Exploration of the relationship between time of myocardial infarction pain onset and the patients morningness-eveningness pattern
by Mary Ellen Fry Davis

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Nursing
Montana State University

Abstract:
The purpose of this study was to explore the relationship between a patient's morningness-eveningness pattern and the time of myocardial infarction (MI) pain onset. An ex post facto/correlational design was used to examine the research questions. Horne and Ostberg's Morningness-Eveningness Questionnaire (1976) (MEQ) was the tool used. The study determined morningness-eveningness pattern, evaluated trends in the time of MI pain onset, and tested the relationship of morningness-eveningness patterns and time of MI pain onset. The sample included 30 patients admitted with a diagnosis of myocardial infarction to the critical care units of a hospital in the northwestern United States.

Correlations were examined between the scores on the MEQ and the time of MI pain onset using Spearman Rank Order correlation test. The results of this test indicated there was no relationship between these two variables ($rs=0.059$, $p>0.05$). A trend in the time of MI pain onset was noted to occur between the hours of 6:00 a.m. and noon (57%).

Twenty-six of the participants (80%) fell into the morning type category, six (17%) into the intermediate category, and one (3%) into the evening category.

These results suggest that MIs tend to occur between 6:00 a.m. and noon without regard to morningness-eveningness pattern, although further validation will be required. An implication for nursing is to administer drugs and treatments during the hours of maximum therapeutic response and minimum adverse reactions. Also, the nurse may help the patient to alter his activity level so that myocardial oxygen requirement is, at a minimum during this time in an effort to reduce mortality and morbidity.

EXPLORATION OF THE RELATIONSHIP BETWEEN TIME OF MYOCARDIAL INFARCTION PAIN ONSET AND THE PATIENT'S MORNINGNESS-EVENINGNESS PATTERN

by

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APPROVAL

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Mary Ellen Fry Davis

This thesis has been read by each member of the thesis committee and has been found to be satisfactory regarding content, English usage, format, citations, bibliographic style, and consistency, and is ready for submission to the College of Graduate Studies.

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VITA

Mary Ellen Fry Davis, the daughter of Elmer and Dorothy Fry, was born September 5, 1951, in Hutchinson, Kansas. She received her secondary education from East High School, Phoenix, Arizona. She graduated from Arizona State University's College of Nursing with a Bachelor of Science in Nursing in 1973.

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TABLE OF CONTENTS

Table of Contents - Continued

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

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CHAPTER 1

INTRODUCTION

The leading cause of death in the United States is disease of the heart and blood vessels. An estimated 64,890,000 Americans have one or more forms of heart or blood vessel disease; 4,870,000 of these have coronary artery disease (American Heart Association [AHA], 1988). In 1988 as many as 1.5 million people were predicted to have heart attacks and about 540,000 of them will die. Each year approximately 350,000 victims of heart attacks die before they reach the hospital as the average victim waits three hours before deciding to seek help.

Patients may present themselves to the hospital emergency room with a complaint of severe chest pain that is typical for angina pectoris or acute myocardial infarction. Angina pectoris is a pain or discomfort caused by poor oxygen supply to the heart muscle. The pain is often described as heavy, crushing, squeezing, or pressing and may be centered or more diffuse throughout the anterior chest. It may radiate to one (more often the left) or both shoulders and/or arms. The pain may also radiate to the neck, jaw, back, or epigastrium. Generally, angina pectoris pain lasts 10 to 15 minutes and is described as a steady discomfort. Any factor that increases myocardial oxygen demand or decreases the supply of oxygen to the heart may precipitate the pain. Coronary atherosclerosis is the most frequent cause of the pain. Angina is promptly relieved by rest and/or nitroglycerin (Herrin & Montgomery, 1985).

Acute myocardial infarction, also known as heart attack, occurs when the demand of the heart muscle (myocardium) for oxygen and blood supply far exceeds the supply. It usually results from severe narrowing or complete blockage of a diseased coronary artery and results in death of the myocardial cells supplied by that artery. The injury to the muscle results in electrical instability that may lead to altered electrical rhythms, including ventricular tachycardia or ventricular fibrillation. The usual symptom of a heart attack is a sudden pressure or pain in the chest that persists for two minutes or longer and is not relieved by rest and/or nitroglycerin (Herrin & Montgomery, 1985).

For those patients fortunate enough to reach coronary care units, mortality from acute myocardial infarction has substantially decreased. This has been accomplished through immediate resuscitation of the cardiac arrest victim. More importantly, cardiac arrest may be prevented by early therapy aimed specifically at prevention of life threatening dysrhythmias, continuous electrocardiographic (EKG) monitoring and other forms of aggressive therapy (McIntyre & Lewis, 1983).

In spite of the frequency and severity of myocardial infarction (MI), little is known about the events that transform a chronic, stable condition to an acute, life threatening illness. It is believed that myocardial infarction is generally preceded by coronary thrombosis. However, recent studies have shown that coronary artery spasm may play a major part in acute infarction, possibly by disrupting the intimal surface or rupturing an atherosclerotic plaque (DeWood et al., 1980). Unfortunately, the circumstances that trigger these pathologic events remain unknown.

