

RECENT STRESSFUL LIFE EVENTS AND SEVERITY
OF SUBSEQUENT CORONARY ILLNESS

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TABLE OF CONTENTS

ACKNOWLEDGEMENTS	iii
TABLE OF CONTENTS	iv
LIST OF TABLES	vi
Chapter	
1. INTRODUCTION	1
Statement of Problem	3
Statement of Purposes	3
Background and Significance	3
Theoretical Framework	8
Assumptions	10
Hypothesis	10
Definition of Terms	10
Limitations	11
Delimitations	12
Summary	13
2. REVIEW OF LITERATURE	14
Models of Stress	14
General Adaptation	
Syndrome	14
Psychosomatic Models	15
Protective Reaction	
Pattern	16
Problem Solving	17
Summary	18
Stress Pathophysiology	18
Myocardial Hypoxia	20
Adrenergic Overactivity	24
Adrenocorticoid	
Overactivity	31
Life Change Events	33
3. PROCEDURE FOR COLLECTION OF DATA	50
Setting	50
Population and Sample	51

Protection of Human Subjects . . .	52
Instruments	52
Data Collection	56
Treatment of Data	58
4. ANALYSIS OF DATA	59
Description of Sample	59
Findings	60
Summary of Findings	66
5. SUMMARY OF THE STUDY	68
Summary	68
Discussion of Findings	69
Conclusions and Implications . . .	72
Recommendations for Further Study	74
APPENDIX A	75
APPENDIX B	76
APPENDIX C	80
APPENDIX D	82
APPENDIX E	84
APPENDIX F	86
APPENDIX G	88
APPENDIX H	90
APPENDIX I	93
APPENDIX J	95
APPENDIX K	99
REFERENCES	103

LIST OF TABLES

1.	Distribution of Subjects' Age	60
2.	Distribution of Subjects' Incomes	61
3.	Summary of Data for Category of Illness and Life Stress Score	62
4.	Occurrence of Subjects in Disease Categories	63
5.	Mean Value of the Variables	65
6.	Standard Deviation of Each Variable	66

CHAPTER 1

INTRODUCTION

Coronary artery disease is the number one cause of death in this country. After 50 years of research, medical science has yet to discover the cause. Since diseased vessels cannot be restored, much of the current focus is aimed at prevention. Circumstances such as obesity, family history of arteriosclerosis, hypertension, diabetes mellitus, cigarette smoking, elevated serum lipids, and high stress levels are thought to be contributing factors to heart disease. These are termed risk factors. Recently emphasis has been placed on reducing the number of risk factors and identifying populations at high risk.

A high risk person might be described as one who is admitted to the coronary care unit to rule out myocardial infarction. He has experienced a high degree of life stress prior to admission. The chest pain is relieved soon after admission and after a day or two in the coronary care unit the patient is moved to a regular room in the hospital. All the laboratory tests are normal. He is reassured that he has not had a heart attack and everyone is relieved that "it wasn't anything to worry about."

He continues to remain stable and is discharged. Following discharge, the chest pain returns. Having been assured that he is not really ill, he feels foolish to run back to the hospital or call the physician. But eventually he does return to the hospital--dead on arrival.

Could this type of unfortunate death be prevented? Studies have demonstrated that patients with suspected myocardial infarction who do not have an infarction in the coronary care unit are a high risk for cardiovascular death after hospital discharge regardless of risk factors (Schroeder, Lamb, & Harrison, 1977). Studies have also demonstrated that persons experiencing a high number of stressful life events have associated incidences of illness (Petricich & Holmes, 1977).

Stress has long been associated with heart disease. It would seem logical that the measurement of recent life stress might be a valid assessment tool if a positive correlation could be found between life stress and the severity of an acute episode of coronary artery disease. Acute coronary illness may or may not be preventable; but it seems that if a patient could be identified as a high risk for a severe coronary illness, anticipation and

therapeutic intervention by the health care team might make a difference in the outcome.

Statement of Problem

The problem identified to study was: Is there a relationship between high levels of life stress and the severity of subsequent episodes of acute coronary artery disease?

Statement of Purposes

The purposes of this study were:

1. To determine the degree of severity of the acute episode of coronary artery disease.
2. To ascertain the number of recent stressful life events the subjects had experienced during the year prior to illness.
3. To determine if a relationship exists between the severity of the acute coronary episode and the number of recent stressful life events the subjects experienced during the one year prior to illness.

Background and Significance

As far back as written records exist, people have been described as suddenly dying during an intense emotional experience. Hate, fear, joy, rage, and humiliation

have all been implicated (Engle, 1971). More recently, the term stress has been used to describe a gamut of stimuli including emotions that influence behavior. Stress is receiving more and more attention as a coronary risk factor. In 1947 the American Heart Association formed a special Committee on Stress, Strain, and Heart Disease. In 1976 at the National Conference on Emotional Stress, emotional stress was identified as a significant risk factor in the pathogenesis of sudden cardiac death and acute myocardial infarction (Eliot, Clayton, Pieper, & Todd, 1977).

While stress is a difficult area to study objectively, there have been attempts to do so. Research has demonstrated a relationship by looking at life events prior to an acute episode of coronary artery disease.

One of the most dramatic studies was the investigation of aerospace engineers in the mid-1960s at the Kennedy Space Center (Eliot et al., 1977). This population had an elevated cardiac death rate of 45% despite an average age of 31 years (Moss, Meltzer, Keily, & Rahe, 1977). Except for stress, normal parameters were in effect for coronary risk factor screening.

Within a highly competitive environment, the manned space effort phased out 88% of its employees from 1965 to

1976. Many specialized aerospace professionals were left with unmarketable skills. Studies showed universal feelings of hopelessness and helplessness and abnormally high anxiety and depression. Socially, these men experienced elevated divorce rates, alcoholism, psychological disorders, and family problems (Reynolds, 1974).

Abnormal resting electrocardiograms were significantly prevalent among these aerospace engineers; and, on autopsy, sudden death victims were found to have cardiac muscle fiber changes that were believed to be the result of catecholamine overdrive (Eliot et al., 1977). These factors may suggest a strong correlation between extraordinary life stress and an increase in occurrence and severity of acute coronary heart disease.

Rahe has studied life changes and subsequent illnesses extensively. Two separate studies involving more than 1000 subjects indicated increases in recent life changes prior to the onset of an acute episode of coronary heart disease (Rahe, Bennett, Romo, Siltanen, & Arthur, 1973; Rahe, Romo, Bennett, & Siltanen, 1974). In both studies, survivors of acute myocardial infarction had a 42% to 69% increase in the number of recent life changes in the 6 months prior to the episode of acute coronary heart disease. Delayed death victims (death within 28

days) had greater recent life changes than survivors of a myocardial infarction, and sudden death victims (death within 1 hour) had greater recent life changes than delayed death subjects. It is of particular interest, however, to note that in a separate study (Rahe & Theorell, 1971) of 54 subjects who survived myocardial infarctions, there was no correlation between life changes 1 year prior to the infarction and the severity of the myocardial infarction.

Medalier and Goldbourn (1976) found a major role played by anxiety and psychosocial factors in the occurrence of angina pectoris in a 5-year study. As the level of psychosocial problems and anxiety rose in those with normal resting electrocardiograms, so did the incidence rates of angina pectoris. When those subjects had electrocardiographic changes (ischemic changes or non-specific T waves) in addition to rising psychosocial problems and anxiety, the incidence rates of angina pectoris approximately doubled. The compounding effects of anxiety and psychosocial problems on the development of angina pectoris were demonstrated, especially in the presence of myocardial insufficiency.

Green, Goldstein, and Moss (1972) found that in at least 50% of the patients with sudden death,

psychological and social factors were associated with the time of sudden death. The authors surmised that perhaps a combination of depressive and arousal affects along with their physiologic concomitants produced disharmonious nervous and hormonal reactions.

In the 1960s at the University of Oklahoma, 65 patients who had undergone documented myocardial infarctions were studied for 7 years to ascertain attitudes and behavioral reactions to everyday challenges and problems. Ten individuals were predicted to succumb to recurrent myocardial infarctions on the basis of depression and social frustration. These 10 individuals were among the 23 deaths during the 4-year period after the predictions were made. Two of the 10 committed suicide, and the remaining 8 died suddenly of presumed or proven myocardial infarction (Wolf, 1971).

On the basis of these studies it is conceivable that stress may adversely affect coronary artery disease. Also, increased levels of life stress as exemplified by anxiety, depression, and life changes may have greater consequences as their severity intensifies. This phenomenon has germane implications for nursing as nurses become increasingly involved in the counseling of cardiac patients.

Theoretical Framework

The theoretical framework used to explain the relationship of life changes and acute episodes of coronary artery disease was Roy's (1976) adaptation model of nursing. According to Roy, man is a biopsychosocial being in constant interaction with a changing environment. Man has coping mechanisms which he utilizes within the changing environment. These mechanisms may be physiologic, such as regulation of body temperature; psychologic, such as the use of defense mechanisms; or social, such as role behavior. A positive response to a changing environment is commonly known as adaptation.

Roy (1976) drew from the work of Helson (1964) to describe the adaptation process. Man's ability to adapt depends upon the degree of change required and the state of the person coping with change. Three types of stimuli interplay to determine the success or failure of adaptation. First, a focal stimulus is the degree of change immediately confronting the person, such as temperature change, illness, loss, and pain. Second, contextual stimuli refer to all other stimuli present in the environment. Third, residual stimuli refer to beliefs, attitudes, and experiences which affect perception of the present situation including previous patterns of coping and

adaptation. An adaptive response is that behavior which is within the individual's ability to cope and maintains the integrity of the organism. A maladaptive response is one that does not maintain integrity and is disruptive to the organism. The adaptive behavior in the case of maladaptation is beyond the capability of the individual at that point in time.

Roy (1976) has identified two main adaptive mechanisms: the regulator and the cognator. The regulator mechanism works mainly through the autonomic nervous system and serves to maintain homeostasis by means of feedback systems. The cognator refers to mental processes. It acts consciously by means of thought and decision and unconsciously through defense mechanisms. Increased regulator activity, i.e., hormone secretion and catecholamine secretion, in the presence of decreased cognator effectiveness, i.e., decreased ability to identify and relate stimuli, is a sign of adaptation failure. The need to maintain physiologic, psychologic, and social integrity stimulates the human organism to utilize the regulator and cognator mechanisms to adapt.

In the present study, life changes represented focal, contextual, and residual stimuli that may have been beyond the individual's cognator adaptive mechanism. The acute

episode of coronary artery disease may have represented the failure of the regulator adaptive mechanism. Failure of both adaptive mechanisms may have resulted in the loss of physiologic integrity and possibly some degree of psychologic and social integrity.

