THE IMPACT OF SOCIOECONOMIC STATUS ON CLINICAL FEATURES AND OUTCOMES OF ACUTE MYOCARDIAL INFARCTIONS

Dr. Julia Uhanova

A Thesis

Submitted to the Faculty of Graduate Studies

In Partial Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

Department of Community Health Sciences

Faculty of Medicine

University of Manitoba

Winnipeg, Manitoba

National Library of Canada
Acquisitions and Bibliographic Services
395 Wellington Street
Ottawa ON K1A 0N4
Canada

Bibliothèque nationale du Canada
Acquisitions et services bibliographiques
395, rue Wellington
Ottawa ON K1A 0N4
Canada

Your file Votre référence

Our file Notre référence

0-612-57588-8

Canada

THE UNIVERSITY OF MANITOBA

FACULTY OF GRADUATE STUDIES

*****

COPYRIGHT PERMISSION PAGE

The Impact of Socioeconomic Status on Clinical Features and Outcomes of Acute Myocardial Infarctions

BY

Dr. Julia Uhanova

A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirements of the degree of

Master of Science

ABSTRACT

An association between lower socioeconomic status and poorer outcomes of acute myocardial infarction (AMI) has been consistently observed for years. The main objective of this study was to examine the relationship between socioeconomic status, clinical covariates, treatments, and outcomes of acute myocardial infarction.

Research Questions:

1. Does socioeconomic status (SES) have an impact on outcomes of AMI?

2. What might be the clinical reasons for socioeconomic differences in mortality?

3. After taking comorbidities and treatment differences into account, does socioeconomic status influence survival after acute myocardial infarction?

Methods: To address the study questions, a Manitoba Centre for Health Policy and Evaluation AMI data set, containing both administrative and clinical data, was used. Information on index hospitalization was obtained from Manitoba Health hospital separation abstracts, which contained the ICD-9-CM code "410" (AMI) in any of the 16 diagnostic fields. Overall, 2,223 Manitoba residents aged 45 to 99, who were discharged from hospital between October 1, 1991 and September 30, 1992, were identified. Clinical information was taken from a detailed chart review of all hospital discharges identified by the above criterion. A final cohort of 1,059 residents of Winnipeg was selected. Income quintile rankings were based upon the 1991 census of the Manitoba population. Logistic regression analysis was used to assess whether and how patients' various demographic, clinical and socioeconomic characteristics were associated with the outcome variables such as mortality or treatment. The odds ratios and 95% confidence

intervals for each model variable, as well as areas under the ROC curves among the various data sets and models were assessed.

Results: Patients from the lowest income quintile tended to be unmarried, older, and female. Addition of socioeconomic status to the logistic regression models revealed that even after controlling for numerous demographic and clinical risk factors, income level was still a significant predictor of mortality after AMI. Patients who belonged to the highest income group (Q5) had a better short-term and long-term survival after AMI. The overall model performance was very good with the ROC area statistics for 30-day, 1-year, and 5-year mortality, being 0.83, 0.84, and 0.86 respectively. Similarly, after controlling for the demographic characteristics and medical therapies received in the hospital, socioeconomic status still played an important role in the outcome of AMI. Socioeconomic status appears to affect utilization of beta-blockers, heparin and thrombolytic therapy.

Conclusion: Even after controlling for the most significant comorbidities, clinical presentation, and treatment covariates, SES contribution to mortality was still significant. In particular, patients who belonged to the highest income quintile (Q5) had a much improved chance to survive an episode of AMI than their lower income counterparts.

DEDICATION

TO MY MOTHER

ACKNOWLEDGMENTS

My thanks to my thesis committee members, Drs. Leslie L. Roos, T. Kue Young and Lindsay E. Nicolle, for their support and valuable input while this work was in progress.

My very special thanks to my thesis advisor, Dr. Leslie L. Roos, whose knowledge, suggestions, and enthusiasm while reading hundreds of pages, carried me through the maze of administrative data and logistic regression to the happy ending in the form of this thesis.

My many thanks to the wonderful people at the Manitoba Centre for Health Policy and Evaluation, where I did most of my analytical work and where I always felt welcomed. In particular, I would like to acknowledge Ruth Bond and Randy Walld, for their assistance when I needed it.

Also, I would like to thank Dr. Bob Tate who willingly reviewed and commented on the final draft of this thesis.

I am also grateful to the Medical Research Council of Canada for its Studentship Award that allowed me to further my educational goals.

Finally, my most heartfelt thanks and appreciation to my wonderful family and friends, both in Canada and in Russia, who always supported me and believed in me.

This research was supported by the Medical Research Council of Canada studentship to Dr. Julia Uhanova.

TABLE OF CONTENTS:

LIST OF FIGURES

LIST OF TABLES

LIST OF APPENDICIES

LIST OF ABBREVIATIONS

ACE inhibitors	Angiotensin Converting Enzyme inhibitors
AMI	Acute Myocardial Infarction
BUN	Blood Urea Nitrogen
CABG	Coronary Artery Bypass Graft surgery
CAD	Coronary Artery Disease
CHD	Coronary Heart Disease
CHF	Congestive Heart Failure
CRF	Chronic Renal Failure
DM	Diabetes Mellitus
ECG	Electrocardiography
ICD-9-CM	International Classification of Diseases, 9th revision, Clinical Modifications
IHD	Ischemic Heart Disease
LOS	Length of Stay
MCHPE	Manitoba Centre for Health Policy and Evaluation
MUGA scan	Multiple Gate Angiography scan
PTCA	Percutaneous Transluminal Coronary Angioplasty
ROC	Receiver Operating Characteristics
SES	Socioeconomic Status

CHAPTER ONE BACKGROUND

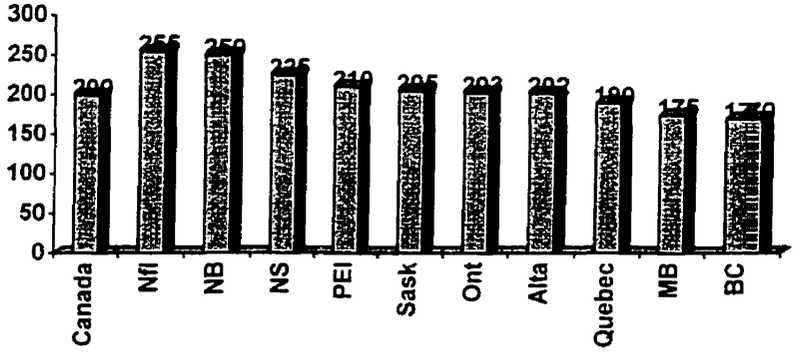
1.1 Acute Myocardial Infarctions (AMI)

Morbidity

Beginning in the middle of the twentieth century, many countries of the developed world were suffering from the "epidemic" of cardio-vascular diseases. This disease imposes a significant burden on individuals, on the society, and on the health system. Substantial evidence suggests that, despite the large resources invested in treatment and prevention, the burden of heart diseases on health care is rising (Naylor, 1999 speech). Although undeniable progress is made in the treatment of acute myocardial infarction, this condition remains one of the leading causes of hospitalizations and deaths in Canada. Acute myocardial infarction becomes an important health problem even in middle aged people. AMI not only affects the elderly but is also the third leading cause of premature death under age 75.

The data from the 1999 Statistical Report on the Health of Canadians illustrates the dimensions of the problem. Annually, almost 45 thousand men and 28.5 thousand women are hospitalized with acute myocardial infarctions in Canada. The disease is somewhat more prevalent among Maritime Provinces and lower in Prairie Provinces and British Columbia. Manitoba enjoys the second lowest average rate of AMI (Figure 1.1).

Figure 1.1 5-Year Average Morbidity Rates (per 100,000) of AMI (1989-93)



J. Uhanova

Mortality

Acute myocardial infarctions account yearly for 10.4% of all deaths (11.4% among men and 9.4% among women) in Canada. AMI death rates vary between the provinces. The Atlantic Provinces had higher mortality rates than the Western provinces, while in both Territories the rates were much below Canadian average due to a significantly younger population (Figure 1.2 and Table 1.1).

Figure 1.2 Age-Sex Adjusted Rates of Death from AMI, Canada 1996

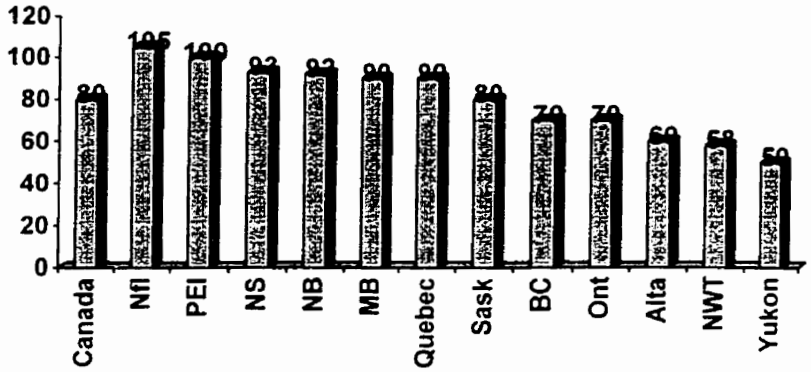


Table 1.1 Percent and Rates of Deaths due to AMI

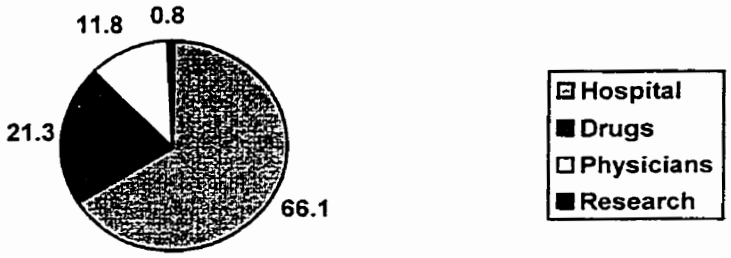
Province	All deaths	AMI	Rates* per 100,000 population
Canada	**212,855**	**10.4%**	**80.0**
Quebec	52,336	11.8%	90.0
Manitoba	9,497	11.0%	90.0
Newfoundland	3,928	11.0%	105.0
Saskatchewan	8,765	10.6%	80.0
PEI	1,268	10.4%	100.0
Ontario	79,099	10.1%	70.0
British Columbia	27,539	10.0%	70.0
New Brunswick	5,896	10.0%	92.0
Nova Scotia	7,751	9.3%	93.0
Alberta	16,391	8.6%	60.0

* Rates = Age-Sex Adjusted Mortality Rates

Economic Burden of AMI

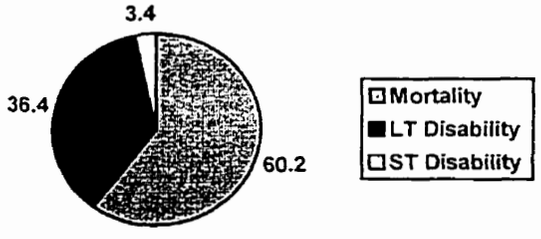
High incidence and mortality determine the overall significant economic impact of acute myocardial infarctions. Cardiovascular diseases are the most "expensive" diseases. The total cost of cardiovascular disease in Canada was estimated to range from $14.1 billion to $20.4 billion in 1994 (Johansen, Nair et al., 1998). Their direct cost (which includes costs of hospitalizations, drugs, physicians' reimbursement and research) comprised over $7,3 billion, which is 17% of the total direct costs of illness in Canada. Approximately one third of these expenditures were consumed by AMI. Almost 4 million physicians' visits were related to the care and treatment of myocardial infarctions. In 1995-96, cardiovascular diseases were the major cause of hospitalizations, accounting for approximately 6 million hospital days in total. More than ten per cent of this were hospital days consumed by AMI. The most significant expenditure (almost $5 billion), comprised 66.1% of direct cost, was the cost of hospitalizations. The cost of drugs was $1,6 billion; payment to physicians came to $867 million; while cardiac research consumed only $60 million (Figure 1.3).

Figure 1.3 Direct Cost of Cardiovascular Diseases (%)



Another estimator of the overall economic impact of illness is its indirect cost, which combines the value of lost productivity due to illness, disability, and loss of future earnings due to premature death. Indirect cost of cardiovascular diseases came to $12,4 billion or 14.5% of the total of indirect costs of illness in Canada in 1993 (this is the second highest rate). Myocardial infarctions were accountable for the significant share of that cost as well. By far the greatest proportion of indirect costs of cardiovascular diseases is associated with mortality (Figure 1.4).

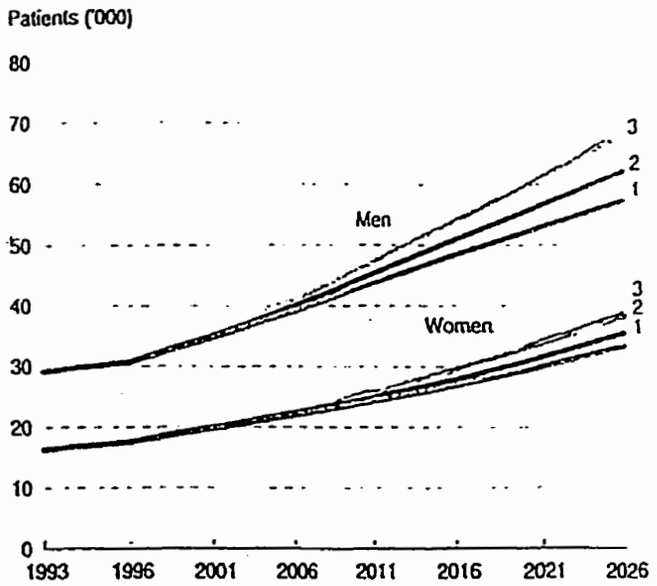
Figure 1.4 Indirect Cost of Cardiovascular Diseases (%)



LT Disability = long-term disability
ST Disability = short-term disability

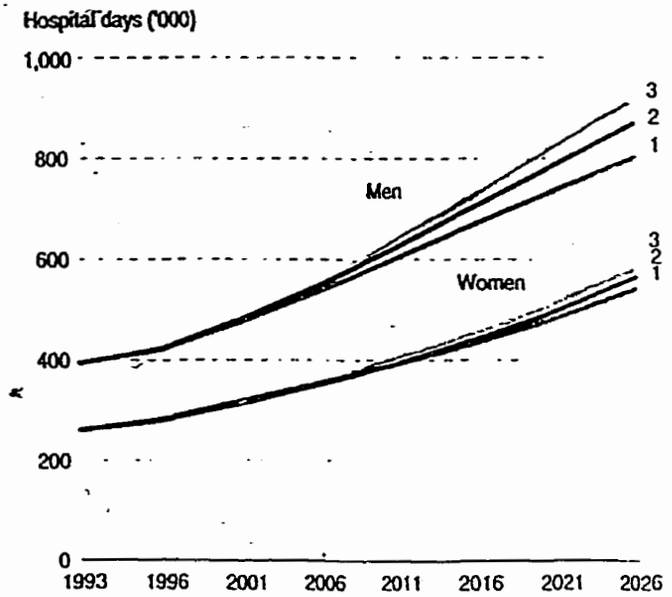
The urgency of identifying and implementing optimal prevention strategies and management of AMI is further intensified by the predicted trends in cardiovascular diseases. Based on the current rates, the number of AMI cases is expected to almost double by the year 2026, due to population growth and aging (Figure 1.5). The increase is approximately 36% in each decade. Accordingly, the number of hospital days used by AMI patients would also rise ranging from 1,340,000 to 1,478,000 in 2026 (Figure 1.6), (Johansen, Nair et al., 1998). Therefore, identifying factors that may adversely influence

Figure 1.5 Projected number of AMI patients, Canada excluding territories, 1993 to 2026



Data source: Population Projections Section, Demography Division, Statistics Canada; Person-Oriented Information Data Base

Figure 1.6 **Projected number of hospital days needed for AMI patients, Canada excluding territories, 1993 to 2026**



Data source: Population Projections Section, Demography Division, Statistics Canada; Person-Oriented Information Data Base
Notes: Projections assume 1993/94 hospitalization rates persist in the future. Low-, medium- and high-growth projections are numbered 1, 2 and 3, respectively.

(from Johansen et al, 1998: Current and Future Hospitalization after Heart Attack)

outcomes of AMI is imperative with future efforts to developing adequate prevention strategies.

1.2 Measurements of Socioeconomic Status in Epidemiological Research

Social class has long been considered important for studying health and disease in populations. "Centuries of evidence – dating back to ancient Greece, Egypt, and China – demonstrate strong associations between socioeconomic position and morbidity and mortality: Poor living and working conditions impair health and shorten lives" (Krieger et al., 1997). Furthermore, even in the world's most developed countries with their high standards of living, the same association is evident. Growing inequalities in income and wealth are accompanied by the increasing socioeconomic inequalities in health (Krieger et al., 1997; Kaplan and Keil, 1993; Liberatos et al, 1988). Moreover, many studies also illustrate that "mortality rates for both children and adults in industrialized countries are directly related not only to poverty but also to degree of income inequality" (Krieger et al., 1997).

The definitions of the meaning of "social class" or "socioeconomic status" are complex. The term "social class" is traditionally used in Europe, while "socioeconomic status" is mostly used in North America. Conceptualization of these terms is a domain of sociologists, while most epidemiologists use the terms interchangeably. To summarize, social class is a broader concept which, according to Weber, incorporates class (or economic interest), status, and power. Class can be described as a membership in a particular social group arising from different socioeconomic relations (based on income,

wealth, ownership, etc). Status is a hierarchy according to prestige and honor in the community (thus bearing different access to life opportunities) (T.K.Young, 1998).

The most widely used indicators of social class are occupation, education, and income (Krieger et al., 1992, Krieger et al., 1997; Kaplan and Keil, 1993; Liberatos et al, 1988). Occupation reflects either status (based on public perception of prestige), or economic wealth (based on education and income), and, therefore, measures the socioeconomic aspect of social class. Similarly, education is an indicator of both status (by placing an individual on different ladders in a social network) and economic position (providing the necessary background for a particular occupation and, therefore, income). Income is an economic indicator also reflecting status and power. Strictly speaking, socioeconomic status, according to Krieger et al., "blurs distinction between two different aspects of socioeconomic position: (a) actual resources, and (b) status, meaning prestige- or rank-related characteristics" (Table 1.2).

Epidemiological and health research employs a wide variety of indicators, ranging from single measurements to composite indexes, to measure different aspects of socioeconomic status (SES). However, in epidemiology the socioeconomic status is a "descriptive term for a person's position in society, which may be expressed on an ordinal scale using such criteria as income, educational level attained, occupation, value of dwelling place, etc." (Last, 1995). The interpretation of the results of research depends on the chosen indicator, and how this indicator is considered. According to Liberatos, epidemiologists were considering SES to be: confounding variable (42%), risk factor (32%), and descriptive variable (26%). A wide variety of SES indicators can be measured on individual or area levels (Table 1.3).

Table 1.2 Definitions of Social Class and Socioeconomic Position

Definitions

Social class	Socioeconomic position
A social category referring to social groups forged by interdependent economic and legal relationships, premised upon people's structural location within the economy—as employers, employees, self-employed, and unemployed, and as owners, or not, of capital, land, or other forms of economic investments; possession of educational credentials and skill assets also contribute to social class position	An aggregate concept that includes both resource-based and prestige-based measures, as linked to both childhood and adult social class position. Resource-based measures refer to material and social resources and assets, including income, wealth, educational credentials; terms used to describe inadequate resources include "poverty" and "deprivation". Prestige-based measures refer to individual's rank or status in a social hierarchy, typically evaluated with reference to people's access to and consumption of goods, services, and knowledge, as linked to their occupational prestige, income, and education level

Implications for data analysis

Levels	Time periods	Modeling of variables
Socioeconomic position can be measured meaningfully at three complementary levels: (*a*) individual, (*b*) household, and (*c*) neighborhood. Each level may independently contribute to distributions of exposures and outcomes	Socioeconomic position can be measured meaningfully at different points in the lifespan, e.g. infancy, childhood, adolescence, adult (current, past 5 yr, past 10 yr, etc). Relevant time periods depend on presumed exposures, causal pathways, and associated etiologic periods; cohort and period effects may also be operative	Social class is, conceptually, a nominal categorical variable; characteristics of socioeconomic position pertaining to material resources can be modeled as ordinal or interval categorical variables; socioeconomic status and other ranked hierarchical measures may be modeled as continuous variables (assuming no threshold effects), with cutpoints, if any, based on the structure of the data (e.g. quintiles)

(from Krieger et al., 1997: Measuring Social Class in US Public Health Research: Concepts, Methodologies, and Guidelines

TABLE 1.3

*Summary of social class measures by five characteristics**

Indicators and measures	Categories and scores	Reliability	Evidence for validity†	Advantages	Disadvantages
Occupation‡		73% agreement at 3 points each 4 weeks apart (133). Correlations between occupation reported 5 years apart, $r = 0.80$ (134); son's proxy report of father's occupation with father's report, $r = 0.74$ (135); two census surveys, $r = 0.87$ (107).	Correlations with income, $r = 0.40$ (107); education, $r = 0.61$ (107).		
Edwards	12 categories; data comparable for censuses 1940–1980; 13 categories for 1980.		Major occupational groups can be ranked on income and education with high correspondence (51).	Used by Census Bureau; provides comparability over time since 1940; widely used since 1940 (51).	Each occupational category contains wide variations in income and education (20).
Nam-Powers OSS	Scores range 0–100; available for 1950, 1960, 1970, and 1980 censuses.	Compared 1950 with 1960 occupations, $r = 0.96$ (51); 1960 with 1970, $r = 0.97$ (51).	Correlation with Duncan, $r = 0.75$ (20).	Each score interpretable as a cumulative percentile; data available for male, female, black, and total labor forces.	Not sufficiently used to provide empirical evidence of its performance.
Siegel	Scores range 0–100; available for 1960 census only.		Correlations with education, $r = 0.84$ (51) and 0.56 (21); income, $r = 0.85$ (51); Duncan, $r = 0.87$ (51); Treiman, $r = 0.93$ (21) and 0.95 (51).	One of few scales using prestige scores.	Based on prestige data collected 20 years ago; not updated to 1980; available for male labor force only.
Treiman	Scores range 0–100; can be grouped into 8 occupational levels.	Mean intercountry correlation, $r = 0.97$.	Correlations with Siegel, $r = 0.93$ (21) and 0.95 (51); Duncan, $r = 0.84$ (21).	Only occupational scale that applies internationally; applies to both industrialized and developing countries.	Based on prestige data collected 20 years ago; not updated to 1980; available for male labor force only.

Education	Usual category range 2–5; sometimes used as a quantitative variable.	89% agreement at 3 points each 4 weeks apart (133). 62% agreement after 3 years (137). Correlation between 2 census surveys, r = 0.93 (21).	Correlations with occupation, r = 0.61 (107); income, r = 0.33 (107).	Stable over lifetime; good predictor of mortality from all causes (105).	Fixed early in adult life cycle; decreasing variability over time; status does not rise monotonically with years.
Income	Categories vary depending on population.	27% agreement at 3 points each 4 weeks apart (133). Correlation between 2 census surveys, r = 0.85 (21).	Correlations with occupation, r = 0.40 (107); education, r = 0.33 (107).	May measure unique aspects of social class (71).	Varies within occupations and is inconsistent with education (20); sensitive to changes in life circumstances; increases with age up to age 65; not comparable across different years or family sizes unless adjusted; sensitive topic in United States (9% refusal rate) (132).
Composites					
Duncan	Scores range 0–99; available for 1950, 1970, and 1980 censuses.		Correlations with Siegel, r = 0.90 (21); Treiman, r = 0.84 (21) and 0.87 (51); education, r = 0.84 (51) and 0.61 (21); income, r = 0.85 (51); Hollingshead, r = 0.74 (51); Nam-Powers SES, r = 0.97 (51); Nam-Powers OSS, r = 0.75 (20).	Most frequently used in social science research (20).	Positively skewed distribution (53); original scale based on 1950 male labor force; updates use studies from 1960s to supplement original 1947 study.
Hollingshead	Original: scores range 11–77, subdivided into 5 classes, available for 1950 census. Revision: scores range 8–66, subdivided into 5 classes, available for 1970 census.		Correlation with Duncan, r = 0.74 (51). Respondent patterns of use of mass media corresponded with a priori expectations (7).	Widely used during 1960s and early 1970s.	Original: based on 1950 census; validated in one small Connecticut city. Revision: scores for each working spouse are averaged; census categories used in revision have been modified requiring additional questions of respondents; not updated to 1980.

TABLE 1—*Continued*
*Summary of social class measures by five characteristics**

Indicators and measures	Categories and scores	Reliability	Evidence for validity†	Advantages	Disadvantages
Nam-Powers SES	Scores range 0–100; available for 1950, 1960, 1970, and 1980 censuses.	Compared 1950 with 1960 occupations, r = 0.96 (51); 1960 with 1970, r = 0.97 (51).	Correlation with Duncan, r = 0.97 (51).	Each score interpretable as a cumulative percentile; data available for male, female, black, and total labor forces; scores are normally distributed (53).	Not sufficiently used to provide empirical evidence of its performance; potentially redundant if used in combination with person's education and income.
Warner	Scores range 12–84.		Compared with prestige ratings of a sample of community residents (51). Correlations with occupation, r = 0.91 (51); income, r = 0.85 (51); house type, r = 0.85 (51); dwelling area, r = 0.82 (51).		Difficult to rate dwelling area and house type; limited applicability since validated on small communities in 1940s.
Indices combining income and education	Ad hoc measures.	Study-specific, if done.	Study-specific, if done.	Can be specifically tailored to study population.	No systematic validation; each scale specific to a given study, making cross-study comparisons difficult.
Other indices	Ad hoc measures.	Study-specific, if done.	Study-specific, if done.	Can be specifically tailored to study population.	No systematic validation; each scale specific to a given study, making cross-study comparisons difficult.
Area-based	Ad hoc measures.	Study-specific if done.	Study-specific, if done.	Relatively inexpensive; easy to use.	"Ecological fallacy."

* Edwards, Edwards' Social-Economic Grouping of Occupations; Nam-Powers OSS, Nam-Powers' Occupational Status Scores; Siegel, Siegel's Prestige Scale; Treiman, Treiman's Standard International Occupational Prestige Scale; Duncan, Duncan's Socioeconomic Index; Hollingshead, Hollingshead's Index of Social Position; Nam-Powers SES, Nam-Powers' Socioeconomic Status Scores; Warner, Warner's Index of Status Characteristics.

† Some correlations are based on the occupational, educational, and income distributions within the US labor force; they tend to be higher than correlations obtained in a sample of individuals (20).

‡ Some reliability and validity data have been collected for occupation without reference to a specific social class measure.

(from Liberatos et al., 1988: The Measurement of Social Class in Epidemiology).

Individual Measures of SES:

- ***Education.*** This measure is becoming the most widely used measure of SES in epidemiologic studies (Kaplan and Keil, 1993); Liberatos et al., 1988). Education incorporates class (ability to acquire certain income) and status (lifestyle, influence). It can be measured as a continuous variable (total number of years of schooling), or a categorical variable (degree / certificate attained). The advantages of this measure include simplicity and relative stability over people's life period, compared to income or occupation. Education is less influenced by poor health in adulthood and is strongly associated to life style, therefore reflecting behavioral risk factors and health practices. The downside of education as a proxy of SES lies in (1) important birth cohort differences in the level of education; (2) existence of regional differences in education; (3) education is not always a reliable predictor of occupational rank and income level; (4) link between education and income vary between different population strata (the relationship is weaker for women and for visible minorities, then for men and for Caucasians); and (5) education may be altered by poor health or unfavorable childhood circumstances.

- ***Income measures.*** Income is a very important indicator of SES in providing access to goods and services and reliably predicting prestige-based status. Measuring income can be complicated. It can be measured at the individual level or adjusted according to family size. Area-based measure of income will be discussed later. Income can be measured as a quantitative or categorical variable. Since respondents view income to be confidential, the categorical approach is more common. The advantage of the

individual income versus family income as a measure of SES is in its sensitivity to the individual's changes in health and disability status and in life circumstances, etc. However, income as a single measure is not a reliable indicator of SES. It has the largest variations among populations, and is often not consistent with education. Different sources of income (beyond employment) exist and change over time. Income is an unstable measurement and changes from early adulthood to retirement. The individual's financial situation at a particular time point may reflect some temporary events. Finally, large inter- regional variations in the cost of living make individual income less suitable for comparisons.

- ***Occupation measures.*** The occupation-based measures became an important characteristic of SES based on both status and class property and are categorical in nature. Many different scales exist to rank occupations according to their perceived status and role in society, power, prestige (status), as well as on educational requirements and monetary rewards (class). These rankings, however, may vary depending on culture, traditions, beliefs, and values of the particular society. The ranking of students, temporarily unemployed and women outside the work (usually ranked according to the husband's occupation) is problematic. Two most commonly used occupational classifications are the British Registrar General's Scale and The US Bureau of the Census (Table 1.4). Many studies reported strong association between the social class (as measured by one's occupation) and all-cause mortality, infant and maternal mortality, low birth weight, prevalence of such risk factors as cigarette smoking and sedentary life style. However, all occupational measures are

based on somewhat subjective prestige rankings. Moreover, the composition of work force changes, new occupations emerge, and old (primarily unskilled) occupations disappear, making comparisons over time difficult due to problematic compatibility of occupational rankings. This highlights the need of frequent revisions of occupational classifications.

