CHAPTER - 1

INTRODUCTION

The 21st century which we are entering into is predisposing us to an ever-increasing psychological and physiological disorders. Of these psycho-physiological disorders, cardiovascular disorders represent the major cause of death. One of the most prominent and prevalent of these is hypertension. As a psycho-physiological disorder hypertension or elevated blood pressure (BP) tends to be asymptomatic and is not usually associated with specific complaints. The occasionally observed symptoms are occipital headache, nausea, enhanced fatigue and cardiac palpitations, all of which are not easily associated with one particular disease and do not necessarily lead to alarming responses. There is a continuum of cardiovascular risk associated with the level of BP, the higher the BP, the higher the risk of both coronary events and stroke (MacMohan, Pito, Cutier, Collins, Sorlie, Neaton, 1990). Although much is known about hypertension-related pathology of the heart, vasculature and kidney, the Central Nervous System (CNS) effects of BP remain poorly understood.

BP levels are normally distributed in a population, as a result an arbitrary definition of hypertension is inevitable. Hypertension is defined as a state of sustained systolic pressure equal to, or greater than 140-150 mm Hg (millimeter of mercury), and/or diastolic values equal to or greater than 90-95 mm Hg (Harrison, 1987). The World Health Organization (WHO, 1978), classifies blood pressure as normal at levels below 140/90 mm Hg. Recently, the WHO expert committee (1993) has re-defined the threshold values of hypertension, and has
identified 140/90 mm Hg the threshold values of systolic/diastolic BP as against 160/95 mm Hg recommended by the 1978 Expert-Committee. The current method of 24 hours continuous monitoring has revealed that there is fluctuation in BP levels to a considerable extent, with falls of 40-70 percent from the maximal level of systolic and diastolic pressure being recorded (Mann, 1986).

Borderline Hypertension is defined as a condition in which BP readings fluctuate above and below the normal levels (Julius & Esler, 1975). This condition is best known predictor of essential hypertension. Malignant hypertension includes those patients with accelerated hypertension with diastolic blood pressure (DBP) greater than 120 to 130 mm Hg and with extremely rapid vascular deterioration, acute renal failure or death if untreated (Ram, 1983). Malignant hypertension usually occurs in patients with previous essential hypertension, but may rarely be the initial presentation in previously normotensive patients (Houston, 1988).

Essential hypertension (EH) defined as a sustained elevation in BP of unknown cause is a major world wide problem affecting 15 to 20 percent of adult population (Griffin & Kee, 1986). The Joint National Committee (1988), defines essential hypertension as a sustained systolic blood pressure (SBP) of greater than 160 mm Hg and/or diastolic pressure greater than 90 mm Hg, and is a chronic disorder of unknown origin affecting 25% of the adult population of the United States (Kaplan, 1983). The American Heart Association has called the disease as the 'silent killer' for there are no typical symptoms sending early signals. By the time the symptoms appear, the disease has become well established (Davison and Neale, 1990; Mehra & Lashkari, 1992). Essential hypertension has been a major focus of biomedical and
psychosomatic research for decades since it has been identified as the largest risk factor for cardiovascular disorders (CVD).

In India 500 percent rise in the incidence of cardiovascular disorders since the advent of high technology has been found and about 20% of patients are below 40 years of age (Pahlanjani, 1992; see also Gupta, 1997; Kaushal, 1994). Epidemiological studies have also shown that irrespective of regional, cultural and religious differences immigrant South Asians (Indians, Pakistanis and Bangladeshis) share a higher mortality from Coronary Heart Disease (CHD) and other cardiovascular diseases than the indigenous white population (Balarajan, Aldelstein, Buluser, Shukla, 1984; McKeigue, Miller & Marmot, 1989). Recent findings by Bhatnagar, Anand, Durrington, Patel, Wander, Mackness, et al. (1995) report a genetic disposition in Punjabis to cardiovascular disease. However, Dhawan and Bray (1994) found no significant difference in the risk profiles or extent of coronary disease between Asian men settled in Britain and their counterparts living in India. Prevalence of hypertension in the US black population is among the highest of any group in the world (Akinkugbe, 1985). EH in blacks is approximately twice than that of white Americans and as a result blacks experience a high rate of hypertension related morbidity and mortality from CHD (Myers, 1986).

