



3 1293 01572 5512

This is to certify that the

thesis entitled

TRENDS IN UTILIZATION OF SELECTED TREATMENTS
FOR ACUTE MYOCARDIAL INFARCTION
ACROSS RACE, GENDER, AGE AND PAYER GROUP
IN A COMMUNITY HOSPITAL, 1990-1994
presented by

VALERIE ROSE LINT

has been accepted towards fulfillment
of the requirements for

~~MASTER~~ degree in ~~EPIDEMIOLOGY~~

Major professor

Date

7/11/96

LIBRARY
Michigan State
University

PLACE IN RETURN BOX to remove this checkout from your record.
TO AVOID FINES return on or before date due.

DATE DUE	DATE DUE	DATE DUE
<hr/>	<hr/>	<hr/>
<hr/>	<hr/>	<hr/>
<hr/>	<hr/>	<hr/>
<hr/>	<hr/>	<hr/>
<hr/>	<hr/>	<hr/>
<hr/>	<hr/>	<hr/>
<hr/>	<hr/>	<hr/>

**TRENDS IN UTILIZATION OF SELECTED TREATMENTS
FOR ACUTE MYOCARDIAL INFARCTION
ACROSS RACE, GENDER, AGE AND PAYER GROUP
IN A COMMUNITY HOSPITAL, 1990-1994**

By

Valerie Rose Lint

A THESIS

**Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of**

MASTER OF SCIENCE

Program in Epidemiology

1996

ABSTRACT

TRENDS IN UTILIZATION OF SELECTED TREATMENTS FOR ACUTE MYOCARDIAL INFARCTION ACROSS RACE, GENDER, AGE, AND PAYER GROUP IN A COMMUNITY HOSPITAL, 1990-1994

By

Valerie Rose Lint

The main purpose of my thesis project was to describe the provision of care for acute myocardial infarction over a five year period among patients admitted at a community hospital. I was interested in assessing whether my data provided evidence that differential provision of thrombolytic therapy, cardiac catheterization, coronary artery bypass grafting, or percutaneous transluminal coronary angioplasty existed among the different patient subpopulation groups of my study population.

I found for time trends, there was no effect modification or confounding by any of the other variables of age, race, gender, insurance, and comorbidity. However, for the different procedures, some of the above mentioned variables were independent predictors of utilization, either as main effects only, or as interactions between themselves. Nevertheless, the inclusion of these significant independent predictors and their interactions did not change my conclusion about the time trends in the utilization of a given procedure.

**Dedicated to my wonderful family and friends for their unconditional love, support,
and guidance.**

ACKNOWLEDGMENTS

This one page acknowledgment is in no way a direct reflection of the amount of appreciation that I have for my family and friends for their encouragement and guidance. The last two years have been a time of reflection, as well as an adjustment period. I am grateful for their unconditional love and support especially during this confusing time. They each continuously reminded me to believe in myself and not to settle for less. Personal goals and dreams that seemed impossible, were again aspired with the strength that they each gave because they never stopped believing. In addition, I wish to acknowledge Dr. Srinivas Bhadriraju and the medical records staff at McLaren Regional Medical Center. With their cooperation and help, this thesis was made possible.

There is one special person that I am especially grateful for, and that is my mother. She has devoted her life to my happiness. This happiness has not always been easy to find; however, my mother gave me the faith to believe in myself and God. Through the many tears and long nights, I could always rely on my mother to provide me with the inspiration and strength to continue, even when the impossible was staring back at me. I could never thank her enough, but can only hope that one day I, too, will be able to provide my children with the comfort and love that she has showed with me.

TABLE OF CONTENTS

LIST OF TABLES	vii
LIST OF FIGURES	x
INTRODUCTION	1
CHAPTER 1	
CORONARY HEART DISEASE	3
General Anatomy	3
Clinical presentation of acute myocardial infarction	4
CHAPTER 2	
EPIDEMIOLOGY OF CORONARY HEART DISEASE	8
Coronary heart disease mortality	8
Hospitalization for acute myocardial infarction	9
Acute myocardial hospital case fatality rates	10
Medical care	10
CHAPTER 3	
THROMBOLYTIC THERAPY	13
Streptokinase	13
Recombinant tissue-type plasminogen activator	15
Anistreplase	19
Contraindications to thrombolytic therapy	20
Thrombolytic therapy in older patients	21
Distribution by demographics	22
CHAPTER 4	
CARDIAC CATHETERIZATION	25
History	25
Procedures	26
Purposes	27
Distribution by demographics	29
CHAPTER 5	
CORONARY ARTERY BYPASS GRAFTING	32
History	32
Procedure	33
Outcome Events	34
Guidelines	35
Distribution by demographics	36

CHAPTER 6	
PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY	40
History	40
Procedure	42
Limitations	46
Restenosis	46
Abrupt reclosure	47
National Heart, Lung, and Blood Institute Registry	48
Guidelines	49
Distribution by demographics	51
 CHAPTER 7	
CORONARY ANGIOPLASTY Vs. BYPASS SURGERY	54
 CHAPTER 8	
METHODS	59
Data Base	59
Patient Population	59
Definition of Variables	61
Procedure Use	62
Statistical Analysis	63
 CHAPTER 9	
RESULTS	65
Patient Population	65
Procedure Use	68
Statistical Analysis	70
 CHAPTER 10	
DISCUSSION	76

LIST OF TABLES
(See Appendix H)

- Table 1 - Characteristics of patients admitted to community hospital with acute myocardial infarction diagnosis: 1990-1994
- Table 2 - Age distribution by gender, race, insurance, and comorbidity; 1990-1994
- Table 3 - Gender distribution by age, race, insurance, and comorbidity; 1990-1994
- Table 4 - No. of AMI's by insurance, distributed across age, race, gender, and comorbidity; 1990-1994
- Table 5 - No. of AMI's by race, distributed across age, gender, comorbidity, and insurance; 1990-1994
- Table 6 - No. of AMI's by comorbidity, distributed across age, gender, race, and insurance; 1990-1994
- Table 7 - Unadjusted odds ratios on thrombolytic therapy utilization for patients up to 64 years of age, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization
- Table 8 - Unadjusted odds ratios on thrombolytic therapy utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization
- Table 9 - Unadjusted odds ratios on cardiac catheterization utilization for patients up to 64 years of age, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization
- Table 10 - Unadjusted odds ratios on cardiac catheterization utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization
- Table 11 - Unadjusted odds ratios on coronary artery bypass grafting utilization for patients up to 64 years of age, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization
- Table 12 - Unadjusted odds ratios on coronary artery bypass grafting utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization

Table 13 - Unadjusted odds ratios on percutaneous transluminal coronary angioplasty utilization for patients up to 64 years of age, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization

Table 14 - Unadjusted odds ratios on percutaneous transluminal coronary angioplasty utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization

Table 15 - Unadjusted odds ratios on thrombolytic therapy utilization and adjusted by statistically significant predictors for other effects of admission year on utilization as the final models

Table 16 - Unadjusted odds ratios on cardiac catheterization utilization and adjusted by statistically significant predictors for other effects of admission year on utilization as the final models

Table 17 - Unadjusted odds ratios on coronary artery bypass grafting utilization and adjusted by statistically significant predictors for other effects of admission year on utilization as the final models

Table 18 - Unadjusted odds ratios on percutaneous transluminal coronary angioplasty utilization and adjusted by statistically significant predictors for other effects of admission year on utilization as the final models

Table 19 - Utilization of Thrombolytic Therapy; Change in Chi-Square for patients up to 64 years of age

Table 20 - Utilization of Thrombolytic Therapy; Change in Chi-Square for patients more than 65 years of age

Table 21 - Utilization of Cardiac Catheterization; Change in Chi-Square for patients up to 64 years of age

Table 22 - Utilization of Cardiac Catheterization; Change in Chi-Square for patients more than 65 years of age

Table 23 - Utilization of Coronary Artery Bypass Grafting; Change in Chi-Square for patients up to 64 years of age

Table 24 - Utilization of Coronary Artery Bypass Grafting; Change in Chi-Square for patients more than 65 years of age

- Table 25 - Utilization of Percutaneous Transluminal Coronary Angioplasty; Change in Chi-Square for patients up to 64 years of age**
- Table 26 - Utilization of Percutaneous Transluminal Coronary Angioplasty; Change in Chi-Square for patients more than 65 years of age**
- Table 27 - Unadjusted odds ratios on thrombolytic therapy utilization across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization**
- Table 28 - Unadjusted odds ratios on cardiac catheterization utilization across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization**
- Table 29 - Unadjusted odds ratios on coronary artery bypass grafting utilization across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization**
- Table 30 - Unadjusted odds ratios on percutaneous transluminal coronary angioplasty utilization across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization**
- Table 31 - Unadjusted and fully-adjusted odds ratios on thrombolytic therapy utilization across all ages**
- Table 32 - Unadjusted and fully-adjusted odds ratios on cardiac catheterization utilization across all ages**
- Table 33 - Unadjusted and fully-adjusted odds ratios on coronary artery bypass grafting utilization across all ages**
- Table 34 - Unadjusted and fully-adjusted odds ratios on percutaneous transluminal coronary angioplasty utilization across all ages**
- Table 35 - Utilization of Thrombolytic Therapy; Change in Chi-Square across all ages**
- Table 36 - Utilization of Cardiac Catheterization; Change in Chi-Square across all ages**
- Table 37 - Utilization of Percutaneous Transluminal Coronary Angioplasty; Change in Chi-Square across all ages**
- Table 38 - Utilization of Coronary Artery Bypass Grafting; Change in Chi-Square across all ages**

LIST OF FIGURES

(See Appendix I)

- Figure 1 - Age-adjusted death rates per 100,000 for ischemic heart disease by gender and race: United States
- Figure 2 - Trends in Mortality Due to Coronary Heart Disease from 1970 to 1990, According to the Location of Death, among Residents of the Twin Cities Area Who Were 30 to 74 Years of Age
- Figure 3 - Number of coronary artery bypass graft (ICD-9-CM 36.1) and coronary angioplasty procedures (removal of coronary obstruction, ICD-9-CM 36.0); United States
- Figure 4 - Trends in Acute Medical Care for Residents of the Twin Cities Area, 30 to 74 Years of Age, Who Were Hospitalized for Definite Acute Myocardial Infarction in 1985 and 1990
- Figure 5 - AMI Admission by Age 1990-1994
- Figure 6 - AMI Admission by Gender 1990-1994
- Figure 7 - % 65 Years of Age & Older, AMI Admission by Insurance, 1990-1994
- Figure 8 - % Less Than 65 Years of Age, AMI Admission by Insurance, 1990-1994
- Figure 9 - AMI Admission by Insurance, 1990-1994
- Figure 10 - AMI Admission by Race, 1990-1994
- Figure 11 - AMI Admission by Comorbidity, 1990-1994
- Figure 12 - % Receiving Thrombolytic Therapy, AMI Admission, 1990-1994
- Figure 13 - % Receiving Cardiac Cath, AMI Admission, 1990-1994
- Figure 14 - % Receiving CABG, AMI Admission, 1990-1994
- Figure 15 - % Receiving PTCA, AMI Admission, 1990-1994
- Figure 16 - % Receiving Thrombolytic Therapy, AMI Admission by Age, 1990-1994
- Figure 17 - % Receiving Thrombolytic Therapy, AMI Admission by Gender, 1990-1994

Figure 18 - % Receiving Thrombolytic Therapy, AMI Admission by Race, 1990-1994

Figure 19 - % Receiving Thrombolytic Therapy, AMI Admission by Insurance, 1990-1994

Figure 20 - % Receiving Cardiac Cath, AMI Admission by Comorbidity, 1990-1994

Figure 21 - % Receiving Cardiac Cath, AMI Admission by Age, 1990-1994

Figure 22 - % Receiving Cardiac Cath, AMI Admission by Gender, 1990-1994

Figure 23 - % Receiving Cardiac Cath, AMI Admission by Race, 1990-1994

Figure 24 - % Receiving Cardiac Cath, AMI Admission by Insurance, 1990-1994

Figure 25 - % Receiving Cardiac Cath, AMI Admission by Comorbidity, 1990-1994

Figure 26 - % Receiving CABG, AMI Admission by Age, 1990-1994

Figure 27 - % Receiving CABG, AMI Admission by Gender, 1990-1994

Figure 28 - % Receiving CABG, AMI Admission by Race, 1990-1994

Figure 29 - % Receiving CABG, AMI Admission by Insurance, 1990-1994

Figure 30 - % Receiving CABG, AMI Admission by Comorbidity, 1990-1994

Figure 31 - % Receiving PTCA, AMI Admission by Age, 1990-1994

Figure 32 - % Receiving PTCA, AMI Admission by Gender, 1990-1994

Figure 33 - % Receiving PTCA, AMI Admission by Race, 1990-1994

Figure 34 - % Receiving PTCA, AMI Admission by Insurance, 1990-1994

Figure 35 - % Receiving PTCA, AMI Admission by Comorbidity, 1990-1994

INTRODUCTION

Sufferers from coronary heart disease are in need of medical attention. The type of medical procedure administered continues to be a topic of controversy. Because of recent technological advances, the once accepted treatment regimen for acute myocardial infarction patients has been under scrutiny. The treatment regimen included receiving thrombolytic therapy, particularly the thrombolytic agent streptokinase, and then surgery. The type of surgery depended upon whether the patient was suffering from single or multiple vessel disease. Percutaneous transluminal coronary angioplasty was a cardiovascular procedure that was an option only for single vessel disease patients. However, with the introduction of the balloon catheter, angioplasty is now commonly used for multiple vessel disease patients as an alternative to bypass surgery. The severity of the patient's condition, comorbidity states, and the patient's demographics are also potential predictors of the utilization of cardiovascular procedures.

These predictors in the utilization of cardiovascular procedures have been suspicioned as the cause of disparity in their provision among certain patient subgroups. Even though the mortality rate of coronary heart disease is declining, the decline is not consistent among all patient subpopulation groups. Many studies suggest a disturbing disparity in the utilization of procedures in myocardial infarction management across different patient subgroups (110,111,112,113,114). However, whether the unequal distribution of treatment is as a result of demographics, is not clearly defined.

The purpose of my analysis is to determine whether a discrepancy exists in the provision of cardiovascular treatment among certain subgroups, in the community teaching hospital to which the patients in my database were admitted. By providing you first with a description of the provision of care for acute myocardial infarction over a five year period (1990-1994) among patients admitted at the hospital, I will examine the provision of the four cardiovascular procedures: cardiac catheterization (CATH), coronary artery bypass grafting (CABG), percutaneous transluminal coronary angioplasty (PTCA), and thrombolytic therapy across age, gender, race, insurance, and comorbidity. I will provide information to answer the following two research questions:

1. Do age, gender, race, insurance, and comorbidity individually or jointly predict the utilization of each of the four cardiovascular procedures (CATH, CABG, PTCA, and thrombolytic therapy)?

2. Is admission year (time) alone or jointly with each or all the variables a predictor of each of the four cardiovascular procedures?

Chapter 1

CORONARY HEART DISEASE

To understand the implications of procedure utilization, one needs to have a general knowledge of the problem at hand, coronary heart disease (CHD). In addition, one needs to have a basic knowledge of the vascular system. This brief summary will be helpful when later discussing the medical procedures of interest.

General Anatomy

The coronary artery and its branches provide nourishment to the coronary muscles of the heart, with oxygen and nutrients. The coronary artery is the branching vessel from the aorta that is the main vessel that routes oxygenated blood out of the left ventricle of the heart. The left ventricle is one of four chambers of the heart. When the left ventricle contracts, it forces the blood out of the chamber. Once circulated through the body, the deoxygenated blood returns to the right side of the heart, where the right atrium accepts the venous blood from the body and the right ventricle forces the blood out to the lungs. Once reoxygenated, the blood returns to the left side of the heart, where the left atrium accepts the oxygenated blood and the left ventricle by way of the aorta, pushes the blood out the heart and through the body.

The normal coronary circulation is able to provide oxygen to the heart under a range of conditions by increasing its blood flow through dilation. Coronary arteries that are diseased with atherosclerosis may fail to do so by lacking the ability to normally dilate under conditions of increased need. Impairment of coronary blood

flow results from narrowing of the coronary arterial lumen by atheromatous plaques. Atheromatous plaques are usually present in the epicardial portions of the coronary arteries. The general pathological sequence of atherosclerosis is intimal smooth muscle proliferation, lipid deposition, and aggregation of platelets in the final development of a complex atheromatous plaque. Thrombosis formation on a coronary artery atheromatous plaque results in a myocardial infarction. Acute myocardial infarction, henceforth known as AMI, is the clinical manifestation of focus in this paper. However, additional manifestations of CHD include angina, arrhythmias, ischemic cardiomyopathy, and sudden death. Myocardial infarction refers to necrosis of heart muscle caused by inadequate blood supply as a result of severe atherosclerotic narrowing of one or more of the coronary arteries.

Clinical presentation of acute myocardial infarction

The textbook presentation of myocardial infarction (MI) describes a patient with the onset of substernal chest pain lasting longer than 30 minutes. The pain is often described as having a heavy object sitting on one's chest. The pain may radiate to the arms (usually left), the neck, or the jaw. High epigastric discomfort may be a manifestation of myocardial ischemia and dismissed as "indigestion." Thus, those "indigestion" cases that occur for an unusual or prolonged time deserve special attention. Accompanying symptoms may include diaphoresis, restlessness and anxiousness, shortness of breath, nausea, and vomiting. Also to note, is the range of pain that myocardial infarction may present. Cases exist with, or without slight chest

discomfort. This is due to the differences in the neural sensory networks of individuals.

To complicate matters, painless myocardial infarctions are common, occurring in up to one-third of the cases (1). The incidence of silent myocardial infarction is greater in women and patients with diabetes mellitus, and it increases with age. A multicenter study showed that symptoms and signs that were predictive of AMI in younger patients (i.e., pain quality) were less helpful in the evaluation of the elderly (2). The study enrolled patients that were evaluated for acute chest pain in the emergency departments of 7 hospitals. The relative risks of pressure-like quality of pain, substernal location, typical pattern of pain radiation and electrocardiographic evidence of ischemia or AMI were consistently closer to 1.0 for the male gender. Meaning, these classic features for the endpoint acute ischemic heart disease (i.e. AMI) among the elderly, were less likely to be predictive of an AMI. This study's data supports the hypothesis that diagnosis of an AMI is especially difficult in elderly patients.

Pathologically, an acute myocardial infarction classification involves two separate categories: transmural myocardial infarction and nontransmural myocardial infarction. The difference is the thickness of affected ventricular wall in the infarct. A transmural myocardial infarction involves more than 50% of the ventricular wall, whereas, a nontransmural myocardial infarction affects less than 50%. However, a clinician would distinguish an AMI as either a Q wave or a non-Q wave myocardial infarction, as opposed to transmural and nontransmural myocardial infarction. A Q-

wave, detected during an electrocardiogram (ECG), is a negative deflection caused by an abnormal ECG if an infarction has occurred. The interpretation of an ECG for a patient who has suffered a nontransmural infarction (non-Q wave infarct) is more difficult.

Besides pathological and clinical classifications, classes of indications for diagnostic procedures and therapeutic interventions have been defined by a Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures that was developed in 1980 by the American College of Cardiology and the American Heart Association. The following defined three classes will be referred to throughout this report (67):

Class I: Usually indicated, always acceptable, and considered useful/effective

Class II: Acceptable, of uncertain efficacy, and may be controversial

- a. Weight of evidence in favor of usefulness/efficacy
- b. Not well established by evidence, can be helpful, and probably not harmful

Class III: Not indicated, may be harmful

Looking more closely at the definitions of the individual classes, one would rightfully assume that Class II is the classification group where the procedural controversy originates. The established Task Force has only provided categories to be used as guidelines for which the physicians will then appropriately modify on an individual basis. These classes provide a foundation for which I will later fit CABG, PTCA, and tPA. However, as long as procedures are classified under *Class II*,

inappropriate and unequal distribution of the treatments available for acute myocardial infarction is possible with the uncertainty factor involved.

Chapter 2

EPIDEMIOLOGY OF CORONARY HEART DISEASE

Coronary heart disease mortality

Coronary heart disease (CHD) remains the number one cause of death in the United States among both men and women. The age-adjusted mortality rates of coronary heart disease increased steadily each year from early in the century to the mid-1960's. Since then, it has been declining. The age-adjusted mortality rate declined 42% between the peak year of 1963 and that of 1985 (4). Despite this decline, in 1990 there were 489,171 deaths attributed to CHD [ICD-9 codes 410-414] in the United States (236,574 women and 252,597 men) (5,6).

Reports indicate on the national level, that there is disparity in the decreasing mortality rates among subpopulation groups. As Figure 1 shows, the US coronary heart disease death rates decreased faster for white men than white women and blacks between 1976 and 1985 (7,8,9). The average annual decrease for 1980 to 1988 by race and gender in greater than or equal to 35 year olds was 3.7% for white men, 3.1% for black men, 2.9% for white women and 2.2% for black women (4). This finding on the national level is consistent on a smaller scale. A study, conducted with the residents of Worcester, Massachusetts as the study population (8), showed that the incidence of acute myocardial infarction decreased less in women than in men from 1975 to 1988, especially among the 25 to 54 and the 65 to 74 year olds.

A more recent study that examined the trends of mortality and morbidity due to CHD was the Minnesota Heart Study (10). It took a closer look at the trends in the second-half of the 1980's. The study population focused on 30 to 74 year olds consisting of 550,719 men and 576,690 women. The target population was from the Twin Cities metropolitan area that is predominantly white. Between 1985 and 1990, the age-adjusted rate of mortality due to CHD declined approximately 25% in both sexes. They measured both in-hospital and out-of-hospital deaths. As Figure 2 shows, the downward trend was seen in both in-hospital and out-of-hospital deaths for both sexes. However, in men, in-hospital mortality declined much more rapidly than out-of-hospital mortality, (9.9% as compared with 3.6% per year; $p < 0.001$). Women had a consistent decline in mortality in both in-hospital and out-of-hospital mortality, (between 5 and 6%).

Hospitalization for acute myocardial infarction

The national discharge rates for patients with acute myocardial infarction decreased between 1988 and 1990. The rates per 100,000 in 1988 were 524 for 45 to 64 year olds and 1,416 for greater than or equal to 65 year olds as compared with respective rates in 1990; 497 and 1,270. The Minnesota Heart Study reported a 12% increase among the men discharged with acute CHD between 1985 and 1990.

Whereas, the women remained about the same during this same time period. Two reasons have been postulated for the increased rate of CHD hospital discharges among men, when other measures of CHD show declines. The first explanation involves the coding expansion in the late 1980's of the ICD-9-CM code to describe previous care

for myocardial infarctions and the second explanation may be the effects of reimbursement (11). With the mortality rate declining, it is certainly possible to assume that the Minnesota's report of increasing discharge rates could be exaggerated with the help of new implemented coding and reimbursement effects.

Acute myocardial hospital case fatality rates

Nationally, the case fatality rates of acute myocardial infarction continue to decline for both sexes. However, reports have indicated that women, especially >70 year olds, have a higher hospital acute myocardial infarction case fatality rate (12,13) than men. In the Minnesota Heart Study, they measured survival after hospitalization for acute myocardial infarction. Overall, the results indicated a lower risk of death both at 28 days and within 3 years in 1990, as compared to 1985. For US hospitals during the 1988 to 1990 period, the in-hospital acute myocardial infarction case fatality rate was 10.1% in women and 8.4% in men aged 55 to 64 years and 14.9% in women and 12.9% in men aged 65 to 74 years.

Medical care

As medical advancements are made in upgrading the treatments and procedures for AMI, it is important to evaluate the trends in the utilization of medical care to assess whether the distribution is biased. Data from the National Hospital Discharge Survey for 1980, 1985, and 1988 to 1990 show a marked increase in the rate of cardiac catheterization and CABG at ages 45 to 64 and >65 years (14,15). However, before 1985, reports indicate that men aged 45 to 64 years underwent both procedures at equal or higher rates than men aged greater than or equal to 65 years. This increase in

utilization among the elderly indicates the greater tendency to diagnose and perform the procedures, (12) or the greater reassurance of the benefits of the procedures due to increased technological advances. Figure 3 shows how the estimated number of coronary angioplasty procedures steadily increased from 1985 to 1990. The recent national data indicate a disproportionate distribution of cardiac procedures among the females and blacks. In 1990, age-adjusted rates per 100,000 were as follows: for coronary angioplasty- men 165.7, women 66.2; for coronary bypass graft surgery-men 155.6, women 57.8; and for cardiac catheterization-men 512.4, women 292.6. And in 1990, the reported rates per 100,000 for blacks as compared to whites: for coronary angioplasty-whites 99.9, blacks 17.6; for coronary bypass graft surgery-whites 98.5, blacks 19.6; and for cardiac catheterization-whites 350.1, blacks 209.7. This disparity in utilization among subpopulation groups is yet to be fully explained. However, these discrepancies raise questions about differential benefits and availability among the groups.

The Minnesota Heart Study reported the utilization of the cardiac treatments and procedures between the two years; 1985 and 1990 (Figure 4). The frequency of administration of the various cardiac therapies largely increased, (as reported in the proportions of patients receiving them) in the following; thrombolytic therapy, (more than doubled; 13 to 30%); coronary angioplasty (5 to 21%); aspirin (27 to 81%); and heparin (53 to 75%). Whereas, only moderate declines were documented among patients given warfarin (20 to 14%) and beta-blockers (56 to 50%). And little change was documented in the use of bypass surgery (8 to 10%).

As previously indicated, the trends in the use of therapy for AMI are reflective of the decreasing mortality due to CHD. The next few chapters will describe in greater detail the kind of impact each cardiovascular procedure has made which is reflective of their increasing use. Each chapter will describe the development of each of the procedures and the recent technological advances that have been made that assure a more effective treatment with less contraindications.

Chapter 3

Thrombolytic Therapy

Thrombolytic therapy is a clot dissolving therapy. Intravenously injected, streptokinase (SK [Streptase/Kabikinase]), recombinant tissue-type plasminogen activator (rt-PA [Activase]) or anisoylated plasminogen streptokinase activator complex (APSAC [Anistreplase]) can dissolve the clot and restore blood flow, interrupt the infarction, reduce myocardial necrosis, and improve the survival rate if administered within six hours of onset of an acute myocardial infarction. Approximately 66% of heart attack victims at hospital entry are prime candidates for thrombolytic therapy, given that an occlusive coronary clot caused the attack (verified with ST segment elevation) (3).

Streptokinase

Streptokinase (SK) was first described in 1933 when it was used in the canine model. It was not investigated in humans until 1949, and was FDA (Food and Drug Administration) approved for use in myocardial infarction patients in 1987. It systematically works in catalyzing the conversion of plasminogen to plasmin, which then stimulates the conversion of fibrin to fibrin degradation products (FDPs). These FDPs are responsible for dissolving the thrombus. These FDPs act as an anticoagulant, thus preventing subacute vessel reclosure.

Reductions in mortality of acute myocardial infarction patients, with the use of SK, has been observed in randomized control trials. One of these trials that studied the

use of SK, was the GISSI trial (Gruppo Italiano per lo Studio della Streptochinasi nell' Infarto Miocardico) (30,31). The study enrolled 11,712 patients who were randomly assigned to the treatment group, (treatment with a 1-hour intravenous infusion of 1.5 million units of SK), or to the control group. All the patients enrolled in the GISSI study were within 12 hours of the onset of acute myocardial infarction and free of contraindications to thrombolytic therapy. The study results proved that the earlier the treatment was started, the more effective it was. For those treated within the first hour of symptom onset, reduction in mortality was 47%, as compared to patients treated within 3 to 6 hours where there was a 17% reduction. In the 0 to 3 hours treatment group, there was a 23% reduction. However, there was no benefit for those patients treated after 6 hours. The 21-day mortality rate, as compared to the control group was reduced by 18%, (10.7% as compared to 13.0%; $p=0.0002$). Additionally, the 1-year mortality rate paralleled that of the 21-day mortality, indicating long-term benefits (30,31).

Another large randomized trial, which confirmed the results of the GISSI study, is the ISIS-2 (the Second International Study of Infarct Survival) (32). A total of 17,187 patients, of no age limit, who were within 24 hours of onset of acute myocardial infarction were enrolled in the study. They were randomly assigned to one of four groups: intravenous streptokinase (1.5 mU over 60 minutes), oral aspirin (160 mg/day for 1 month), to both, or to neither. The 5-year mortality rate experienced by the SK group versus the placebo group was a 23% reduction. This was similar to that seen in the GISSI study; a 18% reduction. In addition, the ISIS-2 results also

concluded that the earlier the treatment, the more effective it was. The observed 5-week mortality at the various times of administration confirmed that the greater reduction was seen during the earlier times. A 32% reduction was observed in those treated within 4 hours, a 13% reduction in those treated between 4 and 12 hours, and a 19% reduction in those treated between 12 and 24 hours. In patients treated within 1 hour, the 5-week mortality rate was reduced by 42% in the SK group. This was similarly observed in the GISSI study, who observed a 47% reduction at 3-weeks. Thus, these two trials, GISSI and ISIS-2, were consistent in their observations and conclusions that show the earlier the administration of the therapy, the more beneficial.

In addition to confirming the results of the GISSI study, the ISIS-2 study provided strong evidence for the additive effect of aspirin on mortality in patients with infarction. There was a reported 21% reduction in mortality by 5 weeks for those who received aspirin as compared to those who received the placebo ($p < 0.00001$). A comparison was made between those patients who received both the SK and aspirin and those who received the placebo, and a 39% reduction in mortality was observed at 5 weeks ($p < 0.00001$) for those in the SK/aspirin treatment group.

Recombinant tissue-type plasminogen activator

Tissue plasminogen activator (t-PA) was first described in 1947, and in 1981 it was synthesized by recombinant DNA technology and administered to nonhumans as recombinant tissue-type plasminogen activator (rt-PA) (107). It was not until 1987 that rt-PA became FDA approved for use in acute myocardial patients. As a second generation agent, rt-PA is considered to have clot specific action which has potential

advantages over the functional capabilities of SK. Because of this, rt-PA's local action activates plasminogen, attached to fibrin, and bypasses the systematic action of SK.

Reductions in mortality of acute myocardial infarction patients have also been demonstrated with the use of rt-PA. The major survival trial for rt-PA is the Anglo-Scandinavian Study of Early Thrombolysis (ASSET) (33). The study population was made up of 5,011 patients, excluding > 75 year olds, who were within 5 hours of onset of suspected myocardial infarction. The enrollees of the study were randomized to either the treatment group or the control group. The treatment group received 100 mg of rt-PA, (10 mg bolus injection, 50 mg in the first hour, 20 mg in the second and third hours) and the control group received a placebo. The overall mortality rate showed a 26% reduction ($p=0.0011$) at 1 month; the rt-PA assigned group had a 7.2% mortality rate as compared to the 9.8% for the placebo group. This led investigators to believe that the superiority of rt-PA over SK was not reflective in mortality rates.

The European Cooperative Study (35) randomly assigned 129 patients to either the rt-PA or the SK group. Both groups received the treatment intravenously after about 3 hours of symptom onset. The study demonstrated that at approximately 90 minutes, the patency rate for the rt-PA group was greater than that for the SK group; 70% for the rt-PA group and 55% for the SK group ($p=0.058$). A second trial conducted to compare the two thrombolytic agents was performed by the investigators in the National Heart, Lung, and Blood Institute Thrombolysis in Myocardial Infarction (TIMI) study (36). They observed a patency rate of 70% for the rt-PA group and a 43% rate for the SK group after 90 minutes of symptom onset. Both studies

demonstrated the same patency rate for those that received rt-PA, and a slight difference in patency rates for the SK group; 55% in the European Cooperative Study and 43% in the TIMI study. In addition, both studies demonstrated that the dissolving of elements of the clotting system was less marked with rt-PA as compared with SK. However, both rt-PA and SK had similar hemorrhagic complications, which were largely due to hematoma formation at the arterial catheterization site.

A second trial conducted by the European Cooperative Group (108), compared the rt-PA with a placebo. It was a randomized, double-blinded controlled trial that enrolled 721 patients, with chest pain and ST segment elevation, that were within 5 hours of symptom onset. The treatment group was given 100 mg of rt-PA over 3 hours and the control groups received a placebo. Both randomized groups received aspirin and heparin. The assigned primary endpoint of the study was detectable differences in left ventricular function. Mortality at 14 days and 3 months were secondary endpoints. At 14-days, patients randomized to rt-PA showed a 51% mortality reduction (NS, $p=0.06$) as compared to the placebo group. The ejection fraction was higher in the rt-PA group, (2.2 ejection points higher) as compared to the placebo group. In addition, the study demonstrated a 20% reduction of infarct size in the treatment group as compared to the placebo group ($p=0.0018$).

Both of these trials did not report statistically significant reductions in the mortality of rt-PA treated patients; however, it was suggested that the size of their study population could be the contributing factor (34). Thus, to determine without any

speculation which agent, SK or rt-PA, is the more beneficial thrombolytic agent, several trials have been conducted.

One of the largest studies to make a comparison between SK and rt-PA is the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO) trial. GUSTO was first presented in May, 1993 and then published (37) with a subsequent substudy (38). The GUSTO trial randomized a total of 41,021 patients from 1,081 hospitals. All patients were within 6 hours of chest pain onset and all had electrocardiographic evidence of infarction. This 26-month study reported on the 30-day mortality (the indicated primary endpoint), 24 hour and 1 year mortality, in-hospital clinical events, and 30 day net clinical benefit (% of patients who were alive at 30 days and free of an in-hospital stroke). All patients received 160 mg of oral aspirin on the day of the myocardial infarction and were given 160-325 mg daily thereafter. The patients were randomly assigned to one of four treatment groups: (1) accelerated t-PA with intravenous IV heparin; (2) IV t-PA plus simultaneous SK with IV heparin; (3) SK plus IV heparin; (4) SK plus subcutaneous heparin. The results from the GUSTO trial strongly indicate that enhanced thrombolysis with accelerated dosing of rt-PA + IV heparin is directly associated with superior early angiographic patency, improved left ventricular function, and reduced 30-day mortality compared to SK with subcutaneous or IV heparin, or the combination of rt-PA plus SK with IV heparin.

One factor probably related to the mortality that was observed in the GUSTO trial was the speed in which the therapy was administered (37). The 30-day mortality

increased as the administration time of the accelerated t-PA increased from symptom onset. The mortality was 4.3% who received the treatment within 2 hours after chest pain, 5.5% for those within 2 to 4 hours, and 8.9% for those between 4 and 6 hours. The elderly patients (≥ 75 years) had a 30-day mortality rate of 20.6% in SK groups as compared to 19.3% in the accelerated t-PA group (37). The GUSTO investigators concluded that the accelerated t-PA will save 10 more lives than SK for every 1,000 patients receiving thrombolytic therapy. The GUSTO investigators believe the mortality difference between the two thrombolytic agents is related to earlier infarct vessel patency in the t-PA group.

The angiographic substudy of 2,400 patients examined patency rates at 90 minutes, 180 minutes, 24 hours, and 7 days after thrombolytic administration (38). There was a significant difference in patency rates at 90 minutes between the four treatment groups. The accelerated t-PA group had a patency rate of 81%, as compared to the t-PA with SK group (73%), the SK with IV heparin group (60%), and the SK with subcutaneous heparin group (54%). However, there was no significant difference in patency rates between groups at 180 minutes, 24 hours, and 5 to 7 days.

Anistreplase

Anistreplase (APSAC) is one of the new thrombolytic agents, also classified as a second generation agent like rt-PA. APSAC functionally works in the same way as rt-PA, by activating plasminogen preferentially on the surface of the clot rather than in the general circulation as SK works. APSAC was FDA approved in 1990.

Many studies have been conducted to assess the patency rate of APSAC, and have found similar rates as that of SK. Bonnier et al. (39) found a patency rate of 64%, which is similar to that found with SK (68%). Bassand et al. (34) demonstrated a 77% patency rate and a 31% reduction in myocardial infarction size with salvage of left ventricular systolic function.

A group from the United Kingdom, the AIMS Trial Study Group, conducted a multicenter, double-blinded controlled trial to study the efficacy of APSAC (109). The 1,004 patients were randomly assigned to the treatment group (30 units of APSAC intravenously over 5 minutes) or to the placebo group. The patients enrolled in the study were <70 years of age and between 30 minutes and 6 hours of symptom onset. They reported a 30-day mortality reduction of 47% of those enrolled within 4 to 6 hours of symptom onset. They reported that there was a greater mortality reduction among those patients entered <4 hours. The 1-year mortality reduction was 44% ($p=0.0006$); 19.4% in the treatment group as compared to 10.8% in the placebo group. APSAC has presently proven to be a viable thrombolytic agent. It joins SK and rt-PA as a useful therapeutic alternative.

Contraindications to thrombolytic therapy

The major side effect to thrombolytic therapy is hemorrhage. Additionally, the Task force has provided a list of absolute contraindications to thrombolytic therapy (3). The task force also provides relative contraindications that should be considered on a case by case analysis of risk versus benefit. (See ACC/AHA Task Force on the

Management of Acute Myocardial Infarction for those) (3). The relative contraindications include:

1. Active internal bleeding.
2. Suspected aortic dissection.
3. Prolonged or traumatic cardiopulmonary resuscitation.
4. Recent head trauma or known intracranial neoplasm.
5. Diabetic hemorrhagic retinopathy or other hemorrhagic ophthalmic condition.
6. Pregnancy.
7. Previous allergic reaction to the thrombolytic agent (SK or APSAC).
8. Recorded blood pressure >200/120 mm Hg.
9. History of cerebrovascular accident known to be hemorrhagic.

Thrombolytic therapy in older patients

I have provided evidence of a steep increase in case fatality rate with age.

Nearly 50% of all deaths in patients hospitalized for acute infarction occur in those >75 years of age (13). Because of the fear of hemorrhaging, most trials, with the exception of ISIS-2 and GISSI have excluded patients either >70 or >75 years of age. The ISIS-2 study reported a greater mortality reduction at 5-weeks in the younger cohorts; however, a mortality reduction was still observed among the treatment group in the eldest cohort. The results were as follows: a 16% reduction (18.2% versus 21.6%) in the SK group >70 years of age; a 26% reduction (10.6% versus 14.4%) in the SK group 60 to 69 years of age; and a 28% reduction (4.2% versus 5.8%) in the SK group <60 years of age.