Table 1.4 Occupational Categories for the British Registrar General's and the US Bureau of the Census Scales

British Registrar General's Scale		US Bureau of Census (1980)
I	Professionals	Executives, administrators, and managers
II	Managerial and lower professionals	Professionals
IIIN	Non-manual skilled workers	Technicians and related support
IIIM	Manual skilled workers	Sales workers
IV	Partly skilled workers	Administrative / clerical support
V	Unskilled workers	Service workers – private household
		Protective service workers
		Service workers (except protective and household)
		Farm operators and managers
		Mechanics and repairers
		Machine operators, assemblers, and inspectors
		Transportation and material moving operatives
		Handlers, equipment cleaners, helpers, and laborers

- ***Indexes of Social Class***. A large number of composite measures combining several individual measures of SES has been developed (Table 1.3). They usually include measures of education attained, income, occupational prestige rankings, and may also include characteristics of neighborhood. An example of such index is Warner's Index of Status Characteristics, which include occupation, source of income, house type, and dwelling area. These indexes attempt to achieve the most comprehensive description of SES, but all of them have the disadvantage of collecting multiple components and determining relative weights of each component.

- ***Measures Based on the Characteristics of Living Conditions.*** These relatively simple measures, such as house or car ownership, had been shown to correlate with income and education, but they may be also a result of differences in the lifestyle.

Area-Based Measures of SES:

To supplement individual characteristic of SES, many studies use socioeconomic characteristics of areas (neighborhoods) in which the subjects reside. Each geographic area can be described in terms of social and economic conditions: housing, crowding, per cent of unemployment in the area, income, education, and occupation. Two examples of convenient and readily available geographic units are Census Enumeration Areas (in Canada) and Census Tracts (in USA), which can be linked to postal codes. These units define populations with relatively similar social and economic characteristics (i.e. the average population of EA is approximately 600 people). To keep the confidentiality of subjects, the census information on areas with a very small population is suppressed (i.e.

less then 250 per EA in Canada). Area-based socioeconomic indicators are derived from the national census data. Examples of these are US census-based measures of socioeconomic position and UK census-based indices of deprivation (Table 1.5). The characteristics of individuals within the area are summarized as average values or proportion of population with certain characteristics (Morgenstern, 1995). Examples of such measures are the average neighborhood income and unemployment: per cent of unemployed people in the area. Area-based measures have several advantages in being relatively inexpensive, easy to use, and employable when no individual data are available. These measures can be similarly applied across ages and genders. They are more stable in describing socioeconomic circumstances, than individual measures. Area-based measures usefully characterize general living conditions that are not measured on the individual level.

As with every measure, there are some important limitations. Principal amongst them is "ecological fallacy", otherwise known as aggregation bias. This type of bias "may occur because an association observed between variables on an aggregate level does not necessarily represent the association that exists at an individual level" (Last, 1995). However, the opposite bias can occur when the area-based measures are not taken into account, and it is assumed that population patterns of outcome can be explained only by individual-level variables. Area-based measures not simply a function of individual measures; area affects health independently of the SES characteristic of individuals living in that area. Area-based effects may help emphasize the need to targeted health promotion on areas where ill health occur rather then people who live there. Individual

and area-based measures do not always have the same patterns, and often complement each other describing different aspects of SES.

In summary, socioeconomic status is a multidimensional concept. This might explain the existence of a variety of SES indicators. They are usually used as single indicators; or several single indicators may be collected to obtain more information; or a combination of indicators is incorporated to construct composite indices. The choice to use area-based SES indicators versus individual indicators is usually based on data availability. Both indicators would show the same direction of association between SES and health outcome; however, the area-based measures usually would underestimate the strength of association. Furthermore, using both sets of indices is desirable, since they are not simply substitutes for one another, but rather independent contributors to health outcome. The degree of inequality in health can not be reliably demonstrated by using only one set of indicators. In explaining health outcomes one should account for both characteristics of the area where people live and characteristics of individuals themselves. By using both types of measurements of SES (area-based and individual), the research would provide insights how social class influences health on every level.

1.3. Study Questions

The main focus of this work was to study the relationship among covariates, treatment, and survival after acute myocardial infarction. After first assessing the importance of covariates derived from both administrative and clinical data, the role of socioeconomic status was assessed. The most important questions were: Does socioeconomic status (as determined by the average neighborhood income) impact

clinical presentation and outcomes after acute myocardial infarction? After controlling for health status using every appropriate covariate (from administrative data and from clinical data), does socioeconomic status influence the treatment after acute myocardial infarction? Furthermore, even after taking comorbidities and treatment differences into account, does socioeconomic status influence survival after acute myocardial infarction?

The following hypotheses were explored in this study:

1. Socioeconomic status is inversely related to the outcomes of AMI;
2. Socioeconomic status negatively influences health status and clinical presentation of AMI;
3. Socioeconomic status is inversely related to the provision of AMI- related care.

Table 1.5 Examples of US census-based measures of socioeconomic position and UK census-based indices of deprivation[a]

US census-based measures of socioeconomic position	UK census-based indices of deprivation
Social class: % working class Defined as % of employed persons in 8 of 13 census-defined occupational groups: Administrative support Sales Private household service Other service (except protective) Precision production, craft, repair Machine operators, assemblers, inspectors Transportation and material moving Handlers, equipment cleaners, laborers	*Townsend index* Unemployment: % economically active residents aged 16–64 and unemployed No car: % households with no car Rented: % households not owner occupied Overcrowding: % >1 person/room Note: index does not weight variables, uses log transformation of % unemployment and % overcrowding; uses Z score for standardization
Working-class neighborhood: ≥66% of employed persons in working-class occupations	*Breadline index* Unemployment: % economically active population unemployed No car: % households with no car Rented: % households not owner occupied Lone parents: % lone parents as proportion of all households Long-term illness: % households with a person with a limiting long-term illness Low social class: % persons in social class IV or V Note: index estimates % poor using weights derived from a validation survey
Poverty: % persons below poverty line	
Poverty area: ≥20% of persons below poverty	
Additional measures: % of persons at <50%, 50–100%, 101–200% of poverty line	
Wealth: % of households owning home % of households owning 1 or more cars % of households with annual family income ≥$50,000 or more	
Education: % of adults age 25 and older with less than a high school degree	*Doe 91 Index of Local Conditions* Unemployment: % unemployed persons Poor children: % households with no earner or one parent in part-time employment Overcrowding: % households with >1 person per room Lack amenities: % households lack or share baths/shower and/or water closet, or in non-permanent housing No car: % households without access to a car Flat children: % children living in flats, not self-contained or non-permanent housing Note: index does not weight variables; uses X^2-standardization
Undereducated neighborhood: ≥25% of adults with less than a high school degree	
Alternative: % of adults age 25 and older who have completed ≥4 years of college	
Crowding: % of persons living in households with ≥1 person/room	
Population density: persons/square mile	

(from Krieger et al., 1997: Measuring Social Class in US Public Health Research: Concepts, Methodologies, and Guidelines

CHAPTER TWO LITERATURE REVIEW

2.1 Outcomes of AMI

Trends and variations in AMI morbidity and mortality highlight the role of the social and economic environment in determining the population patterns of this disease. Marmot and Mustard (1994) argued that "Socioeconomic differences within countries have come to assume such major importance that continued exploration of underlying connections between social position and CHD is likely to be fruitful ". Indeed, many researchers follow that path in order to gain an understanding of SES patterns in outcomes of AMI. Factors such as economic conditions, social environment, nutrition, risk behavior, early life-style are important in investigating how the SES gradient in AMI morbidity and mortality actually operates. Kaplan and Keil (1993) argued that there is still a lack of knowledge about the environmental and biologic pathways by which SES affects cardiovascular disease. However, the available evidence indicates that SES is independent of other known risk factors (Ibid.). This effect persists over time and is observed among different populations, regardless of the type of indicators used to measure SES.

The systematic surveillance of myocardial infarctions revealed considerable variation in mortality associated with the disease among the provinces (Table 1.1, Figures 1.1-1.2). Similar variability exists on a smaller scale: within the provinces and even within the municipalities. Large population-based analysis of the outcomes of acute myocardial infarctions was recently conducted in Ontario by the Institute for Clinical Evaluative Sciences (Tu et al, 1999). Detailed regional and hospital-specific analysis detected the existence of differences in mortality rates among hospitals in Ontario. 30-

day mortality among the province's District Health Councils (DHC) varied from 13.3% to 18.5%. A similar variation was found in 1-year mortality: from 20.8% to 27.4% (the latter was much above the provincial average). Other findings were related to the rank of the hospital. Significant variations in 30-days mortality were found among teaching hospitals (13.8%), large urban hospitals (14.7%), medium (17.0%) and small (15.5%) hospitals in Ontario (Tu et al, 1999). These differences did not persist in the one-year mortality rate, thus emphasizing the crucial role of in-hospital care for immediate survival. For long-term survival, however, other factors (discussed below) played a major role.

Numerous studies had shown SES to be a consistent predictor of mortality after AMI. Thus, Alter et al. have studied access to invasive cardiac procedures and outcomes of AMI in Ontario among a 1994-1997 cohort of patients and found a large effect of income on 1-year mortality after AMI. A "$10,000 increase in neighborhood median income was associated with a 10 percent reduction in the risk of death at one year" (Alter et al., 1999). That effect was consistent among all age groups.

Peltonen and colleagues' study (2000) of social patterns of AMI in Sweden within the population-based MONICA project found similar relations. The AMI cohort was restricted to patients (ages 25-64 years) who had their first-ever AMI during 1985-1994, with the occupational group* as SES indicator.

**Occupational groups were as follows:*

A - unskilled and semi-skilled workers;
B – skilled workers;
C – assistant non-manual employees;
D – intermediate non-manual employees;
E – employed and self-employed professionals (also higher civil servants and executives);
F – self-employed other than professionals (farmers, entrepreneurs);
G - "not classified": includes early retired, unemployed, home workers, housewives, and students.

Figure 2.1 Incidence Rates for first-ever AMI by SES (1985 - 1994)

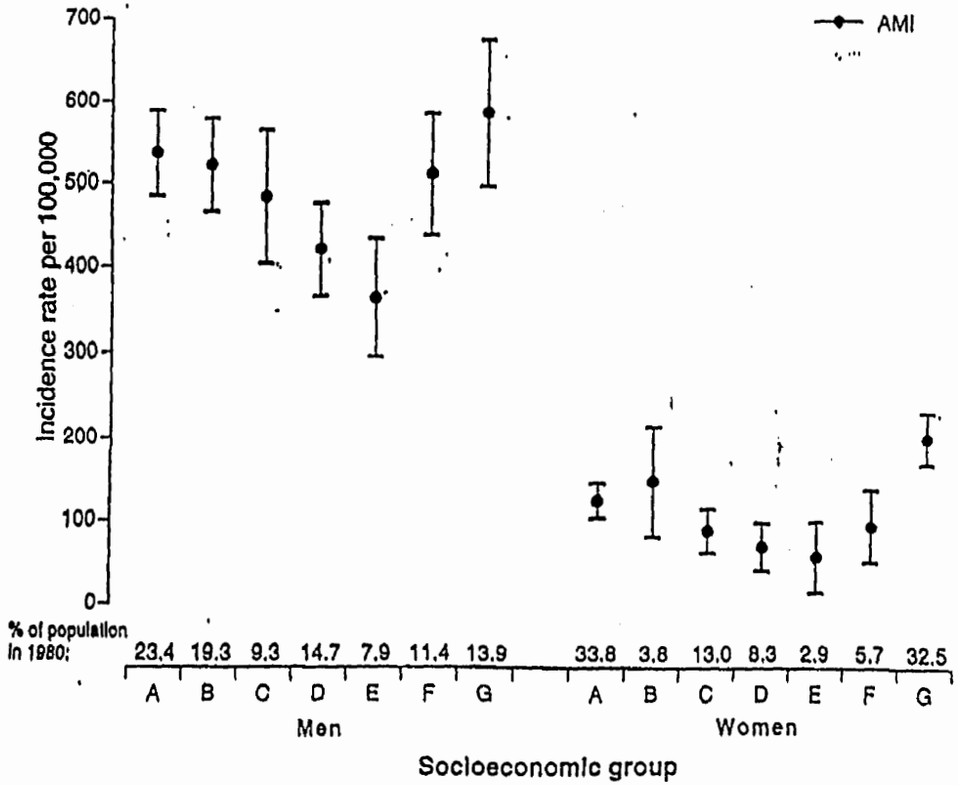


FIGURE 2.1 Incidence rates and 95% confidence intervals (CIs) for first-ever acute myocardial infarction (AMI) by socioeconomic group, Northern Sweden MONICA Project, 1985–1994. AMI: men and women aged 40–50 years in 1980; stroke: men and women aged 40–60 years in 1980. Adjusted for age at onset. MONICA, Multinational Monitoring of Trends and Determinants in Cardiovascular Disease. Socioeconomic groups: A, unskilled and semiskilled workers; B, skilled workers; C, assistant nonmanual employees; D, intermediate nonmanual employees; E, employed and self-employed professionals; F, self-employed (nonprofessionals); G, not classified.

From Peltonen et al., 2000: Social Patterning of Myocardial Infarction and Stroke in Sweden: Incidence and Survival.

FIGURE 2.2 Adjusted 28-Day and One-Year Case Fatality among Patients with first-ever AMI. (From Peltonen et al., 2000: Social Patterning of Myocardial Infarction and Stroke in Sweden: Incidence and Survival).

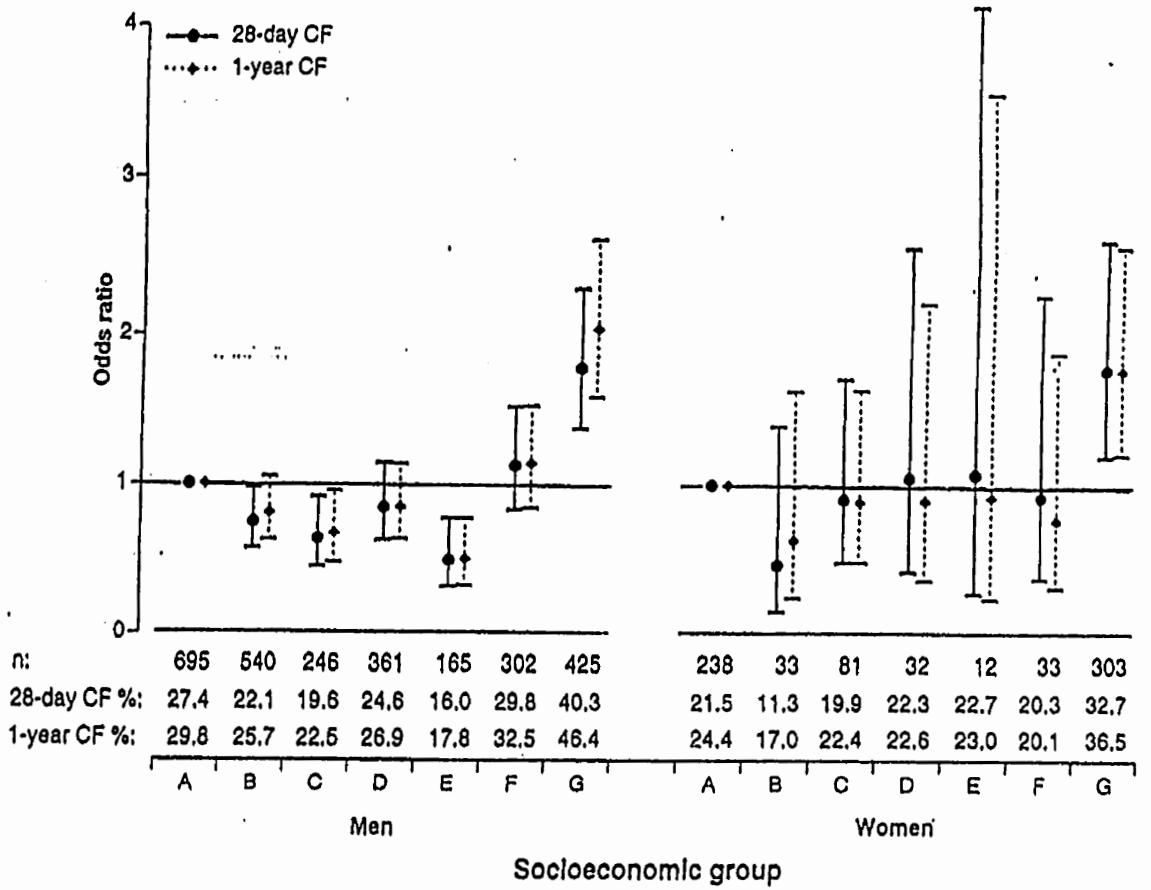


FIGURE 2.2 Adjusted 28-day and 1-year case fatality (CF) with odds ratios and 95% confidence intervals for death among first-ever acute myocardial infarction patients by socioeconomic group in the Northern Sweden MONICA Project, 1985–1994. Men and women aged 40–60 years in 1980. Adjusted for age at onset, time period of onset, and place of living. MONICA, Multinational Monitoring of Trends and Determinants in Cardiovascular Disease. Socioeconomic groups: A, unskilled and semiskilled workers; B, skilled workers; C, assistant nonmanual employees; D, intermediate nonmanual employees; E, employed and self-employed professionals; F, self-employed (nonprofessionals); G, not classified.

Age-adjusted incidence rates of AMI "...showed a distinct social pattern, with high rates in workers and self-employed non-professionals and low rates in professionals [Figure 2.1]. The pattern was similar in men and women" (Peltonen et al., 2000). AMI case-fatality had exhibited similar patterns (Figure 2.2), with the "...proportion of cases of sudden deaths being twice as high in unskilled and semiskilled workers as in professionals" in men (Ibid.). Since specific selection criteria were employed in the study (age up to 64 years as well as only first-ever episode of AMI), the results do not reflect the total burden of AMI in the population. However, the authors pointed out that excluding older people allowed emphasizing of the impact of social stratification on the population patterns of the disease, since that impact tends to decrease with increasing age (Ibid.).

2.2 Studies of Treatment of AMI

Medical Therapies

Drug therapies for acute myocardial infarctions have undergone a significant development in the last three decades. Many clinical trials, on the national and international levels, have clearly proved the survival benefits of using certain drugs and their value in prevention of re-infarctions after an initial episode of acute myocardial infarction. "These medications include aspirin, which reduces the risk of death by 12% and the risk of re-infarction by 31%, and beta-blockers, which reduce long-term mortality by 20% and sudden cardiac death by 34%. Angiotensin converting enzyme (ACE) inhibitors have also been shown to improve survival by 23% in patients with left ventricular dysfunction after an infarct" (Tu et al., 1999). Conversely, clinical studies did

not show any clinical or survival benefits of such therapies as calcium-channel blockers or prophylactic use of lidocaine for this condition (Ayanian et al, 1994). To summarize the results of a large body of research, the American College of Cardiology and American Heart Association published in 1992, and revised in 1996 and 1999 "Clinical Guidelines for the Early Management of Patients with Acute Myocardial Infarction". Clinical practice guidelines based on the latest research evidence provide directions for the appropriate use of a wide range of therapeutic interventions.

During the last decade, U.S. and Canadian studies have invariably shown the existence of a gap between recommendations for practice and actual practice. This applies not only for primary AMI treatment but also for secondary prevention. All drug utilization figures in actual practice are much less than optimal. Rogers et al's (1994) retrospective analysis of treatment of AMI in the USA in 1990-1993 used data from the National Registry of Myocardial Infarction to evaluate how physicians followed the clinical guidelines in AMI treatment. Drug therapy was underutilized: only 35.1% of patients received thrombolytic therapy, and 53.7% received beta-blockers.

In another study by McLaughlin et al. (1996), researchers mailed clinical guidelines to 37 hospitals in Minnesota, and later reviewed the implementation of treatment recommendations. They found that 81% of eligible patients received aspirin therapy, 72% received thrombolytic agents, and only 53% of eligible patients were treated with beta-blockers. Some disparities in the use of these drugs in elderly patients and in women were reported.

A retrospective study of the 1991/92 AMI cohort in Manitoba showed similar rates of aspirin therapy (82%), but lower rates of thrombolytic therapy (59%) in patients

considered eligible according to 1992 ACC/AHA guidelines (those current at the time of hospitalization and treatment) (Murray et al, 1999).

Since myocardial infarctions are treated not only by cardiologists and internists, but also by general practitioners, knowledge and practice patterns may differ between specialists and general practice physicians. Ayanian et al (1994) examined knowledge and practice patterns regarding drug therapy for AMI among 1,211 physicians who treated this condition in New York and Texas. The results were predictable: cardiologists were more likely to prescribe beneficial treatment in accordance to the data of recent clinical trials and guidelines. Ninety-four percent of cardiologists were prescribing thrombolytic agents to treat an acute myocardial infarction, as compared to 82% of internists and 77% of family physicians. Only about half of family practitioners and internists vs. about three-quarters of cardiologists believed in the survival benefits of the immediate use of aspirin. On the other hand, the two treatments proven not to have survival benefits in clinical trials (lidocaine and calcium-channel blockers) were believed to be useful by 40% of general practitioners and 34% of internists compared to 11% of cardiologists, and prescribed by 17%, 13%, and 5% accordingly. Cardiologists were quicker to incorporate the results of clinical trials into their practice style. As specialists, they are more exposed to conferences, workshops, and specialized journals where new results are communicated; they also participate in clinical trials as investigators. The age of a physician also played an important role. Older physicians were less prone to change practice style, while younger practitioners were more likely to utilize the results of recent clinical trials.

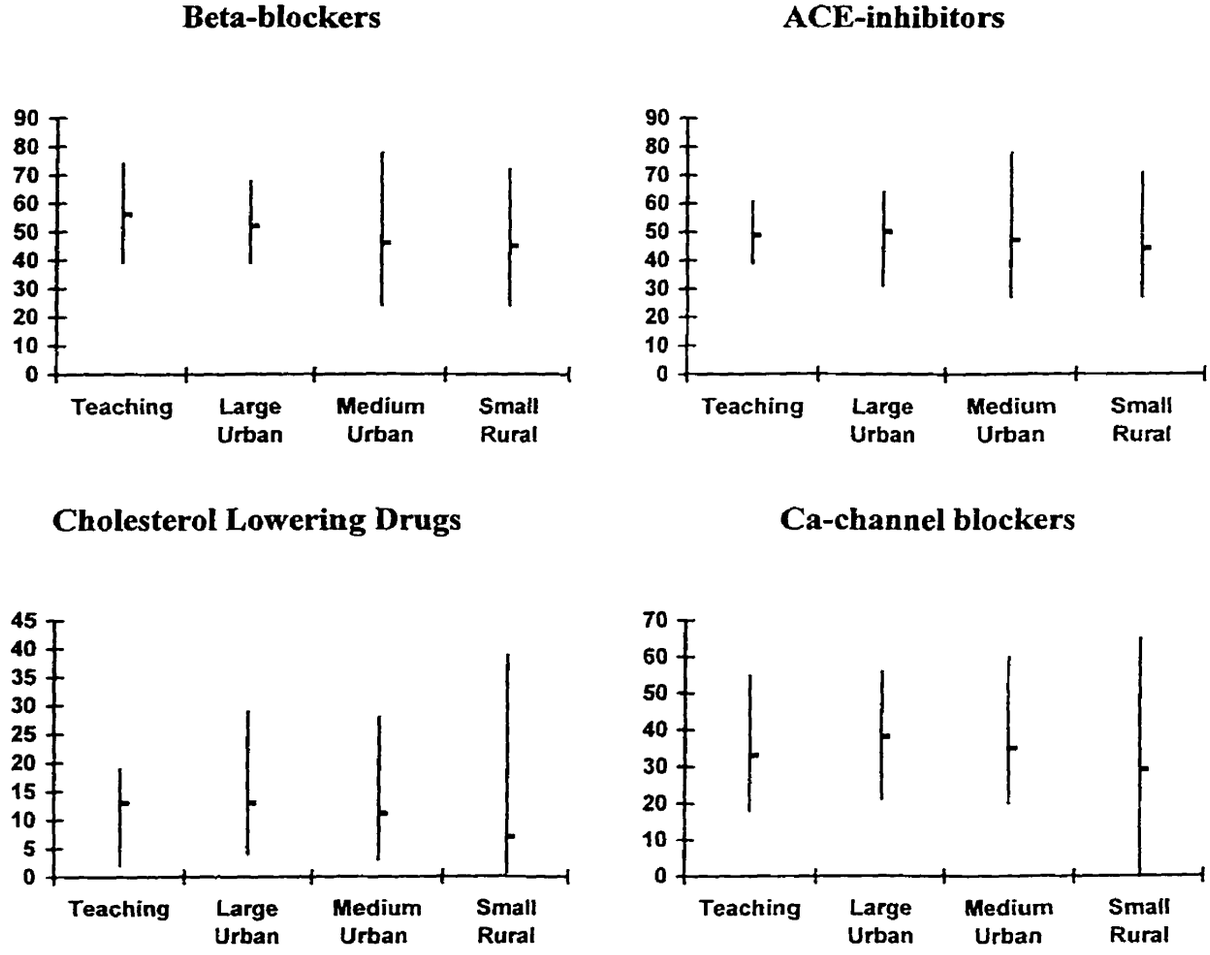
Patients surviving initial myocardial infarction usually remain at increased risk of developing cardiac complications and death. Aspirin and beta-blockers, used in the initial AMI treatment, significantly reduce the risk of re-infarction and death among such patients if used long-term after an initial episode of AMI. "More recently in the 4S (Scandinavian Simvastatin Survival Study) and the Cholesterol and Recurrent Events (CARE) trials, the cholesterol-lowering statin drugs were shown to reduce coronary events and the need for revascularization procedures" (Tu et al., 1999). In general, aspirin and beta-blockers should be used in all post-AMI patients who do not have contraindications to these drugs. One vehicle to improve the treatment outcomes of myocardial infarctions is a greater conformity to clinical guidelines in clinical practice.

Results of the Manitoba study (Murray et al, 1999) showed that the rate of long-term use of aspirin was 69% in the 1991/92 cohort of eligible AMI patients, while the U.S. rate in the same time period was 77-81%. The rates of use of beta-blockers were similar: 42% in Manitoba and 45% in U.S.

Tu with his colleagues (1999) evaluated the secondary prevention in post-AMI patients in Ontario. They found a slight increase in the usage of beta-blockers from 48% in 1994/95 to 51% in 1996-97 and a decrease of use of calcium-channel blockers from 39% in 1994/95 to 34% in 1996/97. Rates of ACE-inhibitor use increased from 45% to 52% in these years, but the largest increase was in the use of cholesterol-lowering drugs: from 7% in 1994/95 to 20% in 1996/97. It was not possible to evaluate each individual case of AMI in order to determine justification of use or failure to use these recommended drugs (Tu et al, 1999). Nevertheless, the existence of large and unexplained variations in prescription practice not only between different regions in

Ontario, but also between similar classes of hospitals, demonstrate the obvious lack of consistency in treatment strategies. For example, seventeen teaching hospitals in Ontario had ranges in 90-day post-discharge prescription rates of recommended drugs from 39% to 74% for beta-blockers, from 39% to 61% for ACE inhibitors. At the same time, the rates for prescribing the no longer recommended calcium channel blockers were unnecessary high: from 55% to 18% (Figure 2.3).

Figure 2.3 90-Days Post-discharge Utilization Rates (%) of AMI Drugs, Ontario Hospitals, 1994-1997.



The difference in the highest and lowest utilization rates of these drugs, even between hospitals of similar capacity, reaches more than 200-300%. These results clearly show

that optimization of drug therapy for a post-infarction patient is the path to follow in attempting to improve outcomes and enhance the health of individuals after myocardial infarction.

Phillips et al. (2000), while studying health and economic benefits of increased use of beta-blockers, reviewed the relevant literature, finding that this effective therapy is still used in rates less than optimal. Their best estimates of beta-blockers use in 2000 are 44% (increase from 30% in 1990), while the eligibility is 92% (i.e., all patients without absolute contraindications). This therapy was particularly underused in women and older patients. Although the issue of whom to consider an appropriate candidate for the beta-blockers therapy is still unresolved, the authors argued that "...even among those elderly patients who are considered "ideal" candidates for treatment, only about half are prescribed beta-blockers on discharge" (Phillips et al. 2000). [The "ideal" candidates are those without both absolute and relative contraindications]. On the population level, the increase in rates of beta-blockers therapy after AMI should have not only an impressive impact on health, but would also be potentially cost saving (Ibid.).

Access to Physician Services

With the decrease in length of hospital stay, more and more diagnostic and treatment procedures are shifted from the hospital to the community. Ontario data show very little variations in post-AMI follow-up by family physicians, but large geographic variations in access to specialty care. On average, patients have their first post-discharge visit to family physicians in two weeks after discharge from the hospital, and then about once every five to six weeks. "Nonetheless, about one in eight patients appear to have no

follow-up by family physician and almost one-quarter are not seen at all by a cardiologist or internist after discharge..." (Chan, 1999). Average waiting time for AMI patients to see a cardiologist after discharge from an Ontario hospital was five to six weeks. In Northern Ontario, the waiting period was extended to nine weeks and 35% of patients did not have any follow-up by cardiologist. These findings suggest that a significant number of AMI patients do not have sufficient follow-up. Clinical guidelines recommend a wide range of interventions after primary hospitalization. Guidelines include not only a long-term use of prescription drugs, but also education and modification of risk factors, diet control, and other health-related activities. In conclusion Chan stated that "...It is difficult to see how all these recommendations can be promoted if there is no primary care physician follow-up in the six month following AMI. These findings raise important questions about the adequacy of follow-up and barriers to care".

Mark and co-authors' (1994) study on the use of medical resources and quality of life after acute myocardial infarction argued that "...the Canadians had more visits to physicians during the follow-up year, but significantly fewer visits to specialists. At 30 days, the functional status was equivalent in the patients from the two countries. However, after one year the U.S. patients had substantially more improvements than the Canadian patients...[who] had more cardiac symptoms...Both more visits to specialist physicians and more use of cardiac-rehabilitation services, is a potential explanation for the observed differences". However, a comparison of medication use revealed a higher rate of long-term use of beta-blockers after discharge in Canada. Canadian physicians less often prescribe drugs no longer considered beneficial, which is more consistent with the clinical guidelines. Although American patients underwent invasive diagnostic and

revascularization procedures more frequently, the mortality rates at one year after AMI were similar in both countries. The described differences accounted only for the better functional status and fewer cardiac symptoms (without proven survival benefits) in U.S. patients as opposed to Canadian patients.

