong and independent associations of age, higher body mass index, central obesity and social class with hypertension in both sexes in both the regions. Higher dietary fat intake and lower physical activity were weakly but significantly associated with hypertension (Singh, Beegom & Naiz, 1996).

Such a review suggests that discussions of psychological correlates of EH need to take into account socio-economic factors such as sample characteristics (race, gender, age, awareness of diagnostic status and socio-economic status) and methods/criteria of EH.

3.3 Life Stress and Cardiovascular Disorders.

A large body of literature demonstrates that personal or life-related stressors such as life-change events are a major source of stress, and can have deleterious effects on health and well-being (e.g. Dohrenwend & Shrout, 1985; Cowen & Cowen, 1988). Research on life stressors thus has occupied a central place in social psychological investigations of psychological and somatic health (Thoits 1983). Stress plays some role in the developing of every disease; and its effect for
better or worse are added to the specific changes and characteristics of the disease in question (Cooper, 1983).

A host of studies have suggested a positive relationship between stressful life events and illness (Chan, 1986; Jandrof et al., 1986; Coyne & Downey, 1993). Pearlstone, Russell and Wells (1994) reported that life events were related to psychosomatic symptoms. Mallinckrodt, Leong and Kralj (1989), earlier, in a comparative study of 92 male and 74 female graduates on measures of psychological stress symptoms and physical health complaints associated with different types of life-change events found women to report significantly more psychological symptoms of stress while men reported fewer life changes. While men elicited concern about debt and economic difficulties, women were concerned with demands of outside job. Tenison (1988) examined the relationship among major life events and daily hassles in physical illness in female and male graduate and upper division university students. It was found that while major life events were significantly related to illness for women, daily hassles were more strongly related to men's physical symptoms. However, both life events and daily hassles were equivalently related to physical symptoms of the women. Further, life event stress has been found to play a significant role in disorder such as asthma (Goreczny et al., 1988; Ghosh, 1992), cancer (Bandopadhyay et al., 1986; Srivastva & Broota, 1987), manic depressive psychosis (Elisabeth, 1984), & neurosis (Gautam & Kamal, 1990).

Life stress plays an important and significant role in the onset and progression of cardiovascular disorders (Jorgensen & Houston, 1989; Fisher, 1996) Elliott (1995) reported a link between psychosocial stress and coronary disease specially in women. He states that given the dichotomy between actual and perceived etiologic links, the
relationship between psychosocial stress and heart disease may depend on the meaning of the situation to the individual and the way she perceives her life situation. Further, females tend to adapt to stress better, but spend more energy complaining of discomfort (Milyaeva, Khaliulin and Barbarash (1995)).

Further, Forest (1996) states that while women experienced a persistent influence from distal events, men were more responsive to recent conditions in terms of global happiness. Overall, there is support that men and women derive their sense of well-being through different pathways. Researchers have also argued in favour of considering recent life events over distal ones as well as gender in research on health/illness (see Suh, Diener & Fujita, 1996). Most recently, Perez, Lopez, Frances, Mendez and Aleman et al., (1998) reported the possible impact of multiple and recent major stressful life events on the development of acute myocardial infarction (MI). MI males experienced more stressful life events in the past one year when compared to the controls.

3.3.1 Life Stress and Coronary Heart Disease (CHD)

Recent research has shown that moderate to severe stress can lead to the development of psychopathology (Coyne & Downey, 1993) and possibly even to increased mortality from CHD (Grossarth-Maticek, Eysenck and Boyle, 1994).

Low, Thoresen, Pattillo and Flieschman (1993) studied the causal attributions and CHD in women. Personal behaviour, stress, luck, family history and martial problems provided for causal attributions.

Bosma, Peter, Siegrist and Marmot (1998) examined the association between two alternative job stress models, effort-reward
balance model and job strain model and risk of CHD among male and female British civil servants. Baseline measures of both job stress models were related to new reports of CHD over a mean 5.3 years of follow-up. The imbalance between personal efforts (competitiveness, work-related overcommitment and hostility) and rewards (poor promotion prospects and a blocked career) was associated with a 2.15 fold higher risk of new CHD. Job strain and high job demands were not related to CHD, however, low job control was strongly associated with new disease.

3.3.2 Life Stress and Essential Hypertension / Elevated Blood Pressure.

The "reactivity hypothesis" states that cardiovascular reactions to episodic psychological stress are involved in the aetiology of essential hypertension (EH) (Light, 1981; Obrist, 1981; Bhargava, Sharma & Agarwal, 1982; and Manuck, et al., 1990). In a survey on female doctors Heim (1991) reported stress to be responsible for health risk with an alarming 10 years lower life expectancy than in general population. Myers, Bastien and Miles (1983) in a study of 191 black University students found that experiences of stress, characteristic patterns of reactions to stress, present health status and family history combined in complex ways to account for differences in SBP/ and DBP. The variables also combined differently in men and women. Variations in normal BP were related to feelings of being under pressure and the experience of distress particularly when these feelings occurred concurrently or resulted from life change stresses. The pathogenic impact of lifestresses on BP was greater for males than for females suggesting a greater male vulnerability to socioecological
stresses. Results support the view that psychological stress factors play an important but not a prime role in the etiology of essential hypertension. Reports state that persons at risk for hypertension (HT) may show elevated BP at rest and during mental stress. Lovallo and Al’ Absi (1998) using parental hypertension history and resting SBP on two screenings identified 21 men at high risk and 21 men at low risk for HT. They examined whether high risk BP elevations reflected greater vascular resistance or cardiac output on a day of extended rest versus a day with prolonged arithmetic and RT tasks. Results reported that high risk men had raised SBP/DBPs and higher vascular resistance with minimal differences in HR and cardiac output.

Uchino, Cacioppo, Malarkey and Glaser (1995) examined the potential mechanism coordinating individual differences in cardiovascular responses to acute psychological stress among healthy subjects who performed a mental arithmetic challenge. Their cardiovascular, endocrine and immune functions were recorded and it was revealed that the acute stressor was associated with changes in them. More important analysis revealed that individual differences in cardiovascular reactivity predicted stress-induced cortisol changes as well as that distinct physiological pathways are activated in response to acute psychological stress. Recently Kamarck, Shiffman, Smithline, Goodie et al., (1998) studied the acute effects of psychosocial processes on cardiovascular activity during daily life by random effects regression. Subjects were healthy adults (n = 120) and were monitored over a 6 day period with ambulatory blood pressure monitors and computer-assisted self-report assessments. Social conflict, task strain, emotional activation, activity posture and other covariates were rated following each ambulatory BP measurement. BP and HR were reported
to be elevated during periods of emotional activation (high negative affect or high arousal). DBP was lower during periods involving high decisional control and HR was lower during high-control, low-demand activities. Substantial individual differences were evident in the effects of psychosocial influences on ambulatory cardiovascular activity. Thus, results state that psychological factors are reliable determinants of ambulatory BP, which may account for the unique predictive value of ambulatory BP.

Shah (1980) studied the impact of stress on 80 officers from cooperative Banks, marketing and consumer society, industrial society and cooperative departments. Hypertension was among the other physiological changes felt by the officers.

Niedhanmer, Goldberg, Lectere, David et al., (1998) examined the cross-sectional relations between psychosocial work variables of psychological demands, decision latitude and social support at work and cardiovascular risk factors of hypertension, hyperlipidemia, diabetes, overweight, smoking and alcohol consumption. Questionnaire data from 13,226,42 to 56 year old male and female French Company employees elicited that psychosocial work aspects and cardiovascular risk factors were different for men and women. Results state that in men, low decision latitude was associated with HT and alcohol consumption while in females with hyperlipidemia.

Several studies have demonstrated an association between psychosocial stressors at work (i.e., work load, conflict with employers) and health-related problems (i.e., cardiovascular disease) Kua, Tian, Lai & Kio, 1989; Lam, Lee, Ong, Wong, Chow & Kleevens, 1987; Makowiec-Dabrowska & Bortkiewiez, 1990). In a multiple regression analysis examining the relationship of job decision latitude, total
psychological stressors, physical exertion and work hours to two dependent variables DBP and serum cholesterol. Sorenson, Lewis and Bishop (1996) reported that physical exertion was related to higher BP levels in women but to lower levels in men.

Curtis, James, Raghunathan and Alcser (1997) examined the association of job strain (or its components, decision latitude and job demands) with elevated BP in 726 African-American adults. Job strain was not associated with BP in both men and women, however, decision latitude proved to be important for hypertension risk among men.

Blumenthal, Thyrum and Siegel (1995) evaluated effects of stress (e.g. job strains, occupational status and marital status) on BP in 99 adults with hypertension. BP was found to be higher at work than at home during daily life. Both men and women with high status occupations had significantly higher blood pressures (BP) during daily life and also during laboratory mental stress testing. High job strain was associated with elevated SBP among women but not among men. However, SBP was comparatively higher in high job status than in low job status men. Both men and women who were married reported elevated BP (SBP) than their unmarried counterparts. Carroll, Smith, Sheffield and Shipley et al., 1997), reported that in men the magnitude of SBP but not DBP changes to stress was positively associated with occupational grade: the higher the grade, the greater the reactions.

