CHAPTER - II

CONCEPTUAL FRAMEWORK

2.1 Cardiovascular Disorders: Categories and Prevalence

The rate of death from Cardiovascular disease though has been decreasing since 1964, it still remains a major source of death. Statistics compiled by WHO (1987) attribute 50 percent of mortalities in various European industrial nations to cardiovascular disorders. Coronary heart disease (CHD) is the greatest cause of death. It is a clinical disorder produced by lesions of the coronary arteries i.e., coronary artery disease (CAD), or atherosclerosis. There are two major manifestations of clinical CHD: (i) Myocardial Infarction (ii) Angina Pectoris. Myocardial Infarction (MI) involves necrosis of heart tissue caused by insufficient oxygen supply. Here a clot in the coronary artery obstructs the artery, thus, diminishing the blood supply leading to sudden death. It is commonly called a heart attack. Angina Pectoris involves a type of chest pain which arises when the heart muscle experiences anoxia because of an inadequate blood supply occasioned by obstruction of one or more coronary arteries. Atherosclerosis is a symptomless disease characterised by thickening of the coronary arteries due to accumulation of fatty deposits in the linings.

CHD has generally been acknowledged as a multifactorial disease. The risk factors found to be associated with CHD are age, gender, socioeconomic status, race, family history of CHD, physical inactivity, personality characteristics (specifically Type A behaviour), smoking, alcohol intake, diabetes mellitus, hypertension, obesity, total serum cholesterol and in women oral contraceptive use (Friedewald,
1988; Seipel and Jahle, 1992). A meta-analysis of CHD prevalence studies in India shows a nine-fold increase of CHD in urban populations from the 1960s to the 1990s and a two-fold increase in rural populations from the 1970s to the 1990s. The study also highlights a greater prevalence in younger age groups (20-39 years) and in urban and rural men and urban women (Gupta and Gupta, 1996). The higher incidence of CHD in the urban areas of developing countries may be attributed to maladaptation, stress of migration and social and ethnic influences involved in urbanization resulting in an epidemiological transition (Feinleib, 1995). Gupta (1995) and Singh and Niaz (1995) state that major coronary risk factors such as hypertension, truncal obesity and sedentary life style are significantly more prevalent in urban populations.

International comparisons show that emigrant South Asians have MI 2.5 to 5 times more frequently and 1.5 to 3 times higher mortality from total coronary heart disease compared to the endemic populations of Singapore, South Africa, England and USA (Marmot, et al., 1984; Mc Keigue, 1989; Enas & Mehta, 1995).

Among the cardiovascular disorders, one of the most common is Essential Hypertension (EH). It is characterised by chronically elevated BP in the absence of obvious organic cause. BP being labile displays marked elevations from time to time along with reductions in pressure. A chronic phase eventually is reached in which the BP remains consistently high. This condition, in turn, causes organic changes in the heart and blood vessels.

Elevated BP meets most criteria for a direct cause of premature death and cardiovascular disease. About 20 percent of adults suffer from hypertension and according to statistics the number keeps
increasing. About 40 percent of all deaths below the age of 65 years are
due to the consequences of high BP. Varying degrees of hypertension
are found in 15 to 33 percent of the adult population of the US
according to the recent estimates. With a prevalence of approximately
20 percent it is the most frequent cardiovascular disease in Germany
(Trenkwalder et al., 1994). Currently some 25 percent of all causes of
death must be reckoned to be associated with hypertension which is
either not treated at all or inadequately treated and this points to the
importance of possible cardiovascular sequelae. It is a powerful
predisposing factor to CHD, now its most common sequelae. Health
officials and clinicians report 35 to 45 percent of the annual toll of
cardiovascular morbidity and mortality to be directly attributable to
hypertension. A correlation between the degree of hypertension and
mortality, as well as the emergence of severe cardiovascular
complications has been established (Marmot, 1986).

Effects of hypertension are mainly borne by the heart but blood
vessels in other parts of the body also take part. The heart maintains a
normal cardiac output against increased peripheral resistance through
an increased action of its fibres which leads to hypertrophy (stage of
compensation). But when the heart no longer hypertrophies and
instead dilates, it leads to cardiac failure (stage of decompensation).
When the heart hypertrophies, its blood supply does not increase in
proportion to its size leading to ischaemia. Hypertension, in association
with atherosclerosis, predisposes to angina pectoris, coronary occlusion
and MI. Apart from its effects on the cardiovascular system it often
causes atheroma of the cerebral vessels causing thrombosis or rupture
of these vessels leading to hypertensive encephalopathy. This condition
is marked by spasms of the cerebral vessels temporarily giving rise to
cerebral ischaemia and oedema in the presence of sudden BP increases due to excitement or any other emotional condition. Benign or malignant nephrosclerosis due to arteriosclerotic changes in the kidney is another consequence of hypertension. Various changes also occur in the retinal vessels according to the increase of the BP.

2.2 Concept and Theoretical Model of Essential Hypertension (EH)

Essential hypertension (EH), commonly known as high blood pressure is a psychophysiological disorder and the main problem linked with it is its risk potential. Since in most cases no symptoms are noticeable over long periods, the disease often is well established before treatment is initiated. It, therefore, is also known as the 'silent killer' as people may go on for years without knowing its presence.

Organically, the elevation in BP is caused by a constriction of blood vessels and malfunction of a set of nerves called baroreceptors which ordinarily depress BP when a critical level is reached. BP may be elevated by increased cardiac output, the amount of blood leaving the left ventricle of heart per minute; by vasoconstriction (i.e. increased resistance to the passage of blood through arteries); and by increase in volume of body fluids. The physiological mechanisms contributing to the regulation of BP are very complex. The central nervous system (CNS), the sympathetic nervous system (SNS) along with the hormones and salt and water mechanisms are involved.

Houston (1989) presents a comprehensive view of the pathophysiological process in which BP is a function of cardiac output (CO) determined by stroke volume (SV) - a function of preload, afterload and inotropic state - and heart rate (HR) - and systematic
FIGURE 2.1 Pressor Mechanism in Essential Hypertension.

vascular resistance (SVR), is a function of peripheral vascular resistance (PVR) and renal vascular resistance (RVR), as can be seen in the following equation:

\[
BP = \frac{CO \times SVR}{SV \times HR \times PVR \times RVR}
\]

The baro-receptor and hypothalamic connection has also been found to play a role in EH.