Conceptual Framework

The current theory of human endogenous, biologic clocks proposes that humans have a daily, 24-hour rhythmic clock for each bodily process (Aschoff, 1985). The oscillating cycles are regulated by a system of internal physiologic clocks (Binkley, 1970; Natalini, 1977). A rhythm consists of recurring events, which are termed cycles. The length of time of a single cycle is called a period. Many biological rhythms studied have a period of about 24 hours, which are called circadian (circa, about: diem, day) rhythms. Periods much less than a day are called ultradian, like those in the 90 minute sleep cycles. Rhythms that are greater than a day are called infradian. Other cycles can range from a month to a year and are considered circamonthly or circannual rhythms. The frequency is the reciprocal of the period; for example, the body temperature peaks once every 24 hours, so the frequency is 1:24 (Natalini, 1977).

The site and nature of this body controlling biological clock are still questioned. Binkley (1979) relayed that in some vertebrate animals, the pineal gland, a small organ embedded in the brain, may be the location of this biologic clock. The exact role of the environment in the establishment, maintenance, and modification of the observed rhythms remains uncertain. It is believed, however, that the biologic clock is capable of gradually adapting its 24-hour rhythm in response to temporary changes in the environment (Luce, 1971). The driving force behind a rhythm is called Zeitgeber or synchronizer. The most common Zeitgeber is the day/night cycle (Natalini, 1977). Circadian rhythmicity has been studied as a potential causative factor in several different physiological variations. The most noticeable of all 24-hour physiologic oscillations is the rhythmic pattern of sleep and wakefulness. Other circadian periodicities have been noted in many circulatory functions, including blood pressure, heart rate, plasma volume, cardiac output, and occurrence of death-producing dysrhythmias (Bunning, 1967).

Nursing Implications

Hours of vulnerability and resistance to drugs and illness also vary according to this 24-hour biologic clock (Luce, 1971). If one's response to drugs or infection depends upon the time of day within the body, the timing of drugs may be, in some cases, as pertinent as the dosage of the drug itself.

The statistics provided by the American Heart Association which were cited earlier reveal that death from myocardial infarction is likely to occur if the symptoms are not recognized early and promptly treated. When working with patients in whom sudden cardiac death is likely to occur, such as those with severe chest pain related to angina pectoris or postmyocardial infarction, it is essential that nursing is aware that a preventative approach needs to be taken to substantially reduce mortality.

A greater awareness of the specific hours that the onset of myocardial infarction pain is likely to occur has implications for alterations in the patient's life style. Drugs and treatments should be administered during the hours of maximum therapeutic response and minimum adverse effects. Activity levels may be adjusted so that myocardial oxygen requirement is at a minimum during this time frame as well.

Of course, these treatment alterations are important for the patient during hospitalization following the acute event, but they are equally as important (or even more so because he will be in an unmonitored situation) at home. The patient may help promote internal stability of his body functions by adjusting medication regimen and activity levels to his individual circadian rhythm. If the patient is aware of his own circadian rhythm and the physiological changes and implications during the 24-hour period, he may be more willing to adjust his life style accordingly. Nurses can facilitate self-care by making the patient aware of circadian variation and the implications of these changing physiological rhythms.

Purpose Statement

The purpose of this study was to explore the relationship between the time of myocardial infarction pain onset and the patient's morningness or eveningness pattern. Specific questions addressed in this study included the following:

1. Are there trends in the time of day or night when myocardial infarction pain onset occurs?

2. Are there trends in times of the day or night when myocardial infarction pain starts for those persons classified as morning types?

3. Are there trends in times of the day or night when myocardial infarction pain starts for those persons classified as evening types?

4. Is there a relationship between the time of myocardial infarction pain onset and the patient's morningness or eveningness pattern?

Definition of Terms

For the purpose of this study, the following definitions were utilized.

1. Myocardial infarction (MI) - Cardiac enzymes and/or EKG results, as read by physicians, were positive for myocardial necrosis. Creatinine phosphokinase (CK) value was greater than 210 IU/L with a CK-MB II isoenzyme value of 1-5% of the total CK value. Creatinine phosphokinase is an enzyme in the blood that elevates when brain tissue, heart tissue, or muscle tissue are damaged. CK-MB II is the isoenzyme that specifically identifies the percentage of CK level due to heart muscle damage.

2. Eveningness - Evening is the time of day an individual functions best. Biological processes such as temperature peak later during the day than a morning type person. Individuals who are evening types arise and retire significantly later than morning types. The concept of eveningness was operationalized by using the Horne and Ostberg Morningness - Eveningness Questionnaire (Horne and Ostberg, 1976). Scores ranging from 16 to 41 classified individuals as evening types.

3. Morningness - Morning is the time of day an individual functions best. Biological processes such as temperature peak earlier during the day than an evening type person. Individuals who are morning types arise and retire significantly earlier than evening types. Morningness was operationalized using the Horne and Ostberg Morningness - Eveningness Questionnaire (Horne and Ostberg, 1976). Scores ranging from 59-86 classified individuals as morning types.

4. Myocardial infarction pain - The patient's report of severe chest pain that occurs as a result of a myocardial infarction.

5. Onset - Self report of the time the patient first noticed the chest pain which caused him to come to the hospital.

Assumptions

1. Humans have circadian rhythms of biological processes.

2. Not all biological processes have the same circadian variation.

3. Circadian rhythms of biological processes can be altered.

CHAPTER 2

LITERATURE REVIEW

A review of the literature pertinent to circadian variation and heart disease was done in an effort to gain information that might present valuable insight to the potential timing of the onset of myocardial infarction pain. The review included circadian rhythm studies pertaining to blood pressure, heart rate, body temperature, ventricular ectopy, blood clotting factors, and variant angina. Circadian variation in the frequency of onset of myocardial infarction and frequency of sudden cardiac death were also included. All of the studies mentioned used available patients for their participants. The terms patients, subjects, and participants have been used as they were in each individual study.