Patel (1994) studied psychosocial risk factors for cardiovascular disease. He observed that, especially men with lower employment status reported more stressful events, had a greater likelihood of smoking, were obese, having diabetes and required treatment for hypertension and elevated BP. In addition they significantly scored higher on hostility and anger than the controls.
In another recent study the relationship between BP and self-reports of physical symptoms and job-related problems was investigated among 262 male and female teachers by Nyklicek et al., (1997). Three groups: treated hypertensives (THT), untreated hypertensives (UHT) and normotensives (NT) were taken. The groups differed significantly with respect to reported physical symptoms, with THT reporting the most and UHT the fewest symptoms. A multiple regression analysis revealed an inverse association between DBP and the number of reported physical symptoms in untreated subjects. A similar trend for SBP did not reach significance. Also, no significant results with respect to work-related problems were obtained, except for a group x gender interaction on job-related irritation. Gender effect was seen as male THT showed the lowest irritation scores whereas the female THT showed the highest irritation scores. Recently Gump and Matthews (1998) investigated the role of sustained search for potential threat in acute cardiovascular stress responses. Male participants (n = 30) in the laboratory were randomly assigned to search on a computer screen for statements that were negative, positive or occurring at known intervals. Relative to participants in the other search conditions, participants in the negative search condition had significantly highest systolic blood pressure (SBP) and diastolic blood pressure (DBP) responses during subsequent stressors.

In another recent study Seibt, Brucsein and Schcuch (1998) compared traditional stress setting and a stress setting of varying task demand and decision latitude with respect to their feasibility to elicit differences in cardiovascular reactivity and recovery in 20 normotensive, 20 borderline hypertensives and 20 non-medicated hypertensives. Also the relationship between laboratory and everyday
BP was investigated. No significant differences among compared groups emerged in their reactivity to the various mental stressors, however, marked differences in their behaviour occurred during the 10 minutes of recovery following each stress situation. Both SBP and DBP in the hypertensives failed to recover during this period. Results also point out a possible role of recovery processes after stress in the development of essential hypertension (EH). Similar findings were reported by O'Brien, Haynes and Mumby (1998).

In a longitudinal study Borghi, Costa, Boschi, Mussi and Ambrosioni, (1986), found that among borderline hypertensives with high sodium levels, DBF reactivity to mental arithmetic, and DBF recovery following mental arithmetic, were significant predictors of sustained hypertension after 5 years. Individuals exhibiting greater reactivity and having recovery pressure at least 6% above baseline levels were at greatest risk for developing sustained hypertension.

Physical stress (e.g. acute exercise) too has been reported to be associated with differing physiology. Bulbulian and Ebert (1994) studied cardiovascular responses to moderate steady state exercise in Type A (n=11) and Type B (n=10) women in ages 18-43 years. Post exercise SBP and DBP reductions were similar in both groups. However, SBP and DBP responses during exercise for Type B, but not Type A were lower at pre-exercise to study state exercise. Results thus state that physiological responses to exercise in females vary according to behavioural types associated with differing physiology.

The relations between stressful life events and cardiovascular activity were investigated during periods of rest and stress which carried as a function of family history of hypertension. The sample included 32 males and 29 females FH + and 29 males and 32 females
FH- subjects. Within the family history of hypertension group (FH +) males exhibited a positive association between the number of negative avoidable events and resting SBP, whereas, an inverse association between these two variables was obtained for females. Among FH + females, resting DBP and the subjective effects of life event and number of avoidable events were inversely associated. Analysis revealed that DBP reactivity to stress was associated with FH + subjects who reported fewer negative life events and less subjective effects for these events (Jorgenson & Houston, 1989). Further, white subjects with a parental history of hypertension were reported to frequently exhibit cardiovascular responses to stress than do offspring of normotensives (Falkner, Kushner, Onesti and Agelakos, 1981; Jorgenson and Houston, 1981; Manuck & Proietti, 1982; Ditto, 1986). Also individuals at an increased risk for hypertension, as a result of parental BP, show greater cardiovascular reactions to psychological stress than people at a lower risk (Drummond, 1985; Ditto, 1986; Fredrikson & Matthews, 1990; Carroll, Harris & Cross, 1991). This illustrates the need to consider life event stress as a consequence of family history of cardiac illness.

Men and women because of the significant differences in gender roles, differ in their appraisal of stressful events (see Milyaeva, Khaliulin and Barbarash, 1995). Van Doornen (1986) showed that physiological factors e.g. serum cholesterol level, SBP and HR were higher in both males and females. During exam day (stressful event) a larger increase in urine adrenaline excretion along with a relation between cholesterol level and achievement motivation was evident in males. In females ambulatory heart rate correlated strongly with the state anxiety and state depression.
In a study on the effect of personality, stress and life style on hypertension in Australia by Mellors, Boyle and Roberts (1994) it was seen that across gender, life style variables were the best predictors of hypertension. Further, the effects on hypertension were magnified when personality, stress and life style variables acted synergistically.

Mechanisms linking ethnicity, psychosocial factors and BP have also been reported. Dressler (1986) studied 128 subjects (mean age 33.1 years) in Brazil. Findings state that psychosocial resources modified Black-White differences in BP. Highest mean BP was observed among mixed-race and black Brazilians who had low psychosocial resources. Afro-Brazilians with high psychosocial resources had low BP than white Brazilians. Fredrikson (1986) examined cardiovascular and non cardiovascular reactivity in three groups of Black and White subjects: established hypertensives, borderline hypertensives and normotensives. Although resting cardiovascular activity was similar in Black and White hypertensives and normotensives, HR and SBP increased less in Black hypertensive and normotensives. However, Light, Obrist, Sherwood, James & Strogatz (1987) reported that the black borderline hypertensives showed greater increases in systolic pressure than their white counterparts to the stressors.

Several studies also report that while patients with hypertension tend to exhibit cardiovascular hyperreactivity in relation to normotensive subjects in response to laboratory stressors (e.g. Steptoe, Melville & Ross, 1984).

Large and expanding literature suggests that both external and internal states are linked to both disease and illness in same causal fashion. While life change approach highlights that any change, irrespective of positive or negative will lead to illness, several studies
have reported that higher levels of negative life events are associated with symptom maintenance and illness exacerbations (Patterson & McCubbin, 1983; Perrin, McLean & Janco, 1988; & Walker & Greene, 1991).

Uchino and Garvey (1997) report that a stressful event (e.g., Speech Stressor) effects the level of BP. In their study in both males and females, the speech stressor was associated with significant increases in SBP, DBP and HR. They also reported that the availability of social support moderated cardiovascular reactivity to the acute stressor.

The other studies that considered stress (life or occupational) along with negative affectivity in the onset and exacerbation of essential hypertension include those by Cottington et al (1986); James (1987); Johnson, Spielberger, Worden & Jacobs, (1987); Henry (1988); Hafner & Miller (1991); Dressler (1993). However, Lazaro et al., (1993) found that the essential hypertensives did not differ in the frequency and intensity of stressful daily events from the controls. More recently, it has been pointed out that negative consequence of life change were stressful and not life changer per se. Friere (1985) states that an event's meaning is not inherent in the event itself. For example, the birth of a child may be seen as either wonderful or tragic. This critical determinant of which meaning is constructed lies in the subject's definition of the situation (Thomas as cited in Blumer, 1979). Furthermore, positive life changes act as stress buffers (Hobfoll & Spielberger, 1992; & Fredrickson & Levenson, 1998).

Individuals' appraisals of and reactions to stressful life events are influenced by their philosophical perspectives on life or their view of the world. Events that shatter a person's view of the world may cause
Review of Related Literature

intense distress and result in subsequent health problems (Antonovsky, 1990; Friedman, 1991). Research suggests that negative aspects of an object event, or choice are weighted more heavily than positive aspects in judgements (Peeters & Czapinski, 1990). Zaraski (1984), David and Paul (1984), Chan (1986) and Jandrof et al (1986) reported undesirable events were better predictors of physical symptoms. Lal, Ahuja and Madhukar (1982) observed that hypertensives reported more number of distressing life events and gave high mean distress rating than normals. This was more characteristic of males over 45 years of age. In coronary disease patients, Bhargava, Sharma and Agarwal (1982) observed that a major change in work responsibility and death of a close relative was significantly reported when compared to the normal controls.

Studies are suggestive of rises in BP and serum cholesterol before and after loss of job. Also, unemployed people consult doctors about cardiovascular conditions including hypertension (HT) more often than do employed people (Smith, 1991). Moreover, men are more adversely affected by unemployment than women (Leeflang, Klein-Hesselink & Spruit, 1992). Brackbill, Siegel and Ackermann (1995) reported similar findings with both unemployed men and women having higher risk of hypertension. The prevalence of self reported hypertension too was higher among these subjects.

Further, low income Blacks report more psychological distress than do higher income Blacks and lower and higher income whites perhaps due to the combined burden of poverty and racism (Kessler & Neighbors, 1986). Also low income blacks are more likely reside in neighborhoods characterized by crowding and other forms of
socioecologic stress. This is associated with hypertension and cardiovascular hyperactivity (Fleming et al., 1987).

