

1-1-1994

## **A comparison of two bathing methods : Effects on the haemodynamic and subjective responses of low risk patients within 48 hours post myocardial infarction**

Heather M. Kidd  
*Edith Cowan University*

Follow this and additional works at: <https://ro.ecu.edu.au/theses>



Part of the [Nursing Commons](#)

---

### **Recommended Citation**

Kidd, H. M. (1994). *A comparison of two bathing methods : Effects on the haemodynamic and subjective responses of low risk patients within 48 hours post myocardial infarction*. Edith Cowan University.  
Retrieved from <https://ro.ecu.edu.au/theses/1094>

This Thesis is posted at Research Online.  
<https://ro.ecu.edu.au/theses/1094>

# Edith Cowan University

## Copyright Warning

You may print or download ONE copy of this document for the purpose of your own research or study.

The University does not authorize you to copy, communicate or otherwise make available electronically to any other person any copyright material contained on this site.

You are reminded of the following:

- Copyright owners are entitled to take legal action against persons who infringe their copyright.
- A reproduction of material that is protected by copyright may be a copyright infringement. Where the reproduction of such material is done without attribution of authorship, with false attribution of authorship or the authorship is treated in a derogatory manner, this may be a breach of the author's moral rights contained in Part IX of the Copyright Act 1968 (Cth).
- Courts have the power to impose a wide range of civil and criminal sanctions for infringement of copyright, infringement of moral rights and other offences under the Copyright Act 1968 (Cth). Higher penalties may apply, and higher damages may be awarded, for offences and infringements involving the conversion of material into digital or electronic form.

**A Comparison Of Two Bathing Methods: Effects On The Haemodynamic  
And Subjective Responses Of Low Risk Patients Within 48 Hours Post  
Myocardial Infarction.**

**BY**

**Heather M. Kidd B. App. Sc., RN.**

**A Thesis Submitted In Partial Fulfilment Of The Requirements For The Award  
Of**

**Master Of Nursing**

**At The School Of Nursing, Edith Cowan University**

**Date Of Submission: 19/9/94**

## USE OF THESIS

The Use of Thesis statement is not included in this version of the thesis.

**A Comparison Of Two Bathing Methods: Effects On The  
Haemodynamic And Subjective Responses Of Low Risk  
Patients Within 48 Hours Post Myocardial Infarction.**

**Abstract**

The purpose of this within subject experimental study was to determine what effect showering by nurses, as compared to bed bathing patients, has on the haemodynamic and subjective responses of low risk patients within 48 hours of having a myocardial infarction.

The sample consisted of 50 patients, 8 female and 42 male, with a mean age of 61 years ( $SD = 10$ ). Twenty-five were randomly assigned to a bed bath, then a shower over 2 consecutive days and the other 25 to the reverse order. The haemodynamic responses consisted of heart rate, rate pressure product (RPP), blood pressure, cardiac rhythm, ST segment changes, arterial oxygen consumption ( $SaO_2$ ) and chest pain. Each of these responses was measured prior to, during, immediately afterwards and 5 minutes after each bathing method. Ratings of perceived exertion and a short questionnaire were completed after both bathing methods to assess patients' subjective responses.

The only significant difference in the cardiovascular responses to both bathing methods was an increase in the  $SaO_2$  measurement, during the shower ( $p < .05$ ). Twelve patients had abnormal cardiovascular responses to the bed bath and 7 to the shower which were not significant ( $p > .05$ ). Either a fall in systolic blood pressure, or changes in heart rate or rhythm accounted for the abnormal cardiovascular responses. Patients overwhelmingly demonstrated their preference for a shower ( $p < .05$ ).

The findings of this research demonstrated that showering low risk patients in the first 48 hours post myocardial infarction does not increase myocardial oxygen demands. Low risk patients can mobilise sooner and therefore be discharged earlier, thus resulting in savings in health care costs and an enhanced sense of patient well being.

### **Declaration**

I certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any institution of higher education; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.

## **Acknowledgments**

I would like to thank my two supervisors; Patricia Percival and Janet Reinbold, who have provided me with the motivation, support, encouragement and guidance to complete this study. My thanks also, to Amanda Blackmore and Pender Pedler for their statistical input. A special thanks to my husband Adrian for his help and on-going support in everything I do. My thanks also to the Nurses Board of Western Australia, for granting me funding, which enabled the most rigorous controls to be applied to the study. Finally, my thanks to the nursing staff of the coronary care unit at Royal Perth Hospital for their eager participation in the study.



## Table of Contents

Abstract .....	ii
Declaration .....	iv
Acknowledgments .....	v
Table of Contents .....	vi
List of Tables .....	x
List of Figures .....	xi
List of Appendices .....	xii
Chapter 1 .....	1
Introduction .....	1
Background to the Study .....	1
Significance of the Study .....	2
Purpose of the Study .....	3
Hypotheses .....	3
Definition of Terms .....	4
Summary .....	5
Chapter 2 .....	6
Literature Review .....	6
Historical Perspective of Management .....	6
Physiological Basis of Early Ambulation .....	8
Selection of Suitable Patients .....	10
Physiological Responses to Bathing Methods .....	13
Ratings of Perceived Exertion .....	18
Safety and Benefits of Early Ambulation .....	19
Summary and Implications for this Research .....	21

Chapter 3.....	22
Theoretical Framework.....	22
Human Response Framework .....	22
Physiological regulatory response. ....	25
Pathophysiological responses.....	28
Experiential responses.....	30
Behavioural responses.....	30
Environmental factors.....	30
Person factors. ....	31
Summary.....	31
Chapter 4.....	31
Methodology.....	32
Design.....	32
Sample and Setting.....	32
Pilot Study.....	33
Sample size.....	34
Trial of the procedure.....	34
Education of the nursing staff.....	35
Instruments .....	36
Cardiovascular responses.....	36
Chest pain. ....	38
Perceived exertion.....	38
Patient preference.....	39
Time factor. ....	39
Procedure .....	39
Investigator's role. ....	39
Nurses' role. ....	40

Data collectors' role.....	41
Bathing procedure.....	41
Ethical Considerations.....	42
Strengths and Limitations.....	43
Chapter 5.....	44
Results .....	44
Characteristics of the Sample.....	44
Comparison of Cardiovascular Responses to Both Bathing	
Methods.....	45
Abnormal Cardiovascular Responses to Bathing Methods.....	47
Selection of Potential Patients at Risk.....	50
Pre heart rate and systolic blood pressure measurements. ....	50
Location of myocardial infarction.....	53
Chest Pain .....	53
Subjective Responses to Bathing Methods .....	53
Subjects perceived exertion. ....	53
Subjects' preferences. ....	54
Time Factor.....	55
Summary.....	55
Chapter 6.....	57
Discussion.....	57
Characteristics of the Sample.....	57
Comparison of Cardiovascular Responses to Both Bathing	
Methods.....	58
Abnormal Cardiovascular Responses to Bathing Methods.....	59
Subjective Responses .....	65
Time Factor.....	66

Selection of Potential Patients at Risk.....	66
Use of the Human Response Framework.....	66
Summary.....	69
Chapter 7.....	70
Conclusions, Implications and Recommendations.....	70
Conclusions.....	70
Implications.....	71
Recommendations for Further Research.....	72
References.....	73
Appendix A.....	86
Appendix B.....	87
Appendix C.....	88
Appendix D.....	89
Appendix E.....	90
Appendix F.....	91
Appendix G.....	92
Appendix H.....	93

### List of Tables

	<b>Page</b>
Table 1: Cardiac Intravenous and Oral Therapy During the Study.	45
Table 2: Means, <u>SD</u> , <u>t</u> and <u>p</u> Values of the Rate Pressure Product (RPP), Heart Rate (HR), Blood Pressure and SaO <sub>2</sub> Between Both Bathing Methods.	46
Table 3: Number of Patients at Risk and Stages and Occurrence of Abnormal Cardiovascular Responses.	48
Table 4: Number of Patients who had Abnormal Cardiovascular Responses and Day on which these Occurred.	50
Table 5: Location of Myocardial Infarction of All Groups.	53
Table 6: Subjective Responses to Bed Bathing and Showering.	55

## List of Figures

	<b>Page</b>
<b>Figure 1:</b> Theoretical Framework: Human Responses of Low Risk Patients Post Myocardial Infarction from Bed Bathing and Showering by Nurses.	<b>24</b>
<b>Figure 2:</b> The Physiological Responses of Low Risk Patients Post Myocardial Infarction from Bed Bathing and Showering by Nurses.	<b>26</b>
<b>Figure 3:</b> The Pathophysiological Responses of Low Risk Patients Post Myocardial Infarction from Bed Bathing and Showering by Nurses.	<b>29</b>
<b>Figure 4:</b> The Means of the Pre Systolic Blood Pressure (SBP) and Heart Rate (HR) of all the Groups to the Bed Bath and Shower.	<b>51</b>
<b>Figure 5:</b> Theoretical Framework: This Studies Specific Human Responses of Low Risk Patients Post Myocardial Infarction from Bed Bathing and Showering by Nurses.	<b>68</b>

**List of Appendices**

	<b>Page</b>
<b>Appendix A</b>	<b>Borg Scale of Perceived Exertion and Questionnaire.</b>
<b>Appendix B</b>	<b>Demographic Data</b>
<b>Appendix C</b>	<b>Medication Data</b>
<b>Appendix D</b>	<b>Bed Bath Procedure</b>
<b>Appendix E</b>	<b>Shower Procedure</b>
<b>Appendix F</b>	<b>Bathing Method Observation Chart</b>
<b>Appendix G</b>	<b>Consent Form</b>
<b>Appendix H</b>	<b>Patient Information Sheet</b>

## **Chapter 1**

### **Introduction**

The continued evolution of professional nursing rests with developing distinctive knowledge about pertinent phenomena. A phenomenon, which has had little attention in the nursing literature, yet is of considerable importance to coronary care nurses, is the patient's haemodynamic and subjective responses to bed bathing and showering by nurses in the first 48 hours post myocardial infarction.

The opening chapter highlights the overwhelming need for the study. The purpose of the research is explained and conceptual definitions of the terms of the research questions are stated.

### **Background to the Study**

Since Mallory, White, and Salcedo-Salgar's (1939) autopsy study demonstrated that it takes up to 6 weeks for a fibrous scar to form after a myocardial infarction, physicians have been reluctant to encourage early physical activity after a myocardial infarction. However, over the past 5 decades researchers have demonstrated that prolonged bed rest, is in fact, physiologically harmful (Convertino, Hung, & Goldwater, 1982; Dock, 1944; Irvin & Burgess, 1950; Levine, 1951; Williams, Amsterdam, & DeMaria, 1976). Consequently, contemporary treatment involves a gradual return to physical activity.

At a major teaching hospital in Western Australia, the current ambulation regimen post myocardial infarction includes strict bed rest for the first 48 hours. The rationale for this regimen is that during the acute phase every effort should be made to salvage any ischemic myocardium. This practice also enables patients to be closely monitored and provides time for their exercise tolerance to increase. This is achieved by interventions that reduce oxygen demand and



improve oxygen supply such as beta-blockers, a reduction in physical activity and thrombolytics respectively. A reduction in cardiac work, although unsubstantiated, is believed to be partly achieved by bed bathing patients in the acute phase, that is, the first 48 hours. It is not until after this time that patients are permitted to shower.

### **Significance of the Study**

Many nurses have noted that bed bathing appears to be tiring and, in some cases, uncomfortable for patients. For those patients who are considered low risk, showering by a nurse in the first 48 hours may actually be less tiresome and more comfortable. The current study is unique, as unlike previous similar research, it focused on the acute phase of the myocardial infarction. Until this study, little was known about the haemodynamic effects and the energy requirements of bed bathing and showering during this phase. This study has enabled these issues to be addressed and in doing so has added valuable information to the existing literature on the nursing care of patients post myocardial infarction.

Research has demonstrated good exercise capacity in patients 3 and 4 days after an uncomplicated myocardial infarction. This has supported the trend towards early discharge, as soon as 3 days after a myocardial infarction (Burek, Kirscht, & Topol, 1989; Topol et al., 1988). Inevitably, early discharge will lead to changes in nursing practice.

By researching one of the potential changes, that is, bathing methods, as this study did, nurses are able to evaluate their practice based on nursing research. Important nursing implications of the current study include individualising patient care, as patients may now choose their bathing method and providing essential information on the safety of early ambulation in a particular population. Furthermore, as bed bathing was proven to be time

consuming, nurses may have more time to assist patients and their families in rehabilitation and to provide psychological support. In addition patients may be identified as suitable for early discharge. If patients are discharged earlier it could potentially lead to savings in health care costs, better use of hospital beds and an enhanced sense of patient well being.

### **Purpose of the Study**

The purpose of this experimental study was to determine what effect showering, as compared to bed bathing patients, had on the haemodynamic and subjective responses of low risk patients within 48 hours of having a myocardial infarction. The study also enabled a major teaching hospital's current practice of bed bathing patients for 2 days post myocardial infarction to be evaluated. Furthermore, it provided the hospital with essential data to serve as a reference for developing a case management system for their clinical population if they desire to do so.

### **Hypotheses**

1. There will be significant differences in the overall cardiovascular responses when low risk patients who have had a myocardial infarction within 48 hours are bed bathed by nurses as compared to those that are showered.
2. There will be a greater number of abnormal cardiovascular responses when low risk patients who have had a myocardial infarction within 48 hours are bed bathed by nurses as compared to those that are showered.
3. There will be no significant difference in the occurrence of chest pain when low risk patients who have had a myocardial infarction within 48 hours are showered or bed bathed by nurses.
4. Low risk patients who have had a myocardial infarction within 48 hours will rate their perceived exertion, using the Borg (1973) ratings of

perceived exertion scale, as greater when being bed bathed by nurses as compared to being showered.

5. The Borg (1973) ratings of perceived exertion scale will correlate with the mean heart rate for each bathing method.

6. Low risk patients who have had a myocardial infarction within 48 hours will prefer being showered to being bed bathed by nurses.

7. It will take longer to bed bath low risk patients post myocardial infarction than it does to shower these patients.

### **Definition of Terms**

Abnormal cardiovascular responses:	those responses described by Wenger (1992), and consisting of the following: heart rate greater than 120 beats per minute (bpm); heart rate less than 50 bpm; heart rate greater than 15 to 20 bpm above resting levels for patients taking beta blocking medication; systolic blood pressure, a fall of greater than 10 to 15 mmHg; systolic blood pressure of greater than 180 mmHg; diastolic blood pressure of greater than 110 mmHg; chest pain; ST segment depression; arrhythmias.
Arterial oxygen consumption (SaO <sub>2</sub> ):	is the percentage of haemoglobin that is combined with oxygen. The normal range is 94 -100% (Schlant & Sonnenblick, 1994).
Low risk patients:	patients who do not have severe myocardial ischemia at rest nor exercise induced angina or severe left ventricular failure (DeBusk et al., 1986).
Myocardial infarction:	occlusive thrombosis on a coronary artery atheromatous plaque (Roberts, Pratt, Morris, & Alexander, 1994).
Early ambulation:	mobilising out of bed within the first 48 hours post myocardial infarction (Wenger, 1984).
Arrhythmias:	occurrence of ventricular or atrial arrhythmias (Wenger, 1992)
ST changes:	ST segment elevation or depression from the base line of more than 0.1 mV before the procedure (ECG Interpretation, 1990).

Rate pressure product	the product of heart rate multiplied by systolic blood pressure, which is a reliable index of myocardial oxygen demand (Gobel, Nordstrom, Nelson, Jorgensen, & Wang, 1978).
Orthostatic intolerance	assumption of the upright posture associated with hypotension and tachycardia (Fareeduddin & Abelmann, 1969).
Frank Starling's Law	the degree of muscle stretch is a determinant in the strength of muscle contraction (Urban, 1990).

### **Summary**

Nurses have a professional responsibility to evaluate nursing practice through research. The current study of examining the haemodynamic and subjective responses of low risk patients post myocardial infarction will increase knowledge of the range of responses to bed bathing and showering in the first 48 hours post myocardial infarction. This information will enable nurses to identify and implement changes in nursing care, which are consistent with the overall changing spectrum of management of these patients.

## **Chapter 2**

### **Literature Review**

In order to better understand and investigate responses of patients with myocardial infarction to bed bathing and showering by nurses in the first 48 hours post myocardial infarction, the following areas have been included in the review. The literature review begins with an historical perspective to trace the development and rationale for current management of patients post myocardial infarction. The physiological basis for early ambulation is then reviewed to provide information on the benefits of early ambulation and the detrimental effects of prolonged bed rest. Literature that identified a specific population suitable for the study and one which would benefit from the study's results in future practice is next reviewed. This is followed by a critique of the research related to bathing methods post myocardial infarction, so that comparisons of results could be discussed with this study's results. To understand the patient's perception of exertion related to the two bathing methods, a review of the relationship between the rate of perceived exertion and physiological measurements was also undertaken. Finally, the literature review concludes with a review of research supporting early ambulation and early discharge from hospital.

#### **Historical Perspective of Management**

The common practice of the 1900's of putting patients with myocardial infarction on strict bed rest for a minimum of 6 to 8 weeks was reinforced by the Mallory et al. (1939) classic autopsy study which demonstrated that it takes up to 6 weeks for a fibrous scar to form after a myocardial infarction. Physicians such as Lewis (1946) were thus legitimately licensed to impose such restrictions as preventing the patients from performing any voluntary movement

up to 6 weeks in order to avert arrhythmias, asystole or myocardial rupture. These patients would have relied on nursing staff for their every need. One can only postulate the associated fear, anxiety and hopelessness that accompanied such management. Further evidence of the need for strict bed rest was provided by Jetter and White (1944), who reported a higher incidence of myocardial rupture among ambulatory patients post myocardial infarction in a mental hospital compared to those confined to bed in a general hospital.

