CHAPTER VI

DISCUSSION

6.1 Stressful Life Events (Percentage and Perceived Impact) in Hypertensives and Normotensives

6.1.1 Percentage of Occurrence of Life Events

When the percentage (total, negative and positive) of life events of the two groups were compared only nonsignificant trends emerged though a greater total percentage of events were reported by the essential hypertension patients. This held good for the total sample as well as its gender (2) or age (2) sub-categories (see Table 5.1).

Lazaro et al., (1993) also reported that both the hypertensives and normotensives did not differ from each other in the frequency of stressful-daily events. Also no difference was evident between the two groups with regard to the quality and magnitude of emotional response and the degree of BP variability. These investigators further failed to provide a psychological and physiological profile for hypertensive patients. Earlier Sevenson and Theorell (1983) also did not find any significant difference on total mean number of life events between the groups of hypertensives and hypotensives. Similarly Ghosh (1989) reported non-significant differences with regard to percentage of occurrence of life events in hypertensives and normotensives.

Some studies reported an inverse association of chronic life events stress with cardiovascular reactivity. Thus suggests that life events may habituate the individual to stressors. Boyce and Chesterman (1990) also observed smaller SBP and HR responses to stress (e.g. video game & cold pressor tasks) in adolescent boys who reported high, rather than low life
event stress. They suggested that the association between low life-event frequency and greater cardiovascular reactivity might reflect an avoidant-denial behavioural style or that life events might have a toughening effect (Dienstbier, 1989). Further, Jorgensen and Houston (1989) also reported smaller DBP reactivity to stress (e.g. arithmetic and background digit counting tasks) in participants with a high frequency of life events. But this was evident only in those with a family history of hypertension. The preceding evidence suggests that either habituation and/or avoidant-denial behavioural style via-a-vis life events stress in case of hypertensives could explain their not being different in terms of frequency of events vis-à-vis surgical/orthopaedic controls.

Contrary to our findings Lal, Ahuja and Madhukar (1982) observed that hypertensives reported more number of distressing life events and gave higher mean distress rating than normals. This observation was characteristic of males who were over 45 years of age.

Not many studies have dealt with frequency of life events vis-à-vis hypertension. It appears that considering an overall index of recent life events in patients with psychosomatic illness is perhaps not considered that meaningful. This contention is supported in subsequent analysis of the present study when separate events were considered. In respect of other illnesses also frequency of life events does not emerge as a significant variable. Talley and Piper, (1986); in respect to dyspepsia; Ghosh, (1992), Dinan et al., (1991), Feldman et al., (1986). Gilligan et al., (1987) in respect of peptic ulcer; Wig, Menon and Chawla (1982) in respect of schizophrenia and organic pain (Stockton, Weinman & McCall, 1985; Hui, Shiu & Lam, 1990).

However, another group of investigators have substantiated the role of life stress in a development or precipitation of physical and psychiatric illness. Stress relating to life events has been demonstrated to be associated
with CHD (Bhargava, Sharma & Agarwal, 1982), ulcerative colitis (Chakroborty, Shah & Parikh, 1983; Khorana, 1983), depression (Prakash et al., 1980; Chaterjee et al., 1981; Gupta et al., 1981). In a study Saxena, Mohan, Dube, Sundaram and Chawla (1983) found that psychiatric outpatients showed significant differences in the frequency of 14 out of 44 events. The mean number of events experienced by them were $2^{1/2}$ times greater than the normal controls. In another study Chattopadhyay and Das (1983) reported that neurotics displayed significantly higher life stress scores than psychotics and normals with regard to both recent and remote events (mostly concerning undesirable events. (Gautam and Kamal (1990) observed that not only the number of stressful life events but also their impact was higher in neurotic patients. Earlier, Bhatti and Channabasavanna (1985) observed that neurotic patients experienced more stressful life events as compared to normal population. With respect to anxiety neurosis also, the frequency of total life events (as well as different categories of life events, experienced during life time and 6 months prior to the onset of the illness were significantly more in patients than in controls (Sharma & Ram, 1988). Similar trend of findings emerged earlier in respect to mania (Singhal, Agarwal & Nathawat, 1984) and peptic ulcer patients (Banerjee & Vyas, 1992).

However, this issue of recent and distal events is quite controversial as researchers are not sure as to which is more important as certain events precede the illness while others may be reported to be greater because of the illness. This and all other issues are highlighted by Sharma (1988) and these issues relate to observed inconsistencies on life events research. The findings of the present study is that when an overall index of recent life events is considered essential hypertensives do not document any differential occurrence of total life events when compared to controls. The inconsistencies in the finding of the above cited studies including the present one, can be
traced to the diversity of approaches to assessing stressful life events and their possible inadequacies (unreliability of measurement, failure to assess separately positive and negative life changes, insensitive methods of quantifying the impact of events) or to other factors. In addition researchers have avoided qualitative analysis of life events and chronic ecological stressors like crowding. Most of these studies are retrospective and there is a confounding role played by retrospective contamination or distortion in life events assessment. For example, a heart attack would seem to be a consequence of stressful life events and stressful life events in its own right. Further, very little is known about the particular type of event that are related to particular types of outcomes. Further, within the category of negative life events there may be certain type of events that are more important than others. It would seem desirable to assess both the things that happen to people and how they appraise them for their possible impact. This implies that the impact (positive and negative) and identification of specific clustering of life events, if any, particular to essential hypertension would be worthwhile to see whether they differentiate the hypertensives from the normal controls. It needs to be mentioned that the present study concerned itself with life events experienced in past 3 years and not during lifetime or more than past 3 years.

6.1.2 Perceived Impact (Intensity) of Life Events

Another finding is that the hypertensives reported (i) a greater overall impact of the life events (regardless of positive or negative impact) when compared to the normotensives (see Table 5.2); (ii) when compared gender (5.3a) and age wise (5.3 b, c) identical trend of results emerged for gender and age-wise analysis.
With regard to hypertension (EH) investigators have found that stressful life events affect the severity and exacerbation of EH. Shah (1980) studied the impact of stress in different organizational settings. He found that hypertension was among the other physiological changes felt by the officers when exposed to stress.

Laboratory paradigms of acute psychological stress have demonstrated reliable changes in cardiovascular functions that appear to provide information on an individual's relations to daily hassles and stressors (Matthews, Owens, Allen & Stoney, 1992; Pollak, 1991). These reactions could possibly be process variables mediating between stress and illness. Consistent with this, there are stable individual differences in cardiovascular reactivity to acute psychological stress (Manuck et al., 1989; Kamarck et al., 1992; Manuck, 1994). Investigators have also stated that immune system alterations in response to acute psychological stress suggests that such changes are greater in individuals characterized by sympathetic - cardiovascular reactivity (see, for reviews, Kiecott Glasser, Cacioppo, Malarkey & Glaser, 1992). Consistent with these results Uchino, Cacioppo, Malarkey and Glaser (1995) revealed that acute stressor was associated with changes in cardiovascular, endocrine and immune systems. Moreso, individual differences in cardiovascular reactivity predicted stress-induced cortisol changes. Results suggest that distinct physiological pathways are activated in response to acute psychological stress. Such studies document the physiological impact of stress.

Further, Baum (1990)is of the view that individuals may develop adverse physical or psychological reactions to stressful events that persist for prolonged periods (chronic). Several findings, however, favour considering recent life events over distal ones, and gender & age interaction (see Suh, Diener & Fujita, 1996). For example, Forest (1996) reported that while
women experienced a persistent influence from distal events, men were more responsive to recent conditions in terms of global happiness. In an earlier research Solomon et al.,(1987) stated that there may be delay in months or years between the stressful events and the emergence of overt disturbances in functioning. This to an extent explains the antecedent role of stressful events in the etiology of EH.

In the present study it was expected that gender or age would influence the perception/appraisal of life events and its subsequent impact because of differential gender role expectation conflicts or age-related experiences. One possible reason for the absence of differential overall impact could be due to sampling of events wherein perhaps, age-specific (middle & early old age) or gender-specific (male & female) life events were not adequately included.

The impact of the stressful life events over other psychophysiological disorders has also been researched upon. Review of literature dealing with CHD states that moderate to severe stress can lead to increased mortality (Grossarth-Maticek, Eysenck & Boyle, 1994). Elliott (1995) reported a link between psychosocial stress and coronary disease specially in women. Teshima et al., (1986) argued that the stress from daily life events influences impact certain biochemical changes which cause bronchialasthma. Imandescu (1987) showed that more frequent psychic stresses have impact in the form of a triggering effect for allergic syndromes related to respiratory disease. Goreczny et al., (1988) reported that both total and impact score on daily stress inventory related significantly to the symptoms of asthma. As stated earlier, Gautam and Kamal (1990) reported that life events had negative impact in neurotic patients in the form of heightened anxiety and depression.
6.1.3 Impact of Positive and Negative Life Changes.

The findings of the present study further revealed that the hypertensives reported higher negative life change score when compared to their normotensive counterparts. As for the positive life changes, a reversal was noted in the case of normotensives wherein a nonsignificant trend towards greater positive impact of life changes emerged. This was true for the total group (see, Table 5.2) and for gender (5.3a) and agewise (5.3, b, c) subgroupings.

Investigators have suggested that physiologic responsiveness to emotional stress might be a marker of pathogenic processes involved in the etiology of EH or CHD (Walker, Garber & Greene, 1994). Further, higher levels of negative life events are associated with symptom maintenance and illness exacerbations (Chase & Jackson, 1981; Patterson & McCubbin, 1983; Perrin et al., 1988). Kamarck et al., (1998) studied the acute effects of psychosocial processes on cardiovascular activity during daily life in a non-clinical sample. BP and HR were reported to be elevated during periods of emotion activation (high negative effect or high arousal). There can be a selective recall of life events. Sevenson and Theorell (1983) in a comparative study of hypertensives, hypotensives and normotensives found that while hypertensives recalled more negative life events, their normotensive counterparts recalled more positive life changes. Canton (1985) concluded that some negative life events can be considered as risk factors of EH. Further, Myers et al., (1983) are supportive of the view that psychosocial stress factors play an important though not a prime role in the etiology of essential hypertension. Dressler (1986) observed highest mean BP among mixed race and black Brazilians who had low psychosocial resources.
Jorgensen and Hosuton (1989) found that males with a family history of hypertension 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 with family history (FH+) of hypertension. Also among the FH+ females, resting DBP and the subjective effects of life event and number of avoidable events were inversely related. Further Forest (1996) reported that both men and women were more similar than different in their responses to undesirable life events.