Canton (1985) in a longitudinal study of stressful life events among 65 hypertensives and 105 normotensive male subjects found that the former reported more life events than the latter. Results also stated that some life events such as loss, can be considered as risk factors for hypertension.

Recent studies measuring chronic stress associated with life events have shown greater cardiovascular reactivity and neuroendocrine responses to acute stressors in individuals under chronic stress (Lepore; Miles & Harvey, 1997; Pike, Smith, Hanger, Nicassio, Patterson, Mc Clintick, Costton & Irwin, 1997). Fleming, Baum, Davidson, Rectanus and McArdle (1987) reported the chronic stress (e.g. crowding) was associated with greater cardiovascular reactivity, higher adrenaline and noradrenaline levels and self-reported stress ratings.

3.4 Type A Behaviour Pattern and Cardiovascular Disorders.

Stress research attributes individual differences in response to environmental strain to personality differences. One such personality variable Type A behaviour pattern (TABP) has been found to provide ample contribution to cardiovascular diseases (CVD). It has been the subject of extensive study as risk factor for coronary heart disease (e.g. Cooper et al., 1981; Houston & Synder 1988; Matthews, 1988; Matthews & Haynes, 1986; Suls and Sanders, 1989). Other reviews have evaluated evidence for Type A as a risk factor according to standard epidemiologic criteria for establishing causality (Matthews & Haynes, 1986)
In a study of 25 males with confirmed MI 25 non-coronary medical control patients and 25 non-hospitalized health controls, Barry and Wassenaar (1996), found that except for TABP none of the other coronary risk factor i.e. anger levels, stress levels, diabetes, previous CHD, physical exercise, smoking and alcohol consumption were able to significantly distinguish the MI group from the controls. Earlier, Spicer, Jackson and Scragg (1993) examined coronary risk associated with TABP, anger-in and limited social support among 206 male and 67 female MI cases and 454 male and 316 female controls. Results are indicative of increase in MI risk when TABP was combined with the tendency to express anger-in an aggressive manner. However, in female Type As, MI risk was increased for those who did not express anger in this way. Female MI cases were characterized by a failure to discuss anger and by fewer available social contacts, relative to controls.

Itzes, Szikriszt, Ortutay, Berecz et al (1998) studied MI patients, patients with psychosomatic disorders and healthy subjects for the development of coronary artery disease (CAD) with respect to rigidity and TABP and the connections between them. Rigidity of Type A patients on JAS was significantly higher than the type Bs. Results state that the rigidity of MI patients substantially deviates from rigidity of certain psychosomatic patient groups and may be a basic component of psychological factors in the development of CAD.

Recently, Julkunen, Salonen, Kaplan and Salonen (1992) examined the role of different aspects of hostility as predictors of the progression of carotid atherosclerosis (PCA). Results indicate that, in addition to previously established risk factors (i.e., higher serum cholesterol concentration, smoking & age), three psychological
variables - cynical distrust, impatience/irritability and anger-control, significantly contributed to a multiple regression that explained nearly one third of the PCA variation. Also high hostility with high anger suppression would predict PCA. However, anger-control seemed to be more important than anger-in as a modifier of the impact of other aspects of hostility on atherosclerosis. Further Lahad et al., (1997) showed that high hostility was associated with an increased risk for MI.

Meesters and Smulders (1994), however, found no significant differences in the mean scores of MI subjects describing themselves as exhibiting higher hostility levels than the controls.

3.4.1 Type A Behaviour Pattern and Coronary Heart Disease (CHD)

There is ample stress literature suggesting that Type A behaviour is related to CHD. Catiporic - Veselica, D- Urijancek, Bracic - Kalan and Amidzic (1997) investigated heart rate and heart rate variability in 82 patients (aged 26 - 70 years) with acute CHD of which 48 patients were classified as Type As and 34 as Type Bs. Mean heart rate variability was significantly higher in the Type A acute CHD patients. Further, Dorado and Fernandez (1997) reported that Type A subjects when compared to Type B subjects had greater arousal disposition.

Kaushik et al., (1991) examined psychological correlates (e.g. coronary prone behaviour (TABP), job involvement, speed & impatience, hard-driving and stressful-life events) and physical correlates (weight, height, SBP & DBP) in 15 male and 9 female CHD patients by administering the JAS. The important precipitating factor for both male and female CHD patients was TABP. Also male CHD
subjects had higher SBP and DBP, had more dominant roles in stressful events, higher weight and greater height than either female CHD subjects or controls. Only the hard-driving factor indicated a significantly higher mean in the female CHD subjects.

Research also supports the evidence that TABP predicts CHD independently of traditional risk factors. Time urgency also has been considered as a noxious aspect of TABP. While there are known gender differences in CHD, the relationship to personality has been largely overlooked. Thayer and Pitarice (1992) examined the relationship among 16 PF and time urgency in 62 female and 253 male engineers. Males exhibited more relationship between personality and time urgency. These differences may explain the higher incidence of CHD in males.

Friedman, Breall, Goodwin, Sparagon, Ghandour and Flieschmann (1996) found that 13 of 32 patients with CHD who also exhibited symptoms and signs of severe time urgency and hostility (the overt components of TABP) reported multiple episodes of silent Myocardial Ischemia (MI).

In a study of 194 employees (80% females) of a health care authority in Australia, Byrne and Reinhart (1994) investigated the possibility that TABP as reflected in JAS scores, influences CHD risk through an effect on the established biological risk markers, either independently or through the mediating influence of occupational dissatisfaction. The findings suggest that some aspects of behaviours falling within the Type A profile that are associated with CHD in men cannot be extrapolated to the prediction of CHD risk in women. In another study Byrne and Reinhart (1995) indicate that the use of TABP
in future studies of coronary risk must take into consideration the multifactor nature of the construct.

Considerable evidence for a relationship between anger/hostility and CHD incidence and mortality has been reported (Barefoot et al., 1987; Dembroski, McDougal, Williams, Haney & Blumenthal, 1985). The potential role of hostility in mediating development of CHD is gaining ground. Consistent with the above findings are results reporting that TABP is associated with high levels of hostile cognition (Weinstein, Davison, DeQuattro and Allen, 1986). Results have also stated that highly hostile individuals respond to interpersonal stressors with increased levels of physiological arousal thus leading to illness (Miller, Smith, Turner, Guijjar & Hallet, 1996).

Research is also suggestive that not all Type A components are predictive of cardiovascular disorders, therefore, the global aspect of TABP is much in question.

Recent contradictory findings in research attempting to determine whether TABP is a risk factor for CHD have spurred much debate. In contrast to the consistent and strong relationship reported before 1979, most subsequent studies failed to find any association between TABP and CHD (for reviews, see Booth-Kewley & Friedman, 1987; Manuck, 1988). The most compelling negative evidence concerning TABP derives from the Multiple Risk Factor Intervention Trial (MRFIT), a clinical trial designed to alter cardiovascular risk factors in high risk men. Both Structured Interview (SI) and JAS questionnaires revealed no relation between TABP and any clinical manifestation of coronary disease (Skekelle et al., 1985).
There has been a trend towards null findings in the recent years. Yet, results from experimental studies suggest that reducing TABP can decrease recurrent CHD (Friedman, Breall, Goodwin, Sparagon, Ghandour & Fleishchmann, 1996). Several explanations for these contradictory findings have been proposed yet have not been entirely satisfactory (Dimsdale, 1988; Matthews, 1988).

3.4.2 Type A Behaviour Pattern and Essential Hypertension/Elevated Blood Pressure.

Personality is extremely important in studies of the pathogenesis of essential hypertension (EH)/elevated BP. Several psychogenic influences throughout the patient's life pave the way for the deformation of psychological structures, make the personality vulnerable to emotive influences and contribute to an unbalance in the vegetative (physiological)nervous system. As indicated earlier, one of the personality factors playing an important role in the etiology of cardiovascular disease is TABP. Anger and hostility are now regarded as core components of the Type A construct (Davison & Neale, 1990).

Peez-Garcia and Sanjuan (1996) reported that Type A individuals reported higher levels of cardiovascular reactivity (BP and HR) than the Type Bs and this difference was explained by competitiveness. Earlier, Ortega and Pipal (1984) in a study of Type A and Type B males found that Type A participants sought greater degree of challenges and had higher cardiovascular reactivity during performance than the Type Bs. Further, Type A behaviour may be translated into heart disease through cumulative deleterious effects of chronic and excessive challenge induced cardiovascular excitation. Holmes, McGilley and Houston (1984) concluded that while working
on the extremely difficult task, the Type A subjects evidenced reliably higher SBP than did the Type Bs. Further, Kelly and Houston (1985) concluded that Type A behaviour was related to both self-reported stress and tension in general and for married women only to poor self-reported physical health.