Appropriate blood volume and BP is maintained in the body by the balance of sodium and water in the body. Inhibited sodium excretion results in volume expansion and elevated BP (Vick, 1984). Sodium exerts its pressor effects through increased vascular resistance and neurogenic vasoconstriction. High levels of plasma renin too have been found to contribute to essential hypertension, as such the renin-angiotension system is being blamed to play a possible role in the development and maintenance of EH. Further the Sympathetic Nervous System (SNS) is claimed to play a permissive role in the maintenance of EH. However, its possible initiating role though plausible awaits further confirmation. Researchers view that extrinsically determined vasoconstriction due to systematic pressor activity could result from the renin-angiotension system or in the adrenergic system or both or from other unidentified factors. It appears that peripheral resistance remains at a higher level in hypertensives than in normotensives. Both the functional and structural components of the increase in vascular resistance may be caused by vasoconstrictor stimuli, either originating from the tissues (autoregulation) or from increased activity on the part of the pressor
system. In essential hypertension no direct information regarding autoregulation is available however. An elevated peripheral resistance with normal or subnormal cardiac output are considered to be the prevalent pattern in essential hypertension. Figure 2.1 demonstrates how sodium excess, stress, genes and obesity activate the pressor system in essential hypertension.

EH is not a single disease entity but rather a heterogeneous disorder potentially initiated and maintained through multiple interacting mechanisms rather than from a single source. Genetic, nutritional, behavioural, psychological, social and environmental factors are significant contributors to the disorder (Anderson, 1988). The importance of behaviourally and environmentally induced changes in sympathetic activity grew out of animal research that demonstrated that factors such as crowding, territorial conflict and other psychosocial stressors produced sustained high BP. This sustained high BP is preceded by transient activation of the "defense" or "fight-flight" pattern of beta-adrenergically mediated sympathetic outflow. Earlier Obrist (1981) hypothesized that the repeated elicitation of this beta-adrenergic response pattern, specially during behavioural challenges that evoke coping, might ultimately lead to sustained high BP. It also increases cardiac output and blood and oxygen supply to bodily tissues, frequently in excess to metabolic needs (Turner & Carroll, 1985). When this occurs, autoregulatory processes are set in motion that eventuate in an elevated peripheral resistance and higher BP.

EH has long been classified as a neurohumorally controllable disease related to the social and psychological factors of human living. In this case the most important social factors are occupation,
FIGURE 2.2 Theoretical model demonstrating the possible relationships among personality factors, emotional factors, various possible mediating factors and essential hypertension.
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professional training, promotion, conflicts at the work place, changes of job, financial position, family life, interpersonal relations, psychological homeostasis in the family, stress-life-physical and psychological and the presence and significance of emotive stimuli. Negative affectivity (e.g. anger, anxiety, hostility) are particularly related to EH. Emotions cause the fixation of processes of the molecular level within the CNS, which occasionally results in irreversible pathological changes in the organs and tissues.

Anger and anxiety have been found to be the most important emotional variables in the etiology of EH. (Hartfield, 1985; Spielberger et al., 1991; Sharma et al., 1996). Of the three modes of anger expression i.e. suppressed anger (anger-in), outwardly expressed anger (anger-out) and anger control (anger-con), anger-in has been found to have a positive relation with EH (Spielberger et al., 1985; Schneider et al., 1986). Though some other researchers did not find any relation between the two (Knight et al., 1987; Smith & Houston, 1987; Mills et al., 1989).

Personality features common to hypertensive patients are stubbornness, secretiveness, dissatisfaction with oneself and others, hostile attitude and aggressive tendencies in behaviour. Very sensitive to criticism, they are unbalanced, tense, anxious and different. Besides the above mentioned factors, other variables included in the etiology of EH are obesity, excessive alcohol intake, smoking, blood lipids, potassium deficiency, glucose, caffeine, genetic disposition racial differences, age and gender. All these are presented in Figure 2.2.

Further the knowledge of one's hypertensive state plays a significant role in self-reports of physical symptoms (Nyklicek et al., 1997).
The influence of any degree of hypertension varies considerably depending on coexistent risk factors. Hence, accurate estimates of the risk require consideration of the entire cardiovascular risk profile.

2.3 Assessment and Classification of Hypertension

EH tends to be asymptomatic and is not usually associated with specific complaints. The occasionally observed symptoms such as nausea, occipital headache, enhanced fatigue, nervousness and cardiac palpitations accompany chronic elevations in blood pressure (BP).

The dividing line between normotension and hypertension is arbitrary. The current definition is that this line is the blood pressure above which intervention has been shown to reduce the risk, (Evans & Rose, 1971). It is well established that lowering even mildly raised pressures reduces morbidity and mortality from cardiovascular disease (Collins, Peto, MacMohan, Hebert, Fiebach, Eberlein et al., 1990)

2.3.1. Measurement of Blood Pressure

Blood pressure generally is measured by the indirect method with a mercury sphygmomanometer. In assessing thresholds several recordings should be obtained - for example, two or more in the sitting position on each visit on up to four separate occasions. Before measurement of BP the patient should be seated for several minutes in a quiet room. A suitable sized cuff is applied to the upper arm kept at heart level. The cuff is then rapidly inflated until the manometer reading is about 30 mm Hg above the level at which the pulse disappears and then slowly deflated at about 2 mm Hg/s. During this time the Korotkoff sounds are auscultated through a stethoscope placed over the brachial artery. The pressure at which the sounds are
first heard is the systolic pressure; the diastolic pressure is the pressure at which the sounds disappear (WHO/ISH guidelines, 1993). The amount of arterial pressure when the ventricle contracts and the heart is pumping constitutes the systolic measure; the diastolic pressure, however, is the degree of arterial pressure when the ventricles relax and the heart is resting. A normal BP in a young adult would be 120 (systolic) over 80 (diastolic) i.e. 120/80 mm Hg. BP is measured in millimeters of mercury. Blood pressure is found to fluctuate both in hypertensives and normotensives (Harrison, 1987).