Use of Invasive Cardiac Procedures after AMI

The large regional and interhospital variations in the rates of invasive diagnostic and revascularization procedures (coronary angiography, percutaneous transluminal coronary angioplasty [PTCA], and coronary artery bypass graft surgery [CABG]) are documented in many studies. Canadian rates remain significantly lower than the U.S. rates, but higher than those in most European countries. Many factors determine the use of these procedures; the major one is the availability of the facilities. Cox et al. (1994) had studied the impact of hospital teaching status and service availability on rates of revascularization procedures after myocardial infarction. AMI patients admitted to a hospital with on-site cardiac services were more likely to undergo invasive diagnostic tests and subsequently angioplasty or bypass surgery than were patients admitted to a hospital without such facilities. The referral rates for angiography determined the rates of subsequent revascularization procedures. Thus, access to angiography was the most important factor on which future rates of surgical procedures depended. A thorough assessment of waiting lists for coronary angiography is of great importance (although public opinion concentrates mostly on the waiting lists for angioplasty or bypass surgery); the rates of these surgical procedures are largely predetermined (and also limited) by the rates of the invasive diagnostic test – angiography. The reasons for the

lower Canadian rates of these procedures, as compared to the U.S. rates, are in the structure of financing of health systems in these countries. " Unlike the private hospital sector in the United States, there is less financial incentive within the Canadian health care system to perform more revascularization. While fee-for-service arrangements give Canadian practitioners some incentives for use of procedures, Canadian hospitals on fixed budgets face considerable pressure to rationalize the use of resources. Indeed, contemporaneous comparisons have consistently shown the practice patterns on the whole are more conservative in Canada than they are in the United States" (Cox et al, 1994).

Anderson et al (1993) who compared the use of CABG surgery in United States and Canada reported similar results. Age-adjusted rates of CABG in New York and California were 80% higher than in 3 Canadian provinces (British Columbia, Manitoba, and Ontario). The income gradient in the rates of CABG was much more pronounced in the US (with the rates rising with increasing income), while in Canada rates varied relatively little by median income of residential areas.

Another study by Tu and his colleagues (1997) compared the use of cardiac procedures in elderly AMI patients in USA and Canada. The comparison was based on 1991 cohort of AMI patients. Even larger differences in rates emerged: angiography 34.9% vs. 6.7%, PTCA 11.7% vs. 1.5%, CABG 10.6% vs. 1.4% respectively (during first 30 days after index AMI). However, the one-year mortality rates were almost identical for both American and Canadian cohorts. "Higher rates of use of cardiac procedures did not translate into better long-term survival rates for elderly patients in the United States" (Tu et al., 1997). Once again, it was suggested that the benefit of revascularization is in

improving the quality of life after AMI, but not in better survival (Tu et al., 1997), (Mark et al., 1994).

However, a trend to earmark resources for cardiovascular care is recently seen in Manitoba. In order to decrease waiting times for diagnostic and surgical cardiac procedures, the provincial government in 1998 announced additional $5.3 million funds for cardiac care. The $1.4 million cardiac catheterization program will "reduce waiting lists for inpatient and outpatient angiograms and angioplasties" (Manitoba Government News Release, 1998). Another capital project, renovation of HSC cardiac step-down unit, will also "significantly increase efficient through-put of cardiac surgery" (Manitoba Government News Release, 1999).

The study of invasive cardiac procedures in Manitoba by Hartford and colleagues revealed that the referral rates for diagnostic angiography largely depended on personal practice patterns of physicians. Rural physicians generally were less likely to refer patients to specialists than were urban physicians. "A 'funnel effect' is found; the fewer patients from a region referred to angiography, the fewer patients from that region who have CABG or PTCA" (Hartford et al, 1998).

While studying the impact of health reform and technological change in treatment of AMI, Roos with his colleagues found that a steady increase in invasive cardiac procedures started in Manitoba in 1977. By the year 1996, the rates for angiography increased by 29% (from 43.7 to 56.5 per 10,000); for bypass surgery by 69% (from 9.8 to 16.5 per 10,000); and for angioplasty by 67% (from 8.9 to 15.0 per 10,000). These rates increased even more for women. However, the Western Manitoba region had the highest rate of AMI, but the lowest rates of both diagnostic and revascularization procedures,

which could be explained only by the referral patterns of physicians from that region. Overall, "...SES was a major predictor of CABG and PTCA after acute myocardial infarction" (Roos et al, 1999). During 1989-1995, patients living in the wealthiest neighborhoods had rates of CABG and PTCA twice the rates of those from the poorest areas. Older patients, and to a lesser degree, female patients had somewhat lower referral rates for revascularization procedures. Both rates of AMI among Manitoba population and mortality (1989-1996) clearly showed a SES gradient.

A similar relationship between the income of patients and the rates of procedures, as well as waiting time, was reported in a recent Ontario study. Both the rates of procedures and the waiting time for these procedures were inversely related to the income of patients: the lower the income, the less was the likelihood of angiography and revascularization and the longer the waiting time for both procedures (Figure 2.4). Such results "...offer a dramatic demonstration of...persisting inequities in access for a cohort of persons who were hospitalized with the same condition and who should, in theory, have been treated similarly" (Alter et al, 1999). The authors concluded that "despite universal health insurance coverage, Ontario residents living in lower-income areas have reduced access to invasive procedures, as compared with residents of wealthier neighborhoods, and have sharply higher mortality one year after hospitalization for acute myocardial infarction. The causes of these socioeconomic disparities in access and outcome remain obscure, but their persistence poses a clear challenge to the egalitarian principles of Canada's publicly funded health care system" (Ibid.).

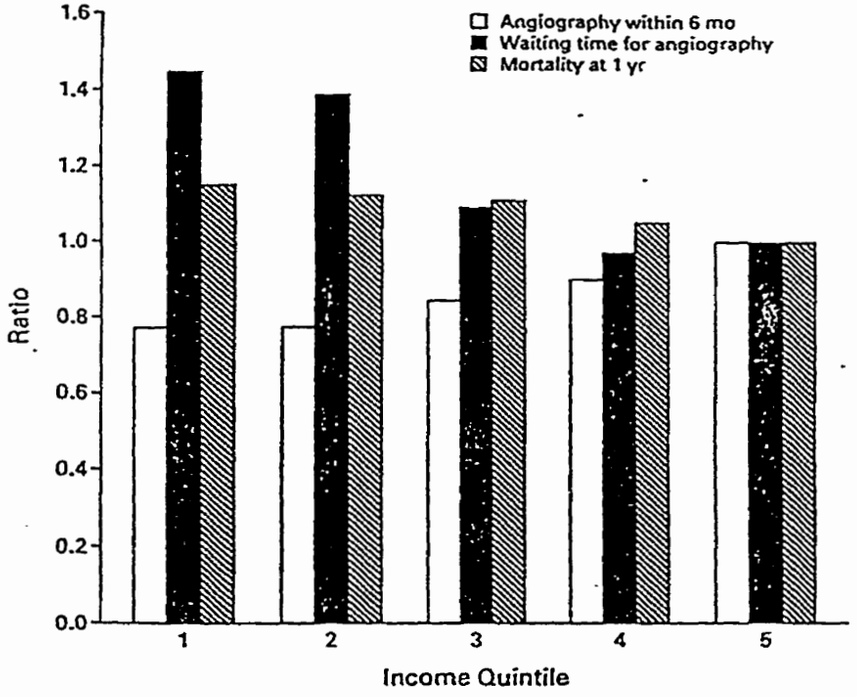


Figure 2.4 Adjusted Relative Rates of Angiography within Six Months after Acute Myocardial Infarction, Waiting Times for Angiography, and One-Year Mortality According to Income Quintile.

Results have been standardized for age, sex, and type of on-site facilities for cardiac procedures. Quintiles for neighborhood median income were derived from 1996 Canadian census data for 493 Forward Sortation Areas (neighborhoods defined by the first three digits of the postal code); the patients in the highest quintile served as the reference group. The bars show the relative differences in adjusted rates and waiting times. The absolute differences between the lowest and the highest income quintile were 7.4 percent for adjusted rates of angiography, 9.2 days for adjusted waiting times for angiography, and 3.1 percent for adjusted mortality rates. P for trend <0.001 for each of the three outcome variables.

(from Alter et al., 1999: Effects of Socioeconomic Status on Access to Invasive Cardiac Procedures and on Mortality After Acute Myocardial Infarction).

2.3 Economic and Psychosocial Factors and Outcomes of AMI

Some population studies of cardiovascular mortality "...have demonstrated an independent role of the psychosocial and economic variables by controlling for the standard cardiovascular risk factors" (Ruberman, 1992). Case et al. (1992) have studied if such factors as living alone or disrupted marriage would be predictive of a higher rate of re-infarctions or cardiac deaths in the cohort of AMI patients who participated in a randomized double-blind drug trail (placebo arm) in the U.S. and Canada. Patients aged from 25 to 75 were followed for 1 to 4 years. Living alone but not disrupted marriage was an independent risk factor for the increased rates of recurrent cardiac events and deaths. The hazard ratio for living alone or having less than 12 years of schooling was similar to the ratio for those who did not have the beta-blocker therapy, and larger than the hazard ratio for prior infarction and ventricular premature complexes $\geq$ 10/hour (Table 2.1). At 6 months after the index AMI, the rates of re-infarctions or cardiac deaths among those living alone was almost twice the rate of those not living alone (Case et al., 1992). Living alone was an independent risk factor for the subsequent major cardiac event.

Ruberman et al. (1984) conducted psychosocial interviews of male patients participating in Beta-blocker Heart Attack Trial. The 20-item questionnaire included questions about life stress, social isolation, and depression. A three-year mortality after AMI was assessed. Patients with a high level of life stress or socially isolated had "...more than four times the risk of deaths of the men with low levels of both stress and isolation" (Ruberman et al., 1984). The increased risk was noted for both total death and sudden cardiac death (Ibid.). Moreover, the inverse educational gradient in mortality was

associated with the high prevalence of adverse psychosocial circumstances (both high stress and social isolation) among the least-educated individuals.

A large prospective cohort study was undertaken by Williams et al (1992) to evaluate how social and economic factors impact on mortality among patients treated for coronary artery disease. Psychological and socioeconomic data were collected on a consecutive sample of patients undergoing angiography at Duke University Medical Center from 1974 to 1980 (Table 2.2). Patients were followed up through 1989 (Williams et al., 1992). After controlling for clinical and treatment variables, as well as for the extent and severity of cardiac disease, the effects of poor social support and economic resources were observed. Patients with higher household income had a much better survival rate. "Those with low incomes were almost twice as likely to die within 5 years of entry into the study as those with high incomes (Cox model adjusted hazard ratio, 1.9; 95% C.I. 1.57 to 2.32)" (Williams et al., 1992). Being married (or having a confidant) was even stronger associated with better survival. In this study, 5-year survival for unmarried patients was three-fold worse than for those with either spouse or confidant (Cox model adjusted hazard ratio, 3.34; 95% C.I. 1.84 to 6.20) (Ibid.). Three socioeconomic variables (income, number of dependants, and marital status) contributed an impressive 12% of the total prognostic information. The results of this study revealed that "...reduced levels of both economic and social resources significantly increased the risk of cardiovascular death over an extended follow-up period in a large cohort of patients with CAD, even after controlling for all known medical prognostic factors, including amount of CAD and left ventricular dysfunction" (Williams et al., 1992).

Table 2.1 Hazard Ratios and 95% Confidence Intervals for Recurrent Cardiac Events Without (Model I) and With (Model II) the Living Alone Variable*

	Model I		***Model II***	
Variable	Hazard Ratio	Confidence Interval (95%)	Hazard Ratio	Confidence Interval (95%)
NYHA class II-IV	1.84	1.21-2.80	1.85	1.21-2.81
LVEF<0.40	1.76	1.24-2.51	1.76	1.24-2.50
Education<12	1.62	1.16-2.27	1.59	1.13-2.22
No β-blockers	1.56	1.09-2.24	1.56	1.09-2.23
Living Alone	...	...	1.54	1.04-2.29
VPCs≥10/h	1.42	0.95-2.13	1.39	0.92-2.08
Prior Infarction	1.37	0.97-1.94	1.32	0.93-1.88

* From Case et al., 1992: Living Alone after Myocardial Infarction. Impact on Prognosis.

**NYHA indicates New York Heart Association; LVEF, left ventricular ejection fraction; VPCs, ventricular premature complexes.

*Table 2.2 Sample of Information Collected and Controlled for in the Study of Social and Economic Factors and CAD

Table 1. – Baseline Characteristics Important for Prognosis

- Left ventricular ejection fraction
- Noninvasive myocardial damage index (Q wave on ECG, history of AMI, cardiomegaly, S3 gallop, history and severity of congestive heart failure)
- Conduction disturbance on ECG (left bundle branch block, right Bundle branch block, nonspecific interventricular conduction defect, left axis deviation)
- Pain/ischemia index (frequency, progressive, stability, nocturnal, ST-T wave abnormalities on resting ECG)
- Mitral regurgitation
- Number of diseased vessels
- Percent stenosis of left main and left anterior descending arteries
- Age
- Left ventricular ejection fraction × number of diseased vessels
- Year in which catheterization was performed

Table 2. – Economic Status and Social Support Questions

Economic Questions

- How far did you go to school? (y)
- Are you currently working? Include housework, volunteer positions, etc.
- Range of present annual family income. Include earnings of spouse if working, investments outside, and etc.
- How many people are dependent on that income including yourself?
- Does your income meet your family's basic needs (i.e. food, clothing, shelter, and medical expenses)?
- Is there any remaining income after your basic needs are met or spending otherwise?

Social Support Questions

Structural

- What is your present marital status?

Functional

- Do you have as much social contact as you would like with friends and relatives?
- Are your family relationships satisfying, that is, do you get along well with members of your family, and do your find interactions with them rewarding?
- Do you have as much contact as you would like with someone you feel close to, someone you can trust and confide in?
- Do you find yourself feeling lonely often?

**From Williams et al., 1992: Prognostic Importance of Social and Economic Resources Among Medically Treated Patients with Angiographically Documented Coronary Artery Disease).*

2.4 SES and Risk Factors for Cardiovascular Diseases

The association between socioeconomic status and cardiovascular disease risk factors has been established and well documented in many studies. One prospective study undertaken by Smith and co-authors (1998) investigated the association between SES (measured both on individual level and area-based) and cardiovascular disease risk factors and mortality. Residents of two towns in Scotland aged 45-64 years were screened during 1972 – 1976 for the evidence of respiratory and heart diseases and followed for 15 years. Examination included BMI, forced expiratory volume (FEV) at 1 sec., blood cholesterol, blood pressure, and EKG). Questionnaires were completed with regard to risk factors (such as smoking habits, occupation, and respiratory/cardiovascular symptoms). Individual SES indicators and area-based measures had an independent contribution into the rates of risk factors, all-cause and coronary mortality. Authors found that "the inhabitants of deprived areas are more likely to be smokers, have poor lung function, be shorter, and for women, have higher body mass indices, even after the occupational social class of these people has been taken into account.... All cause and cardiovascular disease morbidity and mortality rates were both inversely associated with socioeconomic position whether indexed by area-based depravation or social class" (Smith et al., 1998). These results not only confirmed the adverse effects of lower SES but also validated the use of both area-based and individual indices of socioeconomic status.

Kaplan and Keil (1993) conducted a comprehensive review of literature on various aspects of the relationship between socioeconomic factors and cardiovascular diseases. They emphasize the inverse relationship between SES (particularly education)

and almost all risk factors for cardiovascular diseases (with the exception of cholesterol level). "Low SES is related to both prevalence and incidence of hypertension. There seems to be an inverse relation between SES and cigarette smoking, obesity, and some hemostatic factors such as fibrinogen, diabetes, and physical activity " (Kaplan and Keil, 1993). Most of the studies cited by the authors had concentrated on the assessment of selected risk factors. Other studies, however, highlighted similar trends in the prevalence of multiple risk factors. One example of those is the study within the Canadian Health and Canada Fitness Survey. As part of the Survey, adults aged 20-69 were examined between 1978 and 1991 to estimate the prevalence of cardiovascular risk factors (Ibid.). The measure of SES used in this project was education. "The prevalence of cigarette smoking, overweight, obesity, elevated diastolic blood pressure, physical inactivity, excessive alcohol consumption, elevated serum cholesterol, and diabetes mellitus and (in women) the simultaneous use of oral contraceptives and cigarettes tended to be higher among men and women with a lower level of education" (Kaplan and Keil, 1993). Such association is typical for the industrialized world. Authors pointed to the results reported by researchers from many other countries (studies by Jakobsen et al. in Norway, Greiser et al. In Germany, and Gold and Franks in USA), which where largely similar. All found the inverse association between different measures of SES and multiple cardiovascular risk factors (Ibid.).

2.5 Mortality Prediction Models

The growing attention to the assessment of outcomes of AMI, including comparisons on different levels (between hospitals, inter-regional, national, and international) requires developing a statistically sound algorithm. Several models have been developed using either clinical or administrative data. Lee et al (1995) using the large population of the participants enrolled in GUSTO-I trail developed a statistical model for the assessment of 30-day mortality among candidates for thrombolytic therapy. The final model was constructed from the baseline clinical data and included 5 variables: age, lower systolic blood pressure, elevated heart rate, anterior location of infarction, and higher Killip class*. The proposed model accurately estimated patient's risk and prognosis.

Another risk-adjusted model was proposed by Krumholz et al. (1999) based on the analysis of more than 82,000 patients 65 years of age and older, and validated on another group of more than 78,000 patients. After an initial evaluation of 73 variables, seven were selected for the model. These variables described patient's baseline characteristics upon arrival to the hospital: age, cardiac arrest, anterior or lateral location of infarction, systolic blood pressure, white blood cell count, serum creatinine, and congestive heart failure. The ROC area were 0.77 in both initial and validation cohorts. This model was found to perform "...as well as more complex models in comparing hospital outcomes for acute myocardial infarction".

**Killip class is an indicator of severity, where class 4 is the most severe condition, such as cardiogenic shock, and class 1 indicating "no clinical signs of cardiac decompensation".*

An algorithm based on administrative data was developed in Ontario (N=52,616) and validated using data from Manitoba (N=4,386) and California (N=112,234) (Tu et al., 1999 and 2000). Initially, 43 variables were evaluated for significance (Table 2.1). Eleven variables comprised the final model (Table 2.2). The overall model performance was very similar in derivation (ROC area 0.78) and validation (ROC area 0.77) cohorts. The predictive power of an algorithm controlling for comorbidities using administrative data compared very well with that of algorithms derived from clinical information.

Finally, Humphries et al (2000) went back two years before the index hospitalization to compare comorbidities generated from administrative data with comorbidities derived from clinical data. Although comorbidities were slightly underestimated in administrative data, predictions based on them were very similar to those based on chart reviews.

CHAPTER THREE – METHODS

3.1 Data Sources

This research project was based on the data set created by abstracting information from the hospital charts of a relatively large number of patients. The study contains actual clinical information together with the standard administrative data. The collecting of data was performed within the Manitoba Health Reform Impact Study (MHRIS). All index cases of AMI within the province between 1 October 1991 and 30 September 1992 were identified using Manitoba Health hospital separation abstracts. All discharge abstracts which contained code "*410.xx*" according to the *International Classification of Diseases – 9th Revision – Clinical Modifications* (ICD-9-CM) in any of the 16 diagnostic fields were identified (Appendix 1). All records with non-Manitoba postal and municipal codes were excluded from the hospital discharge abstracts, as were all Manitoba hospital discharge records with a hospital number indicating out-of-province location. A team of experienced abstractors screened the charts using the "Abstractors Field Manual: Acute Myocardial Infarction (AMI)", prepared especially for this project (Appendix 2). Additional reference information (such as MHRIS Comprehensive Manual: Acute Myocardial Infarction (AMI)" and MHRIS coding manual were also available (Appendix 3).

Long-term follow-up on 1-year and 5-year mortality was provided by the Manitoba Health Registry. In addition, the postal code of each record was used to assign income quintile (based on average household income for enumeration area) with the income quintile macro developed at MCHPE to allow for comparisons among different socioeconomic groups.

3.2 Income Quintiles

Income quintile rankings were based upon the 1991 census of the Manitoba population. In Winnipeg the average neighborhood household income value was assigned by linkage of the postal code to the census enumeration area via the Statistics Canada postal code conversion file. Based on the mean household income, the enumeration areas were ranked from the poorest to the wealthiest and then grouped into five population quintiles, each quintile containing 20 percent of the city's population. Each quintile contained approximately 130,000 inhabitants. Each resident was linked to an Enumeration Area (EA) by the residential postal code; thus, for each resident a quintile income rank was assigned, with Q1 being the poorest (Roos and Mustard, 1997), (Table 3.1). There were 1,749 EAs in Manitoba, 781 of them in Winnipeg.

Since an Enumeration Area identifies a block face consisting of approximately 600 people, analysis describing individuals' socioeconomic characteristics based on neighborhood income levels can be sensitive to the population's socioeconomic characteristics (Mustard and Frohlich, 1995).

Table 3.1 Minimum and Maximum Value of Average Income by Quintile Rank in Manitoba's Urban Settings ($ CDN)

Quintile	Minimum	Maximum	Mean
Q1	8,767	23,740	18,946
Q2	23,740	28,393	26,037
Q3	28,393	34,150	31,381
Q4	34,150	43,048	38,373
Q5	43,048	126,512	54,967

3.3 Definition of AMI

> ***Acute Myocardial Infarction (AMI) - is an ischemic myocardial necrosis usually resulting from abrupt reduction in coronary blood flow to a segment of myocardium***
>
> (The Merck Manual, 16th edition)

"Acute myocardial infarction occurs during the period when circulation to a region of the heart is obstructed, usually by a thrombus or embolus, and necrosis is occurring: (Dorland's Medical Dictionary, 28th Edition, 1994). Standard criteria for AMI diagnosis include clinical symptoms, electrocardiographic abnormalities, and enzymatic evidence of myocardial injury. For a case to qualify as an acute myocardial infarction any two of the following three groups of criteria need to be present.

Symptoms compatible with AMI include chest pain, angina, unstable angina, neck and arm pain, pallor, nausea, abdominal pain, dyspnea, dizziness, etc.

Electrocardiogram (EKG) abnormalities may include Q-wave, ST segment, and T wave alterations.

Laboratory test results indicative of AMI include creatine kinase (CK) and creatine kinase-MB band (CK-MB) tests. Creatine kinase is an enzyme that reflects tissue catabolism, and its increase above normal serum levels indicates cell injury. CK may be separated into 3 isoenzymes: CK-BB (primarily in brain tissue), CK-MM (in skeletal muscle), and CK-MB (in cardiac muscle). CK levels suggestive of AMI are:

CK – twofold elevation above normal (400 U/L and over)

CK-MB – greater than 5% of total CK.

For this project, a case of AMI was defined as an index event if it fulfilled the following four criteria:

1. ICD-9-CM code of 410.xx was the most responsible (primary) diagnosis (Appendix 6);
2. AMI episode was "fresh", i.e. less then 8 weeks old (according to ICD-9-CM);
3. Discharge dates occurred in the specified period of October 1, 1991 – September 30, 1992;
4. Length of hospital stay at least 5 days for patients discharged alive, in order to eliminate records coded as AMI that were unlikely to actually be AMIs.

3.4 Population

This study includes all hospital discharges with acute myocardial infarction occurring during a pre-selected one-year period. Age of the population in the study was specified at 45-99 years. Overall, 2,223 Manitoba residents hospitalized for AMI had hospital charts eligible for data abstracting. Chart review excluded 345 cases due to the following:

- "not fresh MI" (156);
- no ICD-9-CM diagnosis of 410.xx present on the abstract (105);
- index hospitalization out-of-province (22).;
- stripped charts (21);
- other then AMI diagnosis [hip fractures] (15);
- hospitalization occurred outside of specified time period Oct.1/91-Sept.30/92 (13);
- cerebrovascular accident occurred within 48 hours prior to admission (10);

- other (3);

resulting in the remaining cohort size of 1,878.

Additional 61 cases had either unassignable postal code (22), or postal codes of Personal Care homes (PCH) or other institutions (39), unsuited for linking to the census income data.

This project focussed on Winnipeg residents, since income quintiles are better defined in urban areas and a smaller number of hospitals are involved. This focus also minimized the possibility of bias resulting from the high variability in physician diagnostic tendencies and treatment characteristics across different urban and rural settings. Therefore, additional 758 cases of non-Winnipeg residents were excluded. A final cohort of 1,059 Winnipeg residents was selected (Table 3.2).

For evaluation of long-term outcomes, such as 1-year and 5-year mortality, necessary adjustment was made to reflect cases lost to follow-up. Five and 24 records respectively were eliminated, making the number for the 1-year cohort 1,054 and that for the 5-year cohort 1,035.

3.5 Key descriptive and outcome variables

The final AMI data set contained a wealth of clinical and utilization information (more than four hundred variables) used to study the relationship among covariates and outcomes of acute myocardial infarction and to assess the role of socioeconomic status (Appendix 4). The main variables included the following:

- Independent variable: Income quintile (average neighborhood income level);

- **Dependent variables:** All health outcomes – mortality (in-hospital, 30-day, 1-year and 5-year), complications, length of hospitalization, duration of stay in intensive / coronary care unit (ICU/CCU), etc.

  All health care utilization outcomes relevant to AMI care: rates of pharmacological therapies, diagnostic procedures, laboratory testing, and revascularization procedures performed during index hospitalization.

- **Covariables:** Socio-demographic characteristics (age, gender, marital status), symptoms and the diagnosis on admission, basic physiological parameters on admission (heart rate, blood pressure, respiration, etc.), pre-existing comorbidities and accompanying conditions, location of AMI, etc.

3.6 Statistical analysis

All statistical analyses were conducted using SAS version 6.12.

Descriptive Statistics

A number of techniques were employed to describe the subject's demographics, clinical characteristics, medical interventions, and outcomes of AMI,. Percentages were calculated for each categorical variable. Means and standard deviations were determined for continuous variables, such as age and length of hospitalization.

Mortality and rates of AMI in different income quintiles were expressed as percentages, because a large number of initial cases was excluded for various reasons (as discussed above in p.3.4), making the cohort under investigation no longer representative of a true incidence of AMI in the population and hindering the deriving of a common denominator. Therefore, standardization of rates would not bear much meaning.

Statistical Testing

The Chi-square test of association was used to examine differences in categorical variables: demographic factors, clinical variables, and utilization rates for various diagnostic procedures, laboratory tests, and medical therapies across income quintiles. The rates of health care utilization were compared across income quintiles.

The differences between continuous variables [age, some physiological measurements (blood pressure, heart rate, etc.), and length of hospitalization, etc.] were assessed using the Student's t-test or the analysis of variance.

Statistical significance was considered when a p-value fell below 5% in all analyses. Stratification with regard to the co-morbidity score was employed for the analysis of clinical and resource utilization variables.

Modeling

Logistic regression analysis was used to assess whether and how patients' various demographic, clinical and socioeconomic characteristics were associated with the outcome variables such as mortality or treatment. This method is routinely used in analyzing categorical (presence or absence) outcomes, which can be coded as *death = 1, no death = 0;* or, in case of health care utilization, *procedure performed = 1, not performed = 0*, and so on. Logistic Regression does not need to assume normal distribution of variables, constant variance, or use of continuous variables in the model. Similar to multiple regression, the logistic regression allows using multiple explanatory variables in the model. The unique effect of each explanatory variable on the outcome variable can be determined, while controlling for appropriate demographic and clinical

covariates. Odds ratios and 95% confidence intervals for each model variable were analyzed. The overall model performance was evaluated by the receiver-operator characteristic (ROC) curve.

The initial logistic regression models for administrative data were based on work by Tu and his collaborators using a relatively large cohort of 52,616 patients having an acute myocardial infarction in Ontario between 1994 and 1997 (Tu et al., 1999). The Ontario 11-variable statistical models predicted 30-day and 1-year mortality very well with an area under the receiver operating characteristic (ROC) curve of 0.78 for 30-day mortality and 0.79 for 1-year mortality (Hanley and McNeil, 1982), (Tu et al, forthcoming). Two independent validation data sets from Manitoba (4,836 AMI patients) and California (112,234 AMI patients) showed identical ROC areas for 30-day and 1-year mortality, 0.77 and 0.78 respectively. Therefore, using the Ontario AMI mortality prediction rules for the analysis of a smaller Winnipeg data set seemed appropriate, even though some of predictors of mortality were not statistically significant with the lower number of cases in the Winnipeg sample. The analyses reported here compared areas under the ROC curves among the various data sets and models. The calibration of each model was assessed by comparing the mean observed and predicted 30-day AMI mortality rates among patients sorted into deciles of ascending risk. The number of outcome events per independent variable (EPV) was kept at least 10 (Concato and Feinstein, 1997).