Assumptions

The assumptions for this study were:

1. All living beings experience stress.
2. The human body is continuously engaged in the process of adaptation.
3. Body requirements for adaptation to stress can extend beyond the body's capacity to meet the demands.

Hypothesis

The hypothesis of the study was: There is no significant relationship between the scores on the Social Readjustment Rating Scale, which measures recent stressful life events, and the scores on the Severity Scale for Coronary Illness, which measures the severity of a subsequent episode of acute coronary artery disease.

Definition of Terms

The terms used in the study were defined as follows:

1. Stress--any stimulus, physical or emotional,

internal or environmental, pleasant or unpleasant, that evokes a bodily response.

2. Life stress--the stress that a subject has experienced during 1 year prior to an acute episode of coronary artery disease.

3. Coronary artery disease--irreversible narrowing of the inner lumen of the coronary arteries which restricts blood flow to the myocardium.

4. Subsequent acute coronary illness--the occurrence of the current acute episode of coronary artery disease requiring admission to a critical care area.

5. Life change unit (LCU)--a numerical value given to a potentially stressful life event which requires adaptation.

6. High risk--a high probability that illness will occur in the near future.

7. Critical care area--a coronary care unit and/or an intensive care unit.

Limitations

The limitations of this study were:

1. The level of stress that the subject was exposed to as a result of admission to a critical care area.

2. The ability of the subject to adapt to stress.

3. The effects of drugs the patient received before and after admission to the hospital.

4. The efforts of the health team members to decrease the subjects' exposure to stressful events after admission.

5. The presence or absence of pre-existing coronary artery disease.

6. The measures taken by the medical regimen to prevent an acute episode of coronary artery disease.

7. The educational level, religion, occupation, and personality structure of the subject.

Delimitations

The delimitations of this study were:

1. Subjects suffering from chronic lung disease, diabetes mellitus, and chronic renal disease were excluded from the study.

2. Subjects were able to read and understand English.

3. Subjects were alert, oriented, able, and willing to participate in the interview.

4. Subjects had a physician who utilized the critical care area frequently enough to have standing orders for cardiac admissions.

5. Subjects whose diagnosis, accounting for admission symptom(s), was other than coronary artery disease were excluded.

6. Subjects were male.

7. Subjects were 35 years of age or older.

Summary

A problem for study was defined. Is there a relationship between recent life stress and acute coronary artery disease? Roy's adaptation model of nursing was presented as a theoretical framework from which a null hypothesis was formulated. The hypothesis stated that there is no significant relationship between the scores on the Social Readjustment Rating Scale, which measures recent stressful life events, and the scores on the Severity Scale for Coronary Illness, which measures the severity of a subsequent episode of acute coronary artery disease. The assumptions, limitations, and delimitations of the study were listed. The terms used in the study were defined.

CHAPTER 2

REVIEW OF LITERATURE

In order to review the phenomenon of life change and subsequent illness, several germane aspects will be explored. Models of stress will be presented followed by examination of stress pathophysiology. Finally, a discussion of the pertinence of life change events will be addressed.

Models of Stress

Selected models chosen for discussion were the general adaptation syndrome, psychosomatic models, protective reaction pattern, and problem solving. A description of each follows.

General Adaptation Syndrome

Selye (1976) described a model of stress called the general adaptation syndrome. Biologic stress was defined as the nonspecific response of the body to any demand. The nonspecific response affected all or most parts of the body, hence the name general adaptation syndrome. The general adaptation syndrome was characterized by three stages. The first stage was termed the alarm

reaction and mobilized the defensive forces of the organism. This led to the stage of resistance in which adaptation occurred by means of a set of internal responses that stimulate tissue defense. After prolonged exposure, the acquired adaptation was lost and the organism entered the third stage, the stage of exhaustion. When this occurred, the organism was no longer able to maintain resistance. The causative agent stimulating the alarm reaction was termed the stressor; the resulting condition of organism response was termed stress.

Psychosomatic Models

Alexander (1948) and Grinker (1973) produced similar psychosomatic models of stress. Alexander (1948) described a variety of physical disorders that were the result of excessive autonomic nervous stimulation. This stimulation was the physiological accompaniment of constant or periodically reoccurring psychologic states. Repression of emotion could lead to chronic innervation causing chronic dysfunction of the internal organs.

Grinker (1973) described the human organism as a physiologic, psychologic, and sociocultural system, composed of many small systems that are integrated to maintain a steady state. Activity in any one system was

communicated to other systems. If a stimulus that impinges on one system was of quantity or duration to constitute stress, action was initiated by other systems to return that system to a steady state. When psychologic mechanisms and volitional actions could not allay anxiety, this stress was dissipated to the physiologic system. The biochemical changes constituted psychosomatic disturbances that persisted long after the anxiety itself disappeared.

Protective Reaction Pattern

Simmons and Wolff (1954) formulated a model of stress in which the principal concept was the protective reaction pattern. In this model physical, social, and cultural factors combined to make the whole individual. The capacity of a stressful event to evoke a protective reaction pattern depended upon the significance it held for the individual. The particular protective reaction pattern evoked depended upon the significance of the stressor, the physical capacities of the organism, and the previous experiences associated with the stressor. The stressor was present as a real or as a symbolic threat. The protective reaction patterns were either apt or inept. When aptly used, the protective reaction pattern met specific,

short-term adaptive needs. They were inept and inappropriate when habitually used as long-term patterns of response and damaged the structures they were designed to protect.

Problem Solving

Scott and Howard (1970) developed a stress model based on the problem-solving method. The most important assumption was that each human organism tended to develop a comfortable characteristic level of activity and stimulation determined by behavioral and genetic factors. A problem was defined as a stimulus if it required the organism to exceed its ordinary level of functioning or restricted the usual level of functioning. Problems arose from the internal and external physical environment, from the psychological environment, and from the socio-cultural environment.

Mastery of the problem depended upon the organism's adequate sources of energy and resources, the nature of the problem, the organism's perception of the problem, and the effectiveness of the organism's response to the problem. Successful problem-solving behavior tended to dissipate tension. The organism was then superior to its state prior to the time of the problem.

Failure to master problems required the organism to deal with unresolved tensions. This unresolved tension demanded the use of excess energy and resources to cope with the problem. Stress was experienced to the extent that the organism maintained excess tension. The organism lived with the tension until energy and resources were exhausted or the tension was dissipated through physical, psychological, and/or social mechanisms of tension relief.

Summary

The common theme to all these models of stress was that of a stimuli-response pattern that exceeded the organisms' ability to respond. Implicit to this theme is that illness may occur at some highly individual point when the response required to cope exceeds the ability to respond. Furthermore, the successful response to a stressful stimuli leads to the increased capacity of the organism to cope.

Stress Pathophysiology

Early in the 20th century W. B. Cannon (1929) introduced the fight versus flight concept as a response to stress. The stress "alarm reaction" as later proposed by Selye (1950) described such physical changes as increased

heart rate, increased blood pressure, and increased cardiac output that prepared the organism to meet increased metabolic demands. These changes were activated by the sympathetic nervous system. These demands were well suited to ancient man who was constantly threatened by physical danger. The energy created was utilized to flee or to do battle. Modern man in the American culture, however, encounters more abstract threats such as loss of identity, status, income, security, satisfaction, and loss of environmental control. The energy created in ancient man dissipated almost immediately because of physical exertion. Modern man, on the contrary, is prevented from physical release of this energy due to social and behavioral constraints. This disengagement of the musculoskeletal system from the cardiovascular system results in a prolonged state of sustained sympathetic viscerovascular readiness that subjects the cardiovascular system to excessive work loads (Bove, 1977; Eliot et al., 1977; Lown, Verrier, & Rabinowitz, 1977; Raab, 1966; Selye, 1976; Slay, 1976; Solack, 1979; and Williams, 1979). The cardiovascular response to mental stress is integrated and initiated in the hypothalamus (Gilmore, 1974, 1976).

The mechanisms behind stress-induced acute coronary disease are multifold but owe their ill effects to a single critical phenomenon, namely the delicate electrolyte balance of sodium and potassium in the cardiac cell. The three primary mechanisms that contribute to the electrolyte disturbance were myocardial hypoxia, adrenergic overactivity, and adrenocorticoid overactivity (Raab, 1971). The roles of each of these factors in precipitating acute coronary artery disease are discussed separately in the following sections.

Myocardial Hypoxia

A brief review of myocardial cell physiology helped to clarify the mechanism by which hypoxia upset the electrolyte balance of a myocardial cell. Myocardial cells, having properties of both nervous and skeletal muscle tissue, propagate an action potential as well as contract. Blood-carrying oxygen and nutrients provide the energy required to operate the ionic pump and to contract the myofibrils (Guyton, 1971). It is crucial that enough oxygen is available at a given point in time to meet the oxygen requirements of myocardial tissue at the same given point in time. If a discrepancy exists

between oxygen availability and demand, then hypoxia occurs (Raab, 1966).

The mechanisms behind hypoxia and subsequent myocardial infarction are more complex than simply an occlusion of a coronary vessel. Evidence suggests that nearly two-fifths of the "healthy" population have a silent obstruction. In fact, nearly two-thirds of the individuals studied who died from sudden coronary death had no prior history of heart disease (Eliot, 1979).

There are two safety mechanisms that compensate for sclerotic blocking in coronary vessels. First, the greater the stenosis of a vessel, the greater will be the amount of collateral circulation as this phenomenon is controlled simply by a pressure gradient. Second, hypoxia itself is a powerful vasodilating agent within coronary vessels (Eliot, 1979; Raab, 1966). Again, it is not the vascular supply per se that is predisposed to hypoxia but a balance of oxygen supply and demand at a given point in time.

In myocardial necrosis whether the area of infarction is transmural, extending the entire thickness of the ventricular wall, or nontransmural, extending only through a partial thickness of the ventricular wall, the subendocardium is almost always involved. Eliot (1979)

described the susceptibility of this area and how its consistent involvement may have accounted for a proposed mechanism of hypoxia-induced myocardial infarction.

According to Eliot (1979) several features predisposed the subendocardium to ischemia. These features were:

1. The coronary circulation penetrated from the outer heart wall inwards so that the subendocardium was at the end of the blood supply plumbing. It has the lowest tissue partial pressure of oxygen of any organ or any layer of the heart. Any discrepancy in the oxygen supply and demand ratio was apparent first in the subendocardium.

2. Wall tension was greatest in this inner layer next to the heart chambers creating mechanical stress.

3. Myofibril lengths were longest in these cells so that oxygen demands were greater in the subendocardium than in other areas of the heart.