Blood Pressure, Heart Rate, and Temperature

Intra-arterial blood pressure (BP) and electrocardiograph (EKG) recordings between untreated essential hypertension (HTN) patients (n=20) and normotensive ambulant patients (n=5) were compared by Miller-Craig, Bishop and Raferty (1978). The recording for all subjects in the study was carried out for 48 consecutive hours on an outpatient basis. Twenty-two of the patients were able to return to their normal working day during the course of the study.

Miller-Craig et al. (1978) found that the hypertensive patients' BP was highest at 10:00 a.m. and then progressively declined throughout the day. BP was lowest at 3:00 a.m. during sleep, and then began to rise again. By 6:00 a.m., BP was increasing rapidly and this rate of increase was emphasized after the patients awoke at approximately 7:00 a.m. As recorded in the patients' diaries, changes in BP were not associated with physical activity. Heart rate (HR) in the same patients was maximal at midday and then fell progressively. During sleep, HR maintained at a low level and did not begin to increase until waking at 7:00 a.m. The HR then increased abruptly during the first two hours after waking.

The normotensive patients in Miller-Craig et al.'s (1978) study had similar results, except there was no peak in BP at 10:00 a.m. Another difference was that the BP began to increase at 5:00 a.m., which was two hours before waking. Heart rate began to rise at 6:00 a.m., reached its peak at 1:00 p.m. and then fell progressively to reach a low point at 4:00 a.m.

Kostis et al. (1982) conducted a study to define the effect of age on HR at different times during the day. Using 24-hour ambulatory electrocardiography and maximal stress testing on 101 subjects with normal hearts, they discovered that with increasing age, there was a decrease of maximal HR achieved during the exercise stress test as well as a decrease in maximal heart rate spontaneously recorded during the day or night. While older subjects had lower exercise tolerance, the resting or average heart rates were not affected by age. A diurnal variation of heart rate was seen with maximal HR occurring in the late morning and minimal HR between 3:00 a.m. and 5:00 a.m. These subjects were carefully screened using noninvasive and invasive means prior to entering the study to insure they did not have undetected heart disease. The age range for the subjects was 16 to 68 years with an average age of 48.8 years.

Deryagina and Kraeuskii (1983) studied circadian rhythm of body temperature (BT), BP, and HR in 41 healthy subjects (ages 21 to 45) and 10 subjects (ages 28 to 55) with ischemic heart diseases (IHD) but not suffering from HTN. The intention of the researchers was (1) to ascertain if personality traits determined the development and course of certain diseases of the cardiovascular system; (2) to determine the effect of work activity on the circadian rhythm of BT, BP and HR in normal subjects and patients with cardiovascular disease; and (3) to learn if there was a possibility of correlation between the circadian rhythm of BT, BP, and HR and the state of adrenal hormonal activity during the 24-hour period.

At the conclusion of their study, Deryagina and Kraeuskii (1983) found that behavioral personality features did not determine the circadian variation of BT, BP, or HR in either group of subjects. The researchers concluded there was evidence of the organizing effect of work activity (the rhythm of the working day); therefore, customary work activity was useful for healthy persons and those with IHD. They also determined that adrenal hormone activity was independent of the circadian variation of BT, BP, and HR.

Messerli et al. (1982) studied 11 patients with uncomplicated borderline HTN, 10 patients with established essential HTN, and 13 normotensive subjects to determine if there was a relationship between the urinary concentrations of epinephrine, norepinephrine, and dopamine, and the diurnal variation in BP, HR, and prevalence of ectopic beats. Urinary catecholamine levels were not related to circadian fluctuations or variability in arterial BP, HR, or prevalence of ectopic beats.

Aslanian, Adamian, Grigorian, Bagdassarian, and Assatarian (1980) studied 141 Russian patients with IHD and 25 healthy subjects in an effort to reveal chronobiological differences of the hemodynamics and blood flow of the myocardium in patients with IHD. All subjects rested from 11:30 p.m. to 7:30 a.m. and ate a meal consisting of bread, apples, matzoan, and milk every three hours while they were awake. The studies were carried out on the second and third days after hospitalization before the patient had received any drugs. Every three hours, BP was measured and an EKG was done on each subject. Analysis of the data collected revealed circadian rhythms of HR, BP, and EKG T-wave voltage in both groups; however, the acrophase (peak) occurred at midday or in the evening hours for the healthy subjects while it occurred in the early morning hours or at night for the IHD group.

Ventricular Ectopy

Interesting findings regarding ventricular ectopy and circadian variation have been presented in the literature. Steinbach, Glogar, Weber, Joskowica, and Kaindl (1982) studied 77 patients with premature ventricular complexes (PVCs) detected during routine resting electrocardiograms. These patients were monitored for at least 24 hours under ambulatory conditions to evaluate the number and quality of PVCs. The findings showed the maximal PVC frequency to vary widely, while the minimal PVC frequency showed a clustering during early hours of sleep between midnight and 2:00 a.m.