Table 3.2 Exclusion criteria for AMI cohort

Exclusion criteria	**Removed**	**Remains**
◆ Initial Number		**2,223**
◆ Not fresh AMI	156	2,067
(includes persons admitted for non-cardiac reasons, admitted for non-cardiac or cardiac tests or procedures who developed an AMI as a consequence of the procedure)		
- MI occurred in the hospital	82	
- Elective admission for CABG or other surgery	72	
- MI occurred as a complication	2	
◆ No *ICD-9-CM* diagnosis of *410.xx* present on the abstract	105	1,962
◆ Index hospitalization was out-of-province or at ineligible facility	22	1,940
◆ Stripped charts	21	1,919
◆ Other than AMI diagnosis (hip fractures)	15	1,904
◆ Admission date outside of specified period of October 1, 1991 – September 30, 1992	13	1,891
◆ CVA occurred within 48 hours prior to admission	10	1,881
◆ Hospital could not locate the chart	2	1,879
◆ Duplicate ID number	1	**1,878**
◆ Postal codes of Personal Care Homes (PCH) or other institutions	39	1,839
◆ Unassignable postal codes	22	1,817
◆ Non-Winnipeg residents	758	**1,059**

CHAPTER FOUR RESULTS

4.1 Socio-Demographic Characteristics of AMI Patients

Income Level of AMI Patients

Socioeconomic status is an important factor contributing to the distribution of diseases among the population. In the AMI cohort under investigation, the number of patients declined along with the increase in the average income. The proportion of patients who belonged to the quintile with the lowest income (26.5%) was significantly greater than the proportion of patients who belonged to the quintile with the highest income (16.8%), [x^2 25.6, p 0.001]. The ratio of Q1 (rate in the neighborhood with the lowest income) to Q5 (rate in the neighborhood with the highest income) was 1.58 (Table 4.1 and Figure 4.1), indicating that with the decrease in income the risk of an episode of acute myocardial infarction increases. However, that should be interpreted with caution. The study was concerned only with the cases of AMI, which were hospitalized, and did not have information on AMI cases without making it to the hospital. Also, as described in chapter 3 (part 3.4), the cohort was selected from the total one-year sample of AMI cases that satisfied certain criteria and, as the result, is not as diverse as the true population sample would have been. Therefore, the described cohort is not a representative sample of general AMI population.

Figure 4.1 Distribution of AMI Patients by Average Neighborhood Income

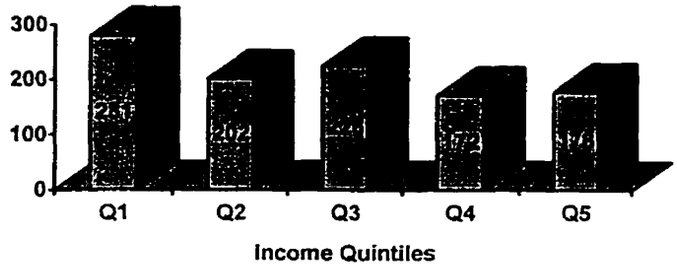


Table 4.1 Demographic Characteristics of AMI patients

Characteristic	Prevalence		Q1	Q2	Q3	Q4	Q5	
	N=1,059		n=281	n=202	n=226	n=172	n=178	P-value
	%	N						
Income Distribution		1059	26.5	19.1	21.3	16.2	16.8	0.001
Age 50-64 years	26.3	278	18.5	20.3	31.4	24.4	40.5	0.001
65-74 years	33.0	349	31.0	38.1	31.9	36.6	28.1	0.197
75+ years	34.2	362	44.5	36.6	31.0	29.7	23.6	0.001
Mean age		69±10.7	72±11.4	71±11.1	69±11.1	68±11.7	65±11.3	0.001
Female sex	43.1	456	53.7	41.1	42.5	40.1	32.0	0.001
Married/ common law	56.8	601	39.2	52.5	59.3	66.3	77.0	0.001
Smoking*	31.2	331	32.7	28.2	32.7	34.8	27.5	0.530

* The data on smoking habits may not be reliable, since the only sources of information were nursing notes in the charts at the time of an index hospitalization.

The Age of AMI Patients

The age of AMI patients in this study was limited by 45 - 99 years, with the mean age being 69.1 ± 11.5 years. A significantly higher proportion of older patients (age 65 and older) – 51% - were residents of the two lower income neighborhoods (Q1-Q2), while only 34.5% of younger patients (45-64 yr.) were from the neighborhoods with that level of income (x^2 26.6, p 0.001). In the highest income group (Q5) the opposite was true: while 24.7% of younger patients comprised that group, only 12.9% of older patients were from the areas with the highest level of income (Figure 4.2).

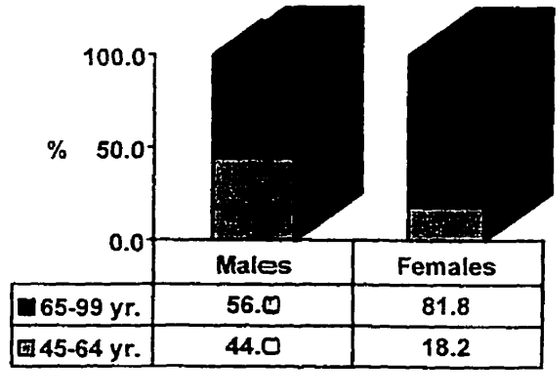
Figure 4.2 The Proportion of Seniors and Younger Adults Across Income Quintiles

	Q1	Q2	Q3	Q4	Q5
45-64 yr.	19.8	14.7	24.1	16.7	24.7
65-99 yr.	29.8	21.2	20.0	16.0	12.9

This is vastly related to the obvious gender difference in the age of AMI patients. Women were having myocardial infarctions much later in their life course than men: only 18.2% of women (83 out of 456) were of pre-retirement age (45 - 64 yr.), while 44% of men (265 out of 603) were of the same age (x^2 95.6, p 0.001), Figure 4.3.

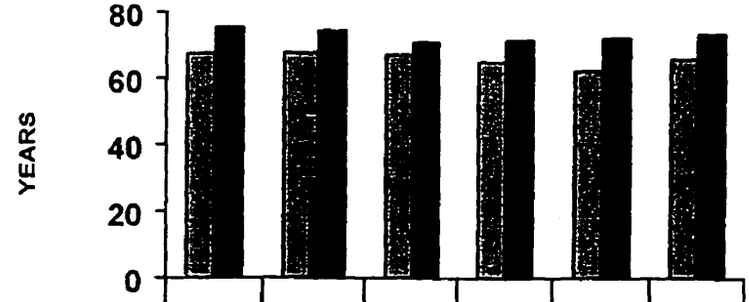
With the increase in income the age of patients decreases in both males and females and in each income quintile (F 16.2, P 0.000). The greatest difference in age at which individuals had AMI -approximately 10 years- was between men and women who belonged to the Q5. Only in the intermediate income quintile the age gap between males and females lessened, with females being only three years older (Figure 4.4).

Figure 4.3 Age Groups of Men and Women with AMI



	Males	Females
65-99 yr.	56.0	81.8
45-64 yr.	44.0	18.2

Figure 4.4 Age of Men and Women from Different Income Quintiles



	Q1	Q2	Q3	Q4	Q5	Mean
Males	67.4	67.8	67.1	65.0	62.4	65.9
Females	75.3	74.5	70.9	71.6	72.2	73.4

Gender

Contrary to the common belief that acute myocardial infarction is a "male problem", in the sample of AMI patients under investigation, the difference between the number of men and women was only 14%. The overall per cent of male patients was 57% (603 out of 1,059), and the female-to-male ratio of the sample was close to 0.75.

The distribution of income between men and women with AMI was very different. Male patients were equally present in each income quintile, while female patients were most highly represented in lower income quintiles (Figure 4.5). One third of all female AMI patients (33.1%) belonged to the lowest income quintile (Q1), while only one-eighth of them (12.5%) were from the highest income group (Q5). The Q1/Q5 ratio was approximately 1.1 for men, but was 2.65 for women (Figure 4.5).

Since significantly more females with AMI were of a lower socioeconomic status, with the increase in the average neighborhood income the proportions of male patients within each quintile also significantly increases (p 0.001), Table 3. Only in the lowest income quintile (Q1) did women outnumber men, while in every other quintile the number of man was greater. The female-to-male ratio in the lowest income quintile (Q1) was 1.16, while in the highest income quintile (Q5) it was 0.47 (Figure 4.6).

Marital Status

Fifty seven per cent of patients were married or living common-law. Notably, the percentage of married men – 73% - was twice the percent of married women (35.5%). The proportion of married man did not change much with the age, in contrast with the sharp decline of the proportion of women who are married. This can be largely attributed

to the longer overall life span of women, who outlive their male partners (Figure 4.7). Patients from the more affluent neighborhoods tend to be married: 77.0% in Q5 vs. 39.2% in Q1.

Figure 4.5 Percentage of Males and Females with Different Level of Income

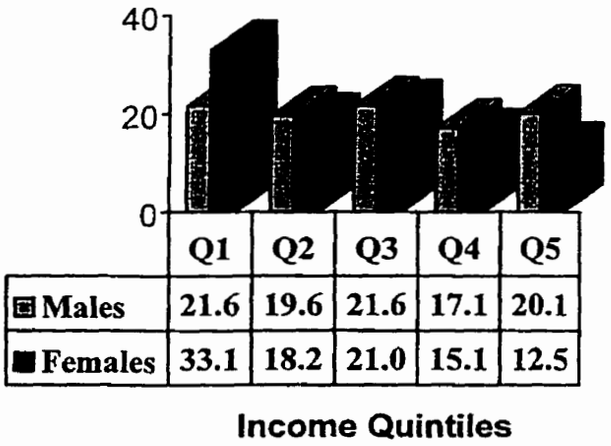


Figure 4.6 Gender Distribution in Different Income Quintiles

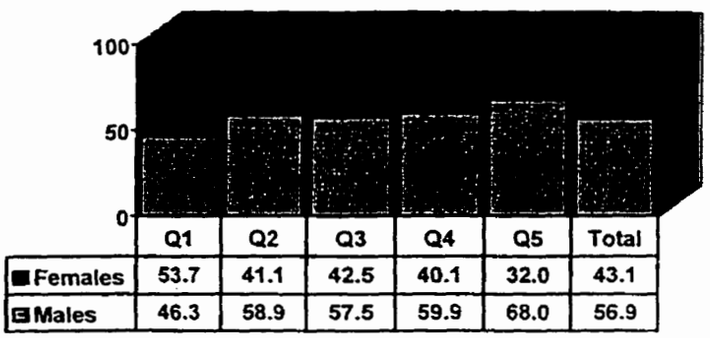
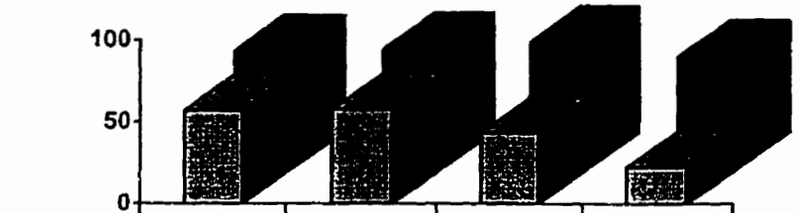


Figure 4.7 Marital Status of Men and Women by Age Category

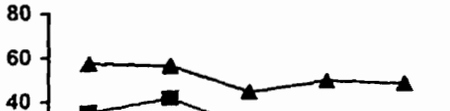


	45-49 yr.	50-64 yr.	65-74 yr.	75+ yr.
Women	57.1	57.9	43.2	22.5
Men	71.4	72.7	76.8	68.1

Association between mortality and socioeconomic status was modified by marital status. Significant differences in mortality between income quintiles exist only for married patients, with much more favorable outcome among high-income patients (p 0.001). Mortality between income quintiles, however, did not differ among unmarried patients (Figure 4.8).

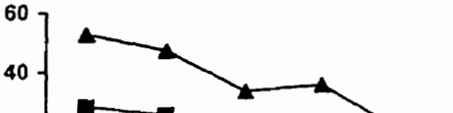
Figure 4.8 Association between SES and Mortality by Marital Status

Unmarried Patients



	Q1	Q2	Q3	Q4	Q5
30-day	20.5	25.0	17.4	22.4	17.1
1-year	35.1	41.7	28.3	31.0	29.3
5-year	57.3	56.3	44.6	50.0	48.8

Married Patients



	Q1	Q2	Q3	Q4	Q5
30-day	21.8	20.8	9.0	10.5	4.4
1-year	28.2	25.5	17.9	15.8	9.5
5-year	52.7	47.2	33.6	36.0	20.4

4.2 Baseline Clinical Characteristics of AMI patients

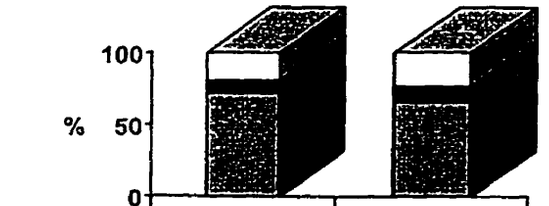
The Timeliness of Patients' Arrival at the Hospital

Information on the time interval between the onset of symptoms and the arrival at the hospital was available for 941 out of 1,059 Winnipeg AMI patients (88.9%). The average time varied across income quintiles; patients from more affluent neighborhoods were arriving at the hospital in a much more timely fashion than their low-income counterparts. On average, patients who belonged to the Q1 were hospitalized within 24.8 hours after the onset of symptoms of an acute myocardial infarction, while patients who belonged to Q5 were hospitalized within only 8.6 hours of onset of symptoms (P 0.009), (Table 4.2). Timeliness of arrival at the hospital after the beginning of AMI symptoms also depended on the gender of patients. A higher proportion of male AMI patients - 72.2%- arrived at the hospital in the first six hours, compared to 65.5% of female AMI patients (P 0.030), (Figure 4.9). Both male and female patients in the higher income groups, however, were arriving at the hospital sooner after their symptoms started then were lower income patients (Figure 4.10).

Table 4.2 Time Interval between the Onset of AMI Symptoms and Arrival at the Hospital

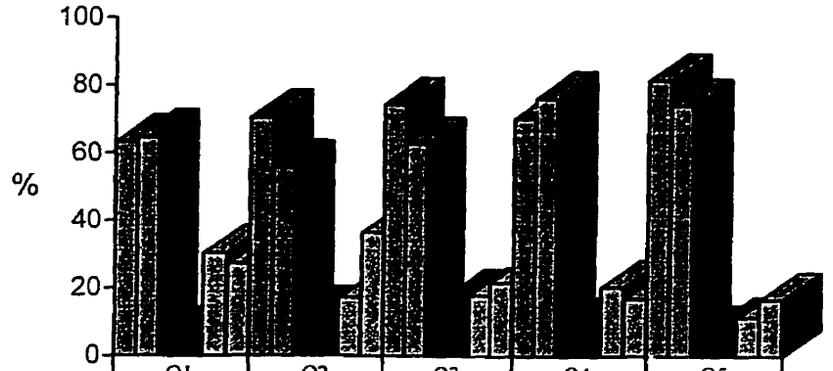
Hours	*Q1* *n=234* *% (N)*	*Q2* *n=182* *% (N)*	*Q3* *n=200* *% (N)*	*Q4* *n=157* *% (N)*	*Q5* *n=168* *% (N)*	*P*
1-6 hrs.	64.1 (150)	64.3 (117)	72.9 (139)	72.6 (114)	79.2 (133)	
7-12 hrs.	7.3 (17)	12.1 (22)	11.0 (22)	8.3 (13)	7.7 (13)	
13+ hrs.	28.6 (67)	23.6 (43)	19.5 (39)	19.1 (30)	13.1 (22)	
Mean (hrs.)	24.8±3.7	15.5±2.9	13.7±2.8	10.4±5.7	8.6±2.0	0.009

Figure 4.9 Timeliness of Patient's Arrival at the Hospital by Gender



	Males	Females
13+ hrs.	19.4	24.0
7-12 hrs.	8.4	10.4
1-6 hrs.	72.2	65.5

Figure 4.10 Time of Arrival at the Hospital by Patient's Gender and Income



	Q1	Q2	Q3	Q4	Q5
Male 1-6	63.4	70.5	74.4	70.4	81.6
Female 1-6	64.8	55.8	62.7	76.3	74.1
Male 7-12	6.3	12.4	7.7	9.2	7.0
Female 7-12	8.2	11.7	17.7	6.8	9.3
Male 13+	30.3	17.1	17.9	20.4	11.4
Female 13+	27.0	36.4	21.7	16.9	16.7

Three most common locations of infarction were inferior (excluding inferiolateral and inferioposterior walls), followed by anterior (excluding anteriolateral), and subendocardial (Table 4.3). Unspecified site of AMI appears to be recorded more often in the discharge abstracts of low-income patients (p 0.015).

Table 4.3 Location of Infarction

ICD-9 Code	AMI location (%)	Q1 n=281	Q2 n=202	Q3 n=226	Q4 n=172	Q5 n=178
410	Anteriolateral wall	4.3	5.9	5.3	11.6	6.2
410.1	Other anterior	19.9	24.8	19.0	20.9	22.5
410.2	Inferiolateral wall	3.6	4.0	2.7	4.7	2.8
410.3	Inferioposterior wall	0.7	1.5	0.9	0.6	0.0
410.4	Other inferior	28.5	27.7	26.1	28.5	37.6
410.5	Other lateral	1.8	2.5	1.8	4.1	3.4
410.6	True posterior infarction	1.1	2.5	2.8	0.0	1.1
410.7	Subendocardial infarction	19.2	17.3	26.6	20.4	16.3
410.8	Other specified sites	3.2	0.5	0.9	0.6	1.7
410.9	Unspecified sites	17.8	13.4	15.0	8.7	8.4

Note: data from the administrative database

Comorbidity and Risk Factors

Table 4.4 depicts baseline clinical characteristics of patients in different income quintiles. The most frequent diagnosis on admission - "AMI / rule out AMI"- was recorded in 55% of all cases. According to the chart reviews, the most frequently experienced symptom -- chest pain -- occurred much more often among the wealthier patients (x^2 29.2, p 0.001). This may be a result of a better ability to communicate symptoms by more affluent patients. Conversely, a decreased level of consciousness was

evident more often among low-income patients (x^2 10.22, p 0.023). They also had higher values of heart rate (F 5.83, 0.040) and respiration (F 7.3, p 0.005) per minute. Similarly, the lower income patients had significantly higher rates of roentgenographic evidence of congestive heart failure (p 0.002), cardiomegaly (p 0.001), and pulmonary edema (p 0.042). Review of medical histories revealed that compared to the more affluent patients, the low-income AMI patients have had much increased rates of congestive heart failure (x^2 11.1, p 0.006), cerebrovascular disease / stroke (x^2 10.5, p 0.013), and chronic pulmonary diseases (x^2 19.7, p 0.001), (Table 4.4). Such findings are similar to those found by Alter et al. in Ontario (1999): congestive heart failure, stroke, and chronic pulmonary diseases were the only comorbidities exhibiting statistically significant differences across income quintiles.

Other base-line clinical characteristics of AMI patients with different levels of income were largely similar, which resulted in similar mean Charlson comorbidity scores (Appendix 5). Sixty-one percent (646 out of 1,059) of patients had no significant comorbidity recorded on their index hospitalization and their Charlson score was zero. One quarter of all patients (282) has had a comorbidity score of 1; followed by 7.8% of patients with the score of 2 and 3.3% of patients with the Charlson comorbidity score of 3. Scores 4 to 7 have had only 1.2% of patients. The Charlson comorbidity score depended on age: a higher proportion of older patients (43%) has had comorbidity score 1 and greater compared to 30% of younger patients (p 0.021), (Figure 4.11). Three per cent of patients were terminally ill (Table 4.4). According to the chart abstracts, approximately one third of AMI patients were current or ex-smokers, and this figure being similar across the income quintiles (Table 4.1).

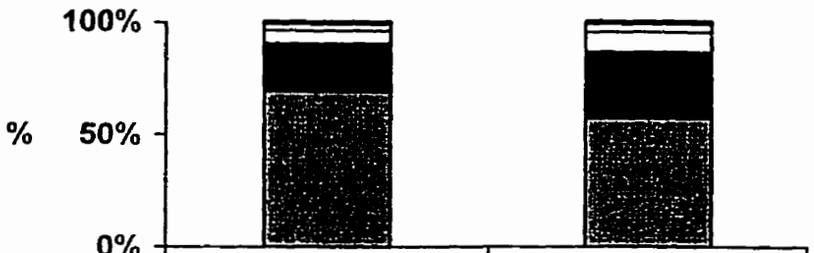
Table 4.4. Base-Line Clinical Characteristics of Patients According to Income Quintile

Condition (%)	Q1	Q2	Q3	Q4	Q5	
	n=281	n=202	n=226	n=172	n=178	P-value
Time from symptoms onset to hospitalization (hrs.)	24.8	15.5	13.7	10.4	8.6	0.009
Diagnosis Recorded on Admission						
AMI / rule out AMI	54.8	54.0	59.7	54.7	51.1	0.632
Cardiac arrest	10.3	7.4	10.6	10.5	7.3	0.608
Congestive heart failure	15.3	9.4	11.1	12.2	6.7	0.055
COPD / Asthma	2.1	1.9	3.1	1.5	0.5	n/a
Myocardial Insufficiency	1.8	1.6	1.4	1.0	1.0	n/a
Pneumonia	2.9	2.5	0.9	0.6	2.8	0.412
Other	8.4	8.4	7.3	3.5	7.1	0.312
Symptoms Recorded on Admission						
Abdominal pain	4.6	3.0	2.2	1.7	2.8	0.136
Chest pain	52.3	61.4	62.0	70.4	70.8	0.001
Decreased level of consciousness	7.8	8.4	4.0	4.1	2.3	0.023
Dyspnea	25.3	23.3	27.9	29.1	22.5	0.945
Nausea	12.1	10.4	11.2	16.9	12.9	0.310
Pulmonary edema	8.9	9.7	8.0	6.7	7.1	0.832
Rales	17.2	15.0	13.9	11.4	14.7	0.600
Shock	5.7	5.5	4.9	4.1	1.7	0.315
3-rd heart sound	3.4	4.1	3.8	3.0	2.5	0.862
Vital Signs Recorded during Admission to the Hospital						
Mean Heart Rate (per min)	87±26	86±25	85±25	82±24	80±23	0.040
Mean Systolic Blood Pressure (mm Hg)	144±35	144±34	141±32	142±33	143±29	0.891
Mean Diastolic Blood Pressure (mm Hg)	85±22	86±19	84±20	85±20	86±19	0.920
Mean Respiration (per min)	23.1±7	22.4±7	22.3±6	21.7±6	20.8±6	0.005
Mean Temperature (C^0)	36.4±1.0	36.4±0.8	36.7±0.8	36.3±0.8	36.3±0.7	0.830
Chest X-ray Recorded during Hospitalization						
Cardiomegaly	29.9	32.2	32.3	24.4	15.7	0.001
Congestive heart failure	19.6	20.8	16.4	14.0	7.9	0.002
Pulmonary edema	15.0	15.4	13.7	13.4	7.3	0.042

Condition (%)	Q1	Q2	Q3	Q4	Q5	
	n=281	n=202	n=226	n=172	n=178	P-value
EKG Abnormalities Recorded during Hospitalization						
Atrial Fibrillation	6.8	5.0	5.2	7.0	5.1	0.837
Bundle Brunch Block	12.0	9.7	10.1	5.5	6.6	0.104
Heart Block	5.7	5.6	5.6	9.5	5.1	0.452
Ischemic Changes	4.7	3.1	3.8	4.0	3.6	0.907
New R/Q-waves	14.4	12.2	12.5	11.9	14.7	0.880
ST elevated / depressed	48.8	47.8	49.1	58.7	64.0	0.001
Other ST changes	56.2	45.5	45.1	43.6	47.2	0.037
Supraventricular Tachycardia	1.0	0.6	0.7	0.5	0.5	N/A
T-wave change	26.6	26.9	27.2	32.8	32.0	0.482
Ventricular Fibrillation	0.5	1.9	0.7	2.5	1.0	N/A
Ventricular Tachycardia	3.7	3.1	2.4	2.5	4.1	0.814
Comorbidity from Medical History						
Previous myocardial infarction	36.7	30.7	27.9	27.9	28.7	0.166
Previous AMI within 8 weeks	4.5	12.2	7.6	8.8	1.9	0.001
Angina / chest pain	29.2	26.2	26.6	31.4	27.5	0.785
Cancer	6.8	7.9	5.3	5.8	6.7	0.848
Cerebro-vascular accident / stroke	14.6	10.4	6.2	7.0	8.4	0.013
Chronic pulmonary diseases	19.2	10.4	12.8	12.2	6.2	0.001
Chronic renal failure	3.6	5.0	1.3	4.7	2.3	0.189
Congestive heart failure	27.8	19.8	19.5	21.5	13.5	0.006
Coronary angioplasty	1.1	1.0	2.7	1.7	2.8	0.466
Coronary artery bypass surgery	4.6	5.5	7.1	4.1	9.6	0.160
Dementia/ Alzheimer's disease	1.6	2.2	1.7	2.5	2.0	0.957
Diabetes mellitus	26.0	22.8	23.0	21.4	15.2	0.051
Hypertension	43.4	43.6	34.1	39.5	37.6	0.202
Ischemic heart disease / coronary artery disease	33.5	28.2	27.7	27.9	24.2	0.202
Peripheral Vascular Disease	9.7	10.3	8.7	8.0	8.6	0.939
Permanent pacemaker	2.5	1.0	2.2	0.0	0.6	0.135
Terminal illness / DNR code	4.2	3.8	2.4	3.0	2.0	0.611

Note: data from the chart review

Figure 4.11 Comorbidity of AMI Patients in Different Age Categories.



	45-64 yr.	65+ yr.
4 to 7	1.1	1.3
3	2.9	3.5
2	5.8	8.9
1	21.0	29.4
0	69.2	57.0

4.3 Clinical Outcomes

Length of hospitalization

The overall length of hospitalization for AMI was between 11 and 13 days. A total of 87% of patients have been treated in a Coronary Care Unit (CCU) for the average time of 3-4 days (Table 4.5). Older patients (65 years of age and older) stayed in the hospital for approximately 3 days longer than younger patients (13.6±16.3 and 10.8±9.5 respectively). However, the duration of the treatment in CCU/ICU was the same regardless of the age (3-4 days). Women were hospitalized at the average 3.5 days longer (p 0.0002, t=3.73) than men (14.6±17.1 vs. 11.1±11.8 respectively). Similar pattern was described by Johansen et al. (1998), for the overall Canadian population. They found that "...female AMI patients tend to have longer hospital stays than male patients" in 1993/94. They felt this to reflect more severe disease and complications among women.

Table 4.5 Duration of Hospitalization across Income Quintiles

	Q1 *n=281*	Q2 *n=202*	Q3 *n=226*	Q4 *n=172*	Q5 *n=178*	P
Treated in CCU (%)	85.0	84.2	86.7	90.7	90.5	
Mean LOS in the CCU/ICU	3.5±3.1	3.5±3.5	4.1±4.2	3.6±2.7	4.2±2.8	0.173
Overall LOS	13.3±15.5	13.6±19.6	11.7±9.7	11.4±7.7	13.1±15.7	0.197

Treatment of AMI during the hospital stay

The analysis of treatment for AMI included evaluation of the use of standard drug therapy (as recommend by ACA/CMA), i.e. aspirin, thrombolytic therapy, heparin, and beta-blockers; utilization of revascularization procedures (coronary angioplasty and coronary artery bypass graft); as well as the use of diagnostic testing (both invasive and non-invasive). Consistent with other studies, the utilization of standard treatment and diagnostic techniques varied markedly among patients with different levels of income. Thus, high–income patients were treated twice as often with the thrombolytic therapy and received heparin fifty percent as often (p 0.001). Low-income patients, conversely, required more aggressive treatment interventions, such as intubation (p 0.010), Table 4.6.

Invasive revascularization procedures, such as coronary angioplasty and coronary artery bypass surgery, were utilized with similar frequency for patients from all income groups. However, this should not undermine the fact that these procedures are usually performed after the discharge from the hospital, and, therefore, do not represent the true patterns, which are known to have a large SES gradient. Two diagnostic procedures were performed more often among patients who belonged to the higher income groups. Stress

exercise testing was performed twice as often among patient from the highest income quintile (20.2%) than among patients belonging to the lowest income quintile (11.0%), (p 0.022). Similarly, the MUGA scan (multiple gate acquisition scan) was also performed more often among the higher income patients (p 0.011), Table 4.6.

Table 4.6. Patterns of Treatment across Income Quintiles

Treatment (%)	Q1 n=281	Q2 n=202	Q3 n=226	Q4 n=172	Q5 n=178	P
Medications:						
Aspirin	71.2	71.8	75.5	75.6	80.3	0.208
Beta-blockers	23.1	27.2	30.5	33.7	39.9	0.002
Heparin	48.8	59.4	57.1	64.0	70.2	0.001
Thrombolytic therapy $\leq$ 24 hr.	21.4	29.7	29.7	39.0	45.5	0.001
Use of Medical and Surgical Services:						
ICU / CCU admission	85.1	84.2	86.7	90.7	90.5	0.173
CPR administered $\leq$ 24 hr.	6.4	8.9	7.5	9.3	2.8	0.311
Intubation	13.9	14.4	11.5	7.6	5.1	0.010
Coronary angioplasty	5.0	4.5	4.0	5.8	3.9	0.902
Coronary artery bypass surgery	2.9	3.0	3.1	2.9	2.8	1.000
Use of Diagnostic Testing:						
Angiography	21.7	19.8	21.2	23.8	28.1	0.345
Stress exercise testing	11.0	11.9	17.7	18.6	20.2	0.022
Echocardiography	41.6	42.6	35.0	42.4	39.3	0.446
Thallium scan	2.9	3.5	3.1	2.9	3.4	0.995
MUGA scan	9.3	9.4	7.1	6.4	16.3	0.011
Other tests 17.1	19.8	15.5	15.7	19.1	0.715	

Note: data from the chart review

Success in the treatment of acute myocardial infarctions depends largely on how soon the treatment is started after the onset of symptoms. Some therapies – thrombolytic therapy with ACE-inhibitors in particular -- are highly effective in the first six hours, less so in the first twelve. Therefore, the proportion of patients who receive thrombolytic therapy depends on the timeliness of arrival at the hospital. Nevertheless, even after stratification by the time of the arrival at the hospital, a significant difference persists in the use of thrombolytic agents and heparin across the income quintiles among patients who arrived within 6 hours from the onset of symptoms (Table 4.7).