The GISSI study observed the same decreasing trend in mortality reduction as the patient population age increased. The GISSI trial noted a 13% reduction in mortality at 3 weeks in those >75 years of age, an 8% reduction in those 65-75 years, and a 26% reduction in those ≤ 65 years. However, the results in the oldest two

patient population cohorts were not statistically significant than that of the reference group for mortality . These studies demonstrated that the weakened affect of SK with increasing age needs to be carefully evaluated when recommending thrombolytic therapy for the elderly population. Recommendations have been provided by the Task Force for administration of thrombolytic therapy to patients with myocardial infarction and without contraindications to the therapy. (See Appendix E).

Distribution by demographics

Thrombolytic therapy is currently used in the United States for only a minority of AMI patients. Abiding by strict inclusion and exclusion criteria, as defined by the current recommendations, have resulted in the underuse of this medical therapy. This is reflective of current estimates that show only 10% of AMI patients in the United States actually receive thrombolytic therapy (115). There are four major medical reasons that AMI patients are not treated with thrombolytic therapy (116): advanced age, nondiagnostic electrocardiogram (ECG), specific contraindications, and excessive delay to treatment.

Advanced age is of particular interest in this study. AMI is the leading cause of mortality in the elderly, and more importantly, the number of elderly patients is projected to increase in the future (116-119). Yet these patients have not been consistently included in the pool of patients that receive thrombolytic therapy, because of fear of the increased risk of hemorrhage from this medical treatment. The increased hemorrhagic risk has not been in all the thrombolytic trials among the elderly. For example, the ISIS-2 study included over 400 patients that were over the age of 80

years, and still there was no significant bleeding complications reported as a result of thrombolytic therapy administration (32). Nevertheless, the efficacy of thrombolytic therapy in the elderly remains unproven, however pooled data suggest possible benefits (115,120-123). For example, the absolute number of lives saved is greater among the elderly group of patients, because of their elevated mortality without thrombolytic therapy (32,115,123).

Demographic, procedural, and outcome data were collected from 1,073 US hospitals on AMI patients during 1990 and 1993 (124). This data comprises the National Registry of Myocardial Infarction (NIMI) Registry. Registry hospitals composed 14.4% of all US hospitals. Among the 240,989 AMI patients enrolled in the study, 84,477 (35.1%) of them received thrombolytic therapy. Overall, the patients to receive the therapy were younger, more likely to be male, presented sooner after onset of symptoms, and were more likely to have localizing ECG changes. A trend analysis from 1990 through 1993, shows that the time from hospital evaluation to administration time of thrombolytic therapy is shortening. The national registry confirms on a large scale what the smaller studies conclude, that thrombolytic therapy is underused among the elderly patients, as well as the late presenters.

In summary, thrombolytic therapy is the medical treatment for AMI patients that is underused among certain subgroup populations. We know that approximately 66% of heart attack victims at hospital entry are prime candidates for thrombolytic therapy, however, only approximately 10% of AMI patients in US hospitals actually receive thrombolytic therapy. Despite the strict inclusion and exclusion criteria as

recommended for administration of thrombolytic therapy, the benefits especially that for the elderly, outweigh that of hemorrhagic risks.

Chapter 4

Cardiac Catheterization

Of the patients surviving to hospital admission, approximately 30% admitted with myocardial infarction will require revascularization within the first 30 days (16,17). With the introduction of thrombolytic agents to treat acute myocardial infarction, the post-infarction evaluation and management have become increasingly complex. Cardiac catheterization is the means used to determine if a patient has responded to thrombolytic therapy. Should the infarct-related artery still be occluded after therapy, the use of catheterization will be important in deciding whether additional cardiovascular procedures should be performed. However, controversy has been raised as to the routine use of cardiac catheterization. An additional important use of catheterization is for diagnostic purposes. Both purposes will be discussed in more detail, but I will first review the development of cardiac catheterization.

History

In 1929, Werner Forssmann was the first to establish cardiac catheterization (18). His interest was in injecting drugs directly into the right atrium. He performed the first cardiac catheterization on himself. Forssmann's motivation spread, and his technique was soon adopted by others. Klein, Cournand, Richards, and others used his technique for studying the physiology of the human circulation (18). The most famous example of this, is the work of Swan and Ganz, who used a catheter to obtain useful physiological measurements. The cardiac catheterization was first used for diagnostic purposes by James Warren, Emmett Brannon, and Heinz Weens. They used the

catheter to diagnose the atrial septal defect, for which the description was published in 1945 (19).

After the creation of cardiac angiography, the right side of the heart was an easy target, while the left side remained a difficult one. Zimmerman, Scott, and Becker were responsible for initiating the development of a technique that allowed for the visualization of the left side. In the late 1940's, Zimmerman, Scott, and Becker performed the first catheterization in which the cardiac catheter passed from the aorta into the left ventricle (20). It was not until Mason Sones in 1958 introduced selective coronary arteriography using the brachial approach (21), that diagnostic and therapeutic work on coronary disease would be possible. Melvin Judkin and others modified his approach, which lead to further developments, such as percutaneous transluminal coronary angioplasty, which will be discussed later in this report.

Procedures

As indicated with the history of the cardiac catheterization, there are two techniques of coronary arteriography; the Sones technique and the percutaneous femoral technique. The Sones technique introduces the catheter by way of the brachial artery, whereas the percutaneous femoral technique uses the femoral artery. The latter approach was introduced in the 1960's and gained popularity. However, the Sones technique has the following advantages over the percutaneous femoral technique (22): (1) only one catheter is necessary to visualize both coronary arteries, the left ventricle, and aortacoronary bypass grafts; (2) this method can be used in patients with severe obstructive disease of the iliofemoral system; (3) the procedure can be done on an

outpatient basis; (4) the depth of insertion of the catheter tip into the left coronary artery can be better controlled; and (5) the catheter tip can be rapidly shifted from one coronary artery to the other. Specifically using the Sones technique, the mortality risk is no more than 0.1 percent (22).

Purposes

Cardiac catheterization has a dual purpose. It is a powerful diagnostic tool and additionally is important in assessing whether a patient has responded to thrombolytic therapy (23). The majority of physicians will agree that cardiac catheterization and coronary arteriography should be performed on patients who survive an acute myocardial infarction (24); however, indications remain controversial (25). Thus, guidelines have been recommended by the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (67). These guidelines are divided into three separate categories, depending on the time after a myocardial infarction has occurred that cardiac catheterization is to be performed: (1) during the initial 6 hours of myocardial infarction; (2) after the initial 6 hours up to but not including predischARGE evaluation; and (3) from immediate predischARGE up to 8 weeks after dischARGE. (See Appendix G). It has been agreed that the following are definite clinical indications for cardiac catheterization and coronary arteriography in the early post infarction period: recurrent ischemia, persistent moderate to severe left ventricular dysfunction, and uncontrollable ventricular tachyarrhythmias (24,25,3,26,27).

The most important use of cardiac catheterization is for diagnostic purposes. Used as a diagnostic tool, cardiac catheterization provides valuable information of anatomic and physiologic change in many cardiovascular diseases. The following are the measurements that can be made using a cardiac catheter (28): (1) pressures in various cardiac chambers and blood vessels; (2) pressure gradients across stenotic cardiac valves; (3) cardiac output; (4) systemic and pulmonary vascular resistances; (5) hemodynamics during stress (eg, supine exercise); and (6) shunts between systemic and pulmonary circulations.

The important role for cardiac catheterization is providing precise anatomic and physiologic details of the cardiac abnormality in patients who are being evaluated for cardiac surgery. During catheterization, contrast material is selectively injected to assist in diagnosis. This material is helpful for defining the anatomy of coronary arteries and various congenital heart diseases, quantifying valvular regurgitation, and calculating the chamber volume, particularly the left ventricular end-diastolic and end-systolic volumes, and the ejection fraction (28). Additionally, for patients who suffer with multi-vessel disease, cardiac catheterization will be helpful in revealing unsuspected abnormalities or misjudged severity of the disease.

Routine use of cardiac catheterization remains to be a topic of controversy. Since there are currently no noninvasive techniques that allow visualization of the complete coronary artery circulation, it is recommended that each patient after an acute myocardial infarction should have selective coronary arteriography (29). However, on the other hand, this recommendation extends to warn that it is not practical to perform

a routine cardiac catheterization on every patient (29). Routine cardiac catheterization and coronary arteriography may be performed unnecessarily in some cases. These cases include patients who may have no evidence of myocardial ischemia and who are at low risk of recurrent cardiac events (29). This is an important reason to promote identification of high-risk patient subsets before any cardiovascular procedure is performed. Many recent technological advances have been made since the development of cardiac catheterization. However, coronary arteriography continues to set the standard for diagnostic measurements and acts as the guide for further cardiovascular care.

Distribution by demographics

Cardiac catheterization is a low-risk procedure performed for the purposes of gaining information on coronary artery anatomy and physiologic abnormalities in order to assess for possible surgical intervention. Not all patients with cardiac symptoms need this procedure, nevertheless, it is most often used on a routine basis. Clinical judgment is key when patients do not clearly meet the strict criteria as recommended by the ACC/AHA guidelines. This judgment in some cases has been shown to result in unequal distribution of cardiac catheterization among certain subpopulation groups.

Men seem to have cardiac catheterization ordered at a rate disproportionately higher than women. A study conducted in 1987 (125), enrolled 390 patients all of which had abnormal exercise radionuclide scans. The results of administering cardiac catheterization between the sexes was alarmingly disproportionate; 40% of the males as compared to 4% of the females were referred for cardiac catheterization. Additionally,

once the researchers controlled for variables of abnormal test results, age, types of angina, presence of symptoms, and confirmed previous myocardial infarction, men were still 6.5 time more likely to be referred for cardiac catheterization than women.

Racial differences in the use of invasive cardiovascular procedures have also been explored. One study in particular, analyzed the used of cardiovascular procedures among black and white male veterans discharged from Veterans Affairs hospitals with primary diagnosis of cardiovascular disease or chest pain during 1987 through 1991 (126). The study concluded that even when financial incentives were absent, whites were more likely than blacks to undergo invasive cardiac procedures. After they adjusted for all the potential confounders, they found that white veterans were more likely than black veterans to undergo cardiac catheterization (odds ratio, 1.38; 95% CI, 1.38 to 1.64).

However, this racial difference for administering cardiac catheterization was not confirmed on the national level. As results from the MITI registry, admitted to 19 hospitals in metropolitan Seattle were a total of 641 blacks and 11,892 white patients with chest pain of presumed cardiac origin since 1988 (127). Black men and women were younger (58 vs. 66 years; $p < .0001$), more often admitted to central city hospitals ($p < .0001$), and developed evidence of AMI less often (19 vs 23%; $p < .01$). During hospitalization, whites had higher rates of coronary angioplasty and coronary artery bypass grafting, although thrombolytic therapy and cardiac catheterization were used equally among the two groups.

Additionally, age-related differences in the utilization of therapies post AMI have been investigated. In particular, a retrospective chart review was performed on all cases with a primary or secondary discharge diagnosis of AMI (110). The total of 771 charts that were reviewed came from two large community hospitals in Milwaukee, Wisconsin from July 1, 1990 to June 30, 1991. They concluded that a very high percentage of those older than 65 years of age received invasive tests and interventions. There was a high cardiac catheterization rate, approximately 77% of all patients, and did not decline until after the age of 75 years, after which it fell steeply.

In summary, according to the recommendation for performing cardiac catheterization, no specifically relate to demographics. These guidelines do suggest to be cautious when performing cardiac catheterization on patients with uncomplicated complete myocardial infarction in whom no acute mechanical or surgical intervention is contemplated. Nevertheless, cardiac catheterization is most often used on a routine basis.

Chapter 5

Coronary Artery Bypass Grafting

Assessing the comparative role and efficacy of coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PTCA) continues to be an issue. Several randomized studies concerning the comparability of these two procedures will be evaluated. Furthermore, addressing the impact that patient demographics have on the utilization of CABG will be of importance. Before examining these critical areas, let's begin with the development of the procedure, coronary artery bypass grafting.

History

The early 1970's marked the beginning of surgical revascularization methods for patients suffering from a thrombus (blood clot) in a coronary artery. This new method was an efficient means to promote normal blood flow to the heart muscle with low morbidity and mortality. Results of studies during this time, confirmed and supported the surgical method of revascularization. One study performed during this period reported that mortality was < 10% and 50% of the infarctions were eliminated in 11 patients from the Brigham Hospital (76).

It was not until the late 1970's that aggressive action was taken to perform surgery for acute evolving myocardial infarction. In fact, in the early 1970's, if the patient had had an uncomplicated transmural myocardial infarction, most clinicians felt that coronary bypass surgery was inappropriate for at least 6 weeks, unless there was

some complicating mechanical effect (77,78). The early results (77,78) were suggestive that speed of reperfusion was critical to decrease long-term mortality and to increase salvage of heart muscle. The results of the long-term investigation conducted in Spokane, Washington confirmed the critical time factor (79,80). This 13-year retrospective study consisted of 387 acute myocardial infarction patients, managed medically or surgically. The surgical (CABG) and medical groups had a statistically significant different rate of sudden cardiac death (7.4% & 17.5%; $p < 0.01$). In addition, the patients who underwent surgery during the late phase of an acute myocardial infarction had a mortality rate similar to that in the medically managed group. Those patients that did undergo the surgery within 2 hours of the onset of the acute myocardial infarction, the mortality rate was only 2%. This confirmed earlier studies that stressed the importance of performing the surgical procedure promptly.

Procedure

During a coronary artery bypass operation, a large vein or artery is removed and used during the procedure. Most commonly removed are short segments of the saphenous vein (in the leg), or alternatively removed are other large veins or arteries in the body, such as the internal mammary artery (in the chest). The removed segment of the vein is used to create an alternate passage for the blood flow to avoid the obstruction. One end of the vein is implanted in the aorta while the other end is connected to the coronary vessel beyond the place of obstruction. This new route, hence the name, 'bypass surgery,' will allow enough blood flow to reach the heart muscles to prevent chest pain, at exertion or at rest.

Outcome Events

CABG is not a curative procedure, rather it has proven effective in reducing the conditions associated with an acute myocardial infarction, particularly angina.

The Coronary Angioplasty versus Bypass Revascularization Investigation (CABRI) is a multinational, multicenter randomized trial comparing the two surgical procedures, PTCA versus CABG (88). This is one of many randomized trials which will be later explored, all of which confirm the results that patients in the PTCA group are more likely to have clinically significant angina after the procedure. In the CABRI study, the PTCA randomized group had a statistically higher risk of angina after one year ($RR=1.54$ [1.09-2.16], $p=0.012$) than the CABG randomized group. This effect was present in both sexes, but statistically significant only among the females. The return of angina is most prevalent of the postoperative ischemic events. The return of angina very early after surgery is primarily due to incomplete revascularization or early closure of grafts. And the return of angina occurring later is usually due to narrowing or closure of one or more grafts and/or the development of native vessel disease.

The surgery is complex and can vary drastically between patients, thus it is important to explore the possible outcomes. Survival after the coronary artery bypass graft operation has been the outcome variable from several reported studies.

Specifically looking at the data from the CASS (Coronary Artery Surgery Study) Randomized Trial, Alderman, et al. conducted a ten-year follow-up study on survival (81). About 98.5% of patients survived at least 1 month after the operation, and 98.1%, 94.7%, and 82%, survived 1, 5, and 10 years respectively, following the

operation. Studies using cardiac death as the outcome variable after the coronary artery bypass grafting operation have suggested that cardiac death depends on: the year in which the operation was performed given recent medical advances in recent years, the severity of the coronary disease, and the severity of the left ventricular dysfunction (82).

Patency rates for coronary artery bypass grafts are dependent on the grafted vessel. The highest patency rates are associated with the use of the left internal mammary (thoracic) artery to bypass proximal stenoses of the left anterior descending coronary artery. Loop et al. reported, after a 10-year survival of patients having the internal mammary bypass, patency rates were approximately 95% (83). However, the use of saphenous veins for the bypass develops disease, contributed to stenoses and occlusions. Reports have indicated variable patency rates for vein grafts in coronary artery bypass grafting. In some reports (84,85), only 50% to 60% overall remain freely open after the 10-year follow-up. While other studies suggest that the patency rate of vein grafts is dependent on the location of its anastomosed artery. It is important to note that the analysis done in this report does not take into account the specific artery used in the coronary artery bypass grafting.

Guidelines

In an era when options are available, The American College of Cardiology and the American Heart Association have designated a Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures, who then appointed a

Subcommittee to develop guidelines and indications for the coronary artery bypass operation (86). Defined are three classes for CABG indications:

**Recommendations for Surgery in the Early Management of Myocardial Infarction
Recommendations for Emergency or Urgent Coronary Bypass Surgery**

Class I

1. Failed angioplasty with persistent pain or hemodynamic instability.
2. Postinfarct angina with left main or three-vessel disease or where coronary angioplasty is not indicated, with two-vessel disease involving the proximal left anterior descending coronary artery or two-vessel disease and poor left ventricular function.

Class II

1. At the time of surgical repair of ventricular septal defect or acute mitral insufficiency.
2. Cardiogenic shock not suitable for angioplasty.

Class III

1. Where the available surgical mortality rate exceeds the mortality rate associated with appropriate medical therapy.

Distribution by demographics

The data from the National Hospital Discharge Survey for 1980, 1985 and 1988 to 1990 show a marked increase in the rate of coronary artery bypass grafting at ages 45 to 64 and > 65 years of age (5,15). During 1988 to 1990, rates for CABG were higher in men at age 65 years or older, than what was seen before 1985, with an equal rate between the two age groups. This indicates a greater tendency to treat the elderly with more aggression as compared to the younger patients. Additionally, as earlier indicated in Chapter 2, recent US rates for cardiac procedures were lower in women than men.

Despite the national rates of CABG disproportionately distributed among the genders, there are no apparent differences in 5 and 10 year survival rates between

males and females after successful CABG (128-132). Nevertheless, several studies have shown that operative mortality has been consistently higher in women than men (128,129,133,134), with a relative risk of death in women after CABG ranging from 1.46 to 4.84. Studies have tried to adjust for preoperative baseline differences between men and women. In conclusion, CASS investigators found that mortality differences between the sexes were as a result of differences in coronary artery size (135).

However, artery size is not a factor that is reason for gender differences in outcome in all studies. Another reason for the increased mortality in women found in some of the studies is possibly referral bias (106). In summary, the studies suggest that CABG outcome may depend more on patient size or coronary size and preoperative risk factors than on gender itself. The women's' longterm survival is similar to that of men, however, studies have shown that women do have a higher surgical mortality rate and less angina relief after CABG than men.

Additionally, studies suggest that there are substantial interracial differences in cardiac procedure rates. One study in particular that suggested just that was a study that obtained discharge data from the Massachusetts Health Data Consortium on all patients discharged from hospitals in Massachusetts during the 1985 fiscal year (114) . Despite that age and sex adjusted admission rates for white and blacks were similar, whites underwent more than twice as many coronary artery bypass grafts than blacks. More specifically, 3,131 coronary bypasses were performed on white patients as compared to only 35 coronary bypasses being performed for blacks. The white-black bypass rate was 2.27 ($p < .05$).

This disproportionate distribution among the races is reflective of the national data. Results from the MITI registry shows that black patients admitted to coronary care units in metropolitan Seattle were less likely to receive coronary bypass surgery as compared to the white patients admitted to the same care units. During the patients' hospitalization, whites had higher rates of coronary artery bypass graft surgery (10 vs 4%; $p=.04$) as compared to the blacks (127).

The Society of Thoracic Surgeons' National Cardiac Database was used in part to determine the changes in preoperative characteristics of patients undergoing CABG during the 10-year period of 1984 to 1993 (136). Data show that an increase of 2.5 years in age and decreases of 3% both in incidence of male patients and in incidence of first operation occurred during this decade. This means that CABG is being utilized more among the elderly patients and less among the male patients and first time patients.

The benefits of performing CABG on the elderly patients are not consistent among the several trials conducted. Additionally, the long-term benefits of CABG outweigh taking a less invasive route with less risks, such as medical therapy. Advancing age was a significant independent predictor of operative mortality in several studies (137-141). However, a study reporting only elective CABG in patients greater than 65 years of age had an operative mortality rate of only 1.6% (142). Additionally, the extent of coronary disease did not affect survival of elderly patients at 5 years in the CASS study (137) ($p=.08$) or at 10 years in the data from the Cleveland Clinic (139). In summary, a decreased risk of sudden death and a better prognosis for 10-year

survival than the normal population, adjusted for age and sex are jointly beneficial (143). There are significantly higher risks among the elderly patients, however, patient selection is currently individualized according to severity of symptoms, coexisting illnesses, and angiographic findings (143). Advanced age is considered an additional risk factor, but does not contraindicate operation.

In summary, CABG is becoming more widely disseminated among the patient population. Studies have proven that there is disproportionate distribution of CABG among certain subgroups, however, as national data indicate, these trends are becoming less as the years progress. Additionally, there is no need for concern that gender or race are factors in differential outcome when having had CABG. The long-term outlook are similar, in particular between males and females. And as indicated earlier, the benefits of surgery outweigh the risks among the elderly patients.

Chapter 6

Percutaneous Transluminal Coronary Angioplasty

Percutaneous transluminal coronary angioplasty (PTCA) has been rapidly growing in popularity, from performance on only a few cases in the 1977-1980 period to 133,000 performed in 1986 (40). PTCA is a surgical procedure commonly used for patients with single-vessel disease; however its use has been extended to treat patients suffering from multi-vessel disease. The United States experienced over the past decade a more than tenfold increase, with approximately 300,000 angioplasty procedures performed in 1990 (41). Despite the popularity of the procedure, studies have suggested that the utilization is not uniform across certain subpopulation groups, such as Kern et al.. (144), Holmes et al.. (145), and Hannan et al.. (146). To begin, a brief summary of the development and the procedure itself will be described.

History

After success with cadaver studies, Dotter found that dilation of localized stenoses without dislodging atheromatous plaques was possible. On January 16, 1964, Dotter performed the first transluminal dilation on a patient, an 82-year old woman. This woman had a 0.5 cm stenosis of the adductor hiatus (in the leg). Her only option was for leg amputation because she was not a candidate for reconstructive vascular surgery. Immediately following Dotter's dilation procedure, he felt distal pulses that were previously undetected in her lower leg. The woman was able to walk for the first time in 6 months; however she later died of congestive heart failure.

After Dotter's initial dilation procedure proved successful, he teamed with Judkin, and by November 1964, they performed 15 dilations on nine patients (42). The initial results were encouraging; the elimination of four amputations and six of the nine patients improved. They concluded that the best results were for those patients with small area stenoses, in contrast to poor results on blockages of long segments. The technique of transluminal angioplasty grew in popularity in Europe during the next decade, while clinicians in the United States remained skeptical. It was not until the invention of balloon catheters that the United States showed interest in transluminal angioplasty.

In 1964, Dotter and Judkin (42) saw a need to have a catheter that featured the ability to produce maximal dilation of a stenotic lesion especially for large vessels. Latex balloons were first tried, but proved ineffective (43). Revisions of angioplasty balloon catheters were underway. In 1973, Porstmann (44) developed a caged or "Korsett" balloon catheter, revised in 1974 by Dotter et al... (43). Neither balloon catheter, the caged nor the "Korsett" balloon catheter, were recommended because of the potential for excessive damage to the intima or the vessel wall. Finally in 1974, Gruentzig introduced a balloon catheter that proved successful. The new type of balloon catheter was capable of producing a rigid balloon that inflated to a preset diameter (4 to 8 mm) and produced a large radial force (3 to 5 atmospheres of pressure) (45,46). In 1977, he had performed the angioplasty using his balloon catheter on 200 patients; 136 with femoropopliteal disease (diseased vessels found in

the posterior region of the knee) and 41 patients with iliac disease (47). These 200 patients were recorded to have a 2-year patency rate of 70 percent (48).

The fear of acute coronary artery occlusion and infarction justifies the apprehension of implementing balloon angioplasty. Gruentzig et al... (48) first tried coronary angioplasty on animals and then dilated coronary arteries of human cadavers. Both attempts were successful using the new balloon catheter of a smaller size. Then on September 16, 1977, Gruentzig performed the first successful percutaneous transluminal coronary angioplasty (PTCA) on a human (49). The patient was a 38-year old man with 85 percent narrowing of the left anterior descending coronary artery.

Procedure

As indicated with the history of the development of coronary angioplasty, the procedure is an extension of diagnostic angiography. The procedure begins when a catheter is guided into the coronary artery from either the arteries in the leg, or less commonly, in the arm. Inserted into the guiding catheter, is a separate catheter; the dilating balloon catheter. This balloon catheter is a double lumen catheter used for the dilation; hence, the name 'double lumen catheter.' This catheter is used to inflate the balloon at the distal end and simultaneously used to inject solutions or measure pressure at the opposite end. Unique qualities such as the elongated and cylindrical structure of the catheter make it possible to travel the lumen of the coronary arteries. In addition, the flexibility of the catheter allows for the mobility through the branching of the coronary arteries by way of a wire. This flexible wire can travel the route of the vascular tree, making way for the balloon system to maneuver through the guiding

catheter into the stenotic coronary artery. The balloon is dilated when the surgeon centers the catheter in the stenosis. Dilation happens once the balloon is inflated in the atherosclerotic plaque (50,51). Dilation was originally believed to result in the compaction and redistribution of transluminal atherosclerotic plaque substance. However, more recent studies have shown that dilation occurs once intimal disruption and stretching of vascular media and adventitia happens (52).

PTCA use as the primary treatment for AMI entails the need to always have a cardiac surgical team available. For this reason, intravenous thrombolysis has become established as the first-line therapy when appropriate. However, thrombolytic therapy has also been administered to patients as an adjunct to PTCA. Thrombolytic therapy administered early to patients with acute myocardial infarction has been shown to decrease mortality. However, this decline in mortality is accompanied by instability of the recanalized vessel that feeds the area of the infarct. This instability probably results from the considerable residual stenosis in the majority of patients after successful thrombolysis (53,54,55). Additionally, after administration of one of the thrombolytic agents (tissue plasminogen activator), thrombolysis is incomplete 1.5 to 3 hours after infusion. And because some thrombolytic agents (tPA included) have been shown to cause platelet activation, it is possible to have an increase risk of hemorrhagic infarction when PTCA is performed immediately after thrombolysis. With the immediate use of PTCA, it may further predispose to platelet deposition (56,57).

Reports have indicated that the delayed use of PTCA following successful thrombolysis has proven beneficial in the following ways: by reducing the narrowing

of the luminal diameter in the underlying plaque, decreasing reocclusion, lessening the residual stenosis, improving coronary blood flow, and by promoting the recovery of myocardial function (58). However, the role and proper timing of PTCA after thrombolysis are still controversial topics.

Two main trials that have been able to provide specifics on the role and most beneficial time of PTCA administration following thrombolysis are the Thrombolysis in Myocardial Infarction (TIMI) Study (59) and the Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) Study (58). Both of these studies investigated the role of recombinant tissue plasminogen activator (rt-PA) prior to a PTCA. The TIMI study indicated that immediate performance of PTCA, compared with delaying the procedures for 18 to 48 hours, provides no advantage and may be harmful. These harmful events include emergency coronary artery bypass surgery, reinfarction, and required transfusions due to immediate bleeding.

The TIMI-II A Study was carried out at seven of the 50 TIMI hospitals. From April 1986 through September 1987, a total of 389 patients were randomly assigned to one of three treatment strategies after r-tPA treatment. The three treatment groups were: (1) immediate coronary arteriography followed by PTCA; (2) 18 to 48 hour arteriography with PTCA; (3) no PTCA unless required by evidence of spontaneous or provokable ischemia. The primary end point for TIMI II A was ventricular function at the time of hospital discharge. Of the 195 patients assigned to the immediate PTCA group, 84% of the attempts were judged to have shown improvement. Of another 194 patients that were assigned to the 18 to 48 hour PTCA group, 93% of the attempts

showed improvement. It was concluded from this trial that immediate PTCA is not required after administration of rt-PA to patients with acute myocardial infarction for beneficial results.

The TAMI study results confirmed results of the TIMI study. The TAMI study group concluded that in patients with initial successful thrombolysis, immediate angioplasty offers no clear advantage over delayed elective angioplasty. This multicenter randomized trial was performed to compare the efficacy of immediate coronary angioplasty after AMI, with that of elective angioplasty (7-10 days) in patients initially treated with intravenous tPA. The incidence of reocclusion was similar in the two randomized groups: 11% in the immediate PTCA group, and 13% in the elective PTCA group. This comparative study resulted in similar improvements in the two defined end points of the study: global left ventricular function and regional wall motion in the infarct zone. In view of the data, a conservative approach after thrombolysis seem indicated. The Task Force has classified the recommendations for angioplasty after intravenous thrombolysis (60) (See Appendix F).

The National Heart, Lung, and Blood Institute (NHLBI) defines a successful angioplasty as one in which a greater than or equal to 20% change in luminal diameter is achieved, with the final-diameter stenosis <50% and without the occurrence of death, acute myocardial infarction, or the need for emergency bypass operation during hospitalization. There are patient-related factors that influence the success rate of a PTCA procedure. Some factors that are associated with increased failure rate are

female gender, age > 65 years, unstable angina, congestive heart failure, chronic renal failure, left main coronary disease, and three-vessel disease (67).

Limitations

Restenosis

Despite the initial success of PTCA, restenosis is of concern, occurring in an average of 20% to 30% of cases within the first 6 months of the procedure (61,62,63,64). Possible contributions to the occurrence of restenosis are platelet adhesion and thrombus formation at the dilation site. These events can occur within one hour after arterial injury (65). If the initial PTCA is unsuccessful, successful revascularization during a second angioplasty occurs approximately 97% of the time (66). The National Heart, Lung, and Blood Institute Registry identified several factors associated with increased incidence of restenosis: male gender, Canadian Heart Classification III or IV angina (see Appendix D for definitions), and dilation of bypass graft (67). Additional factors associated with risk of restenosis include: proximal left anterior descending coronary artery stenosis, diabetes, smoking after PTCA, and multiple lesions in a single-vessel (68,69,63,70). Factors that have not been correlated with an increased incidence of restenosis include age, functional class, history of previous myocardial infarction, hypertension, serum cholesterol, presence of calcification at the site of dilation, morphological features of the lesion, inflation pressure, and medications taken at the time of discharge (60).

Abrupt reclosure

Another limitation of coronary angioplasty is the possibility of incomplete restoration of blood flow upon deflation. At present, 2% to 5% of patients undergoing PTCA will require emergency surgery due to damage done to the coronary arteries (60). As a result, the immediate assistance of a cardiac surgical team is always required. Studies associate a 6% operative mortality and a greater than 50% perioperative myocardial infarction rate following emergency bypass used to treat abrupt reclosure (71,72). With the technological advances and improvements with the balloon catheter, repeat balloon dilation is advantageous in treating abrupt reclosure.

At a hospital in Boston, 1,160 patients who underwent PTCA between December 1981 and December 1986 were enrolled in a study to assess factors that relate to the occurrence and management of abrupt reclosure (73). Abrupt reclosure, experienced by 54 patients (4.7%), developed during the dilation procedure in 43 patients (80%) and 9 to 13 hours following the PTCA in 11 patients (20%). Of the 43 patients that experienced abrupt reclosure during the procedure, 22 (51%) had successful redilation.

Despite the successful redilation, the patients' long-term conditions were not favorable. The health status of the 96% of patients that were discharged alive was obtained between 6 and 60 months. Patients with successful redilated arteries and patients whose reclosure was treated medically were 3 times more likely to have ongoing or recurrent angina than patients who underwent emergency bypass surgery.

However, with the exception of 1 patient who died suddenly, there were no late myocardial infarctions in the redilated group.

National Heart, Lung, and Blood Institute Registry

The National Heart, Lung, and Blood Institute (NHLBI) identified PTCA to be a worthwhile procedure (60). As a result, they sponsored workshops on the procedure, along with establishing an early registry to study the safety and efficacy of the technique. The initial PTCA Registry enrolled patients from September 1977 until September 1982. The registry provided a way to monitor the procedure during the beginning years of utilization. The registry defined strict inclusion criteria:

(1) single-vessel disease; (2) concentric, proximal, noncalcified coronary lesions; (3) recent onset of angina; (4) good left ventricular function; (5) failed medical management; and (6) candidacy for coronary artery bypass graft surgery (CABG).

Despite the strict inclusion criteria, more than 3000 patients were entered into the voluntary registry (74).

The results from the PTCA Registry were first tabulated in the beginning year of setup. The initial success rate was only 68%; failure defined by the inability to cross and/or dilate the lesion. Nearly 30% of the successful cases experienced restenosis of the PTCA site within 6 months of the procedure. In addition, 6.1% had an emergency CABG, 4.9% had a nonfatal myocardial infarction, and 1.3% patients with single-vessel disease died (68).

As a result of improved technique and technology (steerable catheter and revised balloon designs), PTCA has continued to be successful. Meier and Gruentzig (75)

reported that from 1978 to 1982, the success rate increased from 63 percent to 91 percent. In 1985, the NHLBI Registry continued monitoring PTCA among fourteen of the original NHLBI Registry centers after closure of the Early NHLBI Registry in 1982. From the Early Registry to the Late Registry, patient indications for PTCA extended to include patients with unstable angina and multivessel disease. As a result, the late registry had a higher prevalence of older aged patients than that of the early registry. The increase in the number of complex angioplasty in the late registry is reflective of an increasing number of patients with multiple lesions, total occlusions, bifurcation lesions, and prior bypass surgery. This cohort of complex angioplasty patients accounts for nearly 60% of the current PTCA population (67). Reports indicate that success rate increased from the Early to the Late Registry (68% to 91%) as the rate of complications declined. The occurrence of nonfatal myocardial infarction in the late registry was 4.3%, the need for an emergency coronary artery bypass graft surgery was 3.4%, and mortality was 1.0% (69).

Guidelines

In 1980, a task force was formed to provide recommendations on appropriate technology to use for diagnosis and treatment of patients suffering from cardiovascular disease. This task force is referred to as the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures. One technique of particular interest is that of coronary angioplasty. Guidelines were determined for percutaneous transluminal

coronary angioplasty (PTCA) in 1988 (3). Currently, the committee classifies indications for the application of angioplasty into three classes:

Recommendations for Primary Angioplasty of Infarct-Related Artery Only

Class I:

1. Patients presenting within 6 hours of onset of pain and who meet the criteria for thrombolysis but in whom thrombolytic therapy is clearly contraindicated and only if facilities and personnel are immediately available. This recommendation is operative only when data indicate a large amount of myocardium is at risk.

Class IIa:

1. Intermittent continuous pain indicating the possibility of “stuttering” infarction, especially if there are ECG changes, but without clear indication for thrombolytic therapy.
2. Within 18 hours of acute infarction in patients developing cardiogenic shock or pump failure.
3. Patients who have had previous coronary artery bypass graft surgery in whom recent occlusion of a vein graft is suspected.

Class IIb:

1. Patients with known coronary anatomy in whom thrombolytic therapy is not contraindicated, but who develop symptoms and ECG evidence of acute infarction in hospital at a time when rapid access to a catheterization laboratory with personnel experienced in performing expeditious angioplasty for acute myocardial infarction is available (completion within 1 hour).
2. Patients in whom thrombolytic therapy is not contraindicated who present within 4 hours of onset of symptoms of acute infarction at a facility where rapid access to a catheterization laboratory with personnel experienced in performing expeditious angioplasty for acute myocardial infarction is available (completion within 1 hour).

Class III:

This category applies to patients with acute myocardial infarction who do not fulfill the Class I or II criteria:

1. Patients with severe left main coronary artery disease with instrumentation of a more distal occluded artery may be hazardous.
2. Patients in whom only a small area of myocardium is involved, as evidenced by clinical data or previously known coronary anatomy.
3. Dilation of vessels other than the infarct-related artery within the early hours of infarction. (This may not apply to the patient in shock or pump failure.)

The committee also provides indications for angioplasty according to single-vessel coronary artery disease, symptomatic patients with angina pectoris with medical therapy and single-vessel disease, multivessel coronary artery disease, symptomatic patients with angina pectoris with medical therapy and multivessel disease, direct immediate coronary angioplasty for evolving acute myocardial infarction, and after acute myocardial infarction (see ACC/AHA Task Force Report on coronary angioplasty) (60).

Distribution by demographics

As earlier indicated, the use of PTCA has increased steadily over time. The United States use of PTCA has increased over ten-fold in the past decade, resulting in over 300,000 PTCA procedures performed in 1990. Despite the procedure's popularity, studies suggest that this increase may not be equal among certain subpopulation groups. For example, US rates for cardiac procedures were lower in women than in men. In 1990, for example, rates per 100,000 were 165.7 for men and 66.2 for women (147). Additionally, similar disproportionately low rates were previously reported for blacks as compared with white. In 1990, rates per 100,000 were 99.9 for whites and 17.6 for blacks (147).

The National Heart, Lung and Blood Institute (NHLBI) developed a registry to examine gender-related differences in PTCA. The first registry (1977-1982) showed that PTCA risk was higher and efficacy was lower in women. A second registry was developed from 1985 to 1986 to determine if women still had a worse effect after a PTCA than men. The registry collected 2,136 patients (546 women). They concluded

that women undergoing PTCA have a higher procedural mortality rate (2.6% vs. 0.3%; $p < .001$), in addition to having higher initial complications (29% vs. 20%; $p < .001$) as compared to men (148). However, this is explained in part due to women having a worse cardiovascular risk profile, such as more severe angina than men. Otherwise, the success rate (79%) and long-term prognosis after PTCA were similar between men and women. In summary, the female gender was an independent predictor of reduced success with angioplasty (148). In contrast, data from the Medical College of Virginia comparing results from before and after 1985 found no significant gender differences after 1985 (152).

It has been suggested that women have also been at higher risk of complications related to their older age and greater degree of concurrent illness, although few studies actually have adjusted for these discrepancies. As a result of this, data suggests that PTCA carry higher procedure morbidity in women with coronary disease. Nevertheless, long-term outcome suggests that PTCA is a beneficial intervention for women. However, with the increased complications at the time of PTCA performed among women, the procedure needs to be performed with great caution.