Bermúdez and Pérez-Garcia (1996) tested the association between (i) components of coronary-prone behaviour (Hostility, Competitiveness, Impatience, Job involvement and rumination) and (ii) cardiovascular reactivity (SBP, DBP and HR), mood state and performance. 61 undergraduates in a laboratory session performed a mental arithmetic task and a reaction time (RT) task. The results indicated that only hostility, competitiveness and rumination dimensions were significantly associated with psychophysiological activity (mainly SBP). Researchers have proposed exaggerated cardiovascular responses during behavioural challenge as a mechanism linking hostility and style of anger expression with risk of hypertension and coronary heart disease (Diamond, 1982; Krantz & Manuck, 1984). Studies on the association between various anger, hostility or anger expression measures and the magnitude of increase in BP and HR during laboratory challenges relative to resting values, show on balance significant relations (Houston, 1988).

Suls and Wan (1993) in a computer based information search of psychological abstracts and MEDLARS databases yielded 28 reports on the relationship between trait hostility (TH), anger and cardiovascular activity. Results of the series of meta-analysis indicate that high and low scores on most TH measures do not consistently differ in SBP and DBP or HR reactivity to traditional laboratory stressors. Potential for Hostility-Interpersonal Style was predictive of exaggerated SBP and
DBP responses to provocative stressors when stressors were classified as provocative versus non-provocative in accord with Trait X Situation approaches. Larson and Langer (1997) clarify the complex relationship between hostility and cardiovascular reactivity.

Recently, Guyll & Contrada (1998) examined trait-hostility and social interaction in relation to ambulatory cardiovascular reactivity in 40 males and 39 females undergraduates. Interaction effects of overall DBP and HR levels indicated that hostility was positively associated with these variables in men only. In addition hostility (HO) was associated with higher SBP during social interaction in men. These findings are consistent with the hypothesis that cardiovascular reactivity to social interaction mediates the relationship between HO and coronary disease and they may have implications for understanding gender difference in coronary risk.

Several studies examined the age and gender differences in the distribution of anger and hostility in EH and CHD. One such study has been reported by Ricci Bitti et al., (1995). The sample composed of 80 EH patients (mean age 54.48 years), 80 CHD patients (mean age 56.04 years) and 80 controls (mean age 54.39 years) in Italy. Subjects responded to questionnaires on hostility and anger. Results indicate that aggressive responding was linked to the subjects' proneness to CHD, but was not associated with EH. Also the particular components of anger-hostility are important in the study of psychological risk factors of EH and CHD.

Research also supports the idea of the health-related implications of the interaction between emotions. Several studies indicate that anger causes increased BP in primarily hostile as opposed to non-hostile individuals. In their study Suarez and Williams (1990)
found that high hostile men when provoked while performing a mental task reported higher levels of anger and irritation and elevated BP than their low hostility scoring counterparts. Suarez, Kuhn, Schanberg, Williams et al., (1998) reported similar findings with harassed subjects with high hostility scores exhibiting enhanced and prolonged BP and HR. Physiological reactivity in high-hostility subjects was correlated with arousal of negative affect. Also high hostility men showed excessive behaviourally-induced cardiovascular reactivity to interpersonal situations and stress-induced cardiovascular reactivity was associated with the arousal of negative affects such as anger. Similar findings were reported earlier by Jamner, Shapiro, Goldstein and Huy (1991) who studied stress reactions in ambulance workers. High hostility scores workers showed larger BP increases during angry interchanges with emergency room personnel than did those with low hostility scores. Also, the closer link between angry emotions and physiological hyperactivity could contribute to the greater health problems observed among hostile persons.

The relationships of BP reactions to mental stress, cynical hostility and socio-economic status (SES) was investigated by Carroll Smith, Sheffield and Shipley et al., (1997). Results indicate that cynical hostility is negatively related to SBP reactivity and mental stress task performance but unrelated to ratings of task difficulty. The magnitude of SBP but not DBP change scores to stress was positively associated with occupational grade: the higher the grade, the greater the reactions.

In an earlier study by Edguer and Janisse (1994) among other findings the conflict conditions increased the SBP of Type As but not of
Type-Bs. Results state that Type A subjects may attempt to control not only their environment but also their reactions to stressors.

In an earlier study, Pittner and Houston (1980) indicated that Type A subjects manifested higher pulse rate across all conditions and greater SBP and DBP in response to self-esteem than did Type-B subjects. The Type As employed more suppression in response to both the threat to self-esteem and threat of shock and also employed more denial in response to threat to self-esteem than did Type-B subjects. The authors also discuss the implications regarding the means by which TABP increases the risk of premature heart disease.

Aggressiveness, another component of TABP has been found to play an important role in cardiovascular disorders (Johnson, Myers, Webber, Greenland et al., 1997).

Shapiro, Goldstein and Jamner (1996) investigated whether individual differences in 4 personality traits (cynical hostility, anger-out and defensiveness) would predict waking and sleeping ambulatory BP and HR and whether information about these traits would provide a source of racial and gender differences in these measures. Results stated that independent of personality factors, women had lower ambulatory BP and higher HR than men. Blacks had higher BP levels and less of a decrease in HR from waking to sleeping than the whites. Also elevated daytime and nighttime SBP was reported by blacks scoring high on cynical hostility and higher waking DBP was found in blacks scoring high on both anxiety and defensiveness. Effects were also shown for HR as a function of anger-out, anxiety and defensiveness.

The effects of individual characteristics (physical-fitness and personal characteristics) on cardiovascular reactivity and CHD risk was
investigated by Hendrix and Hughes (1997). TABP, trait-anger, trait-anxiety, SBP and DBP measures under a stressful condition were considered. Results elicited mean DBP under video stress condition to be predictive of increased potential of developing CHD, while hard driving TABP was a cardiovascular reactivity (BP & HR) antecedent. Earlier, Bulbulian and Ebert (1994) studied cardiovascular responses to moderate steady state exercise in Type A females. Of the 21 subjects (18-43 years) 11 were categorised as Type As and 10 as Type-Bs according to the JAS scores. Results elicit that post exercise SBP and DBP reductions were similar in both groups. However, DBP responses during exercise for Type B, but not Type A were lower at pre-exercise to steady state exercise. Also steady state exercise SBP was lower in Type Bs compared to Type As.

Several studies providing contradictory results regarding the causal relationship between personality of an individual and his proneness to hypertension have been reported. Gallacher, Beswick Jones and Turkington (1998) studied the association of TABP with greater pressor response stress. Findings did not support physiological response as an explanation of the association between Type A and heart disease. Ivancevich, Matteoson and Preston (1982), earlier too did not find any significant relationship of occupational stress, TABP and physical well being.

However, findings have reported the positive association between TABP and EH/elevated BP. Studies in India have also documented the positive association of Type A behaviour pattern with occupational stress and negative affectivity or elevated blood pressure (Evans, Palsane & Carrere, 1987; Sharma, Sood & Spielberger, 1999; Singh & Srivastava, 1999).
3.5 Emotions and Cardiovascular Disorders.

Emotions have been found to cause a complex physiological transformation in the nervous and cardiovascular systems with sympathetic tendencies prevailing in functional circulatory shifts. There are six basic emotions - anger, fear, surprise, happiness, sadness and disgust. These emotions seem to be culturally universal. Although researchers do not agree on the psychological factors inessential hypertension (Mann, 1986), during the past two decades there has been a surge of interest in the contribution of negative emotions such as anger and anxiety in the etiology of EH (Hartfield, 1985; Spielberger et al., 1991).

In a study by Frasure, Lespérance and Talajic (1995) examining the importance of anxiety, anger-in, anger-out, major depression, depressive symptoms, history of major depression and perceived social support, it was seen that all these, significantly predicted cardiac events.

Ricci Bitti et al., (1995) found that the proneness to CHD was significantly correlated with aggressive responding. The frequency and extent to which one experienced anger arousal had pathogenic effects. They also discussed the evidence which suggested that neurobiology plays a role in the interaction of anger and hostility and coronary risk.

Haynes, Feinleib and Kannel (1980), earlier, investigated the three modes of anger management in relation to CHD and inferred that the inhibition of angry feelings may increase risk for CHD. Dembroski, MacDougal, Williams, and Haney (1985) examined the interaction between anger-in and the potential for hostility, and their relationship to CHD severity in a group of coronary atherosclerosis patients.
Findings indicated that potential for hostility was associated with disease end points, or pathology, for patients high on anger-in. Similar findings have been reported by Julkunen, Salonen, Kaplan and Salonen (1992). Recently, Weeneberg et al., (1997) investigated potential relationships between increased platelet aggregability and psychological characteristics such as hostility and anger. Findings state that mode of anger expression may be associated with increased platelet aggregation and could provide a mechanism for the putative connection between anger/hostility and coronary disease.

Data supports the idea of negative emotions coexisting in configurations and their importance and roles as precursors of CHD and hypertension. Greenglass (1996) found that higher anxiety scores were observed in both women and men when the respondents also had high scores on both anger-in and cynical distrust. Moreover, in males, higher speed and impatience, as measured by Type A scales was found when they had high scores on both cynical distrust and anger-in scales. It is suggested that such an emotional configuration may be a precursor of hypertension. Previous research data is suggestive of the fact that a greater number of hypertensive patients reported higher levels of suppressed anger while scoring higher than normal controls on scales assessing anxiety and depression (DeQuattro et al., 1981).