Table 4.7 Treatment across Income Quintiles

Stratified by the Time of the Arrival at the Hospital

Treatment	*Hours*	*Q1* *n=234*	*Q2* *n=182*	*Q3* *n=200*	*Q4* *n=157*	*Q5* *n=168*	*P*
Aspirin	1-6	75.3	78.6	76.3	73.7	82.0	0.556
	7-12	88.2	86.4	86.4	92.3	69.2	0.510
	13+	74.6	58.1	82.1	76.7	77.3	0.138
Thrombolytics within 24 hr	1-6	36.7	36.8	41.0	48.3	56.4	0.004
	7-12	11.8	18.2	18.2	23.1	15.4	0.948
	13+	3.0	23.3	5.1	20.0	13.6	N/A
Heparin	1-6	61.3	65.0	61.9	69.3	79.0	0.012
	7-12	58.8	68.2	63.6	61.5	76.9	0.865
	13+	37.3	46.5	38.5	50.0	31.8	0.593

After stratification according to the timeliness of one's arrival at the hospital, the rates of procedures were largely similar, with few exceptions. Among men who arrived early, thrombolytic therapy was still administered significantly more often among high-income patients, as was the MUGA scan among women. The numbers of patients and cases in the two groups with later arrivals (7-12 and 13+ hours) were too small to be compared.

Table 4.8 Treatment across Income Quintiles

Stratified by the Time of Arrival at the Hospital and by Gender

MALES	Treatment	Hours	Q1 n=234	Q2 n=182	Q3 n=200	Q4 n=157	Q5 n=168	P
	Aspirin	1-6	81.7	83.8	80.5	73.9	85.0	0.459
	Thrombolytics	1-6	36.6	37.8	48.3	52.2	57.0	0.039
	Heparin	1-6	70.4	70.3	66.7	73.9	81.7	0.208
		13+	38.2	44.4	38.1	55.0	30.8	0.657
	ICU/CCU	1-6	93.0	82.4	85.1	92.8	95.7	0.021
	CPR≤24 hr	1-6	7.0	8.1	4.6	7.3	3.2	n/a
	Intubation	1-6	9.9	10.8	9.2	4.4	3.2	0.299
	Angioplasty	1-6	7.0	6.8	3.5	5.8	4.3	n/a
	Coronary bypass	1-6	2.8	2.7	3.5	2.9	2.2	n/a
	Angiography	1-6	31.0	31.1	26.4	24.6	31.2	0.840
	Stress test	1-6	15.5	18.9	28.7	21.7	25.8	0.280
	Echocardiogram	1-6	46.5	52.7	39.1	43.5	36.6	0.257
		13+	55.9	33.3	28.6	45.0	46.2	0.304
	Thallium test	1-6	5.6	5.4	2.3	1.5	3.2	n/a
	MUGA scan	1-6	9.9	13.5	8.1	5.8	15.1	0.297

FEMALES	Treatment	Hours	Q1 *n=234*	Q2 *n=182*	Q3 *n=200*	Q4 *n=157*	Q5 *n=168*	P
	Aspirin	1-6	69.6	69.8	69.2	73.3	75.0	0.962
	Thrombolytics	1-6	36.7	34.7	28.9	42.2	55.0	0.123
	Heparin	1-6	53.2	55.8	53.9	62.2	72.5	0.288
		13+	36.4	48.0	38.9	40.0	33.3	0.906
	ICU/CCU	1-6	88.6	79.1	90.4	95.6	87.5	0.189
	CPR≤24 hr	1-6	7.6	4.7	15.4	11.1	5.0	n/a
	Intubation	1-6	15.2	9.3	15.4	8.9	12.5	0.770
	Angioplasty	1-6	6.3	2.3	3.9	6.7	7.5	n/a
	Coronary bypass	1-6	2.5	2.3	1.9	2.2		n/a
	Angiography	1-6	24.1	7.0	21.2	20.0	27.5	0.155
	Stress test	1-6	11.4	7.0	9.6	15.6	10.0	0.756
	Echocardiogram	1-6	41.8	20.9	30.8	42.2	40.0	0.134
		13+	39.4	44.0	33.3	30.0	55.6	0.767
	Thallium test	1-6	3.8		7.7	6.7	2.5	n/a
	MUGA scan	1-6	12.7	4.7	7.7	6.7	25.0	0.024

Comparison between two combined groups: quintiles Q1-Q2 (income level $ 8,767 – 28,393) versus quintiles Q4-Q5 (income level $ 34,150 and up) revealed that higher income male patients who arrived at the hospital early were more likely to be treated with thrombolytic therapy and in ICU/CCU, but less often required intubation. No differences were found in the treatment of female patients (Table 4.9).

Table 4.9 Treatment of Lower Income vs. Higher Income Men and Women

Treatment	Males Hours	Q1-Q2 n=217	Q4-Q5 n=212	P	Females Q1-Q2 n=199	Q4-Q5 n=113	P
Aspirin	1-6	82.8	80.2	0.573	69.7	77.4	0.201
	13+	76.9	78.8	0.841	60.3	73.7	0.292
Thrombolytics	1-6	37.2	54.9	0.002	36.1	48.2	0.081
Heparin	1-6	70.3	78.4	0.101	54.1	67.1	0.064
	7-12	65.0	76.5	0.453	63.2	55.6	n/a
	13+	40.4	45.5	0.640	41.4	36.8	0.725
ICU/CCU	1-6	87.6	94.1	0.039	85.2	91.8	0.152
CPR≤24 hr	1-6	7.6	4.9	0.334	6.6	8.2	0.644
	13+	75.0	78.8	0.682	86.2	84.2	n/a
Intubation	1-6	10.3	3.7	0.021	13.1	10.6	0.581
Angioplasty	1-6	6.9	4.9	0.471	4.9	7.1	0.723
Coronary bypass	1-6	2.8	2.5	0.805	3.3	1.2	n/a
Angiography	1-6	31.0	28.4	0.613	18.0	23.5	0.332
	13+	21.2	33.3	0.216	15.5	15.8	n/a
Stress test	1-6	17.2	24.1	0.145	9.8	12.9	0.482
	13+	17.3	27.3	0.273	8.6	5.5	n/a
Echocardiogram	1-6	49.7	39.5	0.074	34.4	41.2	0.325
	7-12	55.0	52.9	0.901	52.6	11.2	n/a
	13+	48.1	45.5	0.816	41.4	42.1	0.951
Thallium test	1-6	5.5	2.5	0.163	2.5	4.7	n/a
MUGA scan	1-6	11.7	11.1	0.867	9.8	15.3	0.232

Complications during Hospitalization

The most frequent complications during hospitalization for the AMI were angina (32.5%), followed by congestive heart failure (19.2%), and bleeding (8.3%). All types of complications were noted with a similar frequency across the income quintiles (Table 4.10).

Table 4.10. Complications during Hospital Stay with the Index AMI Admission

Condition (%)	**Q1**	**Q2**	**Q3**	**Q4**	**Q5**	
	n=281	n=202	n=226	n=172	n=178	P
Angina	36.3	34.2	25.7	32.6	33.2	0.268
Bleeding	10.0	8.4	5.8	5.2	11.8	0.205
Cardiac arrest	4.3	4.5	4.4	5.2	1.7	0.497
Cardiogenic shock	4.6	5.9	3.5	4.1	2.3	0.460
Cardiovascular disease / stroke	3.6	5.0	1.8	2.3	1.7	0.273
Congestive heart failure	21.0	19.8	19.5	16.3	18.0	0.752
Deep vein thrombosis	0.7	0.5	0.0	0.0	0.6	0.747
Pneumonia	3.6	3.5	3.1	1.7	4.5	0.805
Pulmonary emboli	0.4	1.0	1.3	0.0	0.3	0.699
Re-infarction	5.7	5.5	3.1	5.8	3.9	0.900
Other	23.8	27.2	21.7	15.7	18.5	0.060

Mortality and Socioeconomic Status

When the ultimate outcome, death versus survival after an episode of acute myocardial infarction, is compared across income quintiles, the dramatic difference become evident. Mortality was much higher among patients from the lowest income neighborhoods than among their intermediate and high-income counterparts. Both short-term and long-term figures showed an inverse relationship between the patient's income and mortality (Table 4.11 and Figure 4.12). The ratio of Q1 (mortality in the neighborhoods with the lowest income) to Q5 (mortality in the neighborhoods with the highest income) was 2.9 for 30-day mortality, 2.3 and 2.1 for 1-year and 5-year mortality respectively. Lower income patients have a markedly decreased overall chance to survive an AMI episode.

Table 4.11. AMI Mortality by Income Quintile

Mortality	**Q1**	**Q2**	**Q3**	**Q4**	**Q5**	**Q1/Q5**
% (N)	n=281	n=202	n=226	n=172	n=178	**Ratio**
In-hospital*	21.0 (59)	22.8 (46)	12.8 (29)	15.1 (26)	7.9 (14)	2.7
30-Day*	21.0 (59)	22.8 (46)	12.4 (28)	14.5 (25)	7.3 (13)	2.9
1-Year*	34.2 (91)	33.2 (67)	22.1 (50)	20.9 (36)	14.0 (25)	2.3
5-Year*	55.5 (156)	51.5 (104)	38.1 (86)	40.7 (70)	27.0 (48)	2.1

Note: the number of deaths is given in parentheses after the percentages

**P 0.000*

The same inverse trend (decreased mortality with the increase in income) remains within groups stratified by the comorbidity score. The most pronounced difference was evident in the strata of AMI patients with no comorbidities recorded during the index

hospitalization (Charlson score 0). The largest difference was in 30-day mortality (Q1/Q5 ratio 7.3), while for the 1-year and 5-year mortality the Q1/Q5 ratio was 3 (Figure 4.13). In the strata of patients who had one clinically important comorbidity (Charlson Score 1) the overall trend in mortality was similar, although the difference was not that large (Q1/Q5 ratio was 1.9, 2.3, 1.6 for the 30-day, 1-year and 5-year mortality respectively).

Figure 4.12. Mortality after AMI in Different Income Quintiles.

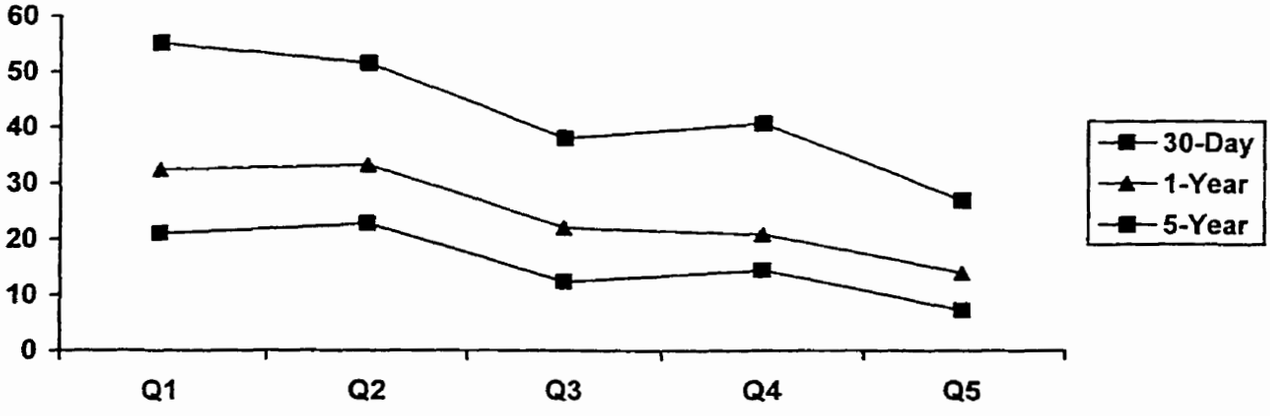
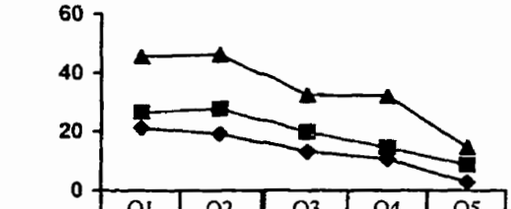


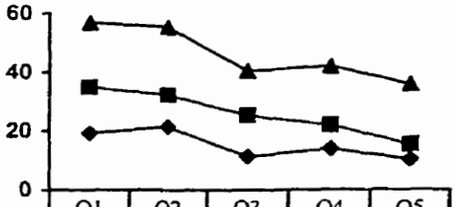
Figure 4.13 Mortality in Income Quintiles, Stratified by the Comorbidity Score

Comorbidity Score=0



	Q1	Q2	Q3	Q4	Q5
30-day	21.2	19.4	13.2	10.7	2.9
1-year	26.5	27.8	19.8	14.5	8.8
5-year	45.6	46.3	32.4	32.1	14.7

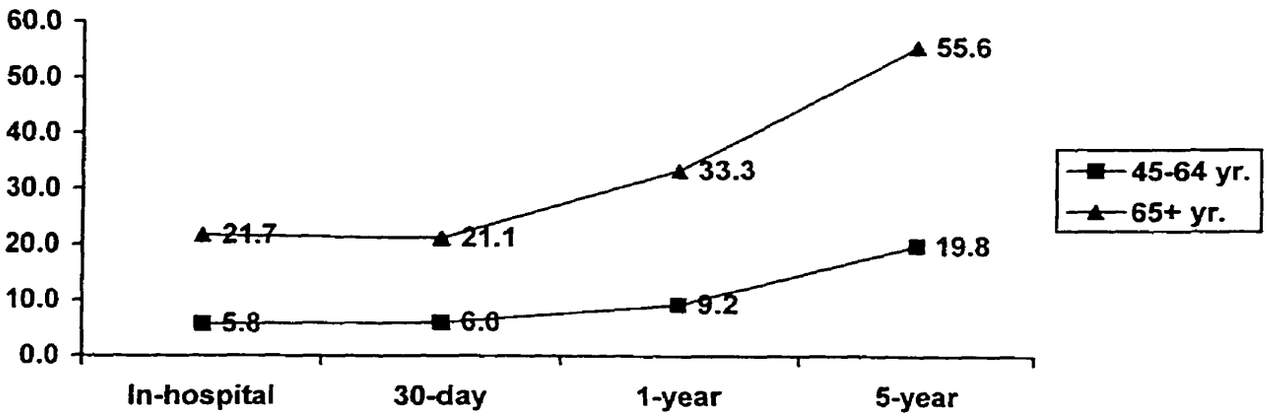
Comorbidity Score=1



	Q1	Q2	Q3	Q4	Q5
30-day	19.3	21.5	11.4	14.0	10.3
1-year	34.9	32.3	25.3	22.0	15.4
5-year	56.9	55.4	40.5	42.0	35.9

Mortality rates of AMI patients also varied depending on the age group to which patients belonged. Mortality during hospitalization was 3.8 times higher among the older patients, 30-days mortality was 3.5 times higher among them, 1-year mortality was 3.6 times higher, and 5-year mortality was 2.8 times higher than one among the younger patients (p 0.001). As many as 80% of pre-retirement age patients survived 5 years after the index episode of AMI, while less than 50% of older patients were alive at that time (Fig. 4.14).

Figure 4.14 Post-AMI mortality in different age groups



Mortality and the Timeliness of Arrival at the Hospital

Among patients hospitalized early in the course of AMI, mortality varied significantly with respect to income quintiles. For those who arrived at the hospital later (more than 6 hours after the onset of symptoms) the difference, although present, was not statistically significant (Table 4.12).

Table 4.12 **AMI Mortality across Income Quintiles**

Stratified by the Time of Arrival at the Hospital

Rate	Hours to Arrival	Q1 *n=234*	Q2 *n=182*	Q3 *n=200*	Q4 *n=157*	Q5 *n=168*	P
In-hospital	1-6	16.7	18.8	10.1	12.3	7.5	0.042
	7+	19.1	20.0	11.5	18.6	5.7	0.261
30-day mortality	1-6	16.0	18.0	9.4	12.3	7.5	0.059
	7+	20.2	21.5	11.5	16.3	2.9	0.090
1-year mortality	1-6	26.0	28.2	18.0	18.4	14.3	0.029
	7+	34.5	33.8	19.7	23.3	11.4	0.036
5-year mortality	1-6	48.0	49.6	33.1	41.2	24.1	0.001
	7+	56.0	47.7	41.0	32.6	31.4	0.041

CHAPTER FIVE STATISTICAL MODELLING

5.1 Construction of AMI Mortality Prediction Model

Mortality after an episode of acute myocardial infarction depends on a wide range of different factors. Not only the timeliness of medical treatment and its type, but also the patient's demographic characteristics and comorbidities that existed prior to AMI contribute to the probability of survival after an AMI. Which factors are the most important and accurate in predicting mortality after an AMI? And which of them should be included in the logistic regression models for prediction of AMI mortality? This chapter deals with the modeling of treatment and survival after AMI, evaluating the significance of risk factors, including socioeconomic status, on the outcomes of AMI.

5.2 A Logistic Regression Model

The initial logistic regression models for administrative data were based on work by J. Tu and his collaborators using a relatively large cohort of 52,616 patients having an acute myocardial infarction in Ontario between 1994 and 1997. Forty variables were considered for the model and subsequently tested. The final model included 11 variables. Those Ontario statistical models predicted 30-day and 1-year mortality very well with an area under the Receiver Operator Characteristic (ROC) curve of 0.78 for 30-day mortality and 0.79 for 1-year mortality (Hanley and McNeil, 1982), (Tu et al, forthcoming). Two independent validation data sets from Manitoba (4,836 AMI patients) and California (112,234 AMI patients), showed identical ROC areas for 30-day and 1-year mortality - 0.77 and 0.78 respectively. Therefore, using the Ontario AMI mortality prediction rules

for the analysis of a smaller Winnipeg data set seemed appropriate, even though some of the predictors of mortality were not statistically significant with the fewer cases in the Winnipeg sample.

The prevalence of risk factors included in the prediction model in Winnipeg AMI cohort was very similar to the one of the Ontario cohort despite the large difference in size (Table 5.1). In both data sets the most prevalent comorbidity was congestive heart failure, followed by cardiac dysrythmias.

The logistic regression model for 30-day mortality prediction worked reasonably well. The strongest predictors of mortality were shock (odds ratio 16.56, 95% C.I. 7.28-37.68), followed by acute renal failure (OR 8.59, 95% C.I. 3.04-24.24), and age greater then 75 years (OR 7.69, 95% C.I. 2.14-27.66). Other significant risk factors for mortality were pulmonary edema (OR 3.41, C.I. 1.34-8.64) and congestive heart failure (OR 1.81, C.I. 1.21-2.69). Although some of the factors were not significant compared to the Ontario cohort, the overall performance of the model yielded very similar results with ROC of 0.77 (Table 5.2).

For the 1-Year mortality prediction model the presence of shock, congestive heart failure, acute and chronic renal failure, as well as age greater then 65 years were significant predictors of mortality. Despite the lack of significance for the other factors in the model (due to sample size), the overall performance of the model was the same as in Ontario – a ROC area was 0.79.

When the same model is tested for the 5-year mortality, all the variables (except for the presence of pulmonary edema and female gender) became statistically significant

predictors of mortality; this is primarily due to the larger number of deaths. The ROC area is 0.82 (Table 5.2). Of note is the fact, that the direction of association for mortality and female gender is the opposite (rather protective) than that of the ICES model, where to be a female was a risk for increased mortality. This may be due to exclusions generated in developing the clinical sample.

5.3 Comparing Administrative and Chart Abstracts Data

The extensive administrative and clinical data for a sample of Winnipeg AMI patients has made it possible to examine the utility of administrative data in some detail. Using British Columbia data, Humphries et al (2000) went back two years before the index hospitalization to compare comorbidities generated from administrative data with those derived from clinical data. Although comorbidities were slightly underestimated in administrative data, predictions using administrative data were very similar to those based on chart reviews.

When selected conditions are compared according to the source of information in the Winnipeg cohort, the most important conditions in terms of outcomes, such as cardiogenic shock, congestive heart failure, diabetes mellitus, and chronic renal failure, were reported with similar frequency in administrative and clinical data. In fact, the administrative database reported a higher percentage of congestive heart failure (consistent with Humphries et al.). Conditions significantly less reported by administrative data included previous AMI, pulmonary edema, cancer, and stroke (Table 5.3). Abstracted clinical information of patients whose comorbidity score was zero according to administrative data, revealed that one third of them had history of

hypertension, 25% had angina, and 20% had an ischemic heart disease and myocardial infarction recorded in the medical chart during index hospitalization. For the reasons that are not clear, these and other comorbidities were particularly under-reported in the discharge abstracts of low-income patients (Table 5.3.1).

The logistic regression models for predicting 30-day, 1-year, and 5-year mortality after AMI using conditions tested by the ICES model (Tu et al, 1999) were constructed separately on the bases of administrative and clinical data. Slight changes were made to facilitate comparisons of the two data sets. Diabetes with complications was not coded in the clinical study; therefore the "diabetes" diagnosis was used for comparison. Also, only "chronic" renal failure was distinguished in the chart review. In addition, conditions known as "cardiac dysrhythmias" were not coded in the chart review. Despite the above noted differences in the frequency of some conditions, the prediction models were strikingly similar (Table 5.4). The most important predictors of in-hospital mortality in both models were age (75 years and older), shock, congestive heart failure, and chronic renal failure for the model based on administrative data. 30-day mortality was best predicted by age (65 years and older), shock, congestive heart failure, pulmonary edema and chronic renal failure for the model based on administrative data. In the chart-based model, only the age (greater than 65 yr.) and shock were significant predictors of mortality. The same variables were significant risk factors for the 1-year mortality. All variables, except for the female gender and pulmonary edema, were significant predictors of the 5-year mortality in the model based on administrative data, while being female was protective in the model based on chart data. Finally, age 50-64 yr. and diabetes failed to reach significance level in the model based on chart abstracts (Table 5.4).

5.4 Models Based on Clinical Data

Detailed information extracted from the charts of AMI patients allows incorporating of some of the most important clinical measures into the mortality prediction model. Both the diagnosis and clinical status reported on admission, together with the basic physiological measurements (blood pressure, heart rate, etc.) and electrocardiographic changes may aid in predicting the outcomes of AMI. After evaluation of the significance of each of these factors (Table 5.5), only those variables significant at the 0.05 level were selected.

Thus, some electrocardiographic changes were important risk factors for in-hospital AMI mortality: ventricular fibrillation and bundle brunch block, as well as supraventricular tachycardia, while the presence of ST or T-wave changes was predictive of a favorable outcome. Cardiac arrest, shock, congestive heart failure, and third heart sound present during admission were risk factors predictive of AMI mortality. Somewhat surprisingly, the history of previous AMI was not a significant factor even in univariate analysis, as was the anterior or lateral location of AMI, which was a risk factor in the model suggested by Krumholz et al, 1999, but not in our analysis. Conversely, the admission diagnosis "chest pain", as well as increased systolic blood pressure and heart rate were "protective" factors for AMI mortality (except for tachycardia for 5-year mortality). Such laboratory findings, as creatine kinase greater then 400 U/L on admission, prothrombin time greater then 14/sec, increased levels of blood urea nitrogen and glucose were also significant predictors of mortality after myocardial infarction (Tables 5.6 and 5.8). Another factor greatly affecting mortality was marital status. Married (and living common law) patients had greatly reduced risk of 1-year and 5-year

mortality. Therefore, the inclusion of these risk factors into the logistic regression model could potentially result in a better statistical model for predicting the outcome of AMI. All these variables were incorporated into multivariate logistic regressions, which also included the variables from administrative data presented in Table 5.2. Only variables with 20 or more cases were included in the analysis. Ventricular fibrillation (N=15) and supraventricular tachycardia (N=10) were eliminated according to this criterion

The above-mentioned model (by Krumholz et al., 1999) was derived from a large population (82,359) of AMI patients 65 years of age and older in the US. That model involved 7 variables describing patient's status on arrival to the hospital: age, cardiac arrest, anterior or lateral location of AMI, systolic blood pressure, white blood cell count, serum creatinine, and congestive heart failure. Each of these variables was included to the testing of the model based on Winnipeg cohort of 1,059. All models appear reasonably robust. Adding the clinical variables moderately increased the area under the ROC curve. The ROC area went from 0.77 to 0.82 for 30-day mortality and from 0.82 to 0.86 for 5-year mortality. Similar results were obtained with comorbidities taken from the clinical, rather than the administrative data. These measures of fit compare very well with those reported in the literature (Krumholz, 1999).

5.5 Treatment

A large number of recent studies have shown the use of routine medical therapies after acute myocardial infarction to improve survival (Topol et al., 1993; Murray et al., 1999; Tu et al., 1999), etc. (for more see chapter two). In the model adjusted by patient demographics, the use of aspirin and beta-blockers significantly improved survival,

while, somewhat surprisingly, the use of early thrombolytic therapy did not affect mortality (Table 9). The choice of therapy is largely dependent on the patient's status and symptoms present during admission. Advanced age (65 years and older) and congestive heart failure was predictive of lesser utilization rates for all drug therapies (age 75+ for beta-blockers). Cardiac arrest negatively affected the use of heparin, beta-blockers, and aspirin, while shock and dyspnea reduced rates of use of aspirin and beta-blockers. Patients admitted with the diagnosis "AMI / rule out AMI" and with chest pain as a major symptom on admission were more likely to receive each drug therapy. Socioeconomic status appears to affect utilization of beta-blockers, heparin and (to an even higher degree) thrombolytic therapy (Table 10). The model was the most sensitive in predicting thrombolytic therapy (ROC area 0.751) and less sensitive in predicting the use of beta-blockers (ROC area 0.693). Gender was not a significant factor in the utilization of the drug therapies in this sample of AMI patients.

5.6 Socioeconomic Status

A number of demographic, diagnostic and clinical measures differ significantly across income quintiles (Table 5.8). Overall, AMI patients of lower socioeconomic status were more likely to be older, female, and not married than their affluent counterparts. Similarly, the conditions such as congestive heart failure, cardiomegaly, tachycardia (heart rate greater than 100 beats per minute), and high level of blood urea nitrogen (BUN) were significantly more prevalent among patients who were residents in lower income neighborhoods. Upper income patients were more likely to have their highest creatine kinase (CK) equal to or greater than 400 U/L and report chest pain on admission.

Addition of socioeconomic status to the logistic regression models revealed that even after controlling for numerous demographic and clinical risk factors, income level was still a significant predictor of mortality after AMI. Patients in the highest income group (Q5) had much-improved chances of both short-term and long-term survival after AMI. The overall model performance improved with the ROC area statistics for 30-day, 1-year, and 5-year mortality, being 0.83, 0.84, and 0.86 respectively. These enhanced AMI mortality prediction models are presented in Table 5.7. Similarly, after controlling for the demographic characteristics and medical therapies received in the hospital, socioeconomic status still plays an important role in the outcome of AMI. Patients residing in the high-income neighborhoods have a significantly decreased risk of mortality (Table 5.11).