Interracial access to selected cardiac procedures for patients hospitalized with coronary artery disease have been evaluated, such as data from national registries. From the NHLBI Percutaneous Transluminal Coronary Angioplasty Registry, the clinical characteristics, in-hospital event rates, and 5-year follow-up results were examined with respect to race for 1985-1986 (153). A total of 2,015 patients (90.8% white, 3.6% black) were enrolled into the registry. Among the black patients, more

were women (50% vs 24%; $p < .001$) and the black patients were more likely to have multivessel disease (72% vs 48%; $p < .001$), hypertension (73% vs 45%; $p < .001$), and diabetes (23% vs 13%; $p < .05$). Additionally, clinical success rates were similar (76.3% for blacks and 79.3% for whites). However, because blacks had more vessels with disease, complete revascularization was achieved in only 26% of the black patients as compared to 44% among the white patients. After PTCA, there was no significant difference in major complications (death, myocardial infarction, or emergency bypass surgery) between the races. Finally, five-year follow-up indicated that the races did not differ in their outcome. There was no significant difference in mortality, myocardial infarction, coronary bypass surgery, or repeat PTCA.

PTCA has proven to be a feasible cardiovascular procedure among the elderly, with angiographic success rates at least 78% (144,149,150). Although, major complications were more frequent in the elderly patients undergoing PTCA. Hartzler et al. (151) reported a 5 to 7 fold increase in mortality in older patients, Kern et al. (144) reported a 19% mortality in elderly patients and Holt et al. (149) noted a 20% incidence of emergency or elective coronary artery bypass grafting in his series.

In summary, in assessing the benefits of PTCA among certain population subgroups, it is important to evaluate the short-term and the long-term outcomes. As indicated, the long-term outcome among the demographics are similar.

Chapter 7

Coronary Angioplasty Vs. Bypass Surgery

Patients suffering from severe angina now have two options for surgical treatment; coronary angioplasty (PTCA) and coronary bypass (CABG). As discussed in former chapters, guidelines will include recommendations to guide clinicians in using the most appropriate procedure for their patient. However, controversy still exists whether one procedure is more beneficial under certain circumstances, as opposed to another.

A meta-analysis conducted by Pocock, et al. (87) compared coronary angioplasty with bypass surgery by combining eight sizeable randomized trials. The trials include: Coronary Angioplasty versus Bypass Revascularization Investigation (88) (CABRI), Randomized Intervention Treatment of Angina trial (89) (RITA), Emory Angioplasty versus Surgery Trial (90) (EAST), German Angioplasty Bypass Surgery Investigation (91) (GABI), The Toulouse trial (92) (Toulouse), Medicine Angioplasty or Surgery study (93) (MASS), The Lausanne trial (94) (Lausanne), and Argentine Trial of PTCA versus CABG (95) (ERACI). Pocock and his colleagues combined the data comparing initial revascularization, of PTCA and CABG, in patients suffering from coronary artery disease. There were a total of 3,371 patients enrolled in the studies that were eligible for either treatment strategy, PTCA or CABG. The mean follow-up was 2.7 years. The trials differed in design, inclusion criteria, and exclusion criteria, (which may be reason for possible heterogeneity between the trials). Reported

results include mortality, cardiac death and myocardial infarction, additional non-randomized procedures, angina, and single versus multi-vessel disease.

The mortality results between the PTCA and CABG recipients gave no indication of a treatment difference (RR for PTCA:CABG 1.08 [CI 0.79-1.50]). Given the various follow-up lengths between trials, Pocock and colleagues calculated a mortality risk (per 100 patient-years of follow-up) beyond the first year of 1.22 for CABG and 1.04 for PTCA. Specifically looking at endpoints, cardiac death and non-fatal myocardial infarction, there was again no indication of a treatment difference for the first year in each study. Though reported during the initial hospital admission, the PTCA group had fewer infarcts. The total number of infarcts in the first year of follow-up in both groups was similar (135 in the PTCA group and 127 in the CABG group). Both CABG and PTCA groups reported having a much lower risk of cardiac death and myocardial infarction in the first follow-up year as compared to during subsequent follow-up years.

The need for additional interventions, once received the randomized procedure, was much greater in the assigned PTCA group, both at one year follow-up and subsequent years, compared to the group initially randomized to CABG. From the combined trials, 17.8% [95% CI 16.0-19.6%] of the PTCA group required CABG within a year following the initial PTCA and 33.7% [95% CI 31.3-35.7%] required at least one additional PTCA and/or CABG. In contrast, only 3.3% of those randomized to CABG required additional interventions during the first year. Again from the

combination of all trials, the reintervention rates were 1.8 (CABG) and 4.5 (PTCA) per 100 patient-years of follow-up.

In conclusion, those randomized to the PTCA groups were more at risk for reinterventions as compared to those randomized for CABG. However, the less severe reintervention risk rate between the two groups in subsequent years suggests that longer follow-up years could explain more. The longest follow-up time among the eight trials was 4.7 years. Thus, it is important to conduct a trial with a lengthy follow-up time to confirm or refute the findings that suggest the possibility that CABG patients could have a similar reintervention risk rate, as that of the PTCA patients several years after the initial procedure.

The studies differ in their prevalence rates of angina. At one-year follow-up, all trials had a higher prevalence rate among the randomized PTCA group as compared to the randomized CABG group (RR 1.56 [CI 1.30-1.88]). Statistics indicate that there is evidence of heterogeneity ($X^2=14.5$; $p=0.054$ at one year). The author assumes this because one of the trials, GABI, had similar angina rates for PTCA and CABG patients, along with recruiting patients with more severe baseline angina (Class 2 or greater) as compared to the other studies. (See Appendix D for angina classifications). Comparing the six trials that had 3-year data, their angina prevalence rates for the randomized PTCA group went from 1.93 [CI 1.50-2.48] at 1 year to 1.23 [CI 0.99-1.54] at 3 years as compared to those patients randomized to the control group.

In assessing the different outcomes for those suffering from single versus multi-vessel disease, the eight trials were combined. Of the eight trials, five strictly enrolled

multi-vessel disease patients, two trials strictly enrolled single-vessel disease patients, and one study included both. The mortality rate for both the PTCA and CABG groups at the first-year was lower in single-vessel disease ($p < 0.01$). Additionally, the endpoints, cardiac death and myocardial infarction, single-vessel disease patients had a lower risk if they were in the randomized CABG groups. Among the multi-vessel disease patients, there was no treatment difference for risk of cardiac death and myocardial infarction. The need for additional intervention (CABG and/or PTCA) once the patient received the initial randomized procedure, was slightly higher among the multi-vessel disease, however not significant (34.5% vs. 30.5%; $p = 0.2$). In addition, the single-vessel disease also had a slightly lower need for an additional CABG once received a PTCA (16.0% vs. 18.3% within one year). The last outcome assessed was prevalence rates of angina. Both at one and three years, the single-vessel disease had a lower rate of angina for both CABG and PTCA procedure groups ($p < 0.01$ for one year and $p = 0.2$ at three years).

Many conclusions can be drawn concerning the two surgical procedures, as indicated from the results of the meta-analysis. There was a low risk of death and myocardial infarction over three years, and neither method (CABG nor PTCA) proved more beneficial. The authors explain the low mortality rates were a reflection of the exclusion of the more severe cases because of their ineligibility for PTCA. The prevalence of angina was higher in the PTCA patients than in the CABG patients. The long-term comparisons (beyond 3 years) of the two procedures were unable to be made with this analysis and are left unknown. What is known is that among CABG patients,

over a 5-10 year period, saphenous vein graft occlusion may lead to a requirement for additional revascularization procedures (96,97). To avoid the higher risk of mortality and morbidity that accompanies a second bypass operation, PTCA is the initial revascularization procedure recommended. The analysis concludes that mortality, cardiac event, and angina prevalence rates were slightly lower for single-vessel disease patients as compared to multi-vessel disease patients. However, there is little evidence to justify a difference in treatment between the single and multi-vessel disease. This analysis only includes two trials that specifically enrolled single-vessel disease patients, so the authors warn not to put much merit on the fact that the single-vessel patients in the CABG group had a lower risk of cardiac death or nonfatal myocardial infarction than that of the PTCA group.

Chapter 8

Methods

Data Base

I performed a retrospective analysis for all patients discharged from a community hospital in Michigan with a diagnosis of acute myocardial infarction (AMI) during the years 1990 through 1994. I used abstracted discharge data supplied by the Medical Records and Coding staff at McLaren Hospital in Flint. The collection of patient-specific data, routinely performed by the Medical Records and Coding staff, is completed for all patients admitted to the community hospital. Fully trained staff members review the accuracy of the medical file of each patient as dictated by the attending physician. The data base, from which I conducted my analysis, included all diagnoses, major procedures, age, gender, race, insurer, marital status, date of birth, admission date, discharge date, discharge status, and the hospital identification chart number.

Patient Population

Using the coded discharge data, I identified a total of 2,685 abstracted charts and included them in the data set. Given readmissions, I performed analysis on the population universe of 2,659 patients; the first discharge of each patient was taken as the index admission. To assure confidentiality, I used coded identifiers for each patient. The original charts were not available; thus, the ability to match chart number with the patient was not possible. Using the clinically modified ninth revision of the International Classification of Diseases (ICD-9-CM) (98), I strictly included patients

with the principal diagnosis of acute myocardial infarction (ICD-9-CM codes 410 through 410.9). (See Appendix A & B).

Each patient in my data set was discharged from the teaching hospital with the primary diagnosis of AMI. In addition, the initial data set also included 1,072 other diagnoses. These diagnoses were abstracted from the patients' charts, as made by the physician. The following is a complete list of the possible ways that an AMI was coded: AMI, anterolateral, initial; AMI, inferolateral, initial; AMI, inferoposterior, initial; AMI, other anterior, initial; AMI, other anterior, subsequent; AMI, other inferior, initial; AMI, other inferior, subsequent; AMI, other lateral, initial; AMI, other lateral, subsequent; AMI, other site, initial; AMI, unspecified site, initial; AMI, unspecified site, subsequent; AMI, subendoinfarction, initial; AMI, subendoinfarction, subsequent; and AMI, true posterior wall, initial. (See Appendix A).

My data base did not include information on the patients' past hospitalizations for myocardial infarctions (fifth-digit classification), nor were the locations of the infarction consistently coded. As a result, I condensed the location and the fifth-digit classifications. After consulting with a cardiologist, I felt it was more beneficial to merge the individually coded AMI diagnoses into one to enhance the power, in addition to simplifying the analysis. I did not want to chance the possibility of not reaching statistical significance due to a small sample size as a result of individually grouping patient's infarct location.

Definition of Variables

The variables I considered as possible predictors of treatment included admission year, age, gender, race, insurance, and comorbidity. I classified race as either white or black and I eliminated the groups Asian, Pacific Islander, and Other. I excluded these groups based on their limited sample size; they only account for 0.4% of the total patient population. I merged age into five groups; 1 through 44 years, 45 through 54 years, 55 through 64 years, 65 through 74 years and 75 through 101 years. I originally classified insurance as private, medicare, medicaid, commercial (including Blue Cross, Blue Net and PPOM), HMO (Health Plus), and all other (worker compensation). However, because insurance is predetermined by age (medicare eligibility is 65 years of age), I merged insurance groups based on age. I combined the seven insurance categories and recoded accordingly for the 65 years and older population: all insurance and medicare. I again combined the seven insurance categories and recoded accordingly for the younger than 65 years old population: all other insurance, HMO, medicare/medicaid, and commercial.

I assessed for comorbidity by creating an index variable that totals certain diagnoses that were weighted according to the seriousness of the condition. I used the diagnoses that were abstracted from the patients' charts and condensed certain diagnoses into weighted assigned groups. These certain diagnoses were chosen as suggested in the Charlson, et al. article (100) on prognostic comorbidity in longitudinal studies. They assigned a value (weighted index) for each condition that a patient was diagnosed with during their hospital stay. The following are those weights and

conditions: 1 for myocardial infarct, congestive heart failure, peripheral vascular disease, cerebrovascular disease, dementia, chronic pulmonary disease, connective tissue disease, ulcer disease, mild liver disease, and diabetes; 2 for hemiplegia, moderate or severe renal disease, diabetes with end organ damage, any tumor, leukemia, and lymphoma; 3 for moderate or severe liver disease; and 6 for metastatic solid tumor and AIDS. I was able to locate the conditions as coded in my data set for the above conditions, except for diabetes with endstage organ damage and AIDS. Diabetes with endstage organ damage was not separately coded in my data set, however, I am confident that it was accounted for within the diabetes category; and none of the patients in my study population had AIDS.

After coding each of the conditions with their assigned weighted index, I created a variable (index) that totaled the weighted index for each patient. I then condensed the individual index values into three to have comparable cells. The three index values were coded accordingly: 0 for having a total weighted index of zero, 1 for having a total weighted index of one, and 2 for having a total weighted index of two or more. Meaning that patients were assigned a 0 for having no additional illnesses besides an AMI, a 1 for having an additional illness besides an AMI, and a 2 for having multiple and/or more severe illnesses in addition to an AMI.

Procedure Use

The original data base included 1,063 procedures (See Appendix B). The treatments of interest included cardiac catheterization (CATH), coronary arterial bypass

surgery (CABG), percutaneous transluminal coronary angioplasty (PTCA), and tissue plasminogen activator (tPA). Thus, I examined the use of cardiac catheterization (ICD-9-CM codes 37.21, 37.22, and 37.23), CABG (ICD-9-CM codes 36.11 through 36.16), PTCA (ICD-9-CM codes 36.01, 36.02, and 36.05), and thrombolytic therapy (ICD-9-CM code 99.29), while disregarding all other procedures that were performed on the patient during their hospital stay. (See Appendix C).

Statistical Analysis

In the primary analysis, I examined the utilization of the four cardiac procedures over the five-year study period (1990 through 1994), the distribution of the study population across age, gender, race, insurance, comorbidity, and admission year, in addition to the distribution of the four procedures across the study population. I performed identical age-stratified analyses for each of the four procedures; CATH, CABG, PTCA, and thrombolytic therapy. I constructed separate logistic regression models to determine the effect of race, gender, age, insurance, comorbidity, and admission year on the use of cardiac catheterization, percutaneous coronary angioplasty, coronary arterial bypass grafting, and thrombolytic therapy (101). Each of these regression models adjusted for the possibility of confounding by the variables previously listed. I also analyzed two-way interactions between variables. I reported odds ratios as the results of the models.

I chose to use the backward hierarchical elimination method when I performed my logistic regressions. I began with four saturated models with CATH, CABG, PTCA, and thrombolytic therapy as the dependent variables and admission

year, age, gender, race, insurance, and comorbidity as the independent variables.

Given insurance is predetermined by age, I chose to evaluate the role of insurance within two age-strata, (younger than 65 years and 65 years and older). This way, I was able to look at the relationship between insurance and the procedures without possible confounding due to age. For each procedure, I repeated the backward hierarchical elimination method within each age-stratum. For each age-stratified analysis (less than 65 years of age and 65 years and older), I determined what variables to keep in a model based on a 0.05 p-value. To assess effect modification, I created two-way interactions between each of these variables with the remaining independent variables that were not statistically significant. I ran each of these two-way interactions in a model with admission year, the main effect variables, and the interactions. In calculating the difference in the chi-square between the interaction term and a model without the interaction term, I was able to report on effect modification (based on a 0.05 p-value). To determine confounding in my models, I assessed a 20% difference between the unadjusted odds ratio and the adjusted odds ratio for admission year. After assessing potential confounding, I determined whether the univariates were important predictors based on the statistical significance when they were adjusted for with admission year in the models. I chose to include those good predictors that added precision to the final model. Personal judgments were made on individual cases of the inclusion of variables, despite statistical significance. The final models are presented for each age-stratum of the four procedures as odds ratios.

Chapter 9

Results

Patient Population

I identified 2,659 admissions with a primary diagnosis of acute myocardial infarction (AMI) at a community hospital during the study period. Table 1 displays the characteristics of the study population. The overall distribution of admissions during the study period remains relatively constant, increasing slightly over the five-years. Accounting for the greatest percentage of admissions is the age group 65 years and older; they accounted for more than half of the discharged each year (Figure 5). Male account for the majority of AMI admissions, between 58% and 67% of the total admission population (Figure 6). Figures 7 and 8 show the age-stratified distribution of admissions across select insurance groups. Figure 7 shows that the majority of my study population aged 65 years and older are receiving medicare. While Figure 8 shows just the opposite. The study population aged less than 65 years are largely distributed over private, commercial, and HMO insurance groups, not medicare. Figure 9 shows that across the entire study population, medicare makes up the greatest proportion of AMI admissions; between 53% and 60% of the total study population. However, this is because Figure 9 shows the distribution across all ages and as seen in Figures 7 and 8, insurance is highly correlated with age. The white race makes up the majority of the AMI admissions, between 93% and 96% (Figure 10). And accounting for the greatest percentage of admissions across the comorbidity indices, is the comorbidity index 0. Between 62% and 69% of the patients did not

receive another diagnosis that was weighted in the comorbidity index scale, besides their primary diagnosis of AMI (Figure 11).

Table 2 shows the age distribution of these patients by gender, race, insurance, and comorbidity. The 75-101 year cohort is the age group that accounts for the greatest percentage of admissions for the females; with the 65-74 year cohort accounting for almost as many female admissions. Whereas, the 65-74 year cohort accounts for the greatest percentage of admissions for males; with the 55-64 year cohort accounting for almost as many male admissions. The age distribution by the black race fluctuates unevenly throughout the study period for each age group. For example, the 55-64 age group accounts for 0% in 1990, 8.7% in 1991, 30.4% in 1992, 39.1% in 1993, and 21.7% in 1994. However, this is understandable given that the black race only accounts between 4% and 6% of the total study population. The youngest age group (1-44 years) accounts between 5% and 7% of the total admissions for the white population during the study period. And, as compared to the youngest cohort of the white population, the percentage of the total admissions quadruples for the oldest age cohort (75-101) of the white population. This reflects data indicating the increasing prevalence of AMI with increasing age. The majority of patients covered by commercial, private, and all other insurances are aged < 65 years. The majority of patients covered by medicare and medicaid are > 64 years of age. Those covered by HMO's are unevenly distributed between the two age cohorts (1-64 and 65-101) throughout the study period. Each of the weighted comorbidity indices are largely

distributed among the older cohorts and it increases with age because comorbidity and age are related.

Table 3 shows the gender distribution of these patients by age, race, insurance, and comorbidity. Accounting for the majority of admissions in all the age groups, with the exception of the 75-101 year cohort, is the male gender. Additionally, the male gender also accounts for the majority of admissions among both the white and the black race. Those covered by HMO's, medicare, and medicaid are almost equally distributed between the males and females, slightly favoring the male gender. Whereas, the males account for the larger proportion covered by private, commercial, and all other insurances. Additionally, the males account for the larger proportion in each of the weighted comorbidity indices, although it is more variable in comorbidity 2.

Table 4 shows the insurance distribution of these patients by age, race, gender, and comorbidity. Accounting for the greatest percentage of all admissions aged 1-64 years, is commercial insurance. Accounting for the greatest proportion of patients older than 64 years, black (except for the 1990 & 1993 admission years), white, female, male, and that have a comorbidity index 1 and 2 are medicare and medicaid. Whereas, accounting for the majority of admissions with a comorbidity index of 0 is almost equally distributed between medicare & medicaid and commercial insurance.

Table 5 shows the race distribution of these patients by age, gender, insurance, and comorbidity. Accounting for the greatest proportion of patients in all age, gender, insurance, and comorbidity groups throughout the study period is the white race. This is reflective of the fact that my study population is almost 95% white.

Table 6 shows the comorbidity distribution of these patients across age, gender, race, and insurance. The distribution of comorbidity across age is the following: accounting for the greatest percentage of admissions with a comorbidity index of 0 is the 1-44 year cohort and the 45-54 year cohort and accounting for the greatest proportion in the remaining age groups (55-64, 65-74, & 75-101) is the comorbidity index 1. The distribution of comorbidity across select demographics shows that the comorbidity index 0 accounts for the greatest proportion among males, females, blacks, whites, and all insurances except for HMO's. Also to note, is that the greatest percentage of admissions with a comorbidity index 2 are the older (75-101), black, female, and who have medicare & medicaid coverage.

Procedure Use

Thrombolytic therapy was performed during the days of the index admission in 509 patients. Figure 12 displays the distribution of the patient population that received thrombolytic therapy over the study period. The rate jumped from 8.8% in 1990 to 20.7% in 1991, where it remained constant during the remainder of the study period (odds ratios of undergoing thrombolytic therapy for a patient during 1990, 1991, 1992, and 1993 as compared with 1994, respectively, .3632 ($p < 0.01$), .9799 ($p < 0.01$), 1.1037 ($p = .4941$), and .9897 ($p = .9439$).

For cardiac catheterization, the corresponding number is 1,541 patients. Figure 13 displays the distribution of the patient population that received a cardiac catheterization over the study period. The rate is 50.2% in 1990, 50.5% in 1991, 51.4% in 1992, 66.9% in 1993, and 68.8% in 1994 (odds ratios of undergoing cardiac

catheterization for a patient during 1990, 1991, 1992, and 1993 as compared with 1994, respectively, .4552 ($p < 0.01$), .4622 ($p < 0.01$), .4788 ($p < 0.01$), and .9164 ($p < 0.01$).

Similarly, 534 patients received a CABG over the study period. Figure 14 displays the distribution of the patient population that received a CABG over the study period. The rate is 12.8% in 1990, 20.8% in 1991, 19.0% in 1992, 23.3% in 1993, and 23.7% in 1994 (odds ratios of undergoing CABG for a patient during 1990, 1991, 1992, and 1993 as compared with 1994, respectively, .4751 ($p < 0.01$), .8429 ($p < 0.2443$), .7527 ($p = 0.0527$), and .9769 ($p = .8679$).

For PTCA, the corresponding number is 692 patients. Figure 15 displays the distribution of the patient population that received a PTCA over the study period. The rate increased during the study period; 19.6% in 1990, 19.8% in 1991, 26.0% in 1992, 28.5% in 1993 and 34.5% in 1994 (odds ratios over the five years, respectively, .4623 ($p < 0.01$), .4678 ($p < 0.01$), .6689 ($p = 0.0021$), and .7586 ($p = 0.0322$). Many independent variables other than admission year, were associated with the use of the four cardiovascular procedures. These variables include age, gender, race, insurance, and comorbidity. Figures 16 through 35 display the distribution of the patient population grouped by age, race, gender, insurance, and comorbidity, that received each of the four procedures (cardiac catheterization, CABG, PTCA, and thrombolytic therapy). Figures 16 through 20 show the distribution of thrombolytic therapy across age, gender, race, insurance, and comorbidity. Additionally, Figures 16 through 20 show that the utilization rate of therapy jumped from the low in 1990 to a maintained

level for the remaining years across each group. The oldest cohort (75-101) was the age group that received thrombolytic therapy the least throughout the study period. Males received thrombolytic therapy more frequently than females in each of the years.

Figures 21 through 25 show the distribution of cardiac catheterization across age, gender, race, insurance, and comorbidity. Overall, the use of cardiac catheterization remained at a constant level for the first three years of the study, where it increased for the remaining two years. It is clear that males and whites received cardiac catheterization more often as compared to their counterparts.

Figures 26 through 30 show the distribution of CABG across age, gender, race, insurance, and comorbidity. With the exception of the distribution across the black race, the figures show a slight increase throughout the study period. Again, the males and the whites (except for the 1990 and 1991 admission years for the race distribution), received a CABG more frequently as compared to their counterparts.

Figures 31 through 35 show the distribution of PTCA across age, gender, race, insurance, and comorbidity. Overall, the procedure was used more frequently as the study years progressed. Again males and whites received a PTCA more frequently as compared to females and blacks. Also, the admissions in the medicare insurance group, received a PTCA less frequently as compared to all other insurance groups.

Statistical Analysis

I used a multiple logistic-regression model to examine the independent contribution of admission year, race, age, gender, insurance, and comorbidity for the use of each of the four cardiovascular procedures. Tables 7 through 14 present the

results of the logistic regression analyses that predict the performance of select cardiovascular procedures across each of the two age-strata (less than 65 years and 65 years and older). The tables provide the unadjusted and the age-adjusted, gender-adjusted, race-adjusted, comorbidity-adjusted and insurance-adjusted odds ratios for each age-strata. While independently controlling for race, age, gender, insurance, and comorbidity, admission year was a statistically significant predictor ($p < 0.01$) for the use of each of the four procedures across both age-strata. The tables provide the unadjusted and respective adjusted odds ratios across both age-strata. More importantly these tables confirm that there is no confounding of the use of any of the four procedures for either age-strata. Tables 15 through 18 provide the unadjusted odds ratios on utilization for each of the four procedures and adjusted by statistically significant predictors for other effects of admission year on utilization as the final model. For example, the odds ratios for undergoing each of the four cardiovascular procedures in 1990 through 1993 as compared to 1994 for both age-strata are reported.

For assessing potential effect modification, two-way interactions were created. Tables 19 through 26 provide the change in chi-square, degrees of freedom, and p-values from which I evaluated the interactions for inclusion into my final models. In some cases, the interactions proved to be statistically significant effect modifiers; however, the interactions did not remain statistically significant in the final models. Therefore, I had to make a judgment as to what variables I included in the final model.

Table 15 includes the odds ratios of the final model for thrombolytic therapy across each age-stratum. The final model for the use of thrombolytic therapy for the

65 year and older age-stratum includes the following variables: admission year, comorbidity, and age. I chose not to include the interaction between admission year and insurance and its main effects, because their prediction of thrombolytic therapy utilization was not statistically significant. More importantly, the inclusion of the variables did not change the time trends in the utilization of thrombolytic therapy. The final model for the 65 year and younger cohort includes the following: admission year and gender. I again chose not to include the statistically significant independent interaction between gender and race for the same reason previously mentioned. I did assess whether race was a statistically significant predictor as a main effect in the final model, and it did not prove statistically significant. Thus, the final model for the age-stratum includes just admission year and gender as the statistically significant predictors for the utilization of thrombolytic therapy.

Table 16 includes the odds ratios of the final model for the use of cardiac catheterization across each age-stratum. For the less than 65 year old cohort, no interactions were reported as statistically significant effect modifiers, thus the final model includes the following variables: admission year, comorbidity, and race. And for the 65 year and older cohort, the final model includes the following: admission year, age, comorbidity, and the interaction between comorbidity and age. The interaction between comorbidity and age is statistically significant and remained as such when included in the final model.

Table 17 includes the odds ratios of the final model for the utilization of CABG across each age-stratum. For the 65 year and older age-stratum, the final model

includes the following variables: admission year, age, and race. I chose not to include the interaction term between race and gender because it was not a statistically significant predictor once added to the final model. I did evaluate whether gender, as a main effect, would be statistically significant; however, once entered into the final model, it lost statistical significance. The final model for the less than 65 age-stratum, includes the following variables: admission year, comorbidity, gender, and the interaction between comorbidity and gender. I chose not to include the interaction term between comorbidity and insurance in the final model, because it was just barely statistically significant once added to the final model. And more importantly, the inclusion of this interaction term did not alter the time trends of admission year on the utilization of CABG. I did assess the possibility of an interaction between gender and insurance, and the interaction was not a statistically significant effect modifier on the utilization of CABG.

Finally, Table 18 includes odds ratios of the final model for the utilization of PTCA across each age-stratum. The final model for the 65 and older age-stratum includes the following: admission year, age, and comorbidity. No interaction terms were statistically significant among this age group. The final model for the less than 65 year age-stratum includes the following: admission year, age, and comorbidity. The interaction term between admission year and age was just barely statistically significant, thus I chose not to include this interaction term in the final model. More importantly, the inclusion of the interaction term did not alter the time trends of admission year on the utilization of PTCA.

The results of this age-stratified analysis confirmed that insurance and age are not confounders of each other. Thus, I additionally assessed the best predictors for the utilization of each of the four procedures across all ages, with the exception of insurance. Because insurance is predetermined by age, I chose to exclude this variable when determining the best predictors across all ages. I used a multiple-logistic regression model to examine the independent contribution of admission year, race, age, gender, and comorbidity for the use of each of the four cardiovascular procedures across all ages. Tables 27 through 30 present the results of the logistic regression analyses that predict the performance of select cardiovascular procedures across all ages. The tables provide the unadjusted odds ratios on the utilization of each procedure across all ages and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization. While independently controlling for race, age, gender, and comorbidity, admission year was a statistically significant predictor ($p < 0.01$) of the use of each of the four procedures. Again, with the exclusion of insurance, Tables 27 through 30 confirm that there is no confounding of the use of any of the four procedures by age, gender, race, or comorbidity.

Tables 31 through 34 provide the unadjusted and fully-adjusted odds ratios and 95% confidence intervals for each of the four procedures across all ages. With the inclusion of all the variables, admission year remains a statistically significant predictor for each of the four procedures. Additionally, the odds ratios for undergoing the four procedures based on select demographics are also displayed in Table 31 through 34. For assessing potential effect modification, two-way interactions were created. Tables

35 through 38 provide the change in chi-square, degrees of freedom, and p-values. For the utilization of thrombolytic therapy (Table 35), the interaction between admission and comorbidity was statistically significant. For the utilization of cardiac catheterization (Table 36), there were no statistically significant effect modifiers. Whereas for the utilization of PTCA (Table 37), the interaction between admission year and age was statistically significant. And finally, for the utilization of CABG (Table 38), there again were no statistically significant effect modifiers.

Chapter 10

Discussion

I found that for time trends, there was no effect modification by any of the other variables of age, race, gender, insurance, or comorbidity. These variables did not modify the effect of admission year on the utilization of thrombolytic therapy, cardiac catheterization, coronary artery bypass grafting, or percutaneous transluminal coronary angioplasty. Additionally, none of these variables acted as confounders for any of the four cardiovascular procedures.

However, for the different procedures, some of the above mentioned variables were independent predictors of utilization either as main effects only, or as interactions between themselves. Thus, the final models vary for the different procedures, for example, sometimes it was only gender that effected the significance and sometimes it was age. Nevertheless, the inclusion of these significant independent predictors and their interactions did not change my conclusion about the time trends with the utilization of a given procedure because they were neither effect modifiers, nor confounders for admission year.

My conclusions were based on the final models for each of the procedures for both age-strata. For a patient less than 65 years of age, they were less likely to receive thrombolytic therapy if admitted in 1990 or were female. This is reflective of the actual number of patients that received thrombolytic therapy during the study period. The use of this particular procedure increased in 1991, where it remained at a constant

level for the remainder of the study period. And the distribution of thrombolytic therapy was greater for the males throughout the study period; more so during the four remaining years of the study. For a patient 65 years and older, they were less likely to receive thrombolytic therapy if admitted in 1990. Again, this is reflective of the actual distribution of thrombolysis during the study period. As Figures 16-20 show, thrombolytic therapy was not widely distributed across all of the variables of interest during 1990. Additionally, patients were almost 2 times more likely to receive thrombolytic therapy if aged 65-74 years, as compared to 75 years and older, and were least likely to receive therapy if they had additional diagnoses and/or more severe conditions beside an AMI. Again, both of these results are reflective of the distribution of thrombolytic therapy during the study period.

My results for administration of thrombolytic therapy confirm current literature for the distribution of thrombolysis across age. As literature suggests, most of the major studies, with the exception of ISIS-2 and GISSI excluded patients greater than 70 or 75 years of age. Nevertheless, these two studies did reveal a greater mortality reduction among the younger cohorts; however, a mortality reduction was still observed among the treatment group in the eldest cohort. Additionally, the recommendations for administration of thrombolytic therapy to patients with myocardial infarction suggest that patients greater than 75 years of age (with specific medical conditions), could have harmful results if given this therapy. In summary, as literature suggests, elderly patients are more at risk if given thrombolysis.

Whereas my results concerning distribution of thrombolytic therapy across gender, do not agree with most of the larger studies comparing gender differences in outcome. My findings show that gender is a statistically significant predictor for the utilization of thrombolytic therapy among the youngest age-stratum (less than 65 years of age). As a result, a woman less than 65 years of age was less likely to receive thrombolytic therapy than a man of the same age-stratum. In general, studies reveal no significant difference in the pharmacokinetics of tPA or coronary artery patency rates in men vs. women (105). In addition, no difference is seen with the reduction in mortality rates in both sexes. Nevertheless, there are subgroup analyses that do conclude gender differences in mortality rates. However, gender was only a statistically significant predictor in the younger age-stratum in my findings. And differences in mortality among the genders is hypothesized to be due to the older age of women presenting with myocardial infarction. This is what is suggested to be contributors to their relatively lesser reduction in mortality after thrombolysis (106).

For a patient less than 65 years of age, they were less likely to receive a cardiac catheterization if admitted in 1990, were white, or had additional diagnoses and/or more severe conditions beside an AMI. This again is reflective of the distribution of the procedure during the study period. And for a patient 65 years and older, they were less likely to receive a cardiac catheterization if admitted in 1990, or had only 1 additional diagnosis beside an AMI. A patient aged 65-74 years was 4 times more likely to receive a catheterization as compared to a patient in the 75-101 year old cohort. This indicates that the elderly were less likely to receive cardiac

catheterization. However, with the interaction between age and comorbidity, this was no longer the case. A 65-74 year old patient with additional diagnoses and/or more severe conditions was less likely to receive a catheterization as compared to a 75-101 year old patient having just an AMI diagnosis.

A patient aged 64 years and younger was less likely to receive a CABG if admitted in 1990. The remaining four years of study period did not effect the distribution of CABG among the study population. This again is reflective of the actual distribution of the procedure during the study period. A patient in this age-stratum who had only one additional condition was almost 2 times more likely to receive a CABG than that of a patient in the same age bracket, who either had only an AMI diagnosis or had more severe and/or multiple conditions. This strongly indicates that the distribution of CABG was based on the patient's medical condition. Gender alone was not a significant predictor, but interacting with comorbidity, it became a statistically significant predictor of CABG utilization. Such that, a 64-75 year old female patient who had multiple and/or more severe conditions, beside an AMI was more than 5 times likely to receive a CABG. A patient 65 years or older again was less likely to receive a CABG in 1990. This is reflective of the actual distribution of CABG over the study period. Whereas, a patient aged 65-74 years was 2 times more likely to receive a CABG, than that of the eldest cohort. Finally, a black patient 65 years and older was less likely to receive a CABG. However, the actual distribution shows that black patients admitted in 1990 and 1991 received more CABGs than the white patients.

A patient less than 65 years of age was less likely to receive a PTCA if admitted in 1990, 1991, and 1992. This again is reflective of the actual distribution. Age was a statistically significant predictor in the utilization of PTCA; however, there was no difference in the chances of receiving a PTCA in any of the age cohorts. Additionally, a patient less than 65 years of age was less likely to receive a PTCA if they had multiple and/or more severe conditions beside an AMI. A patient 65 years of age and older was less likely to receive a PTCA, if admitted in 1990 or 1991, and had multiple and/or more severe conditions besides their primary diagnosis of AMI. On the other hand, the youngest cohort in this age-stratum was 2 times more likely to receive a PTCA than the eldest cohort.

As the above information indicates, my analysis shows an obvious year effect across all age-strata and procedures. The 1990 admission year in particular, is the year when the patients in my study were least likely to receive a procedure. My analysis shows that the cardiovascular procedures were widely disseminated as the years progress. As literature suggests, increasing expertise and technological advances have occurred with cardiovascular procedures over time. I hypothesize that these are contributing factors for the increasing utilization of the procedures as the study progressed.

Additionally, my analysis also shows an age effect across the 65 years and older age-stratum for all four procedures. The eldest cohort was the least likely group of patients in my study to receive a particular procedure. There is a paucity of data on the effects of cardiovascular procedures among AMI patients that are 65 years or older.

For example, two of the larger trials of coronary artery bypass grafting only included persons less than 65 years of age (103, 104). Thus, with the exclusion of this group of patients, I can only recommend that additional studies be conducted that include the elderly to effectively assess the risks and benefits of treatment in this population. The elderly are the fastest growing segment of the population and about two-thirds of the health care costs for heart disease in the US are for those 65 years and older. Thus, it will continue to be an important issue to appropriately assess the most effective means for treatment specifically among this cohort.

My data also strongly linked comorbidity with the decreasing chances of receiving a procedure. The more severe a patient's condition, in addition to the presence of multiple conditions, were strong indications for not receiving a particular procedure. As the literature review states, each cardiovascular procedure has contraindications, and as a result recommendations have been made to assist in evaluating the severity of a patient's case for the distribution of cardiovascular treatments.

In summary, for all the procedures analyzed, there is a definite trend towards higher utilization favoring some subgroups. This is again dependent on the age cohort and the cardiovascular procedure analyzed; however, given there are demographics that are statistically significant predictors in my study, there is reason enough to encourage further investigation among these groups. Such studies could provide information that could be used to develop specific recommendations targeted to eliminate unnecessary biases in the distribution of cardiovascular treatment.

I believe that my study proved informative, however, the study is limited by the following: (1) the data study was abstracted by coding personnel, thus I could not guarantee that coding errors did not occur; (2) I did not address the issue of patient readmission; (3) a clinico-demographic profile was not included, and as a result, I was not able to determine if a procedure was refused by a patient; (4) patient outcomes were not assessed in this study; (5) my study population was insufficiently distributed across all demographics; (6) I did not specifically address infarct location; (7) failure to develop my own comorbidity index scale; (8) not taking into account the specific artery used in the CABG; and (9) not addressing single vs. multiple vessel disease.

Despite these limitations, I believe that my study provides valuable information that can be used to improve health care at McLaren Hospital. In particular is the underuse of thrombolytic therapy. Increasing the number of patients that receive this procedure is a nationwide challenge. I do not propose indiscriminate use of thrombolytic therapy, rather I recommend the increased use in patients who probably will benefit. Furthermore, I encourage the hospital to reevaluate following the strict inclusion and exclusion criteria as recommended for administration of thrombolytic therapy, because the benefits especially that for the elderly, outweigh that of hemorrhagic risk.