As evidence has focused on mechanisms linking psychological and behavioural factors to disease, it has been suggested that physiologic responsiveness to emotional stress might be a marker of pathogenic processes involved in the etiology of EH or CHD (see, Eliot, Buell & Dembroski, 1982; Herd, 1983; Krantz, Baum & Singer, 1983; Schneiderman, 1983). Empirical evidence suggests that psychological stress and emotional adjustment are associated with changes in
symptom complaints disability and physiological indices of disease over the course of illness (e.g. Smith & Gallo, 1994). However, Mann (1986) states that psychological factors do not play a convincing part in the etiology of sustained EH. These inconsistencies as a result call for further research in the related area.

Considerable research has focused on understanding the influence of psychological factors on physical health. Investigators have used prospective studies of community samples (e.g. Delongis, Coyne, Folkman, Dakof & Lazarus, 1982), to identify psychological factors associated with illness onset. Similar studies have been conducted by Smith and Gallo (1994). Kumari (1995), in a study on socio-psychological correlates of EH reported that majority of the patients were highly anxious, highly neurotic and lowly adjusted. Dressler, dos Santos, Jose and Viteri (1986), reported that highest mean blood pressures were observed among mixed race and black Brazilians who had low psychosocial resources. Afro-Brazilians with high psychosocial resources had low BPs than white Brazilians. Patel (1994) found a relationship between psychosocial stress and cardiovascular disease. Similar findings with regard to the relationship between psychosocial stress and cardiovascular disease have been reported by Dressler, dos Santos, Jose and Viteri (1986), Elliot (1995) Smith and Gallo (1994), and Wister, et al., (1994). In fact, the attempts to link psychosocial factors and personality markers to the pathophysiology of essential hypertension (EH) has been a major area of inquiry and dispute (see. Suls, Wan & Costa, 1995; Jorgenson et al., 1996).

Among the other variables influencing hypertension are family history (Anderson, 1988; Anderson & Jackson, 1987; Ditto, 1986; Hohn et al., 1983; Thomas et al., 1994; Waldstein, 1995), stress, due to natural
disasters, culture, urbanization, occupational stress, prolonged illness, experimentally induced stress (Burchfield, 1985; Hughes & Raval, 1989; Somova, Diarra & Jacobs, 1995; Weinberger, Hiner & Tierney, 1987), Type A Behaviour Pattern (TABP), (Seigel & Leitch 1981; Wright, Contrada & Glass, 1985; Sharma, Sood and Spielberger, 1999). Personality (Goldstein, 1981; Mellors, Boyle, Roberts, 1994), negative emotions like anger (Diamond, 1982; Dimsdale et al., 1986; Gentry et al., 1982; James, Yee, Harshfield, Blank et al., 1986; Spielberger et al., 1983; Spielberger, Johnson, Russell, Crane, Jacobs & Worden 1985; Sharma, Krishna & Spielberger, 1996; Suls et al., 1995; Ghosh & Sharma, 1998), anxiety (Chaudhary, Singh & Bhardwaj, 1994; Sharma et al., 1996; Ghosh & Sharma, 1998). The other factors which too play an important role are age (Anderson, 1988; Elias, Robbins, Schultz & Pierce, 1990) obesity (Patel, 1994; Singh & Kaushik, 1994), dietary sodium (Borghi, Costa, Boschi, Mussi & Ambrosioni, 1986; Kaplan, 1980), alcohol (INTERSALT, 1989; Meheshwaran, Gill, Davis & Beevers, 1991; Marmot, Elliott, Shipley, Dyer, Ueshima, Beevers, Stanner, Kesteloot, Rose and Stamler, 1994; Wannamethee & Shaper, 1991), gender (Shapiro & Goldstein, 1982), smoking (Patel, 1994) and caffeine (Reeves & Victor, 1982). Out of these variables, Life Stress, Type A Behaviour Pattern (TABP), Anger and Anxiety are reported to have a special influence on the progression of cardiovascular diseases.