4. The subendocardial capillary bed performed at near capacity at rest.

5. Myocardial arteriole blood flow was dependent upon diastole.

Suppose, that at a given point in time, the alarm reaction to stress was initiated with subsequent acute tachycardia, elevated peripheral resistance, and

catecholamine release. Myocardial metabolic demands would likewise have increased with increased contractility, increased oxygen requirements, increased filling pressures, and decreased diastolic blood-flow time within myocardial capillaries. Under such circumstances the subendocardium was particularly vulnerable to ischemia. Eliot (1979) went on to suggest that the subendocardium was indeed the initial point of hypoxia in acute myocardial infarction.

Once an area of myocardial tissue is hypoxic, the ensuing energy loss causes changes in the myofibrils. The cells begin to lose contractility with a subsequent loss of tone. The cells become bulging, increasingly occupying space, and compressing against the microcirculation. This compression further inhibits the blood flow through the arterioles. A chain reaction ensues as more cells become involved, creating a flaccid area of pressure against surrounding vessels, decreasing cardiac performance, and further contributing to the imbalance of oxygen supply and demand. Without oxygen and energy to activate the ionic pump, propagation of action potential is interrupted and, if prolonged, cellular disintegration begins. Eliot stated that the loss of contractility from hypoxia was the primary event in the sequences

leading to myocardial infarction. Furthermore, after an area of necrosis reached a critical mass, the necrotic process continued cellular destruction in the presence of lowered pH without external forces.

Adrenergic Overactivity

In response to stress the sympathetic nervous system stimulates the adrenal medullae to release epinephrine and norepinephrine into the bloodstream. These two hormones have similar effects as direct sympathetic stimulation of other body organs, except that the effects are greatly enhanced and prolonged. Epinephrine increases the rate of metabolism and especially stimulates the heart. Norepinephrine has little effect on the heart but can greatly increase peripheral resistance, thereby increasing blood pressure. The ratio of secretion of epinephrine to norepinephrine is approximately 3:1, but the effects of norepinephrine last about 10 times as long as those of epinephrine (Guyton, 1971).

The body's response to emotional stress is the same as the response to a physical threat. Specifically, anxiety provoking situations are characterized by high excretion of epinephrine while aggression and depression

trigger the release of norepinephrine (Raab, 1966). Glasser, Clark, and Spoto (1978) demonstrated the deleterious effects of these hormones when the response to stimulation was inappropriate or exaggerated. It was well documented that if these emotionally induced changes were sustained over a period of time without release, cardiovascular disease ensued (Bove, 1977; Eliot et al., 1977; Gilmore, 1974, 1976; Glass, 1977b; Raab, 1966; Rosenman, & Friedman; 1974; Selye, 1950; Slay, 1976; Solack, 1979; Williams, 1979).

In response to physical or emotional stress, epinephrine is capable of raising the metabolic rate as much as 100% above normal. Its affinity for cardiac stimulation can greatly increase cardiac output by increasing the heart rate and the contractility of cardiac muscle. This increase in metabolism greatly increases the oxygen demand (Guyton, 1971).

At rest, the ventricular myocardium utilizes 75% of the arterial oxygen content. Myocardial oxygen consumption is not related to work load per se, but rather to pressure changes in the ventricle. The subendocardium has the lowest partial pressure of oxygen found in the body. As oxygen is utilized in response to increased metabolic demands, its partial pressure in the blood

decreases and facilitates increased arterial blood flow as the principal means of increasing oxygen supply (Rushmer, 1976). It readily becomes apparent how stenosis of coronary vessels, decreased contractility of the myocardium, and/or hypertrophy of the left ventricle interfere with coronary blood flow and alter the delicate balance of oxygen supply and demand in favor of ischemia, especially under the influence of stress-induced epinephrine stimulation.

Necrosis is not the only consequence of hypoxia. The critical factors are the extent of the ischemia and the duration of exposure of the myocardium to ischemia. Ischemia not severe enough nor prolonged enough to cause necrosis can cause serious rhythm disturbances. Bove (1977) described the hyperexcitability resulting from electrolyte derangement that predisposed to arrhythmias. During times of emotional stress, electrocardiographic tracings exhibit ischemic changes (Medalie & Goldbourt, 1976; Raab, 1966) as well as ventricular fibrillation in animals and humans (Lown et al., 1977).

Sudden unexpected coronary death consumes approximately 1200 victims per day. Of these an estimated 25% have no prior recognized heart disease. This implies

that the mechanism for sudden coronary death is frequently unrelated to ischemic processes. Other factors such as neurogenic hormones and catecholamines offer better explanations than ischemia (Eliot, 1979; Reichenbach, Moss, & Meyer, 1977).

In a study of 200 consecutive cases of sudden unexpected coronary death, Baroldi (1975) found that 76% of the victims demonstrated a unique form of myocardial necrosis referred to as coagulative myocytolysis. Coagulative myocytolysis is a category of cell death that results from hyperfunctional overdrive. This hypercontracted form of myocardial necrosis appears to result from catecholamine overdrive. Human and animal studies demonstrated this variety of hyperfunctional necrosis subsequent to the infusion of catecholamines such as epinephrine, norepinephrine, and isoproterenol.

It was hypothesized that cell death occurred secondary to ventricular fibrillation resulting from a barrage of sympathetic stimulation. Since sympathetic outflow differed from one part of the heart to another, simultaneous stimulation found myocardial cells in different refractory periods permitting re-entry and leading to ventricular fibrillation (Eliot, 1979).

As previously mentioned, norepinephrine is released in response to stress elevated blood pressure. Although hypertension has not demonstrated directly to cause coronary artery disease, it is well recognized as a coronary risk factor. Elevations of the diastolic pressure impose an increased mechanical load on cardiac muscle and contribute to hypoxia, particularly of the left ventricular subendocardium. This is even more accentuated in the presence of increased oxygen demands precipitated by epinephrine.

Although the exact mechanism was unknown, Kaplan (1978) succinctly outlined a leading theory. Environmental and genetic factors such as high salt intake, behavioral stress, and inherited disorder acutely and inappropriately raised peripheral vascular resistance causing acute hypertension. Acute hypertension caused increased arterial wall tension that advanced to arterial smooth muscle hyperplasia and hypertrophy. This led to cellular changes that thickened the interior lining of arterial walls. Thickened inner arterial walls decreased the inner lumen of the vessels and thereby increased peripheral resistance, recycling the vicious circle precipitating hypertension. This cycle must have been maintained over a period of time to alter the

arterial walls. In the early stages, behavior modification or reconditioning could interrupt the cycle. If it continued uninterrupted, however, the vascular changes coupled with renal mechanisms became fixed requiring pharmacologic intervention. In sustained essential hypertension the arterial baroreceptors became readjusted upwards reflecting maladaptation. In addition, the arterial baroreceptors became less sensitive in the presence of thickened arterial walls (Eliot, 1979).

In addition to the mechanisms mentioned, adrenergic overactivity seems to contribute to the process of atherosclerosis. Little is known about the actual cause of atherosclerosis, but it is believed to be associated with older age, diabetes, and elevated blood lipids. It is more prevalent in males and is believed to be related to heredity (Guyton, 1971). Hypertension is also believed to contribute to atherosclerosis by causing mechanical damage to the intimal linings of the blood vessels (Eliot, 1979; Raab, 1966). Catecholamines have also shown to produce thickening and inflammatory changes of the intima (Raab, 1966). In fact, in a recent study neural factors alone were found to produce atherosclerosis in rats with hypertension and blood

cholesterol risk factors controlled (Gutstein, Harrison, Parl, Kiu, & Avitable, 1978).

As an energy source to meet increased metabolic needs, catecholamines mobilize free fatty acids from adipose tissue. These free fatty acids are transformed in the liver into triglycerides and phospholipids, which can be utilized for energy. In episodes of emotional stress without physical exertion, these nutrients remain in the circulation possibly contributing to the atherosclerotic process (Raab, 1966). Chronic stimulation of the stress response aggravates atherogenic hypercholesterolemia despite a limited dietary intake of saturated fat.

In summary, epinephrine and norepinephrine induce acute hemodynamic effects that, if sustained, may lead to coronary artery disease. These include elevations of cardiac rate, contractility, and blood pressure. These changes tax the coronary reserve, especially in the presence of coronary atherosclerosis. They may further lead to acute ischemia and loss of effective pump function. Moreover, chronically elevated catecholamine levels directly injure the intima of blood vessels and cause the release of possible atherogenic lipids into the blood.

Adrenocorticoid Overactivity

Stimulation of the adrenal cortex causes secretion of aldosterone and cortisol. These corticosteroids exert profound effects on the cardiovascular system. Each is discussed separately.

Two major outcomes result from the secretion of aldosterone. Aldosterone causes the reabsorption of sodium. As sodium increases blood volume, cardiac output increases. Tissues supplied with too much blood cause the local vessels to constrict in an attempt to return blood flow to normal levels. Consequently, hypertension results with the detrimental effects on the cardiovascular system as previously described (Gilmore, 1976; Guyton, 1971).

The other primary outcome subsequent to aldosterone secretion is the increased excretion of potassium. This net loss of potassium occurs in exchange for the reabsorption of sodium. The loss of potassium may proceed to a point at which the delicate electrolyte balance of myocardial cells is affected, predisposing to arrhythmias and cellular disintegration (Raab, 1966).

Secretion of cortisol by the adrenal cortex results in conversion of amino acids into glucose by the liver and mobilization of fatty acids from adipose tissue.

The latter is converted to lipids that are utilized for energy. It becomes readily apparent that chronic secretion of cortisol might lead to chronically elevated blood glucose and lipids that are risk factors for coronary artery disease (Gilmore, 1976; Glass, 1977a; Guyton, 1977).

Another interesting phenomenon of cortisol is its ability to sensitize heart muscle to the effects of catecholamines. Cortisol seems to accentuate a loss of potassium aggravating a catecholamine cardiotoxicity (Raab, 1966).

In summary, the cardiovascular pathophysiology of stress is a factor of elevated blood pressure, rapid heart rate, and increased oxygen utilization. Any state that initiates and sustains these factors, especially in the presence of a vulnerable coronary vascular system, can cause coronary insufficiency and subsequent electrolyte disturbance. These mechanisms have been described. Emotional stress has been demonstrated to induce these conditions and is a well recognized precipitating factor in acute coronary insufficiency. Whether these factors will induce disease processes depends upon: (a) the state of the coronary vessels; (b) the degree of available coronary reserve; (c) the duration of physical or

psychological stress; (d) the severity of stress; and (e) the degree of individual physical and psychological resistance and resilience (Eliot, 1979).