"White coat hypertension" or "effect" is a condition in which BP is raised only in the presence of a doctor. Whether the effect is an innocent phenomenon is unknown. Indeed, it has been suggested that subjects with a more distinct difference between measurement in the clinic and home may be at somewhat higher cardiovascular risk (Julius, Jamerson, Gudbrandsson, Schork, 1992).

2.3.2 Hypertension Defined

The randomised therapeutic trials on hypertension have defined and treated patients on the basis of diastolic blood pressure (DBP) values, and for this very reason DBP has generally been used to define hypertension specially mild hypertension. However, mounting evidence suggests that systolic values too should be taken into account in defining and managing hypertension (Zanchetti, 1990). Cardiovascular risk, indeed is as strongly associated with systolic as with diastolic values, with no evidence of a threshold below which a decrease in pressure does not reduce risk (Kannel, Dawber, McGee, 1986). Further, findings indicate that cardiovascular events more
Table 2.1

Classification of Hypertension by Blood Pressure (WHO/ISH, 1993)

<table>
<thead>
<tr>
<th>CLASSIFICATION</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(mm Hg)</td>
<td>(mm Hg)</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;140</td>
<td>&lt;90</td>
</tr>
<tr>
<td>Mild hypertension</td>
<td>140-180</td>
<td>90-105</td>
</tr>
<tr>
<td>Subgroup: borderline hypertension</td>
<td>140-160</td>
<td>90-95</td>
</tr>
<tr>
<td>Moderate and Severe hypertension*</td>
<td>≥180</td>
<td>≥105</td>
</tr>
<tr>
<td>Isolated systolic hypertension</td>
<td>≥140</td>
<td>&lt;90</td>
</tr>
<tr>
<td>Subgroup: borderline isolated systolic hypertension</td>
<td>140-160</td>
<td>&lt;90</td>
</tr>
</tbody>
</table>

* Risk to be indicated by reporting actual values of systolic and diastolic blood pressure.
closely correlate with achieved systolic than with diastolic values (MRC working party on Mild to Moderate Hypertension, 1987).

These guidelines thus, define mild hypertension on the basis of diastolic and systolic blood pressure values. In accordance with these guidelines, a diagnosis of Mild Hypertension can be made when DBP values of 90-105 mm Hg or SBP values between 140-180 mm Hg, or both, are repeatedly measured over at least four weeks, with no antihypertensive treatment, though non-pharmacological intervention is to be recommended during this period.

**Established Essential Hypertension**: An individual with chronic SBP greater than or equal to 140 mm Hg and DBP greater than or equal to 90 mm Hg i.e. BP ≥ 140/90 mm Hg can be branded as hypertensive (Harrison, 1987; WHO/ISH, 1993, 1996).

**Malignant Hypertension**: Patients with accelerated hypertension, DBP > 120 to 130 mm Hg and with extremely rapid vascular deterioration, acute renal failure or death if untreated are marked as malignant hypertensives (Ram, 1983). Malignant hypertension usually occurs in patients with previous essential or secondary hypertension but may rarely be the initial presentation in a previously normotensive patient (Houston, 1988).

### 2.3.3 Classification of Hypertension

Table 2.1 shows the operational classification of hypertension by blood pressure as a practical guide to management as proposed by the WHO/ISH (1993, 1996). Subjects with DBP between 85 and 89 mm Hg or SBP between 130 and 139 mm Hg are not classified as "high
normal” (JNCV, 1993), as it carries the risk of labelling a large number of subjects.

To summarize, the model of EH in the present study is based on the psychological factors with special reference to Life Stress, Anger, Anxiety and Personality factors with emphasis on Type A behaviour. It deals with their contribution to the etiology of this widespread cardiovascular disorder. Various socio-demographic factors to have been dealt with (see Fig. 2.1).

2.4 Concept and Assessment of Life Stress

2.4.1 Concept of Life Stress.

During the past two decades an abundance of research has dealt with the relationship of stress (life or occupational) and the indices of low physical and/or psychological well-being (see edited volumes by Pestonjee, Pareek & Aggarwal, 1999; Misra, 1999).

Stress has been defined as a stimulus (for e.g., being exposed to a stressful situation) and a response (for e.g., in the sense of ‘feeling under stress’). Stress also is viewed as a single event or response, when, in fact it may comprise a series of such events and responses. There is good evidence to support the notion that stress may best be seen in terms of an individual’s interaction with the event rather than as a univariate, unidirectional concept. Events perceived and experienced as stressful by the individual are stressors and the reactions elicited in response to these stressors are referred to as stress responses. The entire concept of ‘stress’ is seen as a dynamic interactional process rather than a single event or set of responses. Thus, stressors,
psychological, physical or both impinge on the individual to assess and understand the situation and then respond to it.

All individuals in their entire life period experience a variety of events or life changes, which may be potential stressors, such as death of spouse, marriage, divorce, separation, illness of family member, change in residence, loss of job, major personal illness or injury, new addition in family etc. In our country, the quality of life is changing. Standards of behaviour of individual freedom, of relationship with one another and of values are being altered so rapidly that their impact on society and on individuals is more profound than at anytime in history (Chopra, 1988). These events often require significant social readjustment and adaptation, nobody is immune from experiencing such changes. These changes represent an ongoing source of stress to which all individuals are subject to a greater or less degree.

Since stress is built into life itself, its not so much whether we will experience stress or not but ‘what kind’ and ‘how much’. Some authors suggest that we use two different stresses to distinguish the effects of stress on us. It has been argued that undesirable events (e.g. death of a close family member) may have a very different and possibly a more detrimental effect on individuals than positive events (e.g. outstanding personal achievements). It seems reasonable to conceptualize life stress primarily in terms of events that exert negative impacts. Further, Perkins (1982) views that potentially stressful major life events are seen as “discrete, time-limited events requiring change or adaptation.” Of the variety of ecological stressors, researchers have been more concerned with life stress or stressful life events (Dohrenwend & Dohrenwend, 1974).
Life event is defined as stressful if "it causes changes in and demands readjustment of an average person’s normal routine" (Dohrenwend & Dohrenwend, 1974). This definition highlights that stressful life events include those events that involve change in the usual activities of most individuals who experience them.