In laboratory settings Gump and Matthews (1998), while investigating the role of sustained search for potential threat in acute cardiovascular stress responses, found that participants in the negative search condition had significantly higher SBP and DBP responses during subsequent stressors. Earlier, Uchino and Garvey (1997), reported that a stressful event (e.g. speech stressor) effects the level of BP. They observed that in both males and females, the speech stressor was associated with significant increaser in SBP, DBP and HR.

In India, Lal et al., (1982) observed hypertensive patients to have relatively higher mean distress ratings than the normals. Ghosh (1989) found that essential hypertensives experienced more negative life changes than their controls. The above findings show that these studies considered only the negative life changes and did not include the positive ones. Since positive life changes in the present study elicited non-significant differences in the hypertensives and normotensives, it seems that consideration of such positive life changes and their possible impact vis-a-vis hypertension and related disorders may not be fruitful.

With regard to other cardiovascular/physical/psychophysiological disorders similar findings have been reported. Low et al., (1993) found that stress, along with marital problems, family history and personal behaviour
provided for causal attributions in CHD. Elliot (1995) reported a link between psychosocial stress and coronary disease specially in women. Corse, Manuck, Cantwell, Giordani and Matthews (1982) found that patients with symptomatic coronary disease when compared to the controls, showed greater response to stress on some psychophysiological measures. There are studies that deal with other illnesses. Jandrof et al., (1986) found that daily undesirable life events are better predictors of physical illness. Chan (1986) found that life events have direct effect on physical symptoms. Pefley (1986) found that in both males and females life events were more significantly related to illness (e.g., flu, common cold and minor illness). Earlier, Elisabeth (1984) reported higher number of negative life changes in patients with affective disease i.e. manic depressive psychosis.

In India, Bandopadhyay et al., (1986) reported that two types of cancer patient groups showed a higher degree of exposure to stressful life events prior to the onset of disease than the controls. Srivastava and Broota (1987) also reported that 3 groups of cancer patients (diagnosed, prediagnosed and treated) experienced greater stress than the controls. In a recent study Ghosh (1992) found that patients with bronchial asthma or peptic ulcer reported significantly higher negative life change score than their surgical/orthopaedic controls. The preceding reference to a few studies dealing with stressful life events (negative and positive life stress) vis-à-vis other psychophysiological/physical disorders attest the paucity of relevant comprehensive research in the area of essential hypertension/elevated BP, with patient-controls. Thoits (1983) conducted a careful review of the research literature on the kind of life events that influenced psychological distress (e.g., negative impact), of 20 states that compared the effects of undesirable (negative) change versus change itself, only 3 failed to find that undesirable change was more strongly related to psychological disturbance.
than change alone. Even more compelling, several studies demonstrated that the relationship between life change and disturbance dropped to nearly zero when the effects or impact of undesirable events (negative life change) were partialed out. Indeed, Cohen and Hoberman (1983) reported that positive change can act as a stress buffer, offsetting the negative sequelae that might otherwise follow negative change. The present study also highlights the importance of considering negative life changes (events) and their impact in life events stress research in relation to health/illness.

6.1.4 Life Events Clusters in Hypertensives

Specific clustering of life events for hypertensives is illustrated in Table 5.4. Further, Table 5.5 illustrates the gender (5.5a) and agewise (5.5, b, c) clustering of events in the hypertensives. Taken together these tables demonstrate (i) major cluster pertains to familial events (including financial - spouse's/other family member's unemployment, loan, bereavement, child's failure in exam etc.), and (ii) self-related events (like unemployment, illness, change in eating and sleeping habits). The predominant theme of such events can be subsumed under the little 'LOSS'. Whether familial or personal such a broad cluster holds good for gender or age subgroupings, barring a few. Of these the male-specific events comprised of (i) not having a child after marriage (ii) financial loss or hassles (iii) child's failing an important exam, and (iv) trouble with employer or colleague. The female-specific events were (i) sudden decrease in income, (ii) unemployment - one's own or of a family member, (iii) husband's beginning or ceasing work, (iv) familial conflicts, and (v) large loan.

In an Indian study Ghosh (1989) in aged patients with essential hypertension observed a specific clustering of life events that related primarily to personal, interpersonal, intrafamilial, job retirement and
pending retirement. Bhargava et al., (1982) noticed in the coronary patients more major changes in work responsibility and death of close relatives as compared to normal controls. Canton (1985) found that hypertensives experienced higher exit events, events related to loss, than the normotensives. Patel (1994) observed that, especially men with lower employment status reported more stressful events and required treatment for hypertension and elevated BP.

In an earlier research Smith (1991) reported that unemployed people consult doctors about cardiovascular conditions including hypertension more often than do employed people. Brackbill et al., (1995) found both unemployed men and women having higher risk of hypertension. However, Leeflang et al., (1992) reported that men are more adversely affected by unemployment than women. Further association between psychosocial stressors at work (i.e., work load, conflict with employers) and health-related problems (i.e. cardiovascular disorders) have been found (Kua et al., 1989; Lam et al., 1987; Makowiec - Dabrowska & Bortkiewicz, 1990). Curtis et al (1997) examined association of job strain (or its components decision latitude and job demands) with elevated BP in 726 African-American adults. Though, job strain was not associated with BP in both men and women, decision latitude, however proved to be important for hypertension risk among men. Earlier, Blumenthal et al., (1995) evaluated effects of stress (e.g. job strain, occupational status and marital status) on BP in adults with hypertension. High job strain was associated with elevated SBP among women but not among men. Both men and women with high status occupations had significantly higher BPs during daily life as well as during laboratory mental stress testing. However, SBP was comparatively higher in high job status than in low job status men. Unexpectedly both married men and women reported elevated BP (SBP) than their unmarried counterpart's. In another
recent study, Carroll 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. However, no significant findings in treated hypertensives with respect to work-related problems were obtained, except for a group X gender interaction on job-related irritation with lowest irritation scores being evident in males and highest irritation scores in females. The preceding studies also highlight the importance of considering not only gender-specific life events but also the observation that life events stress has differential impact on males and females.

In a multiple regression analysis examining the relationship of job decision latitude, total psychological stressors physical exertion and work hours to DBP and serum cholesterol, Sorensen et al., (1996) reported that physical exertion was related to higher BP levels in women but to lower levels in men.

Matthews et al., (1997) reported greater DBP responses to behavioural tasks in those who suffered from ongoing, unresolved problems in their lives. Earlier, Fleming et al., (1987) operationalized chronic stress as living in crowded conditions. They found that crowding was associated with greater cardiovascular reactions and slower recovery to a visual problem-solving task, as well as higher adrenaline and nonadrenaline levels and self-reported stress ratings. Recent studies measuring chronic stress associated with life events have also reported greater cardiovascular and neuroendocrine responses to acute stressors in individuals under chronic stress (Lepore, Miles & Levy, 1997; Pike, Smith, Hauger, Nicassio, Patterson, McClintick, Costlow & Irwin, 1997).

Seibt et al., (1978) found no significant differences among borderline hypertensives, non-medicated hypertensives and normotensives on
cardiovascular reactivity to 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 point out a possible role of recovery processes after stress in the development of essential hypertension. Similar findings were reported by O'Brien, Haynes and Mumby (1998).

Clustering of life events has also been observed in other psychophysiological disorders. For example, Misra (1989) found that bereavement, conflicts at job and in married life coupled with change in social activities clustered to precede CHD. Gilligan et al., (1987) observed that duodenal ulcer patients had significantly more life difficulties than those related to personal and family illness, death of a close family member and role change etc. Khorana (1983) reported that marital problems, financial pressure and family problems were positively associated with ulceration. Ghosh (1989, 1992) found that peptic ulcer patients experienced more of intrafamilial, personal, interpersonal and job-related life events than the surgical/orthopaedic controls. Death of a spouse has been found to be significantly associated with the first episode of depression in old age (Sharma et al., 1985). Chaturvedi (1983) demonstrated the distressful impact of unhappy and hazardous married life, death of spouse or offspring, and hazardous friendly relations on psychosomatic disorders. Among neurotics family conflict emerged as the most frequently occurring event present, followed by financial problem or loss, and trouble at work and marital conflict (Gautam & Kamal, 1990). Death of close relative, financial difficulties, death of spouse, etc., turned out to be major life events in manic pathology (Singhal et al., 1984). The clustering of life events can also vary with the recent or remote - life time - perspectives. Sharma and Ram (1988), which studying anxiety neurosis, found four events namely, suspension from
job, theft or robbery, broken engagement or love affair and conflict over dowry, to be significantly more in patients during lifetime. On the other hand, 4 other life events such as, major purchase or construction of house, failure in examination, appearing for interview and getting engaged or married were found to be significantly more in patients during the 6 months prior to the onset of the illness.

The foregoing discussion, thus, highlights the usefulness and meaningfulness of identifying the peculiar clusters of stressful life events vis-à-vis various psychophysiological disorders for developing efficacious intervention programmes.

6.2 JAS-Type A Behaviour Pattern (TABP), Trait - Anger and Trait Anxiety in Hypertensives and Normotensives

6.2.1 TABP in Patient Groups

Table 5.6 illustrates that the hypertensives (either males or females or both) report more of JAS-Type A characteristics when compared to their normotensive (surgical/orthopaedic counterparts. Further, gender (5.7 a) and agewise (5.7 b & c) sub-categorization report similar findings.