Overall, the trends indicate that McLaren has widely distributed each of the four cardiovascular procedures as the years progress. Nevertheless, there was still evidence of unequal distribution among certain population subgroups at the close of the study. I would further recommend that an assessment of what might be accomplished using

currently available knowledge and technology be done, keeping the hospital's patient population in mind.

APPENDICES

APPENDIX A

Appendix A

ICD-9-CM Diagnosis Coding Descriptions (98)

ICD-9-CM code 410: ACUTE MYOCARDIAL INFARCTION - A severe, sudden onset of myocardial necrosis due to formation of a thrombus in the coronary arterial system obstruction arterial blood flow to that section of cardiac muscle.

The following fifth-digit subclassification is for use with category 410:

0 Episode of Care Unspecified

Use when the source document does not contain sufficient information for the assignment of fifth-digit 1 or 2.

1 Initial Episode of Care

Use fifth-digit 1 to designate the first episode of care (regardless of facility site) for a newly diagnosed myocardial infarction. The fifth-digit 1 is assigned regardless of the number of times a patient may be transferred during the initial episode of care.

2 Subsequent Episode of Care

Use fifth-digit 2 to designate an episode of care following the initial episode when the patient is admitted for further observation, evaluation or treatment for a myocardial infarction that has received initial treatment, but is still less than 8 weeks old.

ICD-9-CM code 410.0: OF ANTEROLATERAL WALL - Infarction of the cardiac wall situated in front and below.

ICD-9-CM code 410.1: OF OTHER ANTERIOR WALL - Infarction of the cardiac wall situated in the front.

ICD-9-CM code 410.2: OF INFEROLATERAL WALL - Infarction of the cardiac wall situated below and to one side.

ICD-9-CM code 410.3: OF INFEROPOSTERIOR WALL - Infarction of the cardiac wall situated below and in back.

ICD-9-CM code 410.4: OF OTHER INFERIOR WALL - Infarction of the lower cardiac wall situated below and to one side.

ICD-9-CM code 410.5: OF OTHER LATERAL WALL - Infarction of the cardiac wall situated to one side.

ICD-9-CM code 410.6: TRUE POSTERIOR WALL INFARCTION - Infarction of the cardiac wall situated on the back side.

ICD-9-CM code 410.7: SUBENDOCARDIAL INFARCTION - Infarction situated just beneath the endocardium.

ICD-9-CM code 410.8: OF OTHER SPECIFIED SITES - Note: Use this code when the diagnosis as a myocardial infarction but is not listed above (410.0-410.7).

ICD-9-CM code 410.9: UNSPECIFIED SITE - Note: Use this code when the diagnosis is identified as a myocardial infarction, but is not identified as to site, or type.

APPENDIX B

Appendix B

Diagnoses & Procedures (99)

The Uniform Hospital Discharge Data Set (UHDDS), prepared under the direction of the National Committee on Vital and Health Statistics of the U.S. Public Health Service, provides 14 data items recommended as basic data for hospital discharge statistics. Of the 14 items, diagnoses and procedures (with dates) are exactly defined in the Coding Clinic:

Diagnoses are all diagnoses that affect the current hospital stay. The **principal diagnosis** is designated and defined as the condition established after study to be chiefly responsible for occasioning the admission of the patient to the hospital. **Other diagnoses** to be designated and defined as associated with current hospital stay and are all conditions that coexist at the time of admission, that develop subsequently, or that affect the treatment received and/or the length of stay. Diagnoses related to an earlier episode that have no bearing on the current hospital stay are to be excluded.

Procedures and dates: All significant procedures are to be reported. A significant procedure is one that carries an operative or anesthetic risk, that requires highly trained personnel, or that requires special facilities or equipment. When more than one procedure is reported, the principal procedure is to be designated. In determining which of several procedures is principal, the following criteria apply:

- The principal procedure is one that was performed for definitive treatment rather than for diagnostic or exploratory purposes, or one necessary to take care of a complication.

- The principal procedure is that procedure most related to the principal diagnosis.

Diagnoses other than those required in the UHDDS (principal and associated) may appear in the medical record.

APPENDIX C

Appendix C

ICD-9-CM Procedure Coding Descriptions (98)

ICD-9-CM code 37.2: DIAGNOSTIC PROCEDURES ON HEART AND PERICARDIUM

ICD-9-CM code 37.21: RIGHT HEART CARDIAC CATHETERIZATION -
The insertion of a cardiac catheter into the right heart chambers for the detection of cardiac abnormalities.

ICD-9-CM code 37.22: LEFT HEART CARDIAC CATHETERIZATION -
The insertion of a cardiac catheter into the left heart chambers for the detection of cardiac abnormalities.

ICD-9-CM code 37.23: COMBINED RIGHT AND LEFT HEART CARDIAC CATHETERIZATION - The insertions of cardiac catheters into both the right and left heart chambers for the detection of cardiac abnormalities.

ICD-9-CM code 36.1: BYPASS ANASTOMOSIS FOR HEART REVASCULARIZATION - Restoration of coronary blood flow by a tubular surgical bypass of an occluded coronary artery.

ICD-9-CM code 36.11: AORTOCORONARY BYPASS OF ONE CORONARY ARTERY - Surgical anastomosis from the aorta, distal to one occluded coronary artery.

ICD-9-CM code 36.12: AORTOCORONARY BYPASS OF TWO CORONARY ARTERIES - Surgical anastomosis from the aorta, distal to two occluded coronary arteries.

ICD-9-CM code 36.13: AORTOCORONARY BYPASS OF THREE CORONARY ARTERIES - Surgical anastomosis from the aorta, distal to three occluded coronary arteries.

ICD-9-CM code 36.14: AORTOCORONARY BYPASS OF FOUR OR MORE CORONARY ARTERIES - Surgical anastomosis from the aorta, distal to four or more occluded coronary arteries.

ICD-9-CM code 36.15: SINGLE INTERNAL MAMMARY-CORONARY ARTERY BYPASS - Surgical anastomosis from one internal mammary artery, distal to an occluded coronary artery(s).

ICD-9-CM code 36.16: DOUBLE INTERNAL MAMMARY-CORONARY ARTERY BYPASS - Surgical anastomosis from both internal mammary arteries, distal to occluded coronary artery(s).

ICD-9-CM code 36.0: REMOVAL OF CORONARY ARTERY OBSTRUCTION AND INSERTION OF STENT(S) - The surgical elimination of coronary artery obstructions.

ICD-9-CM code 36.01: SINGLE VESSEL PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY [PTCA] OR CORONARY ATHERECTOMY WITHOUT MENTION OF THROMBOLYTIC AGENT - Dilation of an obstructed coronary artery using a balloon-tipped catheter or the procedural removal of a thickened coronary arterial intima, inserted through the femoral or other artery, without infusion of a thrombus-destroying substance.

ICD-9-CM code 36.02: SINGLE VESSEL PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY [PTCA] OR CORONARY ATHERECTOMY WITH THROMBOLYTIC AGENT - Dilation of an obstructed coronary artery using a balloon-tipped catheter or the procedural removal of a thickened coronary arterial intima, inserted through the femoral or other artery, with infusion of a thrombus-destroying substance.

ICD-9-CM code 36.05: MULTIPLE VESSEL PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY [PTCA] OR CORONARY ATHERECTOMY PERFORMED DURING THE SAME OPERATION, WITH OR WITHOUT MENTION OF THROMBOLYTIC AGENT

ICD-9-CM code 99.2: INJECTION OR INFUSION OF OTHER THERAPEUTIC OR PROPHYLACTIC SUBSTANCE - The forcing of a fluid, or introduction into a vein of a healing or disease-preventative substance, other than listed in (99.11 through 99.19).

ICD-9-CM code 99.29: INJECTION OR INFUSION OF OTHER THERAPEUTIC OR PROPHYLACTIC SUBSTANCE

APPENDIX D

APPENDIX D

CANADIAN CARDIOVASCULAR SOCIETY (102) ANGINA SEVERITY DEFINITIONS

Class I angina occurs with strenuous or prolonged exertion at work or recreation and does not occur with ordinary physical activity.

Class II angina occurs with walking rapidly on level ground or a grade and with rapidly walking up stairs. Ordinary walking for <2 blocks on the level or climbing one flight of stairs does not cause angina except during the first few hours after awakening, after meals, under emotional stress, in the wind, or in cold weather. This implies slight limitation of ordinary activity.

Class III angina occurs when walking <2 blocks on level ground at a normal pace, under normal conditions, or when climbing one flight of stairs. This implies marked limitation of ordinary physical activity.

Class IV angina occurs with even mild activity, and may occur at rest but must be of brief (< 15 min) duration. (If the angina is of longer duration, it is called unstable angina.) This implies inability to carry out even mild physical activity.

APPENDIX E

Appendix E

Recommendations for Administration of Thrombolytic Therapy to Patients with Myocardial Infarction

Patients Without Contraindications to Thrombolytic Therapy (3)

Class I

1. Patients <70 years of age who present with chest pain consistent with the diagnosis of acute myocardial infarction and at least 0.1 mV of ST segment elevation in at least two contiguous ECG leads in whom treatment can be initiated within 6 hours of pain onset.

Class IIa

1. Patients between ages 70 and 75 years who present with chest pain consistent with the diagnosis of acute myocardial infarction and at least 0.1 mV of ST segment elevation in at least two contiguous ECG leads in whom treatment can be initiated within 6 hours of pain onset.

2. Patients with acute myocardial infarction > 6 hours after symptoms onset but with a “stuttering” pattern of pain.

3. Patients who suffer clinically apparent reinfarction in the days after administration of thrombolytic therapy.

Class IIb

1. Patients who present with chest pain consistent with the diagnosis of acute myocardial infarction and at least 0.1 mV of ST segment elevation in at least two contiguous ECG leads in whom treatment can be initiated between 6 and 24 hours after pain onset.

2. Patients > 75 years of age who present with chest pain consistent with the diagnosis of acute myocardial infarction and at least 0.1 mV of ST segment elevation in at least two contiguous ECG leads in whom treatment can be initiated within 6 hours of pain onset where the impending infarction is extensive.

3. Patients who present with chest pain consistent with the diagnosis of acute myocardial infarction with ECG changed less profound than 0.1 mV of ST segment elevation in two contiguous leads who can be treated within 24 hours.

Class III

Patients who have had chest pain when:

- 1. Treatment cannot be initiated within 24 hours of onset of chest pain and pain has not recurred.**
- 2. Chest pain onset is unknown and has receded.**
- 3. The cause of the chest pain is unclear.**

APPENDIX F

Appendix F

Recommendations for Angioplasty After Intravenous Thrombolysis (67)

Class I

Dilation of a significant lesion suitable for coronary angioplasty in the infarct-related artery in patients who are in the low risk group for angiographic-related morbidity and mortality who have a type A lesion (see ACC/AHA Task Force Report on coronary angioplasty ⁴²) and:

1. Have recurrent episodes of ischemic chest pain particularly if accompanied by ECG changes (postinfarction angina).
2. Show evidence of myocardial ischemia while on optimal medical therapy during submaximal stress testing performed before hospital discharge or on maximal stress testing in the early posthospital period.
3. Have recurrent ventricular tachycardia or ventricular fibrillation, or both convincingly related to ischemia while on antiarrhythmic therapy.

Class IIa

Dilation of significant lesions in patients who:

1. Are similar to those in class I but who have type B lesions (anticipated success rate 60% to 85%) (see ACC/AHA Task Force Report on coronary angioplasty⁴²).
2. Are within 18 hours of onset of acute infarction and have cardiogenic shock or pump failure. These patients should be studied and undergo reperfusion as soon as possible.
3. Before hospital discharge in those who have survived cardiogenic shock or pump failure.

Class IIb

Dilation of a lesion in patients who:

1. Have an occluded coronary artery after attempted thrombolytic therapy.
2. Require multivessel angioplasty.
3. Have >90% diameter proximal narrowing of an infarct-related artery with a large area of viable myocardium still at risk.

Class III

All patients in the immediate postinfarct period (during initial hospitalization) who do not fulfill Class I or II criteria. For example:

1. Dilation in patients who are within the early hours of an evolving myocardial infarction and have <50% residual stenosis of the infarct-related artery after receiving a thrombolytic agent.
2. Dilation of lesions in vessels other than the infarct-related artery within the early hours of infarction.
3. Dilation of residual lesions that are borderline in severity (50% to 70% diameter narrowing) of the infarct-related artery without demonstration of ischemia on functional testing.
4. Dilation of the type C lesions (see ACC/AHA Task Force Report on coronary angioplasty for definition⁴²).
5. Undertaking angioplasty in patients in the high risk group for morbidity and mortality (see ACC/AHA Task Force Report on coronary angioplasty for definition⁴²).

APPENDIX G

Appendix G

Recommendations for Angioplasty (3)

Early Evolving Myocardial Infarction (Initial Hours of Myocardial Infarction)

Class I:

1. All patients developing pump failure/shock syndrome.
2. All patients suspected of developing an acute ventricular septal defect.
3. Persistent or recurrent ischemia, or both, despite thrombolytic therapy.

Class IIa:

1. When coronary angiography can be performed within the first 6 hours after the onset of chest pain in patients who are candidates for revascularization therapy utilizing percutaneous transluminal coronary angioplasty or coronary artery bypass surgery (but who are not candidates for thrombolytic therapy).
2. Patients who have had a previous aortocoronary vein graft if the graft is to the suspected infarct-related vessel.

Class IIb:

1. Patients who can be taken quickly for angioplasty or bypass surgery in a facility set up and qualification for these emergency procedures.

Class III:

1. As a routine after early intravenous thrombolytic therapy.
2. Patients with uncomplicated myocardial infarction having no evidence of ongoing ischemia.

Late Evolving Myocardial Infarction (After the Initial 6 hours up to But Not Including PredischARGE Evaluation)

Class I:

1. Patients with recurrent episodes of ischemic chest pain, particularly if accompanied by ECG changes.
2. Patients suspected of having acute mitral regurgitation or a ruptured interventricular septum causing heart failure or shock.
3. Patients suspected of developing subacute cardiac rupture (pseudoaneurysm).
4. Patients with cardiogenic shock or severe pump failure.

Class IIa:

1. Patients with congestive heart failure during intensive medical therapy.
2. Patients with recurrent ventricular tachycardia or ventricular fibrillation, or both, during intensive antiarrhythmic therapy.

Class IIb:

1. Asymptomatic patients who have received thrombolytic therapy during the evolving phase.

Class III:

1. Patients with uncomplicated completed myocardial infarction in whom no acute mechanical or surgical intervention is contemplated.

Convalescent Myocardial Infarction (Immediate PredischARGE up to 8 Weeks After Discharge)

Class I:

1. Postinfarction angina pectoris.
2. Patients with evidence of myocardial ischemia on laboratory testing: exercise-induced ischemia (with or without exercise-induced angina pectoris), manifested by ≥ 1 mm of ST segment depression or exercise-induced reversible thallium perfusion defect or defects, increased lung thallium uptake, or exercise-induced reduction of the ejection fraction or wall motion abnormalities on radionuclide ventriculography or two-dimensional echocardiography.

Class IIa:

1. Patients with the need to return to unusually active and vigorous physical employment.
2. Patients with a left ventricular ejection fraction $< 40\%$.

Class IIb:

1. As a routine in patients receiving thrombolytic therapy during the evolving phase of infarction.
2. Otherwise uncomplicated and asymptomatic patients who are < 45 years of age.
3. Patients with uncomplicated non-Q wave myocardial infarction not otherwise manifesting evidence of myocardial ischemia on noninvasive laboratory testing.

Class III:

1. Patients judged to have a debilitating disease or conditions that preclude their being candidates for invasive intervention.
2. Patients with coexisting disease judged to be primarily responsible for the patient's prognosis, with a greatly shortened life expectancy unless revascularization is determined to be necessary to facilitate treatment of the underlying disease.
3. Patients with very advanced left ventricular dysfunction (ejection fraction $< 20\%$) in the absence of angina pectoris or evidence of ischemia. An exception is the patient who is a candidate for aneurysectomy or cardiac transplantation.
4. Patients with ventricular arrhythmias who have no evidence of ischemia symptomatically or during exercise testing, well preserved exercise tolerance and no suggestion may be the patient with inducible sustained ventricular tachycardia.

APPENDIX H

Table 1: Characteristics of patients admitted to community hospital with acute myocardial infarction diagnosis; 1990-1994

CHARACTERISTIC	1990	1991	1992	1993	1994
Total Admissions-no.	475	511	549	550	574
Age- no. (%)					
1-44	37 (7.8)	37 (7.2)	27 (4.9)	32 (5.8)	33 (5.7)
45-54	68 (14.3)	82 (16.0)	71 (12.9)	68 (12.4)	84 (14.6)
55-64	117 (24.6)	118 (23.1)	135 (24.6)	140 (25.5)	138 (24.0)
65-74	139 (29.3)	120 (23.5)	166 (30.2)	183 (33.3)	162 (28.2)
75-101	114 (24.0)	154 (30.1)	150 (27.3)	127 (23.1)	157 (27.4)
Gender-no. (%)					
Female	158 (33.3)	204 (39.9)	220 (40.1)	229 (41.6)	210 (36.6)
Male	317 (66.7)	307 (60.1)	329 (59.9)	321 (58.4)	364 (63.4)
Payer-no. (%)					
All Other	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (0.5)
Private	6 (1.3)	11 (2.2)	8 (1.5)	4 (0.7)	3 (0.5)
Medicare	252 (53.1)	281 (55.0)	331 (60.3)	320 (58.2)	329 (57.3)
Medicaid	10 (2.1)	15 (2.9)	18 (3.3)	18 (3.3)	28 (4.9)
Commercial	199 (41.9)	170 (33.3)	165 (30.1)	185 (33.6)	184 (32.1)
HMO (Hlth +)	8 (1.7)	34 (6.7)	27 (4.9)	23 (4.2)	28 (4.9)
Race-no. (%)					
Black	27 (5.7)	31 (6.1)	23 (4.2)	36 (6.5)	30 (5.2)
White	448 (94.3)	480 (93.9)	526 (95.8)	514 (93.5)	544 (94.8)
Comorbidity Index-no. (%)					
0	311 (65.5)	357 (69.9)	344 (62.7)	344 (62.5)	377 (65.7)
1	119 (25.1)	116 (22.7)	141 (25.7)	132 (24.0)	139 (24.2)
2	45 (9.5)	38 (7.4)	64 (11.7)	74 (13.5)	58 (10.1)

Table 2: No. of AMI's by age, distributed across gender, race, insurance, and comorbidity; 1990-1994

CHARACTERISTIC	1990	1991	1992	1993	1994
Total Admissions-no.	475	511	549	550	574
(FEMALE)					
Age- no. (%)					
1-44	11 (7.0)	8 (3.9)	5 (2.3)	12 (5.2)	9 (4.3)
45-54	11 (7.0)	23 (11.3)	11 (5.0)	24 (10.5)	17 (8.1)
55-64	32 (20.3)	38 (18.6)	35 (15.9)	44 (17.2)	37 (17.6)
65-74	51 (32.3)	53 (26.0)	82 (37.3)	72 (31.4)	65 (31.0)
75-101	53 (33.5)	82 (40.2)	87 (39.5)	77 (33.6)	82 (39.0)
(MALE)					
Age-no. (%)					
1-44	26 (8.2)	29 (9.4)	22 (6.7)	20 (6.2)	24 (6.6)
45-54	57 (18.0)	59 (19.2)	60 (18.2)	44 (13.7)	67 (18.4)
55-64	85 (26.8)	80 (26.1)	100(30.4)	96 (29.9)	101(27.7)
65-74	88 (27.8)	67 (21.8)	84 (25.5)	111(34.6)	97 (26.6)
75-101	61 (19.2)	72 (23.5)	63 (19.1)	50 (15.6)	75 (20.6)
(BLACK)					
Age-no. (%)					
1-44	8 (29.6)	3 (11.1)	6 (22.2)	6 (22.2)	4 (14.8)
45-54	4 (12.9)	5 (16.1)	3 (22.6)	7 (22.6)	8 (25.8)
55-64	0 (0.0)	2 (8.7)	7 (30.4)	9 (39.1)	5 (21.7)
65-74	3 (8.3)	9 (25.0)	9 (25.0)	9 (25.0)	6 (16.7)
75-101	1 (3.3)	7 (23.3)	12 (40.0)	5 (16.7)	5 (16.7)
(WHITE)					
Age-no. (%)					
1-44	29 (6.5)	33 (6.9)	27 (5.1)	29 (5.6)	32 (5.9)
45-54	65 (14.5)	77 (16.0)	69 (16.0)	59 (11.5)	77 (14.2)
55-64	111(24.8)	111(23.1)	128(23.1)	131(25.5)	126(23.2)
65-74	133(29.7)	113(23.5)	157(29.8)	174(33.9)	157(28.9)
75-101	110(24.6)	146(30.4)	145(27.6)	121(23.5)	152(27.9)

Table 2 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(ALL OTHER, PRIVATE INSURANCE)					
Age-no. (%)					
1-64	6 (100.0)	11(100.0)	8 (100.0)	3 (75.0)	5 (100.0)
65-101	0 (0.0)	0 (0.0)	0 (0.0)	1 (25.0)	0 (0.0)
(HMO)					
Age-no. (%)					
1-64	3 (37.5)	25 (73.5)	16 (59.3)	22 (95.7)	25 (89.3)
65-101	5 (62.5)	9 (26.5)	11 (40.7)	1 (4.3)	3 (10.7)
(MEDICARE & MEDICAID)					
Age-no. (%)					
1-64	33 (12.6)	34 (11.5)	48 (13.8)	37 (10.9)	50 (14.0)
65-101	229 (87.4)	262(88.5)	301(86.2)	301(89.1)	307 (86.0)
(COMMERCIAL)					
Age-no. (%)					
1-64	180 (90.5)	167 (98.2)	161 (97.6)	178 (96.2)	175 (95.1)
65-101	19 (9.5)	3 (1.8)	4 (2.4)	7 (3.8)	9 (4.9)
(COMORBIDITY INDEX 0)					
Age-no. (%)					
1-44	32 (10.3)	37 (10.4)	25 (7.3)	27 (7.8)	30 (8.0)
45-54	58 (18.6)	74 (20.7)	68 (19.8)	58 (16.9)	69 (18.3)
55-64	78 (25.1)	92 (25.8)	104(30.2)	104 (30.2)	105(27.9)
65-74	89 (28.6)	76 (21.3)	94 (27.3)	102 (29.7)	101(26.8)
75-101	54 (17.4)	78 (21.8)	53 (15.4)	53 (15.4)	72 (19.1)

Table 2 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(COMORBIDITY INDEX 1)					
Age-no. (%)					
1-44	4 (3.4)	0 (0.0)	2 (1.4)	5 (3.8)	0 (0.0)
45-54	6 (5.0)	7 (6.0)	2 (1.4)	5 (3.8)	10 (7.2)
55-64	27 (22.7)	21 (18.1)	23 (16.3)	24 (18.2)	25 (18.0)
65-74	40 (33.6)	35 (30.2)	50 (35.5)	55 (41.7)	44 (31.7)
75-101	42 (35.3)	53 (45.7)	64 (45.5)	43 (32.6)	60 (43.2)
(COMORBIDITY INDEX 2)					
Age-no. (%)					
1-44	1 (2.2)	0 (0.0)	0 (0.0)	0 (0.0)	3 (5.2)
45-54	4 (8.9)	1 (2.6)	1 (1.6)	5 (6.8)	5 (8.6)
55-64	12 (26.7)	5 (13.2)	8 (12.5)	12 (16.2)	8 (13.8)
65-74	10 (22.2)	9 (23.7)	22 (34.4)	26 (35.1)	17 (29.3)
75-101	18 (40.0)	23 (60.5)	33 (51.6)	31 (41.9)	25 (43.1)

Table 3: No. of AMI's by gender, distributed across age, race, insurance, and comorbidity; 1990-1994

CHARACTERISTIC	1990	1991	1992	1993	1994
Total Admissions-no.	475	511	549	550	574
(AGE 1-44)					
Gender-no. (%)					
Female	11 (29.7)	8 (21.6)	5 (18.5)	12 (37.5)	9 (27.3)
Male	26 (70.3)	29 (78.4)	22 (81.5)	20 (62.5)	24 (72.7)
(AGE 45-54)					
Gender-no (%)					
Female	11 (16.2)	23 (28.0)	11 (15.5)	24 (35.3)	17 (20.2)
Male	57 (83.8)	59 (72.0)	60 (84.5)	44 (64.7)	67 (79.8)
(AGE 55-64)					
Gender-no. (%)					
Female	32 (27.4)	38 (32.2)	35 (25.9)	44(31.4)	37 (26.8)
Male	85 (72.6)	80 (67.8)	100(74.1)	96(68.6)	101(73.2)
(AGE 65-74)					
Gender-no. (%)					
Female	51 (36.7)	53 (44.2)	82 (49.4)	72 (39.3)	65 (40.1)
Male	88 (63.3)	67 (55.8)	84 (50.6)	111(60.7)	97 (59.9)
(AGE 75-101)					
Gender-no. (%)					
Female	53 (46.5)	82 (53.2)	87 (58.0)	77 (60.6)	82 (52.2)
Male	61 (53.5)	72 (46.8)	63 (42.0)	50 (39.4)	75 (47.8)
(BLACK)					
Gender-no. (%)					
Female	8 (29.6)	10 (32.3)	13 (56.5)	14 (38.9)	12 (40.0)
Male	19 (70.4)	21 (67.7)	10 (43.5)	22 (61.1)	18 (60.0)
(WHITE)					
Gender-no. (%)					
Female	150(33.5)	194(40.4)	207(39.4)	215(41.8)	198(36.4)
Male	298(66.5)	286(59.6)	319(60.6)	299(58.2)	346(63.6)
(ALL OTHER, PRIVATE INSURANCE)					
Gender-no. (%)					
Female	1 (16.7)	4 (36.4)	2 (25.0)	1 (25.0)	1 (20.0)
Male	5 (83.3)	7 (63.6)	6 (75.0)	3 (75.0)	4 (80.0)

Table 3 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(HMO)					
Gender-no. (%)					
Female	4 (50.0)	17 (50.0)	11 (40.7)	5 (21.7)	12 (42.9)
Male	4 (50.0)	17 (50.0)	16 (59.3)	18 (78.3)	16 (57.1)
(MEDICARE & MEDICAID)					
Gender-no. (%)					
Female	102(38.9)	134(45.3)	172(49.3)	161(47.6)	160(44.8)
Male	160(61.1)	162(54.7)	177(50.7)	177(52.4)	197(55.2)
(COMMERCIAL)					
Gender-no. (%)					
Female	51 (25.6)	49 (28.8)	35 (21.2)	62 (33.5)	37 (20.1)
Male	148(74.4)	121(71.2)	130(78.8)	123(66.5)	147(79.9)
(Comorbidity Index 0)					
Gender-no. (%)					
Female	90 (28.9)	130(36.4)	116(33.7)	132(38.4)	119(31.6)
Male	221(71.1)	227(63.6)	228(66.3)	212(61.6)	258(68.4)
(Comorbidity Index 1)					
Gender-no. (%)					
Female	45 (37.8)	56 (48.3)	75 (53.2)	59 (44.7)	57 (41.0)
Male	74 (62.2)	60 (51.7)	66 (46.8)	73 (55.3)	82 (59.0)
(Comorbidity Index 2)					
Gender-no. (%)					
Female	23 (51.1)	18 (47.4)	29 (45.3)	38 (51.4)	34 (58.6)
Male	22 (48.9)	20 (52.6)	35 (54.7)	36 (48.6)	24 (41.4)

Table 4: No. of AMI's by insurance, distributed across age, race, gender, and comorbidity; 1990-1994

CHARACTERISTIC	1990	1991	1992	1993	1994
Total Admissions-no.	475	511	549	550	574
(AGE 1-44)					
Payer-no. (%)					
All Other, Private	3 (8.1)	1 (2.7)	2 (7.4)	1 (3.1)	1 (3.0)
HMO	1 (2.7)	7 (18.9)	2 (7.4)	4 (12.5)	3 (9.1)
Medicare & Medicaid	3 (8.1)	7 (18.9)	5 (18.5)	8 (25.0)	6 (18.2)
Commercial	30 (81.1)	22 (59.5)	18 (66.7)	19 (59.4)	23 (69.7)
(AGE 45-54)					
Payer-no. (%)					
All Other, Private	2 (2.9)	3 (3.7)	4 (5.6)	1 (1.5)	2 (2.4)
HMO	0 (0.0)	8 (9.8)	4 (5.6)	9 (13.2)	6 (7.1)
Medicare & Medicaid	9 (13.2)	15 (18.3)	13 (18.3)	6 (8.8)	16 (19.0)
Commercial	57 (83.8)	56 (68.3)	50(70.4)	52(76.5)	60 (71.4)
(AGE 55-64)					
Payer-no. (%)					
All Other, Private	1 (0.9)	7 (5.9)	2 (1.5)	1 (0.7)	2 (1.4)
HMO	2 (1.7)	10 (8.5)	10 (7.4)	9 (6.4)	16 (11.6)
Medicare & Medicaid	21 (17.9)	12 (10.2)	30 (22.2)	23 (16.4)	28 (20.3)
Commercial	93 (79.5)	89 (75.4)	93 (68.9)	107(76.4)	92 (66.7)
(AGE 65-74)					
Payer-no. (%)					
All Other, Private	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.5)	0 (0.0)
HMO	3 (2.2)	7 (5.8)	4 (2.4)	1 (0.5)	3 (1.9)
Medicare & Medicaid	123(88.5)	110(91.7)	158(95.2)	179(97.8)	150(92.6)
Commercial	13 (9.4)	3 (2.5)	4 (2.4)	2 (1.1)	9 (5.6)

Table 4 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(AGE 75-101)					
Payer-no. (%)					
All Other, Private	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.5)	0 (0.0)
HMO	3 (2.2)	7 (5.8)	4 (2.4)	1 (0.5)	3 (1.9)
Medicare & Medicaid	123(88.5)	110(91.7)	158(95.2)	179(97.8)	150(92.6)
Commercial	13 (9.4)	3 (2.5)	4 (2.4)	2 (1.1)	9 (5.6)
(BLACK)					
Payer-no. (%)					
All Other, Private	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
HMO	1 (3.7)	0 (0.0)	0 (0.0)	0 (0.0)	1 (3.3)
Medicare & Medicaid	11(40.7)	19 (61.3)	15 (65.2)	18 (50.0)	16 (53.3)
Commercial	15 (55.6)	12 (38.7)	8 (34.8)	18 (50.0)	13 (43.3)
(WHITE)					
Payer-no. (%)					
All Other, Private	6 (1.3)	11 (2.3)	8 (1.5)	4 (0.8)	5 (0.9)
HMO	7 (1.6)	34 (7.1)	27 (5.1)	23 (4.5)	27 (5.0)
Medicare & Medicaid	251(56.0)	277(57.7)	334(63.5)	320(62.3)	341(62.7)
Commercial	184(41.1)	158(32.9)	157(29.8)	167(32.5)	171(31.4)
(FEMALE)					
Payer-no. (%)					
All Other, Private	1 (0.6)	4 (2.0)	2 (0.9)	1 (0.4)	1 (0.5)
HMO	4 (2.5)	17 (8.3)	11 (5.0)	5 (2.2)	12 (5.7)
Medicare & Medicaid	102(64.6)	134(65.7)	172(78.2)	161(70.3)	160(76.2)
Commercial	51 (32.3)	49 (24.0)	35 (15.9)	62 (27.1)	37 (17.6)
(MALE)					
Payer-no. (%)					
All Other, Private	5 (1.6)	7 (2.3)	6 (1.8)	3 (0.9)	4 (1.1)
HMO	4 (1.3)	17 (5.5)	16 (4.9)	18 (5.6)	16 (4.4)
Medicare/Aid	160(50.5)	162(52.8)	177(53.8)	177(55.1)	197(54.1)
Commercial	148(46.7)	121(39.4)	130(39.5)	123(38.3)	147(40.4)

Table 4 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(Comorbidity Index 0)					
Payer-no. (%)					
All Other, Private	6 (1.9)	10 (2.8)	8 (2.3)	4 (1.2)	5 (1.3)
HMO	3 (1.0)	30 (8.4)	19 (5.5)	19(5.5)	24 (6.4)
Medicare & Medicaid	152(48.9)	166(46.5)	175(50.9)	175(50.9)	201(53.3)
Commercial	150(48.2)	151(42.3)	142(41.3)	175(50.9)	147(39.0)
(Comorbidity Index 1)					
Payer-no. (%)					
All Other, Private	0 (0.0)	1 (0.9)	0 (0.0)	0 (0.0)	0 (0.0)
HMO	4 (3.4)	4 (3.4)	5 (3.5)	2 (1.5)	3 (2.2)
Medicare & Medicaid	81 (68.1)	97 (83.6)	116(82.3)	105(79.5)	110(79.1)
Commercial	34 (28.6)	14 (12.1)	20 (14.2)	25 (18.9)	26 (18.7)
(Comorbidity Index 2)					
Payer-no. (%)					
All Other, Private	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
HMO	1 (2.2)	0 (0.0)	3 (4.7)	2 (2.7)	1 (1.7)
Medicare & Medicaid	29 (64.4)	33 (86.8)	58 (90.6)	58 (78.4)	46 (79.3)
Commercial	15 (33.3)	5 (13.2)	3 (4.7)	14 (18.9)	11 (19.0)

Table 5: No. of AMI's by race, distributed across age, gender, comorbidity, and insurance; 1990-1994

CHARACTERISTIC	1990	1991	1992	1993	1994
Total Admissions-no.	475	511	549	550	574
(AGE 1-44)					
Race-no. (%)					
Black	8 (21.6)	4 (10.8)	0 (0.0)	3 (9.4)	1 (3.0)
White	29 (78.4)	33 (89.2)	27(100.0)	29 (90.6)	32 (97.0)
(AGE 45-54)					
Race-no. (%)					
Black	3 (4.4)	5 (6.1)	2 (2.8)	9 (13.2)	7 (8.3)
White	65 (95.6)	77 (93.9)	69 (97.2)	59 (86.8)	77 (91.7)
(AGE 55-64)					
Race-no. (%)					
Black	6 (5.1)	7 (5.9)	7 (5.2)	9 (6.4)	12 (8.7)
White	111(94.9)	111(94.1)	128(94.8)	131(93.6)	126(91.3)
(AGE 65-74)					
Race-no. (%)					
Black	6 (4.3)	7 (5.8)	9 (5.4)	9 (4.9)	5 (3.1)
White	133(95.7)	113(94.2)	157(94.6)	174(95.1)	157(96.9)
(AGE 75-101)					
Race-no. (%)					
Black	4 (3.5)	8 (5.2)	5 (3.3)	6 (4.7)	5 (3.2)
White	110(96.5)	146(94.8)	145(96.7)	121(95.3)	152(96.8)
(FEMALE)					
Race-no. (%)					
Black	8 (5.1)	10 (4.9)	13 (5.9)	14 (6.1)	12 (5.7)
White	150(94.9)	194(95.1)	207(94.1)	215(93.9)	198(94.3)
(MALE)					
Race-no. (%)					
Black	19 (6.0)	21 (6.8)	10 (3.0)	22 (6.9)	18 (4.9)
White	298(94.0)	286(93.2)	319(97.0)	299(93.1)	346(95.1)
(Comorbidity Index 0)					
Race-no. (%)					
Black	18 (5.8)	18 (5.0)	14 (4.1)	21 (6.1)	19 (5.0)
White	293(94.2)	33 (95.0)	330(95.9)	323(93.9)	358(95.0)

Table 5 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(Comorbidity Index 1)					
Race-no. (%)					
Black	4 (3.4)	10 (8.6)	3 (2.1)	9 (6.8)	7 (5.0)
White	115(96.6)	106(91.4)	138(97.9)	123(93.2)	132(95.0)
(Comorbidity Index 2)					
Race-no. (%)					
Black	5 (11.1)	3 (7.9)	6 (9.4)	6 (8.1)	4 (6.9)
White	40 (88.9)	35 (92.1)	58 (90.6)	68 (91.9)	54 (93.1)
(ALL OTHER, PRIVATE INSURANCE)					
RACE-no. (%)					
BLACK	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
WHITE	6(100.0)	11(100.0)	8(100.0)	4(100.0)	5(100.0)
(HMO)					
RACE-no. (%)					
BLACK	1 (12.5)	0 (0.0)	0 (0.0)	0 (0.0)	1 (3.6)
WHITE	7 (87.5)	34(100.0)	27(100.0)	23(100.0)	27(96.4)
(MEDICARE & MEDICAID)					
RACE-no. (%)					
BLACK	11 (4.2)	19 (6.4)	15 (4.3)	18 (5.3)	16 (4.5)
WHITE	251(95.8)	277(93.6)	334(95.7)	320(94.7)	341(95.5)
(COMMERCIAL)					
RACE-no. (%)					
BLACK	15 (7.5)	12 (7.1)	8 (4.8)	18 (9.7)	13 (7.1)
WHITE	184(92.5)	158(92.9)	157(95.2)	167(90.3)	171(92.9)

Table 6: No. of AMI's by comorbidity, distributed across age, gender, race, and insurance; 1990-1994

CHARACTERISTIC	1990	1991	1992	1993	1994
(WHITE)					
Comorbidity Index- no.(%)					
0	293 (65.4)	339(70.6)	330(62.7)	323(62.8)	358(65.8)
1	115(25.7)	106(22.1)	138(26.2)	123(23.9)	132(24.3)
2	40 (8.9)	35 (7.3)	58 (11.0)	68 (13.2)	54 (9.9)
(FEMALE)					
Comorbidity Index- no. (%)					
0	90 (57.0)	130(63.7)	111(52.7)	132(57.6)	119(56.7)
1	45 (28.5)	56 (27.5)	75 (34.1)	59 (25.8)	57 (27.1)
2	23 (14.6)	18 (8.8)	29 (13.2)	38 (16.6)	34 (16.2)
(MALE)					
Comorbidity Index- no. (%)					
0	22 (69.7)	227(73.9)	228(69.3)	212(66.0)	258(70.9)
1	74 (23.3)	60 (19.5)	66 (20.1)	73 (22.7)	82 (22.5)
2	22 (6.9)	20 (6.5)	35 (10.6)	36 (11.2)	24 (6.6)
(ALL OTHER, PRIVATE)					
Comorbidity Index- no. (%)					
0	6 (100.0)	10 (90.9)	8 (100.0)	4 (100.0)	5 (100.0)
1	0 (0.0)	1 (9.1)	0 (0.0)	0 (0.0)	0 (0.0)
2	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
(HMO)					
Comorbidity Index- no. (%)					
0	3 (37.5)	30 (88.2)	19 (70.4)	19 (82.6)	2 (85.7)
1	4 (50.0)	4 (11.8)	5 (18.5)	2 (8.7)	3 (10.7)
2	1 (12.5)	0 (0.0)	3 (11.1)	2 (8.7)	1 (3.6)

Table 6 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(WHITE)					
Comorbidity Index- no. (%)					
0	293(65.4)	339(70.6)	330(62.7)	323(62.8)	358(65.8)
1	115(25.7)	106(22.1)	138(26.2)	123(23.9)	132(24.3)
2	40 (8.9)	35 (7.3)	58 (11.0)	68 (13.2)	54 (9.9)
(FEMALE)					
Comorbidity Index- no. (%)					
0	90 (57.0)	130(63.7)	111(52.7)	132(57.6)	119(56.7)
1	45 (28.5)	56 (27.5)	75 (34.1)	59 (25.8)	57 (27.1)
2	23 (14.6)	18 (8.8)	29 (13.2)	38 (16.6)	34 (16.2)
(MALE)					
Comorbidity Index- no. (%)					
0	22 (69.7)	227(73.9)	228(69.3)	212(66.0)	258(70.9)
1	74 (23.3)	60 (19.5)	66 (20.1)	73 (22.7)	82 (22.5)
2	22 (6.9)	20 (6.5)	35 (10.6)	36 (11.2)	24 (6.6)
(ALL OTHER, PRIVATE)					
Comorbidity Index- no. (%)					
0	6 (100.0)	10 (90.9)	8 (100.0)	4 (100.0)	5 (100.0)
1	0 (0.0)	1 (9.1)	0 (0.0)	0 (0.0)	0 (0.0)
2	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
(HMO)					
Comorbidity Index- no. (%)					
0	3 (37.5)	30 (88.2)	19 (70.4)	19 (82.6)	2 (85.7)
1	4 (50.0)	4 (11.8)	5 (18.5)	2 (8.7)	3 (10.7)
2	1 (12.5)	0 (0.0)	3 (11.1)	2 (8.7)	1 (3.6)

Table 6 (cont'd):

CHARACTERISTIC	1990	1991	1992	1993	1994
(MEDICARE & MEDICAID)					
Comorbidity Index-no.(%)					
0	152(58.0)	166(56.1)	175(50.1)	175(51.8)	201(56.3)
1	81 (30.9)	97 (32.8)	116(33.2)	105(31.1)	110(30.8)
2	29 (11.1)	33 (11.1)	58 (16.6)	58 (17.2)	46 (12.9)
(COMMERCIAL)					
Comorbidity Index-no. (%)					
0	150(75.4)	151(88.8)	142(86.1)	146(78.9)	147(79.9)
1	34 (17.1)	14 (8.2)	20 (12.1)	25 (13.5)	26 (14.1)
2	15 (7.5)	5 (2.9)	3 (1.8)	14 (7.6)	11 (6.0)

Table 7: Unadjusted odds ratios a,b on thrombolytic therapy utilization for patients up to 64 years of age, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance B-Adjusted
Admission Year						
1990	.3225	.3203	.3196	.3222	.3270	.3128 c
1991	.9005 c	.9044 c	.9195 c	.8981 c	.8155 c	.8955 c
1992	1.0259 c	1.0195 c	1.0119 c	1.0162 c	1.0038 c	1.0304 c
1993	.7556 c	.7468 c	.7866 c	.7571 c	.7588 c	.7413 c
Age (years)						
1-44	-	.9816 c	-	-	-	-
45-54	-	.7794 c	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	-	-	-	-	-
Gender						
Female	-	-	.5917	-	-	-
Race						
Black	-	-	-	.7790 c	-	-
Comorbidity						
1	-	-	-	-	.6166 d	-
2	-	-	-	-	.5742 c	-
Insurance B						
Other	-	-	-	-	-	.6387 c
HMO	-	-	-	-	-	.8374 c
Medicare/aid	-	-	-	-	-	.7051 c

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; b The reference groups for the variables are as follows; admission year, 1994; age, 55-64 years; gender, male; race, white; comorbidity, 0; and insurance B; commercial; c Not significant; and d Significant at the 0.05 level.