Negative events elicit more causal attributional activity than do the positive events (Peeters & Czapinski, 1990). A negative event is one which has the potential to create adverse outcomes for the individual. Therefore, the definition includes those events that have not occurred but are perceived as potentially threatening, as well as those that have occurred and are perceived as harmful (cf. Lazarus & Folkman, 1984). Much evidence suggestive of a greater role of negative events evoking physiological activity is also implied by research on stressful life events. 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 adjustments, research now indicates that negative events are substantially more potent in this regard than are positive one (e.g. Suls & Mullen, 1981). Earlier, Sarason, Johnson and Siegel (1978) viewed that undesirable or negative events are better predictors of stress. Further, studies have reported that higher levels of negative life events are associated with symptom maintenance and illness exacerbations (e.g. Chase & Jackson, 1981; Patterson and McCubbin, 1983; Perrin, McLean & Janco, 1988; Walker and Greene, 1991).

Studies on family systems perspective suggest that life events experienced by one family member may indirectly affect other members of the family unit. Walker, Garber and Greene (1994) report similar findings in pediatric patients belonging to families with higher
levels of negative life events. Focus on the linkage of psychological and behavioural factors to disease suggests that physiologic responsiveness to emotional stress might be a marker of pathogenic processes involved in the etiology of EH or CHD. For example, the changes in pain perception precede the onset of hypertension. These changes in pain threshold may be due the operation of some of the same mechanisms that might be responsible for the abnormal regulation of resting BP and reactivity to psychological stress (France & Ditto, 1996).

Stress can be beneficial ("the spice of life") or harmful ("the kiss of death"), depending on a person’s interpretation. Evidence states that an event’s meaning is not inherent in the event itself (Freire, 1985). The birth of a child for example, may be seen as either wonderful or tragic. The critical determinant of which meaning is constructed lies in the subject’s definition of the situation (Thomas as cited in Blumer, 1979). In accordance with this view Suedfeld and Bluck (1993) state that the basis of classifying events as positive or negative is of critical importance. Although reliance on a priori conceptualizations about what does or does not constitute a positive (or negative) life event offers methodological parsimony, such an approach ignores the tremendous variation that exists in how life events are experienced. The role that subjective meaning plays in shaping how events are experienced and recounted must be taken into account.

There has been much argument over the consideration of distal or recent events. Baum (1990) views that individuals may develop adverse physical or psychological reactions to stressful events that persist for prolonged periods. Also, there may be delay in months or years between the stressful event and the emergence of overt disturbances in functioning (Solomon, Weisenberg, Schwartzwald &
Mickulincer, 1987). Contrary to this, recent life events have been considered to be more important in research on health/illness (Suh et al., 1996; Fisher, 1996). Further, Selye (1976) and Baum et al., (1983) are of the view that though chronic conditions are more complex and variable than short-term responses, it is quite plausible that reactions to acute events might have consequences for understanding processes related to chronic cardiovascular disorders.

Holmes and Rahe and their associates (Holmes & Masuda, 1974; Holmes & Rahe, 1967; Rahe, 1972, 1974; Rahe, McKean & Arthur, 1967) investigated the relationship between stressful life events and onset of illnesses. They stated that life events cluster significantly in a two-year period preceding the onset of illness and that the onset of illness can be predicted from the total number of life events (Rahe & Others, 1964).

In the clinical arena, links between various illnesses and both major and minor life events have been established (Weinberger, Hiner & Tierney, 1987). Stress as defined in this context is essentially a psychological phenomenon, however, how this psychological activity translates into a physical reality is yet to be extensively researched upon.

2.4.2. Assessment of Life Stress

Holmes and Rahe (1967) prepared the ‘Schedule of Recent Experience’ (SRE-Scale) to measure the weight of several life stress events in the first phase. The second phase sought to perfect the scaling system of the measurement instrument so as to permit clearer vision of the relationship between events and onset of illness. The SRE represents a valuable initial attempt at the quantification of the impact of life change, yet its adequacy has been questioned on several counts.
(Rabkin & Struening, 1976). The SRE was based on the assumption that life change per se are stressful regardless of the desirability of the events experienced. Therefore, both desirable and undesirable events are combined in determining the life stress score. However, several investigators have questioned the logic of combining positive and negative events. It has been argued that negative or undesirable events may have a different and possibly a detrimental effect on individuals than positive events (see Aggarwal & Naidu, 1988).

Later, Sarason, Johnson and Siegal (1978) developed a new assessment measure, the ‘Life Experiences Survey’ (LES). LES is a 57-item self report measure which permits the respondents to indicate events they have experienced in the past one year. Subjects have to rate separately the desirability and the impact of the events they have experienced. They are asked to indicate those events experienced during the past year as well as whether they viewed the event as being positive or negative and the perceived impact of the particular event on their life. Ratings are on a 7-point scale ranging from extreme negative (-3) to extreme positive (+3). A negative change score is derived by summing the impact ratings of those events experienced as negative by the subject. Summing the impact ratings of events designated as positive by the subject provides a positive change score.

Presumptive Stressful Life Events Scale (PSLES), a suitable scale of stressful life events experienced by the Indian population, has been constructed and standardized by Singh, Kaur and Kaur (1984). This has two time space, that is, last one year and life time. PSLES consists of 51 items relating to stressful life events. The scale items have been further divided into desirable, undesirable and ambiguous and also into personal and interpersonal categories. Statistical difference has
also been observed between the desirable and undesirable items, the latter being perceived as more stressful.