These findings are consistent with those of prospective studies supporting the association between TABP and the risk of subsequent hypertension/elevated BP in both men and women. Lazaro et al., (1993) observed the essential hypertensives to be higher on TABP. Schaubroeck, Ganster and Kemmerer (1994) reported that TABP predicted cardiovascular disorder over time. Also the relationship between the two complexity indices (psychological and task-person indices of job complexity) and cardiovascular morbidity were positive among subjects with high TABP ratings but negative among those with low ratings. Further, Hendrix and Hughes (1997) found
that the hard driving TABP is a cardiovascular reactivity (BP & HR) antecedent.

Earlier, Wright, Contrada and Glass (1985) found that Type A patients when compared to Type Bs display larger acute increases in BP, HR, Catecholamines (epinephrine and norepinephrine) and cortisol when confronted by appropriately challenging or stressful tasks. Recently Peéz-Garcia and Sanjuán (1996) found that Type A individuals reported higher levels of BP and HR than the Type B individuals and this difference was explained by competitiveness. Earlier, Krantz et al., (1981) found that cardiovascular responses to challenging social situations revealed some evidence of Type A-B differences in SBP, HR and rate pressure product during the interview. Results also provide evidence for a direct linkage between BP/HR and the atherosclerotic process of coronary arteries.

Research has provided evidence of the mechanism and the link between the various components of Type A and disease status. Steptoe, Melville and Ross (1984) reported that mild hypertensives showed significant greater pressor reactions (both in absolute and percentage terms) than normotensives to task requiring active behavioural coping. Heightened DBP reactions, greater HR and pulse transient time reactions were observed in the transient group, suggesting that exaggerated cardiac responsiveness to active challenges may be characteristic of the prehypertensive profile.

Mathews (1988) opines that the zeitgeist surrounding coronary proneness seems to be moving towards blaming hostility as the key factor in coronary proneness. Researchers are of the view that it is important to recognize the complex and multidimensional nature of the hostility construct because of its potential influence on health, especially in the development of coronary disease (Siegman, Dembroski & Ringle, 1987; Smith, 1992). Lazaro
et al., (1993) observed that the essential hypertensives reported greater hostility.

Exaggerated cardiovascular responses during behavioural challenge have been proposed as a mechanism linking components of TABP like hostility and style of anger expression with risk of hypertension and CHD (Diamond, 1982; Krantz & Manuck, 1984). In a meta-analysis Suls and Wan (1993) showed that 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 situation approaches. Earlier, 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. In another study, Jamner et al., (1991) observed that workers eliciting high hostility showed larger BP elevations during angry interchanges as compared to those who scored low on hostility. Recently, Bermudez, and Pérez-Garcia (1996) found that hostility, competitiveness and rumination dimensions were significantly associated mainly with hypertension. Earlier, Mann (1986) reported that besides other findings behavioral attributes in hypertensives such as repression of hostility are not irrelevant to the understanding of cardiovascular responsiveness.

Ricci Bitti et al., (1995) state that the particular components of TABP like anger-hostility are important in the study of psychological risk factors of essential hypertension (EH) and CHD. In a recent study, Suarez et al., (1998) reported similar findings with harassed subjects with high hostility scores exhibiting enhanced and prolonged BP and HR. The physiological reactivity in the high hostility subjects was correlated with arousal of negative effect. Also, high hostility men showed excessive behaviourally induced cardiovascular reactivity to interpersonal situations, and the stress-induced
cardiovascular reactivity was associated with the arousal of negative affects like anger. Earlier Jamner et al., (1991) stated that the closer link between angry emotions and physiological hyperactivity could contribute to the greater health problems observed among hostile persons. Research literature on hostility and aggression reveals a great deal of conceptual ambiguity and confusion. Spielberger et al., (1983) have, however, tried to remove this ambiguity by stating that, while hostility refers to feelings and attitudes, the concept of aggression generally implied punitive or destructive behaviour directed towards other persons or objects. Further, Larson and Langer (1997) clarified the complex relationship between hostility and cardiovascular reactivity.

Ricci, Bitti et al., (1995) further reported the role of neurobiology in interaction of anger-hostility and coronary risk. With relevance to this aspect Friedman (1996) argues that the neurobiological features are suggested by reports linking disruption of brain stem cardiovascular control and cardiovascular reactivity in challenging tasks, particularly for Type A's.

Suls and Sanders (1989) observed that global TABP may engender high levels of sympathetic arousal to stressful events. Carroll et al., (1997) while investigating the relationship of mental stress, cynical hostility and socio-economic status (SES) found that cynical hostility was negatively related to SBP reactivity and mental stress task performance. Also, 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. Earlier Vickers, Herrig, Rahe and Rosenman (1981), hypothesized that the global TABP and its components, speed and impatience, would be related to the use of defense mechanisms while the TABP attribute, job involvement would be related to high coping. The findings support the
hypothesis by implying that increased coronary risk may be associated with poor coping skills.

Jern (1987) reports that in the case of EH, a specific set of conflicts and traits maintained across the life span are thought to eventually induce EH. Edguar and Janisse (1994) among other findings, reported that the conflict condition increased the SBP of Type As' but not of Type Bs'. Also the Type A subjects may attempt to control not only their environment but also their reactions to stressors. Further, Diamond (1982) and Johnson and Spielberger (1992) reported that persons prone to EH inhibit hostile and aggressive impulses in an attempt to avoid interpersonal conflict and maintain dependent, independent relationships. This to an extent provides explanation for the specific interpersonal clustering in EH patients. Further, aggressiveness, another component of TABP too has been found to play an important role in cardiovascular disorders (Johnson et al., 1997).

Brownley, Light and Anderson (1996) reported that the Type A component, hostility, was related to higher SBP as well as DBP. They also found that high hostility was related to higher systolic and diastolic pressure in whites, while low hostility was related to higher systolic & diastolic pressure in blacks. These relationships were significant for men at home and work and for women at screening. In another study the contribution of gender personality differences was investigated by Campbell and Muncer, (1994). They found that occupational role and sex were both important correlates of individual's representation of aggression and that the impact of gendered personality traits was diminished when occupation and sex were taken into account.

In contrast to the consistent and strong relationship reported, some studies failed to find any association between TABP and EH/elevated BP (see Gallacher, Beswick, Jones & Turkington, 1988).
In sum, findings of the present study reveal that regardless of gender or agewise categorization the hypertensives scored significantly higher on TABP when compared to their normotensive counterparts.

Contrary to the findings of this study, some studies have suggested that gender mediates the relation between Type A behaviour and EH/elevated BP (see, Vögele et al., 1997; Guyll & Contrada, 1998; Felsten & Leitten, 1996; Byrne & Reinhart, 1995).

In their earlier meta-analysis, Suls and Wan (1989) found evidence that self-reported Type A behaviour is reliably associated with chronic dysphoric emotions. Contrary to the traditional view, Type A does have some emotional concomitants and that should not be ignored (see, Sharma, Sood & Spielberger, 1999).

The role of TABP in CHD and other cardiovascular disorders also has been established (Kaushik et al., 1991; Friedman et al., 1996; Catiporic-Veselica et al., 1997; Dorado & Fernandez, 1997) TABP has also been found to be associated with other cardiovascular diseases such as MI (Spicer, Jackson & Scragg, 1993; Barry & Wassenaar, 1996; Lahad et al., 1997), coronary artery disease (Il'tze's et al., 1998) and atherosclerosis (Julkunen et al., 1992).

In summary, this study found evidence that TABP is reliably associated with EH/elevated BP and with chronic dysphoric emotions. Also that Type A has some emotional concomitants of negative effectivity like anger and anxiety which should be further studied. The following section deals with such important findings and issues.

6.2.2 Trait Anger in Hypertensives and Normotensives.

The concept of trait anger (T-Anger) in the present study has been operationalized as "the tendency to perceive a wide range of situations as annoying or frustrating, and the disposition to respond to such situations
with more frequent elevations in state anger.” Table 5.6 illustrates that proneness to anger (T-Anger) was significantly greater in the hypertensive when compared to their normotensive (surgical/orthopaedic) counterparts. Moreover, the nature and magnitude of difference in T-Anger holds good regardless of gender (2) or age subgrouping (2) of the patients (see Table 5.7 a,b,c).

Findings of earlier research report that hypertensives, as a group, tend to experience more anger than normotensives (Baer et al., 1979; Whitehead et al., 1977). Later Crane (1982) explored the relationship of T-Anger with hypertension among hypertensives and a group of general medical patient controls with no family history of hypertension using State-Trait Anger Scale (STAS). Findings state that greater trait anger was reported by the hypertensives. Crane is of the view that the hypertensives are expected to perceive a wider range of situations as anger provoking (e.g. annoying, frustrating, irritating), hence the greater disposition to experience anger (T-Anger) in them. In another study Deshields (1986) compared hypertensives to chronic patients (diabetes) control group and a non-patient control group. He observed that the hypertensive group yielded higher scores on trait anger (T-Anger) in comparison to the non-patient control group. Greater proneness to anger (T-Anger) in hypertensives was also reported by Kearns (1985) and Gorkin et al., (1986). Similarly Coelho et al., (1989) in a comparative study of hypertensives and normotensives observed that the anger score of hypertensives was higher.

In an early study Hartfield (1985) reported that hypertensives experienced longer lasting anger of greater intensity, felt more physical symptoms during anger episodes than the normotensives. Further, 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 (EH). Similarly Boutelle et al., (1987) in a study reported hypertensives (both males and females) to be eliciting greater proneness to anger when compared to their matched normotensive controls. Recently, Suls, Wan and Costa (1995) in a meta-analytic study reported that anger experience was correlated with elevated BP, but the relationship was small and highly variable.

Schwartz, Weinberger and Davidson (1981) investigated cardiovascular correlates of emotions including anger. It was found that anger produced a marked DBP increase with indirect evidence for high peripheral resistance along with activated baro receptor firing which attenuates HR in the presence of a pressor response. Likewise, in a study conducted in India, Ghosh (1989) reported that the essential hypertensive patients scored significantly higher on T-Anger than their surgical/orthopaedic counterparts.