Life Change Events

In 1628 Harvey wrote in his treatise De Motu Cordis: "Every affection of the mind that is attended with either pain or pleasure, hope or fear is the cause of an agitation whose influence extends to the heart" (cited in Jenkins, 1976). Such long standing acceptance of emotions affecting physiologic functioning can be confirmed by such commonplace sayings as "scared to death." Accounts of illness and death related to emotional disturbances have been well documented by Engel (1968, 1971). A major medical text lists psychosocial tensions among the minor risk factors in the etiology of coronary artery disease (DiGirolamo & Schlant, 1974). To further explore the paradigm of life change and illness, the components of research on this subject include: evolution of life change research, replicated studies, confounding variables, methodological issues, and perception as the omnipotent variable.

The study of life change events and their effects on illness occurrence began in the 1950s. By the 1960s

researchers had constructed the Social Readjustment Rating Scale, a life change events list covering a broad spectrum of events from five major categories: health, home and family, work, personal and social, and financial (Holmes & Rahe, 1967). Each event had a numerical stress score. One common theme applied to all the life events--the occurrence of each usually was associated with some adaptive or coping behavior requiring change on the part of the individual. The underlying assumption was that such events serve as precipitating factors, influencing the timing but not the type of illness episodes (Holmes & Rahe, 1967). Within the last 3 decades nursing, medical, psychological, and sociological researchers have utilized the scale in an attempt to determine if an empirical relationship might exist between life change events and the occurrence of illness.

In the 1960s Rahe, a primary researcher of life change events, initially utilized enlisted Navy men to study life change events and subsequent illness. He reported a significant but low order positive relationship between a subject's recent life change magnitude and his near future illness report (Rahe, 1972). These studies were criticized by Goldberg and Comstock (1976)

on the basis of three shortcomings: (a) only a small percentage of the people accounted for a large percentage of the illnesses, (b) most of the sickness reported was minor, and (c) the few life events reported were also minor.

Rahe continued life change research in the early 1970s in Helsinki, Finland, where the Finnish Heart Association maintained a register of all cases of acute coronary heart disease. Utilizing this register, pilot studies indicated that the victims of sudden coronary death and the survivors of myocardial infarction showed increased life change scores prior to death or illness (Rahe & Lind, 1971; Rahe & Paasikivi, 1971; Theorell & Rahe, 1971). As Scandinavian studies proceeded, the most notable one took place in Finland in the early 1970s (Rahe, Romo, Bennett, & Siltanen, 1974). The previous findings were replicated with 279 survivors of myocardial infarction and 226 victims of sudden coronary death. Increased life change scores, 20% to 143%, accompanied death or infarction with death subjects experiencing the sharpest increase.

In the United States information retrieved from a 1962 study concerned with psychosocial forces and sudden death (cited in Wolf, 1971) provided ballistocardiographic

as well as life change data. In this study of 36 survivors of myocardial infarction, there was a significant buildup in recent life changes and abnormal ballistocardiograms for the 18 patients who died over the 6-year follow-up period (Theorell & Rahe, 1975). The 18 patients who survived the 6-year follow-up period showed neither a buildup in life changes nor abnormal ballistocardiograms.

Other studies replicated Rahe's findings. Victims of myocardial infarction showed increased life changes and a greater proportion of severe life change events (Lundberg & Theorell, 1976). Life changes coupled with anxiety and depression were strongly correlated with occurrence of myocardial infarction (Bianchi, Fergusson, & Walshe, 1978). Life change events also predicted seriousness of illness among college students (Garrity, Marx, & Somes, 1978). A similar study of college students demonstrated a latency period between life change and severe illness (Garrity, Marx, & Somes, 1977). Symptoms of minor illnesses tended to emerge soon after life changes; however, life change scores and severe illness correlated in this study most strongly 9 months after the life change assessment.

While studies supported the life change/illness phenomenon, similar research failed to establish a relationship. In a prospective study with random samples, Goldberg and Comstock (1976) found no significant differences between cases and controls. A prospective study of men in the armed forces failed to show any significant relationship between life change scores and illness occurrence (Casey, Thoresen, & Smith, 1970). A prospective study of 6,597 Swedes did not predict near-future myocardial infarction from life-change measurement (Theorell, Lind, & Floderus, 1975). A retrospective study of coronary patients, hospitalized patients, and healthy controls found no differences in life change scores (Glass, 1977b).

In view of the controversial findings in life change/illness research, literature has dealt with confounding variables. The problem seems to lie in the difficulties surrounding the study of human subjects. Humans are continuously interacting with the environment and are susceptible to many factors influencing behavior at a given point in time.

A prominent criticism surrounded the validity of retrospective research. Critics claimed that the subjects tended to explain their illnesses by reporting an

excess of life change events (Bianchi et al., 1978; Goldberg & Comstock, 1976). On the other hand, when survivors of illness under-reported life change events as compared to their spouses' reports, Rahe et al. (1974) hypothesized that these subjects minimized, through denial, their recent life changes and illness. Brown (1974) referred to this as contamination. Jenkins (1976) stated that the use of prospective studies is the best way to eliminate some sources of biased recall. Brown (1974) contended, however, that prospective studies still can be contaminated by virtue of subjective states, such as anxiety and depression, causing the occurrence of both life events and illness.

Another criticism of retrospective studies concerned the subject's ability to recall. Jenkins (1976) questioned the validity of increased recent life change scores simply because recent memory was more complete than distant memory. It was found that for a weight assignment that led to a single presumptive score, events remote in time had less influence than recent events (Horowitz, Schaefer, Hiroto, Wilner, & Levin, 1977). When subjects were tested at 9-month intervals, life change scores were 34% to 46% less than initially reported for the same previous time periods (Jenkins, Hurst, & Rose, 1979). Upon

retest, this sort of unreliability was accounted for by such factors as discretion, denial, forgetting, and ambiguity of items at a given time (Horowitz et al., 1977). It is of interest to note that when subjects ranked the amount of stressfulness associated with a particular life event, the magnitude estimates remained very stable for three sampling periods over 2 years' time (Gerst, Grant, Yager, & Sweetwood, 1978). It was also determined that mood states do not alter recall of life events (Siegel, Johnson, & Sarason, 1979).

The lack of adequate control groups in life change/illness studies came under scrutiny (Goldberg & Comstock, 1976; Jenkins, 1976; and Steele, 1978). A study that employed both a prospective nature and a control group showed a lack of correlation with stress scores and illness (Goldberg & Comstock, 1976). Jenkins (1976) stated that the studies that are methodologically stronger are the ones with negative results.

The concept of illness proved methodologically difficult to study. Illness itself tended to lead to illness, and a major obstacle lay in determining if an illness reported as a life event was truly independent of a subsequent illness counted as an outcome (Goldberg & Comstock, 1976; Hudgens, 1974). Hinkle

(1974) asserted that people with a history of frequent illness were likely to become sick in the future.

Cassem and Hackett (1973) found that after 6 months at home, post-myocardial infarction patients reported depression, sleep disturbances, failure to return to work for psychologic reasons, and weakness. Mechanic (1976) also argued that a central issue to the life event/subsequent illness paradigm was the failure of researchers to differentiate between physiologic illness and illness behavior with all the sociocultural trappings of the sick role.

Another important factor that needed further study was the apparent protection provided by an individual's social support system. The social environment could influence the experience of stress and presumably the vulnerability to certain diseases (Rabkin & Struening, 1976; Solack, 1979; Steele, 1978). Totman (1979) observed that throughout the pre-morbid year, post-infarction patients reported a significantly greater reduction in socializing during this period. These findings certainly stimulated consideration about the nature of the psychosomatic component of illness.

Several methodological issues dominated the literature concerning life change events and illness. These

were the population of events considered stressful, the assessment of stressfulness for each event, and the impact of the stressfulness of pleasant versus unpleasant events. Each of these issues is discussed separately.

How were events to be sampled as stressful defined? From what population of events was a sample of stressful life events to be drawn? Masuda and Holmes (1978) studied 12 disparate groups and found great variation in the occurrence of life event items. Heroin addicts experienced 5 times the number of different events as did medical students using the Holmes and Rahe Social Readjustment Rating Scale. Two sets of events were offered as reasonably stressful or of sufficient magnitude to bring about change in the usual activities of most individuals who experienced them (Dohrenwend, Krasnoff, Askenasy, & Dohrenwend, 1978). The first subpopulation consisted of a set of universal experiences such as marriage, birth, illness, injury, and death, which are common to all settings. The second subpopulation varied with social and cultural settings. This sample of life events were drawn from the experience of the general population living within the appropriate community of values. Dohrenwend et al. (1978) constructed a second subpopulation life events list by asking sample subjects:

"What was the last major event in your life that, for better or for worse, interrupted or changed your usual activities?" In this manner construction of a life events list was attempted that was relevant to the population to be studied.

Originally, Rahe (1972) attached scores to life change events as a result of values given by 400 subjects of differing demographic status. The subjects were instructed that "marriage" had been assigned an arbitrary value of 500; the amount of change associated with 42 other life events was to be compared to that of marriage and scored accordingly. Dohrenwend et al. (1978) asserted that such a sample of convenient judges did not allow for generalization to other sociocultural settings. They proposed that the stress values given to events should be determined by judges selected from the appropriate population. After implementing an elaborate scheme of such a selection of judges and completing the weight assignment task, they conceded that such a group may not be able to complete the task, especially with judges from a relatively uneducated and/or lower class sample.

Once weights of stressfulness were assigned to events, researchers varied on the reliability of such measures. On one hand, a high degree of concordance in

the rank ordering life events was demonstrated by individuals and groups (Horowitz et al., 1977; Masuda & Holmes, 1978; Rahe, 1972; Sands & Parker, 1979-80). On the other hand, different researchers reported cultural contrasts (Brown, 1974; Hough, Fairbank, & Garcia, 1976; Miller, Bentz, Aponte, & Brogan, 1974). For instance, Miller et al. (1974) found that in an urban sample "Marriage" ranked 4th in contrast to 21st in a rural sample in terms of the amount of change and adjustment involved. It appeared that ethnic background was a strong variable in determining the stress values of life events (Dohrenwend & Dohrenwend, 1978; Masuda & Holmes, 1978; Rosenberg & Dohrenwend, 1975). Still other researchers compared various scaling procedures and found no better an index than the simple count of life events (Grant, Sweetwood, Gerst, & Yager, 1978; Ross & Mirowsky, 1979).

Some researchers took issue with the variability of scores that occurred within groups. Rabkin and Struening (1976) stated that life event scores did not predict illness when statistics focused on group means, overlooking extreme variability of individual scores. Horowitz et al. (1977) supported life change scores as an index for obtaining gross, large-group measures of stress, but not

as a reliable solo index since there was no measure of individual adaptive or maladaptive response. Garbin (1979) stated that the current limitations of measurement reflect the difficulties of measuring complex, whole persons by using simple tools that focus on a particular aspect or part of the whole and the results of which may vary from one day to the next.