Saito, Matsuyama, Niki, and Mori (1984) studied non-sustained ventricular tachycardia (VT) in 21 patients ranging from 28 years to 86 years in age. Each patient had at least one ambulatory EKG record containing episodes of ventricular tachycardia (three or more consecutive beats of ventricular origin). Thirteen of the 21 subjects had some type of underlying heart disease, four had hypertension and two patients had diabetes mellitus. The ambulatory EKG was recorded for 24 hours on each patient while he carried out his normal daily routines and recorded a diary of his activities. Findings of the study showed a circadian variation in the frequency of VT in ambulatory patients. The incidence of VT was higher in the morning and in the evening and was reduced during sleep.

Canada et al. (1983) studied the hourly frequency of ventricular arrhythmias in 164 untreated ambulatory patients in order to establish if a diurnal pattern was a significant characteristic of hourly ventricular arrhythmia frequency. In order to qualify for the study, patients were required to have an average of at least 30 ventricular ectopic beats an hour on each of three 24-hour ambulatory recordings. The results showed each day's frequency curve to demonstrate a distinct diurnal pattern. The frequency of ventricular arrhythmias was significantly lower for hours from 1:00 a.m. to 5:00 a.m., while the subjects were presumably sleeping, than the frequency for the "active" hours (12:00 noon to 4:00 p.m.). Zero hourly frequencies were common in the 10:00 p.m. to 6:00 a.m. period.

In an effort to set limits of normality for PVCs, Rasmussen, Jensen, Schnohr, and Hansen (1985) also found diurnal variation of PVCs. They studied 111 healthy subjects within an age range of 20 to 70 years. The subjects without apparent disease were randomly selected from 19,662 persons participating in the Copenhagen City Heart Study. Ambulatory 24-hour EKG recordings were done on each of the subjects. The number of PVCs was reported highest in the daytime; PVCs were higher in males than females, but the difference between the genders was not statistically significant. The findings revealed that the prevalence of PVCs increased with age, but the increase was not statistically significant.

Blood Clotting Factors

Using the same subjects as reported in Aslanian et al.'s article from 1980, Adamian, Aslanian, and Grigorian (1984) reported finding circadian rhythms of individual indicators characterizing the function state of the cardiovascular system and the blood clotting system in healthy subjects and in patients with ischemic heart disease. In healthy subjects, maximal values of arterial BP, HR, cardiac output, and stroke volume were found during the daytime and in the early evening hours. In patients with IHD, a reversal of this periodicity was found which manifests itself by shifting the acrophases of the myocardial nutrition into night time and early morning hours.

In addition to reporting the hemodynamic and EKG values, Adamian et al. (1984) relayed information concerning the investigation of fibrinogen concentration, the prothrombin index, thrombin time, heparin time, plasma heparin tolerance, total content of plasminogen and proactivators, plasminogen activation inhibitors, and antiplasmins and fibrinase concentration in blood serum. They reported that the activity of plasminogen activation inhibitors rose in patients with IHD during the daytime and reached its maximum toward the evening. A tendency toward hypercoagulation was observed in the late morning hours as well. This was manifested in increased plasma heparin tolerances and shortened thrombin time. Therefore, the researchers concluded that hypercoagulation occurs in patients with IHD twice in the course of 24 hours -- in the evening and in the late morning hours.

In an attempt to ascertain whether six blood clotting indices undergo daily variations in healthy subjects and whether such variations, if any, are similar, dissimilar, or absent in subjects with vascular pathology, Petralito et al. (1982) studied 10 healthy subjects and 10 non-diabetic patients with vascular disease. All subjects were put on a synchronized daily program for at least one week, consisting of daytime activity from 7:00 a.m. to 11:00 p.m., nightly rest from 11:00 p.m. to 7:00 a.m., with meals at 8:00 a.m., 12:00 noon, and 6:00 p.m. Blood samples were drawn every three hours to assess blood fibrinogen level, platelet aggregation, Howell's time, partial thromboplastin time

(PTT), thrombin time (TT), and antithrombin III (AT III). The data obtained for the normal subjects demonstrated statistically significant daily variations in blood fibrinogen levels, platelet aggregation, Howell's time, PTT, and TT values. Platelet aggregation, PTT, and fibrinogen levels, all peaked at approximately 12:00 noon. Howell's time peaked at 9:00 p.m., while TT values peaked at 3:00 a.m. No significant variation over the 24-hour cycle was seen in AT III values.

However, in the patients with vascular disease, Petralito et al. (1982) demonstrated the disappearance of the daily variations of blood fibrinogen levels, platelet aggregation, and Howell's time, so at all times these indices showed an increased clotting or thrombophilic tendency. PTT and TT values continued to show daily variations but the time patterns differed from those demonstrated in healthy subjects. Both PTT and TT values peaked at 6:00 p.m. Unlike in normal patients, the AT III values in patients with vascular disease showed highly significant variations over the 24-hour period. It also peaked at 6:00 p.m.

Variant Angina

Variant angina defined as transient ischemia of the myocardium caused by spasm of major coronary arteries has received much attention in the literature. It is characterized by transient attacks of ST-segment elevation caused by coronary artery spasm; episodes may be spontaneous or provoked and occur both with and without pain. Malignant arrythmias such as ventricular tachycardia, ventricular fibrillation, and complete atrioventricular block are common during this type of angina.