Some researchers opined exaggerated cardiovascular response during behavioural stress/challenge to be a mechanism that links T-Anger/hostility to hypertension and coronary heart disease (Diamond, 1982; Krantz & Manuck, 1984). Also studies on the association between anger hostility or anger expression measures and the magnitude of the increase in BP and HR during laboratory challenges relative to resting values, do show-on-balance significant relation (Houston, 1988). Soarez and Williams (1990) reported that 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. Similarly, Jamner et al., (1991) found that high hostility workers showed larger BP increases during angry interchanges. However, contrary to above cited studies Suls and Wan (1993) conducted a meta-analysis of 28 reports
on the relationship between T-Anger/hostility and cardiovascular activity. They 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. Potential for Hostility-Interpersonal style was predictive of exaggerated SBP and DBP responses and the Cook - Medley Hostility Inventory was predictive of DBP responses to provocative stressors when stressors were classified as provocative versus non-provocative in accord with Trait X Situation approaches (see also another related meta-analytic review by Suls, Wan & Costa, 1995). Suls et al., (1995) specifically concluded that “there is little consistent support for a connection between anger and chronically elevated blood pressure” (p. 454). However, it is possible that this conclusion of Suls et al., relates, in part to their focus on trait-anger self-report scores derived by averaging across items, varying in their linkage to or specification of an interpersonal context. It is quite possible that methods with stronger interpersonal foci or contexts (e.g. structured interviews) may affect the strength of the association between anger and EH.

Findings of some studies report that gender and age influence EH/elevated BP. Gentry et al., (1982) reported that gender influenced SBP. Van der Ploeg et al., (1985) found that male hypertensives were more anger-prone than their normotensive counterparts. Further, Vogele, Jarvis and Cheeseman (1997) reported higher cardiovascular reactivity in males than in females and in subjects at a risk for hypertension. Rothenberg (1985) indicated that the frequency of motives for anger increased with age. In another study Ricci Biti et al., (1995) reported that the frequency and extent to which one experiences anger arousal had pathogenic effects. Higher levels of anger and hostility were present in older than in younger women. In addition to gender and age differentials per se perhaps, more important factors in BP for females could be the stress resulting from the interaction of
gender-role related domestic demands and opportunities (or lack of these) for socioeconomic activities (see Steele & McGarvey, 1997)

In the present study no age group difference in T-Anger was found either for males or females. It could be that age ranges were not wide enough to provide significant trends. Focusing on only T-Anger, the post hoc analysis by Suls et al., (1995) also suggest that age and gender do not influence the association of T-Anger and BP. Thus, the observed pattern in this study on the association of T-Anger and EH/elevated BP appear to generalize across gender or age of participants. The patients in this study were on medication and aware of their hypertensive status and there could be a labeling effect i.e. (impact of this awareness) on psychological measures i.e. these may reflect the effects of having a life-threatening disease. Further, cognitive and somatic side effects of antihypertensive medications can confound relationship of EH status and measures of psychological well-being. Moreso, a greater likelihood of people with high negative affectivity receiving a diagnosis of hypertension and making comparatively more routine visits to physicians than those with low negative affectivity cannot be ruled out.

Some researchers have also examined the role of trait anger in CHD and other cardiovascular disorders. Anthony (1989) examined the association of anger/hostility and chest pain with CHD in 542 patients (332 Males & 210 Females) who were administered the State-Trait Personality Inventory, Anger Expression Scale and Cook-Medley Hostility Scale. Patients with high anger/hostility scores were found to have CHD while those with lower scores were free of CHD. Ricci Bitti et al., (1995), further reported that greater levels of anger arousal eliciting situations and hostile outlook were observed more in women with CHD than in men. Earlier, Dembroski et al., (1985) and Suarez (1986) reported a significant interaction between potential for hostility
and anger, specially anger-in, posing a risk for coronary atherosclerosis. However, Julkunnen (1996) reported no such interaction. It may be worthwhile to examine possible interactions of anger proneness (T-Anger) with anger coping styles or anger-sub categories (AX/In or AX/Out or AX/Con) in jointly determining the level of BP and consequential ill-health outcomes.

6.2.3 Trait Anxiety in Hypertensives and Normotensives

In the present study the trait anxiety (T-Anxiety) has been defined as "the disposition to perceive external events or internal cues (thoughts, memories) as personality dangerous or threatening, and a corresponding tendency to respond to such threats with elevation in State-Anxiety." This study further revealed that the hypertensives reported significantly greater trait anxiety when compared to their normotensive controls (see Table 5.6). Similarly this holds good for gender (2) and age(2) sub-groupings (see Table 5.7 a,b & c).

Such a finding is, by and large consistent with the findings of earlier researchers, who have emphasized the role of T-Anxiety in the onset and exacerbation of idiopathic (essential) hypertension. For example, Johnson (1984) documented a significant positive relationship between anxiety and hypertension (see also James et al., 1986). Boutelle et al., (1987) compared 60 hypertensives (30 males and 30 females) aged 25 to 74 years, receiving treatment for EH (all having BP reading above 140/90 mm Hg) to matched controls (n= 60, males = 30, females = 30): They were matched on age and socio-economic status. The results revealed significantly higher scores on anxiety and neuroticism in hypertensives than in the controls (see also DeQuattro et al., 1981). Earlier, Van der Ploeg et al., (1985) investigated the role of anxiety in EH. Essential hypertensive patients (males = 69; females =
from out patient clinics of the University Hospital of Leiden and control group (n=104) from the general population completed the Dutch State-Trait Anxiety Inventory (STAI-DY). Significant differences in the levels of T-Anxiety in hypertensives (only males) was elicited. Such findings underline that as compared to normotensives, hypertensive are characterized by greater subjective feelings of tension, apprehension due to heightened ANS activity and by higher tendency to respond to such situations perceived as threatening is higher.

Average BP outside the clinic has proved to be a more important predictor of hypertensive complications in the study of Schneider et al., (1986). In their comparative study of hypertensives and normotensives they reported that anxiety (both trait and state) 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. Thus, the setting in which BP is recorded could be an important factor.

In a study Sullivan et al., (1981) reported positive correlations of BP with anxiety. York, Borkovec, Vasey and Stern (1987) reported that increased cardiovascular activity is positively related to worry and somatic anxiety. Coelho et al., (1988) reported that total anxiety score of hypertensives was higher than that of the normotensive controls (p < .02). They opine that psychological differences in hypertensives were a consequence of the raised BP and associated biological changes, which suggest that increase in the psychological disturbance parallels the severity of EH. In another study Pogotto, Fallo, Fava, Boscaro and Sonino (1992) reported greater levels of anxiety in hypertensives. Recently, in Canada, Greenglass (1996) observed higher T-Anxiety in both male and female respondents prone to hypertension. In earlier studies not only T-Anger but also T-Anxiety
Discussion

Significantly contributed to the etiology of EH (see Crane, 1982; Hartfield, 1985; Spielberger et al., 1991).

Some Indian studies have investigated the pattern of anxiety in coronary patients. Safar et al., (1978) reported 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. Pestonjee and Bagchi (1979) as compared to controls found coronary patients to be more anxious about their health, success or failure of their ambitions, family, friendship, love, social relations and their future. Later Bhargava, Sharma and Agarwal (1980) observed higher neuroticism, free-floating and somatic anxiety in coronary subjects as compared to the normals. Further, Chaudhary, Singh and Bhardwaj (1994) reported that patients with idiopathic hypertension were significantly more anxious and depressed than the normal controls and medically ill patients. Kumari (1995) later reported that majority of the EH patients were highly anxious, highly neurotic and lowly adjusted. Earlier, Ghosh (1989) found that EH patients experienced significantly higher trait anxiety (T-Anxiety) than the surgical/orthopaedic patient controls. Recent findings by Sharma et al., (1996) report that higher T-Anxiety scores of both male and female hypertensives when compared to their normotensive (surgical/orthopaedic) counterparts, provide strong evidence that high anxiety could be a concomitant of EH. Ghosh and Sharma (1998) further confirmed this conclusion.

However, some researchers have reported contradictory findings. For example, Foster and Bell (1983) after studying essential hypertensives and their normotensive spouses established that no significant differences exist between hypertensives and their normotensive spouses on trait and state...
anxiety. Similarly, Knight et al., (1987) did not find any effect of anxiety on blood pressure in general population.

Several studies provide evidence of the role of trait anxiety in CHD and other cardiovascular diseases. Recently, Frasure, Lesperance and Talajic (1995) reported that besides other factors anxiety significantly predicted cardiac events. Smith et al., (1996) examined relations between psychological factors and CHD in 1994 angina patients (aged 40-69 years), 47 non cardiac patients (aged 40-69 years) and 217 adults (aged 30-70 years). Results stated that in the angina patients anxiety and depression scores were significantly higher than in each of the other two groups and that they also reported the DSM-III criteria of anxiety and depression more often. As compared to the controls, relatively higher trait anxiety was found in CHD, amenorrhoea and psychosomatic Indian patients (e.g. Ansari et al., 1979; Chattopadhyay et al., 1979; Bhargava et al., 1980; Shanmugam & Kalippan, 1982; Katiyar et al., 1989). Pestonjee and Bagchi (1978) in an earlier study reported that anxiety was higher in myocardial infarction (MI) patients during hospitalization as well as after discharge as compared to the normative scores provided on Sinha’s Manifest Anxiety Scale. In another study by Katiyar et al., (1989) MI patients scored significantly higher on neuroticism than the controls.

It could be that individual differences in T-Anxiety (anxiety proneness) alongwith T-Anger, contribute to the etiology of EH and related disorders by enhancing anger suppression (see Sharma et al., 1996). However, an alternative interpretation is that hypertensive patients who are "aware" of their diagnosis (as in this study) experience anxiety more frequently because such individuals may be more concerned about, and sensitized to, the hazards of experiencing and expressing anger.