Fortunately, during this time there were physicians who were less conservative and began to liberalise the activity restrictions of the period. Levine and Lown's (1952) "armchair" treatment revolutionised the trend towards early mobilisation of myocardial infarction patients. Patients were permitted to sit in a chair as early as the first day after a myocardial infarction for 1 to 2 hours. Levine and Lown did not advocate physical activity but believed the sitting position increased venous pooling of the lower extremities and decreased venous return, thereby reducing cardiac work. Other physicians began to be concerned about the risk of thromboembolism, muscular wasting, gastrointestinal and urologic problems (Dock, 1944) and the mental effects (Harrison, 1944) of prolonged bed rest on patients.

By the 1950's, post myocardial infarction management began to change further as physicians recognised the growing evidence of deleterious effects caused by extended periods of bed rest, and recommended earlier ambulation (Irvin & Burgess, 1950). However, statistics for the 1970's in the United States of America (Wenger, Hellerstein, Blackburn, & Castranova, 1982), Britain (Harpur et al., 1971) and Switzerland (Bloch, Maeder, Haissly, Felix, & Blackburn, 1974), showed that bed rest post myocardial infarction varied from 1 day to 4 weeks and the duration of hospitalisation from 2 to 6 or more weeks. Subsequent research has demonstrated that the most serious complications

occur during the initial days and that the majority of patients with uncomplicated myocardial infarction survive with few significant late complications and are suitable for early ambulation (Swan, Blackburn, & DeSanctis, 1976).

There has been a dramatic change in the pathophysiological severity of myocardial infarction over the past decade. This has mainly been related to interrupting an evolving myocardial infarction by thrombolytic agents or percutaneous coronary artery angioplasty, thereby limiting infarct size, improving left ventricular function and enhancing survival (Gruppo Italiano per lo studio della streptochinasi nell' infarto miocardio (GISSI), 1986; Simoons, Serruys, Brand, & De Zwaan, 1985; Topol et al., 1987; Van der Laarse, Vermeer, & Hermens, 1986). Over the past 5 years these practices have led to more rapid mobilisation and early discharge (Burek et al., 1989; Topol et al., 1988). An important part of the nurses' role today is to assess patients' individualised responses to early ambulation and prepare them for discharge.

### **Physiological Basis of Early Ambulation**

The physiological basis for early ambulation post myocardial infarction is related to the need to limit or avert the detrimental effects of the deconditioning response to prolonged bed rest. Researchers have documented numerous adverse effects of prolonged bed rest including reduced pulmonary ventilation, a decrease in skeletal muscle and the development of orthostatic intolerance. Reduced pulmonary ventilation is related to a decrease in lung volume and vital capacity (Williams, et al., 1976), and this is especially of concern in patients with associated pulmonary disease. A decrease by 10 to 15%, within a week, in skeletal muscle mass and muscular contractile strength will render muscular contractions ineffective with more oxygen required for comparable work (Wenger, 1984). This finding highlights the need for active arm and leg exercises in the initial phase post myocardial infarction. A small study by

Ireland and Taylor (1982) on 12 patients within 24 hours of a myocardial infarction demonstrated bed leg and arm exercises did not cause significant cardiovascular changes.

Another detrimental effect of prolonged bed rest, orthostatic intolerance, is characterised by orthostatic hypotension and tachycardia due to both hypovolemia and diminished cardiovascular reflex response (Fareeduddin & Abelmann, 1969). Circulating blood volume will begin to decrease within 24 hours and intravascular volume may decrease by 700 to 800 mls within a week to 10 days of bed rest (Hyatt, Kamenevsky, & Smith, 1969). This circulatory stasis puts patients at further risk of developing thromboembolism related to an increase in blood viscosity because the plasma volume contracts more than the red blood cell mass (Williams et al., 1976). The dangerous effects of orthostatic intolerance can be limited by following the practice established by Levine and Lown (1952) of allowing patients to sit in a chair two or three times daily.

Two major studies have highlighted the effect prolonged bed rest has on the physical work capacity. In Saltin, Blomqvist and Mitchell's (1968) classic bed rest study, 3 sedentary and 2 active male students received bed rest with bathroom privileges, but no standing for 20 days. By the end of the bed rest, the physical work capacity had reduced by 28% and the more active students required an 8 week period of retraining, compared to a 3 week period in the sedentary students, to restore pre bed rest levels of fitness. This 28% reduction in physical work capacity would reflect a comparable decrease in stroke volume and cardiac output which should be considered when assessing patients post myocardial infarction who complain of fatigue.

A more recent study by Convertino et al., (1982) compared cardiovascular responses to upright and supine exercise before and after 10



days of bed rest in healthy middle-aged men, who they believed represented the group most likely to sustain a myocardial infarction. A significantly greater reduction in maximal oxygen uptake occurred with upright exercise (15%) compared with supine exercise (6%) after bed rest. The authors attributed the reduction in physical work capacity to induced reduction in ventricular filling.

Since upright exercise depends more than supine exercise on venous return to the heart and the Frank Starling mechanism for augmenting stroke volume, the subsequent effect would be an increase in the myocardial oxygen demand demonstrated by an increase in the rate pressure product (Hung et al., 1983). Of equal importance, Hung et al. reported that it is not the intensity of physical activity but exposure to gravitational stress that appears to limit hypovolemia, cardiac under filling and deterioration of oxygen transport capacity with effort intolerance.

Although the effects of deconditioning rarely affect low risk patients post myocardial infarction, they commonly complicate the outcome of seriously ill, elderly patients with residual myocardial ischemia and ventricular dysfunction (Wenger, 1992). As improvements in intervention and treatment for myocardial infarction continues, it is likely the average age of a coronary care patient will increase. Early ambulation is designed to limit the detrimental effects of prolonged bed rest and encourage attainment of the activity level required for discharge.

### **Selection of Suitable Patients**

Patients with uncomplicated myocardial infarction who are considered low risk are ideal candidates for early ambulation. Guidelines for the identification of low risk patients post myocardial infarction has received little attention in the medical literature. Much of the literature available on identification of low risk patients post myocardial infarction, stems from the efforts to identify high risk

patients. Madsen, Hougaard, Gilpin and Pedersen (1983) used characteristics of death, cardiac arrest or cardiogenic shock as factors relating to high risk and considered patients to be low risk if none of these events occurred after a myocardial infarction. DeBusk et al. (1986) provided more explicit guidelines by classifying patients into three main risk categories, low, moderate and high, based on the extent of myocardial ischemia and left ventricular dysfunction. More recently, Mark et al. (1991) considered patients to be low risk if they did not experience any of the following for 30 days after myocardial infarction: death, reinfarction, cardiogenic shock, pulmonary oedema, sustained hypotension, sustained ventricular tachycardia, high grade atrioventricular block, acute ventricular septal defect and recurrent ischemia requiring urgent coronary artery bypass grafts.

For the purpose of this study, low risk patients were defined as those who did not have severe myocardial ischemia at rest or activity induced angina and severe left ventricular failure, as described by De Busk et al. (1986). Exclusion criteria were based on the recommendations by Mark et al. (1991) and included sustained ventricular tachycardia, ventricular fibrillation or complete heart block (CHB). Additional exclusion criteria consisted of the presence of invasive haemodynamic and respiratory equipment, or the use of inotropic drugs.

According to Wenger (1992) guidelines for physical activity in the first 48 hours should be primarily dynamic and of low intensity. The activity should be one to two metabolic equivalent units (MET), that is, one to two times the resting metabolic rate. Wenger reported that self-care activities of bathing, feeding, using a bed-side commode and performing leg and arm exercises are within 1-2 MET.

Self bathing has been reported by Johnston, Watt and Fletcher (1981) and Winslow, Lane and Gaffney (1985) to have higher energy costs of 2-4 MET

in patients post myocardial infarction. Mansfield (1971) reported that patients post myocardial infarction who washed themselves had heart rates twice that when washed by a nurse. However, Mansfield did not state if the patients were considered to be high, low or moderate risk from the myocardial infarction. As heart rate is a determinant of myocardial oxygen demand, an increase in heart rate in patients with narrow coronary arteries could be hazardous (Hung et al., 1983). In the present study, it was assumed that as the patients would be bathed by nursing staff the energy costs and therefore myocardial oxygen consumption should be much less. However, these patients were required to take a short walk within their room to the shower. A study by Magder (1985) demonstrated that a short walk within 24-48 hours post myocardial infarction produced few haemodynamic changes to increase myocardial stress and recommended such practice.

The nurse's role is vital in monitoring and assessing the appropriate and abnormal responses to early ambulation. According to Wenger (1992) abnormal responses to low level activity include the following: (a) a heart rate greater than 120 bpm or less than 50 bpm, (b) an increase in heart rate greater than 15 to 20 bpm above the resting heart rate in patients receiving beta blocking drugs, (c) a decrease in systolic blood pressure of more than 10 to 15 mmHg, (d) an increase in systolic blood pressure greater than 180 mmHg, (e) a diastolic blood pressure greater than 110 mmHg, (f) displacement of the ST segment on the electrocardiographic monitor, (g) the occurrence of arrhythmias or (h) chest pain.

The appearance of any of these responses indicates that the cardiovascular system is unable to tolerate such heavy work loads. Under these circumstances the activity should be ceased and the patient's clinical status be evaluated to determine the need for diagnostic and therapeutic

intervention. On the other hand, appropriate responses to physical activity indicates that the patient is able to progress to a greater level of intensity (Wenger, 1992).

### **Physiological Responses to Bathing Methods**

Three published studies which have, to a limited degree, examined the physiological responses to different bathing methods post myocardial infarction were found. The methodology of each study will be described and the results discussed.

Johnston et al. (1981) measured the oxygen consumption, haemodynamic and electrocardiographic responses in 10 patients, between 3 and 5 days post myocardial infarction, to a bed bath, tub bath and a standing shower on 3 consecutive days. Winslow et al. (1985), compared oxygen consumption and cardiovascular responses to bathing activities of 18 patients between 5 and 17 days post myocardial infarction and in 22 control adults. The bathing activities consisted of a basin bath, a tub bath and a standing shower. The third study by Robichaud-Ekstrand (1991) compared heart rate, blood pressure and subjective responses to a sitting shower and a sitting sink bath, in 30 patients between 2 and 9 days post myocardial infarction.

The protocol of the Winslow et al. (1985) study was very similar to the Johnson et al. (1981) study. In both studies, patients were randomised to either a basin bath, tub bath or shower and the patients bathed themselves. During the basin bath in the study by Winslow et al., the patients sat at the edge of the bed compared to remaining in bed in the Johnston et al. study. The pre procedure resting measurements for both studies consisted of determining heart rate by radial pulse palpation and determining blood pressure by a sphygmomanometer. Johnston et al. included cardiac auscultation and a 12 lead electrocardiogram, while Winslow et al. used a holter monitor to record the

heart rhythm. Winslow et al., as part of the pre procedure resting measurements, applied a Max Plank respirator to measure oxygen consumption, whereas Johnston et al. did not commence oxygen consumption reading until the bathing methods had begun and used a Douglas bag to measure oxygen consumption.

In both studies, half way through each bathing method patients would signal the researchers and heart rate was measured. At the end of the procedure, the expired air collection was ceased in both studies. Only Johnston et al. (1981) repeated the pre resting procedure measurements at the end of the study, since Winslow et al. (1985) were only interested in the responses to the different bathing methods rather than the differences in pre and post measurements. The only major differences between these studies appear to be the different choice of oxygen consumption equipment and the time the measurements of oxygen consumption were commenced. Also, in the study by Winslow et al. patients completed the Borg (1973) rating of perceived exertion scale after each bathing method and completed a short questionnaire on their preference for each bathing method.

Although the studies were similar, the results differed markedly. Johnston et al. (1981) found a significantly higher oxygen consumption during shower activity: 3 to 7 METs compared to 2 to 5 METs for the bed bath and tub bath. Winslow et al. (1985) found much lower energy costs: 1 to 7 METs for basin bath and 2 METs for tub bath and shower. These variations may be related to the different techniques for measuring oxygen consumption, but it is more likely that they are the result of Johnston et al. not determining the patient's resting pre procedure oxygen consumption.

Haemodynamic responses to bathing also differed between the two studies. Johnston et al. (1981) found higher rate pressure products in patients

after showering, while Winslow et al. (1985) found higher rate pressure products after tub baths in female patients only. The variability of these results may be related to the difference in the number of days between myocardial infarction and study participation or perhaps a function of individual factors rather than bath type.

There are several scientific flaws in the methodology of both the Johnston et al. (1981) and Winslow et al. (1985) studies. The most important of these in both studies is the absence of defined criteria for normal and abnormal cardiovascular responses for the expected energy costs to different bathing methods post myocardial infarction. Consequently, the results obtained cannot be used as an accurate reflection of the normal cardiovascular responses evoked by different bathing methods post myocardial infarction. Another criticism of both studies, is the wide discrepancy of days between patients post myocardial infarction and commencement of the studies; 3 to 5 days and 5 to 17 days respectively. Up to 7 to 10 days post myocardial infarction the heart undergoes extensive biochemical, electrical, mechanical and vascular changes that occur at different stages (Stewart, 1992). The systemic sequelae of these changes can be an increased sympathetic response with associated increases in heart rate, myocardial contractility in non ischemic areas and systemic vascular resistance (Pipine, 1989). Particularly in the Winslow et al. study, no consideration was made nor were differences in results related to such effects. The results, therefore cannot be generalised to either all patients post myocardial infarction or a specific subgroup of patients post myocardial infarction.

The third study conducted by Robichaud-Ekstrand (1991) used a more sophisticated methodology and partially succeeded to correct the errors in the Johnston et al. (1981) and Winslow et al. (1985) studies. Robichaud-Ekstrand,

as this study did, defined low risk patients using the criteria developed by DeBusk et al. (1986). There are, however, important differences in the exclusion criteria and the criteria to measure abnormal responses between Robichaud-Ekstrand study and this study.

Robichaud-Ekstrand (1991) set a specific creatine kinase (CK) myocardial band (MB) level as greater than 500 units/litre (U/L), (normal range = 25-200 U/L, Roberts, 1988) and a left ventricular ejection fraction (LVEF) of less than 35% as demonstrated by radionuclide studies as exclusion criteria. Research by Roberts (1988), demonstrated that plasma MB-CK activity is elevated within 6 to 10 hours of onset of symptoms of infarction and maximal levels are reached between 14 and 36 hours with return to normal levels after 48 to 72 hours. Bearing this in mind and the fact that the CK of patients who have thrombolytic therapy is often unreliable due to the rapid wash out and altered release of CK (Roberts, 1990), there was no benefit in setting a specific CK level in the present study.

According to the literature, the benefit of a LVEF as a predictor of myocardial dysfunction has been used only to confirm the outcome from 1 to 2 years after hospital discharge (Ahnve et al., 1986; Moss et al., 1983; Mukharji et al., 1984). Considering this and the evidence by Ahnve et al. that the same predictions can be made by using historical and clinical assessment, there was no benefit in using a LVEF in the current study or for that matter in the study by Robichaud-Ekstrand (1991).

Further exclusion criteria used by Robichaud-Ekstrand (1991) also consisted of a history of metabolic or endocrine disorders, thromboembolic complications, musculoskeletal limitations, a resting heart rate greater than 90 bpm, frequent ventricular ectopic beats and obese patients. It is surprising Robichaud-Ekstrand has included risk factors of obesity and endocrine

disorders known to be associated with coronary artery disease (Cunningham, 1992) as exclusion criteria since it would seem likely that these risk factors may have precipitated the myocardial infarction. Exclusion criteria for this study was based on DeBusk et al. (1986) and Mark et al. (1991) criteria for low risk patients. In addition, the presence of invasive haemodynamic monitoring or respiratory assist devices and the use of inotropic drugs were included in the exclusion criteria, as these patients would not be considered low risk.

Robichaud-Ekstrand (1991) choose to follow the recommendations of the American College of Sports Medicine for the abnormal responses to the bathing methods. This study followed the abnormal responses outlined by Wenger (1992) which were specifically for patients post myocardial infarction to ensure consideration had been made for the physiological effects of the myocardial infarction. Like Johnston et al. (1981) and Winslow et al. (1985), Robichaud-Ekstrand assessed patients who were a wide variety of days post myocardial infarction, in this case, 2 to 9 days.

In the Robichaud-Ekstrand (1991) study, pulse, blood pressure and ratings of perceived exertion was recorded 5 minutes prior to the bathing procedure, at the last minute of the bathing procedure and 5 minutes afterwards. Only those patients who required telemetry monitoring had rhythm strips taken during the resting and recovery phase. As expected since the patients were bathing themselves, there were significant differences between the resting and the bathing results despite the bathing measurement being recorded at the last minute of the wash. Ten patients had abnormal responses, though only 2 of the 10 had associated shortness of breath. There were no significant differences between the sitting sink bath and the sitting shower.

The current study applied much more rigid controls. All the patients were in their first 48 hours post myocardial infarction and were washed by nursing



staff following defined methods for both bathing procedures. The haemodynamic data recorded was consistent for all patients and consisted of heart rate, rhythm, ST segment changes, blood pressure, SaO<sub>2</sub>, body temperature and presence of chest pain. Body temperature was only recorded in the resting phase while the remaining variables were recorded in the resting phase, during the procedures at a defined time, immediately after the procedure and in the recovery stage which was 5 minutes after the bathing methods were completed. After the patients had completed both bathing methods they were in a position to compare the two bathing methods. Each patient was then asked to rate their perceived exertion and complete a small questionnaire.

The three studies (Johnston et al., 1981; Robichaud-Ekstrand, 1991; Winslow et al., 1985) and the present research, used a within subject design. Counterbalancing was achieved by randomising the order of the bathing methods. The practical and statistical advantages of this design are that smaller numbers are required and that subjects can act as their own control, which eliminates variability due to individual differences in subjects (Martin, 1985).

### **Ratings of Perceived Exertion**

In order to understand the patients' subjective responses to the two bathing methods, it was important to include a measure of their perceived exertion. The ratings of perceived exertion has historically been accomplished by means of the psychophysical category scale developed by Borg in 1962 (Borg, 1973).