Original life change studies emphasized change from the existing steady state as important and not the psychological meaning, emotion, or social desirability (Holmes & Rahe, 1967). This position came under close examination by subsequent researchers (Brown, 1974; Chiriboga, 1977; Chiriboga & Dean, 1978; Myers, Lindenthal & Pepper, 1974; Ross & Mirowsky, 1979). Mechanic (1975) stated:

In the case of a life event such as "son or daughter leaving home" it makes a great deal of difference if they leave home to marry or attend college as compared with leaving home as a rebellious act or as a result of family disputes. (p. 46)

Mechanic (1975) went on to state that the data based on the Holmes and Rahe Social Readjustment Rating Scale could not resolve the theoretical issue of whether life changes in general or primarily adverse life changes effected the occurrence of illness. Sarason, Johnson,

and Siegel (1978) suggested that the separate assessment of positive and negative change represented a step forward in assessing relationships between life changes and diverse dependent measures, and that it was the negative change measure that should be used if the purpose was to determine the degree of life stress.

In nearly every critique of life change research, mention was made of individual perception of life change events. There were always some individuals who experienced a high degree of life change events and who did not succumb to illness and vice versa (Hinkle, 1974). Steele (1978) described this phenomenon in terms of an inter-relationship of host, agent, and environment as the etiological nature of illness.

Variability of individual perception was alluded to in the discussion of the methodological difficulties encountered in assessing stress value and rank ordering of certain life events. Certain trends in this variability were seen in the literature. Women tended to rate the stress of life events higher than men, and young adults assigned higher values to life events than older adults (Horowitz et al., 1977; Masuda & Holmes, 1978). Psychiatric patients were found to inflate assessments of adjustment to life change (Grant et al., 1978; Horowitz

et al., 1977; Lundberg & Theorell, 1976). Type A coronary prone behavior subjects consistently rated life events as more stressful, especially when associated with perceived loss, than their Type B counterparts (Lundberg & Theorell, 1976; Suls, Gastorf, & Whitenberg, 1979; Theorell, Lind, & Floderus, 1975). Data were conflicting as to whether experienced events were perceived to be more stressful than unexperienced events (Horowitz et al., 1977; Lundberg & Theorell, 1976; Masuda & Holmes, 1978). The influence of education level on the perception of stressfulness of life events was also inconclusive (Masuda & Holmes, 1978; Miller et al., 1974).

Researchers attempted to examine perception in terms of behavioral theory to account for its variability. Behavioral response was viewed as determined by a complex array of internal psychological and physiological components as well as external environmental factors (Brown, 1974; Dohrenwend & Dohrenwend, 1974; Hinkle, 1974; House, 1974; Rabkin & Struening, 1976). Attention was given to the ability of the individual to control his circumstances; feelings of a loss of control were associated with further energy expenditure and negative happenings (Chiriboga, 1977; Dohrenwend & Dohrenwend, 1978; House, 1974; Sarason et al., 1978). Bell (1977) found that

persons exhibiting mental-illness behavior experienced more stressful life events than persons exhibiting mental-wellness behavior during the same 6-month period.

Mechanic (1976) conceptualized the stress experience:

The individual's motivations, skills and defensive capacities do not develop in a vacuum but, rather, reflect the social context in which he is reared and in which he develops his social experience. Psychological stress does not occur without the individual facing a threat of failure or loss; yet the meaning of failure or loss is dependent on social values and the acceptance of cultural definitions of what is valuable. It is the cultural meanings of any subgroup that determine what events will be experienced as stressful.
(p. 5)

Lazarus (1970) concurred with this conceptualization stating that stress reactions were consequences of the coping processes and that these coping processes depended upon cognitive processes of threat appraisal. An environmental demand leads to stress only if individuals anticipate that they will not be able to cope with the demand, and only if the consequences of failure to cope are perceived as important (Garbin, 1979). Consequently, Levine and Scotch (1970) viewed stress as a failure of the individual's adaptive resources or capacities, and Wolf (1971) asserted that death is the ultimate adaptive mechanism. Stress was not entirely detrimental. On the contrary, when life events fell within the individual's capacity

to cope, stress was associated with growth and development (Chiriboga & Dean, 1978; Mechanic, 1976).

In conclusion, Rahe and Arthur (1978) proposed a model of life stress and illness composed of a series of highly individualized processes that occurred between a particular life situation and illness. The first process was perception followed by psychological defenses, psychophysiological response, response management, illness behavior, and finally, illness measure. The influence of life events on illness symptoms and disease were repeatedly documented. Initial conceptions of life changes and illness were simple and straightforward. As evidence for the validity of the general concept mounted, the challenge awaited to develop an all-encompassing model that takes into account not only the environmental variables but also the sociological, psychological, and physiological characteristics of the individual (Rahe & Arthur, 1978).

Summary

Models of stress, stress pathophysiology, and life change events were presented as germane components of the life change/illness phenomenon. The human organism was believed to react to a variety of internal and

external stimuli. This response demonstrated the involved, complex physiological, psychological, and sociocultural determinants.

CHAPTER 3

PROCEDURE FOR COLLECTION OF DATA

The study was a descriptive-correlational research investigation and utilized the questionnaire and content analysis methods of obtaining data. The primary purpose of a descriptive investigation is to identify and describe a phenomenon under study. The hypothesis established an independent variable, life stress, and a dependent variable, the occurrence of an acute episode of coronary artery disease.

Setting

The study was conducted in a 200-bed urban community hospital located in the southwestern United States. The first phase of the study took place in the coronary care unit or the intensive care unit. This was where the patients' charts were reviewed to identify prospective subjects and to determine the severity of the acute episode of coronary artery disease. The intensive care unit consisted of six private rooms arranged in a semi-circle around a central nursing desk. The desk area contained writing space, telephones, and individual patient cardiac

monitors. The actual chart reviews were done at the desk area. In the coronary care unit, five private patient rooms were parallel to two nursing station desks. These desks also contained writing space, telephones, and cardiac monitoring screens. The chart reviews in the coronary care unit took place at the desk areas.

The patient interviews took place in a private or semi-private room in the post-coronary care area. Curtains were available in the semi-private rooms to allow visual privacy. Each room had a sink and private toilet. All beds were electric. Each bed had a console with television, radio, and light controls. Each bed also had intercom access to the nurse's station.

The interviews took place in the late afternoon. This was done so as not to interfere with other activities such as X-rays, tests, meal times, or visiting hours.

Population and Sample

The population was all male patients admitted to a particular hospital to a critical care area with a presenting symptom of chest pain or other symptoms indicative of acute coronary artery disease. The sample was accidental and consisted of all male subjects admitted after the date the investigation began who met the criteria and who agreed to participate in the study.

Protection of Human Subjects

The present study was approved by the Human Subjects Review Committee of Texas Woman's University (Appendix A). This committee assessed the risk factors to human participants that might have been incurred in the research project. Each subject received an explanation of the study and had the option to participate or not; and maintained the option to withdraw at any time (Appendix B). Each subject also signed a consent to act as a subject for research and investigation (Appendix C).

Since all the data were collected in a community hospital, agency permission was obtained from the administrator to review patient charts and conduct interviews (Appendix D). Written permission to interview the patient was obtained from each subject's physician (Appendix E). Prior to collection of data, a form letter was sent to prospectively involved physicians explaining the reason for the interview and soliciting their cooperation (Appendix F).

Instruments

Two instruments were used to obtain the data. The Severity Scale for Coronary Illness (Appendix G) measured the severity of the acute episode of coronary artery

disease. The Social Readjustment Rating Scale (Holmes & Rahe, 1967, Appendix H) measured the recent stressful life events.

The Severity Scale for Coronary Illness was a scale consisting of categories representing various levels of severity of acute coronary illness. Class I was the least severe and Class IV the most severe. This scale was developed in 1978 by the CCU nursing staff in the hospital where data collection took place. Based on the Killip Classifications (Killip & Kimball, 1967), the scale was used to report a patient's clinical status.

Killip and Kimball (1967) utilized the Killip Classifications in predicting the mortality of 250 patients suffering a myocardial infarction. An 81% mortality rate was evident in those subjects classified in the most severe category. Pozen, Stechmiller, and Voigt (1977) misclassified 6% of the 410 subjects with respect to mortality when utilizing the Killip Classifications. In regard to management and treatment of myocardial infarction, Hurst (1978) and Ross, Lesch, and Braunwald (1977) present the Killip Classifications as a predictive index of prognosis.

The Severity Scale for Coronary Illness was used in this study rather than the Killip Classifications because the Killip Classifications are limited to myocardial

infarction. Subjects of the study were suffering from all types of acute coronary artery disease and not just myocardial infarction. The Severity Scale for Coronary Illness contained the Killip Classifications plus classified non-myocardial infarction illnesses as to severity. Documentation as to the reliability and validity of the instrument was not available.

The instrument used to measure stressful recent life events was the Holmes and Rahe Social Readjustment Rating Scale (1967). Permission was obtained for use of this instrument (Appendix I). This instrument consisted of a list of 42 life events reflecting significant change in the life pattern of the individual. The most significant life event listed was "death of spouse," which was given 100, the highest numerical value. The lowest valued life event was "minor violations of the law" and was given a numerical value of 11 (Appendix H--complete list of events and values). The values given each event according to the Social Readjustment Rating Scale were totaled to obtain a score which reflected a numerical value for the subject's recent life stress.

Reliability and validity of this instrument were demonstrated by Rahe (1972) in studies of over 4,000 Navy men. A low order but positive relationship was seen between

subjects' recent life change magnitude and their near-future illness reports. Further studies (Rahe, Romo, Bennett, & Siltanen, 1974) in Sweden strongly correlated incidents of myocardial infarction with increases in recent life changes. Other researchers have demonstrated similar findings. Bramwell, Masuda, & Wagner (1975) demonstrated that among varsity college football players, the risk of injury was proportional to accumulation of life events. Kimball (1971) has drawn attention to the relationship between the onset and exacerbation of diabetes mellitus and psychosocial events. Seyzer and Vinokur (1974) found a significant relationship between the accumulation of life changes and traffic accidents of alcoholic drivers as compared to non-alcoholic drivers.

Life changes scaling studies have been performed in the United States and several foreign countries with strikingly similar results (Rahe, 1969). Life changes scaling by middle-class Americans correlated highly ($r = .90$) with scaling by middle-class Japanese (Masuda & Holmes, 1967). A similar study demonstrated congruent findings ($r = .96$) when scaling of life changes by Americans was compared with scaling of life changes by Swedes (Rahe, Lundberg, Bennett, & Theorell, 1971). The middle-class American scaling sample was also compared to a

group of lower-class Mexican-Americans living in the United States with a lower but significant relationship ($r = .77$) between the two groups' perceptions of life change events (Komaroff, Masuda, & Holmes, 1968).