Some methodological issues in assessment of life stress with particular reference to India have been highlighted by Sharma (1988). These include (i) formation of event list, (ii) severity rating of events, (iii) summation of event scores, (iv) reliability of event reporting, and (v) qualitative analysis of data that adds to the specificity of research beyond a simple counting of events. Aggarwal and Naidu (1988) administered their 'Scale of Stressful Experiences of Students' (SSES) and a strain measure to 100 students of Allahabad University. Correlation for undesirable events with measures of strain was higher than those for desirable and ambiguous events. They, thus, stated that the exclusion of desirable and ambiguous events will result in shorter stress scales with enhanced power of predicting strains.

2.5 Concept and Assessment of Type A Behaviour Pattern (TABP)

2.5.1 Concept of TABP

Type A Behaviour Pattern (TABP) is an "overt behavioural syndrome" or style of living. It is a particular complex of personality traits characterised by excessive competitive drive, impatience, aggressiveness, a harrying sense of time urgency, restlessness, hyperalertness, explosiveness of speech, tensing of facial musculature and being under the challenge of responsibility. Individuals displaying such a pattern seem to be engaged in a chronic, ceaseless and often fruitless struggle with themselves, with others, with circumstances, with time and sometimes with life itself. Free-floating but a rationalized form of hostility and almost always a deep seated sense of
insecurity are also evident in Type A individuals. Also, a time-harassed Type A often indulges in Polyphasic activities in his daily chores. For example, he may shave and scan a newspaper simultaneously or may have his breakfast while dressing up.

Type A behaviour, a coronary-prone behaviour pattern, has been a much acclaimed work of cardiologists Friedman and Rosenman (1974). They described this pattern as "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 required to do so, against the opposing efforts of other things or other persons" (p. 67).

Type B behaviour pattern, originally defined as the relative absence of aggressiveness, hostility, impatience, restlessness, hyper alertness and the various other components of Type A behaviour, is now increasingly being perceived as an alternative style of responding to or coping with environmental challenges. They elicit patterns of relaxation, serenity, less competitive and more easy going than Type As' (Ivancevich & Matteson, 1984).

Research indicates that in achievement situations' Type A individuals, as compared to Type B counterparts tend to be more competitive (e.g. Glass, 1977) and hard-driving in the sense that they respond with greater effort, vigour and persistence (e.g. Weidner & Matthews, 1978) and that they tend to set difficult performance goals (e.g. Grimm & Yarnold, 1984) and obtain higher levels of overall achievement on some measures (e.g. Matthews, Helmreich, Blane & Lucker, 1980).

It may however, be noted that no one Type A individual manifests all the characteristics that constitute the pattern, and also that
Figure 2.3 The Biopsychosocial Interactional Model (Adapted from Smith, T.W. and Anderson, N.B. (1986). Journal of Personality and Social Psychology, 1986, Vol. 50, 6, 1166-1173)
Type B individual will elicit some A-like features. In clinical practice, the designation of a person as Type A or Type B depends upon a summation of the number of Pattern A characteristics and their intensity.

Evidence has accumulated over the years that implicates Type A behaviour pattern (TABP) as a risk factor for CHD. Perspective studies have suggested that TABP is an independent risk factor for CHD in both men and women (Booth-Kewley & Friedman, 1987; Matthews, 1988) and it is independent of and equivalent to traditional risk factors (e.g. age, smoking elevated serum cholesterol). They also indicate that Type A individuals are more likely to (i) develop heart disease (ii) suffer another heart attack following a nonfatal attack, and (iii) show more extensive hardening of the arteries (atherosclerosis) of the heart than Type B individuals.

Type A individuals as compared to Type B's, respond to stimuli with enhanced, sympathetically mediated physiological activity which initiates and hastens the development of coronary artery atherosclerosis, the diseases underlying the clinical manifestation of CHD (i.e. myocardial infarction (MI) angina pectoris & sudden death). They also elicit greater cardiovascular reactivity i.e. systolic blood pressure (SBP) and heart rate (HR) and greater neuroendocrine (i.e. circulating epinephrine, norepinephrine, cortisol) responses to certain stimuli.

Smith and Anderson (1986) in their 'Biopsychosocial Interactional Model' have demonstrated how Type A behaviour and the environmental factors interact leading to coronary disease as can be seen in Figure 2.3.
Western industrialized societies have achieved revolutionary increases in economic standards of living along with marked reductions in mortality and morbidity from infectious diseases. A rise in the prevalence of CHD has been found to be concurrent with these accomplishments. This led the scientists to investigate the potential role of psychological factors associated with modern life styles in the etiology of CHD. Rosenman (1986) investigating cross-cultural and demographic variation in prevalence of Type A behaviour (TAB) and the correlation between TAB and CHD reports that culture does not appear to influence the magnitude of the correlation between the two. However, culture is related to the prevalence of TAB. He also found no relation between sex and the magnitude of the correlation between TAB and CAD.

Significant changes in BP levels especially SBP, have been observed after Type A counselling by Friedman et al., (1996) elucidating the role of this behaviour pattern in essential hypertension (EH).

2.5.2 Assessment of Type A Behaviour Pattern (TABP)

Three measures of Pattern A have been related prospectively to coronary disease. These measures are (i) the Structured Interview (Rosenman, et al., 1975; Chesney, Eagleston and Rosenman, 1980), (ii) by questionnaire scales such as Jenkins Activity Survey (JAS, Jenkins Rosenman & Zyzanski, 1979), and (iii) Framingham Type A Scale (Haynes, Feinleib & Kannel, 1980). The Jenkins Activity Survey (JAS) is a self-report measure. It has three forms - Form A, Form B, and Form C.
2.6 Concept and Assessment of Anger

2.6.1 Anger as a Negative Emotion

In contemporary societies negative emotional states of anger and aggression unquestionably characterize one of the dominant domains of everyday experience. Anger has been recognized as a significant constituent of human life since long. According to the Bhagavadgita, the experience of anger has tremendous consequences for life and well-being of a person. It argues as follows:-

"While contemplating the objects of the senses, a person develops attachment for them, and from such attachment lust develops, and lust anger arises. From anger, complete delusion arises, and from delusion bewilderment of memory. When memory is bewildered, intelligence is lost, and when intelligence is lost one falls down against into the material pool. But a person free from all attachment and aversion, and able to control his sense through regulative principles of freedom, can obtain the complete mercy of the Lord." (Bhagavadgita, Chapter 2, Verses 63-65).