Up until the Winslow et al. (1985) study, no studies had used ratings of perceived exertion in hospitalised patients so the correlations of ratings of perceived exertion had always been based on healthy subjects. Winslow et al.

reported that there was no significant difference in patients perceived exertion among the different bathing methods, compared with the control subjects who felt the basin bath required the most exertion. However, Winslow et al. did not correlate the ratings of perceived exertion with heart rate. Robichaud-Ekstrand (1991) reported low correlations between heart rate and ratings of perceived exertion.

Previous studies (Borg & Linderholm, 1967; Ekblom & Goldberg, 1971) have demonstrated that age, presence of cardiovascular disease, size of muscle mass used and physical training may modify the rating of perceived exertion. According to Morgan (1973), individuals who are depressed or anxious have a tendency to underestimate work intensity especially at moderate loads. However, it seems more likely that although patients in Robichaud-Ekstrand (1991) study bathed themselves they would not have used the same portions of dynamic and isometric exercise that healthy individuals had used when cycling and jogging and on which the best ratings of perceived exertion and heart rate correlations have been made. These results highlight the importance of considering both the energy costs required and the fitness level of the specific sample.

The value of including the patient's ratings of perceived exertion in the present study is to determine the patient's individual perception of the different bathing methods.

### **Safety and Benefits of Early Ambulation**

During the late 1960's and throughout the 1970's the medical literature focused much attention on determining the feasibility of early ambulation and early discharge post myocardial infarction. The first persuasive research by Brumner, Kalio and Tala (1966) demonstrated no significant differences in patients' outcomes when imposed bed rest was reduced from 16 to 10 days

and patients were discharged at 18 days instead of 22 days. Subsequent researchers reported no adverse effects when patients mobilised on day 15 (Grodén, 1971) and reported an associated improvement in psychological status and earlier return to work (Grodén & Brown, 1970). Research in Britain, America and Sweden reported no increases in morbidity or mortality, an earlier return to work and an improvement in psychological factors when patients were mobilised at 8, 12 and 8 days respectively (Harpur et al., 1971; Hutter et al., 1973; Bloch et al., 1974).

In more recent years, researchers using advanced technology have further confirmed the safety of supervised early ambulation for low risk patients post myocardial infarction. Topol et al. (1988) and Burek et al. (1989) demonstrated, using low level exercise thallium studies, that exercise testing can be done as early as day 4 and 3 respectively in patients treated with thrombolytic therapy with no apparent adverse consequences of left ventricular dysfunctioning. Rowe, Jelinek, Liddell and Hugens (1989), showed that rapid mobilisation by day 4 does not affect left ventricular size or ejection fraction in low risk patients post myocardial infarction. Mark et al. (1991), using cardiac catheterisation, identified that patients post myocardial infarction who do not have major complications in the first 1 to 3 days are suitable for early discharge on day 4.

Clearly, there is substantial evidence that low risk patients post myocardial infarction can safely be mobilised and discharged earlier. Low risk patients, post myocardial infarction, will therefore require a more rapid progressive ambulation protocol prior to discharge to reliably ascertain the patients' functional capacity, hence the importance and need for the current study.

### **Summary and Implications for this Research**

Historically, patients post myocardial infarction were confined to prolonged periods of bed rest. In order to limit or avert the deleterious effects of the deconditioning responses caused by such practice, contemporary management includes earlier ambulation. Through research it has been shown that patients who are considered low risk are suitable to be mobilised and discharged sooner.

To date there has been no published research that has specifically investigated bathing methods in the first 48 hours of a myocardial infarction. Research that has studied the physiological effects of different bathing methods on post myocardial infarction patients have reported variable results. The variability of these results may be related to the differences in the number of days between myocardial infarction and study participation, 2 to 17 days, or perhaps a function of individual factors rather than the bathing method.

Two studies were found that investigated the patients subjective responses in association with their haemodynamic responses to different bathing methods. Subjective responses were measured using the Borg ratings of perceived exertion scale.

The present study results will provide a reference on which to base guidelines for the appropriate ambulation of low risk patients post myocardial infarction.

## **Chapter 3**

### **Theoretical Framework**

#### **Human Response Framework**

The framework chosen for this study is the human response framework developed by Heitkemper and Shaver (1989). The American Nurses' Association has defined nursing as "the diagnoses and treatment of human responses to actual or potential health problems" (American Nurses' Association, 1980, p. 9). This definition makes it clear that nursing's domain is the person's response to the health problem not the health problem itself (Mitchell, Gallucci, & Fought, 1991). The Heitkemper and Shaver framework takes an integrated biopsychosocial approach to viewing human responses related to health and is comprised of three main concepts: (a) individual adaptations, (b) environmental factors and (c) person factors (Heitkemper & Shaver, 1989). Clinical nursing therapies can be directed towards any or all of these concepts.

Individual adaptations are viewed from four broad perspectives: physiologic, pathophysiologic, experiential and behavioural. Environmental factors consist of external physical or social factors, which may put patients at risk to negative responses from health problems. Person factors are present within the individual, they are either modifiable or nonmodifiable and can increase a person's vulnerability to negative responses associated with health problems (Heitkemper & Shaver, 1989).

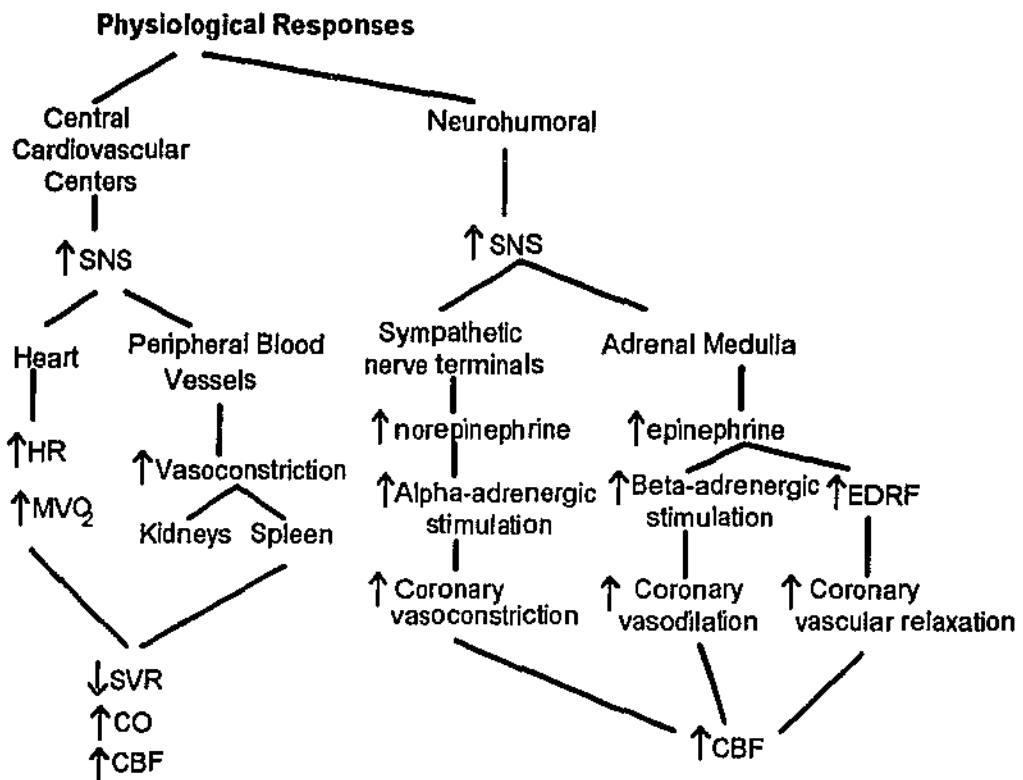
Each of the individual adaptations interact with the environmental and person factors (Heitkemper & Shaver, 1989). By using the human response framework the investigator is able to examine the full range of patient responses, incorporating the interaction of the social, person and the physical

elements associated with the various responses, which is congruent with the holistic caring nature of nursing practice (see Figure 1).

**Physiological regulatory response.**

Physiologic regulatory responses are based on the normal, or usual biological functioning of specific body systems to maintain homeostasis (Heitkemper & Shaver, 1989). Physiologic responses are measured by instruments of the biological sciences for example, stimulation of the sympathetic nervous system in response to a low heart rate.

The acute cardiovascular physiological responses in patients, post myocardial infarction, who are showered or bed bathed by nurses, involve complex interactions between the heart, peripheral vessels, neurohumoral influences and local stimuli (see Figure 2). At the onset of low level activity, for example, getting the patient out of bed to walk to the shower, neuroinputs are received from the metabolic and mechano receptors in the exercising muscle and the central command motor cortex of the brain (Balady & Weiner, 1992). Areas in the cardiovascular centre receive impulses from the spinal cord which are activated by ergoreceptors caused by changes in the mechanical and metabolic condition of the exercising muscle (Mitchell, 1985).



**Figure 2.** The Physiological Responses of Low Risk Patients Post Myocardial Infarction from Bed Bathing and Showering by Nurses (SNS, sympathetic nervous system; HR, heart rate; MVO<sub>2</sub>, myocardial oxygen demand; SVR, systemic vascular resistance; CO, cardiac output; CBF, coronary blood flow; EDRF, endothelium derived relaxing factor).

The cardiovascular centre responds to these impulses by the immediate withdrawal of parasympathetic outflow and increases the sympathetic outflow to the heart, peripheral blood vessels and adrenal medulla (Fletcher & Schlant, 1994). As a result, a series of effects occur. The heart rate increases in association with a linear increase in total body oxygen uptake, thus increasing myocardial oxygen demand (Miers & Arnold, 1990). The ability of the myocardium to respond to increased demands for oxygen is dependent on the functional status of the coronary arteries and the complex interaction of



neuromodulation and endothelial control to augment coronary flow (Balady & Weiner, 1992).

The functional status of the coronary arteries depends on factors such as the diameter of the coronary lumen, coronary perfusion pressure and resistance in the distal coronary vessel to contribute to the coronary flow (Balady & Weiner, 1992). Neuromodulation involves stimulation of the neurotransmitter norepinephrine released from sympathetic nerve terminals and the release of the hormone epinephrine from the adrenal medulla. These catecholamines stimulate alpha-adrenergic and beta-adrenergic receptors resulting in coronary vasoconstriction and coronary vasodilatation respectively (Balady & Weiner, 1992). It has only been in the past 6 years that research has demonstrated that vasomotor tone is also regulated by the release of an endothelium derived relaxing factor (Griffith, Lewis, Newby, & Henderson, 1988; Vanhoutte, 1988). According to Fletcher and Schlant (1994) during low levels of activity the heart rate rises and then levels off within a few minutes when oxygen requirements are met.

The myocardial demand for oxygen is affected by changes in blood pressure, left ventricular contractility and left ventricular wall stress (Clausen, 1976). Clinically the rate pressure product, that is, the product of heart rate multiplied by systolic blood pressure is a reliable index of indirect measurement of myocardial oxygen demand (Gobel et al., 1978).

The increased sympathetic stimulation causing vasoconstriction to the kidney's renal and splanchnic arterioles and vasodilatation in the active tissue results in a reduction in systemic vascular resistance and therefore a reduction in afterload (Miers & Arnold, 1990). The overall effect is an increase in cardiac output and consequently an increase in coronary blood flow and systolic blood

pressure. The diastolic pressure usually remains unchanged or may decrease (Fletcher & Schlant, 1994).

### **Pathophysiological responses.**

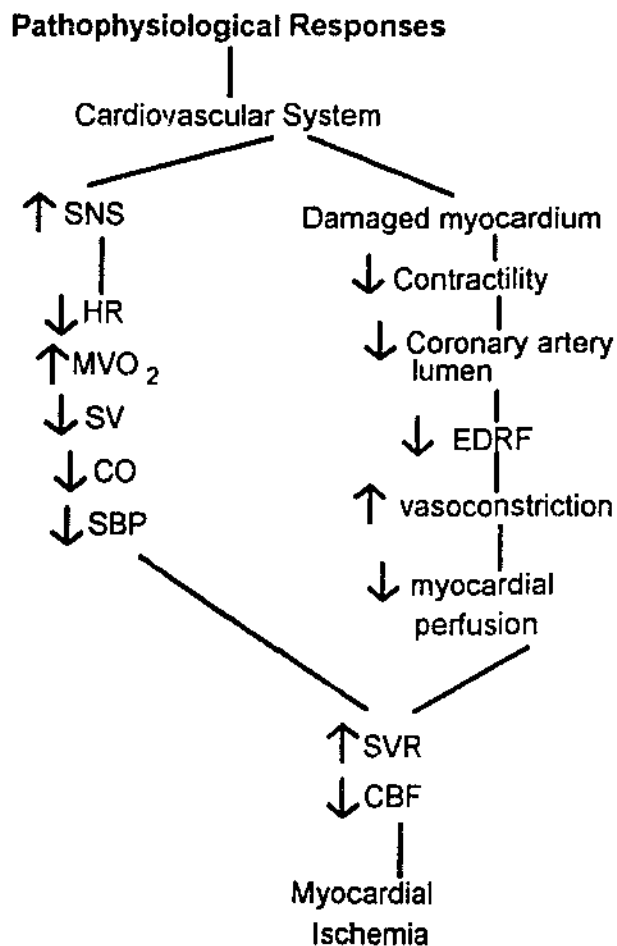
Pathophysiological responses result from an alteration in the normal biological functioning where demands exceed the reserve of compensatory mechanisms and symptoms of distress occur ( Heitkemper & Shaver, 1989; Mitchell et al., 1991). Indicators that haemostasis is no longer being maintained can be observed by instruments of the biological sciences for example, a reduction in cardiac output.

The acute effects of low level activity, for example, being showered, or bed bathed by nurses, on a patient post myocardial infarction will depend on the underlying extent of damaged myocardium. It is important, therefore, to also recognise the pathophysiologic responses.

In patients post myocardial infarction the heart rate increase response to activation of sympathetic stimulation may become blunted. This effect is partly related to an increased diastolic stiffness caused by myocardial dysfunction (Herzil, Leutwyler, & Kravenbuhl, 1985) and partly related to vasoconstriction caused by the inability of damaged endothelial to release the endothelial derived relaxing factor (Marcus, 1988). Despite the lower heart rate, more myocardial oxygen is consumed as the increase in left ventricular end-diastolic pressure requires a greater force in contractility (Braun & Holm, 1989). However, if there is a large portion of ischemia in the left ventricle the myocardium is unable to augment contractility, therefore stroke volume will not increase and cardiac output will be reduced (Balady & Weiner, 1992). As a consequence, systolic blood pressure is likely to fall or remain unchanged, indicating severe coronary artery disease and ischemic dysfunction of the

myocardium with an increased risk for ventricular fibrillation (Fletcher & Schlant, 1994).

Blood pressure, which is a function of cardiac output and peripheral resistance will be maintained in these circumstances by elevated systemic resistance as a result of failure of the resistance vessels to dilate (Francis, 1987). Consequently the patient may not have enough coronary blood flow to supply the metabolic demands of the myocardium as there is an imbalance in the demand and supply for oxygenated blood to the myocardium and myocardial ischemia occurs (see Figure 3).



**Figure 3.** The Pathophysiological Responses of Low Risk Patients Post Myocardial Infarction from Bed Bathing and Showering by Nurses (SNS, sympathetic nervous system; HR, heart rate; MVO<sub>2</sub>, myocardial oxygen

demand; SV, stroke volume; CO, cardiac output; SBP, systolic blood pressure; SVR, systemic vascular resistance; CBF, coronary blood flow; EDRF, endothelium derived relaxing factor).

### **Experiential responses.**

Experiential responses consist of the patients' personal experience to both physiological and pathophysiological responses (Mitchell et al., 1991). The dimensions of a patients experience can only be derived through the patients' verbal report of symptoms, emotions and sensations (Heitkemper & Shaver, 1989). The patients experience to both bathing methods will influence his choice for the preferred type of bathing method.

A patient with myocardial ischemia may verbalise increasing shortness of breath, sweating, chest pain, nausea and feeling frightened (see Figure 1). However, the extent of which the patient will verbalise the experience with nursing staff will depend on factors of past experience, knowledge, culture and the social meaning of the experience (Mitchell et al., 1991).

### **Behavioural responses.**

Behavioural responses are those facial, motor and verbal responses that are observable and indicate the impact a phenomena has on the individual (Heitkemper & Shaver, 1989). Behavioural responses may be divided into physical responses and social responses. A physical response to chest pain would involve the nurse directly observing the patient clutching his chest, looking pale, frightened, sweating and breathing rapidly (see Figure 1). Social responses would include the patient's ability to adapt to imposed restrictions of self caring activities.

### **Environmental factors.**

Environmental factors are risk factors which can be related to a persons health status and are determined by external physical or social influences, for example, personal behaviour, lifestyle and social support. The physical

environmental factors involved in this research include being bed bathed and showered by nurses, use of medications and monitoring equipment (see Figure 1). These factors may result in a greater demand being put on the physiological and behavioural response mechanisms.

### **Person factors.**

Person factors consist of vulnerability factors which are present within the individual and are related to the persons health status (Heitkemper & Shaver, 1989). Vulnerability factors can be nonmodifiable or modifiable.

In this research the normal physiological response to bed bathing and showering patients by nurses may be influenced by the person's nonmodifiable factors, for example, a previous myocardial infarction in the same area of the left ventricle, age, gender, muscular skeletal difficulties, or by modifiable factors, for example, the degree of chest pain and LVF associated with the myocardial infarction, exercise tolerance, weight and anxiety (see Figure 1).

When the person and environmental factors are combined with the four perspectives of human responses, the effect the body, mind and environment has on particular responses to health problems can be examined. Each of these factors can influence the responses of the previous four perspectives in either a negative or positive way (Heitkemper & Shaver, 1989).

### **Summary**

The Heitkemper and Shaver (1989) Human Response framework is ideally suited to drive this study as it enables the researcher to investigate the full range of human responses to the two different bathing methods. Of the three concepts of Heitkemper and Shaver's framework, the clinical nursing therapy of bed bathing and showering patients was mainly directed towards manipulating the environmental factor. To determine the impact of this intervention the investigator assessed all four dimensions of Heitkemper and Shaver's framework.

## **Chapter 4**

### **Methodology**

The purpose of this chapter is to describe the methodology, used in this study. Information is provided on the design, pilot study, procedure, instruments used, ethical considerations and identification of the strengths and limitations.