Araki, Yasushi, Nakagaki, and Nakurmura (1983) and Araki and Nakamura (1984) presented results of their study involving the 24-hour ambulatory EKG recordings of 26 patients with variant angina. The reported results revealed that painless ST-segment elevation of 1 mm or greater was common. Seventy-two percent of the ST-segment elevations occurred from midnight to 10:00 a.m. During this period, the number of attacks of variant angina tended to increase every hour until early in the morning. The ST-segment elevations were observed most frequently (13 percent) between 5:00 and 6:00 a.m. Only five percent of ST-segment elevations occurred between 10:00 a.m. and 1:00 p.m.

Waters, Miller, Bouchard, Bosch, and Theroux (1984) studied 13 hospitalized patients with variant angina to assess circadian variations in disease activity. This study demonstrated that most patients with variant angina show circadian variation, with most attacks occurring in the early morning hours, even though the patient was unaware of them. Most of the episodes of myocardial ischemia in these subjects were asymptomatic.

Yasue et al. (1979) conducted treadmill exercise tests in the early morning and in the afternoon of the same day on 13 patients with variant angina. The attacks of ST elevations were induced repeatedly in all 13 patients in the early morning but only in two patients in the afternoon. They concluded that there is circadian variation of exercise capacity in patients with variant angina caused by coronary artery spasm induced by exercise in the early morning but not in the afternoon.

Frequency of Onset of Myocardial Infarction

Some researchers have become curious after reading the data available and questioned if there might be a circadian variation in the frequency of onset of myocardial infarction. Churina, Ganelina, and Vol'pert (1975) studied 49 patients to determine the distribution of the frequency of onset of acute myocardial infarction during the 24-hour period. They described the frequency of onset of MI in Leningrad as reaching a maximum between 8:00 and 11:00 a.m. and that there are two further peaks: at 5:00 to 6:00 p.m. and 1:00 to 2:00 a.m. Myocardial infarction was least likely to occur between 1:00 to 2:00 p.m.

Circadian variation in the frequency of onset of acute myocardial infarction pain was studied by Muller et al. (1985). The findings of their study conducted with 2,999 subjects revealed a marked circadian rhythm in frequency of onset with a peak from 6:00 a.m. to noon. CK-MB II (the serum enzyme specific for myocardial tissue damage) and estimated timing of pain onset confirmed the existence of a circadian rhythm, with a three-fold increase in the frequency onset at a peak of 9:00 a.m. as compared to a trough at 11:00 p.m.

Frequency of Sudden Cardiac Death

To determine whether sudden cardiac death exhibits a circadian rhythm similar to the rhythm for nonfatal myocardial infarction, James Muller et al. (1987) conducted an investigation. They defined sudden cardiac death as death from cardiac disease occurring less than or equal to one hour after the onset of symptoms.

Dr. Muller et al. (1987) examined the mortality records of the Massachusetts Department of Public Health for the year 1983. They determined that 2,203 individuals died out of the hospital from a primary diagnosis of sudden cardiac death. This group decided to conduct the primary analysis on sudden cardiac death occurring out of the hospital since these deaths occur in the presence of normal endogenous and exongenous circadian rhythms. Normal circadian rhythms are disturbed in hospitalized patients. The results of the study showed a distinct circadian rhythm in the frequency of sudden cardiac death. A low frequency of death occurred during the night while the peak occurrence was from 7:00 a.m. to 11:00 a.m.

Henderson (1982), a registered nurse, conducted a descriptive study of circadian rhythm and the hours of postmyocardial infarction mortality. She examined 214 death certificates from the Bureau of Vital Statistics in a large county of the southwestern United States. Each of the individuals had a final diagnosis of acute myocardial infarction and was hospitalized at the time of death. The one-hour, two-hour and four-hour intervals with the largest mortality rates of hospitalized patients were clustered in the hours between 4:00 p.m. and 8:00 p.m. The lowest one-hour and two-hour incidences of death during the 24-hour period occurred during the hours between 4:00 a.m. and 8:00 a.m.

Her study also examined the hours during each shift that the greatest number of deaths occurred. The hours of the day shift when the greatest number of deaths from MI occurred were 8:00 a.m. to 8:59 a.m., 11:00 a.m. to 11:59 a.m., 1:00 p.m. to 1:59 p.m. Thirty-eight of the 214 patients died during these hours. During the afternoon shift, the greatest number of deaths occurred between the hours of 6:00 p.m. to 6:59 p.m., 7:00 p.m. to 7:59 p.m. and 10:00 p.m. to 10:59 p.m. Forty-three patients died during these hours. The peak hour for death from MI during the night shift was 12:00 midnight to 12:59 a.m. when 14 patients died.

Summary

The review of literature presented indicated there is a circadian variation in various hemodynamic factors, clotting mechanisms, and ventricular ectopy in subjects free of cardiac disease. A rhythmicity of hemodynamic factors, ventricular ectopy, and coronary spasm in subjects with cardiovascular disease was found but the clotting factors did not show a rhythmic 24-hour variance. The onset of frequency of myocardial infarction pain shows a 24-hour variation of maximum number in the morning from 6:00 a.m. to noon, and the fewest number from 11:00 p.m. to 2:00 a.m. Frequency of sudden cardiac death also showed a circadian variation with a low frequency of death occurring during the night and a peak occurrence from 7:00 a.m. to 11:00 a.m.