Anger has been described as a primary emotion, arising when an organism is blocked in the attainment of a goal or in the fulfillment of a need. It is likely to depend on the organism's appraisal of events and assignment of meaning to them. Its an emotional state that consists of feelings of irritation, annoyance, fury, rage and heightened activation or arousal of the ANS. Hostility also involves angry feelings, but this concept is much broader, usually having the connotation of negative destructive attitudes such as hatred, animosity and resentment as well as chronic anger. Aggression generally refers to destructive punitive behaviours directed towards other persons or
objects in the environment. Substantial overlap in the prevailing conceptual definitions of anger, hostility and aggression led to refer them collectively as the “AHA ! Syndrome” by Spielberger, Jacobs, Russell and Crane (1983), who proposed the following working definition of the syndrome:

"Anger usually refers to an emotional state that consists of feelings that vary in intensity, from mild irritation or annoyance to intense fury and rage. Although hostility usually involves angry feelings, this concept has the connotation of a complex set of attitudes that motivate aggressive behaviours directed towards destroying objects or injuring other people..... while anger and hostility refer to feelings and attitudes, the concept of aggression generally implies destructive or punitive behaviour directed towards other persons or objects."

Saul (1976) stated that hostility is usually accompanied by the emotion of anger and is a motivating force which is a conscious or unconscious impulse, tendency, intent or reaction aimed at harming or destroying some object. He viewed it as an element of all destructiveness.

Aggression is regarded as a behavioural reaction to provocation. Further, the concept implies destructive or punitive behaviour directed towards other persons or objects (Spielberger, Johnson, Russell, Crane, Jacobs & Worden, 1985; Spielberger et al., 1988). This notion of aggression mirrors the origin of the term in Latin :'ad gredior', meaning literally "to rush toward something".

It is assumed that a certain type of cause-perception or blame worthiness is a necessary feature of anger. Johnson, Laird and Oatley (1989), however, argued that anger can occur for no known reason.
There is much disagreement on whether anger includes only negative (unpleasant) feelings (e.g. Biaggio, 1987), both negative and positive feelings (Schimmel, 1979) or neither (Alschuler & Alschuler, 1984). Further, Biaggio and Maiuro (1985) and Spielberger et al., (1983) stated that the existing measures of anger and hostility tend to confound the experience and expression of anger with situational determinants of angry reactions and that a coherent theoretical framework that explicitly recognised the state and trait distinction as well as distinguished between anger, hostility and aggression was required.

Following a review of the research literature Spielberger et al., (1983) defined anger as a personality trait (T-anger) in terms of individual differences in the frequency of experiencing state anger over time. Individuals high in trait anger were more likely to perceive a wider range of situations as anger provoking (i.e. annoying, frustrating, irritating), and also to respond to such situations with elevations in state anger than individuals low in T-anger. State anger (S-anger) has been defined as an emotional state or condition that consists of subjective feelings of tension, annoyance, irritation, fury and rage, with concomitant activation or arousal of the autonomic nervous system. It is further assumed that state anger varies in intensity and fluctuates over time as a function of frustration arises from the blocking of goal-directed behaviour.

2.6.2 Anger Expression/Anger Subcategories

Spielberger et al., (1985) stated that, "the expression of anger must be distinguished conceptually and empirically from the
experience of anger as an emotional state (S-anger) and individual
differences in anger as a personality trait (T-anger).

Within the archetypal complex of anger there are two
psychological experiences. One being rage, an instinctive reaction that
happens automatically and unconsciously, specially in response to
perceived threat. The other being anger, a conscious feeling that
includes a sense of judgment, choice and differentiation i.e., the inward
turning movement. Further it is also stated that anger is often directed
towards persons well known to the subject and is instigated mainly by
frustration or an affront to one's honour perceived as unfair.

Styles of anger-expression or anger management vary from one
individual to another. Researchers have stated that individuals manage
their anger in either of the three forms i.e., Anger-In (AX/In), Anger-Out
(AX/Out) and Anger-Control (AX/Con). 'Anger-In' means anger held
in, or suppression of angry feelings. 'Anger-Out' is defined in terms of
the frequency that angry feelings are expressed in verbally or
physically aggressive behaviour. High correlations of AX/Out with T-
Anger/T, and smaller correlations of both AX/Out and AX/In with T-
Anger/R (Spielberger, 1988), suggest that individuals having an angry
temperament are more likely to express their anger outwardly than
suppress it, whereas those who frequently experience anger when they
are frustrated or treated unfairly are equally likely to express or
suppress their anger. 'Anger-Control' refers to attempts to control and
suppress or mitigate the expression of anger. In Western non-clinical
groups, AX/In and AX/Con have been found to be independent
dimensions of anger control, with essentially zero correlations with
each other reflecting different types of mechanisms for controlling and
suppressing anger (Spielberger, 1988). Research states that individuals
relying an anger-control rather than on anger-in were stable personalities with better psychological resources and more capable of coping successfully with various life crises. Anger-control is a more healthier way of dealing with angry feelings, whereas anger-in poses a major health risk. An alternative hypothesis based on the general notion of the benefits of expression feelings, would lead one to expect that, either way, anger not expressed constitutes a health risk (see, e.g. Swan, Carmelli, Dame, Rosenman & Spielberger, 1992).

2.6.3 Assessment of Anger

Evidence for the need to distinguish between anger and hostility was provided in the early 1970s by the appearance of three anger scales. The ‘Reaction Inventory’ (RI) was developed by Evans and Stangeland (1971) to assess the degree to which anger was evoked in a number of specific situations. Subjects report the amount of anger they believe they would experience in each situation by rating themselves on a 5-point scale, from “Not at all” to “Very much.” The ‘Anger Inventory’ (Novaco, 1975) is similar to the RI in conception and format. It consists of 90 statements that describe anger provoking incidents. The subjects rate their degree of provocation on a 5-point scale. A high internal consistency (Chronbach alpha = .96) was reported. Further, Zelin, Adler and Meyerson (1972) designed the ‘Anger-Self-Report’ (ASR) to assess both the experience and expression of anger. The ASR is comprised of seven sub-scales: “Awareness of Anger”, three separate subscales for measuring different modes of “Anger Expression”, “Condemnation of Anger”, “Mistrust”, and “Guilt”. The ASR and RI have been used in only one or two studies over the past 15 years, as such, the construct validity of these scales has yet to be firmly
established. Also none of these three scales adequately distinguishes between anger as an emotional state (angry feelings) and individual differences in anger proneness as a personality trait.