#### **Design**

In order to eliminate variability due to individual differences in subjects and reduce the study sample size a within subject experimental design with randomised counter balancing of the order of the bathing methods was used. The independent variables were (a) a fully assisted bathing method and (b) the 2 consecutive days during which the bathing procedures were performed. Dependent variables included: (a) heart rate, (b) blood pressure, (c) rate pressure product, (d) arrhythmias, (e) ST changes, (f) chest pain, (g) SaO<sub>2</sub>, (h) body temperature, (i) subjective ratings of exertion, (j) bathing preference and (k) duration of each bathing method.

Fifty percent of patients were randomly assigned to a bed bath, then a shower, over 2 consecutive days. The other 50% of patients were randomly assigned to the reverse order, a shower then a bed bath. Randomisation occurred by picking a label indicating the bathing method order out of a carton and not replacing it. The label was then assigned to a patient.

#### **Sample and Setting**

There were 54 patients who enrolled in the study. However 2 of these patients died after completing one of each type of the bathing methods. Neither of the deaths were related to the study treatment. One patient had a cerebral bleed and the other a cardiac arrest. Another patient withdrew prior to

starting the study and another was withdrawn as the patient was transferred out of the coronary care unit prior to the study being completed. The final sample size was  $N = 50$ .

The study was conducted in a 12 bed coronary care unit at a major metropolitan teaching hospital in Western Australia. No changes to the setting were required and the design did not disrupt care of other patients. The sample comprised all patients who were day 1 post myocardial infarction and who met the following inclusion criteria:

- Absence of chest pain at rest.
- Absence of moderate to severe left ventricular failure diagnosed by the medical staff.
- Absence of chest pain induced by exertion, for example by moving around the bed.
- No sustained ventricular tachycardia, ventricular fibrillation or complete heart block since admission.
- No invasive lines in situ, (i.e., a pulmonary artery catheter, arterial line, or an intra aortic balloon pump). All patients had peripheral intravenous cannulas in situ.
- Absence of respiratory assist devices, that is, continuous positive airway pressure.
- Absence of inotropic drugs intravenously.
- Patients who willingly consented to be in the study.

The inclusion criteria was derived from research by DeBusk et al. (1986), Mark et al. (1991) and additional researcher determined criteria.

### **Pilot Study**

One month prior to commencing the main study a pilot study, consisting of 5 patients, was conducted for three reasons. First, the pilot study assisted in

determining a sample size that would provide significant statistical results. The second reason for the pilot study was to trial the procedure and thirdly to familiarise the nursing staff to the study's design and procedure.

### **Sample size.**

In determining the sample size from the pilot study results, the investigator required an estimate of the proportion of patients at risk from both bathing methods. Of the 5 patients in the pilot study, 4 patients were at risk from the bed bath and 1 from the shower. Accordingly, the risk level to the bed bath ( $p_1$ ) was assigned the value 0.4 and the risk level to the shower ( $p_2$ ), was assigned the value of 0.1. For the researcher to be 95% confident of obtaining a significant difference at the 5% alpha level, the values  $1-\beta = 0.95$  and  $\alpha = 0.05$  were assigned. According to the procedure described by Devore (1991) a sample size of  $N = 43$  was necessary. In order to account for withdrawals or unforeseen events, the sample size was set at 54 patients.

### **Trial of the procedure.**

During the pilot study the investigator worked with the nursing staff to identify and solve problems with the proposed methodology. The major problems discovered were issues related to staffing and paper work.

Staffing problems were associated with the investigator, who was responsible for data collection, and the nurses, who were responsible for washing the patient, in arranging a mutually suitable time, within the constraints of their work to participate in the study. Further difficulties arose when on the same shift there were 2 patients enrolled in the study and exacerbated if the same nurse was responsible for washing both patients, as it required the nurse and the investigator to further reorganise their duties.

When the investigator conducted the pilot study on rostered days off it was noted that there was considerably less reorganising of the staff and their



work load. However, this arrangement was not considered a good alternative as it would have limited the study to enrolling only those patients who happened to be admitted on the investigator's days off. To solve these problems and to enhance the rigorous controls of the study, the investigator obtained funding from the Nurses Board Of Western Australia to employ 2 research assistants to be solely responsible for collecting the haemodynamic and subjective data.

The advantages of having one nurse (N1) to wash the patient, and a second nurse (N2) to record and document the haemodynamic and subjective response, was that it avoided both interrupting the wash and introducing subject bias. Also if the nurse who washed the patient administered the questionnaire the patient may have felt uncomfortable in answering honestly in case the answers could be perceived as a reflection of the nurse's practice.

Paper work problems were related to the design of the data collection charts. From using the charts it was clear changes were required in order to include relevant information, for example, a section for intravenous and oral medication. Other changes included, putting the chart used for recording the haemodynamic responses from the bathing methods on each side of a page and labelling them according to the first and second bathing method.

Interestingly, the pilot study also provided an opportunity for the staff to get used to and feel comfortable with being observed while washing the patients.

#### **Education of the nursing staff.**

Two weeks prior to commencing the pilot study, the investigator conducted five in-service education sessions to 5 different groups of nursing staff to ensure they understood the research protocol and standards. After the pilot study was completed and before the research began, a further series of

five education sessions were repeated to the nursing staff and the research assistants, addressing problems and issues raised during the pilot study. As a result, all nursing staff were very familiar with the research methodology prior to the main research project commencing.

### **Instruments**

The instruments used to measure the hypothesis related to the haemodynamic responses consisted of indirect measurements of cardiovascular parameters and questioning and observing the patients responses to the bathing methods. The hypothesis related to the patients subjective responses were measured using a psychophysical scale and asking patients to complete a short questionnaire.

#### **Cardiovascular responses.**

The cardiovascular responses consisted of measuring blood pressure, heart rate, occurrence of arrhythmias, ST segment changes, SaO<sub>2</sub>, rate pressure product, water temperature and body temperature.

Blood pressure was measured using the Korotkoff method of auscultation (Gorny, 1993). The same portable sphygmomanometer (Braun stand by manometer 0250: Perth Surgical, Western Australia) was used on the left arm of each patient with a stethoscope (Littman 110: Protea Surgical, Perth, Western Australia), to account for potential changes caused by hydrostatic differences and alterations in arterial wall elasticity. Further reliability and validity measures included having the same people to record the blood pressure. The research assistants were instructed to assess for equipment errors related to the cuff size, position, leaks and their technique was regularly checked by the investigator to ensure accurate measurements.

The sphygmomanometer has been reported to have a good correlation between direct and indirect measurements of blood pressure (Karlefors, Nilsen,

& Westling, 1966). Arterial blood pressure obtained by the sphygmomanometer can be expected to be slightly below systolic and slightly above diastolic values obtained from invasive measurement techniques (Andreoli, Zikes, Wallace, Fowkes, & Kinney, 1987).

The diaphragm portion of the stethoscope was used to listen for the onset and disappearance of Korotkoff sounds. Use of the diaphragm portion of the stethoscope has been documented as correlating well with direct blood pressure measurements (Byra-Cook, Dracup, & Kazik, 1989).

A Merlin (Hewlett Packard physiologic monitor 78352C: Andover: Massachusetts, USA) telemetry unit or hard wire unit was used to record patient's heart rate, occurrence of arrhythmias and ST changes. Arterial oxygen saturation was recorded by applying either the Merlin monitor or Nellcor's pulse oximeter (N 200 pulse oximeter: Nellcor Incorporated: Hayward, California, USA) finger probe externally. The Merlin monitor, Nellcor's oximeter and the sphygmomanometer were checked for potential malfunctioning by hospital medical technicians prior to the study commencing and each week throughout the study.

Rate pressure product was calculated by multiplying the heart rate with the systolic blood pressure. Research by Gobel et al. (1978), demonstrated that during exercise in patients with angina pectoris the rate pressure product correlated well with myocardial oxygen consumption ( $r = 0.83$ ).

A mercury-in-glass lotion thermometer (Zeal 1L110: Bennet Medical: West Perth, Western Australia) was used to check that the temperature of the bath/shower water was between 35.6°C and 36.7°C. Temperature greater than this is associated with vasodilatation and thus an increase in heart rate (Keatinge & Evans, 1961).

A mercury-in-glass Dew thermometer (Murray Importers & Exporters, Kallaroo, Western Australia) was used to test the patient's body temperature orally. To ensure accurate recording of body temperature, the thermometer was left in the mouth for 3 minutes (Holtzclaw, 1993). If the patient had taken any hot or cold fluids, the research assistants were instructed to wait for 9 minutes before recording the body temperature, to ensure reliable readings (Terndrup, Allegra, & Keally, 1987).

#### **Chest pain.**

Patients were asked at each of the four stages of the bathing procedures, if they had chest pain. At these times their behavioural responses were observed and their verbal response was documented by the data collector.

#### **Perceived exertion.**

After each bathing method, patients rated their perceived exertion using the Borg (1973) ratings of perceived exertion scale (see Appendix A). The Borg ratings of perceived exertion scale was adjusted by Borg from 21 to 15 points in order to increase the linearity of the relationship between rating of perceived exertion value and heart rate (Borg, 1973). Subjects were asked to assign a numeral to represent the subjective sensation of the amount of work being performed. Several studies of homogenous normal healthy subjects have demonstrated correlation coefficients from .75 to .90 between perceived exertion using the Borg rate of perceived exertion scale while cycling and jogging (Borg & Linderholm, 1967; Frankenhaeuser, Post, Nordheden & Sjoeborg, 1969; Stevens, 1971). However, correlations measured in age heterogeneous groups while cycling and jogging were markedly lower, varying from .40 to .70 (Bar-Or, Skinner, Buskirk, & Borg, 1972).

**Patient preference.**

A short questionnaire, developed by the researcher (see Appendix A), was used to assess bathing preference, cleanliness and ranking of ease.

**Time factor.**

A stop watch (deluxe quality stop watch 06231: Dick Smith, Perth, Western Australia) was used to time each bed bath and shower.

**Procedure****Investigator's role.**

A major role of the investigator was being responsible for explaining the study to potential subjects and enrolling them in the study. The investigator then informed the nursing staff and the research assistants of the names of the patients enrolled in the study.

The investigator was responsible for ensuring the study was organised in such a way that it could be conducted with minimal inconvenience to the nursing staff. One of the ways this was achieved was by placing a box containing two files and a carton with labels for randomisation in the most convenient and accessible area for the nursing staff.

One file contained 54 folders, each containing the criteria for selection, the research procedure, bed bath procedure, shower procedure, the data collection charts, the Borg (1973) rate of perceived exertion scale and the questionnaire. All of this information was colour coded. Green charts and labels indicated that the patient was randomised to a shower on the first day and a bed bath the second day. Yellow charts and labels indicated the reverse order of randomisation. In addition, on the inside cover of this file, the research protocol was clearly documented. The file also contained patient consent forms and information sheets.

When a label was picked out of the randomisation container, it was placed in the hygiene section of the standard nursing flow chart. Inside each folder there was also adhesive labels indicating the patient participation in the study and these large colour coded labels were put on the front of the patient's nursing chart.

The second file was for the completed data collection forms. It also contained the master sheet for the patients addressograph and coded numbers, so that the patient's identity did not appear on the data collection forms. At no time was the master sheet with the patient's notes.

In order to impose the most rigorous controls as possible and not introduce a bias into the data collection, the investigator was not responsible for recording the responses to the bathing methods. However, to ensure standardisation of treatment was maintained throughout the study, the investigator did random interrater reliability checks on 20 patients and instructed the research assistants to ensure the nurses adhered to the defined instructions during the bathing methods.

#### **Nurses' role.**

In the absence of the investigator, the nurses were responsible for randomising patients into the study. Their main role was performing the bathing method on the patient they had been assigned to care for. This involved being aware of which patients were enrolled in the study, organising their work and liaising with the research assistant and, on occasions, also the investigator, to arrange a suitable time to conduct the study. The role also included adhering to the defined procedure throughout the bathing methods (see Appendices D and E).

### **Data collectors' role.**

The two research assistants' responsibilities consisted of recording the haemodynamic and subjective responses of the patients to the bathing methods. This involved communicating with the investigator and the nursing staff to determine which patients were enrolled in the study and to organise a suitable time to conduct the study. In the absence of the investigator, the research assistants recorded the patients' demographic and medication data (see Appendices B and C) and also were responsible for ensuring the correct bathing procedure was maintained.

### **Bathing procedure.**

The procedure was divided into four parts. The first part, before testing, involved N1 collecting the material needed for the bathing method (see Appendices D and E). As soon as N1 began to collect the required equipment, N2 started the stop watch and stopped the watch at the end of the bathing procedure. Five minutes prior to the bathing procedure, the following dependent variables were recorded on the data collection chart: (a) heart rate, (b) arrhythmias, (c) ST changes, (d) blood pressure, (e) SaO<sub>2</sub>, (f) presence of chest pain and (g) body temperature (see Appendix F). This was the only time the body temperature was recorded.

In the event a patient experienced chest pain, the nursing staff were instructed to delay the wash until the patient stated he/she was pain free and wished to continue with the wash. During the wash, conversation was limited to events which led to the patient being admitted. Patients who were randomised to showering were assisted out of bed and walked into the bathroom and sat down on the shower chair. All rooms in the coronary care unit have adjoining bathrooms. Water temperature was set between 35.6°C and 36.7°C for both bathing methods.

The second part, the bathing method, was based on guidelines for sponging a patient in bed and showering a patient developed by the Western Australian School of Nursing (1983) (see Appendices D and E). Standardising the bathing method ensured reliability of treatment implementation. During each bathing method, when N1 had washed the patient's buttocks, N2 recorded the dependent variables.

The last two parts consisted of N2 recording the dependent variables immediately after the bathing method and 5 minutes after the bed bath or shower. When the patient had completed both bathing methods, N2 then recorded the Borg (1973) ratings of perceived exertion scale and patient preference (see Appendix A).

### **Ethical Considerations**

Prior to conducting this study, the investigator submitted the research proposal to the university and hospital ethics committees. After rigorous examination, the investigator obtained permission from both these committees to implement the research.

Participants' human rights were protected by the following methods. Only participants who were mentally alert were informed about the study. Participants freely chose whether or not to enter the study and were free to withdraw from the study at any time. The participants voluntarily signed an informed consent that provided a written explanation of the study (see Appendix G), after being given a verbal explanation of the study and reading the patient information sheet (see Appendix H).

Patients rights to protection from discomfort and harm were assured as nurses were informed to continue the current practice of discontinuing the bathing if a patient experienced chest pain and only to resume bathing when the patient was pain free and if the patient so desired.



The investigator maintained confidentiality and anonymity of the participants by ensuring records were coded, therefore, they did not have the participants' name on them. All records, including the master copy of the coded data, will be concealed and kept for 5 years in a safe place under lock and then destroyed.

### **Strengths and Limitations**

Considerable attention was given to the methodology of the study, so that the scope of the study would not be hindered by potential limitations, or poor choice of design.

Many of the possible limitations, for example, carry over effect, standardisation between treatments and researcher bias were dealt with in the design. A major limitation of having a variety of nurses to record the haemodynamic data was avoided, as the investigator obtained funding from the Nurses Board of Western Australia, to employ two nurse research assistants experienced in coronary care nursing to record the haemodynamic data. As the research assistants worked on opposite days, a possible limitation of the study could have occurred if 2 patients required to be washed at exactly the same time, as it would have been difficult to ensure the observations were recorded at the set times. To reduce the potential effects of these limitations, regular interrater reliability checks were carried out by the investigator and nursing staff liaised with the research assistant to ensure 2 patients were not being bathed at exactly the same time.

Although the sample size of this study was determined from the pilot study's results, a larger sample size may have provided information to identify potential patients at risk to either of the bathing methods.

## **Chapter 5**

### **Results**

This chapter presents the study results. Characteristics of the sample, a comparison of the cardiovascular responses to both bathing methods and the abnormal cardiovascular responses to each bathing method are presented. The patient's subjective responses and the identification of potential patients at risk are also described. The chapter concludes with a summary of the study's findings.

#### **Characteristics of the Sample**

The sample consisted of 50 patients, 8 of whom were female and 42 of whom were male, with a mean age of 61 years ( $SD = 10$ ). Twenty-four patients (48%) had an inferior myocardial infarction, while the remaining patients consisted of the following: 14 (28%) anterior myocardial infarction; 9 (18%) multiple location; 2 (4%) lateral myocardial infarction; and in 1 (2%) the site was undefined. Thrombolytic therapy was given to 37 (74%) of the patients who fitted the hospital's criteria for receiving thrombolytic therapy. The mean creatine kinase (CK) was 1038 ( $SD = 844$ ). (Normal range of CK = 25-200 U/L Roberts, 1988). Table 1 describes the cardiac intravenous and oral therapy patients were on while having a bed bath or shower. Table 1 shows the number of patients receiving various types of medication on the day that they were given the bed bath and on the day that they were given the shower. It indicates that each patient was not always on the same medication during each bathing method.

**Table 1****Cardiac Intravenous and Oral Therapy During the Study**

<b>Medications</b>	<b>Number of Patients</b>	
	<b>Bed Bath</b>	<b>Shower</b>
<b>Intravenous</b>		
Glyceryl trinitrate	25	22
Heparin	16	16
Magnesium	4	8
Lignocaine	0	1
<b>Oral &amp; Subcutaneous</b>		
Platelet Inhibitors (Aspirin)	23	22
Platelet Inhibitors (Aspirin) and Subcutaneous (Heparin)	26	27
Beta Blockers	35	35
Calcium Channel Antagonists	9	9
Antiarrhythmic Agents	0	0
Nitrates	7	8
Angiotensin Converting Enzyme Inhibitors	9	6

**Comparison of Cardiovascular Responses to Both Bathing Methods**

The first hypothesis stated that there would be significant differences in the overall cardiovascular responses when patients were bed bathed by nurses, as compared to being showered. To test this hypothesis, paired  $t$  tests comparing the rate pressure product, heart rate, blood pressure and  $\text{SaO}_2$  of the patients taken before, during, immediately after and 5 minutes after each bathing method were done. Table 2 shows there were no significant differences in the means of rate pressure product, heart rate or blood pressure, throughout the four recordings,  $p > .05$ . There was a significant difference in  $\text{SaO}_2$  during the shower as compared to during the bed bath,  $p < .05$ .