Emotional configurations and their roles as precursors of CHD have been reported by several researchers. Booth-Kewley and Friedman (1987) discussed the picture of a coronary-prone individual who exhibit one or more emotions. These may include anger, anxiety, depression etc. Smith, Stemdorff, Röpcke, Gustavsen et al., (1996) examined relations between psychological factors and CHD. Anthony (1989) examined the association of anger/hostility, anxiety and chest
pain with CHD in 542 patients (332 males and 210 females). It was found that a large proportion of patients with lower anxiety and higher anger/hostility scores were found to have CHD. Conversely, a large proportion of patients with higher anxiety and lower anger/hostility were free of CHD.

The relationship between anger and anxiety is borne out by empirical evidence. Anger and anxiety may be closely related, rather anxiety in many situations accompanies anger. Anger discomfort has a positive correlation with trait anxiety and also is positively related to anger suppression and expression (Sharkin & Gelso, 1991).

Iqbal, Nabi and Ahmed (1993) explored the relationship between anger and anxiety in a sample of 100 subjects of which 20 high anxiety and 23 low anxiety subjects were identified. Comparisons on anger direction in high and low anxiety groups reported that high anxiety subjects had a significantly higher score on anger-in than the low anxiety subjects. However, no difference was observed in the two groups on any other score.

Ghosh and Sharma (1998) found that hypertensives not only reported higher trait anxiety but also higher frequency of anger regardless of the direction of anger expression. The role of negative affectivity (anger & anxiety) in the pathogenesis of EH has also been supported by several other researchers (Dimsdale, Pearce, Schoenfeld, Brown, Zusman & Graham, 1986; Schneider, Egan, Johnson, Drobny & Julius, 1986; Harburg, Gleiberman, Russel & Cooper, 1991; Sharma, Krishna and Spielberger, 1996).
3.5.1 Anger, Anger Expression and Essential Hypertension/Elevated Blood Pressure.

In the least few years there has been increasing interest in the role of anger and its expression in physical and psychological well-being/health. A number of studies, both cross-sectional and prospective have provided evidence that individuals who are higher in anger/hostility have higher levels of CAD as well as higher death rates from CHD. The evidence of anger/hostility as a factor influencing health outcome was further confirmed by a recent meta-analytic review of US studies. The review found that hostility/anger is a significant independent risk factor for CHD even when other factors are controlled (Miller et al., 1996).

Earlier research is supportive of systematic study of both of anger and the multidimensional nature of anger and their association with indices of cardiovascular risk (Siegel, 1984). The relationship of anger and anger expression with hypertension (Boutelle et al., 1987; Schneider et al., 1986; Sharma et al., 1996; Ghosh and Sharma, 1998) and elevated blood pressure (Sommers-Flanagan and Greenberg, 1989; Dimsdale et al., 1986; Diamond et al., 1982) has been confirmed.

Crane (1982) explored the relationship of state and trait anger and hypertension. T-Anger is conceptually defined in terms of individual differences in the disposition to experience anger. Individuals higher on T-Anger are expected to perceive a wider range of situations as anger provoking (e.g. annoying, frustrating, irritating), than individuals low in T-Anger, and to respond to such situations with elevation in S-anger. Crane (1982) compared S-Anger and T-Anger of male hypertensives with a control group of general medical patients having no history of hypertension or heart disease using State-Trait
Anger Scale (STAS) (Spielberger, 1988). Greater state and trait anger were reported by hypertensives, though the difference on T-Anger were due almost entirely to the hypertensives' higher score on 'Anger-situation' subscale as compared to the 'Angry-temperament' subscale of trait anger. Results also showed that though the hypertensives reported more intense angry feelings than the controls, they appeared to suppress their feelings in interpersonal situations which resulted in less overt aggressive behaviour.

In a study in Netherlands, Van der Ploeg et al., (1985) also found that male hypertensives were more anger prone than their normotensive counterparts. Boutelle, Epstein and Ruddy (1987) further reported that both male and female hypertensives elicited greater proneness to anger. In a study conducted in Portugal, Coelho, Hughes, Fonseca and Bond (1989) explored the relationship of essential hypertension (EH) and psychological factors including anger. The comparative study of hypertensives and normotensives reported that the anger score of hypertensive patients was higher than that of the normotensives (p=.05). Earlier, Kearns (1985) and Gorkin et al., (1986) also reported greater proneness to anger (T-Anger) in hypertensives. Deshields (1986) further observed that the hypertensive group yielded higher scores on trait-anger (T-Anger) than the non-patients control group. Schneider et al (1986) state that the hypertensives are likely to respond more frequently with intense anger when evaluated negatively. Earlier, Diamond (1982) in a review of literature concluded that trait-anger is a significant contributor to essential hypertension. Recently, Suls, Wan and Costa (1995) in a meta-analysis reported that anger experience was correlated with elevated BP, but the relationship was small and highly variable.
Earlier, Schwartz, Weinberger and Singer (1981) examined that, of anger, sadness, fear, relaxation and control imagery, greatest overall increases in cardiovascular measures was produced by anger in terms of greater DBP, HR and Slower recovery of SBP following exercise.

Hartfield (1985) on comparing scores on anger measures in hypertensives and normotensives found that hypertensives experienced longer lasting anger of greater intensity, felt more physical symptoms during anger episodes and expressed less anger than normotensives. They also used more distancing, self-control and escape-avoidance coping than the normotensive subjects. Müller (1988) reported increase in BP as a consequence of an increased secretion of renin after anger stress particularly suppression of anger.

The direction of association between anger/hostility, anger expression and reactivity has been found to vary across studies. Studies by Diamond (1982) and Krantz and Manuck (1984) opine exaggerated cardiovascular response during behavioural challenge to be a mechanism that links hostility, anger and styles of anger expression with hypertension and CHD. Houston (1988) confirms the association of anger, hostility and anger expression measures to with increase in BP and HR during laboratory challenges. Highly hostile men when provoked while performing a mental task reported higher levels of anger, blood pressure, blood flow to their muscles and stress hormones when compared to their low hostility scoring counterparts (Suarez & Williams, 1990). Similar findings have been reported by Jamner Shapiro, Goldstein and Huy (1991) stating that the closer link between anger and emotions and physiological hypertactivity could contribute to the greater health problems observed among hostile persons.
However, Suls and Wan (1993) in a meta-analysis of 28 reports on the relationship of T-Anger/hostility and cardiovascular activity reported that high and low scores on most trait-hostility measures do not consistently differ in SBP and DBP or HR reactivity to traditional laboratory stressors. Jorgensen, Johnson, Kolodziej and Schreer (1996) reported weak associations of anger-affect expression and negative affectivity.

There is considerable variation across studies regarding the association of dimensions of anger with EH/elevated BP. Some investigators indicate that outward expression of anger (anger-out) is associated with greater blood pressure (BP) (Diamond, Schneiderman, Schwartz, Smith Vorp and Pasin, 1984, Vögele & Steptoe, 1993; Siegman, 1993; Schwenkmezger & Hank, 1996), while others show that holding anger in (suppression of anger) is a critical factor in the development of EH (Holroyd & Gorkin, 1983; Dimsdale et al., 1986; Schneider et al., 1986; Vögelle, Jarvis & Cheesman, 1997).

In a study Goldstein, Edelberg, Meier and Davis (1998) reported that expressed anger was inversely related to SBP and DBP, while family expressed anger was inversely related to SBP only. When subjects were divided into normotensives and hypertensives, the 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. However only females showed the relationship with SBP (see also James et al., 1986).

Further inverse associations between high BP and overt behaviour associated with assertion, dominance, anger-hostility and the emotional expression has been reported by Jern (1986). Otten (1993)
reported negative relationship between externally directed anger and SBP in hypertensives. In 13 international reviews Vögele and Steptoe (1993) found that in 9 studies, externally directed anger expression was associated with SBP and/or high DBP.

In a recent study Schwenkmezger and Hank (1996) examined 40, healthy male normotensive students with extreme high and/or low scores on the anger-out and anger-in subscales of STAXI in laboratory and field studies. In the laboratory setting the students with high anger-out scores showed higher BP than those low in this disposition to overtly express anger. However, no relationship was found between suppressed anger (AX/In) and BP. In the field study, similar results were found though they were moderated by the students' coping capabilities.

While frequently expressed anger is linked to coronary malfunctioning (Mendes de Leon, Carlos, Meesters, 1991), substantial evidence supports that suppressed anger is linked to elevations in BP. (Gentry et al., 1982) reported a significant and positive relationship between suppression of anger (AX/In) and systolic and diastolic blood pressures. In their studies Diamond (1982) and Spielberger, Johnson, Russel, Crane and Worden (1985) replicated these findings. Similarly Van der Ploeg, Buuren and Brummelen (1985) found that hypertensives although feeling irritated and angry retreat to some extent from showing their anger. Schneider, Egan, Johnson, Drobny and Julius (1986) contend that anger suppression (AX/In) rather than anger-proneness is the critical factor in the development of EH. They isolated two groups of borderline hypertensives, one group that maintained high BP outside the clinic and another whose average BP returned to normal at home.
The high home BP group reported greater intensity of anger, although they suppressed their expression of anger to a greater extend. But this group did not differ in anxiety. Also BP variability was not different between the two groups. They suggested that the psychological differences found in the group of higher risk borderline hypertensives may through autonomic arousal contribute to the later development of established hypertension.