Most of the research reviewed was conducted by physicians. Detailed scientific methodology and analysis of data were clearly presented in the articles. Many of the projects used ambulatory EKG monitors that required the patient to keep a diary of his daily activities while wearing the monitor. None of the researchers asked for a description of the subject's morningness or eveningness pattern while he was not wearing the monitor.

CHAPTER 3

METHODOLOGY

Design

The study was conducted using an ex post facto/ correlational research design. This type of design allows for the exploration of the relationship between variables (Brink & Woods, 1983). The goal of using this style of research design is to describe the relationship among variables rather than to infer cause and effect relationships. A retrospective approach to the study was used so that the researcher could determine the subject's morningness or eveningness pattern and time of myocardial infarction pain onset.

There are several advantages of the ex post facto design. The first is that often it may be morally and ethically impractical to assign humans to experimental treatment groups (Brink & Wood, 1983). Secondly, correlational research is often an efficient and effective means of collecting a large amount of data about a problem area (Polit & Hungler, 1983). Finally, ex post facto research is done in the actual environment where a problem may occur, rather than in an artificial location such as an experimental laboratory. The results of studies in laboratory research are often less generalizable to other settings than are the results from research done in realistic settings.

Although the ex post facto design will always have to be used for this type of study, Polit and Hungler (1983) offered some limitations of this method. First, the researcher is unable to manipulate the independent variables of interest. The second limitation is the inability to randomly assign individuals to an experimental treatment group. The last potential problem is the possibility of faulty interpretation of study results.

Patients who had myocardial infarctions were chosen to be the study group. Purposive sampling method was used because a specific group was chosen for the study (Polit & Hungler, 1983). A total of 30 patients participated.

Sample

A hospital located in the northwestern region of the United States in a city with a population of approximately 58,000 was the area from which the sample population was drawn. This agency also provided care for persons living within a 150 mile radius of the city. It had a capacity of housing 288 patients with acute care needs and 124 patients with extended care needs.

All persons admitted to the hospital with chest pain of cardiac nature were eligible to participate in the research study. Patients with chest pain were identified in the six bed acute coronary care unit (ACCU), the 20 bed step-down coronary care unit (CCU), or the 12 bed intensive care unit (ICU). The criteria requirements for participation were as follows:

1. The patient must have had a diagnosis of myocardial infarction pain.

2. The patient was able to document the time of myocardial infarction pain onset.

3. The patient had medically stable cardiovascular, neurological, and pulmonary systems for a minimum of 24 hours. Cardiovascular stability was evidenced by hemodynamic stability in HR, BP, and cardiac rhythm. Neurological stability was evidenced by the participant remaining alert and oriented to time, place, and person. Pulmonary stability was evidenced by absence of mechanical assistance to breathe and absence of severe hypoxia.

Demographic data was collected. This data included age, marital status, gender, race, education level, occupation, and number of previous myocardial infarctions.

Data Collection

In order to identify which participants met study criteria, the researcher checked with the charge nurses of ACCU, CCU, and ICU every day during the data collection time period to determine if any new patients had been admitted with a diagnosis that may relate to coronary artery disease. If the answer was yes, permission to review the medical record to determine if the new patient met criteria for entry into the study was obtained.

If the newly admitted patient met the study criteria, the charge nurse of the respective critical care unit was contacted to obtain a status report regarding medical stability of the participant. When a patient had been medically stable for 24 hours, the researcher then sought permission from the charge nurse to talk with the patient regarding participation in the study.

The next step was to approach the patient. The investigator briefly explained the purpose of the research and the time involved in filling out the questionnaire. If the patient indicated initial willingness, he was asked to read the explanation of the study form and the consent to participate form (see Appendix A). Those who agreed to participate signed the consent form.

Since there was inconsistency in reporting the initial start of pain on the patient's record, the researcher interviewed the participant to establish the time of myocardial infarction pain onset. After establishing the time of pain onset, the researcher read the questionnaire instructions to the participant to insure that each participant was alerted to all of the instructions. Directions for answering the questions were also provided on a cover sheet attached to the questionnaire so the participant had them for future reference. Upon completion of this task, the researcher left the participant so that he could complete the questionnaire in privacy.

The participant was revisited in 24 hours to determine if the questionnaire was completed. If the questionnaire was not completed, an additional 24 hours was granted to finish it. If it was not completed by the end of the second 24 hours, the questionnaire was collected and classified as an unusable tool.

If the participant decided he did not want to complete the tool, he was instructed to put the unfinished questionnaire in the envelope provided, seal it, and hand it to a staff member. The staff member placed the sealed envelope in the receptacle provided by the investigator on each of the three units.

An alternate procedure was to read the questionnaire to the participant. If the participant told the researcher he was willing to participate but would like the questionnaire read to him, the investigator read the questions and possible answers to him. The researcher was cautious not to add to, subtract from, or interpret the questions for the participants. The participants were instructed that they could refuse to answer a question or decide not to complete the questionnaire even while the researcher was reading it to them.

Instrument

Horne and Ostberg's Morningness-Evenigness Questionnaire (MEQ) (1976) was the instrument used to determine the subject's morningness or eveningness pattern (see Appendix B). Ordinal level data produced from the questionnaire placed a participant into one of five categories: (1) definitely morning type (score 70-86), (2) moderately morning type (score 59-69), (3) neither type (score 42-58), (4) moderately evening type (score 31-41), and (5) definitely evening type (score 16-30). The MEQ took about 20 minutes to complete.