As such cross-sectional research, however, does not permit cause-effect generalization, as such studies do not clarify whether high trait anxiety
as another marker of negative affectivity is an antecedent or concomitant of EH/elevated BP. Even if it turns out to be both a predisposing as well as a concomitant variable it will be beneficial to know the extent. Thus, more research, particularly longitudinal, would be needed to clarify the role of T-Anxiety in the onset and exacerbation of EH/elevated BP. As was the case with T-Anger, the influence of knowledge of EH status on T-Anxiety (a kind of labelling effect) cannot be ruled out.

6.3 Anger Expression and its Modes (Anger-In, Anger-Out and Anger-Control) in Hypertensives and Normotensives

6.3.1 Anger-Expression (AX/EX) in Patient Groups

Total anger expression (AX/EX) comprises the responses of the 24 items of the AX/In, AX/Out and AX/Con scales, and provides for a general index of the frequency that anger is expressed, regardless of the direction of expression. Thus, AX/EX score is a composite score and is computed by subtracting AX/Con score from the total AX/In and AX/Out scores and adding a constant of 16. This constant of 16 precludes the possibility of a negative AX/EX score. The equation thus is

\[ AX/In + AX/Out - AX/Con + 16 \]

Vide Table 5.8, it was seen that the hypertensives reported significantly higher over-all anger expression than the normotensives (surgical/orthopaedic controls). In other words the hypertensives were relatively higher on the frequency of anger expression regardless of direction of expression when compared to their normotensive counterparts. On following Table 5.9 similar trends can be seen. When hypertensives were
matched to the normotensives with respect to gender (5.9 a) and age 5.9 b & c), the hypertensives reported significantly greater total anger expression regardless of direction of expression.

A comparative study of anger expression and its various dimensions/modes reveals that the hypertensives reported more suppression (AX/In) and relatively less expression (AX/Out) and also less control (AX/Con) of their angry feelings than their normotensive (surgical/orthopaedic) counterparts. The latter, however, reported relatively higher anger expression (AX/Out) as well as control (AX/Con) of angry feelings. This evidence of higher overall anger expression (AX/EX) in the hypertensives is mainly due to the greater suppression and lower expression as well as lower control of angry feelings. As such, the higher suppression of anger which was evident in the hypertensives may be viewed as having a greater role in the etiology of essential hypertension.

The direction of the association between anger-expression and reactivity has been found to vary across studies. Diamond (1982) and Krantz and Manuck (1984) opine exaggerated cardiovascular response during behavioural challenge to be a mechanism that links styles of anger expression and hostility with hypertension and coronary heart disease. Johnson (1984) found that total anger expression significantly and positively correlated with SBP, however, it was opposite in direction for both males and females. In an earlier study Gentry et al., (1982) reported that anger expression was related to SBP but only in females. Sommers-Flanagan and Greenberg (1989) in review of findings concluded that the connection between elevated BP and anger-expression was confirmed more strongly. Coelho, Hughes, Fonnesca and Bond (1989) in a comparative study of hypertensives and normotensives found that the hypertensives reported greater anger than the normotensives.
Recently, Vögele et al. (1997) found that the hypertensive males reported higher cardiovascular reactivity than subjects at a risk of hypertension.

In a recent Indian study, Ghosh and Sharma (1998) also reported significantly higher total anger expression (p < .001) in hypertensives than in normotensives.

Earlier, Engebretson et al., (1989) argued that expressing anger in a preferred or non-preferred way has quite different effects on cardiovascular responses during the following anger expression. Further, regardless of the preferred mode of anger expression involving expression or suppression of angry affect it was expected that cardiovascular reactivity will be more elevated among subjects who were induced to use a manner of anger expression that was inconsistent with their preferred mode of anger expression stating that individuals get used to their preferred mode of anger coping and may be differentially reinforced. Engebretson et al., in their study found that participants who perceived the confederate to have negative characteristics exhibited elevated SBP responses during the tasks, concluding that enduring negative conditions about another person may be more important factors than more transient angry effect in determining physiological responses. Also that individuals who by virtue of their environment or behaviour are often in anger-provoking situations have heightened BP, even after they no longer are directly confronted by the situation and accordingly, should be at elevated risk for cardiovascular disease. This further explains the higher incidence of EH and other cardiovascular disorders in populations facing racial discrimination as they are consistently exposed to anger-provoking situations.

Similarly, Glass et al., (1983) and Diamond et al., (1984), state that an annoying and harassing confederate led to elevated BP and HR in the participants regardless of their style of coping with anger-producing
situation. The harassed participants still showed elevated SBP relative to the pretask baseline values even 25 minutes after working with the annoying confederate thus suggesting that it takes a substantial period of time to recover from harassing interpersonal situations. Also that individuals frequently exposed to unpleasant and annoying interpersonal interactions at work and at home may be exhibiting elevated systolic blood pressure substantially after an interaction is terminated.

Studies report that anger causes elevation in BP in primary hostile, as opposed to non-hostile individuals. Suarez and Williams (1990) reported that anger does not have similar effects on the BP of non-hostile individuals. Consistent with these findings Jamner et al., (1991) found that high hostility score workers showed larger BP increases during angry interchanges than did those with low hostility scores. Christensen and Smith (1993) reported heightened BP reactivity during social interaction in hostile subjects when compared to those low in hostility.

Recently Suarez et al., (1998) reported that harassed subjects with high hostility scores exhibited enhanced and prolonged BP and HR, thus, highlighting the importance of situational context. Continuing with the contextual approach these authors also found that while stress-induced cardiovascular reactivity was associated with the arousal of negative affects such as anger, excessive behaviourally-induced cardiovascular reactivity was associated with interpersonal situations. Earlier Houston (1988) reported that studies between various anger, hostility or anger expression measures and magnitude of the increase in blood pressure and heart rate during laboratory challenges, relative to resting values, do show, on balance, significant relations. Ricci Bitti et al., (1995) recently stated that the particular components of anger-hostility are important in the study of psychological risk factors of EH and CHD. These findings thus state that the closer link between anger
and emotions and physiological hyperactivity could contribute to the greater health problems observed among hostile persons.

Further, investigators have also stated that the degree to which the expressive, experiential and physiological emotion components correspond to one another varies depending on a number of social cultural and situational factors (e.g. Ekman, Frieson & Ellsworth, 1982; Aldelmann & Zajonc, 1989; Lang, Bradley & Cuthbert, 1990; Miller & Kozak, 1993; Singh & Misra, 1997; Guyll & Contrada, 1998).

Anger-expression and hostility have become key concepts for researchers and have potentially important links to human well-being, ranging from health risks to extreme modes of violence. Although the exact nature of their role cannot be pinpointed yet they play an important role in various cardiovascular disorders such as CHD and atherosclerosis (Siegman, Dembroski & Ringel, 1987; Smith, 1992; Williams, Haney, Lee, Kong, Blumenthal & Whalen, 1980). Wenneberg et al., (1997) state that modes of anger expression may be associated with increased platelet aggregation and could provide a mechanism for the punitive connection between anger/hostility and CHD. Seigel (1984) states that both the multidimensional nature of anger and of the association of the dimensions of anger with indices of cardiovascular risk requires systematic study.

6.3.2 Anger-In (AX/In) in Patient Groups

Suppressed anger (anger-in) refers to “individual differences in the frequency that angry feelings are held in or suppressed.”

As evident from Table 5.8, the present study revealed that the hypertensives used significantly more anger-in mode of anger expression than the normotensive (surgical/orthopaedic) controls. Further, the same mode of anger expression (i.e. anger-in) was evident in the hypertensives
when sub-categorised into gender or age groups (see Table 5.9 a,b & c).

These findings are consistent with earlier related research. Findings state that although the angry and hostile impulses are mobilized in preparation for over aggression, yet, this physiologic activation is not discharged because people with EH ostensibly fear retaliation and loss of social approval by those who provide interpersonal distress. As such persons with EH have been described as passive, submissive, unassertive and prone to suppress anger and hostility (Diamond, 1982; Jern, 1986; Jorgensen & Houston, 1986; Weiner & Sapira, 1987; Sommers - Flanagan & Greenberg, 1989; Johnson & Spielberger, 1992; Jorgensen et al., 1992). Similarly, Hartfield (1985) in a comparative study of hypertensives and normotensives found that the hypertensives expressed less anger and used more distancing, self-control and escape-avoidance coping than the normotensives.

Further, Holroyd and Gorkin (1983) and Dimsdale et al., (1986) indicate that holding anger-in is a critical factor in the development in EH. Van der Ploeg et al., (1985) reported that hypertensives although feeling irritated and angry retreat to some extent from showing their anger. Schneider et al., (1986) contend that anger-in rather than anger proneness is a critical factor in the development of EH. Further Cottington et al., (1986) reported that the interaction between suppressed anger and job stress significantly predicts hypertension status. Julius et al., (1986) found that subjects suppressing their anger had significantly higher mortality risk than those expressing their anger and that the excess mortality rate among these subjects seemed to be mediated by elevated BP in persons high on anger-in (AX/In). The importance of suppressed anger (anger-in) in the etiology of essential hypertension (EH) has further been emphasized by and Vögelle et al., (1997).
Earlier, Diamond (1982) reported that the chronic inhibition of verbal and motoric expression of intense emotions fosters a chronic activation of the sympathetic nervous system which contributes to changes in the vasculature underlying essential hypertension (EH). Moreso, Boutelle, Epstein and Ruddy,(1987), found that inhibition of anger is the cause of heightened autonomic arousal and hypertension.