Further, Cottingham, Mathews, Talbott and Kuller (1986) reported that the interaction between suppressed anger and job stress significantly predicts hypertension status. In contrast Siegman and Snow (1997), however, reported that only the anger-out condition was associated with high cardiovascular reactivity levels. Anger-in was associated with near zero or very low reactivity levels. Among others, this study showed that the full-blown expression of anger, in all of its intensity is pathogenic and that the mere inner experience of anger is not. In an earlier study by Johnson (1984), suppressed anger or anger-in was found to be significantly and positively correlated with SBP for males ($r = .47; \ p < .001$) and for females ($r = .27; \ p < .001$). However, anger-out was found to be negatively correlated with SBP ($r = .13; \ p < .01$), the correlation being similar in magnitude for males and females. The correlation between total anger expression and BP measures was similar in magnitude to that of anger-in and SBP, but opposite in direction for both males and females. Anger-in was significantly and positively correlated with DBP (males : $r = .29; \ p < .001$ and females : $r = .16; \ p < .001$), but less in magnitude than that of correlation between anger-in and SBP. The correlation between anger-out and DBP was negative and very small in males ($r = -.09; \ p < .05$ ) whereas no such relationship was found in females. In
analysis of variance differences in SBP for males and females the five anger-in groups were evaluated. The main effects of anger-in were significant (df = 4, f = 49.19, p < .001) i.e. for higher anger-in SBP was also higher in males and females. The main effects of gender were also reported to be significant (f = 43.44; p < .001) which indicated that SBP for males was consistently higher than for females at every level of anger-in variable. The main effects of anger-in by gender were significant (f = 4.44; p < .001) stating that SBP for males began to increase at a lower level of anger-in than for females. Higher DBP was found to be associated with higher anger-in scores for both the genders, though average DBP of females was comparatively higher. ANOVA also revealed that for higher levels of AX/Out, SBP for both men and women was substantially lower (f = 5.48, p < .001). Significant gender differences indicated that SBP for males was substantially higher than for females at every level of anger-out variable (f = 51.98; p < .001). The SBP for males was found to be uniformly higher than for females at every level of anger-out variable. Study trends showed lower DBP to be associated with higher anger-out scores.

Further, research has called attention to the different ways men and women experience and express anger (Thomas, 1989; 1993). Contrary to the previous research with the Framingham Anger Scales (Haynes et al., 1978) in which women were more likely to suppress anger than men, Thomas (1989) did not find any gender differences in either anger suppression (anger-in) or anger expression (anger-out). However, women were found to discuss their anger, had more anger-related physical symptoms and trait anger as measured by the State-Trait Anger Scale (STAS), strongly related to perceived stress, especially vicarious stressors arising from women’s concern about
others and their drive to care for them. Correlations between the Framingham anger-in and anger-out measures were essentially zero for both men and women. This finding was consistent with that reported by Spielberger et al. (1985).

High anger-in scores indicate that an individual frequently experiencing intense angry feelings tends to suppress rather than express them (Spielberger, 1988). Anger-in may then be seen as an emotional-focused coping strategy as it tends to be used to regulate one's emotional reactions or make one feel better without solving the problem. Sharma and Acharya (1989) studied coping with organizational stress and anger expression in 150 engineers in Himachal Pradesh (India). They found that suppressed anger was associated with avoidance coping.

In a recent study Bongard, Pfeiffer et al, Absi, Hodapp et al., (1997) investigated whether effortful active coping and anger provocation add in their effects on cardiovascular responses. Results indicated that provocation led to an increase in anger, but not in fear or negative or positive affect. Effortful active coping and provocation elevated cardiovascular activity, with provocation affecting particularly DBP and HR. The effects of active coping and provocation on HR and DBP but not on SBP were additive and probably were produced by different physiological mechanism. However the relationship between anger expression and coping styles has generally received less attention. It could be that higher levels of stress are related to less effective coping strategies.

Another study examining the effects of habitual anger-coping styles gender on SBP/DBP levels was conducted by Gentry, Chesney, Gary, Hall and Harburg (1982). They studied 495 Black and white
males and 511 Black and white females residing in high/low stress areas of Detroit with respect to provocative interpersonal situations. Race and anger expression were found to be major determinants of DBP and race and gender influenced SBP. Anger expression was related to SBP but only in females. Findings further illuminate the importance of sociodemographic factors and socioecological niche in predisposing humans to vascular disease and the magnitude of difference in mean BP and risk for hostility attributable to chronic suppressed anger.

When compared to other groups Black people residing in the United States have consistently shown a disproportionate risk for hypertension and the mortality, morbidity and sequelae of this disease (Myers and McClure, 1993). In industrialized nations the Blacks are more likely to experience chronic sociocultural stressors (e.g. racism, lower SES and living in high crime neighbourhoods) than the whites (Anderson et al, 1989; Anderson & McNeilly, 1993; Dressler, 1993; Myers & McClure, 1993). Anderson et al. (1992) contend that chronic sociocultural stressors are thought to interact with psychological risk factors (e.g., suppressed anger, anxiety and depression) in the frequent triggering of the physiological stress reactions (e.g. SNS mediated sodium retention and vasoconstriction) thought to contribute to the development of EH (Anderson et al., 1989, 1992; Anderson & McNeilly, 1993; Myers & McClure, 1993).

In a recent study Somova, Diarra and Jacobs (1995) reported a significant link between suppressed anger and BP level in 2,902 Black, White and Indian students in two African developing countries. Thus, the combination of chronic sociocultural stress and psychological risk
as suppressed anger may induce greater pathophysiological activity related to essential hypertension (EH) than either factor alone.

Rüdel, Schächinger, Quirrenbach and Otten (1993) studied anger, anger expression and BP over a 24-hour period. Though no relation between occasional measurements of BP and anger expression variables was found, however, aggregated 24-hour DBP scores had a significant positive correlation with suppressed anger and a negative correlation with externally directed anger, and also there were diurnal variations.

Julkunen and Korhonen (1993) empirically addressed the question. "Is the anger-in-BP relationship independent of the situational characteristics, or is it affected by characteristics of the context or other personality factors?" The study sample comprised of 351 women from a Health promotion Program conducted in North Karelia. They hypothesized that the association between anger-in and BP would be positive and more pronounced in a setting where the workplace atmosphere was tense and hostile. Results of a two-way analysis of covariance (ANCOVA) revealed a significant and positive association between anger-in and elevated BP in women who experienced their work unit as hostile and tense. Findings offer the impact of a contextual variable (i.e., the emotional quality of perceived environment) on the person-health relationship. On parallel lines, Christensen and Smith (1993) reported heightened BP reactivity during social interaction in hostile subjects when compared with those low in hostility. The AX/In-BP interactions were also dealt with by Baron and Kenny (1986), and Cox and Ferguson (1991); and Thomas (1997).

Furthermore, Sharma, Krishna and Spielberger (1996) reported in Indian context that regardless of gender high AX/In and lower
AX/Out was observed in hypertensives than in normotensives. The correlations of the hypertensives for both AX/In and AX/Out with BP were highly significant, with suppressed anger more strongly related to elevated SBP than DBP, thus stating the importance of suppressed anger in the etiology of hypertension. Higher inverse correlations between AX/In and AX/Out scales, especially in hypertensives, considered alongwith the substantial correlations of these scales with BP, point to a striking East-West cultural differences. Consistent with the above findings Ghosh and Sharma (1998) also highlighted the association of negative emotions (particularly suppressed anger) on patients with essential hypertension.

Earlier, Boutelle, Epstein and Ruddy (1987) found that inhibition of anger is the cause of heightened autonomic arousal and hypertension. They stated that there is a relationship between hypertension and self-reported measures of autonomic arousal, proneness of anger and avoidance of or guilt over the expression of anger. The correlational nature of the study provides three possible interpretations of the findings. One, that the inhibition of anger or guilt over its expression causes the heightened autonomic arousal, which causes hypertension. Two, the reactions to hypertension cause the inhibition of anger and the heightened autonomic arousal. Three, that hypertension and the three psychological variables (anger, anxiety & depression) are all caused by another factor.

Vögele et al., (1997) investigated gender-related differences in cardiovascular reactivity and role of anger inhibition and risk for future hypertension. Results show higher cardiovascular reactivity in males than in females and in subjects at risk for hypertension. A combination of hypertension risk and AX/In led to the highest
reactivity in males, while in females, differences in anger-in had no effect on reactivity. Another explanatory approach views increase in BP as a consequence of an increased secretion of renin after anger-stress particularly suppression of anger (Miller, 1988).

However, Sharma et al., (1996) reported positive and significant correlations between AX/In and BP in both males and females. These correlations were more significant with SBP than with DBP for both sexes.

There are also studies reporting that suppressed anger was unrelated to BP. Mann (1986) was rather skeptical about the impact of anger and anger-expression on the development of hypertension. Knight, Paulin and Wall-Manning, (1987); Mills, Schneider and Dimsdale (1989) and Smith and Houston (1987) reported similar findings. Vögele and Steptoe (1993) with reference to the 13 international studies reviewed concluded that externally directed anger was more strongly related to BP increases than suppressed anger. Siegman (1993) provided for similar conclusions.