**Table 2**

Means SD t and p Values of the Rate Pressure Product (RPP), Heart Rate (HR), Blood Pressure and SaO<sub>2</sub> Between Both Bathing Methods

Stage of Recording	Bed Bath		Shower		t (49)	p > .05
	Mean	SD	Mean	SD		
<b>RPP</b>						
Pre	8155	1754	8190	2534	.09	.926
During	8369	2305	8641	2668	.79	.435
Immediate	8375	2253	8356	2790	.06	.955
Post	8199	1940	8105	2886	.25	.802
<b>HR</b>						
Pre	68	9	68	13	.15	.885
During	68	11	70	14	1.44	.156
Immediate	67	10	69	14	1.09	.282
Post	67	10	66	14	.54	.593
<b>SBP</b>						
Pre	118	17	117	19	.02	.981
During	121	20	120	19	.16	.870
Immediate	122	19	118	20	1.18	.245
Post	120	17	119	21	.35	.726
<b>DBP</b>						
Pre	71	14	72	13	.64	.527
During	71	14	73	11	1.20	.236
Immediate	72	13	72	11	.28	.782
Post	74	12	72	13	1.08	.284
<b>SaO<sub>2</sub></b>						
Pre	96	2	96	2	.21	.835
During	95	2	97	2	3.06	.004
Immediate	96	2	96	2	1.86	.068
Post	96	2	96	2	.67	.508

The patient's body temperature was also examined and found not to be significantly different between the bed bath (M = 36, SD = .5) and the shower (M = 36, SD = .6)  $t (49) = 1.15, p > .05$ .

### **Abnormal Cardiovascular Responses to Bathing Methods**

The second hypothesis stated that there would be a greater number of abnormal cardiovascular responses when patients were bed bathed by nurses as compared to being showered. To test this hypothesis, the nonparametric McNemar Change Test was used to test for significant differences in patients at risk from either bathing methods. In assessing the subjects cardiovascular responses to the two bathing methods, their physiological data were compared with the criteria set for abnormal responses to low level activity (as given on page 4 under definition of terms). Any patient who met the criteria on any variable, during any of the four recordings was classified "at risk".

There were 12 (24%) patients at risk during the bed bath and 7 (14%) patients at risk during the shower. As the number of patients at risk was small the McNemar Change Test, recommended in these circumstances by Siegal and Castellan (1988), was used to test for significant differences in patients at risk from either bathing method. There were no significant differences in abnormal cardiovascular responses to either bathing method, ( $p > .05$ ).

Table 3 describes the number of patients at risk and the stages and occurrence of the physiological data responsible for the abnormal cardiovascular responses.

**Table 3**

**Number of Patients at Risk and Stages and Occurrence of Abnormal Cardiovascular Responses.**

<b>Abnormal Cardiovascular Responses</b>	<b>Number of Patients At Risk</b>	
	<b>Bed Bath</b>	<b>Shower</b>
<b>Systolic Blood Pressure (SBP) &gt; 180 mmHg</b>		
At any stage	0	0
<b>SBP decreased &gt; 10 - 15 mmHg</b>		
<b>Stage SBP dropped</b>		
From pre through to post	2	1
From pre to during only	5	1
From pre at immediate only	1	2
From pre at immediate & post only	1	1
From pre at post only	0	2
From during to post	1	0
From immediate to post	1	0
<b>Diastolic Blood Pressure (DBP) &gt; 110 mmHg</b>		
At any stage	0	0
<b>Heart Rate &lt; 50 BPM</b>		
From pre at post only	1	0
<b>Heart Rate &gt; 120 BPM</b>		
At any stage	0	0
<b>Heart Rate &gt; 15 - 20 BPM &gt; Resting on Beta Blockers</b>		
From pre at immediate only	2 <sup>a</sup>	0
<b>ST Segment Changes</b>		
At any stage	0	0
<b>Angina</b>		
At any stage	0	0
<b>Arrhythmias</b>		
From during to post	1 <sup>b</sup>	0
<b>Total</b>	<b>12</b>	<b>7</b>

<sup>a</sup>These 2 patients also had a drop in SBP, therefore these responses are included with the SBP and are not represented separately in the total responses.

<sup>b</sup> This patient also had a drop in heart rate, therefore the abnormal response has already been accounted for and should not be included in the overall total.

In all of the patients, with the exception of 3, the abnormal responses were solely caused by a drop in SBP by a mean of 14 mmHg ( $SD = 13$ ) during the bed bath and by a mean of 9 mmHg ( $SD = 12$ ) during the shower. In the other 3 patients, 2 had an increase in heart rate greater than 19 bpm, while on beta blockers and in association with a drop in SBP. The third patient had a drop in heart rate less than 50 bpm and developed transient CHB, though reverted spontaneously to sinus rhythm (SR) with first degree atrioventricular heart block ( $1^\circ$ AVB) prior to the end of the bed bath and without being compromised.

Other arrhythmias were also observed, but were not included as abnormal responses to the bathing methods since they were not caused by the study treatment. One patient had atrial fibrillation (AF) and another had sinus tachycardia (ST) with  $1^\circ$ AVB prior to and throughout both study treatments. Another patient had ventricular ectopic beats prior to the bed bath and throughout this particular treatment. None of these patients, or any patient during the bathing methods, developed ST segment changes, or experienced symptoms of chest pain.

Table 4 describes the number of patients and on which day the abnormal cardiovascular responses from the two bathing methods occurred. No obvious patterns can be seen from Table 4. Chi square could not be used to test for significance because expected frequencies were less than five in two of the four cells.

**Table 4**

Number of Patients who had Abnormal Cardiovascular Responses and Day on which these Occurred.

Day	Bed Bath	Shower
1	8	3
2	4	4
<b>Total</b>	<b>12</b>	<b>7</b>

### **Selection of Potential Patients at Risk**

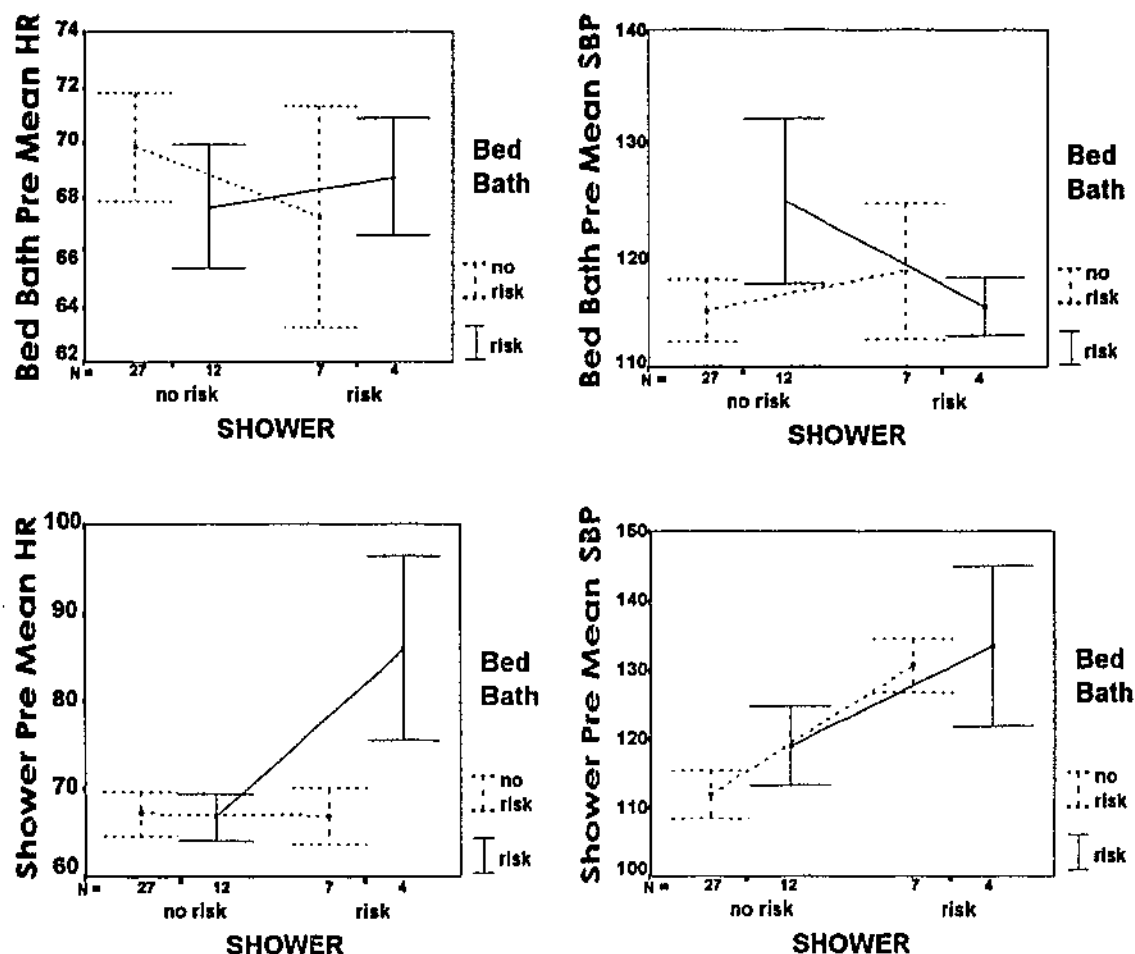
#### **Pre heart rate and systolic blood pressure measurements.**

Data were further examined in an attempt to select variables that may enable nurses to identify potential patients at risk from either bathing methods. The systolic blood pressure and heart rate measurements were used because changes in these variables were mainly responsible for the abnormal responses. Also, routine practice of bathing asymptomatic patients does not involve recording cardiovascular parameters during or after the procedure, therefore only the pre recordings were used. Subjects were divided into 4 groups depending if they were at risk, or not at risk in the shower or bed bath. One group consisted of patients at risk in both bathing methods, 2 groups were divided into those patients at risk only in the shower and those at risk only in the bed bath and the last group consisted of patients not at risk in either the bed bath or shower.

Figure 4 shows the means for the pre systolic blood pressure and heart rate of all the groups. The top two graphs show the means and standard errors of heart rate and systolic blood pressure prior to the bed bath, while the bottom two graphs show the means and standard errors of the heart rate and systolic blood pressure to the shower. Subjects who were at risk to the bed bath are



shown by the unbroken line, while those not at risk to the bed bath are shown by the broken line. Subjects who were at risk to the shower are seen to the right of the graph, while those not at risk to the shower are seen left of the graph.



**Figure 4.** The Means of the Pre Systolic Blood Pressure (SBP) and Heart Rate (HR) of all the Groups to the Bed Bath and Shower.

Figure 4 also shows that there were uneven numbers in the 4 groups and only 4 people were at risk from both the bed bath and shower. Because of the overlap in standard error bars it would not be possible to reliably identify patients at risk to either the bed bath or shower from physiological measurements taken before the bathing methods. A one way analysis of

variance (ANOVA) comparing the groups pre bed bath heart rate  $F(3, 46) = .02$ ,  $p > .05$ , and pre bed bath systolic blood pressure  $F(3, 46) = .09$ ,  $p > .05$ , showed there was no significant differences.

Of the four graphs in Figure 4 the pre mean heart rate for the shower stands out. A one way ANOVA showed there was a significant difference between the groups and the pre shower heart rate  $F(3, 46) = 2.1$ ,  $p < .05$ . Furthermore, the Tukey HSD test showed that there was a significant difference between the group who were not at risk to either of the bathing methods and the group who were at risk to both the bed bath and the shower. From a clinical perspective, this information is not helpful for two reasons. Firstly, the group only consisted of 4 patients and secondly the patients were at risk in both of the bathing methods. Consequently, their data do not identify patients who may be at risk to one or other of the bathing methods, which was the purpose of this exercise. One way ANOVA also showed there was a significant difference between the groups in their pre shower systolic blood pressure  $F(3, 46) = 3.1$ ,  $p < .05$ . However, the Tukey HSD test showed that no 2 groups were significantly different at the .05 level.

### **Location of myocardial infarction.**

The location of the myocardial infarction was also examined to determine if it provided evidence of patients who were more susceptible to risk from the bed bath or shower. Table 5 shows the location of myocardial infarction for all groups and shows no obvious patterns to suggest that a particular myocardial infarction site may contribute to patients being at risk to either bathing methods.

**Table 5**

#### **Location of Myocardial Infarction of All Groups**

<b>Myocardial Infarction Location</b>	<b>GROUP</b>			
	<b>Bed Bath At Risk N = 12</b>	<b>Neither At Risk N = 27</b>	<b>Both At Risk N = 4</b>	<b>Shower At Risk N = 7</b>
Anterior	3	7	1	3
Inferior	6	14	1	3
Lateral	1	1	0	0
Multiple	2	5	1	1
Site undefined	0	0	1	0

### **Chest Pain**

The third hypothesis stated that there would be no difference in the occurrence of chest pain when patients were showered or bed bathed by nurses. No patient developed chest pain during either of the bathing methods.

### **Subjective Responses to Bathing Methods**

#### **Subjects perceived exertion.**

The fourth hypothesis stated that patients would rate their perceived exertion, using the Borg (1973) ratings of perceived exertion scale, as greater when bed bathed by nurses as compared to being showered. To test this hypothesis a paired  $t$  test was used to test the effect of the bathing method and

perceived exertion. As predicted, patients rated their exertion greater when bed bathed ( $M = 10$ ,  $SD = 3$ ) as compared to being showered by nurses ( $M = 9$ ,  $SD = 2$ ),  $t(49) = 3.43$ ,  $p < .001$ .

The fifth hypothesis stated that the Borg (1973) ratings of perceived exertion scale would correlate with the mean heart rate for each bathing method. To test this hypothesis, Pearson's product moment correlation coefficient was used to measure the degree of association between the Borg ratings of perceived exertion and the mean heart rate for each bathing method. The correlation coefficient differed significantly from zero for both bathing methods: for the bed bath  $r(48) = -.19$ ,  $p > .05$  and for the shower  $r(48) = -.07$ ,  $p > .05$ , therefore the Borg exertion scale did not correlate significantly with the mean heart rate.

#### **Subjects' preferences.**

The sixth hypothesis stated that patients would prefer being showered to bed bathed by nurses. To test this hypothesis, chi-square analysis was used to test for differences in preference. Of the 50 patients enrolled in the study, 49 preferred being showered by nurses rather than being bed bathed  $\chi^2(1, N = 50) = 46$ ,  $p < .0001$ . Forty-four reported the shower was easier than the bed bath  $\chi^2(1, N = 50) = 28$ ,  $p < .0001$  and 48 reported they felt cleaner when showered  $\chi^2(1, N = 50) = 42.32$ ,  $p < .0001$ . These results demonstrate most patients preference for the shower to the bed bath for a variety of reasons. Table 6 describes the patients' subjective responses to preference, ease and cleanliness of the two bathing methods.

**Table 6****Subjective Responses to Bed Bathing and Showering.**

Subjective Responses	Number of Patients	
	Bed Bath	Shower
Preference	1	49
Ease	6	44
Cleanest	2	48

**Time Factor**

The last hypothesis stated that it would take longer to bed bath low risk patients post myocardial infarction than it does to shower these patients. To test this hypothesis a paired  $t$  test comparing differences in the duration of the bathing methods was used. Although there was not a large difference in the time required to bed bath and shower patients, the  $t$  test demonstrated it took significantly greater time ( $M = 35$  min,  $SD = 6$ ), to bed bath patients than shower patients ( $M = 28$  min,  $SD = 6$ )  $t(49) = 5.34$ ,  $p < .001$ .

**Summary**

The study was unable to support one of the major hypotheses, that there would be significant differences in the overall cardiovascular responses between the bathing methods. The only significant differences in cardiovascular responses, was that the  $SaO_2$  was greater during showering than during the bed bath.

However, the majority of the hypotheses were supported by the study. The study demonstrated that more patients had abnormal cardiovascular responses when bed bathed  $n = 12$ , compared to when showered  $n = 7$ . However, there was no significant differences found in the numbers of patients at risk from either bathing method. Either a fall in systolic blood pressure or

changes in heart rate and the occurrence of an arrhythmia accounted for the abnormal cardiovascular responses to both bathing methods.

As predicted, patients preferred being showered to being bed bathed. Patients' exertion was rated significantly higher when bed bathed compared to being showered, although this did not correlate with heart rate. This study also showed that it takes a longer time to bed bath than shower patients. From a clinical perspective it was reassuring that there was no occurrence of chest pain in either bathing method. Unfortunately the study was unable to define variables that may enable nurses to identify potential patients at risk to either of the bathing methods.

## Chapter 6

### Discussion

This chapter provides a comprehensive discussion of the study findings. For consistency and easy reading, it follows the same format as the results chapter.

#### **Characteristics of the Sample**

The sample consisted of a typical population in the coronary care unit of the metropolitan centre in which the study was conducted. The majority of the sample were men with an average age of 61 years. Male dominance of the sample gender is not surprising, since according to the Health Department of Western Australia (1991), more men than women aged between 25 to 64 years in Perth experience coronary heart disease.

The anatomical location of myocardial infarction was varied. People who experience anterior myocardial infarction have been shown to have a poorer prognosis with a significantly higher 1 year mortality compared to those with inferior myocardial infarction (Hands, Lloyd, & Robinson, 1986). Infarcts in the lateral surface area of the left ventricle are typically smaller (Healy, 1990). In the present study the majority of patients received thrombolytic therapy. Perhaps the wide range of the SD (844) for the creatine kinase reflects not only the varied size of myocardial infarction, but also the rapid wash out and altered creatine kinase release into the circulation by the thrombolytic therapy.

Current practice includes titrating intravenous, subcutaneous and oral medications in the first 48 hours post myocardial infarction, which would account for changes in medication during the study. However, regardless of this practice a within subject design was used, therefore each subject acted as his or her own control.