The potential link between emotional suppression and cardiovascular reactivity is suggested by the stress tradition. The finding that inhibiting moderate levels of emotional expressive behaviour leads to increased sympathetic activation of the cardiovascular system raises the possibility that suppression may activate some elements of the classic stress response, which in turn may influence the nature and course of immune responding (Pennebaker, Kiecolt-Glaser & Glaser, 1988; Esterling, Antoni, Kumar & Schneirman, 1990; Felten & Felten, 1994). An explanatory approach views increase in BP as a consequence of an increased secretion of renin after anger stress particularly suppression of anger (Thailer, Friedman, Harshfield & Pickering, 1985; Muller, 1988). Spielberger et al., (1985) reported a significant and positive relationship between anger-in an systolic and diastolic blood pressure. Earlier Johnson (1984) reported similar findings in both men and women. The anger-in-BP interactions were also reported by Baron and Kenny (1986), Sommers-Flanagan and Greenberg (1989) and Cox and Ferguson (1991). One of the 13-international, well-controlled laboratory studies on non-clinical population reviewed by Vogele and Steptoe (1993) states that “anger-suppressors” exhibited the strongest SBP reactions. Gentry et al., (1982) in an earlier study of blacks and whites also reported that anger-in is associated with hypertension i.e. SBP in black females living in high sociological stressful area. They also illuminated the importance of sociodemographic factors and socioecological niche influencing the
magnitude of difference in mean BP and risk for hostility attributable to chronic suppressed anger. Further, the view that chronic hostility and anger inhibition (anger-in) may be linked to hypertension and CHD has also been supported by Friedman and Booth-Kewley (1987 and Steptoe, 1993). Julkunen and Korhonen (1993) revealed a significant and positive association between anger-in and elevated BP in women who experienced their work unit as hostile and tense.

Further, regardless of gender higher anger-in (suppressed anger)) was observed in hypertensives than in normotensives in two Indian studies (Sharma et al., 1996; Ghosh and Sharma, 1998). These investigators reported that the correlations in respect of the hypertensives demonstrated that suppressed anger was more strongly related to elevated SBP as well as DBP. However, no such relationship was observed in the normotensive counterparts, thus stating the importance of suppressed anger in the etiology of essential hypertension.

The meta-analysis by Jorgensen et al., (1996) to date is the most comprehensive evaluation of the empirical validity of the psychogenic evaluation of the empirical validity of the psychogenic perspective with regard to hypertension. This metanalysis of 295 relevant effect sizes obtained from 25,469 participants confirmed expectations that elevated blood pressure (BP) and essential hypertension (EH) would be associated with lower effect expression or higher anger-suppression. They further demonstrated that the prediction of EH can be maximised if anger-suppression measures are used that are linked to specific social contexts (p.312).

There are also some researchers that report findings which are not supportive of the one observed in the present study. Among them are Mann (1986), Knight, Paulin and Wall-Manning (1987), Mills, Schneider and Dimsdale (1989) and Smith and Hosuton, (1987).
However, majority of findings are consistent with those of the present study stating suppressed anger to be a significant variable in the etiology of EH/elevated BP. It appears that inhibition of anger causes heightened autonomic arousal, which eventually leads to hypertension. The light control of negative emotions may adversely affect physical health. Though it is not clear how it might happen, but the underlying premise usually is that inhibiting emotion leads to acute increase in physiological response parameter that may in the long run, do damage. The frequent and dominant use of anger-in by the hypertensives can be attributed to their fear of retaliation as well as loss of social approval and social diserability. Analyzing the recent empirical studies one finds that both men and women’s expressive behaviour is particularly susceptible to modification by various social factors (Buck et al., 1992; Frijda, 1993; Fridlund, 1994; Levenson, 1994; Halberstadt et al., 1995; Gross & John, 1997). Particularly expressive behaviour in social situations is believed to be influenced by socially and culturally determined display rules - i.e., social and cultural standards about how and when to express emotion. Another reason for this suppression of anger could be due to the defined gender roles and that men and women are differentially reinforced for expressing emotion.

The relationship between anger expression and coping styles has received less attention but there is a strong reason to believe that people differing in anger expression will also show differences in pattern of coping. Although few studies have directly examined the relationship between anger expression and coping styles there are those that have suggested that individuals differing in anger expression also differ in coping. Among engineers in India, Sharma and Acharya (1989) found that suppressed anger was associated with what they term avoidance coping (also see Schneider et al., 1986). One significant psychological process mediating between
suppressed anger and EH could thus be the more frequent use of avoidance coping.

Similar findings with regard to the role of anger-in in the progression of other cardiovascular disorders have been reported by some investigators. Hynes et al., (1980) reported that the inhibition of angry feelings may increase the risk for CHD. Julkunen et al., (1992) observed a synergistic impact of hostility and anger-suppression (AX/In) on the progression of atherosclerosis. Similar findings were earlier observed by Dembroski et al., (1985) and Suraez, (1986).

6.3.3 Anger-Out (AX/Out) in Patient Groups.

A further follow up of Table 5.8 reveals that the normotensives (surgical/orthopaedic) reported significantly higher anger-out (AX/Out) than the hypertensives. Similar trends were observed in the two gender and age subcategories (Table 5.9, a,b & c). This is converse of what was seen in case of anger-in (AX/In).

Anger-out is defined as individual differences in the frequency that state-anger is expressed in aggressive behaviour directed towards other people or objects in the environment. This elicits that people with essential hypertension resort less to expressing their anger towards other people or objects in the environment when compared with their normotensive controls.

Johnson (1984) found that for higher levels of anger-out, SBP for both non-clinical men and women was substantially lower (F= 5.48, p < .001) and lower DBP was associated with higher anger-out scores. Earlier, Goldstein, Edelberg, Meier and Davis (1988) reported that expressed anger (anger-out) was inversely related to SBP and DBP. The normotensives showed significant associations between expressed anger and DBP, family expressed anger and SBP and experienced anger and DBP (see also James et al., 1986).
Earlier Jern, (1986) reported inverse association between high BP and overt behaviour associated with assertion, dominance, anger hostility and emotional expression. Recently Rüddel, Schächinger, Quirrenbach and Otten (1993) found no relation between occasional measurements of BP and anger expression. Further analysis revealed that while aggregated 24 hour DBP scores had a significant positive correlation with suppressed anger, a negative correlation with externally directed anger (anger-out) was found and that there were diurnal variations.

Gentry et al., (1982) found that in hypertensives externally directed anger (anger-out) score was associated with lower blood pressure. Mills, Schneider andDimsdale (1989) state that the ability to express anger outwardly may be related not only to reduce reactivity but to reduce blood pressure and incidence of heart disease. They also argued that reactivity is a potential risk factor for high blood pressure and cardiovascular disease. Manuck and Krantz, (1986) opine that, it may be the expression of anger outwardly and its possible association with lower reactivity is a mechanism mediating the maintenance of lower BP.

However, in some contradictory studies frequently expressed anger is linked to coronary malfunctioning (Mendes de Leon, Carlos and MecSteres, 1991). Diamond et al, (1984) reported anger-out to be associated with elevated BP. In a review of 13 international well-controlled studies on normal populations published between 1985 and 1992, Vögele and Steptoe (1993) found that in nine studies externally directed anger expression, or theoretically similar variables such as hostility were associated with SBP and/or high DBP. Also that externally directed anger was more strongly related to BP increases than suppressed anger. The authors also reviewed clinical studies and studies at-risk groups and found similar heterogeneous findings. However, they concluded that externally directed anger was
related more strongly to BP increases than suppressed anger. Siegman (1993) provided for similar conclusions. In the field and laboratory studies of anger-out and anger-in subscales of the STAXI, among healthy male normotensive students, Schwen Knezger and Hank (1996), found that in both the settings the students with higher anger-out scores showed higher BP than those who in this disposition to overtly express anger. No relationship was, however, found between suppressed anger and BP. In a recent study Seigman and Snow (1997) found that, of anger-out, anger-in and mood in congruent speech conditions only anger-out was associated with high cardiovascular reactivity levels. They state that the full-blown expression of anger in all of its paraverbal intensity is pathogenic and that mere inner experience of anger is not.

In an epidemiological study of men, Otten (1993) found no general relation between anger expression and cardiovascular activity when controlling for the following covariates: body weight, body size, alcohol consumption and age. However, he did find a negative (i.e. not positive) relation between extremely directed anger expression and SBP in a subgroup of hypertensives. It could be that such differential association of AX/In or AX/Out with SBP and/or DBP varies with the diagnostic status and its awareness by the respondent. Vögele and Steptoe (1993) considered that this inconsistency is also particularly due to the lack of standardized procedures for measuring habitual forms of anger induction. One central problem is that internally and externally directed anger expression are seen as the two poles of a bipolar dimension in certain cultures (see Sharma et al., 1996) and not as largely independent (see Spielberger, 1988).
6.3.4 Anger-Control (AX/Con) in Patient Groups

Anger-control has been defined as individual differences in the frequency that individuals attempt to control the outward expression of angry feelings.

This study revealed that respective of gender or age hypertensives reported lower anger control that the patient controls i.e. the normotensives. Stated otherwise, the hypertensives had less control over their angry feelings than the normotensives (see Tables 5.8 & 5.9).

A group of researchers argue that anger-control and anger-in scales reflect two distinct ways of dealing with anger (Spielberger, 1988; Spielberger et al., 1988; Julkunen et al., 1994). These authors further report that whereas anger-in is significantly associated with negative coping styles, a reverse pattern of features related to anger-control. Tanzer, Sim and Spielberger, (1996) found that in Singaporean Chinese women anger-control appeared to be an extremely important personality trait.

Ghosh and Sharma (1998) also observed lower anger (AX/Con) in male EH patients than controls. While in an earlier study Sharma et al., (1996) reported that there were no significant differences in the AX/Con scores of the hypertensives and normotensive groups for either gender. The higher AX/Con means relative to AX/In and AX/Out means in these studies either reflect better anger control among all subgroups or reflect to some extent the impact of social desirability variable.