Psycho-social stress forms an inseparable part of life and upto a degree may be essential for adequate personality growth. All individuals in the course of life experience a variety of events or life changes which may be considered potential stressors (such as death of spouse, marriage, divorce, unemployment etc.) and often require significant social readjustment and adaptation. These changes
represent an ongoing source of stress to which individuals are subject to a greater or lesser degree (Sarason, Johnson & Siegal, 1978). In the current studies, life event is defined as stressful if “it causes changes in and demands readjustment of an average person’s normal routine” (Dohrenwend & Dohrenwend, 1974; Holmes & Masuda, 1974). Canton (1985) in a longitudinal study reported that some life events such as loss, can be considered as a risk for hypertension (see also, Cottington et al., 1986; Hanfner & Miller, 1991; Hobfoll & Spielberger, 1992). It has been argued that undesirable events may have a very different and possibly a more detrimental effect on the individual than positive events (see Aggarwal & Naidu, 1988). Although research initially suggested that both positive and negative events were capable of producing physical disorders because of their capacity to force the individual to make changes and accommodations, research now indicates that negative events are substantially more potent in this regard than are positive ones (e.g. Suls & Mullen, 1981). Negative consequences of changes were stressful and not life change per se (Hobfoll & Spielberger, 1992). Studies by Lal, Ahuja and Madhukar (1982) and Mellors, et al., (1994) showed that the effects of hypertension were magnified when personality and life stress variables acted synergistically. Brackbill et al., (1995) documented that job loss was strongly related to self-rated hypertension.

Some Indian studies also showed that hypertensives reported more number of distressing life events and gave higher mean distress rating than normals (Lal, Ahuja & Madhukar, 1982). Gender differences in the perception of stressful life events have been observed (Dube et al., 1980). In CHD patients, a major change in work responsibility and death of a close relative have been reported by
significantly greater number of patients than normal controls (Bhargava, Sharma & Aggarwal, 1982). Some Indian researchers have attempted to identify specific clustering of life events that act as elicitors of a particular disease such as bronchial asthma, peptic ulcer and depressive disorders (see Dogra, 1991; Ghosh, 1992). No Indian study has dealt with specific clustering of life events that precede EH/CVD.

A number of physiological changes are observed during the experience of stress. These include elevations in heart rate, systolic and diastolic blood pressure, increased secretions of epinephrine and norepinephrine (Abott & Sutherland, 1991), and increased release of cortisol. In fact, with respect to the relationship between stress and well-being, the research literature is clear in pointing to a negative relationship between stress and both psychological and physical well-being (see related review by Stein & Miller, 1993).

Type A behaviour Pattern (TABP), one of the most promising coronary-prone behaviour pattern, with its combination of rush, impatience and competition is the most closely studied of the various personality factors involved in coronary diseases. It is a behavioural syndrome that includes aggressiveness, competitiveness, impatience and a sense of urgency in overcoming obstacles to task performance (Ivancevich & Matteson, 1984; Gastorf, 1980). Individuals not manifesting the preceding characteristics have been classified as Type B individuals.

Several studies have indicated that the relationship between Type A Behaviour and resting BP is weak and inconsistent, yet TABP individuals generally respond to stressful situations with marked increases in heart rate (HR) and BP (see Evans, Palsane & Carrere,
Type A scores for female college students have been observed to be associated with a family history of hypertension (Waldron et al., 1980). The Western Collaborative Group Study (Rosenman et al., 1976), the Framingham Study (Haynes, Feinleib & Kannel, 1980) and the French-Belgian Collaborative Group (1982), found that Type A behaviour predicated heart disease independently of other known risk factors. TABP has been linked to an increased risk of CHD in retrospective and perspective studies (Booth-Kewley & Friedman, 1987; Cooper, Detre & Weiss, 1981; Matthew, 1988; & Matthew & Haynes, 1986).