The tool was externally validated using circadian variation of oral temperature measurement and sleep-wake logs compiled by the subjects (Horne and Ostberg, 1976). Spearman rank order correlations were calculated between pairs of dimensions taken from morningness-eveningness, peak oral temperature time, bed time, arising time, and sleep length. Bed time and arising time were both significantly correlated with Morningness-Eveningness (rs=-0.67, -0.79 respectively, p:001 [p:=p≤ in the United States]).

Mecacci and Zani (1983) also presented tool validation in their study when they evaluated the sleep-wake logs of 475 subjects and their scores from the MEQ. A t-test for matched samples was used to compare the expressed preferences of the Morningness-Eveningness Questionnaire and the sleep-waking habits. A significant difference was found in morningness workers between the expressed preference of morningness and eveningness and the sleep log data, both in rising time (t=4.38, p<0.001) and time in bed (t=11.05, p<0.001), but not in bedtime. Significant differences were found for eveningness workers in bedtime (t=3.21, p<0.01), rising time (t=10.44, p<0.001), and time in bed (t=6.14, p<0.001).

Posey and Ford (1981) tested the reliability of the MEQ using 259 college students. They found the score distribution to be essentially normal and compatible with the five point scale suggested by Horne and Ostberg. They reported a test-retest reliability coefficient of .89.

Permission to use the tool was sought from both of the authors. Dr. Ostberg returned the consent (see Appendix C) and signed permission for both authors. The publishing company holding the copyright also authorized use of the questionnaire (see Appendix C).

A pilot study using 15 participants was done to test for readability of the instrument. Eight hospital employees and seven local restaurant employees completed the questionnaire. The results of the pilot study indicated that the participants did not always see the terms a.m. or p.m. on the questionnaire. As a result, several indicated they marked incorrect answers.

To alleviate this problem, the word "morning" was added in parentheses each time the term a.m. appeared in the questionnaire. In a similar manner, the term p.m. was followed by the word "evening" (see Appendix B). In addition, the terms representing the same time frame were color coded. Morning and a.m. were coded blue while evening and p.m. were coded orange.

Protection of Human Subjects

After a patient was identified as eligible for the study, the researcher approached the person and explained the nature of the research and time involved. The participant then read the explanation of the study and the consent form (see Appendix A). Before deciding to participate the patient was given the opportunity to look through the questionnaire and ask questions of the researcher. Once he had decided to participate in the study, he signed the consent form implying that he understood the information presented. He was given the explanation of the study form for his records.

The participant had the right not to participate in the study and was clearly informed of this verbally and in the consent form. If the patient decided not to participate, his wish was respected. No form of coercion or punishment was used in an effort to persuade him to comply. Participants experiencing fatigue or any other medical symptoms were encouraged to discontinue answering the questionnaire.

Confidentiality was maintained by removing the signed consent form from the questionnaire. The questionnaire was left with the participant in order to provide privacy to complete the form. Each participant was instructed to complete the questionnaire within 24 hours and to place the completed questionnaire in the envelope provided and seal it. Any staff member could then take the sealed envelope and place it in the receptacle provided. The researcher collected the sealed envelopes at regular intervals.

Upon completion of data collection, the signed consent forms are kept in a sealed envelope in a locked file in the College of Nursing at Montana State University (MSU) for three years. After this time period, they will be shredded and burned per MSU protocol.

Feedback regarding the results of the research is available as group data in the thesis completed by the investigator. The bound copy of the completed work will be available for public examination at the Montana State University Library in Bozeman.

Analysis of Data

The purpose of this study was concerned with relationships of two variables. According to Brink and Wood (1983), this style of question is a level II research question. At level II there are two steps in data analysis. The first is to put the data into tables for descriptive statistics to describe trends in the data and peaks of onset of chest pain in the morning group and the evening group. The second is to test for the significance between the onset of pain and morningness or eveningness. For this particular study, the researcher used the Spearman's rank order test because the morningness or eveningness was determined using ordinal level data measurements. Polit and Hungler (1983) indicate that nonparametric tests should be used to test for significance when using ordinal level data.

CHAPTER 4

RESULTS

Sample

Over a period of three months, a total of 34 people met the study sample requirements, and of that group, 30 agreed to participate. All 30 completed usable questionnaires. Of the 30 who participated, 28 (93%) elected to have the questionnaire read to them. The demographic data collected for this group included age, race, gender, marital status, educational level and occupation.

The sample consisted of 23 males (77%) and seven (23%) females, all of whom were Caucasian. The age range was from 39 to 80 with a mean of 60.5 years (Table 1). Twenty-five (83%) of the participants were married at the time of the study. Two (7%) of the study participants were widowed while three (10%) were divorced (Table 2). Seven (23% had less than a high school education, nine (30%) of the participants graduated from high school, and 14 (47%) had more advanced education (Table 3).