Table 5.1. Risk Factors Included in the Mortality Prediction Model for Ontario and Manitoba AMI Cohorts

Risk Factors	**ICD-9 CM Codes**	**Prevalence**		
		Ontario N=52,616	**Winnipeg** N=1,059	
		%	%	N
Age 50-64		27.2	26.3	278
65-74		28.9	33.0	349
70+		32.8	34.2	362
Female		36.9	43.1	456
Shock	785.5	2.5	3.5	37
Diabetes w/ complications	250.1-250.9	2.0	3.5	37
Congestive heart failure	428.x	20.7	23.9	253
Malignancy	140.0-208.9	1.9	2.1	22
Cerebrovascular disease	430.0-438.x	4.1	6.1	65
Pulmonary edema	518.4, 514.x	1.3	2.2	23
Acute renal failure	584.x, 586.x, 788.5	1.5	2.0	21
Chronic renal failure	585.x, 403-404.x, 996.7 394.2, 399.4, v451	2.4	3.5	37
Cardiac dysrhythmias	427.0-427.9	14.7	18.9	200

Table 5.2. Predicting AMI Mortality

Risk Factors	In-hospital			30-day		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age* 50-64 yr.	0.4485	1.57	0.40-6.12	0.4481	1.57	0.44-5.96
65-74 yr.	1.0966	2.99	0.80-11.16	1.266	3.55	0.98-12.85
75+ yr.	2.1665	8.73	2.37-32.07	2.0397	7.69	2.14-27.66
Female	0.1022	1.11	0.76-1.62	-0.1094	0.90	0.61-1.31
Shock	2.6196	13.73	6.15-30.66	2.8072	16.56	7.28-37.68
Diabetes w/complications	0.7096	2.03	0.86-4.81	0.3518	1.42	0.29-4.54
Congestive heart failure	0.4997	1.65	1.11-2.50	0.5907	1.81	1.21-2.69
Malignancy	-0.2437	0.78	0.23-2.73	-0.3494	0.71	0.20-2.49
Cerebrovascular disease	0.3499	1.42	0.75-2.68	0.0754	1.08	0.56-2.09
Pulmonary edema	0.7195	2.05	0.79-5.31	1.2251	3.41	1.34-8.64
Acute renal failure	2.1355	8.46	3.00-23.87	2.1503	8.59	3.04-24.24
Chronic renal failure	0.7014	2.02	0.87-4.65	0.7568	2.13	0.92-4.93
Cardiac dysrhythmias	0.2503	1.28	0.84-1.98	-0.0776	0.93	0.59-1.45
Intercept	-3.6316			-3.4747		
N / Deaths	1059 / 174			1059 / 171		
Area Under the Curve	0.791			0.768		

*Reference group for odds ratio is those of 45-49 years

Table 5.2. Predicting AMI Mortality

Risk Factors	1-year			5-year		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age* 50-64 yr.	0.6218	1.86	0.58-5.98	0.9947	2.70	1.07-6.97
65-74 yr.	1.457	4.29	1.38-13.32	1.7945	6.02	2.38-15.24
75+ yr.	2.5273	12.52	4.05-38.71	3.0143	20.37	7.97-52.08
Female	-0.2092	0.81	0.58-1.13	-0.2139	0.81	0.59-1.11
Shock	2.7422	15.52	6.06-39.78	1.8894	6.62	2.40-18.24
Diabetes w/complications	0.2019	1.22	0.52-2.89	1.4615	4.31	1.74-10.68
Congestive heart failure	0.6741	1.96	1.38-2.79	1.2606	3.53	2.13-31.77
Malignancy	0.8246	2.28	0.85-6.13	2.108	8.23	2.13-31.77
Cerebrovascular disease	0.2174	1.24	0.69-2.25	1.3273	3.77	1.82-7.82
Pulmonary edema	0.7858	2.19	0.84-5.70	0.0941	1.10	0.37-3.29
Acute renal failure	2.2909	9.88	2.91-33.55	1.6367	5.14	1.27-20.86
Chronic renal failure	2.0666	7.90	3.24-19.26	4.0402	56.84	6.92-466.50
Cardiac dysrhythmias	0.2288	1.26	0.85-1.85	0.4249	1.53	1.04-2.25
Intercept	-3.4747			-2.7489		
N / Deaths	1054 / 269			1035 / 464		
Area Under the Curve 0.801				Area Under the Curve 0.824		

*Reference group for odds ratio is those of 45-49 years

Table 5.3 Comparing Reported Comorbidities by Data Source

Comorbidity	Administrative Data		Chart Abstract				%	
	%	N	%	N	X^2	P	Agreement	Kappa
Shock	3.49	37	4.53	48	2.45	0.223	9•3.5	0.25
Diabetes	18.89	200	22.19	235	3.54	0.057	9•3.3	0.81
Congestive heart Failure	23.89	253	21.06	223	1.21	0.118	7•9.8	0.42
Malignancy	2.08	22	6.52	69	25.3	0.000	9□.7	0.24
Cerebrovascular Disease	6.14	65	9.73	103	9.34	0.002	9□.3	0.48
Pulmonary edema	2.17	23	9.25	98	49.3	0.000	9□.7	0.16
Chronic renal Failure	3.49	37	31.3	35	0.37	0.810	98.1	0.77
Previous AMI	9.35	99	27.00	286	112.4	0.000	8□.8	0.39

Table 5.4 Comparing Models Based on Adminstrative Data and on Chart Abstracts

30-Day Mortality

	Administrative Data			Chart Abstracts		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age 50-64	0.4672	1.60	0.43-5.99	0.3021	1.35	0.37-4.97
65-74	1.3509	3.86	1.08-13.75	1.3686	3.93	1.13-13.66
75+	2.1100	8.25	2.33-29.25	2.0952	8.13	2.34-28.18
Female	-0.1811	0.83	0.57-1.21	-0.2401	0.79	0.54-1.14
Shock	2.8356	17.04	7.54-38.50	2.6436	14.06	6.82-29.01
Diabetes	0.0831	1.09	0.69-1.72	0.2539	1.29	0.85-1.96
Congestive heart failure	0.5300	1.70	1.16-2.50	0.3349	1.40	0.91-2.14
Malignancy	-0.2709	0.76	0.22-2.59	-0.1254	0.88	0.44-1.77
Cerebrovascular disease	0.0964	1.101	0.57-2.12	-0.1700	0.84	0.47-1.52
Pulmonary edema	1.3251	3.76	1.51-9.35	0.3239	1.38	0.79-2.43
Renal failure	0.8805	2.41	1.05-5.53	0.6375	1.89	0.83-4.29
Intercept	-3.4501			-3.3554		
N / Deaths	1,059 / 171					
Area Under the Curve	*0.759*			*0.760*		

Table 5.4 Comparing Models Based on Adminstrative Data and on Chart Abstracts (continued)

5-Year Mortality						
	Administrative Data			**Chart Abstracts**		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age 50-64	0.9409	2.56	1.02-6.42	0.7758	2.17	0.91-5.19
65-74	1.7503	5.76	2.34-14.18	1.6405	5.16	2.20-12.11
75+	3.0036	20.16	8.08-50.27	2.7179	15.15	6.38-35.98
Female	-0.268	0.77	0.56-1.05	-0.3618	0.70	0.51-0.95
Shock	2.0373	7.67	2.82-20.89	2.4007	11.03	4.14-29.36
Diabetes	0.4209	1.52	1.04-2.23	0.3367	1.40	0.99-1.99
Congestive heart failure	1.322	3.75	2.63-5.36	1.3986	4.05	2.69-6.09
Malignancy	2.005	7.43	1.93-28.54	0.6307	1.88	1.04-3.38
Cerebrovascular disease	1.2973	3.66	1.78-7.52	0.9707	2.64	1.57-4.44
Pulmonary edema	0.2251	1.25	0.42-3.73	0.2921	1.34	0.74-2.41
Renal failure	4.0213	55.77	6.90-450.54	2.3906	10.92	2.34-51.03
Intercept	-2.6363			-2.4196		
N / Deaths	1,035 / 464					
Area Under the Curve	*0.817*			*0.810*		

Table 5.5 Significant Univariate Predictors of Mortality after AMI

Variable	Prevalence		Mortality		
	%	N	30-day	1-year	5-year
Married / living common law	56.8	601	+	+	
Laboratory results (earliest after hospital admission)					
1-st CK >=400	42.7	452		+	
Highest CK >=400	89.3	946	+	+	+
High WBC	39.3	416	+	+	+
High PT	10.1	107			+
High BUN	31.3	331	+	+	+
Hyperglycemia	85.1	901	+		
Cardiomegaly on X-ray	16.2	292			+
Electrocardiography results (earliest after hospital admission)					
Other ST changes	31.3	331	+	+	+
Bundle brunch block	10.2	108		+	+
Results of Physical examination (results during admission)					
Heart rate <60 /min	11.2	119			+
Heart rate >100 /min	19.7	209	+	+	+
SBP<90 mm Hg	2.7	29	+	+	
SBP>140 mm Hg	51.1	541	+	+	+
Respiration>20/min	45.1	478	+	+	+
3-rd heart sound	4.0	42	+	+	+
Diagnosis on admission					
Chest pain	62.1	658	+	+	+
Cardiac arrest	9.4	99	+		
N			1,059	1,054	1,035
Deaths			171	269	464

Variables included were significant at the .05 level for one or more of the mortality intervals. Only variable with 20 or more cases were included; ventricular fibrillation showed a statistically significant relationship w mortality but only 15 cases were recorded.

Table 5.6 Predicting post-AMI Mortality using Adminstrative and Clinical Data

	30-Day			5-Year		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age: 50-64 yr.	0.5378	1.46	0.38-5.56	1.0576	2.88	1.10-27.52
65-74 yr.	1.1398	3.13	0.85-11.45	1.8065	6.09	2.36-15.75
75+ yr.	1.8616	6.43	1.75-23.67	2.8484	17.26	6.56-45.39
Female	-0.2459	0.78	0.52-1.17	-0.5180	0.60	0.41-0.86
Married / common law				-0.4770	0.62	0.44-0.88
Administrative data						
Shock	2.5322	12.58	5.40-29.33	1.5050	4.50	1.59-12.75
Diabetes w/complications	-0.0349	0.97	00.36-2.56	1.1841	3.27	1.26-8.49
Congestive heart failure	0.5236	1.69	1.11-2.57	0.8034	2.23	1.50-3.34
Malignancy	-0.3973	0.67	0.19-2.35	2.1495	8.58	2.16-34.16
Cerebrovascular disease	-0.1442	0.87	0.0.43-1.75	1.1859	3.27	1.56-6.86
Pulmonary edema	1.0013	2.72	1.00-7.43	-0.4193	0.66	0.21-2.10
Acute renal failure	2.0759	7.97	2.59-24.57	1.6217	5.06	1.03-24.89
Chronic renal failure	0.889	2.43	1.01-5.88	4.1112	61.02	6.13-607.63
Cardiac dysrhythmia	-0.2937	0.75	0.46-1.20	0.2575	1.29	0.86-1.95
Laboratory results						
High WBC count				0.4542	1.58	1.13-2.21
High PT	0.7636	2.15	1.22-3.77	0.7363	2.09	1.24-3.52
High BUN	0.4519	1.57	1.04-2.38	0.6930	2.00	1.40-2.90
Hyperglycemia	0.6853	1.98	1.03-3.81			
Cardiomegaly on X-ray				0.5672	1.76	1.21-2.56

Table 5.6 Predicting post-AMI Mortality using Administrative and Clinical Data (continued)

	30-Day			5-Year		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Physical examination						
Heart rate <60 /min				-0.6958	0.50	0.29-0.85
Heart rate >100 /min				0.4769	1.61	1.04-2.49
SBP>140 mm Hg	-0.7313	0.48	0.32-0.72	-0.4246	0.65	0.49-0.91
Respiration >20/min				0.3759	1.46	1.04-2.03
3-rd heart sound	0.7845	2.19	1.03-4.69			
Diagnosis						
Chest pain	-0.6043	0.55	0.37-0.81	-0.3658	0.69	0.50-0.97
Cardiac arrest	0.7369	2.09	1.18-3.70			
Intercept	-3.5221			-2.5272		
N	1,059			1,035		
Deaths	171			464		
Area Under the Curve	*0.816*			*0.863*		

Table 5.7 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status

Risk Factors	30-Day			5-Year		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age: 50-64 yr.	0.4257	1.53	0.40-5.83	1.1352	3.11	1.72-8.26
65-74 yr.	1.1064	3.02	0.83-11.04	1.8421	6.31	2.40-16.58
75+ yr.	1.8106	6.11	1.67-22.41	2.8816	17.84	6.68-47.67
Female Sex	-0.2595	0.77	0.51-1.16	-0.5206	0.59	0.41-0.86
Married / common law				-0.3729	0.69	0.48-0.99
Administrative data						
Shock	2.4973	12.15	5.16-28.64	1.5145	4.55	1.57-13.21
Congestive heart failure	0.4937	1.64	1.07-2.51	0.8235	2.28	0.52-3.41
Malignancy	-0.4242	0.65	0.19-2.32	2.2842	9.82	2.42-39.78
Cerebrovascular disease	-0.1139	0.89	0.44-1.82	1.0711	3.57	1.69-7.53
Pulmonary edema	0.9146	2.50	0.90-6.93	-0.4375	0.65	0.20-2.11
Acute renal failure	2.0669	7.90	2.52-24.80	1.4669	4.34	0.89-21.08
Chronic renal failure	0.8264	2.29	0.93-5.65	4.0470	57.23	5.83-561.80
Diabetes w/complications	0.0646	1.07	0.40-2.86	1.2327	3.43	1.29-9.12
Cardiac dysrhythmias	-0.2990	0.74	0.46-1.20	0.2775	1.32	0.88-1.99
Laboratory results						
High WBC				0.4958	1.64	1.17-2.31
High PT	0.7590	2.14	1.21-3.78	0.7797	2.18	1.28-3.71
High BUN	0.4493	1.57	1.03-2.38	0.6747	1.94	1.37-2.82
Hyperglycemia	0.6786	1.97	1.03-3.79			
Cardiomegaly on X-ray				0.5407	1.72	1.18-2.51

Table 5.7 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status (continued)

	30-Day			5-Year		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Physical examination						
Heart rate <60 /min				-0.6978	0.50	0.29-0.86
Heart rate >100 /min				0.4707	1.60	1.03-2.48
SBP>140 mm Hg	-0.7360	0.48	0.32-0.72	-0.4211	0.66	0.47-0.92
Respiration >20/min				0.3527	1.42	1.02-1.99
3-rd heart sound	0.7355	2.09	0.97-4.50			
Diagnosis on admission						
Chest pain	-0.5813	0.56	0.38-0.83	-0.3203	0.73	0.52-1.02
Cardiac arrest	0.8066	2.24	1.26-3.98			
Socioeconomic status						
Income Quintile* Q2	0.2260	1.25	0.74-2.11	-0.1952	0.82	0.51-1.32
Q3	-0.5298	0.59	0.33-1.05	-0.5421	0.58	0.37-0.92
Q4	-0.1080	0.90	0.49-1.63	-0.3088	0.73	0.44-1.21
Q5	-0.7258	0.48	0.24-0.98	-0.9743	0.38	0.22-0.66
Intercept	-3.3278			-2.3189		
N	1,059			1,035		
Deaths	171			646		
Area Under the Curve	*0.825*			*0.868*		

*Reference category for odds ratio is those from the neighborhoods with the lowest income (Q1).

Table 5.8 Socioeconomic Status and Distribution of Model Variables

Risk Factors	Prevalence N=1059 %	N	Income Quintile Q1 N=281	Q2 N=202	Q3 N=226	Q4 N=172	Q5 N=178	P
Age 50-64 years	26.3	278	18.5	20.3	31.4	24.4	40.5	0.001
65-74 years	33.0	349	31.0	38.1	31.9	36.6	28.1	0.197
75+ years	34.2	362	44.5	36.6	31.0	29.7	23.6	0.001
Female	43.1	456	53.7	41.1	42.5	40.1	32.0	0.001
Married / common law	56.8	601	39.2	52.5	59.3	66.3	77.0	0.001
Adminstrative data								
Shock	3.5	37	5.0	3.5	4.4	2.9	0.6	0.129
Diabetes with complications	3.5	37	4.6	1.0	4.0	3.5	3.9	0.297
Congestive heart failure	23.9	253	28.1	29.7	22.6	19.2	16.9	0.008
Malignancy	2.1	22	2.5	2.5	0.9	1.2	3.4	n/a
Cerebrovascular Disease	6.1	65	5.0	8.4	5.3	5.2	7.3	0.493
Pulmonary edema	2.2	23	1.8	4.5	2.2	1.2	1.1	n/a
Acute renal failure	2.0	21	2.5	2.5	2.2	2.2	1.1	n/a
Chronic renal failure	3.5	37	3.6	5.0	2.2	4.7	2.3	0.423
Cardiac dysrhythmias	18.9	200	19.2	20.8	19.5	19.2	15.2	0.705
Physical examination								
Heart rate <60 /min	11.2	119	11.4	8.4	10.2	11.1	15.7	0.241
Heart rate >100 /min	19.7	209	25.6	22.8	17.7	16.3	12.9	0.006
SBP<140 mm Hg	51.1	541	50.9	52.0	50.4	50.0	52.3	0.991
3rd heart sound	4.0	42	4.3	5.5	3.5	3.5	2.8	0.724
Laboratory results								
CK>=400	22.7	820	22.8	25.7	21.2	19.8	23.0	0.700
Highest CK>=400	69.1	732	63.4	66.8	66.8	74.4	78.7	0.004
High BUN	31.3	331	38.4	33.2	29.2	27.9	23.6	0.01
WBC>11*109	39.3	416	38.1	41.1	42.0	36.6	38.2	0.780
PT>14mm/sec	10.1	107	10.0	10.4	11.1	8.7	10.1	0.961
Cardiomegaly on X-ray	16.2	292	29.9	32.2	32.3	24.4	15.7	0.001
Diagnosis on admission								
Chest pain	62.1	658	52.3	61.4	62.0	70.4	70.8	0.001
Cardiac arrest	9.4	99	10.3	7.4	10.6	10.5	7.3	0.599

Table 5.9 Medical Therapies for AMI

Variable	30-day Mortality			1-Year Mortality			5-Year Mortality		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age 50-64 yr.	0.3443	1.410	0.39-5.06	0.4924	1.64	0.54-4.94	0.9190	2.510	1.07-5.90
65-74 yr.	1.1985	3.320	0.97-11.28	1.3839	3.99	1.37-11.59	1.8027	6.070	2.62-14.03
75+ yr.	1.7392	5.690	1.67-19.39	2.1730	8.79	3.02-25.54	2.7071	14.990	6.39-35.13
Female	-0.2801	0.760	0.52-1.10	-0.3368	0.71	0.51-0.99	-0.3347	0.720	0.53-0.97
Aspirin	-0.8151	0.440	0.30-0.65	-0.8817	0.41	0.29-0.59	-0.6291	0.530	0.38-0.75
Heparin	-0.0718	0.930	0.61-1.43	-0.1902	0.83	0.57-1.19	-0.4897	0.610	0.44-0.85
Thrombolytics	0.0466	1.050	0.64-1.73	-0.1849	0.83	0.54-1.28	-0.2859	0.751	0.52-1.08
Beta-blockers	-3.5501	0.029	0.01-0.12	-1.7731	0.17	0.10-0.28	-1.4524	0.230	0.17-0.33
Intercept	-2.0747			-1.6422			-1.0371		
N	1,059			1,054			1,035		
Deaths	171			269			464		
ROC area	*0.799*			*0.800*			*0.805*		

Table 5.10 Predictive Models for Therapy

Risk Factors	Thrombolytic Therapy			Aspirin			Heparin			Beta-blockers		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age 50-64	-0.3245	0.72	0.41-1.26	-0.9495	0.38	0.14-1.06	-0.1787	0.83	0.44-1.58	0.1432	1.15	0.66-2.02
65-74	-0.5158	0.60	0.34-1.05	-1.3311	0.26	0.09-0.71	-0.6383	0.52	0.28-0.98	-0.0406	0.96	0.55-1.68
75+	-1.4337	0.24	0.13-0.44	-1.6366	0.19	0.07-0.52	-1.2911	0.27	0.14-0.51	-0.4850	0.62	0.34-1.10
Female	0.0382	1.04	0.76-1.41	-0.1679	0.85	0.61-1.16	-0.1938	0.82	0.88-1.54	-0.0003	1.00	0.74-1.35
Chest pain	0.5801	1.79	1.29-2.47	0.8092	2.25	1.61-3.13	0.7397	2.10	1.56-2.82	0.5216	1.69	1.23-2.30
Congestive heart failure	-1.4250	0.24	0.11-0.52	-0.6919	0.50	0.34-0.77	-0.6529	0.52	0.34-0.80	-1.5010	0.22	0.11-0.17
Dyspnea				-0.5173	0.60	0.42-0.85	-0.2959	0.74	0.54-1.01	-0.4336	0.65	0.46-0.91
Cardiac arrest				-0.5492	0.58	0.35-0.94	-0.5296	0.59	0.34-0.95	-0.6075	0.55	0.30-0.98
Myocardial infarction	0.7265	2.07	1.52-2.80	0.3725	1.45	1.05-2.02	0.5528	1.74	1.31-2.31			
3-rd heart sound							0.7809	2.18	1.08-4.42			
Shock				-1.8891	0.15	0.08-0.30				-2.7398	0.07	0.01-0.48
Rales	-0.5026	0.61	0.39-0.94									
Income Quintile: Q2	0.4115	1.51	0.96-2.37	-0.2170	0.80	0.51-1.25	0.3254	1.39	0.93-2.06	0.0966	1.10	0.71-1.70
Q3	0.2549	1.29	0.83-2.00	0.0018	1.00	0.64-1.56	0.1044	1.11	0.76-1.63	0.2481	1.28	0.84-1.95
Q4	0.6913	2.00	1.26-3.16	-0.0526	0.94	0.58-1.54	0.4317	1.54	1.01-2.36	0.3783	1.46	0.94-2.28
Q5	0.9394	2.56	1.62-4.03	0.0397	1.04	0.62-1.73	0.5904	1.81	1.17-2.79	0.4499	1.57	1.02-2.42
Intercept	-1.3431			2.4929			-0.0251			-0.9797		
N / received therapy	1,059 / 335			1,059 / 796			1,059 / 623			1,059 / 318		
Area Under the Curve	0.751			0.746			0.722			0.693		

Table 5.11 Prediction of Mortality by Medical Therapy and Socioeconomic Status

Variable	30-day Mortality			1-year Mortality			5-year Mortality		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age 50-64 yr.	0.3808	1.46	0.40-5.30	0.5156	1.68	0.55-5.09	1.0048	2.73	1.15-6.49
65-74 yr.	1.1658	3.21	0.94-11.01	1.3648	3.92	1.34-11.44	1.8216	6.18	2.65-14.43
75+ yr.	1.7083	5.52	1.61-18.93	2.1497	8.58	2.93-25.11	2.7195	15.17	6.42-35.87
Female	-0.2941	0.75	0.51-1.09	-0.3558	0.70	0.50-0.98	-0.3682	0.69	0.51-0.94
Aspirin	-0.8214	0.44	0.30-0.65	-0.8966	0.41	0.29-0.60	-0.6371	0.53	0.37-0.75
Heparin	-0.0552	0.95	0.62-1.46	-0.1796	0.84	0.58-1.21	-0.4901	0.61	0.44-0.85
Thrombolytics	0.0768	1.08	0.65-1.79	-0.1420	0.87	0.56-1.34	-0.2361	0.79	0.56-1.14
Beta-blockers	-3.5157	0.03	0.01-0.12	-1.7444	0.18	0.11-0.29	-1.4289	0.24	0.17-0.34
Income Quintile Q2	0.1992	1.22	0.75-1.98	0.1702	1.19	0.76-1.84	-0.0466	0.95	0.63-1.46
Q3	-0.4452	0.64	0.38-1.09	-0.3015	0.74	0.47-1.17	-0.5308	0.59	0.39-0.89
Q4	-0.2379	0.79	0.45-1.39	-0.3732	0.69	0.42-1.14	-0.3061	0.74	0.47-1.16
Q5	-0.8366	0.43	0.22-0.85	-0.6555	0.52	0.30-0.90	-0.8262	0.44	0.27-0.70
Intercept	-1.9028			-1.4831			-0.8048		
N / Deaths	1,059 / 171			1,054 / 269			1,035 / 464		
ROC area	*0.809*			*0.807*			*0.813*		

CHAPTER SIX TESTING MODELS ON DIFFERENT POPULATIONS

The ICES mortality prediction model was tested on different populations of AMI patients in Manitoba. The model was applied to the overall cohort of 1,817 patients, as well as to 1,388 Manitobans residing in urban areas (mainly Winnipeg and Brandon). In addition, the model was tested on the subgroup of patients with so-called "definite AMI". This group was identified during the chart review process if the clinical presentation compatible with the diagnosis of AMI (chest pain, angina, etc.) was accompanied by certain EKG changes: "Q-wave / transmural AMI", or "loss of R forces", or "Q waves (new, significant, large)", and creatine kinase above the upper limit of normal (200U/L) (Ducas, 1998). Out of 1,059 Winnipeg's AMI patients, two thirds (66.7%) fitted the above criteria.

Testing the eleven-variable ICES model (chapter 5) on different populations of AMI patients revealed that the highest reliability of the model was achieved when applied to the most homogeneous population of patients – Winnipeg residents who were classified as cases of "definite AMI" (Table 6.1). All ROC curves were above 0.80, and higher than ones for the two other populations (Tables 6.2 and 6.3). However, the EPV values were less then 10 for in-hospital and 30-day mortality models with only 84 and 86 deaths. However, the number of events (deaths) was sufficient for higher EPV values in the logistic regression models predicting 1-year and 5-year mortality. Significant for predicting 1-year mortality were age greater than 65 years, shock, congestive heart failure, acute and chronic renal failure. The ROC statistics was 0.83. When this same

model was tested for the 5-year mortality, all variables but three (pulmonary edema, cardiac dysrhythmia, and female gender) became statistically significant predictors of mortality. The ROC area was also 0.83 (Table 6.1).

Testing the model on a larger and more diverse patients' population (residents of urban areas) produces slightly smaller ROC statistics (Table 6.2). 30-day mortality was best predicted by presence of shock, congestive heart failure, pulmonary edema, and acute renal failure, as well as advanced age (65 years and older), with ROC curve 0.75. A 1-year mortality was predicted by the same variables but pulmonary edema, with addition of malignancy and chronic renal failure, with the ROC statistic 0.80. The same variables as for the "definite AMI" were significant in the 5-year model (ROC statistics 0.81).

When the same model was tested on the total cohort of Manitoba's AMI patients, more variables were significant in predicting mortality than in any other, although, due to the diversity of patients, the corresponding ROC statistic was somewhat smaller then one for the other populations (Table 6.3). For the 1-Year mortality prediction model presence of shock, congestive heart failure, malignancy, pulmonary edema, and acute renal failure, as well as advanced age (65 years and older were significant predictors of mortality. Although some of the factors were not significant compared to the Ontario cohort, the overall performance of the model yielded very similar results with ROC of 0.77 (Table 6.3). For the 5-year mortality prediction model nine out of eleven model variables (the two being pulmonary edema and female gender) were significant predictors of mortality (ROC statistic was 0.80).

As in the previous chapter, several clinical variables were tested for their significance in predicting AMI mortality and included in the extended risk model. Model

combining administrative and clinical data. Variables significant in predicting 30-day and 5-year mortality are listed in Table 6.4 for the urban Manitoba population and in Table 6.5 for the population with "definite AMI". The models first were tested without socioeconomic variables, and then with addition of socioeconomic status on both populations. Addition of SES variable slightly improved the robustness of the overall models: for the 30-day and 5-year models based on urban population of Manitoba the ROC statistics increased from 0.81 and 0.85 to 0.82 and 0.86, and in the 5-year model based on "definite AMI" subset it changed from 0.86 to 0.87. The most important finding, however, was not the size of improvement, but that SES was an independent factor influencing risk of death after AMI. Comparisons of the odds ratios of death for each risk variable between the models without and with SES revealed that all individual odds ratios remained unchanged after the addition of SES variable (Tables 6.4 and 6.5). Therefore, the contribution of SES to the mortality after an AMI is independent of the other risk factors. For each of these models, residence in the highest income neighborhoods greatly decreased risk of post-AMI death when other factors were controlled for. For urban residents of the most affluent neighborhoods, the odds ratio for 30-day mortality for was 0.48 (95% C.I. 0.25-0.92), while for the 5-year mortality it was even more impressive: 0.36 (95% C.I. 0.22-0.60). In the "definite AMI" subgroup, the most affluent patients had the odds ratio for 5-year mortality only one quarter of those of their lowest-income counterparts: 0.23 (95% C.I. 0.11-0.49).

Similarly, the contribution of SES into AMI mortality was independent of the patients' demographic characteristics and treatment received in the hospital, when it was tested on the clinically more homogeneous population of patients with "definite AMI".

(Tables 6.6 and 6.7). Addition of the SES variable improved the overall model performance results (ROC curve risen from 0.80 to 0.82 for 30-day and 1-year mortality, and from 0.78 to 0.80 for the 5-year mortality). All adds ratios of individual variables remained unchanged after the SES variable was introduced into the model, indicating the independent mechanism of action of SES on mortality. Residents of the highest income neighborhoods had significantly lower odds of deaths (0.3 for 30-day and 5-year, and 0.5 for 1-year mortality). Another interesting observation was that while the use of beta-blockers, aspirin, and heparin reduces mortality, thrombolytic therapy was not significant factor in reducing mortality (these results are similar to the ones obtained from the Winnipeg cohort, chapter 5). The use of beta-blockers was significant in reducing 30-day mortality, aspirin and beta-blockers independently reduced 1-year mortality and the latter and heparin reduces 5-year mortality, thrombolytic therapy was not significant factor in reducing. As with the Winnipeg cohort, the gender variable did not reach the level of statistical significance.