### **Comparison of Cardiovascular Responses to Both Bathing Methods**

As discussed in the physiological section under human responses of the theoretical framework, the appropriate responses to low level activity in low risk patients post myocardial infarction consist of the following: a slight increase in heart rate, which then levels off and a proportionate rise in systolic blood pressure, while the diastolic blood pressure usually remains unchanged or may decrease slightly (Fletcher & Schlant, 1994). This effect is shown in Table 2. It is also clear from Table 2 that during both bathing methods the haemodynamic parameters only altered slightly, indicating the low intensity of the level of physical activity required during both bathing methods. As there was no significant difference in the pre procedure body temperature between both bathing methods, changes in heart rate and blood pressure could not be related to activation of the vasodilator system.

The only significant difference in the overall cardiovascular responses was that the  $\text{SaO}_2$  was significantly greater during showering than bed bathing. It is likely this haemodynamic benefit was a consequence of sitting upright during the shower as compared to being supine during the bed bath. The improved cardiac output would increase the delivery of oxygenated blood, which is the goal of nursing interventions in the first 48 hours post myocardial infarction. Also, research has shown that exposure to gravitational stress improves the oxygen transport capacity with effort intolerance (Hung et al., 1983).

From a clinical perspective, this study provides reassurance that neither bathing methods causes an increase in cardiac work, as throughout both bathing methods there was no significant difference in the rate pressure product, heart rate or blood pressure. Similar findings have also been reported by Robichaud-Ekstrand (1991). However, Johnston et al. (1981) reported a



higher rate pressure product after showering, while Winslow et al. (1985) reported a higher rate pressure product after tub baths in females. In each of these three studies the patients bathed themselves, thus the increase in rate pressure product cannot solely be explained by the expected normal physiological response to increased physical activity.

A variety of reasons could account for these conflicting results. There was a wide variety of days between patients having a myocardial infarction and commencement of each of the studies. Robichaud-Ekstrand's (1991) study commenced on day 2, compared with Johnston et al. (1981) on day 3 and Winslow et al. (1985) on day 5. The increase in heart rate which both Johnston et al. and Winslow et al. reported as being responsible for the increased rate pressure product may have been a result of the deconditioning response to prolonged bed rest. It is important to also consider postural differences. In Robichaud-Ekstrand study, the patients had a sitting shower. According to Wenger (1992) cardiac work is less in the seated than in the supine position. In the Johnston et al. study patients stood in the shower, while the patients in the Winslow et al. study climbed in and out of a tub. Both of these involve much more physical activity and therefore an increase in cardiac work. Neither Johnston et al. or Winslow et al. defined abnormal responses to the level of expected physical activity. Therefore, it is possible a lower rate pressure product was seen as being normal when it may have been abnormal and a consequence of left ventricular dysfunction.

### **Abnormal Cardiovascular Responses to Bathing Methods**

The acute effects of physical activity on low risk patients post myocardial infarction relate to the imbalance that exists between myocardial oxygen supply and demand (Balady & Weiner, 1992). The ability of the coronary circulation to provide an adequate supply of blood in response to increased myocardial

demand, depends on the severity of myocardial ischemia and the degree of impairment of myocardial function. Symptoms of dysfunctioning myocardium that occur when oxygen demands exceed oxygen supply are hypotension, arrhythmias, bradycardia, tachycardia and angina (Balady & Weiner, 1992).

As hypothesised, there were a greater number of abnormal cardiovascular responses during the bed bath as compared to during the shower, although they were not significantly different. In the present study the major abnormal response from the bathing methods was a drop in systolic blood pressure in 18 patients (11 patients during the bed bath and in 7 patients during the shower). This phenomenon, is described in the pathophysiological section under the human responses in the theoretical framework.

Such a drop in systolic blood pressure during low levels of physical activity has been reported by Wenger (1992) to be indicative of activity induced ischemic ventricular dysfunction, resulting in an inadequate cardiac output to meet the demand. However, it is important to consider other potential causes for the drop in systolic blood pressure. The excessive effect of vasodilator drugs such as glycerol trinitrate and calcium channel antagonists should not be overlooked. Also, 50% of patients with inferior myocardial infarction, during the acute phase, become hypotensive and bradycardiac at rest as a result of excessive parasympathetic stimulation (Webb, Adgey, & Pantridge, 1982). Exercise induced hypotension in patients with known ischemic heart disease indicates a poor prognosis and has a predictive value of 50% for left main trunk or three vessel disease (Hammermeister, DeRouen, Dodge, & Zia, 1983).

In the present study as well as the drop in systolic blood pressure, the other abnormal responses, which occurred to a much lesser degree and only during the bed bath, consisted of changes in heart rate and rhythm. Only 1 patient had a drop in heart rate to less than 50 bpm, without an associated drop

in SBP, but in association with the development of transient CHB and reverted to SR with 1°AVB. As this patient had an inferior myocardial infarction, it is quite likely the drop in heart rate and the conduction disturbance was related to ischemia of the sinus node. In 55% of people the sinus node is supplied by the right coronary artery which would be occluded in an inferior myocardial infarction. The left circumflex supplies the sinus node in the remaining 45% of people (Anderson, Ho, & Anderson, 1979). According to Gersh (1987), sinus bradycardia occurs in 14% to 36% of patients with inferior myocardial infarction. In this case, the drop in heart rate may also have been related to an extrinsic factor such as suppression of automaticity by a beta blocker or a calcium channel blocker.

Another 2 patients on beta blockers exhibited an increase in heart rate greater than 19 bpm in association with a drop in systolic blood pressure. According to Wenger (1992), this is an unfavourable prognostic sign and reflects progressive left ventricular dysfunction. However, another explanation may be the initiation of compensatory mechanisms to maintain the cardiac output by overcoming the suppressed automaticity, increasing sympathetic stimulation and withdrawing parasympathetic outflow. In one of these patients, the sympathetic stimulation may have been further enhanced as a result of excess sympathetic outflow owing to the myocardial infarction being located in the anterior surface area of the left ventricle (Webb et al., 1982).

The present study and also the Winslow et al. (1985) and Johnston et al. (1981) studies found that no patient experienced symptoms of chest pain. Robichaud-Ekstrand (1991) reported that only 2 of the 10 patients with atypical heart rate and blood pressure responses experienced associated symptoms of shortness of breath. These findings support other research that have reported that myocardial dysfunction can occur without angina (Herzli et al., 1985).

However, perhaps in the present study if the patients had washed themselves the symptoms of chest pain may have developed.

The most common symptom reported by Robichaud-Ekstrand (1991) in patients without atypical changes in heart rate or blood pressure was fatigue and related this to a reduction in cardiac output caused by beta blockers. It may be that the reduction in cardiac output was also related to an increase in diastolic stiffness causing an increase in left ventricular diastolic pressure and a reduction in myocardial contractility as a result of myocardial dysfunction. This results in a reduced supply in oxygenated blood to the exercising muscles causing fatigue.

It is of interest to note that Robichaud-Ekstrand's (1991) criteria for atypical responses was based on those recommended by the American College of Sports Medicine. Therefore, specific responses related to ischemic myocardium may not have been considered and consequently missed.

No patient in this study or in the Robichaud-Ekstrand (1991) study developed ST segment changes. This further emphasises the low level of physical activity required during the bathing methods as ST segment depression is the most common manifestation of exercise induced myocardial ischemia (Bruce, Fisher, Pettinger, Weiner, & Chaitman, 1988). Winslow et al. (1985) reported ST segment elevation during the standing shower and Johnston et al. (1981) reported ST segment depression in all bathing methods without associated angina. This same phenomenon has been reported more recently by Dagenais et al. (1988), who found that asymptomatic ischemia occurs in approximately 60% of patients with coronary artery disease.

The variability in these results may have been related to the similar medications taken by patients in this study and Robichaud-Ekstrand (1991) study. Winslow et al. (1985) did not report the medications taken during the

study. Only 2 patients in the Johnston et al. (1981) study had beta blockers and none were on calcium channel antagonists. These drugs have been reported to delay ST segment depression, delay time to ischemia and improve exercise capacity until higher work loads (Fletcher & Schlant, 1994).

Other considerations are that the studies by Johnston et al. (1981) and Winslow et al. (1985) involved much more dynamic and isometric exercise, than the current study, which may have been associated with ST segment changes. Bruce et al. (1988) reported that approximately 50% of patients with a recent anterior myocardial infarction develop ST segment elevation during exercise. However, Johnston et al. reported the ST segment changes occurred only in patients with inferior myocardial infarction. Winslow et al. did not report the location of the myocardial infarction in relation to the ST segment changes.

In the present study 1 patient developed CHB then reverted spontaneously to SR with 1°AVB during the bed bath. Prior to beginning and throughout both bathing methods, 1 patient had ST with 1°AVB, another remained in AF and another had occasional unifocal ventricular ectopic beats. Therefore, in the present study 4 patients had arrhythmias, which is concurrent with the findings of Robichaud-Ekstrand (1991). Conversely, both Johnston et al. (1981) and Winslow et al. (1985) reported only occasional premature contractions and seven episodes of premature atrial and ventricular contractions respectively. In Robichaud-Ekstrand's study, 1 patient had 1°AVB, 2 patients had 2°AVB Mobitz II and another patient had occasional unifocal premature ventricular contractions.

Most patients with a myocardial infarction have some abnormality of cardiac conduction or rhythm disturbance during the first 24 hours. These abnormalities of rhythm and conduction disturbance are the result of ischemia and necrosis as well as of altered autonomic tone, hypoxia, electrolyte and acid

base disturbances (Janse & Wit, 1989). Atrial ischemia or infarction and pericarditis are relatively common in myocardial infarction and may cause atrial premature complexes (Nielson, Anderson, Gram-Hansen, Sorensen, & Kalusen, 1992). Conduction disturbances of CHB occur in 5% of patients and is most commonly seen in inferior myocardial infarction (Clemmensen et al., 1991).

The patient in the present study who developed transient CHB had an anterior myocardial infarction which is associated with a high mortality rate of 80% (Kostuk & Beanlands, 1970). Exercise induced CHB is rare though 1°AVB occasionally occurs at the end of exercise or during the recovery phase (Fletcher & Schlant, 1994). First degree heart block is especially common in inferior myocardial infarction though has minimal clinical significance (Roberts et al., 1994). The patients in this study who had 1°AVB had inferior myocardial infarction. It is most likely that the 1°AVB was related to acute ischemia and beta blocker therapy.

In the present study, the patient who had AF does not fit the typical criteria researchers have described when AF occurs post myocardial infarction. According to Nielson et al. (1992) AF is usually associated with large myocardial infarction complicated with cardiac failure and occurs in 10 to 15% of patients with myocardial infarction. The size of the myocardial infarction in this study's patient would be considered moderate (CK = 1147), (normal range = 25-200 U/L Roberts, 1988) and the patient had no evidence of cardiac failure or any other condition that might increase left atrial pressure such as pericarditis. Hod et al. (1987) have related the onset of AF in the acute phase of a myocardial infarction to be a consequence of left atrial ischemia. Exercise induced AF is rare, occurring in less than 1% of patients who undergo testing (Atwood et al., 1988).

According to Fletcher and Schlant (1994), exercise induced arrhythmias can either be generated by enhanced sympathetic tone and catecholamines, or an increase in myocardial oxygen demand. Ectopic ventricular contractions are commonly seen in most patients post myocardial infarction. They are generally related to electrolyte disturbances, such as hypokalemia and hypomagnesemia, cardiac failure and recurrent myocardial ischemia (Nordrehaug, Johannessen, & Von der Lippe, 1985). Ectopic ventricular contractions are the most frequent type of cardiac arrhythmia that develops during exercise.

Since this was the first study to examine patients haemodynamic responses when bed bathed and showered by nurses in the first 48 hours post myocardial infarction, it was important to assess the number of abnormal responses that occurred on each day. Similar studies have not reported this information. Eight patients had abnormal cardiovascular responses during the bed bath on day 1 and 4 patients on day 2. During the shower, on day one, 3 patients had abnormal cardiovascular responses and 4 patients on day 2. Although statistically these numbers were too small to test for a significant change, clinically they provide reassurance to nursing staff on the safety of changing current practice from bed bathing to showering patients and in doing so providing individualised care based on the patient's preference.

### **Subjective Responses**

Nurses are not only concerned with the patients' different physiological responses to bathing methods but also to their psychophysical and psychosocial responses, as described under the environmental factors of the theoretical framework. It would be expected that the patients' person factors as outlined in the theoretical framework would influence their subjective responses.

In this study, as predicted, patients reported their perceived exertion was greater when bed bathed as compared to showered by nurses. They also reported they preferred the shower as it made them feel cleaner and was considered easier than the bed bath. These findings were also reported by Robichaud-Ekstrand (1991) and Winslow et al. (1985), although in these studies the patients bathed themselves. Johnston et al. (1981) did not report subjective responses. Clearly if patients were given a choice in the first 48 hours post myocardial infarction as to which bathing method they would like, they would choose a shower.

### **Time Factor**

The reality of caring for patients in a busy coronary care unit requires nurses to prioritise their time effectively. As this study demonstrated it took significantly greater time to bed bath than shower patients, a change in nursing practice would lead to a saving in nurses' time. Nurses could allocate this extra time to other care such as rehabilitation of the patient and family members.

### **Selection of Potential Patients at Risk**

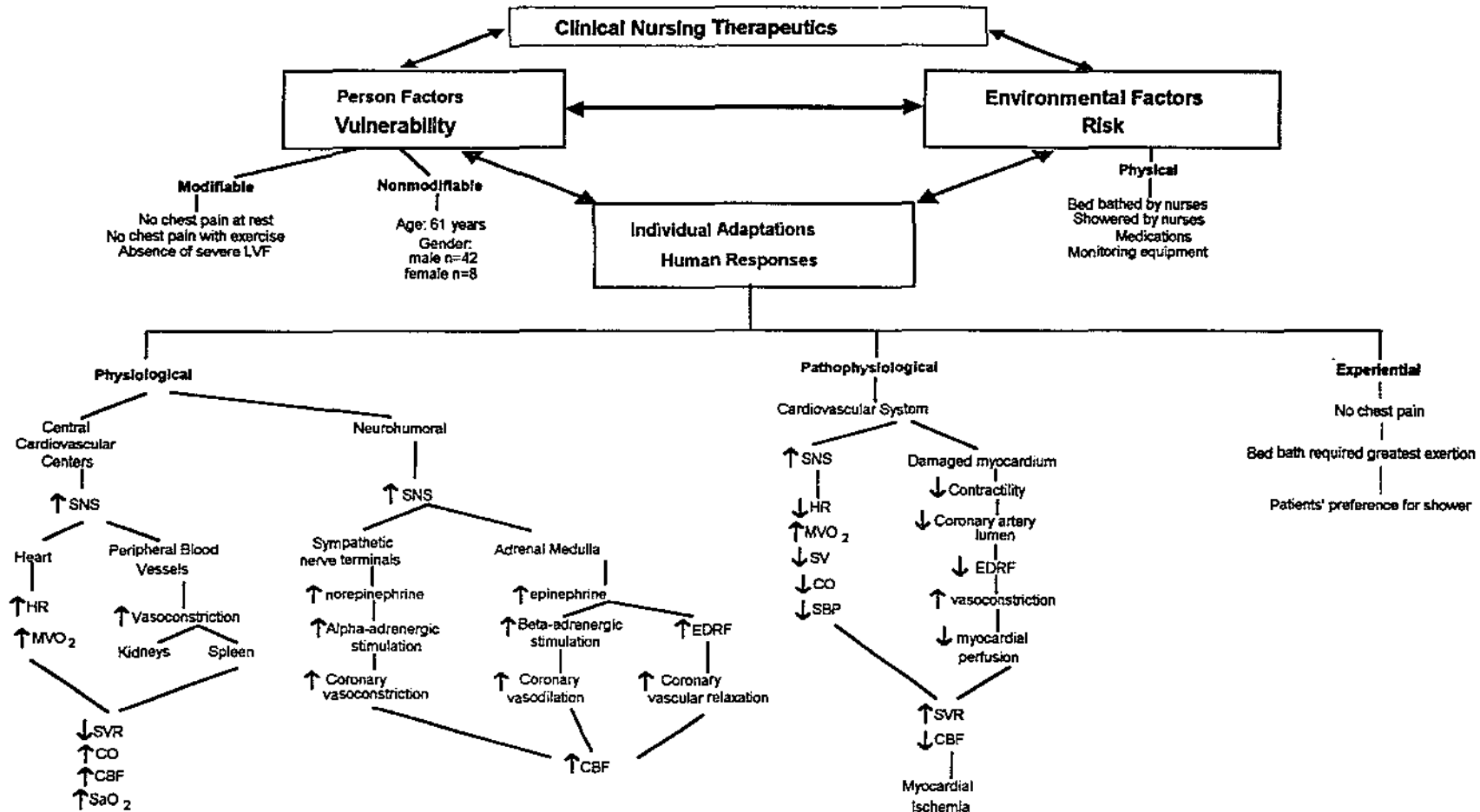
It is essential that nurses monitor, and evaluate patients' responses to all nursing care. To be aware of specific parameters that may induce abnormal responses to bathing methods would be very valuable. This study attempted to provide nurses with such criteria, but was unable to do so as no obvious patterns were found. However, it is important that nurses are aware of and recognise patients' individual abnormal cardiovascular responses to bathing, so that their level of physical activity can be altered appropriately.

### **Use of the Human Response Framework**

None of the studies by Johnston et al. (1981), Winslow et al. (1985) or Robichaud-Ekstrand (1991) reported that they used a theoretical framework to guide their research. Using Heitkemper and Shaver's (1989) Human Response



framework for this study, the investigator manipulated the physical environmental factor by introducing the nursing clinical therapy of bed bathing and showering patients. To determine the impact of this intervention, the investigator assessed all four dimensions of Heitkemper and Shaver's individual adaptations and found that most effects occurred in the physiological, pathophysiological and experiential responses (see Figure 5).