Table 1. Age Distribution of Participants (N=30)

Age	Frequency	Percentages
39 to 49	6	20%
50 to 59	7	23%
60 to 69	11	37%
70 to 80	6	20%

Mean = 60.5 Standard Deviation = 11.3

Table 2. Marital Status of Participants (N=30)

Marital Status	Frequency	Percentages
Married	25	83%
Widowed	2	7%
Divorced	3	10%

Table 3. Educational Level of Participants (N=30)

Educational Level	Frequency	Percentages
Less than High School	7	23%
High School Graduate	9	30%
More than High School	14	47%

Mean = 13.1 years of education

Standard Deviation = 3.1 years of education

No pattern was seen when examining the occupations of the participants. Eight (27%) were retired from a variety of jobs. Examples of their careers prior to retiring included dentist, farmer, highway maintenance supervisor, and gas field superintendent. Twenty-two (73%) of the participants were still working. A few examples of their occupations included truck driver, nurse, school teacher, rancher, and legal secretary.

Fifteen (50%) of the participants scored between 70 and 86 on Horne and Ostberg's Morningness-Eveningness Questionnaire (MEQ), which classified them as definite morning types. Nine (30%) scored between 59 and 70; therefore, they were classified as moderate morning types. Five members (17%) of the sample scored between 42 and 58, which classified them as intermediate (neither) types. No one scored between 31 and 41, which was the moderate evening type category. Only one (3%) participant scored between 16 and 30. She was classified as a definite evening type.

Table 4. Morningness-Eveningness Classification (N=30)

Score	Frequency	Percentages
70-86	15	50%
59-69	9	30%
42-50	6	17%
31-41	0	0%
16-30	1	3%

A summary of the MEQ scores indicated that the majority (80%) of the participants classified as morning types as operationally defined for this study. The second largest group was intermediate types. Evening type was the smallest classification of participants.

Findings

In this study, four research questions were evaluated to explore the relationship between time of myocardial infarction pain onset and the patient's morningness or eveningness pattern. The first question examined trends in the time of day or night when myocardial infarction pain onset occurred for the entire study group. The data collected showed that in the first six hour time period, 12:00 midnight to 5:59 a.m., chest pain began for seven participants (23%). During the next six hour time block, 6:00 a.m. to 11:59 a.m., 17 participants (57%) reported onset of chest pain. From 12:00 noon to 5:59 p.m., three participants (10%) reported onset of pain as did three participants (10%) from 6:00 p.m. to 11:59 p.m. (Figure 1 & Table 5).

Figure 1. Hourly Distribution of Myocardial Infarction Pain Onset (N=30)

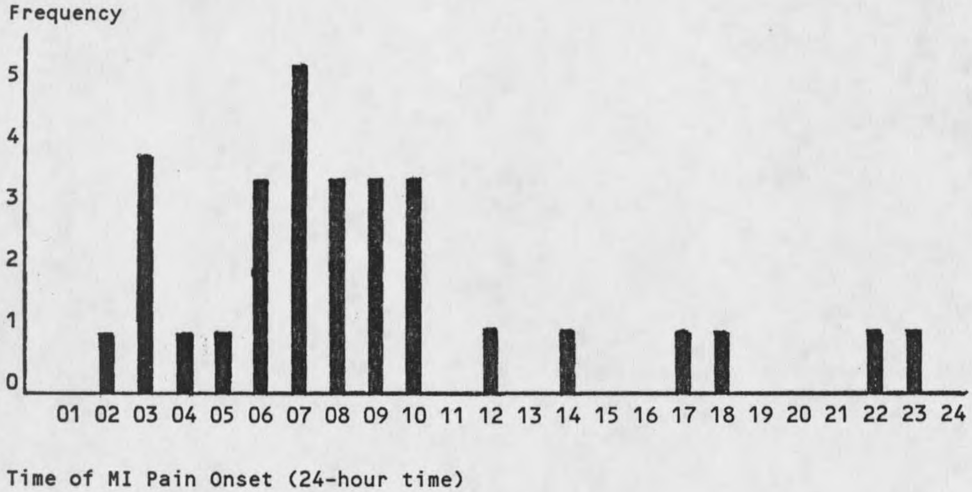

Table 5. MI Pain Onset Distribution (N=30)

Time Period	Frequency	Percentages
12:00 midnight to 5:59 a.m.	7	23%
6:00 a.m. to 11:59 a.m.	17	57%
12:00 noon to 5:59 p.m.	3	10%
6:00 p.m. to 11:59 p.m.	3	10%

Data analysis showed that the peak incidence of myocardial infarction pain in this study occurred between 6:00 a.m. and 12:00 noon. The hours from 12:00 midnight to 5:59 a.m. encountered the second peak in myocardial infarction pain. A tie for the lowest number of MIs occurred between the 12:00 noon to 5:59 p.m. and the 6:00 p.m. to 11:59 p.m. time blocks.

Trends of MI Pain Onset for Morning Types

Research question two evaluated the time of MI pain onset for those classified as morning types to determine if there were trends in the time pain began. The 15 participants (50%) scoring 70 to 86 on the Horne and Ostberg Morningness-Eveningness Questionnaire (MEQ) were classified as definite morning types. Nine (30%) scored 59 to 69 on the MEQ and were classified as moderate morning types. As operationally defined, these two groups (80%) were combined and evaluated as one group for this study.

The range for MI pain onset for morning types was from 2:00 a.m. to 10:00 p.m. (Figure 2). Myocardial infarction pain began between 12:00 midnight and 5:59 a.m. for seven (29%) of this group. Onset of pain for 13 (54%) of the morning types occurred between 6:00 a.m. and 11:59 a.m. Between 12:00 noon and 5:59 p.m., two (8%) of the morning participants reported onset of pain as did two (8%) between 6:00 p.m. and 11:59 p.m. (Table 6).