To measure the fundamental properties of anger, it is essential to assess the intensity of angry feelings that are experienced at a particular time, the frequency that anger is experienced, and whether anger is held in (suppressed) or expressed in aggressive behavior directed toward other persons or objects in the environment. Recent research emphasizes the importance of evaluating the degree to which a person endeavours to control anger (Spielberger et al., 1985). The State-Trait Anger Scale (STAS) was designed to assess the intensity of anger as an emotional state and individual difference in anger proneness as a personality trait (Spielberger, 1980; Spielberger et al., 1983). Seigel (1986) standardized the "Multidimensional Anger Inventory", a scale that is claimed to be sensitive to the multidimensional nature of the anger construct. Its test-retest reliability is 0.75 and alpha reliability coefficients range from 0.84 to 0.89.

Spielberger et al., (1985) developed a highly competent tool, the "Anger Expression" (AX Scale), a dichotomous self-report rating scale assessing anger expression as a personality trait. AX Scale assesses how often subjects respond in a particular manner, rather than how they respond to a particular situation. Scores on this scale provide an index of the frequency that an individual expresses or suppresses anger across a variety of anger providing situations that are typically encountered in daily life. AX scale comprises of 24 items and yields four different scores. The three AX subscales assess individual differences in the tendency to express anger toward other people or objects in the environment (AX/Out), experience but hold in
(Suppress) angry feelings (AX/In), control/or resist the experience and expression of anger (AX/Con) and total score (AX/EX). The analysis of the AX-scores of students classified as "anger-in" and "anger-out" on the basis of modified Harburg procedure (Johnson, 1984) provides evidence of the concurrent and construct validity of the AX Scale and its sub-scales. Significant gender effects were found for the AX/EX and the AX/In scores; females had somewhat higher AX/EX (total anger expression) scores than males and somewhat lower scores on the AX/In subscale. Further, convergent and divergent validity of the AX and its subscales was found (Johnson, 1984), which reported its correlation with other anger and personality measures. The AX/In and AX/Out subscales correlated more highly with STPI anger measures than did the AX/EX total anger expression scores. A small but significantly high correlation of AX subscales with state and trait anxiety was also found.

The State-Trait Anger Expression Inventory (STAXI) (Spielberger, 1988; Spielberger et al., 1983, 1985) provides concise measures of the experience and expression of anger. The STAXI was developed primarily to (i) provide a method of assessing components of anger that could be used for detailed evaluations of normal and abnormal personality, and (ii) provide a means measuring the contribution of various components of anger to the development of medical conditions including hypertension, coronary heart disease and cancer.

The experience of anger, as measured by the STAXI, is conceptualized as having two major components - State and Trait anger. These components have been defined earlier. In addition to these two components, scores on AX/In, AX/Out and AX/Con are also provided
by the 44-items of STAXI. The names, the number of items and the components of anger assessed by each scale are as follows:

**State Anger (S-Anger)**

A 10-item scale which measures **the intensity** of angry feelings at a particular time.

**Trait Anger (T-Anger)**

A 10-item scale which measures individual differences in the disposition to experience anger. The T-Anger scale has two subscales i.e. Angry temperament and Angry Reaction.

**Angry Temperament (T-Anger/T)**

A 4-item test T-Anger subscale which measures a general propensity to experience and express anger without specific provocation.

**Angry Reaction (T-Anger/R)**

A 4-item T-Anger subscale which measures individual differences in the disposition to express anger when criticized or treated unfairly by other individuals.

**Anger Expression (AX/EX)**

A research scale based on the responses of the 24-items of the AX/In, AX/Out and AX/Con scales which provides a general index of the frequency that anger is expressed regardless of the direction of expression.
Anger-In (AX/In)

An 8-item anger expression scale which measures the frequency with which angry feelings are held in or suppressed.

Anger-Out (AX/Out)

An 8-item anger expression scale which measures how often an individual expresses anger toward other people or objects in the environment.

Anger-Control (AX/Con)

An 8-item scale which measures the frequency with which an individual attempts to control the expression of anger.

The individuals in responding to each of the 44-items of the STAXI, rate themselves on four-point scales that assess either the intensity of their angry feelings or the frequency that anger is experienced, expressed, suppressed or controlled.

Significant correlation of the T-Anger scale with three hostility measures e.g., Buss Durkee Hostility Inventory (BDHI; 1957), the Hostility (HO; Cook & Medley, 1954) and Overt Hostility (HO; Schultz, 1954) scales of Minnesota Multiphasic Personality Inventory (MMPI) was found across samples for both males and females. This provides a strong concurrent validity of the T-Anger scale as a measure of anger. The moderate correlations between the T-Anger Scale and the T-Anxiety and EPQ Neuroticism Scales are consistent with clinical observations and theory that individuals high in neuroticism and trait anxiety frequently experience angry feelings that they cannot readily express. Low to moderate correlations that were found between the S-Anger Scale and the T-Anxiety and EPQ Neuroticism and Psychoticism
Scales indicate that individuals with psychopathological personality traits experience more intense angry feelings than emotionally stable people.

The Hindi language version of Anger Expression AX Scale (AX/In, AX/Out and AX/Con) and of the total STAXI have been developed by Krishna (1988) and Rana (1990) respectively. They have provided ample evidence of the cross-language equivalence (English-Hindi) of these measures in terms of comparable means, standard deviation, inter-correlations, item-remainder correlations, alpha reliability, etc.