**Figure 5.** Theoretical Framework: This Studies Specific Human Responses of Low Risk Patients Post Myocardial Infarction from Bed Bathing and Showering by Nurses (LVF, left ventricular failure; SNS, sympathetic nervous system; HR, heart rate; MVO<sub>2</sub>, myocardial oxygen demand; SVR, systemic vascular resistance; CO, cardiac output; CBF, coronary blood flow; SaO<sub>2</sub>, arterial oxygen saturation; SV, stroke volume; SBP, systolic blood pressure; EDRF, endothelium derived relaxing factor). Adapted from "Nursing Research Opportunities in Enteral Nutrition" by M. Heitkemper and J. Shaver, 1989 *Advances in Nutritional Support*, 74 (2), p. 416.

## **Summary**

This is the first study to demonstrate that when low risk patients in the first 48 hours post myocardial infarction are bed bathed or showered by nurses, there is no increase in cardiac work. There have been conflicting reports supporting and opposing this finding in previous similar research. However, in these studies the patients washed themselves.

In this study the major abnormal responses to the bathing methods was a drop in systolic blood pressure in 11 patients during the bed bath and in 7 patients during the shower. Although this effect has been reported to be indicative of activity induced ischemia, other factors such as, vasodilating drugs and the effects of excessive parasympathetic stimulation should also be considered.

Changes in heart rate and rhythm accounted for the other abnormal responses and have been reported in similar studies. Factors contributing to abnormalities in conduction and rhythm include the location of the myocardial infarction, ischemia of the sinus node and altered autonomic tone.

The only significant difference in the overall cardiovascular responses was that the  $\text{SaO}_2$  was significantly greater during showering than bed bathing. It is likely this haemodynamic benefit was a consequence of sitting upright during the shower as compared to being supine during the bed bath. Consistent with similar studies the patients overwhelmingly reported their preference for a shower.

Similar previous research has not used a theoretical framework to guide their research. This study used Heitkemper and Shaver's Human Response framework to enable the researcher to investigate all human responses associated with bed bathing and showering low risk patients in the first 48 hours post myocardial infarction.

## **Chapter 7**

### **Conclusions, Implications and Recommendations**

The purpose of this experimental study was to determine what effect showering as compared to bed bathing, has on the haemodynamic and subjective responses of low risk patients within 48 hours of having a myocardial infarction. The hypotheses associated with determining the haemodynamic responses, were related to the effects each bathing method had on the cardiovascular system, whereas a psychophysical scale and a short questionnaire were used to determine those relating to subjective responses.

In the present study it was important to assess the human responses associated with the clinical nursing intervention of bed bathing and showering patients in order to understand the effects of this intervention. The use of Heitkemper and Shaver's (1989) Human Response Framework enabled the researcher to investigate the full range of human responses of low risk patients post myocardial infarction from bed bathing and showering.

#### **Conclusions**

This study demonstrated, for the first time, that low risk patients post myocardial infarction can safely be showered within the first 48 hours. It did not however, prove that bed bathing required more cardiac work than showering, as apart from significant changes in the  $\text{SaO}_2$ , there was no significant differences in the cardiovascular response to both bathing methods. The appropriate physiological responses, as described in the theoretical framework occurred during both bathing methods, with the exception of the abnormal responses, which were consistent with the pathophysiological effects, outlined in the theoretical framework. The majority of abnormal responses did, as hypothesised, occur during the bed bath, but were not significantly different.

As it is the nurse's responsibility to ensure optimal care is taken to reduce myocardial oxygen demands in the first 48 hours post myocardial infarction, it was reassuring to demonstrate that neither bathing methods provoked the occurrence of chest pain. The study also demonstrated that low risk patients in the first 48 hours post myocardial infarction can safely get out of bed, have a short walk within their room and sit on a chair, without increasing myocardial oxygen demands.

Other important results of this study for nurses are that, as predicted, it took significantly greater time to bed bath than shower patients and also nurses learned of the patients' preferences to different bathing methods. The subjective responses of the patients, as hypothesised, overwhelming demonstrated their preference for a shower to a bed bath. Their preference for the shower to the bed bath was on the basis that less exertion was required and an improved feeling of cleanliness was achieved by showering.

### **Implications**

The study results have several implications which affect not only nurses, but medical staff, patients and the community. The clinical implications are that nurses can change their practice to showering rather than bed bathing patients, based on a scientific rationale and the patients' preference. The overall management of these patients may be changed, as from as early as day 1 post myocardial infarction, they will no longer be restricted to bed rest. This will affect other aspects of nursing care, for example, patients may walk into the bathroom to use the toilet or wash at the sink.

An important part of patients' recovery and a major role of nurses, is to ensure patients have a good understanding of coronary artery disease and are aware of ways to modify particular risk factors predisposing them to heart disease. The time saved from changing nursing practice could be allocated to

rehabilitation of the patient and family members. As the patients in this study fitted a defined criteria for low risk post myocardial infarction, there is no reason these changes in nursing practice cannot be applied internationally.

Medical staff may decide to use the data obtained by nurses on patients' physiological responses to increased levels of physical activity to assist in selecting suitable patients for early exercise testing and earlier discharge. An increase in physical activity in the acute phase post myocardial infarction could thus facilitate a shorter hospital stay. Both medical and nursing staff, at the center the study was conducted, will be able to review and amend the current 7 day ambulation regimen, so that patients reach their discharge activity level sooner than was previously thought possible. As a result of this study's findings, the current hospital stay for low risk patients post myocardial infarction, could be reduced, resulting in savings in health care costs and potentially improved use of coronary care unit beds.

Implications for patients are that they can participate in their care by choosing either of the bathing methods, with the knowledge that neither of them will increase cardiac work. Thus, promoting independence and an enhanced sense of well-being with improved functional status. Patients also benefit by returning home sooner.

### **Recommendations for Further Research**

Further similar research with a larger sample size with equal numbers of men and women to determine specific parameters that identify potential patients at risk to either a bed bath or shower should be instigated. Research is also required to investigate patients cardiovascular responses to bathing themselves in the first 48 hours post myocardial infarction in an effort to determine if the ambulation regimen could be increased further.

## References

- Ahnve, S., Gilpin, E., Henning, H., Curtis, G., Collins, D., & Ross, J. (1986). Limitations and advantages of the ejection fraction for defining high risk after acute myocardial infarction. American Journal of Cardiology, 58, 872-878.
- American Nurses' Association. (1980). Nursing: a social policy statement (Published No. NP-63). Kansas City, MO: The Association.
- Anderson, K. R., Ho, S. Y., & Anderson, R. H. (1979). British Heart Journal, 41, 28-32.
- Andreoli, K. G., Zikes, D. P., Wallace, A. G., Fowkes, V. H., & Kinney, M. (1987). Comprehensive cardiac care (6th ed.). St Louis: CV Mosby.
- Atwood, J. E., Myers, J., Sullivan, M., Forbes, S., Friis, R., & Pewen, W. (1988). Maximal exercise testing and gas exchange in patients with chronic atrial fibrillation. Journal of American College of Cardiology, 11, 508-513.
- Balady, G. J. & Weiner, D. A. (1992). Physiology of exercise in normal individuals and patients with coronary heart disease. In N. K. Wenger and H. K. Hellerstein (Eds.), Rehabilitation of the coronary care patient, (3rd ed.) (pp. 103-122). New York: Churchill Livingstone.
- Bar-Or, O., Skinner, J. S., Buskirk, E. R., & Borg, G. (1972). Physiological and perceptual indications of physical stress in 41-60 year old men who vary in condition level and body fatness. Medicine and Science in Sports, 4, 96-100.

- Bloch, A., Maeder, J-P., Haissly, J- C., Felix, J., & Blackburn, H. (1974). Early mobilization after myocardial infarction. A controlled study. American Journal of Cardiology, 34, 152-157.
- Borg, G. A. V. (1973). Perceived exertion: A note on 'history' and methods. Medicine and Science in Sports, 5(2), 90-93.
- Borg, G., & Linderholm, H. (1967). Perceived exertion and pulse rate during graded exercise in various age groups. Acta Medicine Scandinavian Supplement, 472, 194-206.
- Braun, L. T., & Holm, K. (1989). Preservation of ischemic myocardium through activity management. The Journal of Cardiovascular Nursing, 3(4), 39-48.
- Bruce, R. A., Fisher, L. D., Pettinger, M., Weiner, D. A., & Chaitman, B. R. (1988). ST segment elevation with exercise: A marker for poor ventricular function and poor prognosis. Circulation, 77, 897-905.
- Brumner, P., Kalio, V., & Tala, E. (1966). Early ambulation in the treatment of myocardial infarction. Acta Medicine Scandinavian, 180, 231-237.
- Burek, K. A., Kirscht, J., & Topol, E. J. (1989). Exercise capacity in patients 3 days after acute, uncomplicated myocardial infarction. Heart & Lung, 18(6), 575-580.
- Byra-Cook, C., Dracup, K., & Kazik, A. (1989). Direct and indirect blood pressure in critical care patients. Nursing Research, 39, :85-288.
- Clausen, J. P. (1976). Circulatory adjustments to dynamic exercise and effects of physical training in normal subjects and in patients with coronary artery disease. Progressive Cardiovascular Disease, 18, 459-495.



- Clemmensen, P., Bates, E. R., Califf, R. M., Hlatky, M. A., Aronson, L., & George, B. S. (1991). Complete atrioventricular block complicating inferior wall acute myocardial infarction treated with reperfusion therapy. American Journal of Cardiology, 67, 225-230.
- Convertino, V. A., Hung, J., & Goldwater, D. (1982). Cardiovascular responses to exercise in middle-aged men after 10 days of bed rest. Circulation, 65, 134-140.
- Cunningham, S. (1992). The epidemiologic basis of coronary disease prevention. Nursing Clinics of North America, 27(1), 153-170.
- Dagenais, G. R., Rouleau, J. R., Hochart, P., Magrina, J., Cantin, B., & Dumesnil, J. G. (1988). Survival with painless strongly positive exercise electrocardiogram. American Journal of Cardiology, 62, 892-895.
- DeBusk, R. F., Blomqvist, C. G., Kouchoukos, N. T., Luepker, R. V., Miller, H. S., Moss, A. L., Pollock, M. J., Reeves, T. J., Selvester, R. H., Stason, W. B., Wagner, G. S., & Willman, V. L. (1986). Identification and treatment of low risk patients after acute myocardial infarction and coronary-artery bypass graft surgery. New England Journal of Medicine, 314(3), 161-166.
- Devore, J. L. (1991). Probability and statistics for engineering and the sciences (3rd ed.). California: Brooks Cole.
- Dock, W. (1944). The evil sequelae of complete bed rest. Journal of American Medical Association, 125, 1083-1088.
- ECG Interpretation Clinical Skillbuilders. (1990). Pennsylvania: Springhouse Corporation.

- Ekblom, B., & Goldberg, A. N. (1971). The influence of physical training and other factors on the subjective rating of perceived exertion. Acta Physiological Scandinavica, 83, 399-406.
- Fareeduddin, K., & Abelmann, W. H. (1969). Impaired orthostatic tolerance after bed rest in patients with myocardial infarction. New England Journal of Medicine, 280, 345-350.
- Fletcher, G. F., & Schlant, R. C. (1994). The exercise test. In R. C. Schlant, R. W. Alexander, R. A. O'Rourke, R. Roberts and E. H. Sonnenblick (Eds.), Hurst's the heart (8th. ed.), (pp. 423-440). New York: McGraw-Hill.
- Francis, G. S. (1987). Hemodynamic and neurohumoral responses to dynamic exercise; normal subjects vs. patients with heart disease. Circulation, 76, (suppl. VI):VI-11-V16.
- Frankenhaeuser, M. J., Post, B., Nordheden, B. & Sjöberg, H. (1969). Physiological and subjective reactions to different work loads. Perception Motor Skills, 28, 343-349.
- Gersh, B. J. (1987). Acute myocardial infarction: Arrhythmias. In R. O. Brandenburg, V. Fuster, E. R. Giuliani, & D. C. McGroon (Eds.). Cardiology: Fundamentals and Practice. (pp. 1220-1249). Chicago: year book medical publishers.
- Gorny, D. A. (1993). Arterial blood pressure measurement technique. AACN: Clinical Issues, 4(1) 66-80.
- Gobel, F. L., Nordstrom, L. A., Nelson, R. R., Jorgensen, M. D., & Wang, Y. (1978). The rate-pressure product as an index of myocardial oxygen

- consumption during exercise in patients with angina pectoris. Circulation, 57(3), 549-556.
- Griffith, T. M., Lewis, M. J., & Newby, A. C., & Henderson, A. H. (1988). Endothelium derived relaxing factor. Journal of American College of Cardiology, 12(3) 797-806.
- Groden, B. M. (1971). The management of myocardial infarction. A controlled study of the effects of early mobilisation. Cardiac Rehabilitation, 1, 13-18.
- Groden, B. M. & Brown, R. I. F. (1970). Differential psychological effects of early and late mobilisation after myocardial infarction. Scandinavia Journal of Rehabilitation Medicine, 2, 40-46.
- Gruppo Italiano per lo studio della streptochinasi nell'infarto miocardio (1986). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. The Lancet, 1, 397-401.
- Hammermeister, K. E., DeRouen, T. A., & Dodge, H. T., & Zia, M. (1983). Prognostic and predicative value of exertional hypotension in suspected coronary heart disease. American Journal of Cardiology, 51, 1261-1265.
- Hands, M. E., Lloyd, B. L., Robinson, J. S., De Klerk, N., & Thompson, P. L. (1986). Prognostic significance of electrocardiographic site of infarction after correction for enzymatic size of infarction. Circulation, 73, 885-891.
- Harpur, J. E., Kellett, R. J., Conner, W. T., Galbraith, H.-J. B., Hamilton, M., Murray, J. J., Swallow, J. H., & Rose, G. A. (1971). Controlled trial of early mobilisation and discharge from hospital in uncomplicated myocardial infarction. The Lancet, December 18, 1331-1336.

- Harrison, T. R. (1944). Abuse of rest as a therapeutic measure for patients with cardiovascular disease. Journal of American Medical Association, 125, 1075-1080.
- Health Department of Western Australia. (1991). Our state of health: an overview of the Western Australian population; Perth, Western Australia: Health department of Western Australia.
- Healy, B. (1990). Pathology of coronary atherosclerosis. In J. W. Hurst (Ed. ), The Heart, (7th ed.),. New York: McGraw-Hill.
- Heitkemper, M. M., & Shaver, J. F. (1989). Nursing research opportunities in enteral nutrition. Nursing Clinics of North America, 24(2), 415-426.
- Herzil, H. G., Leutwyler, R., & Krayenbuhl, H. P. (1985). Silent myocardial ischemia: haemodynamic changes during dynamic exercise in patients with coronary artery disease despite absence of angina pectoris. Journal of American College of Cardiology, 6(2), 275-284.
- Hod, H., Lew, A. S., Keltai, M., Cercek, B., Geft, I. I., & Shah, P. K. (1987). Early atrial fibrillation during evolving myocardial infarction: A consequence of impaired left atrial perfusion. Circulation, 75, 146-150.
- Holtzclaw, B. J. (1993). Monitoring body temperature. Clinical Issues in Critical Care Nursing, 4(1), 44-55.
- Hung, J., Goldwater, D., Convertino, V. A., McKillop, J. H., Gorris, M. L., & DeBusk, R. F. (1983). Mechanisms for decreased exercise capacity after bed rest in normal middle aged men. American Journal of Cardiology, 51, 334-348.

- Hutter, A. M., Jr., Sidel, V. W., Shine, K. I., & DeSantis, R. W. (1973). Early hospital discharge after myocardial infarction. New England Journal of Medicine, 288(22), 1141-1144.
- Hyatt, K. H., Kamenetsky, L. G., Smith, W. M. (1969). Extravascular dehydration as an etiologic factor in post recumbency orthostatism. Aerospace Medicine, 40, 644-649.
- Ireland, C., & Taylor, D. J. E. (1982). The effect of exercises starting within 24 hours of infarction. Physiotherapy, 68, 191-192.
- Irvin, C. W. Jr., & Burgess, A. M. Jr., (1950). The abuse of bed rest in the treatment of myocardial infarction. New England Journal of Medicine, 243, 486-492.
- Janse, M. J., & Wit, A. L. (1989). Electrophysiology mechanisms of ventricular arrhythmias resulting from myocardial ischemia and infarction. Physiology Review, 69, 1049-1169.
- Jette, W. W., & White, P. D., (1944). Rupture of the heart in patients in mental institutions. Annual Intern Medicine, 21, 783-802.
- Johnston, B. L., Watt, E. W., & Fletcher, G. F. (1981). Oxygen consumption and hemodynamic and electrocardiographic responses to bathing in recent post-myocardial infarction patients. Heart & Lung, 10(4), 666-671.
- Karlefors, T., Nilsen, R., & Westling, H. (1966). On the accuracy of indirect auscultatory blood pressure measurements during exercise. Acta Physiology Scandinavian Supplement, 449, 81-87.

- Keatinge, W. R., & Evans, M. (1961). The respiratory and cardiovascular response to immersion in cold and warm water. Quarterly Journal of Experimental physiology, 46, 83-94.
- Kostuk, W. J., & Bearlands, D. S. (1970). Complete heart block associated with acute myocardial infarction. American Journal of Cardiology, 26, 380-384.
- Levine, S. A. (1951). The myth of strict bed rest in the treatment of heart disease. American Heart Journal, 42, 406-413.
- Levine, S. A., & Lown, B. (1952). "Armchair" treatment of acute coronary thrombosis. Journal of American Medical Association, 148, 1365-1369.
- Lewis, T. (1946). Diseases of the heart. London: Macmillan.
- Madsen, E. B., Hougaard, P., Gilpin, E., & Pedersen, A. (1983). The length of hospitalization after acute myocardial infarction determined by risk calculation. Circulation, 68(1), 9-16.
- Magder, S. (1985). Assessment of myocardial stress from early ambulatory activities following myocardial infarction. Chest, 87, 442-447.
- Mallory, G. K., White, P. D., & Salcedo-Salgar, J. (1939). The speed of healing myocardial infarction: A study of the pathologic anatomy of 72 cases. American Heart Journal, 18, 647-671.
- Mansfield, L. W. (1971). Continuous electrocardiographic monitoring and simultaneous patient observation as a method of nursing research. Nursing Research, 4, 108-126.