Table 6.1 Predicting AMI Mortality Among "Definite AMI" Subgroup

	In-hospital			30-day		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age* 50-64 yr.	-0.0356	0.97	0.17-5.41	0.1653	1.18	0.22-6.26
65-74 yr.	0.8481	2.34	0.46-11.94	1.0846	2.96	0.60-14.57
75+ yr.	2.0901	8.09	1.62-40.28	1.8411	6.30	1.29-30.78
Female	0.0855	1.09	0.64-1.86	-0.1099	0.90	0.52-1.53
Shock	2.7886	16.26	5.52-47.89	3.1559	23.48	7.72-71.36
Diabetes w/complications	-0.0529	0.95	0.22-4.05	-0.5404	0.58	0.12-2.94
Congestive heart failure	0.6756	1.97	1.12-3.46	0.8818	2.42	1.36-4.28
Malignancy	0.1514	1.16	0.22-6.06	0.017	1.02	0.20-5.16
Cerebrovascular disease	0.4618	1.59	0.64-3.95	0.2002	1.22	0.47-3.18
Pulmonary edema	0.5609	1.75	0.53-5.84	0.6593	1.93	0.57-6.56
Acute renal failure	1.8132	6.13	1.57-24.00	1.9682	7.16	1.81-28.25
Chronic renal failure	1.2003	3.32	1.14-9.70	0.5192	3.01	1.01-8.96
Cardiac dysrhythmias	0.126	1.13	0.61-2.10	1.1005	0.70	0.36-1.37
Intercept	-3.8176			-3.6648		
N / Deaths	706 / 86			706 / 84		
Area Under the Curve	0.827			0.804		

*Reference group for odds ratio is those of 45-49 years

Table 6.1 Predicting AMI Mortality Among "Definite AMI" Subgroup

Risk Factors	1-year			5-year		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age* 50-64 yr.	0.9392	2.56	0.49-13.48	1.5548	4.73	1.26-17.77
65-74 yr.	1.6545	5.23	1.02-26.92	2.2306	9.31	2.49-34.74
75+ yr.	2.7771	16.07	3.15-81.94	3.1772	23.98	6.37-90.25
Female	-0.2299	0.80	0.50-1.27	-0.2074	0.81	0.55-1.21
Shock	3.1717	23.85	6.38-89.17	2.3961	10.98	2.59-46.61
Diabetes w/complications	-0.796	0.45	0.10-2.03	1.6811	5.37	1.61-17.97
Congestive heart failure	0.9417	2.56	1.57-4.19	1.4802	4.39	2.80-6.91
Malignancy	0.7325	2.08	0.54-7.99	3.3876	29.59	3.22-271.91
Cerebrovascular disease	0.3854	1.47	0.63-3.43	1.4233	4.15	1.67-10.32
Pulmonary edema	0.3576	1.43	0.44-4.65	-0.7392	0.48	0.14-1.60
Acute renal failure	2.4605	11.71	2.65-51.82	2.1766	8.817	1.60-48.47
Chronic renal failure	2.4574	11.67	3.65-37.34	4.0792	59.10	6.23-560.24
Cardiac dysrhythmias	0.3455	1.41	0.83-2.41	0.2484	1.28	0.79-2.09
Intercept	-3.9325			-3.4341		
N / Deaths	706 / 135			706 / 261		
Area Under the Curve 0.833				Area Under the Curve 0.831		

*Reference group for odds ratio is those of 45-49 years
Population: "definite AMI"

Table 6.2. Predicting AMI Mortality Among Urban Manitoba Population

Risk Factors	In-hospital			30-day		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age* 50-64 yr.	0.5746	1.78	0.46-6.83	0.6482	1.91	0.52-7.10
65-74 yr.	1.2569	3.52	0.95-13.03	1.4482	4.26	1.19-15.28
75+ yr.	2.308	10.05	2.75-36.75	2.1948	8.98	2.53-31.93
Female	0.0992	1.10	0.80-1.52	-0.0856	0.92	0.67-1.26
Shock	2.6981	14.85	6.83-32.31	2.833	17.00	7.67-37.66
Diabetes w/complications	0.9578	2.61	1.26-5.40	0.6321	1.88	0.89-3.97
Congestive heart failure	0.4894	1.63	1.16-2.30	0.5391	1.72	1.22-2.42
Malignancy	-0.0929	0.91	0.30-2.74	0.0454	1.05	0.37-2.96
Cerebrovascular disease	0.387	1.47	0.83-2.60	0.2055	1.23	0.69-2.20
Pulmonary edema	0.7251	2.07	0.87-4.90	1.102	3.01	1.29-7.04
Acute renal failure	1.7708	5.88	2.42-14.24	1.8385	6.29	2.59-15.25
Chronic renal failure	0.5263	1.69	0.77-3.72	0.6658	1.95	0.90-4.22
Cardiac dysrhythmias	0.1896	1.209	0.82-1.78	-0.1215	0.89	0.59-1.32
Intercept	-3.723			-3.5663		
N / Deaths	1388 / 231			1388 / 234		
Area Under the Curve	0.775			0.748		

*Reference group for odds ratio is those of 45-49 years

Table 6.2. Predicting AMI Mortality Among Urban Manitoba Population

Risk Factors	1-year			5-year		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age* 50-64 yr.	0.7652	2.15	0.68-6.80	1.1011	3.01	1.19-7.62
65-74 yr.	1.5744	4.83	1.57-14.88	1.955	7.06	2.82-17.68
75+ yr.	2.5444	12.74	4.16-39.00	3.0693	21.53	8.59-53.94
Female	-0.0653	0.94	0.71-1.25	-0.1297	0.88	0.67-1.15
Shock	2.7551	15.72	6.31-39.19	1.9807	7.25	2.70-19.45
Diabetes w/complications	0.478	1.61	0.78-3.34	1.489	4.43	2.04-9.65
Congestive heart failure	0.6666	1.95	1.43-2.65	1.1914	3.29	2.40-4.52
Malignancy	1.2929	3.64	1.48-8.96	2.5079	12.28	3.31-45.51
Cerebrovascular disease	0.3126	1.37	0.80-2.34	1.074	2.93	1.58-5.42
Pulmonary edema	0.7886	2.20	0.91-5.33	-0.0757	0.93	0.36-2.42
Acute renal failure	2.2645	9.63	3.29-28.21	1.5612	4.76	1.43-15.86
Chronic renal failure	2.0422	7.71	3.37-17.61	4.2318	68.84	8.59-552.05
Cardiac dysrhythmias	0.1817	1.20	0.85-1.70	0.5158	1.68	1.19-2.35
Intercept	-3.3448			-2.9173		
N / Deaths	1388 / 356			1388 / 611		
Area Under the Curve 0.779				Area Under the Curve 0.814		

*Reference group for odds ratio is those of 45-49 years
Population: urban residents of Manitoba

Table 6.3 Predicting AMI Mortality in Manitoba, 1991/92

	In-hospital			30-day		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age* 50-64 yr.	0.9899	2.69	0.77-9.46	0.7485	2.11	0.70-6.40
65-74 yr.	1.5339	4.64	1.35-15.94	1.4174	4.13	1.40-12.19
75+ yr.	2.4883	12.04	3.54-40.97	2.0979	8.15	2.78-23.90
Female	0.1621	1.18	0.89-1.56	0.0075	1.01	0.76-1.33
Shock	2.973	19.55	9.72-39.35	3.0181	20.45	10.01-41.78
Diabetes w/complications	0.9734	2.65	1.40-4.99	0.6819	1.98	1.04-3.78
Congestive heart failure	0.3519	1.42	1.04-1.95	0.4649	1.59	1.17-2.17
Malignancy	0.112	1.12	0.41-3.05	0.1856	1.20	0.46-3.14
Cerebrovascular disease	0.3958	1.49	0.86-2.56	0.2647	1.30	0.75-2.25
Pulmonary edema	0.6891	1.99	0.92-4.33	0.945	2.57	1.20-5.52
Acute renal failure	1.7568	5.79	2.65-12.68	1.9495	7.03	3.17-15.57
Chronic renal failure	0.4415	1.56	0.74-3.27	0.5192	1.68	0.81-3.48
Cardiac dysrhythmias	0.2337	1.26	0.89-1.79	-0.0198	0.98	0.69-1.40
Intercept	-3.9456			-3.5142		
N / Deaths	1817 / 298			1817 / 310		
Area Under the Curve	0.765			0.745		

*Reference group for odds ratio is those of 45-49 years

Table 6.3 Predicting AMI Mortality in Manitoba, 1991/92

Risk Factors	1-year			5-year		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age* 50-64 yr.	0.9171	2.50	0.92-6.82	0.9179	2.50	1.20-5.24
65-74 yr.	1.6142	5.02	1.88-13.45	1.7027	5.49	2.65-11.36
75+ yr.	2.4798	11.94	4.48-31.79	2.7652	15.88	7.66-32.92
Female	-0.0536	0.95	0.74-1.22	-0.1009	0.90	0.72-1.14
Shock	2.885	17.90	7.93-40.45	2.0949	8.13	3.39-19.46
Diabetes w/complications	0.5202	1.68	0.90-3.16	1.1265	3.09	1.60-5.96
Congestive heart failure	0.6957	2.01	1.52-2.65	1.2629	3.54	2.65-4.71
Malignancy	1.1918	3.29	1.43-7.59	2.2579	9.56	3.04-30.10
Cerebrovascular disease	0.2925	1.34	0.81-2.23	0.8685	2.38	1.34-4.23
Pulmonary edema	0.9249	2.52	1.14-5.57	0.2286	1.26	0.53-3.00
Acute renal failure	2.1743	8.80	3.37-22.96	1.6962	5.45	1.74-17.12
Chronic renal failure	1.9208	6.83	3.16-14.74	3.3783	29.32	6.47-132.93
Cardiac dysrhythmias	0.1982	1.22	0.89-1.67	0.5378	1.72	1.26-2.33
Intercept	-3.3254			-2.6290		
N / Deaths	1817 / 460			1817 / 786		
Area Under the Curve 0.769				Area Under the Curve 0.804		

*Reference group for odds ratio is those of 45-49 years
Population: overall 1-year cohort of AMI cases in Manitoba

Table 6.4 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status among "Definite AMI" Subgroup

	30-Day Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age: 50-64 yr.	0.3895	1.48	0.25-8.61	0.4056	1.50	0.26-8.67
65-74 yr.	1.1796	3.25	0.60-17.71	1.1314	3.10	0.57-16.76
75+ yr.	2.0478	7.75	1.44-41.76	2.0058	7.43	1.40-39.60
Female Sex	-0.0816	0.92	0.53-1.62	-0.0239	0.98	0.55-1.74
Administrative data						
Shock	3.0956	22.10	7.09-68.86	3.0671	21.48	6.78-68.07
Congestive heart failure	0.7796	2.18	1.21-3.93	0.7723	2.17	1.19-3.94
Malignancy	-0.0037	1.00	0.19-5.28	0.1586	1.17	0.21-6.57
Cerebrovascular disease	-0.0544	0.95	0.34-2.62	-0.0250	0.98	0.35-2.69
Pulmonary edema	0.3538	1.43	0.40-5.07	0.3638	1.44	0.39-5.27
Acute renal failure	2.0411	7.70	1.73-34.32	1.9548	7.06	1.55-32.14
Chronic renal failure	1.2833	3.61	1.19-10.96	1.1253	3.08	0.98-9.71
Diabetes w/complications	-0.6958	0.50	0.10-2.57	-0.6487	0.52	0.10-2.79
Cardiac dysrhythmias	-0.4563	0.63	0.32-1.24	-0.4377	0.65	0.33–1.27
Physical Examination						
ST /T wave changes	-0.9636	0.38	0.19-0.78	-0.9930	2.18	0.18–0.78

Table 6.4 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status (continued)

	30-Day Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Laboratory results						
Hyperglucemia	1.5310	4.62	1.44-14.89	1.4892	4.43	1.38-14.25
CK>400 U/L	0.8450	2.33	1.29-4.21	0.8841	2.42	1.33-4.40
Diagnosis on admission						
Chest pain	-0.8300	0.44	0.23-0.82	-0.7768	0.46	0.24-0.87
Socioeconomic status						
Income Quintile* Q2				0.4607	1.56	0.75-3.33
Q3				-0.0237	0.98	0.42-2.28
Q4				0.3585	1.43	0.61-3.38
Q5				-0.6423	0.53	0.18-1.57
Intercept	-4.5375			-4.6398		
N	706			706		
Deaths	84			84		
Area Under the Curve	*0.847*			*0.859*		

*Reference category for odds ratio is those from the neighborhoods with the lowest income (Q1).

Note: "definite AMI" population

Table 6.4 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status (continued)

	5-Year Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age: 50-64 yr.	1.6034	4.97	1.32-18.77	1.7693	5.87	1.49-23.04
65-74 yr.	2.1696	8.76	2.32-33.02	2.2821	9.80	2.49-38.48
75+ yr.	3.058	21.28	5.56-81.52	3.1995	24.52	6.14-97.90
Female Sex	-0.4429	0.64	0.40-1.02	-0.4498	0.64	0.40-1.02
Married / common law	-0.5649	0.57	0.36-0.89	-0.4282	0.65	0.41-1.03
Administrative data						
Shock	2.2520	9.51	2.26-40.06	2.1553	8.63	1.95-38.18
Congestive heart failure	1.1875	3.28	2.02-5.33	1.2452	3.47	2.12-5.69
Malignancy	3.0312	20.72	2.36-181.68	3.5745	35.68	3.69-344.87
Cerebrovascular disease	1.2792	3.59	1.40-9.20	1.2748	3.58	1.36-9.40
Pulmonary edema	-0.8224	0.44	0.12-1.60	-0.8991	0.41	0.11-1.57
Acute renal failure	1.9036	6.71	1.09-41.34	1.7847	5.96	0.95-37.51
Chronic renal failure	3.9314	50.98	4.61-563.70	3.8147	45.36	4.30-478.10
Diabetes w/complications	1.2966	3.66	1.07-12.45	1.3219	3.75	1.08-13.05
Cardiac dysrhythmias	0.1620	1.18	0.70-1.96	0.1926	1.21	0.72-2.03
Physical Examination						
Heart rate > 100/min	0.5797	1.79	1.02-3.12	0.5583	1.75	0.99-3.08

Note: "definite AMI" population

Table 6.4 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status (continued)

	5-Year Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Laboratory results						
High WBC count	0.7338	2.08	1.38-3.15	0.8322	2.30	1.50-3.51
High PT	0.7559	2.13	1.15-3.95	0.8102	2.25	1.19-4.25
High BUN	1.0679	2.91	1.87-4.58	1.0596	2.89	1.83-4.55
Socioeconomic status						
Income Quintile* Q2				-0.1907	0.83	0.46-1.48
Q3				-0.5414	0.58	0.32-1.05
Q4				-0.0762	0.93	0.50-1.73
Q5				-1.4517	0.23	0.11-0.49
Intercept	-3.6232			-3.5169		
N	706			706		
Deaths	261			261		
Area Under the Curve	*0.862*			*0.873*		

*Reference category for odds ratio is those from the neighborhoods with the lowest income (Q1).

Note: "definite AMI" population

Table 6.5 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status among Urban Manitoba Population

	30-day Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age: 50-64 yr.	0.6370	1.89	0.50-7.21	0.6693	1.95	0.51-7.49
65-74 yr.	1.3654	3.92	1.05-14.56	1.2961	3.66	0.98-16.66
75+ yr.	2.0134	7.49	2.03-27.68	1.9276	6.87	1.85-25.53
Female Sex	-0.0879	0.92	0.64-1.32	-0.0640	0.94	0.65-1.36
Administrative data						
Shock	2.5892	13.32	5.98-29.65	2.5934	13.38	5.94-30.13
Congestive heart failure	0.4021	1.50	1.04-2.15	0.3947	1.48	1.03-2.14
Malignancy	0.1295	1.14	0.41-3.19	0.1290	1.14	0.40-3.25
Cerebrovascular disease	0.0725	1.08	0.58-1.98	0.1546	1.17	0.63-2.16
Pulmonary edema	1.0637	2.90	1.17-7.18	1.0298	2.8	1.11-7.05
Acute renal failure	1.5879	4.89	1.84-13.04	1.6098	5.00	1.88-13.30
Chronic renal failure	0.6763	1.97	0.87-445	0.6420	1.90	0.83-4.36
Diabetes w/complications	0.3931	1.48	0.68-3.25	0.4499	1.57	0.71-3.44
Cardiac dysrhythmias	-0.2821	0.75	0.50-1.15	-0.2692	0.76	0.50-1.17
Physical Examination						
SBP > 140 mm Hg	-0.8348	0.43	0.31-0.61	-0.8187	0.44	0.31-0.62
Respiration >20/min	1.0496	2.86	1.30-6.27	1.0672	2.91	1.31-6.46

Table 6.5 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status (continued)

	30-day Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Laboratory results						
High WBC count	0.4934	1.64	1.17-2.29	0.5102	1.67	1.19-2.33
High BUN	0.5513	1.74	1.23-2.46	0.5564	1.74	1.23-2.47
Hyperglucemia	0.7701	2.16	1.19-3.94	0.7555	2.13	1.17-3.89
ST/T wave changes	-0.5309	0.59	0.39-0.88	-0.5148	0.60	0.40-0.90
Admission diagnosis						
Chest pain	-0.5151	0.60	0.43-0.83	-0.4738	0.62	0.45-0.87
Socioeconomic status						
Income Quintile* Q2				0.1607	1.17	0.77-1.79
Q3				-0.4654	0.63	0.38-1.03
Q4				-0.2246	0.80	0.46-1.38
Q5				-0.7373	0.48	0.25-0.92
Intercept	-3.7297			-3.6016		
N	1,388			1,388		
Deaths	234			234		
Area Under the Curve	*0.813*			*0.822*		

*Reference category for odds ratio is those from the neighborhoods with the lowest income (Q1).

Note: urban population

Table 6.5 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status (continued)

	5-Year Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Age: 50-64 yr.	1.0687	2.91	1.15-7.37	1.1302	3.10	1.20-7.97
65-74 yr.	1.8881	6.61	2.63-16.58	1.8810	6.56	2.57-16.74
75+ yr.	2.8648	17.55	6.97-44.20	2.8299	16.94	6.62-43.37
Female Sex	-0.3928	0.68	0.50-0.92	-0.3973	0.67	0.49-0.92
Married / common law	-0.3252	0.72	0.54-0.98	-0.2401	0.79	0.58-1.07
Administrative data						
Shock	1.4138	4.11	1.49-11.38	1.4624	4.32	1.52-12.27
Congestive heart failure	0.7407	2.10	1.49-2.96	0.7691	2.16	1.53-3.05
Malignancy	2.4547	11.64	3.11-43.64	2.6936	14.78	3.77-57.97
Cerebrovascular disease	0.9811	2.67	1.42-5.01	1.081	2.95	1.57-5.53
Pulmonary edema	-0.5046	0.60	0.23-1.62	-0.4886	0.61	0.23-1.68
Acute renal failure	1.2212	4.23	1.11-16.13	1.3597	3.90	1.03-14.74
Chronic renal failure	4.1461	63.19	6.85-582.68	4.1424	62.95	6.80-583.22
Diabetes w/complications	1.3456	3.84	1.70-8.68	1.3562	3.88	1.71-8.82
Cardiac dysrhythmias	0.3343	1.40	0.98-2.00	0.3719	1.45	1.01-2.08
Physical Examination						
Heart rate < 60/min	-0.8089	0.45	0.28-0.71	-0.7877	0.46	0.28-0.73
Heart rate > 100/min	0.4712	1.60	1.11-2.31	0.4699	1.60	1.11-2.31
SBP < 90 mm Hg	0.952	2.59	1.05-6.41	0.9148	2.50	1.00-6.26
SBP > 140 mm Hg	-0.4059	0.67	0.50-0.88	-0.4053	0.67	0.50-0.89
Respiration >20/min	0.3783	1.46	1.10-1.93	0.3631	1.44	1.08-1.91

Table 6.5 Predicting AMI Mortality from Administrative and Clinical Data with Addition of Socioeconomic Status (continued)

	5-Year Mortality					
	Without SES			*With SES*		
Risk Factors	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**	**Parameter Estimate**	**Odds Ratio**	**95% C.I.**
Laboratory results						
High WBC count	0.4785	1.61	1.22-2.14	0.5135	1.67	1.25-2.23
High BUN	0.6643	1.94	1.44-2.63	0.6792	1.97	1.46-2.67
Cardiomegaly on X-ray	0.5929	1.81	1.31-2.49	0.582	1.79	1.30-2.47
Admission diagnosis						
Chest pain	-0.3052	0.74	0.55-0.98	-0.2500	0.78	0.58-1.04
Socioeconomic status						
Income Quintile* Q2				-0.0687	0.93	0.64-1.36
Q3				-0.5591	0.57	0.39-0.85
Q4				-0.3390	0.71	0.46-1.11
Q5				-1.0108	0.36	0.22-0.60
Intercept	-2.7037			-2.5136		
N	1,388			1,388		
Deaths	611			611		
Area Under the Curve	*0.851*			*0.857*		

*Reference category for odds ratio is those from the neighborhoods with the lowest income (Q1).

Note: urban population

Table 6.6 Medical Therapies for AMI among "Definite AMI" Subgroup

Variable	30-day Mortality			1-Year Mortality			5-Year Mortality		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age 50-64 yr.	-0.0150	0.99	0.20-4.82	0.5406	1.72	0.38-7.86	1.0342	2.81	0.95-8.36
65-74 yr.	1.0971	3.00	0.67-13.46	1.5211	4.58	1.04-20.08	1.8435	6.32	2.16-18.53
75+ yr.	1.7417	5.71	1.26-25.80	2.4117	11.15	2.53-49.19	2.6296	13.87	4.64-41.43
Female	-0.1456	0.86	0.52-1.44	-0.2733	0.76	0.49-1.19	-0.1855	0.83	0.57-1.20
Aspirin	-0.1812	0.83	0.47-1.49	-0.5570	0.57	0.35-0.94	-0.5968	0.55	0.35-0.86
Heparin	-0.1161	0.89	0.49-1.61	-0.3697	0.69	0.42-1.14	-0.5095	0.60	0.40-0.91
Thrombolytics	-0.1149	0.89	0.46-1.72	-0.0335	0.97	0.56-1.69	-0.1669	0.85	0.55-1.30
Beta-blockers	-3.1217	0.04	0.01-0.18	-1.8694	0.15	0.08-0.30	-1.3595	0.26	0.17-0.39
Intercept	-2.3261			-1.8505			-0.9675		
N	706			706			706		
Deaths	84			135			261		
ROC area	*0.801*			*0.804*			*0.783*		

Table 6.7 Prediction of Mortality by Medical Therapy and Socioeconomic Status among "Definite AMI" Subgroup

Variable	30-day Mortality			1-year Mortality			5-year Mortality		
	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.	Parameter Estimate	Odds Ratio	95% C.I.
Age 50-64 yr.	0.0620	1.06	0.21-5.24	0.6062	1.83	0.40-8.47	1.1141	3.05	1.02-9.12
65-74 yr.	1.0853	2.96	0.65-13.45	1.5579	4.75	1.07-21.05	1.8499	6.36	2.15-18.80
75+ yr.	1.7757	5.91	1.29-26.97	2.4739	11.87	2.66-52.91	2.6433	14.06	4.67-42.37
Female	-0.1584	0.85	0.51-1.44	-0.2936	0.75	0.48-1.71	-0.2540	0.78	0.53-1.13
Aspirin	-0.1789	0.84	0.46-1.51	-0.5931	0.55	0.34-0.91	-0.6280	0.53	0.34-0.84
Heparin	-0.0839	0.92	0.51-1.67	-0.3313	0.72	0.43-1.19	-0.5047	0.60	0.40-0.92
Thrombolytics	-0.0834	0.92	0.47-1.79	0.0121	1.00	0.58-1.78	-0.0868	0.92	0.59-1.42
Beta-blockers	-3.1294	0.04	0.01-0.18	-1.8815	0.15	0.08-0.30	-1.3476	0.26	0.17-0.39
Income Quintile Q2	0.3660	1.44	0.74-2.80	0.4114	1.51	0.84-2.72	-0.1574	0.85	0.51-1.42
Q3	-0.0774	0.93	0.44-1.94	0.1577	1.17	0.63-2.19	-0.5180	0.60	0.35-1.01
Q4	0.0943	1.10	0.51-2.36	-0.0742	0.93	0.47-1.84	-0.3344	0.72	0.41-1.26
Q5	-1.0968	0.33	0.12-0.94	-0.7853	0.46	0.21-1.00	-1.1827	0.31	0.17-0.56
Intercept	-2.3454			-1.9190			-0.5950		
N / Deaths	706 / 84			706 / 571			706 / 261		
ROC area	*0.815*			*0.816*			*0.799*		

CHAPTER SEVEN DISCUSSION AND CONCLUSIONS

7.1 Introduction

This work has focused on the issues related to clinical characteristics of AMI and their association with the patients' socioeconomic characteristics, as reflected by the assigned income quintile. Additionally, the extensive administrative and clinical data for a sample of AMI patients has made it possible to discuss the utility of administrative data in some detail.

Politics and economics play an important role in the shaping of both the patterns of population health and disease and the structure and delivery of health care. Many combinations of objective and subjective factors and circumstances working on individual and aggregate levels are not yet fully explained. While examining medical care provided to hospitalized patients with acute myocardial infarctions, the focus of the study was on exploring how the variations in socioeconomic status affect clinical features, outcomes, and treatment.

Patients with acute myocardial infarctions receive medical care that requires continuity and consistency. The elements of cardiac care can be systematized in several equally important categories. They include immediate hospital treatment; post-infarction invasive diagnostic procedures and surgical revascularization; rehabilitation and secondary prevention of cardiac death and re-infarction by generalist and specialist physicians on a long-term basis. And, practically in each category of interventions, studies have found the inverse relationship between socioeconomic status and rates of access or utilization of interventions being investigated.

The cardiac care and services available to patients with the acute myocardial infarctions should be assessed in relation to some fundamental principles of Canada's national health care system. Canadian Medicare was designed to provide access to universal and comprehensive coverage for all medically necessary hospital, diagnostic, and physician services, with equality in access to these services being one of the milestones of a universal health care system. The Canada Health Act (1984) specified five fundamental principles on which Canada's national health care system is based. They are Public Administration, Portability, Universality, Comprehensiveness, and Accessibility. The last principle is particularly important in the evaluation of the relationship between SES and outcomes of acute myocardial infarctions (since the latter also depend on the type of treatment interventions patients receive). Accessibility implies that "...the [provincial] plan must provide, on uniform terms and conditions, reasonable access to insured hospital and physician services without barriers... No one may be discriminated against on the basis of income, age, health status, etc." (Canada's Health System, Health Canada, 1999).

Was this the case with AMI patients? While examining the socio-economic differences in outcomes of acute myocardial infarctions, the question of the accessibility of services for diverse patients' population needs to be addressed. Did patients receive medical care and treatment based on uniform standards? Were all diagnostic and surgical services equally available to any qualified patient? A number of national and international studies on myocardial infarction suggest that significant inter-regional variations exist in treatment practice and outcomes of the disease, as well as in the rates of invasive revascularization procedures (Roos and Mustard, 1997; Hartford et al., 1998;

Alter et al., 1999; Murray et al., 1999). There is some evidence that "...women with proven heart disease are investigated and treated differently than men" (Naylor, 1999). Moreover, despite Canada's universal health care system, it fails to provide equality in health care delivery. Results of one of the most recent studies in Ontario (Alter et al, 1999) support this statement. The researchers found that although "...Canada's federal – provincial Medicare plan covers all medically necessary services provided by hospitals and physicians without any user fees and is based on the principle of access according to need rather than income...those of a low socioeconomic status remain less likely to receive specific services than wealthier patients...and... socioeconomic status had a pronounced effect on the access to specialized cardiac services and on mortality one year after acute myocardial infarction". A similar effect of socioeconomic status on mortality and medical treatments was found in this study.

7.2 Socioeconomic Status

Socio-economic status is associated with the distribution of diseases in the population. In the study cohort the number of AMI patients declined along with the increase in the average income. Forty-six percent of patients from the AMI cohort belonged to the two lower income quintiles (Q1 and Q2). As noted elsewhere (Schwartz et al., 1997), women had AMI episodes much later in their life than men. High-income patients tend to be younger, male, and married.

Socioeconomic status contributes to patients' health status prior to the episode of AMI, and, ultimately, to the outcomes of AMI. Low-income patients had higher rates of such conditions as congestive heart failure, cerebrovascular disease / stroke, and chronic

pulmonary diseases, which existed prior to the index AMI event. These findings are consistent with the work of Alter et al. (1997) in Ontario and Peltonen et al. (2000) in Sweden.

Higher-income patients were admitted to hospital much sooner after the onset of symptoms of acute myocardial infarction than lower-income patients. This may be a result of a higher awareness by high-income patients about symptoms and signs of AMI, or such patients might have the means to get to the hospital without delay. The environment may play a role, in that the chance of having a more knowledgeable person around when AMI happens is higher for the more affluent subjects. Once been admitted to the hospital with AMI, lower income patients had communicated chest pain less frequently than higher income patients, but had more frequently decreased level of consciousness, and higher rates of tachycardia and shortness of breath. However, they received several standard treatment and diagnostic interventions at a lesser rate. In particular, more affluent patients received thrombolytic therapy and heparin almost twice more often as their low-income counterparts. Stress exercise testing and multiple gate angiography (MUGA scan) were also performed more often among higher income patients. Invasive revascularization procedures, such as coronary angioplasty and coronary artery bypass surgery, were utilized with similar frequency for patients from all income groups during the index hospitalization. However, for the most part these procedures are performed after the initial AMI hospitalization and treatment have been completed, and, therefore, these figures do not represent the persistent SES trends in invasive cardiac procedures.

When mortality after an episode of acute myocardial infarction is compared across income quintiles, dramatic differences become evident. Mortality was much higher among patients from the lowest income quintiles than in their intermediate and high-income counterparts. Both short-term and long-term figures showed a striking inverse relationship between the patient's income and mortality. Lower income patients had a markedly decreased overall chance to survive an AMI episode. Additionally, to be married / or to live in a common law relationship was predictive of a better survival after AMI. Kawachi et al. (1996), while studying the relationship between social networking and cardiovascular disease incidence in men in the United States reported that socially isolated individuals had a much-increased risk of the disease. Moreover, "...of the individual components of the social networks index, being unmarried was the strongest predictor of cardiovascular disease mortality..." (Ibid.). The substantial intercorrelation between socioeconomic status and marital status (higher income patients were married twice as often as those of lower income) stresses out the need to include both variables in understanding the role of social factors in health.

The differences in post-AMI survival among income quintiles appear to depend on differences in both health status and treatment. Although variations in rates of AMI and survival among income quintiles are well known, the reasons for the differences in survival have been less well researched.

Significant progress has been made in the past 30 years in the drug therapy associated with acute myocardial infarction. A number of national and international clinical trials clearly proved the survival benefits of thrombolytic agents, aspirin, and beta-blockers, and their value in prevention of re-infarctions after the initial episode of

AMI. However, in the 1990, several U.S. studies (and only a few Canadian studies) assessed the treatment practice of AMI. These studies invariably showed the existing gap between recommendations for practice and actual practice (Mark et al., 1994; Meehan et al., 1995; Murray et al., 1999). Deviations from the optimal standard in actual practice are significant, indicating that equality in receiving high-quality medical care is still a desirable goal, not a reality. For example, Rogers et al. (1994) reported that drug therapy was underutilized: only 35.1% of patients received thrombolytic therapy and 53.7% received beta-blockers in a retrospective analysis of treatment of AMI in 1990-1993. In another study, after mailing clinical guidelines to 37 hospitals in Minnesota and later reviewing how the treatment recommendations were implemented, McLaughlin et al. (1996) found that 81% of eligible patients received aspirin therapy, 72% received thrombolytic agents, and only 53% were treated with beta-blockers; most disparities in the use of these drugs were found in the elderly and in women. Phillips et al. (2000) pointed out that "Recent surveys indicate that beta-blockers are prescribed to as few as 40% of eligible patients... and they are particularly underused in women and elderly patients".

Murray et al. (1999) assessed the utilization rates and survival benefits for the AMI patients treated with the therapies recommended by the AHA and ACC in 1992 and showed similar rates of aspirin therapy (82%), but somewhat lower rates of thrombolytic therapy (60%) in eligible patients in 1991/92. A one-year mortality was higher among patients with inappropriate omission of either of these therapies. Appropriate medical intervention has clearly played a role in the increased survival after AMI observed in recent years.