Although a growing body of research has been reported with the AX/In scale, there is considerably less research in which AX/Con is measured. Given research interest in both AX/In and AX/Con, as well as their theoretical importance, studies are needed to establish their impact on health.
Recently Julkunen (1996) raised the issue titled as “Anger-In and Anger Control: Two Ways of suppressing Anger.” In general, the results of Julkunen et al., (1994) support the hypothesis that AX/In and AX/Con are designed to reflect two distinct ways of dealing with anger as proposed by the authors of the AX Scale (Spielberger, 1988). Julkunen (1996) also observed that AX/Con and AX/In were both indicators of anger expression. It has further been observed that AX/Con was significantly associated with several positive personality features, such as, self esteem and strong sense of coherence, optimism and less tendency toward depression. However, they seemed to be somewhat defensive, and relied more on repression than did persons scoring high on AX/In. In contrast AX/In was significantly associated with many negative coping styles, such as, resignation, withdrawal, avoidance and pessimism; has low self esteem along with a weak sense of coherence (see Julkunen et al., 1994; Sharma & Achaiya, 1989).

Given these results, it could be argued that persons relying on anger control rather than anger-in have more stable personalities with better psychological resources, and therefore are more capable of coping successfully with various life crisis. These data suggest that, in comparing two modes of suppressing anger, anger control should be a healthier way of dealing with angry feelings, whereas anger-in would pose a major health risk. An alternate hypothesis is, based on the general notion of the benefits of expressing feelings, would lead one to expect that either way, anger not expressed constitutes a health risk (e.g.Suan, Carmelli, Dame, Rosenman & Spielberger, 1992).

In any case, the present study highlights that the hypertensives had lower frequency with which they attempt to control expression of anger than the normotensives. Such findings if replicated in different settings have strong implications for developing anger-control interventions.
When the findings with respect to three modes of anger expression (coping) namely AX/In, AX/Out and AX/Con are seen together (Table 5.8, 5.9) it is clear that regardless of gender or age subgroup, the hypertensives had significantly higher Anger-In (AX/In) lower Anger-Out (AX/Out) and lower Anger-Control (AX/Con) than their surgical/orthopaedic controls. Similar findings were observed in earlier two studies on Indian hypertensives (Sharma et al., 1996; Ghosh & Sharma, 1998) and also with patients of chronic gastric ulcer - a non CHD illness (Sharma, Ghosh, & Spielberger, 1995). These findings suggest that Hindi AX/In, AX/Out and AX/Con scales define a unidimensional bipolar continuum, with AX/In and AX/Out as opposite poles and AX/Con at same point in the middle range of this continuum, and there are not independent dimensions as documented in North American cultures (e.g. Spielberger, 1988; Spielberger, Reheiser, & Sydeman, 1995). This contention is further supported by findings of Houseth, (1996); Sharma et al., (1996); Sharma et al., (1999) and Singh and Misra (1997) wherein highly significant inverse (negative)correlations were observed between AX/In and AX/Out subscales on different samples - suggesting a single Anger-In/Anger-out dimension. In the Singh and Misra (1997) study AX/Con was relatively and significantly related to AX/Out, and positively and significantly related to AX/In. In any case the factor structure for the Hindi AX scale is yet to be determined for large samples (clinical and nonclinical) and hence firm statement on the three modes of coping with anger cannot be made.

6.4 Life Stress, Type A Behaviour Pattern, Anger and Anxiety as Discriminators of Hypertensives and Normotensive Controls

Stepwise discriminant analysis (two-groups) was considered appropriate because the dependent variable is categorical (hypertensives vs.
normotensives), while all the independent variables were metric such as life events stress, TABP, T-Anger, AX/In, AX/Out, AX/Con and T-Anxiety (SPSS-PC +).

a) Relative Power of Discriminating Variables

Tables 5.10, 5.11(a), and 5.11(b), provided the following major findings:

(i) JAS - Type A Behaviour Pattern emerged as the leading discriminator;
(ii) Anger/Out, T-Anger and Anger/In were identified the next powerful discriminators in that order;
(iii) Negative impact of life events stress was the next potent discriminator; and
(iv) T-Anxiety, though significant, had the least discriminating power.

Thus, the best set of six discriminators was identified that significantly separated the patients with essential hypertension and their normotensive surgical/orthopaedic (control) counterparts.

With gender-wise comparisons (Tables 5.11 (a), 5.11(b), similar sets of discriminators in the same order of strength emerged for males or females. However, in both these gender groups, T-Anxiety (otherwise too a weaker variable) did not emerge as a discriminator of significance.

A special feature of this analysis is that the identification of these discriminators and their relative power has been achieved by a simultaneous use of multiple indices of discrimination, namely, F-to-remove, Wilk's Lamda - Wilk's Decrement, and Standardized Discriminant Function (SDF) Coefficients.

Furthermore, AX/EX was not included in the analysis because it is a linear combination of or sum of AX/In, AX/Out and AX/Con. Earlier, Klecka (1985) had also argued that no discriminating variables may be a
linear combination of other discriminating variable(s). As stated earlier, F-to-remove values indicate the rank order of discriminating variable power of selected variables (larger F-values indicate greater discriminating power); Wilk's Lambda and its decrement also provide similar information, including additional information on the unique contribution of the discriminating variable above and beyond its preceding counterpart. Further, SDF coefficients also demonstrate which variables contribute most to the determining of scores on the function as well as the classificatory strength. Thus the rank order of the discriminating strength (power) of a set of six discriminators (as observed in this study) was arrived at also by examining the magnitude of the SDF coefficients (ignoring signs). Since the SDF values were the highest for TABP (for total as well as for gender groupings), TABP has been identified as the most powerful or leading discriminator.

Furthermore, the positive signs of loadings for TABP, AX/In; T-Anger and negative impact of life events stress (for either male or female group) indicated that they made a positive contribution to the discriminant function. Likewise, AX/Out made a negative contribution to this function. The directions of group-means differences, as reported earlier, are generally consistent with the signs of these loadings.

The emergence of JAS-TABP and T-Anger along with anger coping styles (AX/Out, AX/In) as leading discriminators was anticipated. A research stream in life/occupational stress that has come from health sciences has focused on the understanding of stress-prone syndrome called Type A Behaviour Pattern. Since their pioneering work decades ago, Friedman and Rosenman (1974) described TABP as an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, instant struggle to achieve more and more in less and less time. The components of Type A Behaviour involve hostility, aggressiveness, competitiveness, and a
sense of time urgency, and all these together have been postulated and evidenced to be linked to chronic dysphoric emotions and an increased risk of coronary heart disease in retrospective and prospective studies (see Suls & Wan, 1989). The present study involved the use of Jenkins Activity Survey (JAS) Form-B for estimating TABP. As a consequence, the total TABP score in this study was the sum of the three independent factors labelled as: Factor S (Speed and Impatience), Factor J (Job Involvement); and Factor H (Hard-Driving and Competitive Speed and impatience (S) deals with the time urgency revealed in the style of behaviour of Type A person. Job involvement (J) refers to the degree of dedication to occupational activity. Hard-driving and competitive (H) involves perceptions of oneself as being hard-driving, competitive, conscientious, putting forth more effort than other people. Respectively, these three factors speak of the style of behaviours that characterize Type A behaviour. It is claimed that these three factor scores of the JAS make relatively independent contribution to the assessment of Type A tendencies. It needs to be highlighted that three components that comprise overall JAS-Type A Behaviour do not clearly and sufficiently include or emphasize the AHA! (Ager-Hostility-Agression) Syndrome. Hence the emergence of T-Anger and anger coping styles as the next powerful discriminators. Taken together, the JAS-TABP and Anger meet the requirements of complete conceptual description of TABP. Two recent meta-analytic reviews also document that chronic anger/hostility act as independent risk factors for elevated blood pressure and/or CHD (Jorgenson et al., 1996; Miller et al., 1996).

It is also important to recognize that intercorrelation between the self-report measures influences the outcome of discriminant analysis. For example, TABP and T-Anger either did not relate significantly ($r = 0.05$) in hypertensives or had a weak, though significant correlation in normotensives
(r = 0.32; \( p < .001 \)). Thus the constructs of JAS-TABP and T-Anger did not indicate substantial overlap and hence emerged as 2 out of 3 leading discriminators.

Findings further revealed T-Anxiety as significant but a weak discriminator (i.e. with the least strength in the set of 6 variables). In the present study, T-Anxiety had a highly significant and substantial relationship with T-Anger in hypertensives \( (r = 0.44, p < .001) \) and normotensives \( (r = 0.64, p < .001) \), indicating considerable overlap in these two constructs. In view of this, the small SDF loading (0.09) may indicate that either its corresponding variable T-Anxiety is not relevant, or that it has been partialled out of the relationship/discrimination because of its high degree of association with T-Anger. The emergence of T-Anger as a powerful discriminator had preceded the weak emergence of T-Anxiety as a discriminating variable.

The emergence of negative impact of life events stress as a significant discriminator not only emphasized its importance as a situational factor but also suggested that stress is more complicated than merely an accumulation of life events. There are certain mediating factors (like TABP, T-Anger) specific to each individual that may play more crucial role in their appraisal/impact than life events per se in determining the level of distress and health outcomes (see Sharma et al., 1999; Singh & Srivastava, 1999). The adults with chronic hostility/anger have been shown to have higher levels of intrapersonal distress and more stressful life events (Smith, 1994). Stressors include intrapersonal (e.g. divorce), financial and job-related stress (see Falger & Schouter, 1992). Most of these stressors are similar to the cluster of life events identified in the present study. Retrospectively it is also seen that EH/NT patients could be asked to recall life events of past 5 years i.e. at least 2 years prior to their diagnosis of essential hypertension/elevated blood pressure.
b) Classification accuracy

In respect of discriminant function analysis, another crucial question is the classification accuracy based on significant discriminators identified in preceding steps. The question of classification accuracy is crucial if the percentage of correct classification is significantly larger than that would be expected by chance (in two-groups, equal sample sizes, chance is 50%), the classification (predictive) accuracy of, for instance, 90% justify moving to interpretation stage. Some authors suggest that classification accuracy should be at least 25 percent greater than that achieved by chance. That is, if chance accuracy is 50 percent, the classification accuracy should be 62.5%. This criterion is easy to apply to groups with equal size.