Table 8: Unadjusted odds ratios a,b on thrombolytic therapy utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance A-Adjusted
Admission Year						
1990	.3970	.3806	.3961	.3983	.3853	.3983
1991	1.0541 c	1.1136 c	1.0556 c	1.0668 c	1.0378 c	1.0543 c
1992	1.2387 c	1.2276 c	1.2428 c	1.2473 c	1.3228 c	1.2396 c
1993	1.3224 c	1.2505 c	1.3236 c	1.3346 c	1.3884 c	1.3216 c
Age						
1-44	-	-	-	-	-	-
45-54	-	-	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	2.1548	-	-	-	-
Gender						
Female	-	-	.9564 c	-	-	-
Race						
Black	-	-	-	.5368 c	-	-
Comorbidity						
1	-	-	-	-	.5494	-
2	-	-	-	-	.3492	-
Insurance A						
Other	-	-	-	-	-	.9448 c

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; comorbidity, 0; and insurance A; medicare; c Not significant; and d Significant at the 0.05 level.

Table 9: Unadjusted odds ratios a,b on cardiac catheterization utilization for patients up to 64 years of age, and individually adjusted by age, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance B-Adjusted
Admission Year						
1990	.3954	.3915	.3948	.3923	.3955	.3796
1991	.4390	.4327	.4417	.4333	.4006	.4374
1992	.4874	.4909	.4848	.4720	.4545	.4843
1993	1.2882 c	1.2996	1.3065 c	1.2989 c	1.3219 c	1.2718 c
Age (years)						
1-44	-	1.3778 c	-	-	-	-
45-54	-	1.2272 c	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	-	-	-	-	-
Gender						
Female	-	-	.8542 c	-	-	-
Race						
Black	-	-	-	.5308	-	-
Comorbidity						
1	-	-	-	-	.7024 c	-
2	-	-	-	-	.2402	-
Insurance B						
Other	-	-	-	-	-	.7561 c
HMO	-	-	-	-	-	.7093 c
Medicare/aid	-	-	-	-	-	.8097 c

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; b The reference groups for the variables are as follows; admission year, 1994; age, 55-64 years; gender, male; race, white; comorbidity, 0; and insurance B; commercial; c Not significant; and d Significant at the 0.05 level.

Table 10: Unadjusted odds ratios a,b on cardiac catheterization utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance A-Adjusted
Admission Year						
1990	.4527	.3870	.4460	.4538	.4257	.4516
1991	.4294	.4300	.4314	.4332	.4045	.4293
1992	.4563	.4043	.4633	.4583	.4730	.4560
1993	.7826 c	.6718 d	.7846 c	.7885 c	.8134	.7830 c
Age						
1-44	-	-	-	-	-	-
45-54	-	-	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	3.9705	-	-	-	-
Gender						
Female	-	-	.7846 d	-	-	-
Race						
Black	-	-	-	.6305 c	-	-
Comorbidity						
1	-	-	-	-	.4636	-
2	-	-	-	-	.3393	-
Insurance A						
Other	-	-	-	-	-	1.0425 c

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; comorbidity, 0; and insurance A; medicare;
c Not significant; and d Significant at the 0.05 level.

Table 11: Unadjusted odds ratios a,b on coronary artery bypass grafting utilization for patients up to 64 years of age, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance B-Adjusted
Admission Year d						
1990	.6127 d	.6153 d	.6123 d	.6127 d	.5985 d	.6035 d
1991	1.1251 d	1.1387 c	1.1319 c	1.1244 c	1.1585 c	1.1203 c
1992	.8704 c	.8617 c	.8671 c	.8685 c	.8897 c	.8681 c
1993	1.2666 c	1.2550 c	1.2817 c	1.2672 c	1.2607 c	1.2604 c
Age (years)						
1-44	-	.7455 c	-	-	-	-
45-54	-	.7868 c	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	-	-	-	-	-
Gender						
Female	-	-	.8711 c	-	-	-
Race						
Black	-	-	-	.9454 c	-	-
Comorbidity						
1	-	-	-	-	1.5295 d	-
2	-	-	-	-	1.6273 c	-
Insurance B						
Other	-	-	-	-	-	.9986 c
HMO	-	-	-	-	-	.8817 c
Medicare/aid	-	-	-	-	-	.9039 c

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; b The reference groups for the variables are as follows; admission year, 1994; age, 55-64 years; gender, male; race, white; comorbidity, 0; and insurance B; commercial; c Not significant; and d Significant at the 0.05 level.

Table 12: Unadjusted odds ratios^{a,b} on coronary artery bypass grafting utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance A-Adjusted
Admission Year						
1990	.3804	.3588	.3745	.3817	.3795	.3855
1991	.6559 d	.6880 c	.6607 d	.6654 d	.6544 d	.6562 d
1992	.6745 d	.6568 d	.6873 c	.6797 d	.6800 d	.6765 d
1993	.7948 c	.7365 c	.7984 c	.8035 c	.7935 c	.7925 c
Age						
1-44	-	-	-	-	-	-
45-54	-	-	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	2.3580	-	-	-	-
Gender						
Female	-	-	.7629 d	-	-	-
Race						
Black	-	-	-	.4223 d	-	-
Comorbidity						
1	-	-	-	-	.7946 c	-
2	-	-	-	-	.9796 c	-
Insurance A						
Other	-	-	-	-	-	.7740 c

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; comorbidity, 0; and insurance A; medicare; ^c Not significant; and ^d Significant at the 0.05 level.

Table 13: Unadjusted odds ratios ^{a,b} on percutaneous transluminal coronary angioplasty utilization for patients up to 64 years of age, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance B-Adjusted
Admission Year						
1990	.3975	.3997	.3975	.3960	.3993	.3786
1991	.4056	.4012	.4049	.4025	.3677	.3953
1992	.6278 d	.6300 d	.6285 d	.6158	.5835	.6230 d
1993	.7666 c	.7785 c	.7641 c	.7689 c	.7680 c	.7505 c
Age (years)						
1-44	-	.8764	-	-	-	-
45-54	-	1.4485	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	-	-	-	-	-
Gender						
Female	-	-	1.0379 c	-	-	-
Race						
Black	-	-	-	.6235 c	-	-
Comorbidity						
1	-	-	-	-	.3463	-
2	-	-	-	-	.2111	-
Insurance B c						
Other	-	-	-	-	-	1.0126 c
HMO	-	-	-	-	-	.7545 c
Medicare/aid	-	-	-	-	-	.6543 d

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 55-64 years; gender, male; race, white; comorbidity, 0; and insurance B, commercial; ^c Not significant; and ^d Significant at the 0.05 level.

Table 14: Unadjusted odds ratios ^{a,b} on percutaneous transluminal coronary angioplasty utilization for patients 65 years and older, and individually adjusted by age, gender, race, comorbidity, and insurance for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted	Insurance A-Adjusted
Admission Year						
1990	.4880	.4591	.4857	.4897	.4569	.4749
1991	.4921	.5144	.4934	.4981	.4632	.4910
1992	.7160 c	.6958 c	.7207 c	.7209 c	.7774 c	.7115 c
1993	.7330 c	.6714 d	.7342 c	.7398 c	.7678 c	.7365 c
Age						
1-44	-	-	-	-	-	-
45-54	-	-	-	-	-	-
55-64	-	-	-	-	-	-
65-74	-	2.6228	-	-	-	-
Gender						
Female	-	-	.9131 c	-	-	-
Race						
Black	-	-	-	.4885 c	-	-
Comorbidity						
1	-	-	-	-	.3302	-
2	-	-	-	-	.1811	-
Insurance A						
Other	-	-	-	-	-	1.4938

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; comorbidity, 0; and insurance A; medicare; ^c Not significant; and ^d Significant at the 0.05 level.

Table 15: Unadjusted odds ratios a,b on thrombolytic therapy utilization and adjusted by statistically significant predictors for other effects of admission year on utilization as the final model.

Variables		Less than 65 years of age		65 years of age and older	
Admission Year		Unadjusted	Final Model	Unadjusted	Final Model
1990		.3225 (.1961,.5304)	.3196 (.1941,.5265)	.3970 (.2226,.7081)	.3721 (.2074,.6674)
1991		.9005 c (.6049,1.341)	.9195 c (.6164,1.371)	1.0541 c (.6766,1.6422)	1.0919 c (.6955,1.714)
1992		1.0259 c (.6921,1.521)	1.0119 c (.6815,1.503)	1.2387 c (.8164,1.879)	1.2971 c (.8483,1.984)
1993		.7556 c (.5034,1.134)	.7866 c (.5227,1.1837)	1.3224 c (.8735,2.002)	1.3154 c (.8619,2.008)
Age					
1-44		.9446 c (.6348,1.406)	-	-	-
45-54		.7867 c (.5792,1.068)	-	-	-
55-64		-	-	-	-
65-74		-	-	2.1216 (1.5737,2.8603)	1.9479 (1.433,2.647)

Table 15 (cont'd):

Gender				
Female	.5983 d (.9310, .8304)	.5917 (.4246, .8245)	.9991 c (.7531, 1.325)	-
Race				
Black	.7561 c (.4308, 1.327)	-	.5527 (.2356, 1.2964)	-
Comorbidity				
1	.5922 d (.3821, .9179)	-	.5682 (.4105, .7866)	.5973 (.4286, .8325)
2	.5344 c (.2683, 1.065)	-	.3777 (.2258, .6318)	.3920 (.2322, .6619)
Insurance A				
Other	-	-	.8187 c (.4145, 1.672)	-
Insurance B				
Other	.6761 c (.2754, 1.660)	-	-	-
HMO	.9700 c (.5858, 1.606)	-	-	-
Medicare/aid	.7512 c (.5142, 1.097)	-	-	-

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows: admission year, 1994; age, 55-64 years for less than 65 years & 75-101 years for 65 years and older; gender, male; race, white; comorbidity, 0; insurance A, medicare; insurance B, commercial; ^c Not significant; and ^d Significant at the 0.05 level.

Table 16: Unadjusted odds ratios a,b on cardiac catheterization utilization and adjusted by statistically significant predictors for other effect of admission year as the final model.

Variables	Less than 65 years of age		65 years of age and older	
Admission Year	Unadjusted	Final Model	Unadjusted	Final Model
1990	.3954 (.2627, .5949)	.3922 (.2583, .5954)	.4527 (.3233, .6339)	.3647 (.2531, .5254)
1991	.4390 (.2927, .6584)	.3960 (.2617, .5991)	.4294 (.3086, .5974)	.4058 (.2842, .5792)
1992	.4874 (.3235, .7341)	.4410 (.2900, .6704)	.4563 (.3322, .6265)	.4152 (.2946, .5854)
1993	1.2882 c (.8131, .2041)	1.3291 c (.8315, .2124)	.7826 c (.5705, 1.074)	.6953 d (.4940, .9786)
Age				
1-44	1.2990 c (.8814, 1.915)	-	-	-
45-54	1.1850 c (.8929, 1.573)	-	-	-
55-64	-	-	-	-
65-74	-	-	3.8422 (3.092, 4.775)	4.0280 (2.950, 5.500)

Table 16 (cont'd):

Gender				
Female	.9024 c (.6810, 1.196)	-	.7827 d (.6375, .9610)	-
Race				
Black	.5863 d (.3705, .9276)	.5471 d (.3368, .8889)	.6144 c (.3649, 1.035)	-
Comorbidity				
1	.7243 (.5050, 1.037)	.7065 c (.4880, 1.023)	.4744 (.3763, .5981)	.5085 (.3501, .7386)
2	.2863 c (.1720, .4767)	.2429 (.1420, .4155)	.3610 (.2621, .4972)	.6038 d (.3815, .9557)
Insurance A				
Other	-	-	.9520 c (.5991, 1.513)	-
Insurance B				
Other	.6647 c (.3220, 1.372)	-	-	-
HMO	.8120 c (.5097, 1.294)	-	-	-
Medicare/aid	.8380 c (.6004, 1.170)	-	-	-

Table 16 (cont'd):

Comorbidity* Age (years)							
1*65-74	-	-	-	-	-	-	1.0511 c (.6364, 1.736)
2*65-74	-	-	-	-	-	-	.4446 d (.2277, .8679)

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 55-64 years for less than 65 years & 75-101 years for 65 years and older; gender, male; race, white; comorbidity, 0; insurance A, medicare; insurance B, commercial; ^c Not significant; and ^d Significant at the 0.05 level.

Table 17: Unadjusted odds ratios a,b on coronary artery bypass grafting utilization and adjusted by statistically significant predictors for other effects of admission as the final model.

Variables	Less than 65 years of age		65 years of age and older	
	Unadjusted	Final Model	Unadjusted	Final Model
Admission Year				
1990	.6127 d (.3796,.9891)	.5943 d (.3308,.9647)	.3804 (.2398,.6037)	.3601 (.2255,.5749)
1991	1.1251 c (.7370,1.717)	1.1982 c (.7805,1.839)	.6559 d (.4410,.9754)	.6993 c (.4669,1.047)
1992	.8704 c (.5598,1.353)	.9171 c (.5870,1.433)	.6745 d (.4617,.9855)	.6634 d (.4512,.9752)
1993	1.2666 c (.8359,1.919)	1.3108 c (.8599,1.998)	.7948 c (.5485,1.152)	.7443 c (.5102,1.087)
Age				
1-44	.7388 c (.4779,1.142)	-	-	-
45-54	.7874 c (.5736,1.081)	-	-	-
55-64	-	-	-	-
65-74	-	-	2.3061 (1.753,3.035)	2.3750 (1.798,3.167)
Gender				
Female	.9059 c (.6579,1.2470)	.8217 c (.5599,1.206)	.7781 c (.5995,1.010)	

Table 17 (cont'd):

Race				
Black	.9629 c (.5542, 1.673)	-	.4213 d (.1803, .9842)	.4017 d (.1701, .9491)
Comorbidity				
1	1.4884 d (1.014, 2.184)	1.9436 (1.244, 3.037)	.8004 c (.5965, 1.074)	-
2	1.5643 c (.8881, 2.755)	.6853 c (.2594, 1.811)	1.0058 c (.6915, 1.463)	-
Insurance A				
Other	-	-	.6933 c (.3610, 1.331)	-
Comorbidity*				
Gender				
1*Female	-	.4389 c (.1715, 1.123)	-	-
2*Female	-	5.3776 (1.529, 18.915)	-	-

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 55-64 years for less than 65 years & 75-101 years for 65 years and older; gender, male; race, white; comorbidity, 0; insurance A, medicare; insurance B, commercial; ^c Not significant; and ^d Significant at the 0.05 level.

Table 18: Unadjusted odds ratios a,b on percutaneous transluminal coronary angioplasty utilization and adjusted by statistically significant predictors for other effects of admission year on utilization as the final model.

Variables	Less than 65 years of age		65 years of age and older	
	Unadjusted	Final Model	Unadjusted	Final Model
Admission Year				
1990	.3975 (.2705,.5840)	.4029 (.2715,.5978)	.4880 (.3118,.7639)	.4347 (.2733,.6913)
1991	.4056 (.2783,.5910)	.3668 (.2494,.5394)	.4921 (.3183,.7608)	.4844 (.2992,1.120)
1992	.6278 d (.5374,1.093)	.5815 (.4009,.8436)	.7160 c (.4865,1.054)	.7476 c (.4992,1.120)
1993	.7666 c (.5374,1.093)	.7764 c (.5386,1.119)	.7330 c (.4979,1.079)	.7115 c (.4749,1.066)
Age ^d				
1-44	.8397 c (.5815,1.212)	.7432 c (.5083,1.087)	-	-
45-54	1.4063 d (1.083,1.827)	1.2895 c (.9816,1.694)	-	-
55-64	-	-	-	-
65-74	-	-	2.6063 (1.948,3.488)	2.2500 (1.665,3.041)

Table 18 (cont'd):

Gender					
Female	1.0396 c (.7965,1.357)	-		.9206 c (.7031,1.205)	-
Race					
Black	.6470 c (.3940,1.062)	-		.4746 c (.2025,1.112)	-
Comorbidity					
1	.3585 (.2385,.5391)	.3495 (.2299,.5312)		.3393 (.2428,.4742)	.3616 (.2572,.5085)
2	.2324 (.1137,.4754)	.2091 (.1012,.4320)		.1915 (.1068,.3433)	.2080 (.1151,.3758)
Insurance A					
Other	-	-		1.3807 c (.7910,2.410)	-
Insurance B					
Other	.9195 c (.4465,1.834)	-		-	-
HMO	.8314 c (.5277,1.310)	-		-	-
Medicare/aid	.6964 d (.5005,.9690)	-		-	-

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows: admission year, 1994; age, 55-64 years for less than 65 years & 75-101 years for 65 years and older; gender, male; race, white; comorbidity, 0; insurance A, medicare; insurance B, commercial; c Not significant; and d Significant at the 0.05 level.

Table 19: Utilization of Thrombolytic Therapy; Change in Chi-Square for patients up to 64 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Age	15.168	8	0.250 < p < 0.500
Admission*Gender	2.507	4	0.500 < p < 0.750
Admission*Race	2.339	4	0.500 < p < 0.750
Admission*Insurance	14.895	12	0.100 < p < 0.250
Admission*Comorbidity	9.099	8	0.250 < p < 0.500
Interaction with Gender			
Gender*Age	0.805	2	0.500 < p < 0.750
Gender*Race	4.645	1	0.025 < p < 0.050
Gender*Insurance	1.572	3	0.500 < p < 0.750
Gender*Comorbidity	1.001	2	0.500 < p < 0.750

Table 20: Utilization of Thrombolytic Therapy; Change in Chi-Square for patients more than 65 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Age	4.151	4	0.250 < p < 0.500
Admission*Gender	0.615	4	0.950 < p < 0.975
Admission*Race	5.163	4	0.250 < p < 0.500
Admission*Medicare	11.847	4	0.010 < p < 0.025
Admission*Comorbidity	12.105	8	0.100 < p < 0.250
Interaction with Age			
Age*Gender	0.750	1	0.500 < p < 0.750
Age*Race	0.460	1	0.250 < p < 0.500
Age*Medicare	0.048	1	0.750 < p < 0.900
Age*Comorbidity	5.031	2	0.050 < p < 0.100
Interaction with Comorbidity			
Comorbidity*Gender	5.153	2	0.050 < p < 0.100
Comorbidity*Race	5.959	2	0.050 < p < 0.100
Comorbidity*Medicare	2.741	2	0.250 < p < 0.500

Table 21: Utilization of Cardiac Catheterization; Change in Chi-Square for patients up to 64 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Age	8.837	8	0.250 < p < 0.500
Admission*Gender	4.750	4	0.250 < p < 0.500
Admission*Race	0.675	4	0.950 < p < 0.975
Admission*Insurance	16.941	12	0.100 < p < 0.250
Admission*Comorbidity	2.666	8	0.950 < p < 0.975
Interaction with Race			
Race*Age	0.148	2	0.900 < p < 0.950
Race*Gender	0.043	1	0.750 < p < 0.900
Race*Insurance	5.618	2	0.050 < p < 0.100
Race*Comorbidity	4.213	2	0.100 < p < 0.250
Interaction with Comorbidity			
Comorbidity*Age	3.301	4	0.500 < p < 0.750
Comorbidity*Gender	5.341	2	0.050 < p < 0.100
Comorbidity*Race	4.213	2	0.100 < p < 0.250
Comorbidity*Insurance	5.623	5	0.250 < p < 0.500

Table 22: Utilization of Cardiac Catheterization; Change in Chi-Square for patients more than 65 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Age	3.802	4	0.250 < p < 0.500
Admission*Gender	1.059	4	0.900 < p < 0.950
Admission*Race	2.435	4	0.250 < p < 0.500
Admission*Medicare	6.107	4	0.100 < p < 0.250
Admission*Comorbidity	4.293	8	0.750 < p < 0.900
Interaction with Age			
Age*Gender	0.691	1	0.250 < p < 0.500
Age*Race	0.061	1	0.750 < p < 0.900
Age*Medicare	0.494	1	0.250 < p < 0.500
Age*Comorbidity	6.409	2	0.025 < p < 0.050
Interaction with Comorbidity			
Comorbidity*Gender	1.340	2	0.500 < p < 0.750
Comorbidity*Race	0.974	2	0.500 < p < 0.750
Comorbidity*Medicare	1.205	2	0.500 < p < 0.750

Table 23: Utilization of Coronary Artery Bypass Grafting: Change in Chi-Square for patients up to 64 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Comorbidity			
Comorbidity*Race	0.201	2	0.900 < p < 0.950
Comorbidity*Gender	12.284	2	0.001 < p < 0.005
Comorbidity*Insurance	12.737	5	0.025 < p < 0.050
Comorbidity*Age	2.991	4	0.500 < p < 0.750
Comorbidity*Admission	7.763	8	0.250 < p < 0.500
Interaction with Admission Yr			
Admission*Age	6.713	8	0.500 < p < 0.750
Admission*Gender	5.504	4	0.100 < p < 0.250
Admission*Race	3.213	4	0.500 < p < 0.750
Admission*Insurance	19.156	12	0.050 < p < 0.100

Table 24: Utilization of Coronary Artery Bypass Grafting; Change in Chi-Square for patients more than 65 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Age			
Age*Race	0.374	1	0.500 < p < 0.750
Age*Gender	0.280	1	0.500 < p < 0.750
Age*Medicare	0.138	1	0.500 < p < 0.750
Age*Admission Year	2.662	4	0.500 < p < 0.750
Age*Comorbidity	3.438	2	0.100 < p < 0.250
Interaction with Race			
Race*Gender	5.072	1	0.010 < p < 0.025
Race*Medicare	0.834	1	0.250 < p < 0.500
Race*Age	0.374	1	0.500 < p < 0.750
Race*Comorbidity	1.429	2	0.250 < p < 0.500
Race*Admission Year	4.647	4	0.250 < p < 0.500
Interaction with Admission Yr			
Admission*Age	2.662	4	0.500 < p < 0.750
Admission*Gender	1.767	4	0.750 < p < 0.900
Admission*Comorbidity	6.630	8	0.500 < p < 0.750
Admission*Medicare	4.361	4	0.250 < p < 0.500

Table 25: Utilization of Percutaneous Transluminal Coronary Angioplasty; Change in Chi-Square for patients up to 64 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission Yr			
Admission*Age	15.942	8	0.025 < p < 0.050
Admission*Gender	1.793	4	0.750 < p < 0.900
Admission*Race	0.235	4	0.990 < p < 0.995
Admission*Insurance	5.178	12	0.950 < p < 0.975
Admission*Comorbidity	8.711	8	0.250 < p < 0.500
Interaction with Age			
Age*Gender	0.426	2	0.750 < p < 0.900
Age*Race	2.682	2	0.250 < p < 0.500
Age*Insurance	4.739	6	0.500 < p < 0.750
Age*Comorbidity	2.538	4	0.500 < p < 0.750
Interaction with Comorbidity			
Comorbidity*Gender	3.732	2	0.100 < p < 0.250
Comorbidity*Race	2.266	2	0.250 < p < 0.500
Comorbidity*Insurance	6.152	5	0.250 < p < 0.500

Table 26: Utilization of Percutaneous Transluminal Coronary Angioplasty; Change in Chi-Square for patients more than 65 years of age

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission Yr			
Admission*Age	8.556	4	0.050 < p < 0.100
Admission*Gender	0.636	4	0.950 < p < 0.975
Admission*Race	7.957	4	0.050 < p < 0.100
Admission*Medicare	1.268	4	0.750 < p < 0.900
Admission*Comorbidity	5.406	8	0.750 < p < 0.900
Interaction with Age			
Age*Gender	0.270	1	0.500 < p < 0.750
Age*Race	3.512	1	0.050 < p < 0.100
Age*Medicare	0.132	1	0.500 < p < 0.750
Age*Comorbidity	3.310	2	0.100 < p < 0.250
Interaction with Comorbidity			
Comorbidity*Gender	2.210	2	0.250 < p < 0.500
Comorbidity*Race	1.904	2	0.250 < p < 0.500
Comorbidity*Medicare	1.871	2	0.250 < p < 0.500

Table 27: Unadjusted odds ratios a,b on thrombolytic therapy utilization across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted
Admission Year					
1990	.3632	.3471	.3576	.3634	.3584
1991	.9799 c	.9949 c	.9916 c	.9822 c	.9483 c
1992	1.1037 c	1.1083 c	1.1183 c	1.1006 c	1.1318 c
1993	.9897 c	.9592 c	1.0079 c	.9934 c	1.0176 c
Age					
1-44	-	2.8952	-	-	-
45-54	-	2.3102	-	-	-
55-64	-	2.9439	-	-	-
65-74	-	2.1771	-	-	-
Gender					
Female	-	-	.6904	-	-
Race					
Black	-	-	-	.7365 c	-
Comorbidity					
1	-	-	-	-	.5137
2	-	-	-	-	.3644

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; **b** The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; **c** Not significant; and **d** Significant at the 0.05 level.

Table 28: Unadjusted odds ratios a,b on cardiac catheterization utilization across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted
Admission Year					
1990	.4552	.3817	.4453	.4554	.4319
1991	.4622	.4234	.4651	.4631	.4177
1992	.4788	.4350	.4822	.4762	.4725
1993	.9164 c	.8472 c	.9354 c	.9212 c	.9576 c
Age					
1-44	-	7.4259	-	-	-
45-54	-	6.6226	-	-	-
55-64	-	5.4824	-	-	-
65-74	-	3.9421	-	-	-
Gender					
Female	-	-	.6590	-	-
Race					
Black	-	-	-	.9212 d	-
Comorbidity					
1	-	-	-	-	.4116
2	-	-	-	-	.2497

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; c Not significant; and d Significant at the 0.05 level.

Table 29: Unadjusted odds ratios a,b on coronary artery bypass grafting utilization across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted
Admission Year					
1990	.4751	.4612	.4710	.4755	.4755
1991	.8429 c	.8693 c	.8489 c	.8450 c	.8453
1992	.7527 c	.7416 d	.7582 c	.7502 c	.7515
1993	.9769 c	.9373 c	.9879 c	.9808 c	.9728
Age					
1-44	-	1.5468 c	-	-	-
45-54	-	1.6150	-	-	-
55-64	-	2.0582	-	-	-
65-74	-	2.3404	-	-	-
Gender					
Female	-	-	.7971 d	-	-
Race					
Black	-	-	-	.7172 c	-
Comorbidity					
1	-	-	-	-	.9697
2	-	-	-	-	1.1270 c

a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; c Not significant; and d Significant at the 0.05 level.

Table 30: Unadjusted odds ratios ^{a,b} on percutaneous transluminal coronary angioplasty across all ages, and individually adjusted by age, gender, race, and comorbidity for other effects of admission year on utilization.

Variables	Unadjusted	Age-Adjusted	Gender-Adjusted	Race-Adjusted	Comorbidity-Adjusted
Admission Year					
1990	.4623	.4235	.4576	.4626	.4388
1991	.4678	.4429	.4707	.4687	.4209
1992	.6689	.6596	.6739	.6661	.6753
1993	.7586 d	.7294 d	.7676 d	.7618 d	.7788
Age					
1-44	-	3.9295	-	-	-
45-54	-	6.4765	-	-	-
55-64	-	4.5046	-	-	-
65-74	-	2.6013	-	-	-
Gender					
Female	-	-	.7779	-	-
Race					
Black	-	-	-	.6870 c	-
Comorbidity					
1	-	-	-	-	.2736
2	-	-	-	-	.1557

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; ^c Not significant; and ^d Significant at the 0.05 level.

Table 31 : Unadjusted and fully-adjusted odds ratios ^{a,b} on thrombolytic therapy utilization across all ages.

Variables	Unadjusted	Fully-adjusted
Admission Year		
1990	.3632 (.2496, .5284)	.3468 (.2373, .5066)
1991	.9799 c (.7309, 1.314)	.9799 c (.7267, 1.321)
1992	1.1037 c (.8318, 1.465)	1.1217 c (.8405, 1.497)
1993	.9897 c (.7427, 1.319)	.9940 c (.7415, 1.332)
Age (years)		
1-44	2.6941 (1.753, 4.140)	2.1564 (1.374, 3.383)
45-54	2.2439 1.586, 3.175)	1.7386 (1.204, 2.510)
55-64	2.8521 (2.114, 3.848)	2.3775 (1.736, 3.257)
65-74	2.1216 (1.574, 2.860)	1.9457 (1.433, 2.642)
Gender		
Female	.7171 (.5844, .8806)	.8333 c (.6717, 1.034)
Race		
Black	.7321 c (.4604, 1.164)	.7226 c (.4498, 1.161)
Comorbidity Index		
1	.5193 (.40311, .6689)	.6068 (.4649, .7920)
2	.3779 (.2517, .5673)	.4567 (.3003, .6946)

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; ^c Not significant; and ^d Significant at the 0.05 level.

Table 32: Unadjusted and fully-adjusted odds ratios ^{a,b} on cardiac catheterization utilization across all ages.

Variables	Unadjusted	Fully-adjusted
Admission Year		
1990	.4552 (.3538, .5857)	.3758 (.2856, .4943)
1991	.4622 (.3608, .5921)	.4027 (.3073, .5278)
1992	.4788 (.3754, .6106)	.4316 (.3309, .5629)
1993	.9164 ^c (.7133, 1.177)	.8877 ^c (.6761, 1.165)
Age (years)		
1-44	6.7780 (4.611, 9.964)	5.6866 (3.788, 8.537)
45-54	6.1827 (4.676, 8.175)	5.0931 (3.773, 6.876)
55-64	5.2177 (4.137, 6.581)	4.6146 (3.602, 5.912)
65-74	3.8422 (3.092, 4.775)	3.6276 (2.890, 4.554)
Gender		
Female	.6738 (.5752, .7893)	.9395 ^c (.7856, 1.123)
Race		
Black	.7016 ^d (.5029, .9788)	.5814 (.4030, .8391)
Comorbidity Index		
1	.4271 (.3553, .5134)	.5748 (.4686, .7050)
2	.2759 (.2117, .3598)	.3670 (.2741, .4915)

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; ^c Not significant; and ^d Significant at the 0.05 level.

Table 33 : Unadjusted and fully-adjusted odds ratios ^{a,b} on coronary artery bypass grafting utilization across all ages.

Variables	Unadjusted	Fully-adjusted
Admission Year		
1990	.4751 (.3414, .6611)	.4587 (.3286, .6402)
1991	.8429 c (.6321, 1.123)	.8845 c (.6610, 1.184)
1992	.7527 c (.5647, 1.003)	.7393 d (.5529, .9884)
1993	.9769 c (.7413, 1.287)	.9382 c (.7093, 1.241)
Age (years)		
1-44	1.5000 c (.9535, 2.360)	1.6218 d (1.012, 2.598)
45-54	1.5987 (1.138, 2.247)	1.6651 (1.161, 2.387)
55-64	2.0305 (1.524, 2.706)	2.1028 (1.556, 2.843)
65-74	2.3061 (1.753, 3.034)	2.3762 (1.795, 3.146)
Gender		
Female	.8144 d (.6679, .9931)	.8469 c (.6887, 1.041)
Race		
Black	.7247 c (.4596, 1.143)	.6886 c (.4341, 1.092)
Comorbidity Index		
1	.9660 c (.7695, 1.213)	1.0827 c (.8507, 1.378)
2	1.1412 c (.8406, 1.549)	1.3587 c (.9836, 1.877)

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; ^c Not significant; and ^d Significant at the 0.05 level.

Table 34 : Unadjusted and fully-adjusted odds ratios ^{a,b} on percutaneous transluminal coronary angioplasty across all ages.

Variables	Unadjusted	Fully-adjusted
Admission Year		
1990	.4623 (3.478, .6145)	.4182 (.3098, .5645)
1991	.4678 (.3544, .6175)	.4073 (.3035, .5466)
1992	.6689 (.5174, .8646)	.6446 (.4901, .8478)
1993	.7586 d (.5892, .9767)	.7457 d (.5693, .9767)
Age (years)		
1-44	3.7075 (2.466, 5.572)	2.6881 (1.753, 4.123)
45-54	6.2114 (4.531, 8.515)	4.6243 (3.302, 6.476)
55-64	4.4166 (3.308, 5.897)	3.5656 (2.626, 4.841)
65-74	2.6063 (1.948, 3.488)	2.2841 (1.690, 3.089)
Gender		
Female	.7860 (.6558, .9421)	1.1382 c (.9318, 1.390)
Race		
Black	.6854 c (.4523, 1.039)	.6154 d (.3955, .9576)
Comorbidity Index		
1	.2826 (.2196, .3636)	.3575 (.2744, .4656)
2	.1667 (.1067, .2606)	.2085 (.1318, .3300)

^a Unless otherwise indicated, odds ratios are significant for $P < 0.01$; ^b The reference groups for the variables are as follows; admission year, 1994; age, 75-101 years; gender, male; race, white; and comorbidity, 0; ^c Not significant; and ^d Significant at the 0.05 level.