Data Collection

Daily rounds of the critical care areas were made utilizing census records and patient records to identify prospective subjects. Charts were reviewed and subjects selected on the basis of admission diagnosis, laboratory data, and nurses' notes. The subjects were initially categorized utilizing the Severity Scale for Coronary Illness. This categorization was provisional and may have changed as the subject's condition changed. If a subject's category changed during the hospitalization, the most severe category that a particular subject experienced was the category considered in the data analysis.

Once physician permission had been obtained, several preliminaries were necessary at the time of the interview. These preliminaries were: (a) the patient was approached in his hospital room and asked if he was experiencing any chest pain, nausea, or other problem that would necessitate postponing the interview to a later time; (b) the study was explained, (c) confidentiality was assured,

and (d) the patient permission form was signed. After these conditions had been met, the Schedule of Recent Experience (Appendix J) was administered. The subject was asked if the events had occurred within the past year and also the number of times the event had occurred if it had taken place more than once.

Prior to the collection of data, the patient's name and the category of illness obtained from the Severity Scale for Coronary Illness were written on an index card. To ensure confidentiality, at the conclusion of the subject's interview the numerical value for stress and the severity category number as well as the subject's age, gross annual income range, and marital status were placed on a separate index card. The card containing the subject's name, severity of illness category, and his schedule of recent experience was destroyed at the end of the interview. When all the data were collected, there were 30 index cards containing demographic data, a number indicating the numerical value of life stress, and a number indicating the category of the severity of the illness. In order to ascertain the most frequently occurring event out of the 42 on the Social Readjustment Rating Scale, a separate copy of the Schedule of Recent Experience was

kept with hatch marks placed at each event each time it occurred throughout the data collection.

Treatment of Data

Once the data had been collected, analysis determined if the variables were related and if the dependent variable could be predicted to occur given the occurrence of the independent variable. That is, as the number of recent stressful life events increased, did the severity of an acute episode of coronary artery disease also increase? Frequency, mean, and standard deviation were computed for disease, stress, age, and income in order to describe the sample. Bivariate correlations and multivariate regression were determined to ascertain if a relationship existed between the variables. Tables were constructed depicting the following data: (a) the distribution of subject's ages, (b) the distribution of subject's incomes, (c) a summary of the data for category of illness and life stress score, (d) the occurrence of subjects in disease categories, (f) the mean value of the variables, and (g) the standard deviation of each variable.

CHAPTER 4

ANALYSIS OF DATA

This chapter presents the findings of the data collected in this study. The sample is described using the demographic data provided by the subjects. The findings are used to accept or reject the following hypothesis: There is no significant relationship between the scores on the Social Readjustment Rating Scale, which measures recent stressful life events, and the scores on the Severity Scale for Coronary Illness, which measures the severity of a subsequent episode of acute coronary artery disease. In addition, demographic variables are analyzed to determine if there is any correlation between the demographic variables and either the independent or dependent variables.

Description of Sample

The sample consisted of the first 30 male subjects admitted to a critical care area, who met the aforementioned criteria. The demographic data obtained were: age, marital status, and gross annual income. Since marriage was a constant variable in all but three instances, it was discarded in the analysis for lack of variability.

Ages ranged from 40-78 years with 59 the most frequently occurring age. Fifty percent of the subjects were age 58 or below and 50% were age 59 or above. Table 1 illustrates the distribution of ages throughout the sample.

Table 1
Distribution of Subjects' Ages

Age range	Frequency of Occurrence
40-48	6
49-55	4
56-63	10
64-71	6
72-79	4

Note. $n = 30$.

Gross annual incomes ranged from \$5,000-\$52,000. Fifty percent of the subjects earned \$25,000 or less and 50% earned \$25,000 or more. Table 2 illustrates the distribution of the subjects' incomes throughout the sample.

Findings

The findings of the study reflected the numerical representations of the variables. The hypothesis stated that there was no significant relationship between the

Table 2

Distribution of Subjects' Incomes

Income range ^a	Frequency of occurrence
< 10	5
10-15	6
15-20	3
20-25	1
25-30	5
30-35	5
35-40	3
> 40	2

Note, $n = 30$.

^aReported in thousands of dollars.

scores on the Social Readjustment Rating Scale, which measured recent stressful life events, and the scores on the Severity Scale for Coronary Illness, which measured the severity of an acute episode of coronary artery disease. A description of the findings follows. Table 3 summarizes the data representing the independent and dependent variables.

The occurrence of subjects was more heavily distributed in the less severe Category I. Twenty-four subjects,

Table 3
Summary of Data for Category of Illness
and Life Stress Score

Subject	Category of illness	Life stress score
1	I	421
2	I	237
3	I	90
4	I	171
5	I	619
6	I	131
7	I	90
8	III	433
9	I	64
10	I	261
11	I	254
12	I	211
13	I	261
14	I	359
15	II	187
16	I	207
17	I	241
18	II	173
19	I	299
20	I	271
21	I	254
22	I	163
23	II	16
24	II	489
25	I	456
26	I	270
27	I	619
28	I	91
29	II	329
30	I	63

or 80%, were classified as disease Category I; 5 subjects, or 17%, were classified as disease Category II; 1 subject, or 3%, was classified as disease Category III; and no subjects were classified as disease Category IV. Table 4 illustrates the occurrence of subjects in the different disease categories.

Table 4
Occurrence of Subjects in Disease Categories

Category	Number of Subjects	Percent
I	24	80
II	5	17
III	1	3
IV	0	0

Note. $n = 30$.

In testing the hypothesis, Bivariate Correlations were used to determine the relationship between the scores on the Social Readjustment Rating Scale, which measured recent stressful life events, and the scores on the Severity Scale for Coronary Illness, which measured the severity of a subsequent acute episode of coronary artery disease. A correlation coefficient of .11 was determined

between stress and disease with a p value of .55. The null hypothesis was accepted that there was no significant relationship between recent stressful life events as measured by the scores on the Social Readjustment Rating Scale, and the severity of a subsequent acute episode of coronary artery disease as measured by the scores on the Severity Scale for Coronary Illness.

Several additional findings were apparent after data were analyzed. Inverse relationships were found between stress and income, stress and age, age and income, and income and disease. Of these, one inverse relationship was significant. Age and income showed a $-.40$ correlation with $p = .03$, which is a significant finding. As age increased, income could be expected to decrease given the occurrence of the former variable.

Stress, age, and income were computed using multivariate regression to predict the occurrence of the disease, given the occurrence of any of the former. An 8% explained variance of disease occurred with $R\text{-square} = .08$. Ninety-two percent of disease occurrence therefore was unexplained in terms of stress, age, and income variability.

The most frequently occurring life change event was taking a vacation, which occurred to 15 subjects; followed

by a change in the number of family gatherings, which occurred to 14 subjects; followed by a major change in the health or behavior of a family member, which occurred to 13 subjects. Two life change events occurred to 12 subjects. These were sexual difficulties and major changes in sleeping habits. Three life change events occurred to 11 subjects. These were revising personal habits, experiencing a major personal injury or illness, and experiencing the death of a close friend. See Appendix K for a complete listing of frequency for each life change event.

The data were analyzed to determine the mean for each variable. The results are shown in Table 5.

Table 5
Mean Value of the Variables

Variable	Mean value
Disease	Category I
Life Stress Score	258 LCUs
Age	59 years
Income	\$24,000

The data were analyzed to determine the standard deviation of each variable. The results are shown in Table 6.

Table 6
Standard Deviation of Each Variable

Variable	Standard deviation
Disease	.50
Life Stress Score	155 LCUs
Age	10 years
Income	\$13,000

Summary of Findings

The findings of this study are summarized as follows:

1. There is no significant relationship between recent stressful life events as measured by the scores on the Social Readjustment Rating Scale, and the severity of a subsequent acute episode of coronary artery disease as measured by the scores on the Severity Scale for Coronary Illness. The findings were not significant at $p = .55$ and .11 Bivariate Correlation Coefficient.
2. Of all the Bivariate Correlations, only an inverse relationship between age and income was

significant with a correlation coefficient of $-.40$ and $p = .03$.

3. Eight percent of disease occurrence was explained by multivariate regression in terms of stress, age, and income variability.

4. The most frequently occurring life change event, taking a vacation, occurred to 15 subjects, or 50% of the sample.

5. The mean category for disease was Category I with a standard deviation of $.50$. The mean value of life stress scores was 258 LCUs with a standard deviation of 155 LCUs. The mean value of age was 59 years with a standard deviation of 10 years. The mean value of income was \$24,000 with a standard deviation of \$13,000.

CHAPTER 5

SUMMARY OF THE STUDY

The study was undertaken to determine if there was a relationship between recent stressful life events and the severity of subsequent episodes of acute coronary artery disease. A null hypothesis was proposed: There is no significant relationship between the scores on the Social Readjustment Rating Scale, which measures recent stressful life events, and the scores on the Severity Scale for Coronary Illness, which measures the severity of a subsequent episode of acute coronary artery disease.

Summary

In order to determine if a relationship existed between recent stressful life events and subsequent acute coronary artery disease, data were collected on 30 male subjects admitted to a critical care area who were experiencing an acute episode of coronary artery disease. Two instruments were employed to measure the independent and dependent variables. The subjects were categorized according to the severity of their illness utilizing the Severity Scale for Coronary Illness and interviewed utilizing the

Social Readjustment Rating Scale to determine their recent life stress. Demographic data were also collected. The data were analyzed to test the hypothesis and to ascertain additional findings. The null hypothesis was accepted that there is no relationship between the scores on the Social Readjustment Rating Scale, which measures recent stressful life events, and the scores on the Severity Scale for Coronary Illness, which measures the severity of a subsequent episode of acute coronary artery disease.

Discussion of Findings

The findings of the study indicated that no relationship existed between recent stressful life events and the severity of subsequent acute coronary artery disease. An insignificant correlation of .11 was found with $p = .55$. Eight percent of disease occurrence was explained by stress scores, age, and income. One factor that may have skewed the statistical results was the fact that all but 6 of the 30 subjects were classified into a single illness category. Had the sample size been larger than 30 subjects, a greater variety of disease categories might have occurred.

These findings are consistent, however, with those of Theorell and Rahe (1971). The life change scores reported

by subjects over 1 year prior to infarction showed no correlation with various indices of the severity of the subjects' myocardial infarctions. In contrast, Garrity, Marx, and Simes (1978) found that recent life change correlated at .33 with the seriousness of a wide variety of illnesses in a sample of college freshmen. Garrity, Marx, and Simes (1977) also demonstrated that minor illnesses tended to emerge within 3 months after life change while there was a substantial latency period of 9 months and possible longer between life change and its production of a more severe illness.