- Marcus, M. (1988). Regulation of myocardial perfusion in health and disease. Hospital Practice, 22, 105-160.
- Mark, D. B., Sigmon, K., Topol, E. J., Kereiakes, D. J., Pryor, D. B., Candela, R. J., & Califf, R. M. (1991). Identification of acute myocardial infarction patients suitable for early hospital discharge after aggressive interventional therapy. Results from the thrombolysis and angioplasty in acute myocardial infarction registry. Circulation, 83(4), 1186-1193.
- Martin, D. W. (1985). Doing psychology experiments (2nd ed.). California; Brooks/Cole.
- Miers, L. J., & Arnold, R. (1990). The cardiovascular response to exercise in the patient with congestive heart failure. The Journal of Cardiovascular Nursing, 4(3), 47-58.
- Mitchell, J. H. (1985). Cardiovascular control during exercise: central and reflex neural mechanisms. American Journal of Cardiology, 55, 34D-41D.
- Mitchell, P. H., Gallucci, B., & Fought, S. G. (1991). Perspectives on human response to health and illness. Nursing Outlook, 39(4), 154-157.
- Morgan, W. P. (1973). Psychological factors influencing perceived exertion. Medicine and Science in Sports, 5(2), 97-103.
- Moss, A. J., Bigger, T. J., Case, R. B., Gillespie, J. A., Goldstein, R. E., Greenberg, H. M., Kronc, R., Marcus, F. I., Odoroff, C. L., & Oliver, G. C. (1983). The multicenter postinfarction research group: risk stratification and survival after myocardial infarction. New England Journal of Medicine, 309, 331-336.

- Mukharji, J., Rude, R. E., Poole, W. K., Gustafson, N., Thomas, L. J., Strauss, H. W., Jaffe, A. S., Muller, J. E., Roberts, R., Raabe, D. S., Croft, C. H., Passamani, E., Braunwald, E., Willerson J. T., & the MILIS study group. (1984). Risk factors for sudden death after acute myocardial infarction: two year follow up. American Journal of Cardiology, 54, 31-36.
- Nielson, F. E., Anderson, H. H., Gram-Hansen, P., Sorensen, J. T., & Kalusen, I. C. (1992). The relationship between ECG signs of atrial infarction and the development of supraventricular arrhythmias in patients with acute myocardia infarction. American Heart Journal, 123, 69-72.
- Nordrehaug, J. E., Johannessen, K. A., & Von der Lippe, G. (1985). Serum potassium concentration as a risk factor of ventricular arrhythmias early in acute myocardial infarction. Circulation, 71, 645-649.
- Pipine, C. (1989). New concepts in the pathophysiology of acute myocardial infarction. American Journal of Cardiology, 64, 2B-8B.
- Roberts, R. (1990). Enzymatic estimation of infarct size. Thrombolysis induced its demise: Will it now rekindle its renaissance?. Circulation, 81, 707-710.
- Roberts, R. (1988). Enzymatic diagnosis of acute myocardial infarction. Chest, 93, 3S-6S.
- Roberts, R., Pratt, C. M., Morris, D., & Alexander, R. W. (1994). Pathophysiology, recognition, and treatment of acute myocardial infarction and its complications. In R. C. Schlant, R. W. Alexander, R. A. O'Rourke.



- R. Roberts and E. H. Sonnenblick (Eds.), Hurst's the heart (8th. ed. ), (pp. 1107-1184. New York: McGraw-Hill.
- Robichaud-Ekstrand, S. (1991). Shower versus sink bath: Evaluation of heart rate, blood pressure and subjective response of the patient with myocardial infarction. Heart & Lung, 20(4), 374-482.
- Rowe, M. H., Jelinek, M. V., Liddell, N., & M. Hagens (1989). Effect of rapid mobilization on ejection fractions and ventricular volumes after acute myocardial infarction. American Journal of Cardiology, 68, 1037-1041.
- Saltin, B., Blomqvist, G., & Mitchell, J. H. (1968). Responses to exercise after bed rest and after training. Circulation, 38, (suppl VII) VII-1-VII-55.
- Schlant, R. C., & Sonnenblick, E. H. (1994). Normal physiology of the cardiovascular system. In R. C. Schlant, R. W. Alexander, R. A. O'Rourke. R. Roberts and E. H. Sonnenblick (Eds.), Hurst's the heart (8th. ed. ), (pp. 113-151. New York: McGraw-Hill.
- Siegal, S., & Castellan, N.J., Jr. (1988). Nonparametric statistics for the behavioural sciences (2nd ed.). New York; McGraw-Hill.
- Simoons, M. L., Serruys, P. W., Brand, M. V., & De Zwaan, C. (1985). Improved survival after early thrombolysis in acute myocardial infarction. The Lancet, 2, 578-582.
- Stevens, S. S. (1971). Issues in psychophysical measurement. Psychology Review, 78, 426-450.
- Stewart, S. L. (1992). Acute MI: A review of pathophysiology, treatment, and complications. The Journal of Cardiovascular Nursing, 6(4), 1-25.

- Swan, H. J. C., Blackburn, H. W., & DeSanctis, R. (1976). Duration of hospitalization in "uncomplicated completed acute myocardial infarction." An ad hoc committee review. American Journal of Cardiology, 37, 413-419.
- Terndrup, T. E., Allegra, J. R., & Keally, J. A. (1987). A comparison of oral, rectal and tympanic membrane derived temperature changes after ingestion of liquids and smoking. American Journal of Emergency Medicine, 7, 150-154.
- Topol, E. J., Burek, K. A., O'Neill, W. W., Kewman, D. G., Kander, N. H., Shea, M. J., Schork, M. A., Kirscht, J., Juni, J. E., & Pitt, B. (1988). A randomized controlled trial of hospital discharge three days after myocardial infarction in the era of reperfusion. New England Journal of Medicine, 318, 1083-1088.
- Topol, E. J., Califf, R. M., Kereiakes, D. J., George, B. S., Abbot-Smith, C. W., Candela, R. J., Lee, K. L., Pitt, B., Stack, R. S., & O'Neil, W. W. (1987). A randomized trial of immediate versus delayed elective angioplasty after intravenous tissue plasminogen activator in acute myocardial infarction. New England Journal of Medicine, 317, 581-588.
- Urban, N. (1990). Hemodynamic clinical profiles. Clinical Issues in Critical Care Nursing, 1, 119-130.
- Van der Laarse, A., Vermeer, F., & Hermens, W. T. (1986). Effects of early intracoronary streptokinase on infarct size estimated from cumulative enzyme release and on enzyme release rate: a randomized trial of 533

patients with acute myocardial infarction. American Heart Journal, 112, 672-681.

Vanhoutte, P. M. (1988). The endothelium-modulator of vascular smooth muscle tone. New England Journal of medicine, 319(8), 512-513.

Webb, S. W., Adgey, A. A., & Pantridge, J. F. (1982). Autonomic disturbance at onset of acute myocardial infarction. British Medical Journal, 1, 121-125.

Wenger, N. K. (1984). Early ambulation physical activity: Myocardial infarction and coronary artery bypass surgery. Heart & Lung, 13(1), 14-18.

Wenger, N. K. (1992). In-hospital exercise rehabilitation after myocardial infarction and myocardial revascularization: physiologic basis, methodology, and results. In N. K. Wenger and H. K. Hellerstein (Eds.), Rehabilitation of the coronary care patient, (3rd ed.) (pp. 351-365). New York: Churchill Livingstone.

Wenger, N. K., Hellerstein, H. K., Blackburn, H. & Castranova, S. J. (1982). Physician practice in the management of patients with uncomplicated myocardial infarction: Changes in the past decade. Circulation, 65(3), 421-427.

Western Australian School Of Nursing. (1983). Guidelines for nursing procedures (2nd ed.). Perth: Author.

Williams, D. O., Amsterdam, E. A., & DeMaria, A. N. (1976). Physical activity in the rehabilitation of patients following myocardial infarction. 1. Basis of early ambulation. Heart & Lung, 5, 317-321.

Winslow, E. H. , Lane, L. D., & Gaffney, F. A. (1985). Oxygen uptake and cardiovascular responses in control adults and acute myocardial infarction patients during bathing. Nursing Research, 34(3), 164-169.

## Appendix A

### Borg Scale of Perceived Exertion and Questionnaire

The Borg rating scale of perceived exertion (please circle one of the following statements which best indicates the amount of exertion you felt each bathing method had on you).

---

6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very, very hard
20	

---

From "Perceived exertion: a note on history and methods" by G. A. V. Borg, 1973, Medicine and Science in Sports, 5(2), p.92.

### Questionnaire

Please complete this questionnaire when the patient has completed both bathing methods.

	Bed Bath	Shower
1. Which bathing method do you feel was easiest for you.	<input type="checkbox"/>	<input type="checkbox"/>
2. Which bathing method do you feel made you feel cleanest.	<input type="checkbox"/>	<input type="checkbox"/>
3. Which bathing method did you prefer.	<input type="checkbox"/>	<input type="checkbox"/>

**Appendix B**

**Demographic Data**

Sex:	Male	<input type="checkbox"/>	Female	<input type="checkbox"/>
Age:	<input type="text"/>			
Site of Infarction:				
Anterior:	<input type="checkbox"/>			
Inferior:	<input type="checkbox"/>			
Lateral:	<input type="checkbox"/>			
Multiple location - please state:	<input type="checkbox"/>			
	<input type="text"/>			
	<input type="text"/>			
	<input type="text"/>			
Site undefined:	<input type="checkbox"/>			
Cardiac enzymes (CK):	<input type="text"/>			

**Past Medical History**

Appendix C

Medication Data

Intravenous

Drug Name	Dose	Mls/hr
GTN		
Heparin		
Magnesium		
Lignocaine		
OTHER:		
OTHER:		

Oral

Medication	Drug Name	Dose	Time Given
Platelet Inhibitors			
Beta Blockers			
Calcium Channel Antagonists			
Antiarrhythmic Agents			
Nitrates			
Angiotensin Converting Enzyme Inhibitors			

## Appendix D

### Bed Bath Procedure

Prior to commencing the bed bath offer the patient a bed pan/urinal. Do not include the time taken to use the bed pan/urinal in the duration of the bed bath.

#### Bed Bath

##### Equipment

Trolley	Clean bed linen
Wash bowl	Clean pyjamas/nightie
Two bath towels	Soiled linen carrier
One wash towel	Patients own toilet requisites
One sponge blanket	

##### Bed Bath Protocol

1. Cover patient with the sponge blanket.
2. Remove top bed clothes and put them on the bed end.
3. Remove patient's clothing.
4. Fill wash bowl with water and check temperature is between 35.6°C and 36.7°C.
5. Using face cloth, wash patients face and neck, then dry.
6. Place one towel under the arm farthest away. Wash and rinse arm from shoulder to wrist. Immerse hand in basin and wash. Dry with second towel.
7. Repeat for other arm.
8. Wash chest, axilla and abdomen, then dry.
9. Place towel under leg farthest away and wash from groin to ankle. Immerse foot in basin, wash and dry.
10. Repeat for other limb.
11. Change water.
12. Roll patient on side. Place towel along his/her side and wash back and dry with second towel.
13. Change bottom sheet.
14. Roll patient to other side. Place towel in position to protect bed linen. Wash back and dry.
15. Place towel under buttock. Check pressure areas. Allow patient to wash his/her own genitalia. Wash and dry buttocks and apply creams if necessary. N2 to record BP, SaO<sub>2</sub>, heart rate rhythm, ST changes and presence of chest pain.
16. Dress patient in clean pyjamas/nightie.
17. Attend to mouth care and hair.
18. Complete bed making, remove sponge blanket. N2 to record BP, SaO<sub>2</sub>, heart rate rhythm, ST changes and presence of chest pain.
19. Discard wash towel, soiled linen and put away toiletries. Record the time the bed bath was completed.

Bathing protocol from Guidelines for nursing procedures (p. A21-A22) by Western Australian school of nursing, 2nd ed. 1983, Perth: Author.



## **Appendix E**

### **Shower Procedure**

Prior to commencing the shower offer the patient a bed pan/urinal. Do not include the time taken to use the bed pan/urinal in the duration of the shower.

#### **Shower**

##### **Equipment**

- Two bath towels
- One wash towel
- Clean bed linen
- Clean pyjamas/nightie
- Patients own toilet requisites, including mouth care and hair brush
- Soiled linen carrier

#### **Shower Protocol**

1. Assist patient out of the bed.
2. Walk patient to the bathroom and sit patient on the shower chair.
3. Undress the patient.
4. Adjust and test the water temperature is between 35.6°C and 36.7°C.
5. Wash patient in the following order: face, neck, arms, hands, chest, axilla, abdomen, legs, and feet. Stand patient up so that buttocks can be washed and allow patient to wash his/her own genitalia. When the patient is sitting down again the N2 is to record BP, SaO<sub>2</sub>, heart rate, rhythm, ST changes and presence of chest pain.
6. Dry and dress the patient in clean pyjamas/nightie.
7. Sit the patient by the sink and allow the patient to attend mouth care and hair care.
8. Walk patient back to a chair by the bed.
9. N2 to record BP, SaO<sub>2</sub>, heart rate, rhythm, ST changes and presence of chest pain.
10. Make the bed.
11. Assist patient into the bed.
12. Discard linen and put away toiletries. Record time shower completed.

Shower Protocol from Guidelines for nursing procedures (p. A21-A22) by Western Australian school of nursing, 2nd ed. 1983, Perth: Author.

## Appendix F

### Bathing Method Observation Chart

#### Haemodynamic Data

At 5 minutes prior to bed bath/shower - time =	Heart rate/ min	Rhythm	ST Segment	Blood pressure mmHg	SaO <sub>2</sub>	Presence of chest pain
_____ hrs						
Record time the nurse started to collect equipment = _____ hrs. Body Temp.:						
During wash after buttocks record the time =	Heart rate/ min	Rhythm	ST Segment	Blood pressure mmHg	SaO <sub>2</sub>	Presence of chest pain
_____ hrs						
Immediately after bed bath/shower is completed	Heart rate/ min	Rhythm	ST Segment	Blood pressure mmHg	SaO <sub>2</sub>	Presence of chest pain
_____ hrs						
Record time the bed bath/shower is fully completed = _____ hrs						
Five minutes post bed bath/shower record the time =	Heart rate/ min	Rhythm	ST Segment	Blood pressure mmHg	SaO <sub>2</sub>	Presence of chest pain
_____ hrs						

Record a 12 lead ECG if the patient had any ST segment changes, atrial fibrillation, atrioventricular blocks and ventricular fibrillation or ventricular tachycardia during the bathing method.

## Appendix G

### Consent Form

**STUDY TITLE:** A comparison of two bathing methods on patients who have had a heart attack within 48 hours. Effects on the heart and subjective response of patients.

**INVESTIGATOR:** Heather Kidd

Heather Kidd is a registered nurse who is employed as a clinical nurse in the coronary care unit at Royal Perth Hospital. As part of her Master's degree through Edith Cowan University she is reviewing the hospital's current practice of bed bathing patients for two days after a heart attack. The study has been approved by your Cardiologist, Royal Perth Hospital's and Edith Cowan University's research and ethics committees.

The study involves patients being allocated to either a bed bath the first day, then a shower the next day, or the reverse. The current literature supports mobilisation within the first two days after a heart attack. Consequently, participation in this study should not put you at any further risk. Your blood pressure, heart rate, rhythm and blood oxygen content will be recorded using non-invasive methods by the nurse, before, during and after the bathing method. Since the purpose of this research includes discovering what type of bathing method you prefer, you will be asked a series of questions. It is hoped that results from this study will lead to improvements in quality of patient care and promote a more rapid recovery after a heart attack which will reduce the length of stay in hospital.

This is to certify that I \_\_\_\_\_ hereby consent to take part in the above named study. I have been given the opportunity to ask questions and to have a member of my family, or a friend present while the study was explained to me. If I need to ask Heather Kidd any further questions, I know I can reach her at work by asking any nurse to contact her for me. I am also aware that my participation in this study is strictly voluntary and that I have the right to withdraw from this study at any time without prejudice.

I understand that my identity will be concealed and I give my consent for the data collected to be published.

\_\_\_\_\_  
Participant

\_\_\_\_\_  
Witness

\_\_\_\_\_  
Date

## **Appendix H**

### Patient Information Sheet

**A comparison of two bathing methods on patients who have had a heart attack within 48 hours. Effects on the heart and subjective response of patients.**

**Investigator: Heather Kidd**

Heather Kidd is a registered nurse who is employed as a clinical nurse in the coronary care unit at Royal Perth hospital. As part of her Master's degree through Edith Cowan University she is reviewing the hospital's current practice of bed bathing patients for two days after a heart attack.

The purpose of this study is to compare the effects of two bathing methods on low risk patients in their first two days after a heart attack. The study involves patients being allocated to either a bed bath the first day, then a shower the next day or the reverse. The current literature supports mobilisation within the first two days after a heart attack. Consequently, participation in this study should not put you at any further risk.

Your blood pressure, heart rate, rhythm and blood oxygen content will be recorded using non-invasive methods by the nurse, before, during and after the bathing method. Since the purpose of this research includes discovering what type of bathing method you prefer, you will be asked a series of questions.

It is hoped that results from this study will lead to improvements in the quality of patient care and promote a more rapid recovery after a heart attack which will reduce the length of stay in hospital.

Your participation in this study is strictly voluntary and you have the right to withdraw from the study at any time without prejudice. This study has been approved by your Cardiologist, Royal Perth Hospital's and Edith Cowan University's research and ethics committees.

If you have any concerns about this study, or you need to ask any further questions, please do not hesitate to contact Heather Kidd at work by asking any nurse to contact her for you. If so desired, correspondence regarding any concerns about this project can be directed to Dr JM White, Chairperson, Ethics Committee, c/- Medical Administration, Royal Perth Hospital, Wellington St, Perth 6001.