2.7 Concept and Assessment of Anxiety

2.7.1 Anxiety as a Negative Emotion

Anxiety, a very mobile feature which can be reversible when the individual’s somatic condition and social circumstances return to normal, is a stable state associated with a sense of vague danger whose nature and direction is unknown. It can last for a long time before undergoing a reverse development or progressing. Anxiety is perceived as a fairly stable trait in patients suffering from psychosomatic diseases (e.g. essential hypertension) with a protracted chronic course. It is characterized by tension, nervousness, restlessness, a sense of danger, some unknown threat, etc.

The term "anxiety" is most often used to describe an unpleasant emotional state or condition which is characterized by subjective feelings of tension, apprehension and worry by activation or arousal of autonomic nervous system. At various times, anxiety has been conceptualized as a response, a stimulus, a trait, a motive and a drive
(see Spielberger, 1976). An important source of confusion and ambiguity in the theory and research on anxiety is the interchangeable use of the terms 'stress' and 'anxiety'. Another problem faced is the inability to distinguish between anxiety as a state (transitory) and anxiety as a trait (individual differences in anxiety proneness). The trait-state distinction has been emphasized by Spielberger (1976). Spielberger proposed that the terms stress' and 'threat' be used to denote different aspects of a temporal sequence of events that result in the evocation of an anxiety reaction. While ‘stress’ denotes the objective stimulus properties of a situation, ‘threat’ refers to an individual’s perception of a situation as more or less dangerous or personally threatening to them. Thus, a person who perceives the situation as threatening will experience an increase in state anxiety (S-Anxiety). Thus,

\[ \text{Stress} \rightarrow \text{Perception of danger (threat)} \rightarrow \text{Increase in S-Anxiety} \]

Trait anxiety (T-Anxiety) refers to relatively stable individual differences in anxiety proneness that are manifested in behaviour in the frequency with which an individual experiences S-Anxiety elevations over time. An anxiety state (S-Anxiety) is defined in terms of the intensity of the subjective feelings of tension, apprehension, nervousness, worry and by heightened activity of the ANS that accompanies these feelings. Anxiety states vary in intensity and duration and fluctuate over time as a function of the amount of stress that impinges upon an individual’s interpretation of the stressful situation as personally dangerous or threatening. It is also referred to as a complex psychobiological process. It can also be regarded as a
psychological defense mechanism and as an interference in the organized purposive behaviour of the organism, or its block, resulting in vegetative activation.

The schematic diagram of Trait-State Anxiety Theory is presented in the Figure 2.4. The theory assumes that the arousal of anxiety states involves a process or sequence of temporally ordered events initiated by either external or internal stimuli that one perceives to be dangerous or threatening. Previously noted situations or circumstances in which personal adequacy is evaluated are likely to be perceived as more threatening by high T-Anxiety individuals, than by persons who are low on T-Anxiety. It should be noted, however, that the appraisal of a particular stimulus or situation as threatening is also influenced by a person's aptitude abilities and past experiences, as well as by his level of T-Anxiety and the objective danger that is inherent in the situation.

Anxiety as a process refers to a sequence of cognitive, affective, psychological and behavioural events. The concept of anxiety as a process is reflected in the traditional distinction between fear and anxiety. 'Fear' denotes an emotional reaction in anticipation of injury or harm from some real objective danger in the external environment. It is also stated that fear reaction is proportional to the magnitude of the danger that evokes it. In contrast, 'anxiety' is traditionally regarded as an 'objective' emotional reaction because either the stimulus conditions that evoke it are unknown or the intensity of the emotional reaction is disproportionately greater than the magnitude of the objective danger.

In the present study trait anxiety (T-Anxiety) is defined as the individual differences in anxiety proneness, i.e. differences between people in the predisposition to perceive external events or internal cues
(thoughts or memories as personally dangerous and threatening, and a corresponding tendency to respond to such threats with elevation in state anxiety (S-Anxiety). Individuals high in T-Anxiety experience elevations in S-Anxiety more frequently, and of greater intensity, than persons who are low in A-Trait as they tend to perceive a large number of situations as dangerous.

Thus, once a stimulus situation is appraised as threatening it is assumed: (i) a S-Anxiety reaction will be evoked, (ii) the intensity of this reaction will be proportional to the amount of threat the situation poses for the individual, and (iii) the duration of S-Anxiety reaction will depend upon the persistence of the evoking stimuli and the individual's previous experience in dealing with similar circumstances. The evocation of S-Anxiety, however, would be different in persons who differ in T-Anxiety: (i) individuals with high T-Anxiety appear to interpret circumstances in which their personal adequacy is evaluated as more threatening than do low T-Anxiety individuals, and (ii) situations that are characterized by physical danger are not interpreted as differentially threatening by high and low T-Anxiety subjects. As such, differential elevations in S-Anxiety would be expected for persons who differ in T-Anxiety under circumstances characterized by some threat to self-esteem, but not in situations that involve physical danger unless personal adequacy is also threatened.

2.7.2 Assessment of Anxiety

The State-Trait Anxiety Inventory (STAI) was developed by Spielberger, Gorsuch and Lushene (1970) to provide reliable relatively brief self-report measures of both S-Anxiety and T-Anxiety. The STAI, S-Anxiety scale consists of 20 items or statements which ask people to
describe how they feel at a particular moment in time. The STAI T-Anxiety scale also consists of 20 statements measuring the individual's anxiety proneness (i.e. how he generally feels). Each T-Anxiety item has been determined to be impervious to situational stress and is relatively stable over time.

General (Trait) anxiety scale developed in India include Sinha's Anxiety Scale (Sinha, D., 1962, 1965) and its shorter version (Khan & Hassan, 1981); Cattel and Scheier's IPAT Anxiety Scale (Hundal and Kaur, 1974); Comprehensive Test of Anxiety (Sinha & Sinha, 1969; Krishna, 1970); Hindi version of State-Trait Anxiety Inventory (Spielberger, Sharma & Singh, 1973; Spielberger & Sharma, 1976). Tripathi and Rastogi (1978) later developed an anxiety scale in which items for state-trait and free-floating anxiety have been included.

The STAI has been translated and adapted into more than 50 languages and dialects (Spielberger and Sydeman, 1994), making it an excellent tool for cross-cultural research.