Table 35: Utilization of Thrombolytic Therapy; Change in Chi-Square across all ages

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Comorbidity	15.890	8	0.025 < p < 0.050
Admission*Age	22.428	16	0.100 < p < 0.250
Admission*Gender	2.450	4	0.500 < p < 0.750
Admission*Race	5.346	4	0.250 < p < 0.500
Interaction with Comorbidity			
Comorbidity*Age	9.001	8	0.250 < p < 0.500
Comorbidity*Gender	5.839	2	0.050 < p < 0.100
Comorbidity*Race	6.780	2	0.025 < p < 0.050
Interaction with Age			
Age*Gender	8.863	4	0.050 < p < 0.100
Age*Race	3.476	4	0.250 < p < 0.500

Table 36: Utilization of Cardiac Catheterization; Change in Chi-Square across all ages

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Race	1.533	4	0.750 < p < 0.900
Admission*Comorbidity	4.533	8	0.750 < p < 0.900
Admission*Age	20.344	16	0.100 < p < 0.250
Admission*Gender	0.937	4	0.900 < p < 0.950
Interaction with Comorbidity			
Comorbidity*Race	0.044	2	0.975 < p < 0.990
Comorbidity*Age	15.080	8	0.050 < p < 0.100
Comorbidity*Gender	0.823	2	0.500 < p < 0.750
Interaction with Age			
Age*Gender	4.905	4	0.250 < p < 0.500
Age*Race	0.233	4	0.990 < p < 0.995
Interaction with Race			
Race*Gender	1.195	1	0.250 < p < 0.500

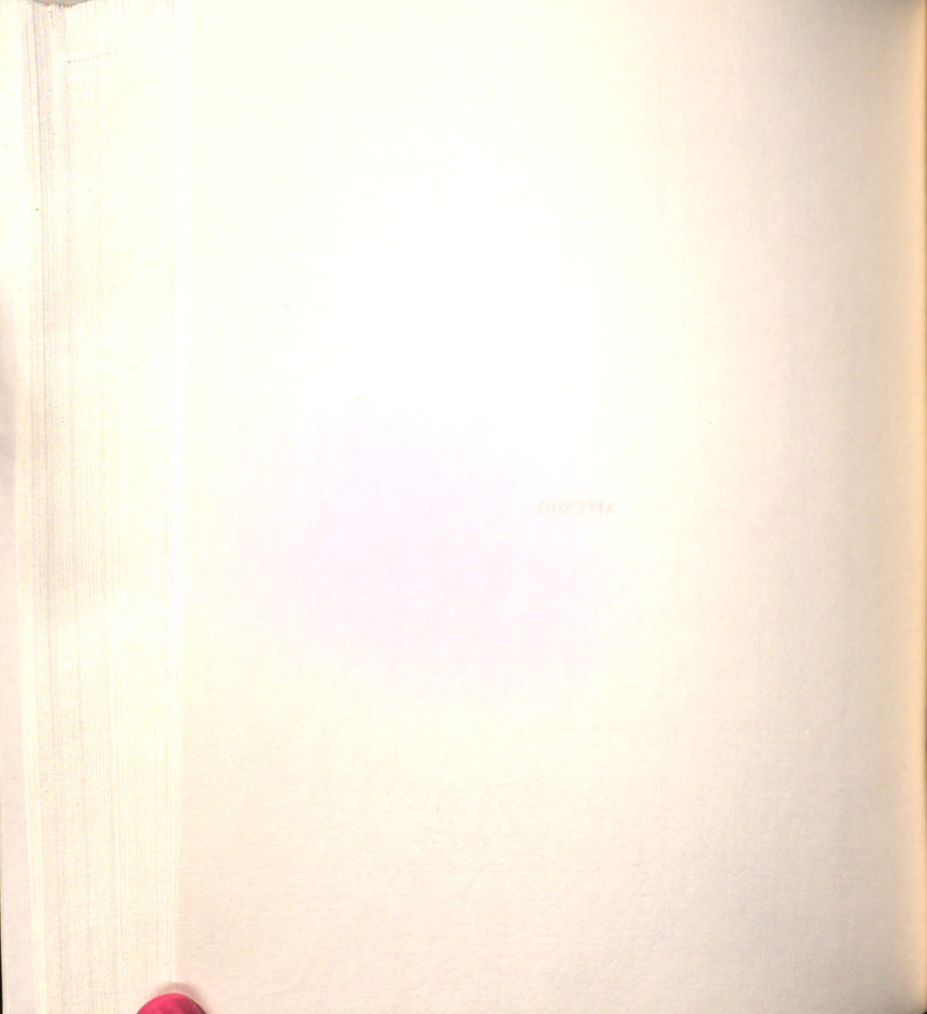
Table 37: Utilization of Percutaneous Transluminal Coronary Angioplasty; Change in Chi-Square across all ages

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Race	1.919	4	0.750 < p < 0.900
Admission*Comorbidity	3.960	8	0.750 < p < 0.900
Admission*Age	26.481	16	0.025 < p < 0.050
Admission*Gender	1.242	4	0.750 < p < 0.900
Interaction with Comorbidity			
Comorbidity*Race	2.967	2	0.100 < p < 0.250
Comorbidity*Age	5.928	8	0.500 < p < 0.750
Comorbidity*Gender	5.680	2	0.050 < p < 0.100
Interaction with Age			
Age*Gender	0.652	4	0.950 < p < 0.975
Age*Race	6.599	4	0.100 < p < 0.250
Interaction with Race			
Race*Gender	0.665	1	0.250 < p < 0.500

Table 38: Utilization of Coronary Artery Bypass Grafting; Change in Chi-Square across all ages

Variable	Change in Chi-Square	Degrees of freedom	p-value
Interaction with Admission			
Admission*Comorbidity	5.349	8	0.500 < p < 0.750
Admission*Age	14.324	16	0.500 < p < 0.750
Admission*Gender	2.682	4	0.500 < p < 0.750
Admission*Race	3.502	4	0.250 < p < 0.500
Interaction with Age			
Age*Comorbidity	9.801	8	0.250 < p < 0.500
Age*Gender	2.156	4	0.500 < p < 0.750
Age*Race	7.015	4	0.100 < p < 0.250

APPENDIX I



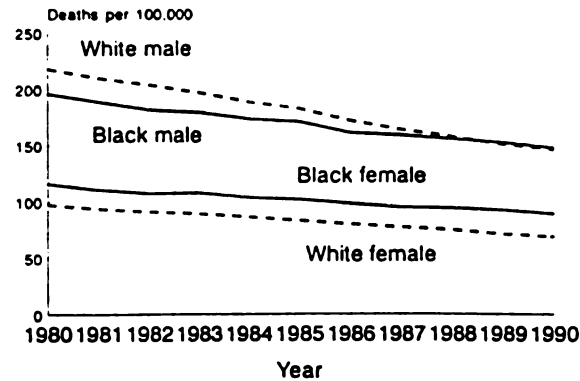


Figure 1: Age-adjusted death rates per 100,000 for ischemic heart disease by gender and race: United States.

Source: Gillum, RF. Trends in Acute Myocardial Infarction and Coronary Heart Disease Death in the United States. American College of Cardiology 1994; 23:1274.

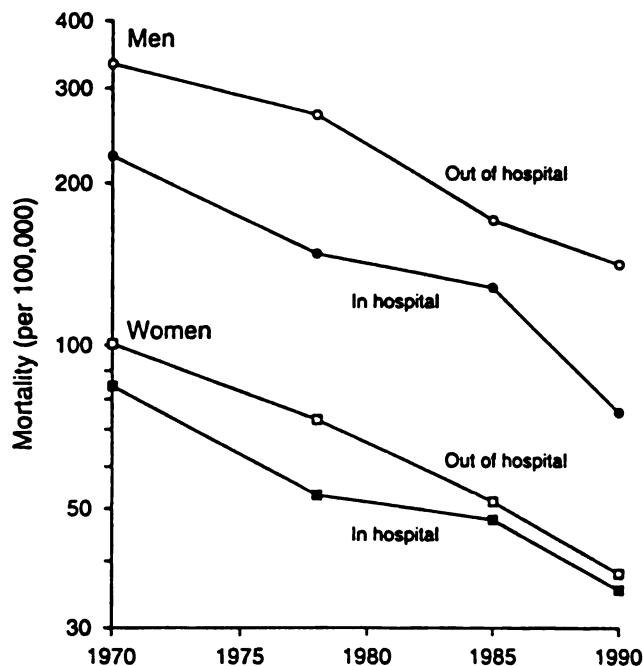


Figure 2: Trends in Mortality Due to Coronary Heart Disease from 1970 to 1990, According to the Location of Death, among Residents of the Twin Cities Area Who Were 30 to 74 Years of Age.

Source: McGovern PG, Pankow JS, Shahar E, et al. Recent Trends in Acute Coronary Heart Disease. The New England Journal of Medicine 1996; 334:886.

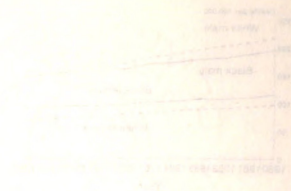


Figure 1: Trends in Death Rates for Coronary Heart Disease, by Race and Sex, 1950-1980. Source: National Center for Health Statistics, *Health Statistics*, 1981, Table 1-1.1.

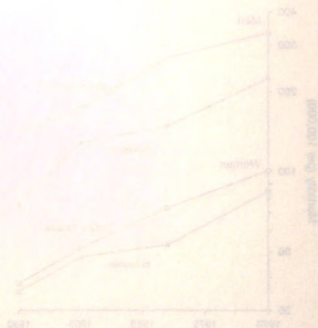


Figure 2: Trends in Death Rates for Coronary Heart Disease, by Age Group, 1950-1980. Source: National Center for Health Statistics, *Health Statistics*, 1981, Table 1-1.2.

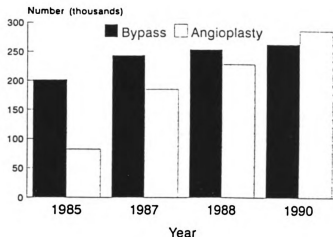


Figure 3: Number of coronary artery bypass graft (ICD-9-CM 36.1) and coronary angioplasty procedures (removal of coronary obstruction, ICD-9-CM 36.0): United States.

Source: Gillum, RF. Trends in Acute Myocardial Infarction and Coronary Heart Disease Death in the United States. American College of Cardiology 1994; 23: 1276.

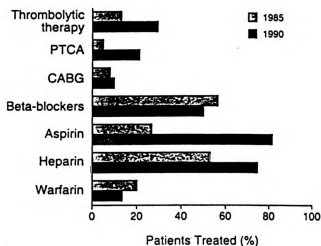


Figure 4: Trends in Acute Medical Care for Residents of the Twin Cities Area, 30 to 74 Years of Age, Who Were Hospitalized for Definite Acute Myocardial Infarction in 1985 and 1990.

Source: McGovern PG, Pankow JS, Shahar E, et al. Recent Trends in Acute Coronary Heart Disease. The New England Journal of Medicine 1996; 334: 888.

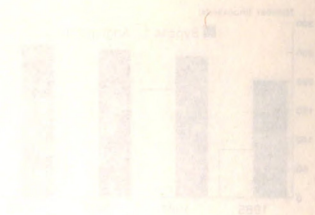


Figure 3: Number of cases of various types of meningitis in the United States from 1965 to 1975. The Y-axis represents the number of cases (0 to 2000). The X-axis represents the year (1965, 1970, 1975). The legend indicates: Cryptococcus (dark grey), Meningococcus (light grey), Pneumococcus (white), and Tuberculosis (hatched).

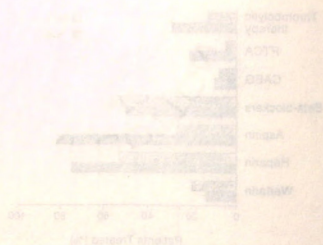


Figure 4: Trends in Acute Meningitis Cases and the Incidence of the Twin Cities Area, 1965 to 1975. The Y-axis represents the percentage of cases (0 to 100). The X-axis represents the year (1965, 1970, 1975). The legend indicates: Cryptococcus (dark grey), Meningococcus (light grey), Pneumococcus (white), and Tuberculosis (hatched).

FIGURE 5
AMI ADMISSION BY AGE
1990-1994

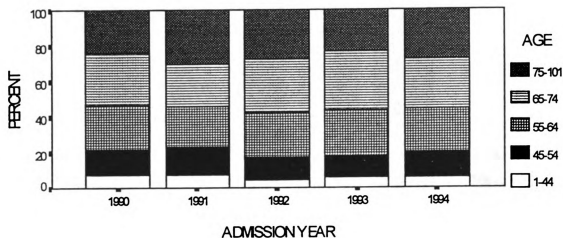


FIGURE 6
AMI ADMISSION BY GENDER
1990-1994

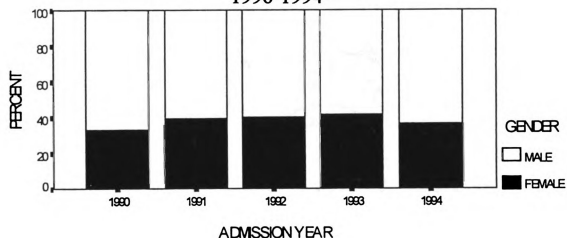
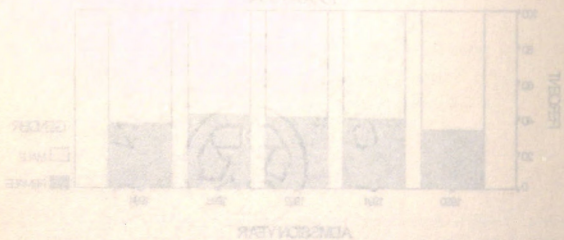


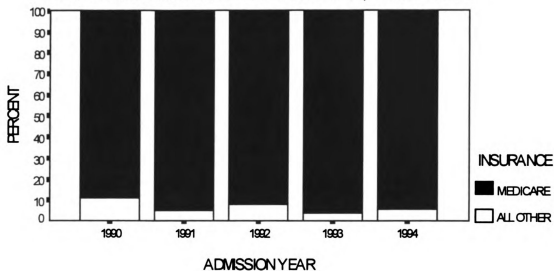
FIGURE 2
AMI ADMISSION BY AGE
1990-1994



FIGURE 3
AMI ADMISSION BY GENDER
1990-1994



**FIGURE 7: % 65 YEARS OF AGE & OLDER
AMI ADMISSION BY INSURANCE, 1990-1994**



**FIGURE 8: % LESS THAN 65 YEARS OF AGE
AMI ADMISSION BY INSURANCE, 1990-1994**

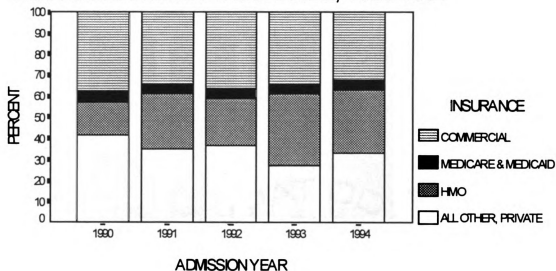


FIGURE 7: % 65 YEARS OF AGE & OLDER
AMI ADMISSION BY INSURANCE, 1980-1994

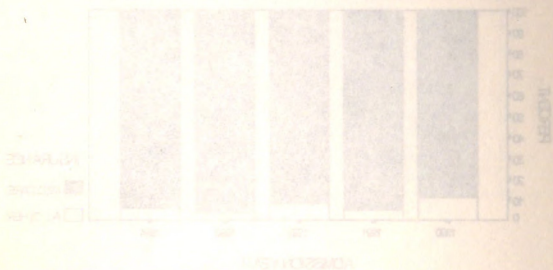


FIGURE 8: % LESS THAN 65 YEARS OF AGE
AMI ADMISSION BY INSURANCE, 1980-1994

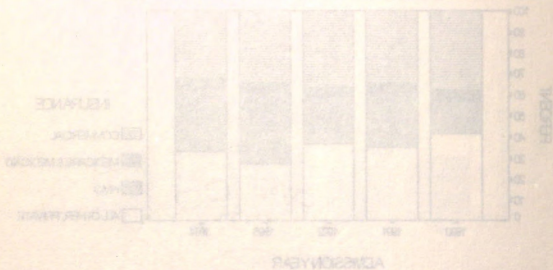


FIGURE 9
AMI ADMISSION BY INSURANCE, 1990-1994

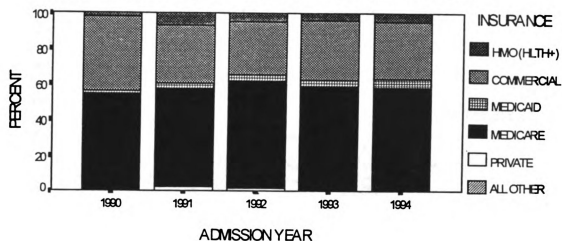


FIGURE 10
AMI ADMISSION BY RACE, 1990-1994

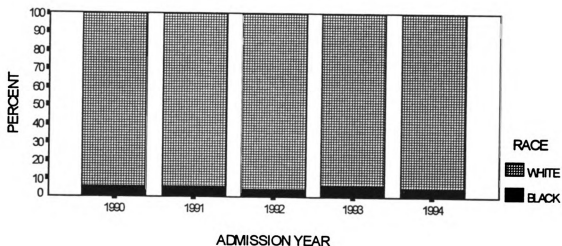


FIGURE 9
AMI ADMISSION BY INSURANCE TYPE, 1997-1998



FIGURE 10
AMI ADMISSION BY RACE, 1997-1998



FIGURE 11
AMI ADMISSION BY COMORBIDITY

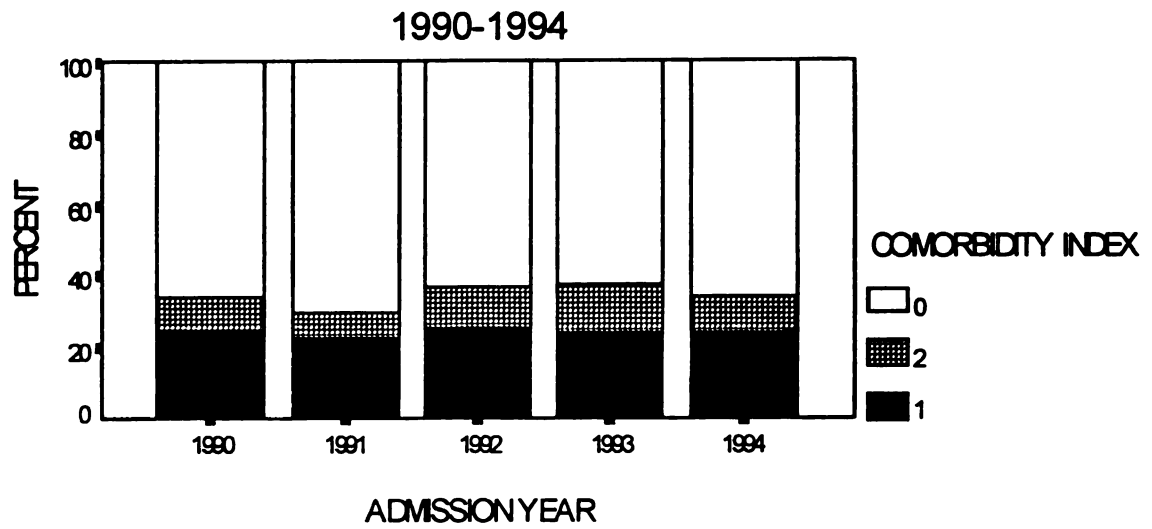


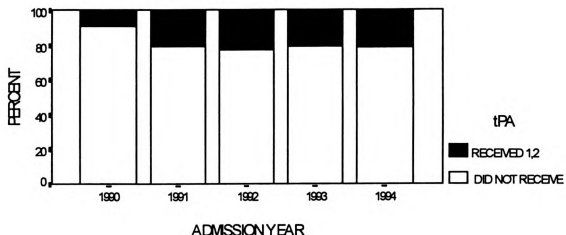
FIGURE 11

AMM ADMISSION BY COMPOSITION

1990-1995



**FIGURE 12: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION, 1990-1994**



**FIGURE 13: % RECEIVING CARDIAC CATH
AMI ADMISSION, 1990-1994**

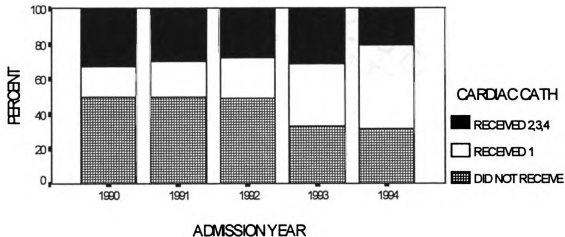


FIGURE 12: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION, 1990-1991

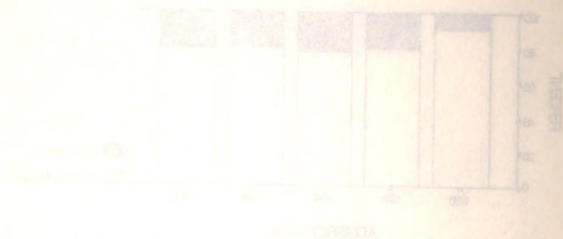
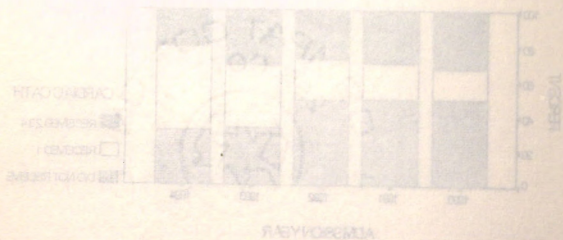
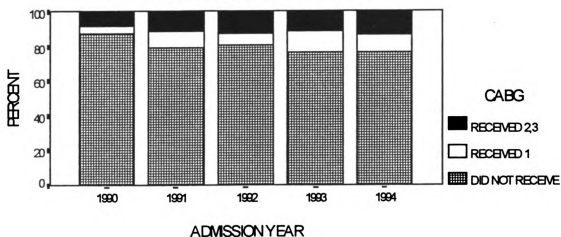


FIGURE 13: % RECEIVING CATHETERIZATION
AMI ADMISSION, 1990-1991



**FIGURE 14: % RECEIVING CABG
AMI ADMISSION, 1990-1994**



**FIGURE 15: % RECEIVING PTCA
AMI ADMISSION, 1990-1994**

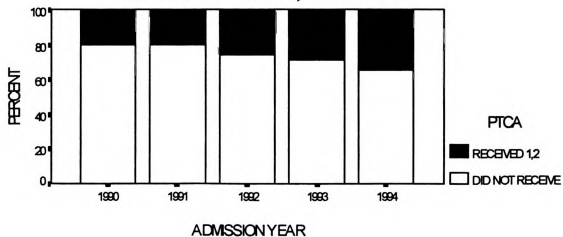


FIGURE 14: RECEIVING CARD
AND ADMISSION, 1960-1964

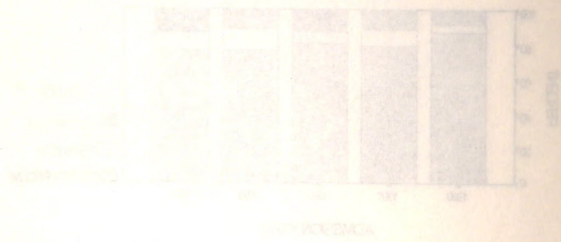
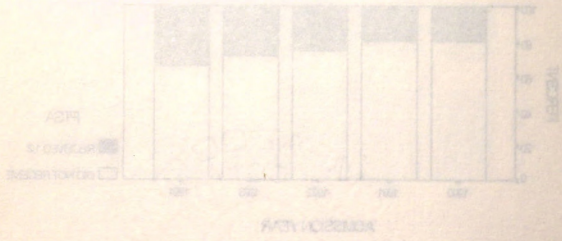
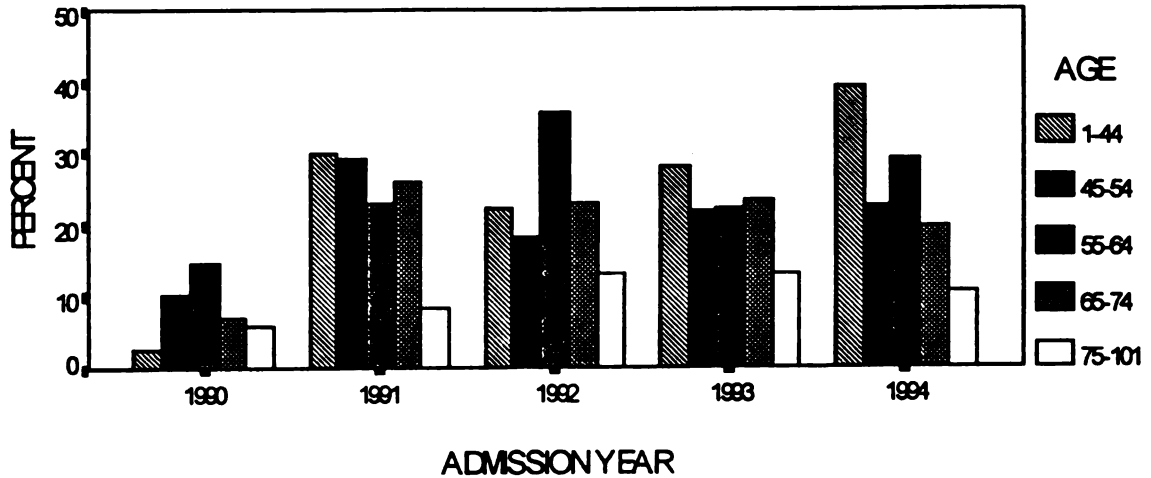


FIGURE 12: RECEIVING CARD
AND ADMISSION, 1960-1964



**FIGURE 16: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION BY AGE, 1990-1994**



**FIGURE 17: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION BY GENDER, 1990-1994**

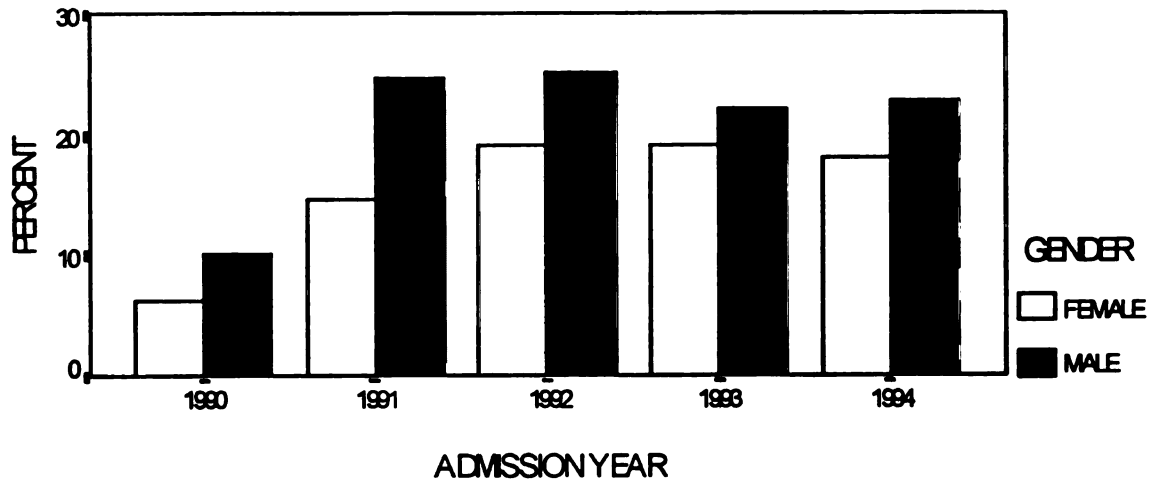
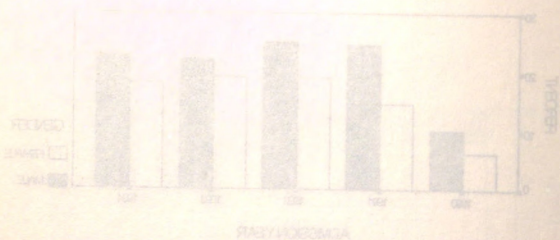


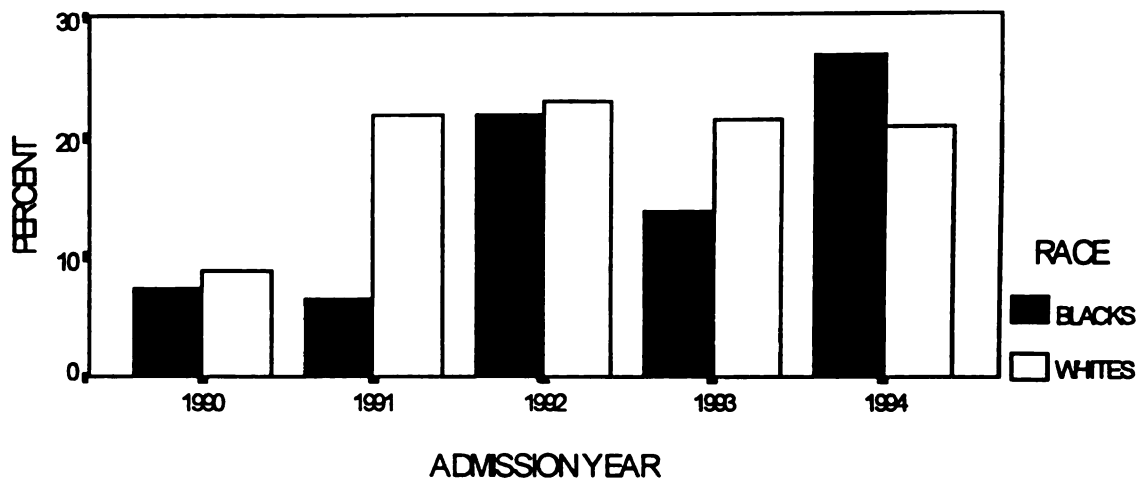
FIGURE 16: % RECEIVING THROUGHOUT THERAPY
AMI ADMISSION BY AGE GROUP



FIGURE 17: % RECEIVING THROUGHOUT THERAPY
AMI ADMISSION BY GENDER



**FIGURE 18: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION BY RACE, 1990-1994**



**FIGURE 19: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION BY INSURANCE, 1990-1994**

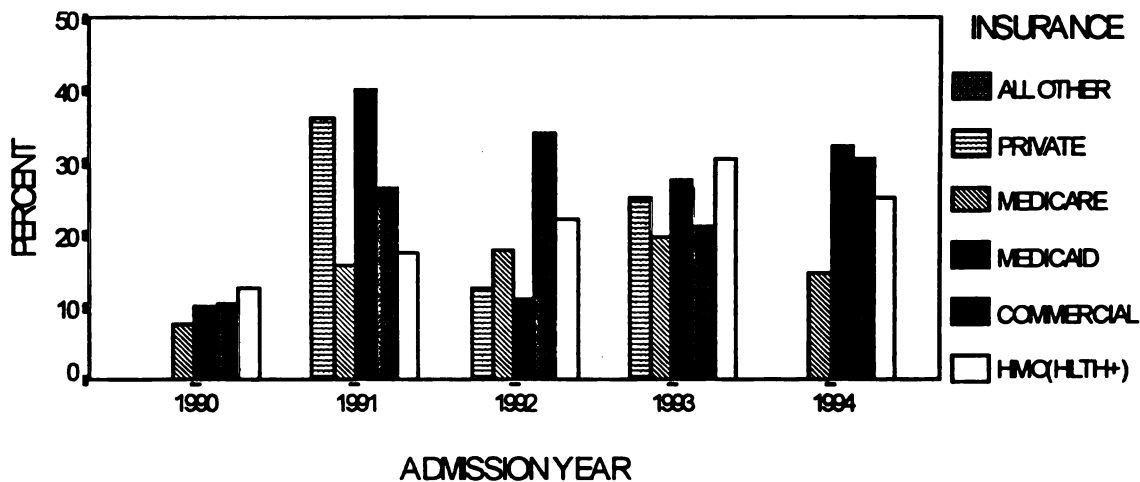


FIGURE 18: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION BY YEAR, 1997-1999



FIGURE 19: % RECEIVING THROMBOLYTIC THERAPY
AMI ADMISSION BY INSURANCE TYPE, 1997-1999



**FIGURE 20: % RECEIVING THROMBO THERAPY
AMI ADMISSION BY COMORBIDITY, 1990-1994**

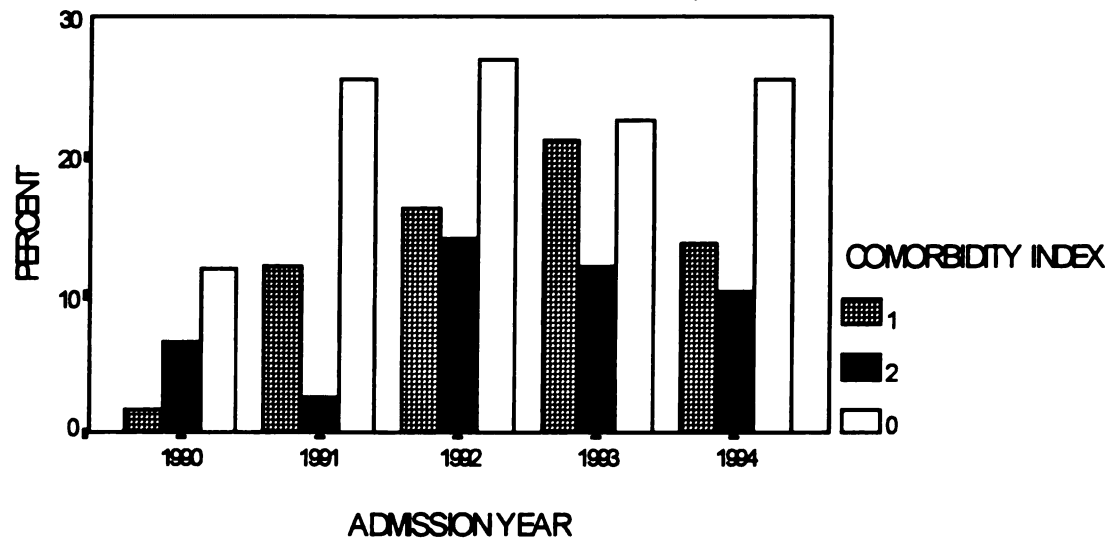
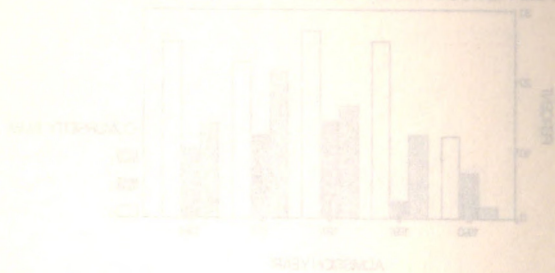
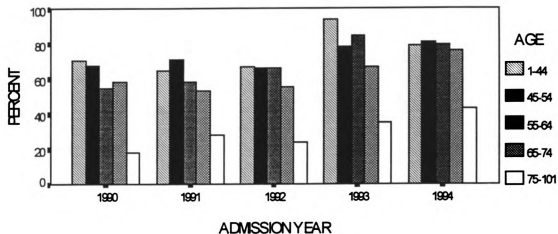


FIGURE 20: % RECEIVING THERAPY
AM ADMISSION BY COMORBIDITY, 1990-1994



**FIGURE 21: % RECEIVING CARDIAC CATH
AMI ADMISSION BY AGE, 1990-1994**



**FIGURE 22: % RECEIVING CARDIAC CATH
AMI ADMISSION BY GENDER, 1990-1994**

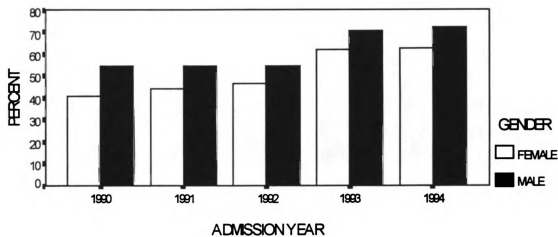
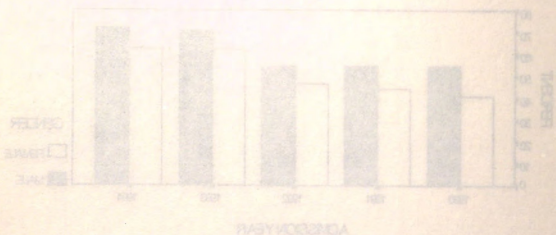


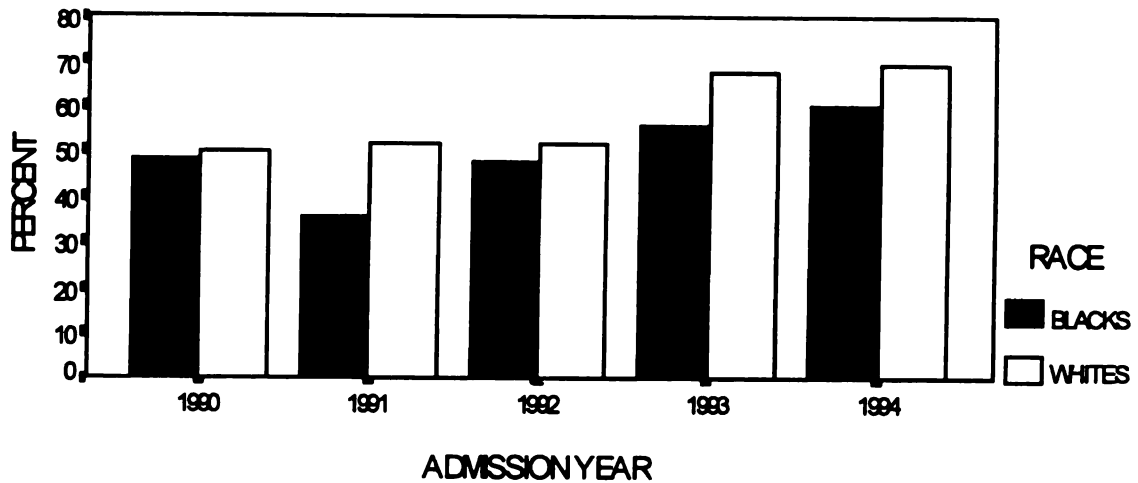
FIGURE 21: % RECEIVING CARDIAC CATH
AMI ADMISSION BY AGE 1991-1994



FIGURE 22: % RECEIVING CARDIAC CATH
AMI ADMISSION BY GENDER 1991-1994



**FIGURE 23: % RECEIVING CARDIAC CATH
AMI ADMISSION BY RACE, 1990-1994**



**FIGURE 24: % RECEIVING CARDIAC CATH
AMI ADMISSION BY INSURANCE, 1990-1994**

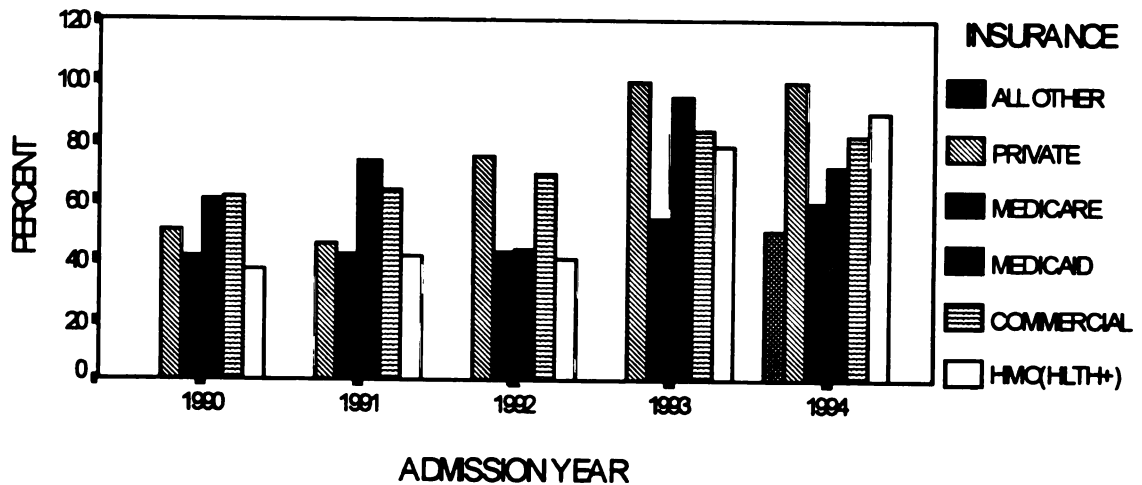
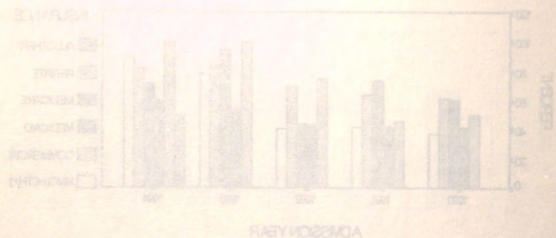