In contradiction to these findings researchers reported no association of anger with hypertension/elevated BP. In their study Mills et al., (1989) studied the relationship between anger expression and physiological reactivity. 40 male normotensives aged 20-40 years (mean age = 32.5 years) were administered AX-Scale (Spielberger et al., 1985). Correlation run between the change scores of the physiological variables with the anger scores revealed no significant association between SBP/DBP and anger-in, anger-out and total anger expression scores. Knight et al., (1987) replicated these findings.

Emotional Phenomena are increasingly being approached from a cognitive and sociocultural perspective. Singh and Misra (1997)
analyzed the experience of anger and aggression in a young adult sample with a focus on gender differences and subjective construal of anger. Using a narrative account of recent episodes of anger experiences, the participants were asked to provide accounts of various aspects of anger in relation to their experiences. The results revealed many similarities and some differences across males and females and indicated the role of appraisal and interpretation in emotional regulation, pointing toward Socio-cultural roots of emotions. The study also pointed to possible linkage of anger with depression and life satisfaction; and also to a moderately positive relationship between anger-in and anger-out subscales of Ax scale. The possibility that AX/In and AX/Out scales define poles of a unidimensional scale for populations in Eastern and other countries cannot be ruled out (see Sharma et al., 1996; Schwenkmezger & Hank, 1996).

Although a larger body of Western research supports the anger-in - blood pressure hypothesis, there is also conspicuous body of evidence that supports the anger-out - blood pressure hypothesis. Thus even in western social/cultural context no clear cut pattern emerges on the relationship of mode of anger expression and elevated blood pressure. This calls for further research.

3.5.2 Anxiety and Essential Hypertension/Elevated Blood Pressure.

The psychological changes that underline behaviour like tension, apprehension, nervousness, worry etc., are mediated by secretions of adrenalin and other stress hormones which elevate BP, HR, dilate pupils and make breathing faster (Spielberger & Krasner, 1988), thus emphasizing the role of anxiety in the onset and exacerbation of idiopathic hypertension (see Coelho, Hughes, Fonseca...
and Bond, 1989). These authors examined the relationship between EH and psychological factors in Portugal on essential hypertensives (n = 165) and normotensives (n= 152) attending a general hospital. The matched groups did not differ significantly with respect to age (EH, M = 46.61 + 11.10 years, NT, M= 47.45 + 12.31 years), sex, marital status or social class. Hypertensives scored higher on neuroticism ( p < .001) than the normotensives and also their total anxiety scores were higher (p<.02). Psychological differences in hypertensives were a consequence of the patients raised BP and associated biological changes, which suggest that increase in the psychological disturbance parallels the severity of EH.

In an early study Safar et al., (1978) compared the anxiety levels of borderline and established hypertensives. It was seen that in sustained hypertensives there was an inability to express anxiety in a symbolic fashion and that they have a predominantly somatic issue in place of the psychological conflicts observed in borderline hypertensives. In another study Pestonjee and Bagchi (1979) found that coronary patients were more anxious about their health, success or failure of their ambitions, family, friendship, love, social relations and their future. Also higher somatic and free-floating anxiety and neuroticism was found in the coronary subjects when compared to the normals (Bhargava, Sharma & Agarwal, 1980). In a Dutch study Van der Ploeg, Buuren and Brummelan (1985) investigated the role of anxiety in essential hypertension. EH patients(males = 69; females = 35) from the out patients clinic of the University Hospital of Leiden, were matched to a control group (n= 104) from the general population of Leiden. They were matched with regard to age, level of education and gender. Significant differences in levels of anxiety in males only
was elicited. Average level of A-state of hypertensives (39.3) was higher than that of A-state of normotensives (35.4, p < .05). Similar results were obtained on A-Trait as well, with hypertensives scoring higher (39.9) than normotensives (36.8; p < .10). However, females did not exhibit such differences in anxiety (State/Trait). This study underlines that hypertensives are characterized by subjective feelings of tension, apprehension due to heightened ANS activity (A-State)) and their tendency to respond to situations perceived as threatening is higher (A-Trait) as compared to normotensives.

In another study James, Yee, Harshfield, Blank Mee and Pickering (1986) examined differences in BP associated with reported anxiety, happiness and anger among 90 borderline hypertensives (aged 16-70 years) during 24 - hour BP monitoring. Results indicate that emotional arousal significantly increases SBP and DBP. Pressure during reported anxious and anger states were higher than those during a happy state. Emotional effects were also related to the degree of individual daily pressure variation such that the greater the variability the larger the BP change associated with emotions. Results state that anxiety, happiness and anger increase BP to differing degrees and that emotional effects may be greater in individuals with more labile BP.

Further Boutelle et al., (1987) compared 60 hypertensives (30 males + 30 females) aged 25 to 74 years, receiving treatment for EH to matched controls (n= 60, males = 30, females = 30). Results revealed significantly (.05 level) higher scores on anxiety, neuroticism and anger in hypertensives than in the controls.

In a comparative study of hypertensives and normotensives Schneider et al., (1986) reported that anxiety (both State and Trait)
correlated significantly and positively with SBP in the group maintaining high BP outside the clinic, while anxiety in the normal home BP group was not related to either SBP or DBP. In another study, York, Borkovec, Vasey and Stem (1987) examined negative thought instructions in 36 undergraduates following emotion inductions designed to produce either worrisome somatically anxious or neutral states. Worry induction produced a significantly greater increase in negative instructions, than the neutral condition. Both worry and somatic anxiety inductions generated significant increases in cardiovascular reactivity.

In yet another study, Sharma, Krishna and Spielberger (1996), examined the role of anxiety in hypertensives (being aware of their disease status) and normotensives in both males and females. The mean age of the subjects was 46.2 years and 44.3 years respectively and all belonged to middle social class having minimum high school education. Hypertensives of both the sexes reported higher T-Anxiety than the normotensives. However, the correlation of the T-Anxiety scale with the BP measures were not statistically significant for either group. Though the absence of the relationship between anxiety and BP raises questions regarding the contribution of this negative emotion to the etiology of EH, the higher T-Anxiety scores of the hypertensives provide strong evidence that high anxiety is a concomitant of EH. The role of individual differences in T-anxiety (anxiety-proneness) by enhancing anger suppression seems quite plausible. An alternative interpretation to this is that hypertensives on being “aware” of their diagnosis experience anxiety more frequently for they are more concerned about and sensitized to the hazards of experiencing and expressing anger.
In an earlier study by Chaudhary, Singh and Bhardwaj (1994), it was found that patients with idiopathic hypertension were significantly more anxious and depressed than the normal controls and medically ill patients.

In a recent study, Ghosh and Sharma (1998) compared the anxiety levels in essential hypertensive and surgical/orthopaedic patients. The subjects were males (n= 40), married out-patients with mean age of 46.53 and 40.38 years respectively. Results state that the EH patients reported higher trait anxiety (T-Anxiety) when compared to their matched surgical/orthopaedic controls.

Similar findings of greater anxiety in hypertensives were reported in earlier studies (e.g. Bullpitt, Hoffbrandt & Dollery, 1976; Esler, Julius, Zweifler et al., 1977; Banahan, Sharpe, Baker et al., 1979; Sullivan, Procci, DeQuattro, Schoentgen, Levine, Van der Meulen & Bornheimer, 1981; Johnson, 1984).

Some investigators state that males and females differ on T-Anxiety. While some state that females report experiencing more emotion (T-Anxiety) than men and thus are more expressive too (Schwartz et al., 1980; Choti; Martson, Holston & Hart, 1987; Greenwald et al., 1989; Gross & Levenson, 1993), others report male hypertensives to be higher on T-Anxiety (see Van der Ploeg et al., 1985; Vögele et al., 1997).

Several studies, however, have provided inconsistent results (e.g. Whitehead, Blackwell De Silva & Robinson, 1977). Similar findings were reported by Knight et al. (1987) in a study conducted in New Zealand on an adult general population (n = 1127, hypertensives and normotensives combined). Anxiety Response Scale (ARS) measuring intensity of anxiousness was administered. One way
ANCOVA suggested that age connected scores (ARS) were not associated with SBP and DBP, which suggests that anxiety responsiveness or the propensity to feel anxious in stressful situations, does not predict levels of BP. Also Santonastaso, Canton, Ambrosio and Zambani (1984) found no correlation between anxiety, neuroticism and BP. Likewise Foster and Bell (1983) and Boutelle, Epstein and Ruddy (1987) reported no differences while comparing the anxiety levels of hypertensives with normotensives.

Thus, research on anxiety and essential hypertension/elevated BP has produced mixed results.