The results of the present investigation were consistent with the equivocal nature of other findings in the literature. In a prospective study Theorell, Lind, and Floderus (1975) found that life-change measurement did not predict near-future myocardial infarction. Goldberg and Comstock (1976) found no relationship between life changes and subsequent illness. On the other hand, other researchers have demonstrated significant increases in life changes prior to myocardial infarction and coronary death (Bianchi, Fergusson, & Walshe, 1978; Rahe, Romo, Bennett, & Siltanen, 1974).

As Roy (1976) proposed in the adaptation model of nursing, man has physiologic, psychologic, and social

coping mechanisms that he utilizes to adapt within a changing environment. It would seem that, according to Roy's adaptation model of nursing and the results of the research cited, some individuals fail to cope and maintain the integrity of the organism while others successfully utilize coping mechanisms to adapt to environmental change.

There was a fairly even distribution of ages and incomes. Ten subjects, or 33% of the sample, were between the ages of 56 and 63. Fifteen subjects, or 50% of the sample, earned gross annual incomes greater than \$25,000. This might suggest that these men were at their peak career earning capacity. Ten subjects, or 33% of the sample, were from 64-70 years of age while 11 subjects, or 36% of the sample, earned gross annual incomes of less than \$5,000. This might suggest that this group's income largely came from retirement funds, social security, pension, and the like. Bivariate correlation indicated a significant inverse relationship ($p = .13$) of $-.40$ between age and income.

The life change event that occurred most frequently was the taking of a vacation. This event occurred to 15 subjects, or 50% of the sample. This fact is not surprising since short weekend trips were considered vacations

as well as longer trips. Nine out of the 40 events on the Schedule of Recent Experience were not experienced by any of the subjects. Events not experienced tended to be high scoring events such as "death of spouse," "marriage," "detention in jail," and "fired from work." (See Appendix K for a complete listing.) Sarason, Johnson, and Siegel (1978) stated that one of the three characteristics that a life stress instrument should possess is a list of events experienced with at least some degree of frequency in the population being investigated. Twenty-one percent of the events on the instrument used were not experienced by any of the subjects. Masuda and Holmes (1978) concluded that the differences in demographic characteristics can lead to differences in the accumulation of the quality and quantity of life events; much of what occurs is a product of a lifestyle or culture.

Conclusions and Implications

Based on the results of the study, the following conclusions and implications were drawn:

1. Although recent stressful life events may not be related to severity of illness, studies have demonstrated that the adaptation required by increased numbers of life change events might exceed the individual's ability to

cope with the environmental changes and predispose the individual to illness (Bianchi, Fergusson, & Walshe, 1978; Lundberg & Theorell, 1976; Rahe & Lind, 1971; Rahe & Paasikivi, 1971; Rahe, Romo, Bennett, & Siltanen, 1974; & Theorell & Rahe, 1971, 1975). This implies a challenge for nursing to consider the relationship of stressful life events to health maintenance and illness prevention (Bell, 1977). In assisting the individual to adapt to life stress, the nurse must first understand the concept of stress and recognize its manifestations (Smith & Selye, 1979). The inclusion of life stress assessment tools in nursing assessment has been advocated (Bell, 1977; McNeil & Pesznecker, 1977; & Speich, 1979).

2. The finding that increasing age is accompanied by decreasing income has great implications for assessment and discharge planning by nurses. Low-income elderly individuals are particularly subject to inadequate diets, housing, safety, and health care needs (Dinsmore, 1979). These conditions could be potential life stress precursors. A life stress assessment tool might be developed specifically for an elderly population. Susceptible persons should be identified and referrals made to the appropriate community resources.

Recommendations for Further Study

The following are recommendations of the study:

1. Repeat the study using a larger sample, a group of female subjects, and a control group of hospitalized subjects.
2. Repeat the study and compare two different time periods for life stress events and illness outcomes for each subject.
3. Repeat the study and utilize an instrument that allows the individual to subjectively determine life stress.
4. Repeat the study utilizing a more highly refined instrument for measuring severity of illness.

APPENDIX A

TEXAS WOMAN'S UNIVERSITY

Human Research Committee

Name of Investigator: Mary Anne Secrist Center: Dallas
Address: 917 N.E. 82, Norman, Okla 73071 Date: 9/7/79

Dear Ms. Secrist:

Your study entitled Recent Stressful Life Events and Severity of Subsequent Coronary Illness. has been reviewed by a committee of the Human Research Review Committee and it appears to meet our requirements in regard to protection of the individual's rights.

Please be reminded that both the University and the Department of Health, Education and Welfare regulations require that written consents must be obtained from all human subjects in your studies. These forms must be kept on file by you.

Furthermore, should your project change, another review by the Committee is required, according to DHEW regulations.

Sincerely,

Estelle J. Katz

Chairman, Human Research
Review Committee

at _____.

APPENDIX B

ORAL EXPLANATION OF STUDY TO
PROSPECTIVE SUBJECTS

"Hello, Mr. _____. My name is Mary Anne Secrist. I am a registered nurse and I am conducting a research study to see if there is a relationship between the stress of life and getting sick. Do you feel well enough to talk to me for a few minutes?"

Pause for patient response. Return later if indicated.

"The reason I would particularly like to talk to you is because you came to the hospital with symptoms of heart trouble, and I am interested in whether or not life stress might aggravate heart trouble. Dr. _____ has given his/her permission for you to participate in the study. To participate in the study all you have to do is mark whether any of a list of 42 life events has happened to you during the past year."

Show the patient the list of questions and answer any questions he may have. Reassure the patient he has a choice to agree or decline.

"Your response to these questions is kept completely confidential. All I will do is add up the total number of life events that have happened to you and give you a score for life stress."

If visitors are present make a return appointment.
If not, ask if there are any questions and proceed.

"Before you mark the questions I would like you to sign a consent form to participate in the study. You may change your mind about participating at any time."

APPENDIX C

TEXAS WOMAN'S UNIVERSITY
HUMAN RESEARCH REVIEW COMMITTEE

Title of Project: "Recent Stressful Life Events and
Severity of Subsequent Coronary
Illness"

Consent to Act as a Subject for Research and Investigation:

I have received an oral description of this study, including a fair explanation of the procedures and their purpose, any associated discomforts or risks, and a description of the possible benefits. An offer has been made to me to answer all questions about the study. I understand that my name will not be used in any release of the data and that I am free to withdraw at any time.

Signature

Date

Witness

Date

Please be advised that there is no medical treatment or compensation for physical injuries incurred as the result of participating in this research.

Certification by Person Explaining the Study:

This is to certify that I have fully informed and explained to the above-named person a description of the listed elements of informed consent.

Signature

Date

Position

Witness

Date

APPENDIX D

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE Norman Municipal Hospital

GRANTS TO Mary Anne Secrist
a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem.

Review patient charts in ICU and CCU in order to determine the severity of illness of selected patients. Interview these selected patients with the attending physician's permission. Administer the Schedule of Recent Experience in order to determine the amount of recent life stress. The data will then be correlated to see if a relationship exists between recent stressful life events and severity of subsequent coronary illness.

The conditions mutually agreed upon are as follows:

1. The agency (may) ~~(may not)~~ be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) ~~(may not)~~ be identified in the final report.
3. The agency (wants) ~~(does not want)~~ a conference with the student when the report is completed.
4. The agency is (willing) ~~(unwilling)~~ to allow the completed report to be circulated through interlibrary loan.
5. Other _____

Date: 10/31/79

Richard C. Little
Signature of Agency Personnel

Mary Anne Secrist
Signature of Student

Jane D. Lawrence
Signature of Faculty Advisor

*Fill out & sign three copies to be distributed as follows:
Original - Student; First copy - Agency; Second copy - TWU College of Nursing.

APPENDIX E

Date:

To : Dr. _____.

From: Mary Anne Secrist, R.N.

_____ has been selected as a prospective study subject. He meets the criteria as set forth in the research study "Recent Stressful Life Events and Severity of Subsequent Coronary Illness." I would like your permission to interview this patient to administer the Schedule of Recent Experience.

☐ Yes, you may interview this patient.

☐ No, please do not interview this patient.

_____, M.D.

APPENDIX F

917 SE 82
Norman, Oklahoma 73071

Dear Dr. :

In order to fulfill the requirements for a Master's degree in nursing from Texas Woman's University, it is necessary for me to complete a research study. The study is entitled "Recent Stressful Life Events and Severity of Subsequent Coronary Illness." Mr. Luttrell has given me permission to interview Norman Municipal Hospital patients to obtain data.

During the coming weeks patients admitted to the critical care areas with acute coronary artery disease who meet certain criteria will be categorized according to the severity of their illness. After they are stable and moved to a regular room, you will see a permission form on the chart. With your permission, I will interview the patient and determine the amount of recent life stress. This numerical value for the amount of stress will be correlated with the severity of illness to see if a relationship exists.

The patient will have the opportunity to agree or decline to participate in the study. The patient's well-being is, of course, our main concern. This is why your judgment is requested.

Enclosed please find the list of questions included in the interview and the permission form. I look forward to sharing the study results with you.

Thank you for your time and consideration.

Sincerely,

Mary Anne Secrist, R.N.

APPENDIX G

Severity Scale for Acute Coronary Illness

Class	Documented MI	Non-MI
I	Uncomplicated; no rales, gallop; transient or controlled arrhythmias	Intermittent or transient pain easily controlled; no or transient arrhythmias; questionable embolus
II	Moderate ventricular failure; rales, gallop, or rub; persistent or difficult to control arrhythmias	Equivocal lab results, pain difficult to control, mild CHF; newly diagnosed embolus; persistent arrhythmias
III	Pulmonary edema associated with MI; poorly controlled arrhythmias; extension of recent MI	Pulmonary edema without recent MI; poorly controlled arrhythmias; complicated embolus
IV	Cardiogenic shock; cardiac arrest; recurrent death-producing arrhythmias	Severe myocardial ischemia without infarction; irretractable pulmonary edema; acute respiratory distress associated with embolus

APPENDIX H

SOCIAL READJUSTMENT RATING SCALE

Rank	Life Event	Mean Value
1	Death of spouse	100
2	Divorce	73
3	Marital separation	65
4	Jail term	63
5	Death of close family member	63
6	Personal injury or illness	53
7	Marriage	50
8	Fired at work	47
9	Marital reconciliation	45
10	Retirement	45
11	Change in health of family member	44
12	Pregnancy	40
13	Sex difficulties	39
14	Gain of new family member	39
15	Business adjustment	39
16	Change in financial state	38
17	Death of close friend	37
18	Change to different line of work	36
19	Change in number of arguments with spouse	35
20	Mortgage over \$10,000	31
21	Foreclosure of mortgage or loan	30
22	Change in responsibilities at work	29
23	Son or daughter leaving home	29
24	Trouble with in-laws	29
25	Outstanding personal achievement	28
26	Wife begin or stop work	26
27	Begin or end school	26
28	Change in living conditions	25
29	Revision of personal habits	24
30	Trouble with boss	23
31	Change in work hours or conditions	20
32	Change in residence	20
33	Change in schools	20
34	Change in recreation	19
35	Change in church activities	19
36	Change in social activities	18
37	Mortgage or loan less than \$10,000	17
38	Change in sleeping habits	16
39	Change in number of family get-togethers	15
40	Change in eating habits	15

41	Vacation	13
42	Christmas	12
43	Minor violations of the law	11

Source: Holmes, T. H., and Rahe, R. H. Journal of Psychosomatic Research. 11:213-218, 1967.