Meta-analytic reviews have, however, questioned the reliability of this metaconstruct of TABP as a predictor of EH/CHD incidence (see Jorgenson et al., 1996), though it could be an important influence as a precipitating factor. Moreover, TABP is composed of many components and not all of them predict EH/CHD. In fact, past research has paid relatively less attention to the emotional component of TABP (see Suls et al., 1995).

The best prediction of heart disease can possibly be obtained by measuring hostility/anger, the most important feature of TABP. A number of studies, both cross-sectional and prospective, have provided evidence that individuals who are higher in anger/hostility have higher levels of CAD (Coronary Artery Disease) as well as higher death rates from CHD. The evidence of hostility as a factor influencing health outcome was further confirmed by a recent meta-analytic review of 45 studies investigating relationship between hostility, CHD and all-cause mortality. This review found that hostility/anger is a significant independent risk factor for CHD even when other factors are controlled (Miller, Smith, Turner, Guijarro & Hallet, 1996).
Exaggerated forms of cardiovascular response during behavioural challenge have been proposed as a mechanism linking hostility/style of anger expression with risk of hypertension and coronary heart disease (Diamond, 1982; Krantz & Manuck, 1984). Research on the association between several 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 relations (Houston, 1988). The role of anger in the etiology of EH has been reported by some researchers (Crane, 1982; Hartfield, 1985; Spielberger et al., 1991; Sharma et al., 1996). However, the direction of association between anger expression and cardiovascular reactivity varies across studies. Findings have revealed that outward expression of anger (anger-out) is related to CHD, whereas suppression of anger (anger-in) is more common to hypertension (Seigel, 1984; Spielberger et al., 1985; Cottington et al., 1986; Schneider et al., 1986; Boutelle, Epstein and Ruddy, 1987; Suarez & Williams, 1990). In a recent study Sharma et al., (1996) examined the role of anger and anxiety in essential hypertension and found that, (i) the hypertensives of both sexes had a higher trait anxiety (T-Anxiety) than normotensives, and (ii) regardless of gender, the hypertensives had higher anger-in and lower anger-out than normotensives (see also Somova et al., 1995; Julkunen, 1996). All such studies support the traditionally dominant hypothesis in the psychological and psychosomatic literature on the impact of anger suppression on BP.

A significant positive relationship between anxiety and BP has also been reported in several studies (e.g. Schneider, Egan, Johnson, Drobny & Julius, 1986; Boutelle, Epstein & Ruddy, 1987; Sharma et al., 1996). However, Whitehead, Blackwell, De Silva & Robinson (1977)
found no relationship between the two. In comparative studies of anxiety levels in hypertensives and normotensives there were either no differences (Boutelle et al., 1987; Foster & Bell, 1983), or significant differences were found only for hypertensive males who reported greater anxiety than their normotensive counterparts (Van der Ploeg, Buuren & Brummelen, 1985; Chaudhary, Singh & Bhardwaj, 1994). High anxiety subjects had a significantly higher score on anger-in than low anxiety subjects (James, Yee, Harshfield, Blank et al, 1986). Likewise Sharkin and Gelso (1991) report that anger discomfort has a positive correlation with trait-anxiety. The relationship between T-Anger and T-Anxiety needs further scrutiny.

Many authors today argue that most important coronary prone element would be anger. In fact, emotional vital signs that are most critical to an individual’s well-being include not only anger but also anxiety and depression (Spielberger & Sydeman, 1994). All these are markers of negative affectivity and it might be important to examine these emotional configurations as precursors of EH.