The preceding research, by and large thus predicts positive associations between markers of negative affect (anger, anxiety, hostility, depression, low self-esteem, & guilt) and hypertension (Sharma et al., 1996; Ghosh & Sharma, 1998) and blood pressure (Julkunen and Korhonen, 1993; Christensen and Smith, 1993). Byrne (1992) states that empirical research has linked anxiety, tension, depression, interpersonal sensitivity and guilt to elevated BP. Other studies report high casual BP levels to covary with covert negative affective states of anger, resentment, hostility and irritability (Johnson, Schork & Spielberger, 1987; Johnson, Spielberger, Worden and Jacobs, 1987; Durel et al., 1989). Further, Johnson and Spielberger (1992) and Byrne, (1992) also state that these negative affective experiences among persons with EH is triggered by a conflict between unfulfilled competitive and status needs and dependency on the approval of others. This illustrates the important role of emotions in the onset and exacerbation, of essential hypertension/elevated blood pressure.

Finally, it can be stated that a mosaic of factors interacting and changing synergistically across one’s life span is likely to produce
several different pathophysiologic manifestations of links among behaviour, personality, emotions (specially negative affectivity) and essential hypertension/elevated blood pressure).

Overview.

Essential hypertension (EH) is a significant risk factor for morbidity and mortality. The etiology and pathophysiology of EH has been a major focus of biomedical and psychosomatic research for decades. However, the attempts to link stress and personality/emotional factors to the pathophysiology of EH have produced a large body of conflicting findings. Such inconsistent findings relate to sample characteristics (race, culture, gender, age, SES, awareness of diagnostic status, medication) and methods (types of psychological instruments, adequacy of casual BP readings and SBP or DBP as the criterion).

As elicitors of EH, life events stress (LES: Operationalized as negative and positive life changes) has been a subject of inquiry of a number of Western and some Indian researches with inconclusive findings. While some studies dealing with the total frequency of life event occurrence do not report any significant difference between the hypertensives and the normotensives (Sevenson & Theorell, 1983; Boyce & Chestman, 1990, Lazaro et al., 1993) others have found a greater occurrence of life events in the hypertensives (Lal, Ahuja & Madhukar, 1982). However, not many studies have dealt with frequency of life events vis-à-vis hypertension. Later research, however, points out that negative consequences of life change were stressful and not life change per se. Positive life changes, however, act as stress buffer (Hobfoll & Spielberger, 1992; Fredrickson & Levenson,
1998). Recently, some researchers have either concentrated on a single life event (i.e. loss of job) or at the identification of culture-specific clustering of life events that act as elicitors of particular psychosomatic illness. Specific-clustering of life events in hypertension related primarily to personal, interpersonal, occupational, intrafamilial, unemployment, loss, chronic stress, etc., has been found across studies (Ghosh, 1989; Brack bill et al., 1995; Blumenthal et al., 1995; Lepore et al., 1997). Other researchers have also considered stress (life or occupational) along with negative affectivity in the onset and exacerbation of EH (Myers & Miles, 1981; Cottington et al., 1986; James, 1987; Johnson et al., 1987; Henry, 1988; Hafner & Miller, 1991; Dressler, 1993; Lazaro et al., 1993).

With regard to the relationship of life stress or stressful life events and other cardiovascular disorders (such as CHD, MI, atherosclerosis, etc.), most of the studies have reported a positive, significant relationship between the two (Grossarth-Maticek et al., 1994; Fisher, 1996; Perez et al., 1998). Positive evidence of the role of life stress in other psychophysiological and psychiatric illnesses too has been confirmed (Ghosh, 1989, 1992; Pefley, 1986; Srivastava & Broota, 1987; utam-Kamal, 1990; Coyne & Downey, 1993).

Investigators have also attempted to determine the personality profiles of patients with EH and other cardiovascular diseases and found higher measures of Type A Behaviour Pattern (TABP), Anger and Anxiety in such patients. Hypertensive patients have been found to manifest more Type A characteristics (Lazaro et al., 1993; Hendrix & Hughes, 1997., Bermudez & Perez Garcia, 1996). Anger and hostility now regarded as the core components of the Type A construct have been found to play an important role in EH/elevated BP (Ricci Bitti et
al., 1995., Suarez et al., 1998). There are also suggestions in the literature that negative affectivity (e.g. anger experienced by the Type A individuals may be predisposing elements in cardiovascular disorders (Evans, Palsane & Carrere, 1987., Sharma et al., 1999., Singh & Srivastava, 1999). Further TABP has been found to provide ample contribution to other cardiovascular diseases like CHD (Kaushik et al., 1991., Catiporic - Veselica et al., 1997), MI (Barry & Wassenaar, 1996, Friedman et al., 1996., Lahad et al., 1997) and CAD (Ittzes et al., 1998). Despite some inconsistent findings (Goldberg, et al., 1981., Skekelle, et al., 1985, Gallacher, et al., 1988; Ragland & Brand, 1988), most of the studies however, agree that patients with EH and other cardiovascular disorders exhibit more Type A characteristics. Further the role of anger as a negative emotion and its multidimensional nature has been confirmed in the progression of EH/elevated BP (Sharma et al., 1996; Ghosh & Sharma, 1998; Dimsdale et al., 1986; Sommers-Flanagan & Greenberg, 1989). While some investigators have observed greater suppression of anger in hypertensives (Cox & Ferguson, 1991; Johnson & Spielberger, 1992, Jorgensen et al., 1992; Sharma et al., 1996; Ghosh & Sharma, 1998) others reported outward expression of anger is associated with elevated BP (Diamond et al., 1984; Vögele & Steptoe, 1993; Schwenkmezger & Hank, 1996). Anger has also been found to play a positive role in other cardiovascular disorders (Ricci Bitti et al., 1995; Weeneberg et al., 1997). It is, however, important to highlight that health-related consequences of anger have not been sufficiently studied in India. Also, the experience, expression and control of anger and related ill-health consequences may not be the same in Eastern and Western societies. Anxiety, too has been implicated in the progression of EH/elevated BP (Coelho et al., 1989,)
Review of Related Literature

Van der Ploeg & Brummelan, 1985., Chaudhary, Singh & Bhardwaj, 1994., Ghosh & Sharma, 1998) and has been observed as a significant emotion among the EH patients (Pestonjee & Bagchi, 1979., Sullivan et al., 1981., Bhargava et al., 1980., Johnson, 1984., Schneider et al., 1986; Boutelle et al., 1987). Anxiety has also been found to significantly predict cardiac events (Frasure, Lesperance & Talajic, 1995). The role of anxiety vis-à-vis EH/elevated BP yet needs to be extensively researched upon in India.

Some researchers have recently stressed the importance of examining emotional configurations (anger, anxiety, depression) vis-à-vis their role as precursors of EH/elevated BP (De Quattro et al., 1981., Dimsdale et al., 1986; Harburg et al., 1991; Byrne, 1992; Julkunen & Korhonen, 1993; Christensen & Smith, 1993; Sharma et al., 1996; Greenglass, 1996; Ghosh & Sharma, 1998), and other cardiovascular disorders. (Booth-Kewley & Friedman, 1987; Sharkin & Gelso, 1991; Iqbal, Nabi & Ahmad, 1993; Smith et al., 1996). There is also evidence to suggest that chronic socio-cultural stressors interact with psychological risk factors in frequent triggering of the physiological stress reactions to contribute to the development of EH.

Given the presumed importance of psychosocial stressors TABP and negative emotions in the development of EH, it would seem important to reconcile the inconsistent findings by further research using psychological tools with trans-cultural validity so that the integration of evidence is made possible at the international level.

Viewed as a whole the following issues/gaps emerge in this area of research:
(i) A large section of the research has been conducted in the Western set-up, and their findings cannot be safely generalized to the oriental set-up.

(ii) Validated tests to our culture still are called to be translations.

(iii) Use of narratives in such research limits the findings of the research as the patient's perspective is not talked of.

(iv) Another issue which comes to light is whether the findings can be generalized to other samples as the mechanisms for regulating anger and cardiovascular reactivity could take a different form in other samples.

(v) None of the studies have so far considered together life stress, TABP, anger and anxiety vis-à-vis essential hypertension.

(vi) A few multivariate attempts have been made to see the joint effect of the selected variables in EH/elevated BP and other coronary disorders simultaneously.

The review further highlights the gaps in such research in the case of EH and other coronary disorders in India.

3.6 Hypotheses

In view of the objectives of the study, the research gaps and the general trend of findings, following hypotheses were framed.

I. The hypertensives would not only report higher number of recent life events (changes) but also their greater impact when compared with their normotensive (surgical/orthopaedic) counterparts.
II. The impact of negative life events (changes) would be greater in hypertensives than their normotensive (surgical/orthopaedic) counterparts. However, no such difference would emerge with respect to positive life events (changes).

III. There would be a disease-specific clustering of recent life events (changes) in EH patients when compared to their normotensive (surgical/orthopaedic) counterparts.

IV. The hypertensives would be higher on Type A Behaviour Pattern (TABP), Trait Anger (T-Anger) and Trait Anxiety (T-Anxiety) than their normotensive (surgical/orthopaedic) counterparts.

V. The hypertensives would report greater anger suppression (AX/In) and relatively less outward anger expression (AX/Out) as well as anger control (AX/Con) than the normotensive controls.

VI. A subset of Life Events Stress (negative impact of life changes), TABP, T-Anger, T-Anxiety and Anger Coping Styles (AX/In, AX/Out, AX/Con) in combination would maximally separate/discriminate hypertensives and their matched normotensive counterparts.