FIGURE 23: % RECEIVING CARDIAC CATH
AMI ADMISSION BY RACE, 1990-1994



FIGURE 24: % RECEIVING CARDIAC CATH
AMI ADMISSION BY RACE, 1990-1994



**FIGURE 25: % RECEIVING CARDIAC CATH
AMI ADMISSION BY COMORBIDITY, 1990-1994**

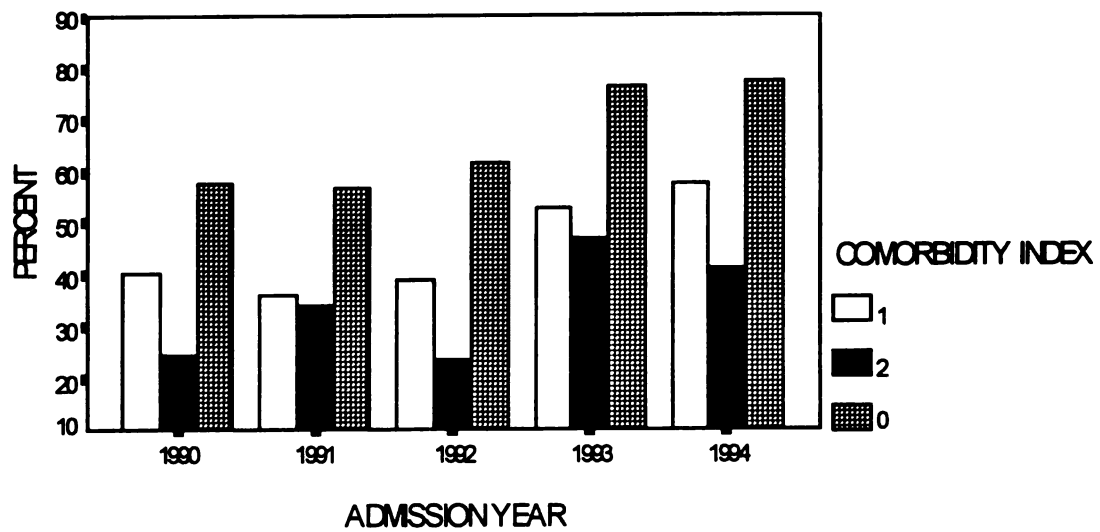
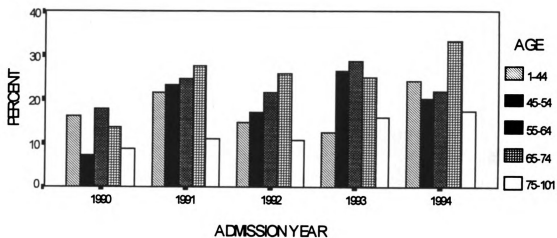


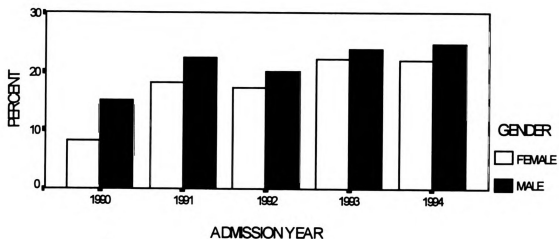
FIGURE 25: % RECEIVING CARDIAC CATH
 AND ADMISION BY COMORBIDITY, 1990-1991



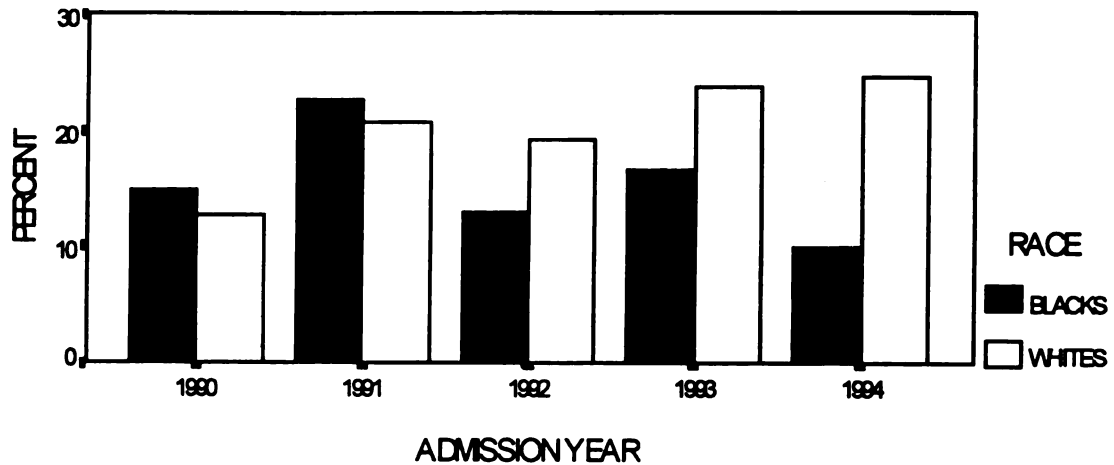
**FIGURE 26: % RECEIVING CABG
AMI ADMISSION BY AGE, 1990-1994**



**FIGURE 27: % RECEIVING CABG
AMI ADMISSION BY GENDER, 1990-1994**



**FIGURE 28: % RECEIVING CABG
AMI ADMISSION BY RACE, 1990-1994**



**FIGURE 29: % RECEIVING CABG
AMI ADMISSION BY INSURANCE, 1990-1994**

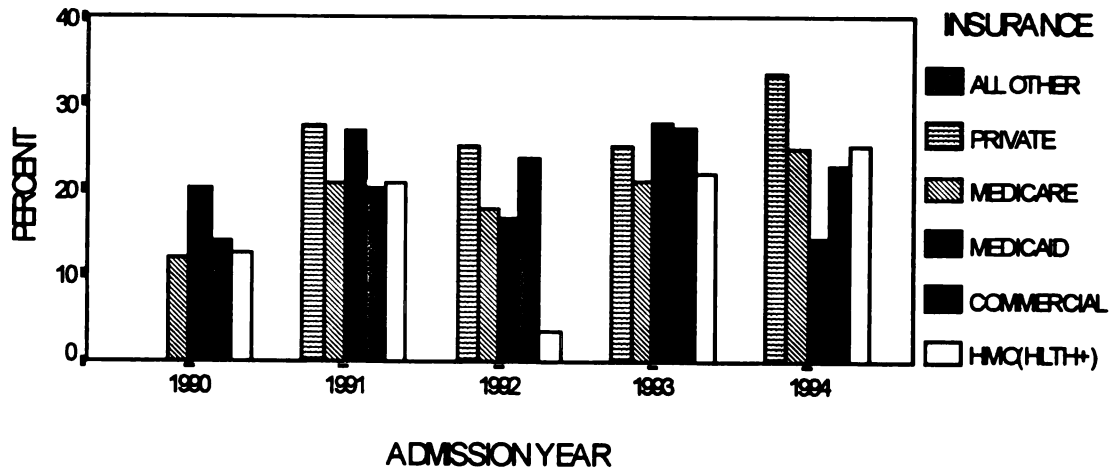


FIGURE 28: RECEIVING CASE
AMI ADMISSION BY RACE, 1990-1994

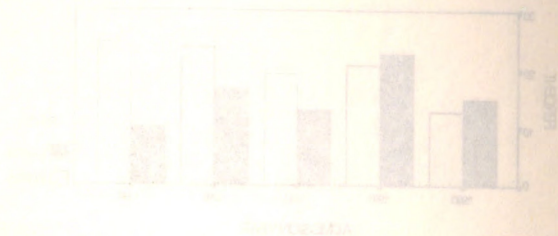


FIGURE 29: RECEIVING CASE
AMI ADMISSION BY INST. TYPE, 1990-1994

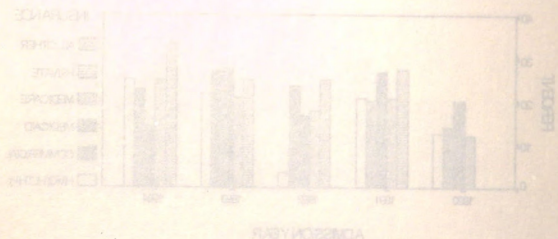


FIGURE 30: % RECEIVING CABG
AMI ADMISSION BY COMORBIDITY, 1990-1994

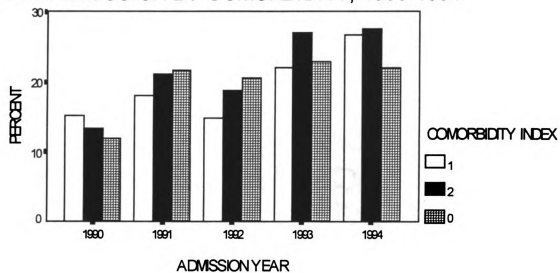
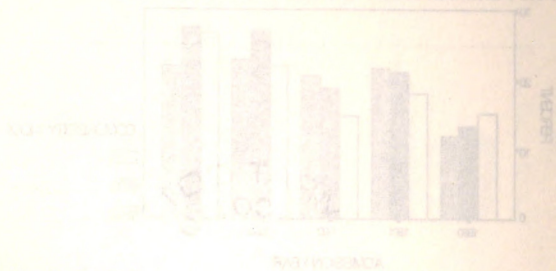
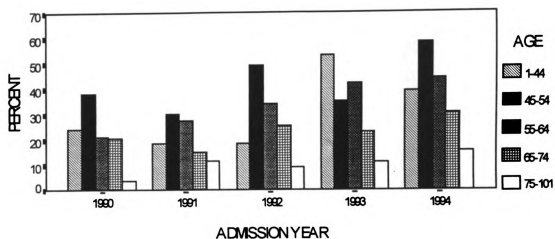


FIGURE 30: % RECEIVING CARE
AMMADITION BY COMORBIDITY, 1990-1994



**FIGURE 31: % RECEIVING PTCA
AMI ADMISSION BY AGE, 1990-1994**



**FIGURE 32: % RECEIVING PTCA
AMI ADMISSION BY GENDER, 1990-1994**

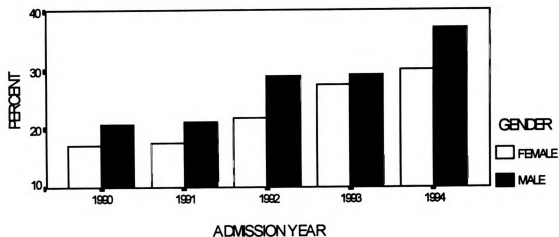
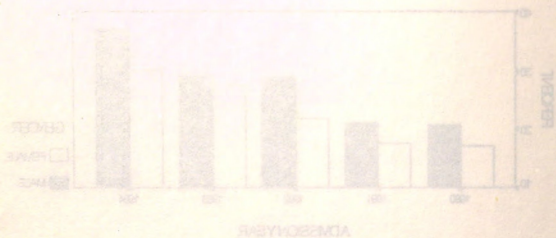


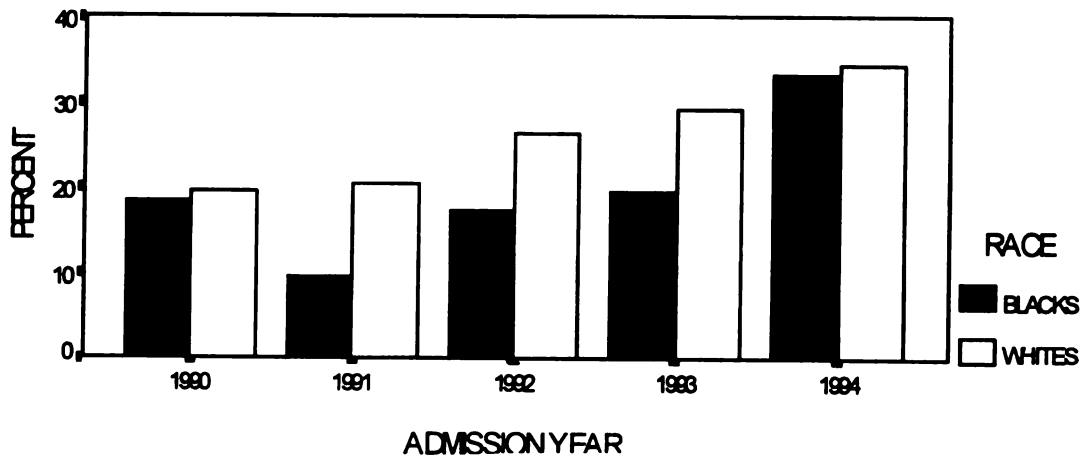
FIGURE 31. 4 RECEIVING PTCA
AMI ADMISSION BY AGE, 1990-1994



FIGURE 32. 4 RECEIVING PTCA
AMI ADMISSION BY GENDER, 1990-1994



**FIGURE 33: % RECEIVING PTCA
AMI ADMISSION BY RACE, 1990-1994**



**FIGURE 34: % RECEIVING PTCA
AMI ADMISSION BY INSURANCE, 1990-1994**

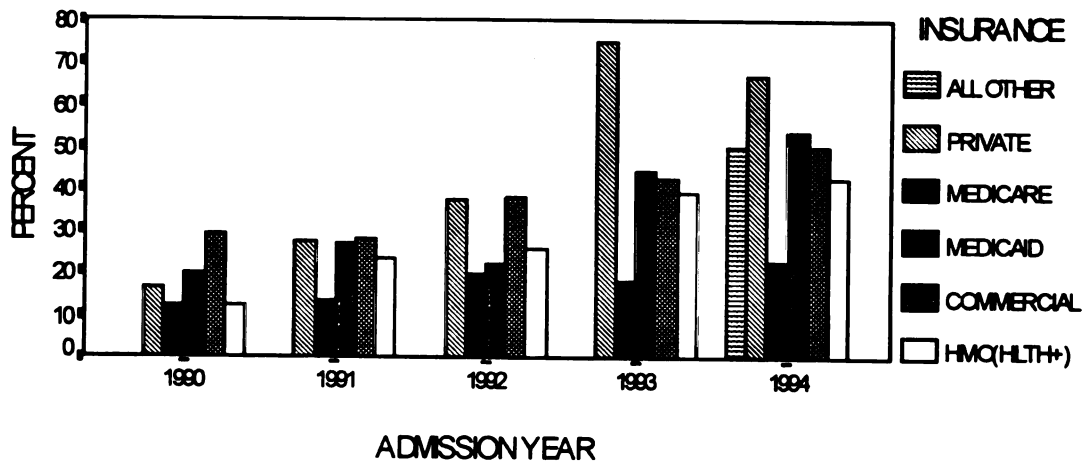


FIGURE 11 - RECEIVING BY
AND ADMISSION BY RATE 1970-1980



FIGURE 12 - RECEIVING BY
AND ADMISSION BY RATE 1970-1980



**FIGURE 35: % RECEIVING PTCA
AMI ADMISSION BY COMORBIDITY, 1990-1994**

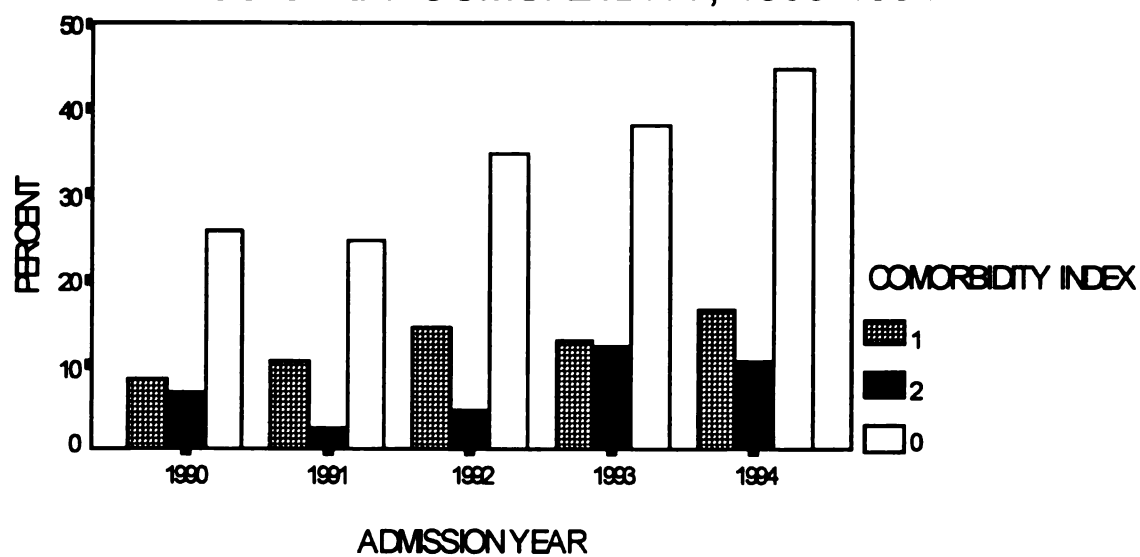


FIGURE 35. % RECEIVING PTO
AMI ADMISSION BY COMORBIDITY, 1980-1994



LIST OF REFERENCES



LIBRARY OF THE

LIST OF REFERENCES

1. Margolis JR, Kannel WB, Feinleib M et al: Clinical features of unrecognized myocardial infarction-silent and symptomatic: eighteen year follow-up: the Framingham study. *Am J Cardio.* 32:1, 1973.
2. Solomon CG, Lee TH, Cook EF, et al. Comparison of Clinical Presentation of Acute Myocardial Infarction in Patients Older than 65 Years of Age to Younger Patients: The Multicenter Chest Pain Study Experience. *The American Journal of Cardiology.* 1989; 63:772-776.
3. Gunnar RM, Bourdillon PDV, Dixon DW, et al. ACC/AHA Guidelines for the Early Management of Patients With Acute Myocardial Infarction. A report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee to develop guidelines for the early management of patients with acute myocardial infarction). *Circulation* 1990; 82(no.2):664-707.
4. CDC, Trends in Ischemic Heart Disease Mortality, 1980-1988. *MMWR* 41(1992): 548-56.
5. National Center for Health Statistics. *health, United States, 1992.* Hyattsville, (MD): Public Health Service, 1993.
6. National Heart, Lung and Blood Institute. *Advance Report of Final Mortality Statistics, 1990. Monthly Vital Statistics Report, Vol 41, No 7, Suppl,* Hyattsville, (MD): Public Health Service, 1993.
7. Gillum R.F., Feinleib M. Cardiovascular disease in the United States: mortality, prevalence and incidence. In: Kapoor AS, Singh BM, editors. *Prognosis and Risk Assessment in Cardiovascular Disease.* New York: Churchill Livingstone, 1993; 49-59.
8. Goldberg RJ, Gorak EJ, Yarzebski J, et al. A community-wide perspective of gender differences and temporal trends in the incidence and survival rates following acute myocardial infarction and out-of-hospital deaths due to coronary heart disease. *Circulation* 1993;87:1947-53.
9. Sempos C, Cooper R, Kovar MG, McMillen M. Divergence of the recent trends in coronary mortality for the major race-sex groups in the United States. *Am J Public Health* 1988;78:1422-7.

LIST OF REFERENCES

1. Marmot JG, Kannel WB, Feinleib M, et al. Clinical features of myocardial infarction in men and women: a long-term follow-up study. *Ann Intern Med*. 1979;91:1-9.
2. Selzer CG, Lee TH, Cook EF, et al. Management of clinical trials in acute myocardial infarction in patients with heart failure. *Am J Cardiol*. 1990;65:1172-1176.
3. Gosselin RM, Boardman P, Lurie D, et al. AHA guidelines for the early management of patients with acute myocardial infarction. *Am J Cardiol*. 1990;65:1172-1176.
4. CDC. Trends in Ischemic Heart Disease Mortality, 1960-1980. *MMWR*. 1981;30:11-15.
5. National Center for Health Statistics. *Health, United States, 1980*. (DHED-80-001). Public Health Service, 1980.
6. National Heart, Lung, and Blood Institute. *Annual Report of the National Heart, Lung, and Blood Institute, Vol. 11, 1980-1981*. (DHED-81-001). Public Health Service, 1981.
7. Gillum RF, Feinleib M. Cardiovascular diseases in the United States: prevalence and incidence. In: Kaplan AS, Singh RM, editors. *Prevalence and Incidence in Cardiovascular Disease*. New York: Churhill Livingstone, 1981; 49-59.
8. Goldberger RL, Gorek EJ, Yarneski J, et al. A community-wide perspective on gender differences and temporal trends in the incidence and survival rates following acute myocardial infarction and out-of-hospital deaths due to coronary heart disease. *Circulation*. 1983;67:1247-53.
9. Selzer CG, Cooper R, McMillan M. Divergence of the recent trends in coronary mortality for the major race-sex groups in the United States. *Am J Public Health*. 1988;78:1452-5.

10. McGovern PG, Pankow JS, Shahar E, et al. Recent trends in acute coronary heart disease mortality, morbidity, medical care, and risk factors. *The New England Journal of Medicine*. 334(14); 1996:884-890.
11. Asaf AR, Lapane KL, McKenney JL, et al. Possible influence of the prospective payment system on the assignment of discharge diagnoses for coronary heart disease. *N Engl J Med* 1993; 329:931-5.
12. Gillum, RF. Acute myocardial infarction in the US, 1970-1983. *Am Heart J*. 1987; 113:804-11.
13. Roig E, Castaner A, Simmons B, et al. In-hospital mortality rates from acute myocardial infarction by race in US hospitals: findings from the National Hospital Discharge Survey. *Circulation*. 1987; 76:280-8.
14. National Center for Health Statistics. *Health, US, 1992*. Hyattsville (MD): Public Health Service, 1993.
15. Graves EJ. *National Hospital Discharge Survey: Annual Summary, 1990*. Hyattsville (MD): National Center for Health Statistics, 1992: Public Health Service (Vital & Health Statistics: Series 13, No. 112).
16. GISSI: Long-term effects of intravenous thrombolysis in acute myocardial infarction: final report on the GISSI study. *Lancet* 2:871, 1982.
17. Rogers WJ, Baim BS, Gore JM et al, for the TIMI IIA Investigators: Comparison of immediate invasive, delayed invasive and conservative strategies after tissue-type plasminogen activator. Results of the Thrombolysis in Myocardial Infarction (TIMI) phase IIA trial. *Circulation* 81:1457,1990.
18. Forssmann W: The catheterization of the right side of the heart. *Klin Wochenschr*, 8:2085, 1929.
19. Brannon ES, Weens HS, Warren JW: Atrial septal defect: study of hemodynamics by the technique of right heart catheterization. *Am J Med Sci*. 210:480, 1945.
20. Zimmerman HA, Scott RW, Becker NO: Catheterization of the left side of the heart in man. *Circulation* 1:357, 1950.
21. Sones FM Jr, Shirey EK: Cine coronary arteriography. *Mod Concepts Cardiovasc Dis*, 31:735-738, 1962.

10. McKoven FC, Fainlow JS, Soward B, et al. System trends in acute coronary heart disease mortality, morbidity, medical care, and risk factors. *Am J Epidemiol*. 1994;139:334-343.
11. Arai AR, Lippman H, et al. Possible influence of the payment system on the diagnosis of coronary artery disease. *Am J Med*. 1993;95:329-333.
12. Gillum RF. Acute myocardial infarction in the US, 1970-1985. *Am J Med*. 1987;81:804-811.
13. Klig B, Cantone A, Simmons B, et al. In-hospital death rates for acute myocardial infarction by race in US hospitals. *Am J Epidemiol*. 1987;125:450-454.
14. National Center for Health Statistics. *Health, US, 1994*. Washington, DC: NCHS; 1995.
15. Graves EL. National Hospital Discharge Survey. *Annual Report*. 1994. Hyattsville (MD): National Center for Health Statistics; 1995.
16. GISSI. Long-term effects of intravenous thrombolysis in acute myocardial infarction: final report on the GISSI study. *Lancet*. 1992;340:1-6.
17. Rogers WJ, Bain R, Gore JM, et al. for the TIMI II Investigators. Efficacy and safety of intravenous and combined intravenous and aspirin therapy after myocardial infarction: Results of the Thrombolysis in Myocardial Infarction (TIMI) phase IIa trial. *Circulation*. 1990;81:1437-1449.
18. Forrester W. The contribution of the right side of the heart. *Circulation*. 1979;60:508-519.
19. Brannon ES, Wooten HS, Wooten LW. Atrial septal defect: study of hemodynamics by the technique of right heart catheterization. *Am J Med Sci*. 1945;150:187-192.
20. Zimmerman HA, Scott RW, Becker NO. Catheterization of the left side of the heart in man. *Circulation*. 1957;15:757-760.
21. Scott RW, A. Shiley BK. Cine coronary arteriography. *Med Clin North Am*. 1962;36:733-738.

22. Heupler, F Jr. Coronary Arteriography and Left Ventriculography: Sones Technique in Coronary Arteriography and Angioplasty by Spencer B. King III and John S. Douglas, Jr. McGraw-Hill Company, 1985.
23. Califf RM, O'Neill W, Stack RS, et al: Failure of simple clinical measurements to predict perfusion status after intravenous thrombolysis. *Ann Intern Med* 108:658,1988.
24. Kulick DL, Rahimtoola SH: Assessment of the survivors of acute myocardial infarction: the case for coronary angiography, p. 429. In Gersh B, Rahimtoola SH (eds): *Myocardial Infarction*. Elsevier, New York, 1991.
25. Crawford MH, O'Rourke RA: The role of cardiac catheterization in patients after myocardial infarction. *Cardiol Clin* 2(1):105, 1984.
26. O'Rourke RA: Clinical decision in post infarction patients. *Mod Conc Cardiovasc Dis* 55:55, 1986.
27. Kelly DT: Clinical decisions in patients following myocardial infarction. *Curr Probl Cardiol* 10(1):1, 1985.
28. Bamrah VS, and Wann LS. *Cardiology in Concise textbook of Medicine*. Elsevier, 1990.
29. O'Rourke, RA. Invasive and noninvasive risk stratification of postmyocardial infarction patients, pg. 135 In McCall, David (ed): *Acute Myocardial Infarction*. Churchill Livingstone, New York, 1991.
30. GISSI: Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986; 1:397-402.
31. GISSI: Long-term effects of intravenous thrombolysis in acute myocardial infarction: Final report of the GISSI study. *Lancet* 1987; 2:871-874.
32. ISIS Collaborative Group: Randomized trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet* 1988; 2:349-360.
33. Wicox RG, von der Lippe G, Olsson CG, Jensen G, Skene AM, Hampton JR. Trial of tissue plasminogen activator for mortality reduction in acute myocardial infarction: Anglo-Scandinavian study of early thrombolysis (ASSET). *Lancet* 1988; 2:525-30.

23. Hengler J, L. Coronary Atherosclerosis and Left Ventricular Hypertrophy. *Techniques in Coronary Atherosclerosis and Angiography* by Spencer R. King III and John S. Douglas Jr. McGraw-Hill Company, 1987.
24. Giblin MM, O'Neill W, Stock RJ, et al. Failure of single channel measurement to predict perfusion status after intravenous thrombolysis. *Ann Intern Med* 103:688, 1985.
25. Kellick DL, Rabinowitz SR. Assessment of the accuracy of single channel perfusion: the case for coronary angiography. *Am J Cardiol* 57:103-108, 1986 (abst). Myocardial infarction. Elsevier, New York, 1987.
26. Crawford MH, O'Rourke RA. The role of the single channel perfusion measurement in myocardial infarction. *Circulation* 73:1112-1118, 1986.
27. O'Rourke RA. Clinical decision in post infarction patients. *Circulation* 73:432, 1986.
28. Kelly DT. Clinical decision in patients following myocardial infarction. *Am J Cardiol* 10(1):1, 1982.
29. Harnish VS, and Wain LK. *Cardiology: A Clinical Approach* 1st ed. Philadelphia, 1990.
30. O'Rourke RA. Intravenous and coronary artery thrombolysis in acute myocardial infarction patients. pp. 125 in *McCullough* 1987. *Heart and Lung Transplantation*. Churchill Livingstone, New York, 1987.
31. GISSI. Effectiveness of intravenous thrombolysis in myocardial infarction. *Lancet* 1986; 1:397-401.
32. GISSI. Long-term effects of intravenous thrombolysis in acute myocardial infarction. Final report of the GISSI study. *Lancet* 1987; 2:871-874.
33. ISIS Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected acute myocardial infarction. *Lancet* 1988; 2:341-349.
34. Wolk RF, von der Lippe G, Olsson CG, Jensen O, Skene AM, Thompson JR. Trial of tissue plasminogen activator for mortality reduction in acute myocardial infarction. Anglo-Scandinavian study of early thrombolysis (ASSET). *Lancet* 1988; 2:823-826.

34. Bassand J-P, Machecourt J, Cassagnes J, et al and the APSIM Study Investigators. Multicenter trial of intravenous anisoylated plasminogen streptokinase activator complex (APSAC) in acute myocardial infarction: effects on infarct size and left ventricular function. *J Am Coll Cardiol* 1989; 13:988-97.
35. Verstraete M, Bernard R, Bory M, et al. European Cooperative Study Group for Recombinant Tissue-Type Plasminogen Activator: Randomized trial of intravenous recombinant tissue-type plasminogen activator versus intravenous streptokinase in acute myocardial infarction. *Lancet* 1985; 1:842-847.
36. Chesebro JH, Knatterud G, Roberts R: Thrombolysis in Myocardial Infarction (TIMI) trial, Phase I: A comparison between intravenous tissue plasminogen activator and intravenous streptokinase. *Circulation* 1987; 76:142-154.
37. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. The GUSTO investigators. *N Engl J Med* 1993; 329: 673-82.
38. The effects of tissue plasminogen activator, streptokinase, or both on coronary-artery patency, ventricular function, and survival after acute myocardial infarction. The GUSTO Angiographic Investigators. *N Engl J Med* 1993; 329:1615-22.
39. Bonnier HJRM, Visser RF, Klomps HC, Hoffman HJML and the Dutch Invasive Reperfusion Group. Comparison of intravenous anisoylated plasminogen streptokinase activator complex and intravenous streptokinase in acute myocardial infarction. *Am J Cardiol* 1988; 62:25-30.
40. National Center for Health Statistics: 1986 Summary: National Hospital Discharge Survey. Advance Data from Vital and Health Statistics. No. 145. US Dept of Health and Human Services publication No. (PHS) 87-1250. Public Health Service. Hyattsville, Md, National Center for Health Statistics, 1987.
41. Graves EJ. National Hospital Discharge Survey: Annual Summary, 1991. National Center for Health Statistics 1993; DHHS publication (PHS):93-1775;44.
42. Dotter, CT and Judkins, MP: Transluminal treatment of arteriosclerotic obstruction: Description of a new technique and a preliminary report of its application. *Circulation* 30: 654, 1964.
43. Dotter, CT, Rosch, J, Anderson JM, et al: Transluminal iliac artery dilation: Non-surgical catheter treatment of atheromatous narrowing. *JAMA* 230:117, 1974.
44. Porstmann, W: Ein neuer Korsett-Ballonkatheter zur transluminalen Rekanalisation nach Dotter unter besonderer Berücksichtigung von Obliterationen an den Beckenarterien. *Radiol Diagn (Berl)* 14:239, 1973.

34. Johnson J-P, Mobergson L, Cassinger J, et al: The AFIRM study: investigation of the effect of intracranial aneurysms on the development of atherosclerosis. *J Am Coll Cardiol* 1989; 13:988-93.
35. Weinstein M, Bandyk E, Bory M, et al: Intracranial aneurysms: a review of the literature. *Neurosurgery* 1987; 21:1182-91.
36. Chassin D, Kinsman G, Roberts R, et al: Intracranial aneurysms: a review of the literature. *Neurosurgery* 1987; 21:1182-91.
37. An international multicenter trial comparing two different techniques for aneurysm resection. *The GUSTO Investigators*. *Stroke* 1993; 24:1055-62.
38. The effect of blood pressure on the development of aneurysms: a review of the literature. *Stroke* 1993; 24:1055-62.
39. Bandyk EP, Kinsman G, Roberts R, et al: Intracranial aneurysms: a review of the literature. *Neurosurgery* 1987; 21:1182-91.
40. National Center for Health Statistics: 1992 and 1993 National Health Interview Survey. *Health Statistics Reports* 1993; 145:1-100.
41. Grimes EJ, National Hospital Discharge Survey Annual Summary, 1991. *National Center for Health Statistics* 1992; 145:1-100.
42. Dornier CT and Johnson M: Transcatheter treatment of aneurysms: a review of the literature and a preliminary report of its application. *Circulation* 1993; 88:1055-62.
43. Dornier CT, Bandyk E, Bory M, et al: Transcatheter treatment of aneurysms: a review of the literature and a preliminary report of its application. *Circulation* 1993; 88:1055-62.
44. Bandyk EP, Kinsman G, Roberts R, et al: Intracranial aneurysms: a review of the literature. *Neurosurgery* 1987; 21:1182-91.

45. Athanasouleis, CA: Percutaneous transluminal angioplasty: General principles. AJR 135:893, 1980.
46. Grüntzig, A and Kumpe DA: Technique of percutaneous transluminal angioplasty with the Grüntzig balloon catheter. AJR 132:547, 1979.
47. Grüntzig, A: die perkutane transluminale Rekanalisation chronischer Arterienverschlüsse mit einer neuen Dilatationstechnik. Gerhard Witzstrock, Baden-Baden, 1977.
48. Gruentzig, AR, Myler, RK, Hanna, ES, et al: Coronary transluminal angioplasty (abstr.) Circulation 56 (Suppl 3):84, 1977.
49. Gruentzig AR. Transluminal dilation of coronary artery stenosis. Lancet 1978; 1:263.
50. Dotter CT, Judkins MP. Transluminal treatment of arteriosclerotic obstruction. Circulation 1964; 30:654-70.
51. Hill JA, Margoli JR, Feldman RL, et al. Coronary arterial aneurysm formation after balloon angioplasty. Am J Cardiol 1983; 52:261-4.
52. Castaneda-Zuniga WR, Formanek A, Tadavarthy M, et al. The mechanism of balloon angioplasty. Radiology 1980; 135:565-71.
53. Lee G, Low RI, Takeda P, et al. Importance of follow-up medicare and surgical approaches to prevent reinfarction, reocclusion, and recurrent angina following intracoronary thrombolysis with streptokinase in acute myocardial infarction. American Heart Journal. 1982; 104:921-4.
54. Meyer J, Merx W, Schmitz H, et al. PTCA immediately after intracoronary streptolysis of transmural myocardial infarction. Circulation 1982; 66:905-13.
55. Swan HJC. Thrombolysis in acute myocardial infarction: treatment of the underlying coronary artery disease. Circulation. 1982; 66:914-6.
56. Fitzgerald DJ, Catella F, Roy L, Fitzgerald GA. Marked platelet activation in vivo after intravenous streptokinase in patients with acute myocardial infarction. Circulation. 1988; 77:142-150.
57. Sebuski RJ, Ohlstein EA. Attenuation of platelet responsiveness to prostacyclin (PGI₂) after tPA. Circulation. 1987; 76:(Suppl IV):IV-338.

58. Topol EJ, Califf RM, George BS, et al. A randomized trial of immediate versus delayed elective angioplasty after intravenous tissue plasminogen activator in acute myocardial infarction. *N Engl J Med* 1987; 327:581-8.
59. TIMI Research Group. Immediate vs delayed catheterization and angioplasty following thrombolytic therapy for acute myocardial infarction: TIMI IIA results. *JAMA* 1988; 260:2849-58.
60. Ryan TJ, Faxon DP, Gunnar RM, Kennedy JW, King SB III, Loop FD, Peterson KL, Reeves TJ, Williams DO, Winters WL Jr, et al. Guidelines for percutaneous transluminal coronary angioplasty: a report of the American College of Cardiology/American Heart Association Task force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures. *Circulation* 1988; 78:486-502.
61. Gruentzig AR, King SB III, Schumpf M, Siengenthaler W. Longterm followup of percutaneous transluminal coronary angioplasty: the early Zurich experience. *N Engl J Med* 1987; 316: 1127-32.
62. Leimgruber PP, Roubin GS, Hollman J, et al. Restenosis after successful coronary angioplasty in patients with single-vessel disease. *Circulation* 1986; 73:710-717.
63. Kent KM. Restenosis after percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1988; 61:67G-70G.
64. Leimgruber PP, Roubin GS, Anderson HV, et al. Influence of intimal dissection on restenosis after successful coronary angioplasty. *Circulation* 1985; 72:530-5.
65. Lam JYT, Cheseboro JH, Steele DM, et al. Deep arterial injury during experimental angioplasty. Relationship to a positive 111 indium-labelled platelet scintigram, quantitative platelet deposition and mural thrombus. *J Am coll Cardiol* 1986; 8:1380.
66. Meier B, King SB, Gruentzig AR, et al. Repeat coronary angioplasty. *J Am Coll Cardiol* 1984; 4:436.
67. Sipperly, Mary Ellen. Expanding role of coronary angioplasty: Current implications, limitations, and nursing considerations. *Heart & Lung* 1989; 18:507-13.
68. Dorros G, Cowley MJ, Simpson J, et al. Percutaneous transluminal coronary angioplasty: report of complications from the National Heart, Lung, and Blood Institute PTCA Registry. *Circulation* 1983; 67: 723-30.
69. Detre K, Holubkov R, Kelsey S, et al. Percutaneous transluminal coronary angioplasty in 1985-1986 and 1977-1981. The National Heart, Lung, Blood Institute Registry. *N Engl J Med* 1988; 318:265-270.