Most of the preceding research, particularly in India, has favoured single-factor, univariate analysis disregarding possible interaction of age and gender. Moreover, it would be useful to study the combination of socio-cultural stressors and emotional vital signs (such as anger, anxiety) as they synergistically induce greater psychophysioligic activities related to essential hypertension. This suggestion is reinforced by other studies which have demonstrated significant correlations between stress and anger/hostility and its expression (Browman & Johnson, 1988; Houston & Kelly, 1989; Sharma, Sood & Spielberger, 1999).
The Present Study

The present research work considered the following:

(i) Psychophysiological disorder group of essential hypertension patients.

(ii) Control group of normotensive (surgical/orthopaedic patients).

(iii) The use of systematically developed culturally relevant Hindi language versions of Life Stress Scale (Ghosh, 1992), Jenkins Activity Survey - Form B (JAS Form B) (Sood, 1989), State-Trait Anger Expression Inventory : STAXI (Krishna, 1988; & Rana, 1990), and A - Trait Scale of STAI (Spielberger, Sharma & Singh, 1973). Since, there are inherent problems of applying a western-based knowledge to a culturally distinct country like India (Sinha & Holtzman, 1984), it necessitates the certification of the validity of such a research using well standardized culturally appropriate adapted tools of data collection. This will help establishing the validity of the western research across cultures.

Operational Definitions

(i) Essential Hypertension: Chronically elevated blood pressure (SBP ≥ 140 mm Hg and DBP ≥ 90 mm Hg) in the absence of obvious organic cause. (Harrison, 1987; WHO/ISH, 1993).

(ii) Life Stress: A life event is defined as stressful if it causes changes in and demands readjustment of an average person’s normal routine. The life events are distinguished as negative life changes and positive life changes.
(iii) **Total Frequency of Events**: Number of events regardless of positive, negative or neutral perception.

(a) **Negative Frequency**: Number of events perceived as negative.

(b) **Positive Frequency**: Number of events perceived as positive.

(iv) **Total Impact of Life Changes**: Total impact of events regardless of negative or positive perception.

(a) **Negative Impact of life changes**: Total impact of events perceived as negative.

(b) **Positive Impact of life changes**: Total impact of events perceived as positive.

(v) **Type A Behaviour Pattern**: It is an action-emotion complex that can be observed in any person who is aggressively involved in a chronic, incessant struggle to achieve more and more in less and less time, and if required to do so, against the opposing efforts of other things or other persons.

(vi) **Trait Anger (T-Anger)**: Individual differences in anger proneness, i.e. the tendency to perceive a wide range of situations as annoying or frustrating, and the disposition to respond to such situations with elevations in State-Anger.

(vii) **Trait Anxiety (T-Anxiety)**: Individual differences in anxiety proneness, i.e. differences between people in 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.
(viii) Anger-Expression (AX/EX): Anger expression (AX/EX) comprises the responses to the 24 items of the AX/In, AX/Out, AX/Con scales, and provides a general index of the frequency that anger is expressed regardless of the direction of expression.

(a) Anger-in (AX/In): Individual differences in the frequency that angry feelings are held in or suppressed.

(b) Anger-out (AX/out): Individual differences in the frequency that State-Anger is expressed in aggressive behaviour directed toward other people or objects in the environment.

(c) Anger-Control (AX/Con): Individual differences in the frequency that individuals attempt to control the outward expression of angry feelings.

Objectives of the Study

Main Objective

How do Life Event Stress, Type A Behaviour Pattern, T-Anger, T-Anxiety and modes of anger expression (AX/In, AX/Out, AX/Con) synergistically influence the pathophysiology of essential hypertension.

Specific Objectives

When compared to surgical/orthopaedic normotensive patient controls, this research will determine:

(i) relative magnitude of the total as well as the negative and positive impact of life events (changes) in EH patients,

(ii) disease-specific clustering of various stressful life events (changes) in EH patients,
(iii) levels of TABP, T-Anger and T-Anxiety in EH patients,
(iv) dominant mode of coping with anger (AX/In, AX/out, or AX/Con) employed by EH patients, and
(v) Optimal set of variables (Life Events Stress, TABP, T-Anger, T-Anxiety and modes of anger expression/coping) which in combination would maximally separate/discriminate EH patients from their normotensive controls.