Note. The listing of life change events by rank order of their mean LCU scores creates a scale that is known as the Social Readjustment Rating Scale. "Christmas" was included in the scaling studies only and has never been included in the Schedule of Recent Experience. Therefore, 43 items appear in the Social Readjustment Rating Scale and 42 items appear in the Schedule of Recent Experience.

APPENDIX I



DEPARTMENT OF THE NAVY
NAVAL HEALTH RESEARCH CENTER
SAN DIEGO, CALIFORNIA 92152

IN REPLY REFER TO:
15 August 1978

Ms Mary Anne Secrist, R.N., B.S.
813 East Acres
Norman, OK 73071

Dear Ms Secrist:

In response to your letter of 11 August 1978, I've enclosed a packet of information for researchers on the derivation of the RLCQ. I believe this is the questionnaire you should use in your upcoming studies. I've also enclosed a recent reprint (74-41) which gives the questionnaire in its appendix. You may also want to use the subjective scaling technique along with the use of the standard life change units. Appropriate references for this scaling will be found in the enclosed editorial, which I recently wrote for Psychosomatic Medicine. I look forward to learning of your results.

Sincerely yours,

RICHARD H. RAHE
Captain, MC USN
Commanding Officer

Encls.

APPENDIX J

SCHEDULE OF RECENT EXPERIENCE
Holmes and Rahe, 1964

Occurrence in
past 1 year

Item

-
- | | |
|-------|---|
| _____ | 1. Mark the number of times there has been either a lot more or a lot less trouble with the boss. |
| _____ | 2. Mark the number of times there was a major change in sleeping habits (sleeping a lot more or a lot less, or change in part of day when asleep). |
| _____ | 3. Mark the number of times there was a major change in eating habits (a lot more or a lot less food intake, or very different meal hours or surroundings). |
| _____ | 4. Mark the number of times there was a revision in your personal habits (dress, manner, association, etc.). |
| _____ | 5. Mark the number of times there was a major change in your usual type and/or amount of recreation. |
| _____ | 6. Mark the number of times there was a major change in your social activities (e.g., clubs, dancing, movies, visiting, etc.). |
| _____ | 7. Mark the number of times there was a major change in church activities (e.g., a lot more or a lot less than usual). |
| _____ | 8. Mark the number of times there was a major change in number of family get-togethers (e.g., a lot more or a lot less than usual). |
| _____ | 9. Mark the number of times you had a major change in financial state (e.g., a lot worse off or a lot better off than usual). |
| _____ | 10. Mark the number of times you had in-law trouble. |
| _____ | 11. Mark the number of times you had a major change in the number of arguments with spouse (e.g., either a lot more or a lot less than usual regarding child rearing, personal habits, etc.). |
| _____ | 12. Mark the number of times you had sexual difficulties. |

- _____ 13. Mark the number of times you experienced major personal injury or illness.
- _____ 14. Mark the number of times you have lost a close family member (other than spouse) by death.
- _____ 15. Mark the number of times you have experienced the death of spouse.
- _____ 16. Mark the number of times you have experienced the death of a close friend.
- _____ 17. Mark the number of times you have gained a new family member (e.g., through birth, adoption, elderly person moving in, etc.)
- _____ 18. Mark the number of times there has been a major change in the health or behavior of a family member.
- _____ 19. Mark the number of times you have had a change in residence.
- _____ 20. Mark the number of times you have experienced detention in jail or other institution.
- _____ 21. Mark the number of times you have been found guilty of minor violations of the law (e.g., traffic tickets, jay walking, disturbing the peace, etc.).
- _____ 22. Mark the number of times you have undergone a major business readjustment (e.g., merger, reorganization, bankruptcy, etc.).
- _____ 23. Mark the number of times you married.
- _____ 24. Mark the number of times you were divorced.
- _____ 25. Mark the number of times you had marital separation from your mate.
- _____ 26. Mark the number of times you had an outstanding personal achievement.
- _____ 27. Mark the number of times you had a son or daughter leaving home (e.g., marriage, attending college, etc.).
- _____ 28. Mark the number of times you have experienced retirement from work.
- _____ 29. Mark the number of times there was a major change in working hours or conditions.
- _____ 30. Mark the number of times you had a major change in responsibilities at work (e.g., promotion, demotion, lateral transfer).
- _____ 31. Mark the number of times you have been fired from work.
- _____ 32. Mark the number of times there was a major change in living conditions (building a new

home, remodeling, deterioration of home or neighborhood).

- _____ 33. Mark the number of times your wife began or ceased working outside the home.
- _____ 34. Mark the number of times you took on a mortgage greater than \$10,000 (e.g., purchasing a home, business, etc.).
- _____ 35. Mark the number of times you took on a mortgage or loan less than \$10,000 (e.g., purchasing a car, T.V., freezer, etc.).
- _____ 36. Mark the number of times you experienced a foreclosure on a mortgage or loan.
- _____ 37. Mark the number of times you have taken a vacation.
- _____ 38. Mark the number of times you have changed to a new school.
- _____ 39. Mark the number of times you have changed to a different line of work.
- _____ 40. Mark the number of times you have begun or ceased formal schooling.
- _____ 41. Mark the number of times you had a marital reconciliation with your mate.
- _____ 42. Mark the number of times you had a pregnancy.

APPENDIX K

SCHEDULE OF RECENT EXPERIENCE
Holmes and Rahe, 1964

Occurrence in past 1 year	Item
<u>3</u>	1. Mark the number of times there has been either a lot more or a lot less trouble with the boss.
<u>12</u>	2. Mark the number of times there was a major change in sleeping habits (sleeping a lot more or a lot less, or change in part of day when asleep).
<u>7</u>	3. Mark the number of times there was a major change in eating habits (a lot more or a lot less food intake, or very different meal hours or surroundings).
<u>11</u>	4. Mark the number of times there was a revision in your personal habits (dress, manner, association, etc.).
<u>14</u>	5. Mark the number of times there was a major change in your usual type and/or amount of recreation.
<u>9</u>	6. Mark the number of times there was a major change in your social activities (e.g., clubs, dancing, movies, visiting, etc.).
<u>10</u>	7. Mark the number of times there was a major change in church activities (e.g., a lot more or a lot less than usual).
<u>14</u>	8. Mark the number of times there was a major change in number of family get-togethers (e.g., a lot more or a lot less than usual).
<u>9</u>	9. Mark the number of times you had a major change in financial state (e.g., a lot worse off or a lot better off than usual).
<u>2</u>	10. Mark the number of times you had in-law trouble.
<u>1</u>	11. Mark the number of times you had a major change in the number of arguments with spouse (e.g., either a lot more or a lot less than usual regarding child rearing, personal habits, etc.).
<u>12</u>	12. Mark the number of times you had sexual difficulties.

- | | |
|-----------|--|
| <u>11</u> | 13. Mark the number of times you experienced major personal injury or illness. |
| <u>6</u> | 14. Mark the number of times you have lost a close family member (other than spouse) by death. |
| <u>0</u> | 15. Mark the number of times you have experienced the death of spouse. |
| <u>11</u> | 16. Mark the number of times you have experienced the death of a close friend. |
| <u>3</u> | 17. Mark the number of times you have gained a new family member (e.g., through birth, adoption, elderly person moving in, etc.) |
| <u>13</u> | 18. Mark the number of times there has been a major change in the health or behavior of a family member. |
| <u>3</u> | 19. Mark the number of times you have had a change in residence. |
| <u>0</u> | 20. Mark the number of times you have experienced detention in jail or other institution. |
| <u>1</u> | 21. Mark the number of times you have been found guilty of minor violations of the law (e.g., traffic tickets, jay walking, disturbing the peace, etc.). |
| <u>3</u> | 22. Mark the number of times you have undergone a major business readjustment (e.g., merger, reorganization, bankruptcy, etc.). |
| <u>0</u> | 23. Mark the number of times you married. |
| <u>2</u> | 24. Mark the number of times you were divorced. |
| <u>3</u> | 25. Mark the number of times you had marital separation from your mate. |
| <u>5</u> | 26. Mark the number of times you had an outstanding personal achievement. |
| <u>4</u> | 27. Mark the number of times you had a son or daughter leaving home (e.g., marriage, attending college, etc.). |
| <u>3</u> | 28. Mark the number of times you have experienced retirement from work. |
| <u>4</u> | 29. Mark the number of times there was a major change in working hours or conditions. |
| <u>3</u> | 30. Mark the number of times you had a major change in responsibilities at work (e.g., promotion, demotion, lateral transfer). |
| <u>0</u> | 31. Mark the number of times you have been fired from work. |
| <u>6</u> | 32. Mark the number of times there was a major change in living conditions (building a new |

home, remodeling, deterioration of home or neighborhood).

- | | |
|---------------|---|
| <u>3</u> | 33. Mark the number of times your wife began or ceased working outside the home. |
| <u>1</u> | 34. Mark the number of times you took on a mortgage greater than \$10,000 (e.g., purchasing a home, business, etc.). |
| <u>7</u> | 35. Mark the number of times you took on a mortgage or loan less than \$10,000 (e.g., purchasing a car, T.V., freezer, etc.). |
| <u>0</u> | 36. Mark the number of times you experienced a foreclosure on a mortgage or loan. |
| <u>15</u> | 37. Mark the number of times you have taken a vacation. |
| <u>0</u> | 38. Mark the number of times you have changed to a new school. |
| <u> </u> | 39. Mark the number of times you have changed to a different line of work. |
| <u>2</u> | 40. Mark the number of times you have begun or ceased formal schooling. |
| <u> </u> | 41. Mark the number of times you had a marital reconciliation with your mate. |
| <u> </u> | 42. Mark the number of times you had a pregnancy. |

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