52. Topol EJ, Chlibk RM, George RS, et al. A randomized trial of immediate versus delayed elective angiography after myocardial infarction. *N Engl J Med* 1987; 317:91-8.
53. TIMI Research Group. Intravenous ascorbic acid in the treatment of patients following thrombolytic therapy for acute myocardial infarction. *N Engl J Med* 1988; 319:2649-58.
54. Ryan TJ, Faxon DP, Gunnar RM, Kinney TW, King III DL, Lissner L, et al. Report of the Committee to Reassess the Use of Coronary Catheterization in the Elderly. *Circulation* 1984; 69:1311-21.
55. Kannel WB, Castelli WP, Hjortskov L, Sornavalle R, Castelli WP. Lifetime risk of coronary heart disease. *N Engl J Med* 1984; 311:351-6.
56. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
57. Greenwald AR, King III DL, Braunholtz H, et al. Intravenous ascorbic acid in the treatment of patients with angina pectoris. *Am J Med* 1987; 82:117-22.
58. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
59. Kannel WB, Castelli WP, Hjortskov L, Sornavalle R, Castelli WP. Lifetime risk of coronary heart disease. *N Engl J Med* 1984; 311:351-6.
60. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
61. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
62. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
63. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
64. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
65. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
66. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
67. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
68. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
69. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.
70. Lemberg P, Rabinowitz D, Rabinowitz D, et al. The effect of intravenous ascorbic acid on the outcome of percutaneous transluminal coronary angioplasty. *Am J Med* 1987; 82:117-22.

70. Galan KM, Deligonul U, Kern MJ, et al. Smoking increases the risk of restenosis following coronary angioplasty (PTCA). *Circulation* 1986; 74: II-281.
71. Cowley MJ, Dorros G, Kelsey SF, Van Rader M, Detre KM. Emergency coronary bypass surgery after coronary angioplasty: The National Heart, Lung, and Blood Institute's PTCA Registry Experience. *American Journal of Cardiology*. 1984; 53:22C-26C.
72. Bredlau CE, Roubin GS, Leimgruber PP, et al. In-hospital morbidity in patients undergoing elective coronary angioplasty. *Circulation* 1985; 72:1044-52.
73. Sinclair IN, McCabe CH, Sipperly ME, et al. Predictors, therapeutic options and long-term outcome of abrupt reclosure. *American Journal of Cardiology*. 1988; 61:615-66.
74. Kent KM, Bentivoglio LG, Block PC, et al. PTCA: Report from the Registry of the National Heart, Lung, and Blood Institute. *American Journal of Cardiology*. 1982; 49:2011-2020.
75. Falk, E: Plazue rupture with sever pre-existing stenosis precipitating coronary thrombosis : Characteristics of coronary atherosclerotic plazues underlying fatal occlusive thrombi. *Br Heart J* 50:127, 1983.
76. Cohn LH, Gorlin R, Herman M, et al.: Surgical treatment of acute coronary occlusion. *J Thorac Cardiovasc Surg* 1972; 64:503-13.
77. Phillips SJ, Kongtahworn C, Skinner JR, et al: Emergency coronary artery reperfusion. A choice therapy for evolving myocardial infarction. *J Thorac Cardiovasc Surg* 1983; 86:679-688.
78. Phillips SJ, Kongtahworn C, Zeff RH, et al: Emergency coronary artery revascularization. A possible therapy for acute myocardial infarction. *Circulation* 1979; 60:241.
79. DeWood MA, Notske RN, Berg R, Ganji JH, Simpson CS, Hinnen ML, Selinger SL, Fisher LD. Medical and surgical management of early Q wave myocardial infarction. I. Effects of surgical reperfusion on survival in recurrent myocardial infarction, sudden death and functional class at 10 or more years of follow-up. *J Am Coll Cardiol* 1989; 14:65-77.
80. DeWood MA, Leonard J, Grunwald RP, Hensley GA, Mouser LT, Burroughs RW, Berg R, Fisher LD. Medical and surgical management of early Q wave myocardial infarction. II. Effects on mortality and global and regional left ventricular function at 10 or more years follow-up. *J Am Coll Cardiol* 1989; 14:78-90.

70. Gilman RM, Deligdisch L, Kern MJ, et al. Smoking increases the risk of a tamoxifen-induced coronary angiopathy (PTEA). *Circulation* 1993; 87: 11-24.
71. Conley MJ, Doros G, Koleski RJ, Van Buren N, Kline RM. A tamoxifen-induced coronary artery and coronary angiopathy. The National Heart, Lung, and Blood Institute's PECA Registry. *American Journal of Cardiology* 1994; 73: 25C-26C.
72. Bradburn CE, Roubin GS, Litwakson WF, et al. Induced coronary artery aneurysms in tamoxifen-treated coronary angiopathy. *Circulation* 1992; 85: 1007-1012.
73. Shachar IN, McCabe CH, Sperry RW, et al. Tamoxifen-induced coronary artery aneurysms: a possible mechanism of abrupt death. *Journal of Intensive Cardiology* 1993; 4: 61-65.
74. Kern MJ, Bradburn CE, Litwakson WF, et al. Tamoxifen-induced coronary artery aneurysms. *Journal of Intensive Cardiology* 1993; 4: 2011-2022.
75. Belli B. Tamoxifen therapy with tamoxifen-induced coronary angiopathy. *Circulation* 1993; 87: 11-24.
76. Gilman RM, Deligdisch L, Kern MJ, et al. Smoking increases the risk of a tamoxifen-induced coronary angiopathy (PTEA). *Circulation* 1993; 87: 11-24.
77. Phillips SJ, Kozlowski C, Gilman RM, et al. Tamoxifen-induced coronary angiopathy. A clinic therapy for tamoxifen-induced coronary angiopathy. *Circulation* 1993; 87: 11-24.
78. Phillips SJ, Kozlowski C, Gilman RM, et al. Tamoxifen-induced coronary angiopathy. A clinic therapy for tamoxifen-induced coronary angiopathy. *Circulation* 1993; 87: 11-24.
79. DeWood MA, Moore HW, Gault JM, Simpson CF, Hirsch MJ, Selinger SL, Fisher LD. Medical and surgical management of early Q wave myocardial infarction. I. Effect of surgical revascularization on survival in coronary artery aneurysm, sudden death and recurrent chest pain 10 or more years of follow-up. *J Am Coll Cardiol* 1989; 14: 65-77.
80. DeWood MA, Lawrence J, Garwood RP, Hamley GA, Moore LT, Partridge BW, et al. Fisher LD. Medical and surgical management of early Q wave myocardial infarction. II. Effect on mortality and global and regional left ventricular function at 10 or more years follow-up. *J Am Coll Cardiol* 1989; 14: 78-90.

81. Alderman EL, Bourassa MG, Cohen LS, Davis KB, Kaiser GG, Killip T, Mock MB, Pettinger M, Robertson TL (CASS Investigators): Ten-year follow-up of survival and myocardial infarction in the randomized Coronary Artery Surgery Study (CASS). *Circulation* 1990; 82:1629-1647.
82. Califf RM, Harrell FE Jr, Lee KL, Rankin JS, Hlatky MA, Mark DB, Jones RH, Muhlbaier LH, Oldham HN Jr, Pryor DB: The evolution of medical and surgical therapy for coronary artery disease: A 15-year perspective. *JAMA* 1989; 261:2077-2086.
83. Loop FD, Lytel BW, Cosgrove DM, Stewart RW, Goormastic M, Williams GW, Golding LAR, Gill CC, Taylor PC, Sheldon WC, Proudfit WL: Influence of the internal-mammary-artery graft on 10-year survival and other cardiac events. *N Engl J Med* 1986; 314:1-6.
84. Bourassa MG, Fisher LD, Campeau L, Gillespie MJ, McConney M, Lesperance J: Long-term fate of bypass grafts: The Coronary Artery Surgery Study (CASS) and Montreal Heart Institute experiences. *Circulation* 1985; 72:V-71-V-78.
85. Grondin CM, Campeau L, Lesperance J, Enjalbert M, Bourassa MG: Comparison of late changes in internal mammary artery and saphenous vein grafts in two consecutive series of patients 10 years after operation. *Circulation* 1984; 70(suppl I):I-208-I-212.
86. Kirklin, JW, Blackstone, EH, Califf, RM, et al. ACC/AHA Guidelines and Indications for Coronary Artery Bypass Graft Surgery. A report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee on Coronary Artery Bypass Graft Surgery). *Circulation* 1991; 83(no.3):1125-1173.
87. Pocock SJ, Henderson RA, Rickards AF, et al. Meta-analysis of randomised trials comparing coronary angioplasty with bypass surgery. *Lancet* 1995; 346:1184-89.
88. CABRI Trial Participants. First-year results of CABRI (Coronary Angioplasty vs Bypass Revascularization Investigation). *Lancet* 1995; 346: 1179-84.
89. RITA Trial Participants. Coronary angioplasty versus coronary artery bypass surgery: the Randomised Intervention Treatment of Angina (RITA) trial. *Lancet* 1993; 343: 573-80.
90. King, SB, Lembo NJ, Kosinski AS, et al. A randomised trial comparing coronary angioplasty with coronary bypass surgery. *N Engl J Med* 1994; 331:1044-50.

91. Hamm CW, Riemers J, Ischinger T, et al. A randomised study of coronary angioplasty compared with bypass surgery in patients with symptomatic multi-vessel coronary disease. *N Engl J Med* 1994; 331: 1037-43.
92. Puel J, Karouny E, Marco F, et al. Angioplasty versus surgery in multi-vessel disease: immediate results and in-hospital outcome in a randomised prospective study. *Circulation* 1992; 86 (supp I):372.
93. Hueb W, Arie S, Oliveira SA, et al. The Medicine, Angioplasty or Surgery Study (MASS): a prospective randomised trial for single proximal left anterior descending artery stenoses. *J Am Coll Cardiol* (in press).
94. Goy J-J, Eeckhout E, Burnand B, et al. Coronary angioplasty versus left internal mammary grafting for isolated proximal left anterior descending artery stenosis. *Lancet* 1994; 343:1449-53.
95. Rodriguez A, Bouillon F, Perez-Balina N, et al. Argentine randomised trial of percutaneous transluminal coronary angioplasty versus coronary artery bypass surgery in multi-vessel disease (ERACI): in-hospital results and 1-year follow-up. *J Am Coll Cardiol* 1993; 22:1060-67.
96. Fitzgibbon GM, Leach AJ, Kafka HP, Keon WF. Coronary bypass graft fate: long-term angiographic study. *American Journal of Cardiology*. 1991; 17:1075-80.
97. Loop FD, Lytle BW, Cosgrove DM, et al. Influence of the internal-mammary artery graft on ten-year survival and other cardiac events. *New England Journal of Medicine*. 1986; 314:1-6.
98. The international classification of diseases. 9th Revision. Clinical modification. 2nd ed. Washington, D.C.: Department of Health and Human Services, 1980. (DHHS publication no. (PHS) 80-1260.
99. Coding Clinic May/June 1984; pg. 9-14.
100. Charlson ME, Pompei P, Ales KL, and MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chron Dis* 1987; 40(no.5): 373-383.
101. Cox DR. Regression models and life tables. *J R Stat Soc*. 1972; 34:187-220.
102. Campeau, L: Grading of angina pectoris. *Circulation* 1976; 54:522-523.
103. CASS Principal investigators and their associates. Coronary artery Surgery Study (CASS): a randomized trial of coronary artery bypass surgery. Survival data. *Circulation* 1983; 68:939-950.

91. Hansen CW, Haganek J, Jackson T, et al. A randomized study of coronary angiography compared with bypass surgery in patients with symptomatic multivessel coronary disease. *N Engl J Med* 1990; 323: 1072-81.
92. Patel I, Kohnen E, Marzke E, et al. Angioplasty versus surgery in multivessel disease: immediate results and in-hospital mortality in a multicenter prospective study. *Circulation* 1991; 83 (suppl 1): 117.
93. Hersh W, Aris S, Oliveira EA, et al. The MARS procedure: Angioplasty or bypass? (MARS): a prospective randomized trial for single proximal coronary artery disease. *J Am Coll Cardiol* (in press).
94. Guy J-L, Beckson B, Brumard B, et al. Efficacy of angioplasty versus coronary artery bypass grafting for isolated proximal left anterior descending coronary artery disease. *Lancet* 1990; 335: 1448-53.
95. Rodriguez A, Brullon F, Soria Jarama B, et al. Angioplasty versus coronary artery bypass grafting in multivessel disease (BRAC): a prospective study and follow-up. *Am J Cardiol* 1990; 65: 1000-61.
96. Pridmore GM, Leach AJ, Kiefe HP, et al. A multicenter study of coronary artery bypass grafting versus angioplasty. *Am J Med* 1990; 88: 107-20.
97. Loop FD, Lytle BW, Cosgrove DM, et al. Long-term results of the arterial anastomosis coronary graft on the year survival and late cardiac events. *J Am Coll Cardiol* 1986; 7: 11-21.
98. The International Classification of Diseases. 9th Revision, Clinical Modification. 2nd ed. Washington, D.C.: Department of Health and Human Services, 1980. (784112 publication no. (DHHS) 80-1260).
99. Coding Clinic Magazine 1990; pg. 9-14.
100. Charlson ME, Pompei P, Ales KL, and MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chron Dis* 1957; 40 (no. 2): 373-381.
101. Cox DR. Regression models and life tables. *J R Stat Soc* 1972; 34: 187-220.
102. Campbell L. Grading of angina pectoris. *Circulation* 1956; 13: 527-529.
103. CASS Principal Investigators and their associates. Coronary artery surgery study (CASS): a randomized trial of coronary artery bypass surgery. *Circulation* 1982; 66: 939-950.

104. European Coronary Surgery Study Group. Long-term results of prospective randomized study of coronary artery bypass surgery in stable angina pectoris. *Lancet* 1982; 2:1173-1180.
105. Becker RC. Coronary thrombolysis in women. *Cardiovasc Dis Women*. 1990;77(suppl 2):110-123.
106. Eysmann SB, Douglas PS. Reperfusion and Revascularization strategies for coronary artery disease in women. *JAMA*. 1992;268(14):1903-1907.
107. Talley JD. Review of thrombolytic intervention for acute myocardial infarction-is it valuable? *Journal of the Arkansas Medical Society*. 1994;91(2):70-79.
108. Van de Werf F, Arnold AER. Intravenous tissue plasminogen activator and size of infarct, left ventricular function and survival in AMI. *Br Med J*. 1988;297:1374-1379.
109. AIMS Trial Study Group: Effect of intravenous APSAC on mortality after AMI: Preliminary report of a placebo-controlled clinical trial. *Lancet*. 1988;1:545-549.
110. Malone ML, Sial Shahid, Battiola RJ, et al. Age-related Differences in the Utilization of Therapies Post Acute Myocardial Infarction. *Journal of American Geriatric Society* 1995; 43 (no. 6): 627-633.
111. Udvarhelyi IS, Gatsonis, C, Epstein AM, et al. Acute Myocardial Infarction in the Medicare Population. *JAMA* 1992; 268 (no. 18): 2530-2536.
112. Wenneker MB, Weissman JS, and Epstein AM. The Association of Payer With Utilization of Cardiac Procedures in Massachusetts. *JAMA* 1990; 264 (no. 10): 1255-1260.
113. Ayanian, JZ and Epstein AM. Differences in the Use of Procedures Between Women and Men Hospitalized for Coronary Heart Disease. *The New England Journal of Medicine* 1991; 325 (no.4): 221-225.
114. Wenneker, MB and Epstein AM. Racial Inequalities in the Use of Procedures for Patients With Ischemic Heart Disease in Massachusetts. *JAMA* 1989; 261 (no. 2): 253-257.
115. Grines CL, De Maria AN. Optimal utilization of thrombolytic therapy for acute myocardial infarction: concepts and controversies. *J Am Coll Cardiol*. 1990; 16:223-231.
116. American Heart Association. 1991 Heart and Stroke Facts. Dallas, Tex: American Heart Association; 1991.

104. European Coronary Project Group. Long-term results in angioplasty: randomized study of coronary artery bypass surgery in infarcted patients. *Lancet* 1982; 2:1133-1136.
105. Sackier RC. Coronary thrombolysis in women. *Am Heart J* 1980; 100:110-113.
106. Eymann SB, Douglas ES. Reperfusion and myocardial damage in coronary artery disease in women. *JAMA* 1981; 245:1071-1074.
107. Tebbe H. Review of thrombolytic treatment of acute myocardial infarction: a valuable journal of the American Heart Association. *Am Heart J* 1981; 101:1071-1074.
108. Van de Werf R, Arntz AE. Intravenous streptokinase in the treatment of infarcted ventricular function and survival. *Am J Med* 1981; 70:1174-1179.
109. AHA. Third Study Group. Effect of streptokinase on survival after acute myocardial infarction: a placebo-controlled clinical trial. *Lancet* 1981; 1:124-128.
110. Malin ML, Shi S, Shih H, et al. Streptokinase in the treatment of acute myocardial infarction. *Am J Med* 1981; 70:1174-1179.
111. Liberman IS, Gerson C, Gerson C, et al. Streptokinase in the treatment of acute myocardial infarction. *JAMA* 1981; 245:1071-1074.
112. Weinacker MB, Weinstein JS, and Epstein AJ. The association of heart with the treatment of cardiac procedures in Massachusetts. *JAMA* 1980; 244:1071-1074.
113. Agazzi M and Epstein AJ. Differences in the use of thrombolytic therapy in women and men hospitalized for coronary heart disease. *The New England Journal of Medicine* 1981; 325:1071-1074.
114. Weinacker MB and Epstein AJ. Racial inequalities in the use of thrombolytic therapy for patients with ischemic heart disease in Massachusetts. *JAMA* 1980; 244:1071-1074.
115. Gerson C, De Meis A. Optimal utilization of thrombolytic therapy for acute myocardial infarction: concepts and controversies. *J Am Coll Cardiol* 1980; 16:124-127.
116. American Heart Association. 1981 Heart and Stroke Facts. Dallas, Tex: American Heart Association; 1981.

117. Smith SC Jr, Gilpin E, Ahnve S. Outlook after acute myocardial infarction in the very elderly compared with that in patients aged 65 to 75 years. *J Am Coll Cardiol.* 1990; 16:784-792.
118. Adolph RJ. The elderly, the very elderly and traditional practice patterns. *J Am Coll Cardiol.* 1990; 16:793-794.
119. Topol EJ, Califf RM. Thrombolytic therapy for elderly patients. *N Engl J Med.* 1992; 327: 45-47.
120. Collins R, for the ISIS Collaborative Group. Optimizing thrombolytic therapy of acute myocardial infarction: age is not a contraindication. *Circulation.* 1991; 84:II-230. Abstract.
121. Peto R. Which patients to treat with fibrinolysis: overview of ISIS-3 and Estudio Multicentrico Estreptoquinase Republicas de America del Sur. Presented at the 40th annual scientific session of the American College of Cardiology; March 3, 1991; Atlanta, Ga.
122. Pepine CJ. Thrombolytic use in the elderly. *J Myocardial Ischemia.* 1991; 3:10-12.
123. Rich MW. Acute myocardial infarction in the elderly. *J Myocardial Ischemia.* 1991;3:10-12.
124. Robers WJ, Bowlby LJ, Chandra NC, et al. Treatment of myocardial infarction in the United States (1990 to 1993). Observations from the National Registry of Myocardial Infarction. *Circulation.* 1994;90(4):2103-2114. Abstract.
125. Tobin JN, Wassertheil-Smoller S, Wexler JP, et al. Sex bias in considering coronary bypass surgery. *Ann Intern Med.* 1987; 107:19-25.
126. Whittle J, Conioliaro J, Good CB, et al. Racial differences in the use of invasive cardiovascular procedures in the department of veterans affairs medical system. *The New Engl Journ of Med.* 1993; 329(9): 621-627.
127. Maynard C, Litwin PE, Martin JS, et al. Characteristics of black patients admitted to coronary care units in metropolitan Seattle; results from the Myocardial Infarction Triage and Intervention Registry (MITI). *Am J Cardiol.* 1991;67(1): 18-23.
128. Loop FS, Golding LR, MacMillan JP et al. Coronary artery bypass surgery in women compared with men: analysis of risks nad long-term results. *J Am Coll Cardiol.* 1983;4:383-390.

117. Smith SC Jr, Gupta R, Alvario R. Canine distal aortic aneurysm and rupture: the very elderly compared with that in younger aged dogs. *J Vet Cardiol*. 1990; 16:784-792.
118. Adolph NJ. The elderly: the very elderly and traditional medicine. *J Am Coll Cardiol*. 1990; 16:793-798.
119. Topol EJ, Chait RM. Thrombolytic therapy in elderly patients. *N Engl J Med*. 1993; 327:43-47.
120. Collins R, for the ISIS Collaborators. Treatment with aspirin and aspirin plus clopidogrel in patients with acute myocardial infarction: age as a covariate. *Lancet*. 1990; 335:828-32.
121. Peto R. Which patients to treat with aspirin? *Lancet*. 1990; 335:1024-1026.
122. Mulcaugh EO, Bortnick SE. Aspirin in the elderly. *Am J Geriatr*. 1990; 38:10-12.
123. Fajno G. Thrombolytic use in the elderly. *JAMA*. 1991; 265:310-311.
124. Rich MW. Acute myocardial infarction in the elderly. *J Geriatr*. 1991; 36:10-12.
125. Robert WJ, Bonney LJ, Lander H. et al. Treatment of myocardial infarction in the United States (1990 to 1995). *Circulation*. 1994; 90:1100-1107.
126. Topol DJ, Watersham Z, Wexler J, et al. Late time to reperfusion: coronary bypass surgery. *Ann Intern Med*. 1987; 107:16-23.
127. White H, Conolly J, Cook EF, et al. Random differences in the use of invasive cardiovascular procedures in the treatment of various medical systems. *The New Engl J Med*. 1993; 329:611-617.
128. Maynard C, Linn PE, Martin JS, et al. Characteristics of black patients admitted to coronary care units in metropolitan Seattle: results from the Myocardial Infarction Triangulation and Intervention Registry (MITI). *Am J Cardiol*. 1993; 71:18-23.
129. Cook EF, Conolly J, Wexler J, et al. Coronary artery bypass surgery in women compared with men: analysis of rates and long-term results. *J Am Coll Cardiol*. 1993; 21:383-390.

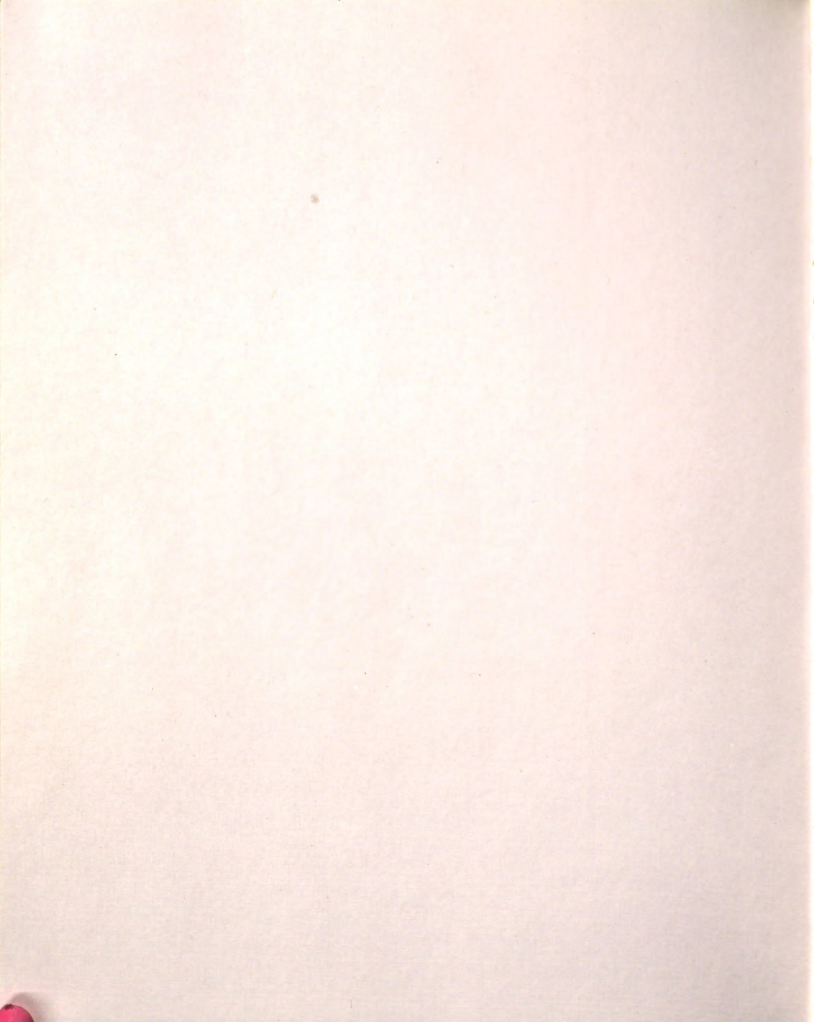
129. Tyras DH, Barner HB, Kaiser GC et al. Myocardial revascularization in women. *Ann Thorac Surg.* 1978; 25:449-453.
130. Killen DA, Reed WA, Arnold M, et al. Coronary artery bypass in women: long-term survival. *Ann Thorac Surg.* 1982;34:559-563.
131. Hall RJ, Elayda MA, Gray A, et al. Coronary artery bypass: long-term follow-up of 22,284 consecutive patients. *Circulation.* 1983;68(suppl II):20-25.
132. Myers WO, Davis K, Foster ED, et al. Surgical survival in the Coronary Artery Surgery Study (CASS) Registry. *Ann Thorac Surg.* 1985;40:245-260.
133. Douglas JS, King SB, Jones EL, et al. Reduced efficacy of coronary bypass surgery in women. *Circulation.* 1981;64(suppl 2):11-16.
134. Eaker ED, Kronmal R, Kennedy JW, et al. Comparison of long-term, postsurgical survival of women and men in the Coronary Artery Surgery Study (CASS). *Am Heart J.* 1989; 117:71-81.
135. Davis KB. Coronary artery bypass graft surgery in women. In: Eaker ED, Packard B, Wenger NK, et al, eds. *Coronary Artery Disease in Women.* New York, NY: Haymarket-Doyma; 1987:247-250.
136. Clark RE, Edwards FH, and Schwartz M. Profile of preoperative characteristics of patients having CABG over the past decade. *Ann Thorac Surg.* 1994;58(6):1863-1865.
137. Gersh BJ, Kronmal RA, Frye RL, et al. Coronary arteriography and coronary artery bypass surgery: Morbidity and mortality in patients aged 65 years or older. *Circulation.* 1983;67:483-491.
138. Montague NT, Kouchoukos NT, Wilson TAS, et al. Morbidity and mortality of coronary artery bypass grafting in patients 70 years of age and older. *Ann Thorac Surg.* 1985;39:552-557.
139. Loop FD, Lytle BW, Cosgrove DM, et al. Coronary artery bypass graft surgery in the elderly. *Cleve Clin J Med.* 1988;55:23-24.
140. Hibler BA, Wright JO, Wright CB, et al. Coronary artery bypass surgery in the elderly. *Arch Surg.* 1983; 118:402-404.
141. Horneffer PJ, Gardner TJ, Manolio TA, et al. The effects of age on outcome after coronary bypass surgery. *Circulation.* 1987;76(suppl V):6-12.

130. Tynes DH, Rennie HB, Kojan DE, et al. Myocardial revascularization in women. *Ann Thorac Surg*. 1978;22:449-453.
131. Eklund DA, Reed WA, Arnold M, et al. Coronary artery bypass in women: long-term survival. *Ann Thorac Surg*. 1982;34:519-523.
132. Hall RJ, Edwards MA, Gray A, et al. Coronary artery bypass: long-term follow-up of 22,384 consecutive patients. *Circulation*. 1983;68:1173-8.
133. Myers WO, Davis K, Pomeroy H, et al. Long-term survival in the coronary artery surgery group (CASS) Registry. *Ann Thorac Surg*. 1985;40:247-250.
134. Douglas JS, King SB, Jones EL, et al. Reduced mortality in coronary artery surgery in women. *Circulation*. 1981;64(suppl 1):1-6.
135. Eklund DA, Kinnison B, Kennedy JW, et al. Comparison of long-term postoperative survival of women and men in the Coronary Artery Surgery Study (CASS). *Am Heart J*. 1980;100:1173-41.
136. Davis EB. Coronary artery bypass graft surgery in women. In: Eklund DA, Kinnison B, Wanger NK, et al, eds. *Coronary Artery Bypass in Women*. New York: Harper & Row; 1983:147-150.
137. Clark RL, Edwards FH, and Schwartz M. Effects of postoperative characteristics of patients having CABG on long-term survival. *Ann Thorac Surg*. 1983;36:1567-1569.
138. Gersh BJ, Kannel WB, Folsom AR, et al. Coronary angiography and coronary artery bypass surgery: Mortality and morbidity in patients aged 65 years or older. *Circulation*. 1983;67:463-467.
139. Monaghan MT, Hochberger MT, Wilson LYS, et al. Morbidity and mortality of coronary artery bypass grafting in patients 70 years of age and older. *Ann Thorac Surg*. 1985;39:232-237.
140. Loop FD, Lytle BW, Cosgrove DM, et al. Coronary artery bypass graft surgery in the elderly. *Cleve Clin J Med*. 1988;55:57-64.
141. Hibler BA, Wright JO, Wright CB, et al. Coronary artery bypass surgery in the elderly. *Arch Surg*. 1983;118:402-404.
142. Hornsby PJ, Gardner TJ, Manolis TA, et al. The effects of age on outcome after coronary bypass surgery. *Circulation*. 1987;76(suppl V):6-15.

142. Matangi MF, Strickland JA, Burgess JJ, et al. Elective aortocoronary bypass grafting in the elderly. *Can J Cardiol* 1987;3:342-244.
143. Mohan R, Amsel BJ, Walter PJ. Coronary artery bypass grafting in the elderly-a review of studies on patients older than 64, 69 or 74 years. *Cardiology*. 1992;80:215-225.
144. Kern MJ, Deligonal U, Galan K, et al. Percutaneous transluminal coronary angioplasty in octogenarians. *Am J Cardiol*. 1988; 61:457-458.
145. Holmes DR Jr, Holubkov R, Vliestra RE, et al. Comparison of complications during percutaneous transluminal coronary angioplasty from 1977 to 1981 and from 1985 to 1986: the NHLBI Angioplasty Registry. *J Am Coll Cardiol*. 1988; 12:1149-1155.
146. Hannan EL, Kilburn H Jr, O'Donnell JF, et al. Interracial access to selected cardiac procedures for patients hospitalized with coronary artery disease in new york state. *Medical Care*. 1991;29(5):430-441.
147. Gillum RF. Coronary artery bypass surgery and coronary angiography in the United States, 1979-1983. *Am Heart J*. 1987;113:1255-1260.
148. Cowley MJ, Mullin SM, Kelsey SF, et al. Sex differences in early and long-term results of coronary angioplasty in the NHLBI PTCA registry. *Circulation*. 1985;71:90-97.
149. Holt GW, Sugrue DD, Bresnahan JF, et al. Results of percutaneous transluminal coronary angioplasty for unstable angina pectoris in patients 70 years of age and older. *Am J Cardiol*. 1988;61:994-997.
150. Dorros G, Janke L. Percutaneous transluminal coronary angioplasty in patients over the age of 70 years. *Cathet Cardiovasc Diagn* 1986;12:223-229.
151. Hartzler GO, Rutherford BD, McConahy DR, et al. "High-risk" percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1988; 61:33G-37G.
152. Cowley MJ, Kelsey SF, Costigan TM, et al. Percutaneous transluminal coronary angioplasty in women: gender differences in outcome. In: Eaker ED, Packard B, Wenger NK, Clarkson T, Tyroler HA, eds. *Coronary Artery Disease in Women*. New York, NY: Haymarket-Doyma; 1987;251-256.
153. Scott NA, Kelsey SF, Detre K, et al. Percutaneous transluminal coronary angioplasty in African-American patients (the National, Heart, Lung, and Blood Institute 1985-1986 Percutaneous Transluminal Coronary Angioplasty Registry). *Am J Cardiol* 1994; 73(16): 1141-1146.

143. Manning MB, Strickland JA, Burgess JJ, et al. Exercise measurement of angiotensin in the elderly. *Can J Cardiol* 1983;7:343-344.
144. Mannan R, Anand BJ, Walter ET. Coronary artery bypass grafting in the elderly: review of studies on patients older than 60, 65 or 70 years. *Catheterizing*. 1982;36:175-225.
145. Kern MJ, Deligonis U, Galan K, et al. Postoperative morbidity and mortality in angioplasty in octogenarians. *Am J Cardiol* 1985;55:453-456.
146. Holmes DR Jr, Holubsky R, Williams III, et al. Treatment of coronary disease during percutaneous transluminal coronary angioplasty from 1977 to 1981 and from 1985 to 1986: the NHLBI Angioplasty Registry. *Circulation* 1988;77:1144-1152.
147. Hannan EL, Kilburn H Jr, O'Connell B, et al. Intervessel disease in atherosclerotic coronary procedures for patients angioplasty with coronary artery disease: a multi-center study. *Medical Care* 1981;19(2):430-441.
148. Gilliam RP. Coronary artery bypass surgery and percutaneous angioplasty in the United States, 1979-1983. *Am Heart J* 1987;113:121-126.
149. Cowley MJ, Mellin SM, Kelsey SE, et al. Age differences in early and late results of coronary angioplasty in the NHLBI PTCII Registry. *Catheterizing*. 1987;37:97-107.
150. Holt DW, Sagarin DD, Bransford JR, et al. Effects of percutaneous transluminal coronary angioplasty for unstable angina pectoris: results in series of 100 and older. *Am J Cardiol* 1988;61:964-967.
151. Dorros G, Janke L. Percutaneous transluminal coronary angioplasty in patients over the age of 70 years. *Cathet Cardiovasc Technol* 1986;13:123-129.
152. Hartzler GO, Rutherford BD, McCannay DB, et al. "High-risk" percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1988;61:730-736.
153. Cowley MJ, Kelsey SE, Costigan JM, et al. Percutaneous transluminal coronary angioplasty in women: gender differences in outcome. In: Baim ES, Pasterkamp WG, et al. *Coronary Artery Disease in Women*. New York: Haymarket-Dovner; 1987:251-256.
154. Scott NA, Kelsey SE, Dotz K, et al. Percutaneous transluminal coronary angioplasty in African-American patients (the National Heart, Lung, and Blood Institute 1985-1986 Percutaneous Transluminal Coronary Angioplasty Registry). *Am J Cardiol* 1994;73(10):1141-1146.

143. Manning MT, Swickard JA, Burgess JL, et al. Preoperative coronary bypass grafting in the elderly. *Can J Cardiol* 1987; 3:341-346.
144. Minton R, Arnold BJ, Walker M. Coronary artery bypass grafting in the elderly: a review of studies on patients older than 65. *Can J Geriatr* 1987; 30:225-235.
145. Kern MJ, DeGroot LJ, Gelin R, et al. Preoperative myocardial ischemia: angiography in octogenarians. *Am J Cardiol* 1982; 51:447-450.
146. Holmes DR Jr, Holstebek R, Villanar JR, et al. A review of the complications during percutaneous transluminal coronary angioplasty from 1967 to 1981 and from 1982 to 1986: the NHLBI Angioplasty Registry. *Circulation* 1988; 77:1149-1155.
147. Hannan EL, Kliborn H Jr, O'Rourke D, et al. Intracoronary thrombolytic agents: cardiac procedures for patients undergoing early coronary artery bypass in new York state. *Medical Care* 1991; 29(2):140-46.
148. Gilliam RF. Coronary artery bypass surgery and coronary angiography in the United States, 1979-1983. *Am Heart J* 1987; 113:1125-30.
149. Cowley MJ, Mullins SM, Kealey SP, et al. Age-related trends in early and late results of coronary angioplasty in the NHLBI PIIA Registry. *Circulation* 1987; 75:973-977.
150. Holt GW, Sledge DO, Brannan JR, et al. Results of percutaneous transluminal coronary angioplasty for unstable angina pectoris in patients 70 years of age and older. *Am J Cardiol* 1988; 61:904-907.
151. Durrer G, Jaisie L. Percutaneous transluminal coronary angioplasty in patients over the age of 70 years. *Cathet Cardiovasc Techn* 1986; 15:221-229.
152. Hancher GO, Rubenford BD, McCarthy DE, et al. "High-risk" percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1988; 61:300-307.
153. Cowley MJ, Kealey SP, Costigan TM, et al. Percutaneous transluminal coronary angioplasty in women: gender differences in outcomes. In: Haber ED, Franklin B, Wenger NK, Charlson T, Tyroler HA, eds. *Coronary Artery Disease in Women*. New York: NY: Hemisphere-Penguin; 1987:251-256.
154. Scott WA, Kealey SP, Durrer G, et al. Percutaneous transluminal coronary angioplasty in African-American patients (the National Heart, Lung, and Blood Institute 1983-1986 Percutaneous Transluminal Coronary Angioplasty Registry). *Am J Cardiol* 1994; 73(10): 1141-1146.



MICHIGAN STATE UNIV. LIBRARIES



31293015725512