Table 5.12, 5.13(a), and 5.13(b) clearly documented a near-perfect overall classification (between 98.70% to 92.7%) based on self-report measures of TABP, T-Anger, Anger/Out, Anger/In, negative life events stress and T-Anxiety. For the gender groups, the hit-ratios or classification accuracy for hypertensive males and females were 100% and 97.1% respectively. The corresponding values for normotensive males and females were 96.25% and 92.90%.

Such a very high percentage of cases correctly classified (all higher than minimum classification accuracy of 62.5%) indicates not only the accuracy of the procedure, but also indirectly confirms the degree of group separation i.e. between hypertensives and normotensives). In other words, very high percentage of "known" cases (essential hypertensives or normotensives) which are correctly classified provides an additional measure of group differences. As was the case in this study, Klecka (1985) also argued that we can use it along with overall Lamda and SDF coefficients to indicate the amount of discrimination contained in the variables.
In sum, the present investigation describes parameters of essential hypertension in males and females (middle age upwards) with particular focus on TABP, T-Anger, modes of anger coping (AX/In, AX/Out, AX/Con), negative impact of life events stress (LES) and T-Anxiety in a broader and different socio-cultural context. The patients with essential hypertension were clearly distinguished from control participant by elevation on TABP, T-Anger, Anger/In, negative impact of life events stress and trait anxiety.

The co-occurrence of situational variable (negative impact of life events stress) in interaction with personality/emotions (TABP, T-Anger, T-Anxiety) vis-à-vis essential hypertension suggests that psychological stressors, personality styles, experience and mode of expression of anger, trait anxiety and corresponding behavioural tendencies can synergistically influence the pathophysiology and etiology of essential hypertension.

Conclusion

The major conclusions of this study are:

(i) No significant differences emerged between the two patient groups on the total number of life events experiences by them in the past three years. However, specific clusterings of life events preceded EH which were predominantly familial and self-related events and pertained to 'LOSS'. Such a cluster holds good for gender or age subgroups, barring a few. However, no such clustering of life events was evident in normotensives.

(ii) Regardless of gender or age EH patients reported greater total as well as negative impact of life events than their surgical/orthopaedic normotensive controls. However a reversal in positive life changes was
observed in normotensives who experienced non-significant but greater positive impact.

(iii) Patients with EH (regardless of gender or age) were significantly higher on TABP, T-Anger and T-Anxiety than their matched normotensive counterparts.

(iv) The EH patients (irrespective of gender or age) in comparison to the normotensives reported significantly higher overall anger expression (AX/EX) as well as greater suppression of anger (AX/In). This implies the important role of the negative emotion anger in the etiology of EH.

(v) Anger-out and Anger-Control characterized the normotensive controls (regardless of gender or age) when compared to the hypertensives. This to an extend implied that the relative lesser use of these modes of coping with anger might be contributory factors to EH.

(vi) The significant discrimination between the hypertensives and the normotensives was mainly on account of TABP, T-Anger and anger-coping (AX/Out, AX/In) negative impact of life events and T-Anxiety. Similar findings emerged for male and female subgroupings, the exception being T-Anxiety.

Clinical Implications

This study assumes significance in view of the ever increasing incidence of psychophysiological disorders including EH - a major precursor of CVD, which in turn is a major cause of death in modern terms. In findings provide a clearer picture of the linkages of life events stress TABP
emotional configuration (anxiety, anger and the three modes of anger management (AX/In, AX/Out, AX/Con), and EH. The findings can lead to the emergence of an indigenous intervention package for Indian patients, which besides yoga, bio-relaxation, behavioural and cognitive technique would also necessitate the inclusion of anxiety and anger management. Effective strategies for controlling anger are urgently needed in treatment planning (Deffenbacher, 1992). Effective treatment requires that all aspects of anger phenomenon be assessed carefully, along with behaviours triggered by or associated with anger. Deffenbacher, (1992) found that high T-Anger individuals reported strong tendencies toward verbal and physical antagonism and less constructive behaviour, which suggested that these individuals are generally more abrupt, abrasive and intimidating. The verbal and nonverbal cues associated with such behaviour may elicit anger in others, leading them to withdraw or counterattack - the latter response is likely to stimulate further anger and aggression through T-Anger individuals. Effective treatment will require raising the high T-Anger person's awareness of the vicious cycle, and then training him or her to control the tendency to counterattack.

According to Deffenbacher, (1992) therapeutic strategies for dealing with anger and anxiety should include psychodynamic, behavioral and cognitive interventions to help people/patient perceive the world as less threatening. Research evidence indicates that relaxation exercises, social skills training and cognitive-behavioural interventions have proved effective in decreasing levels of anxiety and anger (Deffenbacher, 1992).

Research with the STAI and more recently with the STAXI and its subscales provides encouraging evidence of the utility of these inventories in treatment planning, and in the evaluation of treatment process and outcome.
There is a great potential for the use of STAXI and STAI to significantly further our understanding of important stress-based and stress-influenced syndromes and to help in identifying means by which such disorders may be reversed and prevented.

Limitations

This study has some limitations:

(i) This is a cross-sectional study and in a cross sectional analysis it is difficult to make assumptions on casualty. The negative emotions could be seen as consequences rather than the cause of essential hypertension.

(ii) The personality and emotional correlates of EH that are employed represent an excessive reliance on self-report measures that may not, for instance capture the behavioural tendencies linked to the suppression of anger within specific social contexts.

(iii) The hypertensives in this study were under medication and this might have affected their self-reports of negative affectivity. In other words the obtained scores and their relationships could in part be influenced by somatic side effects of antihypertensive medication. Since the BP (SBP & DBP) of EH patients were controlled to a large extent by medicines the variation (SD) was restricted. Hence BP could not be used as a continuous variable in the relationship of various indicators of TABP and negative emotions.

(iv) “Retrospective contamination” too cannot be ruled out. For instance, the hypertensive patients, because of awareness of their diagnosis may simply recall more negative life changes and assign more negative weightings to reported events than normotensives.
Suggestions

In the backdrop of the present study, some suggestions for future research are given below:

(i) Identification of specific/differential clusterings of life events in cardiovascular diseases needs to be stressed upon. Such an information will greatly assist in developing effective strategies of health promoting behaviours in such patients.

(ii) Alongwith monitoring stressors associated with daily hassles, the role of various situational mediators such as available social support needs to be further analyzed vis-à-vis cardiovascular/psychophysiological disorders.

(iii) Little is known about how combinations of coping responses patterns and constellations - influence well-being over a broad range of life settings (see Caplan, Naidu & Tripathi, 1984). As such future research is required to determine the extent to which different combinations of coping and defence aid or exacerbate cardiovascular disorders.

(iv) Future studies also need to consider socio-demographic variables like rural - urban, migration (rural to urban; from one culture to the other), socio-economic status, family set up, education, profession, gender along with psychosocial variables that contribute to the onset of such disorders.

(v) Prospective studies are required to clarify whether psychological factors are of aetiological significance in coronary disorders or are the result of the disabling illness itself.
(vi) Researches are needed to identify the role of personality characteristics (like locus of control, habitual coping, hardiness) vis-à-vis coronary disorders.

(vii) Future psychological research in coronary disorders needs to employ a multi-dimensional stress model, encompassing both environmental variables (stressors) and person-specific psychological and social variables.

(viii) Tight control of emotions may adversely affect physical health. Just how this might happen is not known, but the underlying premise usually is that inhibiting emotion leads to acute increases in physiological response parameters, that may, over long term, do damage (Krantz - Manuck, 1984). As such the issue “is the anger-in - BP relationship independent of the situational characteristics, or is it affected by characteristics of the context or other personality features” needs to be addressed (Julkunen & Korhonen, 1993).

(ix) Future research studies also need to take into consideration the issue of “what are the psychological implications of anger-in as a way of managing anger or hostility?” and “will anger-in pose a major health risk?”

(x) The fundamental psychophysiological mechanisms underlying the connection between anger and BP response are little known. As such to understand this mechanism future research needs to follow concepts based on psychophysiological reactivity during stress or anger exposure (Vögele & Steptoe, 1993), postulate pathophysiological reactions as a consequence of changes in behaviour following exposure to stress (e.g. increased consumption of alcohol & nicotine following emotional stress; Scherwitz &
Rugulies, 1992), as well as consider that there might be interactive relations (Krohne, 1990).

(xi) Several recent studies suggest that anger/hostility may be closely related to other risk factors for poor physical health, including age, gender, social status, smoking, family history, excessive alcohol intake, higher caffeine consumption, greater fat and caloric intake, lower physical activity, greater body mass, sleep problem etc. Such negative health behaviours are plausible links between anger/hostility and EH/CHD (see Siegler, 1994). The issue needs to be addressed is the extent to which statistical control for such risk factors diminishes the strength of the association between anger/hostility and EH/CHD, etc.

(xii) Psychophysiological studies on the impact of anger and its expression are particularly promising. Such studies have been carried out on samples from the normal population and in person already suffering from essential hypertension. Confounds with the consequences of disease (as in the present study) or risk factors can be avoided by studying normal populations and measuring cardiovascular reactivity in persons with different levels of habitual anger expression after experimentally induced exposure to stress or anger.

(xiii) The synergistic influence of occupational stressors, personality styles and emotional vital signs on EH/CHD and other illnesses need to be extensively proved across occupational/gender/age group/social class etc.

(xiv) Future research studies in this area may examine the effects of stress with measures other than self-report instruments as primary variables. For
example, physiological data pertaining to different psychophysiological disorders might be used in an attempt to identify the patients' response to stress and to try to elucidate the stress-psychophysiological disorder relation.

(xv) Future research studies need to focus the research on "salutogenesis" which involves identifying the factors that predict movement toward the health as well as the disease end of the health continuum (Antonovsky, 1987). This will aid in considering these factors in developing effective pharmacological as well as non-pharmacological interventions in cardiovascular/psychophysiological disorders.