While the study confirmed that socioeconomic status has played an important role in outcomes of AMI, the question remains: Through what mechanisms did those effects come to life? Dramatic changes in coronary heart disease (CHD) incidence and mortality have taken place since the early twentieth century. First it was described as a "disease of affluence" (Marmot and Mustard, 1994). With the increment of wealth in all industrialized countries, the disease "...reached epidemic proportions..." (Ibid.). Now-days, however, the pattern is opposite. The "... association between SES level and coronary heart diseases changed markedly from a direct to an inverse association over the past 60 years" (Beaglehole, 1990). This raises a question on its own: How have the dramatic changes between SES level and coronary heart disease come about? And why - despite all the changes in health and social environment – does the SES gradient in mortality persist? Many complementing explanations have been proposed to date, but none completely explains the persistent association. Genetics, behavioral risk factors, social environment and social isolation, stress, etc. – all these factors definitely play a role in the development of CHD. Link et al (1998) cite Susser, Watson, and Hopper (1985) on one of the principles of social epidemiology: "...Societies in part create the disease they experience and, further, they materially shape the way in which diseases are to be experienced". This principle emphasize the fact, that "...the connection between social conditions and disease are dynamic – social forces actively create and shape patterns of disease" (Link et al., 1998). Social conditions include not only monetary wealth and its distribution within the society, but also knowledge, power, and prestige, etc. Individuals who possess "more" can use their assets to "... their advantage to avoid risk and to adopt protective strategies that enhance health and well-being" (Ibid.). These

individuals are able to modify their exposure to risk factors (e.g. smoking) when such knowledge emerges and to strengthen the effect of protective factors. Also, being better informed about the newest treatments, those who hold more resources have also an improved access to the newest technologies and therapies (although the latter should have less influence in Canadian versus U.S. health care system). Many studies have shown greater reduction of CHD among more affluent members of the society by a more rapid and dramatic reduction of risk behaviors (i.e. smoking) and adhesion to beneficial practices (exercise, low fat diet, etc), when such knowledge emerges. A higher prevalence of smoking, inactivity, unbalanced diet, alcohol abuse, etc. among lower-income populations is well documented, as well as their increased risk of having multiple risk-factors (Johansen et al., 1998; Kaplan and Keil, 1993).

This mechanism of SES patterns in CHD seems even more compelling when compared to another possible explanation. The role of social status was explored by Evans et al, 1994; Marmot et al., 1999; McEwen (1999). They argued that the advantage of being at the top of the social hierarchy and the stress of being at the bottom produce different effects on the immune and nervous systems. The damaging effects of chronic stress of those at the lowest social level, in turn, make them more susceptible to diseases. Several studies on primates also confirmed the difference in physiological response to stress depending on social status of animals (Shively and Clarkson, Shively et al., Sapolsky et al., 1999). This biological mechanism of SES differences, however, does not explain the direct association between SES and coronary heart disease in the early 20th century. If having a higher status is inherently beneficial, this must have been a case in the past as well, and this theory needs to be supplemented with other explanations.

Therefore, social epidemiology provides a better understanding of the dynamic association between SES and CHD. "...SES patterns in exposure to risk and protective factors and SES patterns in morbidity and mortality will change when there are historical shifts in the profile of diseases, treatments, risk factors, and knowledge thereof" (Link et al., 1998). An effective health innovation, such as a new procedure, can become a mechanism that links SES to mortality: the implementation of every new procedure is taking place within society, with its inequality in many aspects: wealth, power, knowledge, social network, etc. Various strata of society become differently exposed to the new procedure, and therefore, this fact creates new trends in rates of disease, clinical characteristics, and mortality. Thus, the persistent SES gradient in the invasive cardiac procedures, such as coronary artery bypass graft surgery, may become a new mechanism linking SES to AMI, for instance, by reducing (or delaying) re-infarction. And, although the overall rates of cardiac procedures, such as coronary angioplasty or CABG, are steadily rising in Canada, the population-based studies in Manitoba and Ontario have demonstrated the persisting SES gradient in mortality and in revascularization. Alter et al. (1999) reported that "...Ontario residents living in low-income areas have reduced access to invasive procedures, as compared with residents of wealthier neighborhoods, and have sharply higher mortality one year after hospitalization for AMI".

The introduction of new drug therapies may also result in similar mechanisms linking mortality and SES. By receiving beneficial drugs at higher rates (as was found in this and other studies), more affluent patients may experience improved outcomes, therefore, widening the gap in mortality between different SES groups. Possible clinical reasons for not administering particular treatments among lower income strata include:

poorer overall health status, higher rates of comorbidities and clinical contraindications, more advanced age, delay in hospitalization, problems of compliance with treatments, etc. However, this study found an independent contribution of SES to AMI mortality while controlling for various demographic and clinical variables; those from the higher income neighborhoods had much improved survival, compared to the lower-income subjects.

Levels of social inequality, however, are responsive to policy. For example, attention to the questions of differences in clinical evaluation and management of CHD in women resulted in positive changes in clinical practice. Roos et al. (1998) examined the technological changes in AMI treatment in Manitoba between 1977 and 1996 and found " a steady increase " in rates of cardiac procedures. Furthermore, "angiography and PTCA rates for women increased substantially more than those for men (46% versus 21% for angiography and 106% versus 53% for PTCA)". This is only one instance substantiating the claim that levels of inequality can, to some degree, be combated by the change of policy.

Although policy changes do have an impact on access to treatment, new procedures etc., and although for all persons affected by some disease equal treatment is expected according to policy, it is nevertheless the case that inequalities in the application of policy prevail. But if inequalities in the application of measures prescribed by policy are not due to policy itself because it ought to hold for all afflicted persons, then the reasons for inequality have to be found somewhere else. Social status seems to present itself as a factor that generates inequalities of policy application if (and only if) one and the same policy holds for the whole of a population.

There seems to be a discrepancy between the "ought" of policy – prescribed procedures, treatments etc. for all people equally – and the "is" of reality, which is described and not prescribed. The discrepancy between the "ought" (what is prescribed) of policy and the "is" (what is described as being the case in reality) seems to be generated by socioeconomic determinants.

The impact of socioeconomic factors on the realization of prescribed policy measures may in the future be lessened, for instance, by the introduction of care maps, which are currently being developed by the Winnipeg Regional Health Authority. Care maps are supposed to include prescribed measures (such as appropriate diagnostic and treatment procedures) in form of a list the conjunction of which will have to be followed as a "package deal" for all patients that have the disease regardless of socioeconomic or other circumstances. The care maps may be applied only to selected diseases because not all conditions are suited to be treated via unified measures of care maps. And AMI is the disease that suits perfectly the standardization of treatment via care maps.

7.3 Administrative versus clinical data

Population-based administrative data have numerous advantages for conducting population health research, such as generating a denominator for calculating rates, a relatively large number of cases, and information for calculating comorbidity. They provide a great deal of convenience in using existing information without embarking on a complex and lengthy process of data collection. When linked with readily available demographic information (such as marital status), and neighborhood income (from the small areas used in national censuses), administrative data facilitate comparing

individuals of varying socioeconomic status as to rates of disease, comorbidity, treatments, and such outcomes as re-admissions and mortality.

However, administrative data have the following potential drawbacks. First, they have a potential for generating a high false positive rate in coding AMI in hospital discharge abstracts (Van Walraven et al., 1990). Secondly, they may underestimate the comorbidities associated with admission for a condition such as acute myocardial infarction. Moreover, severity of the AMI may differ across socioeconomic groups; such severity is not captured in administrative data. This may produce biased estimates of the differences in health status associated with differences in income, since both the severity of the AMI and the comorbidities associated with the disease might be associated with socioeconomic status.

Extensive administrative and clinical data for a sample of Winnipeg AMI patients have made it possible to explore data quality in detail. The strength of this unique data set permitted comparing the accuracy of predictive models constructed using administrative and clinical data for predicting 30-day, 1-and 5-year mortality were. The false-positive coding of AMI as the most responsible diagnosis in this study was 5.4%, which is in line with the results reported by Meehan et al., 1995, Cox et al., 1997, or Osler et al., 1999. The comorbidities that influence AMI survival the most were reported similarly in both sources of data, while others were reported less frequently in the administrative data. The prediction algorithms based on information generated from administrative data were very similar to the ones derived from the clinical data; this corresponds with the findings of several earlier mentioned works (by Humphries et al, 2000; Tu et al., 2000). Therefore, the administrative data should be considered providing fairly reliable information on

comorbidities; such data are a valuable instrument in facilitating population-based health research.

7.4 Strengths and Limitations

The large comprehensive database utilized in this study was a major strength. Only a few studies have used both administrative data and chart review as the sources of information. The large number of clinical variables explored is another important advantage of this study. A vast collection of information on clinical presentation, laboratory test results, diagnostic procedures, and treatments of AMI, not routinely available in health research, was used. The previously validated mortality prediction models based on administrative data were supplemented with important clinical variables. That resulted in the enhanced control of covariables influencing the outcomes of AMI; nevertheless, an independent effect of SES on AMI mortality was identified. Furthermore, restriction of the study population to only Winnipeg residents allowed minimizing of inter-regional differences in diagnostic and treatment practices, and removing the bias arising from the differences in urban versus rural settings.

Several limitations apply to this research. The study population includes patients hospitalized and treated for acute myocardial infarctions during a one-year period, and does not include AMI cases not making it to the hospital. Therefore, the work does not present a true annual incidence of AMI and does not provide numerators for the calculation of rates. Because the population is limited to Winnipeg residents only, the analysis can not be reliably generalized to the whole Manitoba population.

Furthermore, the index hospitalization occurred in 1991-92, and treatment practices have changed dramatically since. The algorithm for the routine use of early thrombolytic therapy has been implemented; a wider use of beta-blockers has been encouraged, and so forth. The results of this study may not represent current trends in treatment of AMI or the SES difference in utilization rates of drug therapies for AMI. Saying that, however, does not undermine the fact that gradients in utilization of drug therapies and cardiac procedures, as well as differences in outcomes are likely to persist over time, despite increases in overall rates of utilization of medical procedures.

Finally, while several studies reported have correlations between aggregate (using small area-based measures) and individual measures of socioeconomic status, finding that both types of measures were similarly associated with the health outcomes (Krieger, 1992), this study can not separate the ecological and individual effects of socioeconomic status.

7.5 Conclusions

This study has focused on the issues related to clinical characteristics and outcomes of AMI and their association with patients' socioeconomic characteristics. Socioeconomic status has been confirmed as a significant factor independently contributing to the outcomes of AMI. Socioeconomic status appears important in shaping the overall health status of individuals, evaluated on the basis of the presence of comorbid conditions measured during the index hospitalization for AMI. Also significant, as emphasized by previous researches, was the impact of marital status on survival after

AMI; the mortality was significantly higher among unmarried patients both shortly after AMI and long-term.

Some deviations from equality in access to treatment were detected as well. After admission to hospital with AMI, lower income patients received several standard treatment and diagnostic interventions at a lesser rate than their more affluent counterparts.

The results of this study are very important for several reasons. Other studies have examined the relationship between SES and AMI mortality controlling for demographic and clinical covariates derived from the administrative sources. This study is unique in the range of variables examined (several hundred) and in using both administrative and clinical data as a source of information. The data permitted the inclusion of significant clinical information not otherwise available into multivariate statistical models. The final models using demographic variables, clinical risk factors, and socioeconomic data generated ROC statistics as high as 0.83-0.87 for mortality outcomes. Even after such rigorous control of covariates, the significant survival advantage of those from the high-income groups was evident.

From a research prospective, many questions remain. There is a need to account for individual characteristics such as education, since considerable evidence points to an even stronger relationship between health outcome and measure of SES by education than by income. As discussed in chapter one, incorporating both community and individual characteristics would allow detecting different aspects of SES. Finally, incorporating some measure of social isolation would be of additional value. All these factors are important in producing the SES gradient in AMI mortality, and to evaluate

their relative importance could be a next step in the understanding (with the goal of reducing) socioeconomic inequalities in AMI and associated outcomes.

REFERENCES

Alter DA, Naylor CD, Austin P, Tu JV. Effects of Socioeconomic Status on Access to Invasive Cardiac Procedures and on Mortality after Acute Myocardial Infarction. N Engl J Med 1999; Vol. 341, No 18:1359-67.

Anderson GM, Grumbach K, Luft HS, Roos LL, Mustard C, Brook R. Use of Coronary Artery Bypass Surgery in the United States and Canada. JAMA, 1993; Vol 269, No 13:1661-1666.

Ayanian JZ, Hauptman PJ, Guadagnoli E, Antman EM, Pashos CL, McNeil BJ. Knowledge and Practices of Generalist and Specialist Physicians Regarding Drug Therapy for Acute Myocardial Infarction. N Engl J Med 1994; Vol. 331, No 17:1136-42.

Beaglehole R. International Trends in Coronary Heart Disease Mortality, Morbidity, and Risk Factors. Epidemiologic Reviews, 1990; Vol. 12: 1-16.

Brophy JM. The Epidemiology of Acute Myocardial Infarction and Ischemic Heart Disease in Canada. Can J Cardiol 1997; 13:474-78.

Carol JJ et al. Why do Women Receive Thrombolysis Less Often for Acute Myocardial Infarction? Circulation 1993; 88:2737-42.

Case RB, Moss AJ, Case N, McDermott M, Eberly S. Living Alone after Myocardial Infarction. Impact on Prognosis. JAMA, 1992, Vol. 267, No 4:515-519.

Charlson ME, Pompei P, Ales KL, MacKenzie CR. A New Method of Classifying Prognostic Comorbidity in Longitudinal Studies: Development and Validation. J Chron Dis., 1987; Vol.40, No 5:373-383.

Concato J, Feinstein AR. Monte Carlo Methods in Clinical Research: Applications in Multivariable Analysis. J Investig Med., 1997; Vol.45, No 6:394-400.

Coordinated Myocardial Infarction Registry Workshop (notes), held on February 25, 1996, Toronto, Ontario.

Cox JL, Chen E, Naylor CD. Revascularization after Acute Myocardial Infarction: Impact of Hospital Teaching Status and On-site Invasive Facilities. J of Gen Int Med, 1994: Vol. 9: 674-678.

Cox JL, Melady MP, Chen E, Naylor CD. Towards Improved Coding of Acute Myocardial Infarction in Hospital Discharge Abstracts: A Pilot Project. Can J Cardiol, 1997; Vol. 13, No 4: 351-358.

Daly MC, Duncan GJ, Kaplan GA, Lynch JW. Macro-to-Micro Links in the Relation between Income Inequality and Mortality. The Milbank Quarterly, 1998: Vol. 76, No 3: 315-339.

Ellerbeck EF, Jencks SF, Radford MJ, Kresowik TF, Craig AS, Gold JA, et al. Quality of Care for Medicare Patients with Acute Myocardial Infarction. JAMA, 1995; Vol. 273: 1509-1514.

Evans RG, Barer ML, Marmor TR (editors). Why are Some People Healthy and Others Not? Aldine De Gruyter, New York, 1994.

Frasure-Smith N et al. Randomized Trial of Home-based Psychosocial Nursing Intervention for Patients Recovering from Myocardial Infarction. Lancet 1997; 350:473-9

Hartford K, Roos LL, Walld R. Regional Variations in Angiography, Coronary Artery Bypass Surgery, and Percutaneous Transluminal Coronary Angioplasty in Manitoba, 1987 to 1992. The Funnel Effect. Med Care, 1998; Vol.36:1022-1032.

Health Canada-Statistics Canada-Canadian Institute for Health Information (1999). Statistical Report on the Health of Canadians. Toward a Healthy Future: Second Report on the Health of Canadians.

Humphries KH, Rankin JM, Carere RG, Buller CE, Kiely FM, Spinelli JJ. Co-morbidity Data in Outcomes Research. Are Clinical Data Derived from Administrative Databases a Reliable Alternative to Chart Review? J Clin Epidemiol 2000; Vol. 53: 343-349.

Jagal SB et al. Sex Differences in the Use of Invasive Coronary Procedures in Ontario. Can J Cardiol 1994; 10: 239-44.

Johansen H, Nargundkar M, Nair C, Taylor G, ElSaadany S. At Risk of First or Recurring Heart Disease. Health Reports, spring 1998, Vol. 9, No 4: 19-29.

Johansen H, Nair C, Taylor G. Current and Future Hospitalization after Heart Attack. Health Reports, autumn 1998; Vol. 10, No 2: 21-28.

Kaplan GA, Keil JE. Socioeconomic Factors and Cardiovascular Disease: A Review of the Literature. Circulation, 1993; Vol. 88, No 4, Part 1:1973-1988.

Katz SJ, Hofer TP, Manning WG. Hospital Utilization in Ontario and the United States: The Impact of Socioeconomic Status and Health Status. Can J Pub Health, July-August 1996; Vol. 87 N0 4: 253-56.

Kawachi I, Colditz GA, Ascherio A, Rimm EB, Giovannucci E, Stampfer MJ, Willett WC. A Prospective Study of Social Networks in Relation to Total Mortality and

Cardiovascular Disease Incidence in Men in the United States. J Epidemiol Comm Health 1996; Vol.50: 245-251.

Kawachi I, Kennedy BP, Wilkinson RG (editors). The Society and Population Health Reader. Volume 1: Income Inequality and Health. The New Press, New York 1999.

Krieger N. Overcoming the Absence of Socioeconomic Data in Medical Records: Validation and Application of a Census-Based Methodology. Am J Public Health, 1992; Vol. 82, No 5: 703-710.

Krieger N, Williams DR, Moss NE. Measuring Social Class in US Public Health Research: Concepts, Methodologies, and Guidelines. Ann. Rev. Public Health, 1997; Vol. 18: 341-378.

Krieger N. Letter to the Editor. Re: "Use of Census-Based Aggregate Variables to Proxy for Socioeconomic Group: Evidence from National Samples" in Am J Epidemiol, 1999; Vol.150, No 8:892-894.

Krumholz HM, Chen J, Wang Y, Radford MJ, Chen YT, Marciniak T. Comparing AMI Mortality Among Hospitals in Patients 65 years of Age and Older: evaluating Methods of Risk Adjustment. Circulation, 1999: Vol. 99, No 23: 2986-2992.

Last JA. Dictionary of Epidemiology, 3rd edition. Oxford University Press, New York, 1995.

Lee KL, Woodlief LH, Topol EJ, Weaver WD, Betriu A, Col J, Simoons M, Aylward P, Van de Werf F, Califf RM. Predictors of 30-Day Mortality in the Era of Reperfusion for Acute Myocardial Infarction. Results from an International Trial of 41,021 Patients. GUSTO-I Investigators. Circulation, 1995; Vol. 91, No 6: 1659-1668.

Lenfant C. Conquering Cardiovascular Disease. Progress and Promise. JAMA 1999; Vol. 282, No 21.

Liberatos P, Link BG, Kelsey JL. The Measurement of Social Class in Epidemiology. Epidemiologic Reviews, 1988; Vol. 10: 87-121.

Link BG, Northridge ME, Phelan JC, Ganz ML. Social Epidemiology and the Fundamental Cause Concept: On the Structuring of Effective Cancer Screens by Socioeconomic Status. The Milbank Quarterly, 1998: Vol. 76, No 3: 375-402.

Mant J, Hicks N. Detecting Differences in Quality of Care: The Sensitivity of Measures of Process and Outcome in Treating Acute Myocardial Infarction. BMJ 1995; 311: 793-6.

Marciniak TA, Ellebreck EF et al. Improving the Quality of Care for Medicare Patients with Acute Myocardial Infarction: Results from Cooperative Cardiovascular Project. JAMA 1998; 279:1351-57.

Mark DB, Shaw LK, DeLong ER, Califf RM, Pryor DB. Absence of sex bias in the referral of patients for cardiac catheterization. N Engl J Med 1994; Vol. 330:1101-06.

Mark DB, Naylor CD, Hlatky MA, Califf RM, Topol EJ, Granger CB et al. Use of Medical Resources and Quality of Life after Acute Myocardial Infarction in Canada and the United States. N Engl J Med 1994; Vol. 331:1130-35.

Marmot MG, Mustard JF. Coronary Heart Disease from a Population Perspective. In Why are Some People Healthy and Others Not? Aldine De Gruyter, New York, 1994, edited by Evans RG, Barer ML, Marmor TR. Chapter 7, 189-214.

Marmot MG, Kogevinas M, Elston MA. Social/Economic Status and Disease. Ann. Rev. Public Health, 1987, Vol. 8:111-135.

Marmot MG, Wilkinson RG (Editors). Social Determinants of Health. Oxford University Press, New York, 1999.

Maynard C, Every NR, Weaver WD. Factors Associated with Rehospitalization in Patients with Acute Myocardial Infarction. Am J Cardiol 1997; 80:777-779.

McEwen BS. Protective and Damaging Effects of Stress Mediators. Chapter 32, 379-392 in The Society and Population Health Reader. Volume 1: Income Inequality and Health. The New Press, 1999, edited by Kawachi I, Kennedy BP, Wilkinson RG.

McLaughlin TJ, Soumerai SB, Willison DJ, Gurwitz JH, Borbas C, Guadagnoli E, et al. Adherence to National Guidelines for Drug Treatment of Suspected Acute Myocardial Infarction. Arch Intern Med., 1996; Vol.156: 799-805.

Meehan TP, Hennen J, Radford MJ, Petrillo MK, Ballard DJ. Process and Outcome of Care for Acute Myocardial Infarction among Medicare Beneficiaries in Connecticut: A Quality Improvement Demonstration Project. Ann Intern Med, 1995; Vol. 122, No 12: 928-936.

Morgenstern H. Ecological Studies in Epidemiology: concepts, principles, and methods. Ann Rev Public Health, 1995; Vol.16: 61-81.

Murray RP, Silversides CK, Dukas J, Bond R, Fisher ES, Tate RB, Wright B, Viring B, Plohman J. Five Year Survival of Ideal Candidates vs. Clinical Candidates for Medical Therapies for Acute Myocardial Infarction. (2000, unpublished).

Mustard CA, Frohlich N. Socioeconomic Status and the Health of the Population. Med. Care, 1995; Vol. 33, No 12, DS43-DS54 *Supplement.*

Naylor CD, Slaughter PM (editors). Cardiovascular Health & Services in Ontario: An ICES Atlas. Toronto, Institute for Clinical Evaluative Sciences & Heart and Stroke Foundation, 1999.

Naylor CD. Text of Speech Launching Cardiovascular Health & Services in Ontario: An ICES Atlas, 23.02.1999.

Naylor CD et al. Placing Patient in Queue for Coronary Surgery: Do Age and Work Status Alter Canadian Specialists' Decisions? J Gen Int Med 1992; 7:492-98.

Osler M, Rostgaard K, Sorensen TIA, Madsen M. The Effect of Recurrent Events on Register-Based Estimates of Level and Trends in Incidence of Acute Myocardial Infarction. J Clin Epidemiol 1999, Vol. 52, No 7, 595-600.

Peltonen M, Rosen M, Lundberg V, Asplund K. Social Patterning of Myocardial Infarction and Stroke in Sweden: Incidence and Survival. Am J Epidemiol 2000; Vol. 151:283-92.

Phillips KA, Shlipak MG, Coxson P, Heidenreich PA, Hunink MGM, Goldman PA, Williams LW, Weinstein MC, Goldman L. Health and Economic Benefits of Increased Beta-Blocker Use Following Myocardial Infarction. JAMA, 2000; Vol. 284, No.21: 2748-2754.

Romano PS, Zach A et al. The California Hospital Outcomes Project: Using Administrative data to Compare Hospital Performance. Comm J Qual Improve 1995; 21:668-682.

Roos LL, Gelskey DE. Exploration of Cardiovascular Surgery Outcomes in Manitoba and New England. Can J Card, 1993; Vol. 9:1860.

Roos LL, Walld R, Soodeen RA, Roos NP. Health Reform and Technological Changes in Manitoba: Treatment of Acute Myocardial Infarction. 15.12.1998.

Roos NP, Mustard CA. Variation in Health and Health Care Use by Socioeconomic Status in Winnipeg, Canada: Does the System Work Well? Yes and No. The Milbank Quarterly, 1997; Vol. 75, No 1: 89-111.

Ruberman W, Weinblatt E, Goldberg JD, Chaudhary BS. Psychosocial Influences on Mortality after Myocardial Infarction. N Engl J Med 1984; Vol. 311 No 9:552-559.

Ruberman W. Psychosocial Influences on Mortality of Patients with Coronary Heart Disease (editorials). JAMA, 1992, Vol.267, No 4:559-560.

Ryan TJ, Anderson JL et al. ACC/AHA Guidelines for the Management of Patients with Acute Myocardial Infarction. J Am Coll Cardiol 1996; 28:1328-1428.

Ryan TJ, Graham TP, Annas GJ, DeMaria AN, Fost NC, Fuster V et al. Task Force III: Perspectives on the Allocation of Limited Resources in Cardiovascular Medicine. J Am Coll Cardiol, 1990; Vol. 16, No 1: 17-23.

Sapolsky RM, Alberts SC, Altmann J. Hypercortisolism Associated with Social Subordinance and Social Isolation among Wild Baboons. Chapter 35, 421-432 in The Society and Population Health Reader. Volume 1: Income Inequality and Health. The New Press, 1999, edited by Kawachi I, Kennedy BP, Wilkinson RG.

Schwartz LM, Fisher ES, Tosteson ANA, Woloshin S, Chang CH, Virnig BA, Plohman J, Wright B. Treatment and Health Outcomes of Women and Men in a Cohort With Coronary Artery Disease. Arch Intern Med. 1997; Vol. 157:1545-1551.

Shively CA, Clarcson TB. Social Status and Coronary Artery Atherosclerosis in Female Monkeys. Chapter 33, 393-404 in The Society and Population Health Reader. Volume 1: Income Inequality and Health. The New Press, 1999, edited by Kawachi I, Kennedy BP, Wilkinson RG.

Shively CA, Laber-Laird K, Anton RF. Behavior and Physiology of Social Stress and Depression in Female Cynomolgus Monkeys. Chapter 34, 405-420 in The Society and Population Health Reader. Volume 1: Income Inequality and Health. The New Press, 1999, edited by Kawachi I, Kennedy BP, Wilkinson RG.

Smith GD, Hart C, Watt G, Hole D, Hawthorne V. Individual Social Class, Area-based Deprivation, Cardiovascular Disease Risk Factors, and Mortality: the Renfrew and Paisley Study. J Epidemiol Community Health 1998; 52:399-405.

Smith GD. Income Inequality and Mortality: Why are they related? BMJ 1996; Vol. 312: 987-988.

Stamler J, Stamler R, Neaton JD, Wentworth D, Daviglus ML, Garside D, Dyer AR, Liu K, Greenland P. Low Risk-Factor Profile and Long-term Cardiovascular and Noncardiovascular Mortality and Life Expectancy. JAMA 1999; Vol. 282, No 21.

Topol E et al. (The GUSTO Investigators). An International Randomized Trial Comparing Four Thrombolytic Strategies for Acute Myocardial Infarction. N Engl J Med, 1993; Vol. 329, No 10: 673-682.

Tu JV, Austin P, Naylor CD, Iron K, Zhang H. Acute Myocardial Infarction Outcomes in Ontario. In Naylor CD, Slaughter PM (eds.). Cardiovascular Health & Services in Ontario: An ICES Atlas. Toronto, Institute for Clinical Evaluative Sciences & Heart and Stroke Foundation, 1999:83-110.

Tu JV, Austin P, Naylor CD. Technological Changes in Heart Attack Care in Ontario, Canada, 1981 to 1995. Presented at the International Conference on Technological Change and Medical Expenditure Growth, feb.26-28, 1998.

Tu JV, Pashos CL, Naylor CD, Chen E, Normand SL, Newhouse JP, McNeil BJ. Use of Cardiac Procedures and Outcomes in Elderly patients with Myocardial Infarction in the United States and Canada. N Engl J Med, 1997; Vol. 336, No 21:1500-1505.

Tu JV, Sykora K, Naylor CD. Assessing the Outcomes of Coronary Bypass Graft Surgery: How Many Risk Factors Are Enough? J Am Coll Cardiol, 1997; Vol. 30, No 5: 1317-1323.

Tu JV, Wu K. The Improving Outcomes of Coronary Artery Bypass Graft Surgery in Ontario, 1981 to 1995. CMAJ, 1998; Vol. 159, No 3: 221-227.

Tu JV, Austin PC, Walld R, Roos LL, Agras J, McDonald KM. Development and Validation of the Ontario Acute Myocardial Infarction Mortality Prediction Rules. J Am Coll Cardiol, 2001; Vol.37, March 15, No 4.

Udvarhelyi IS, Gatsonis C, Epstein AM, Pashos CL, Newhouse JP, McNeil BJ. Acute Myocardial Infarction in the Medicare Population. Process of Care and Clinical Outcomes. JAMA, November 11, 1992-Vol 268, No 18:2530-2536.

Van Walraven C, Wang B, Ugnat AM, Naylor CD. False-Positive Coding for Acute Myocardial Infarction on Hospital Discharge Records: Chart Audit Results from a Tertiary Centre. Can J Cardiol, 1990; Vol. 6, No 9: 383-386.

Williams RB, Barefoot JC, Califf RM, Haney TL, Saunders WB, Pryor DB, Hlatky MA, Siegler IC, Mark DB. Prognostic Importance of Social and Economic Resources among Medically Treated Patients with Angiographically Documented Coronary Artery Disease. JAMA, 1992; Vol. 267, No 4:520-524.

Young TK. Population Health. Concepts and Methods. Oxford University Press, New York, 1998.

APPENDIX ONE

Codes For Acute Myocardial Infarctions

International Classification of Diseases, 9th Revision, Clinical Modifications

ISCHEMIC HEART DISEASE (410-414)

Includes: that with mention of hypertension

Use additional code to identify presence of hypertension (401.0-405.9)

➡ AHA Coding Clinic: J-A, '84, 5

410 Acute myocardial infarction A: 15–124

➡ **Def:** A sudden insufficiency of blood supply to an area of the heart muscle; it is usually due to a coronary artery occlusion.

Includes: cardiac infarction
coronary (artery):
embolism
occlusion
rupture
thrombosis
infarction of heart, myocardium, or ventricle
rupture of heart, myocardium, or ventricle
any condition classifiable to 414.1-414.9 specified as acute or with a stated duration of 8 weeks or less

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

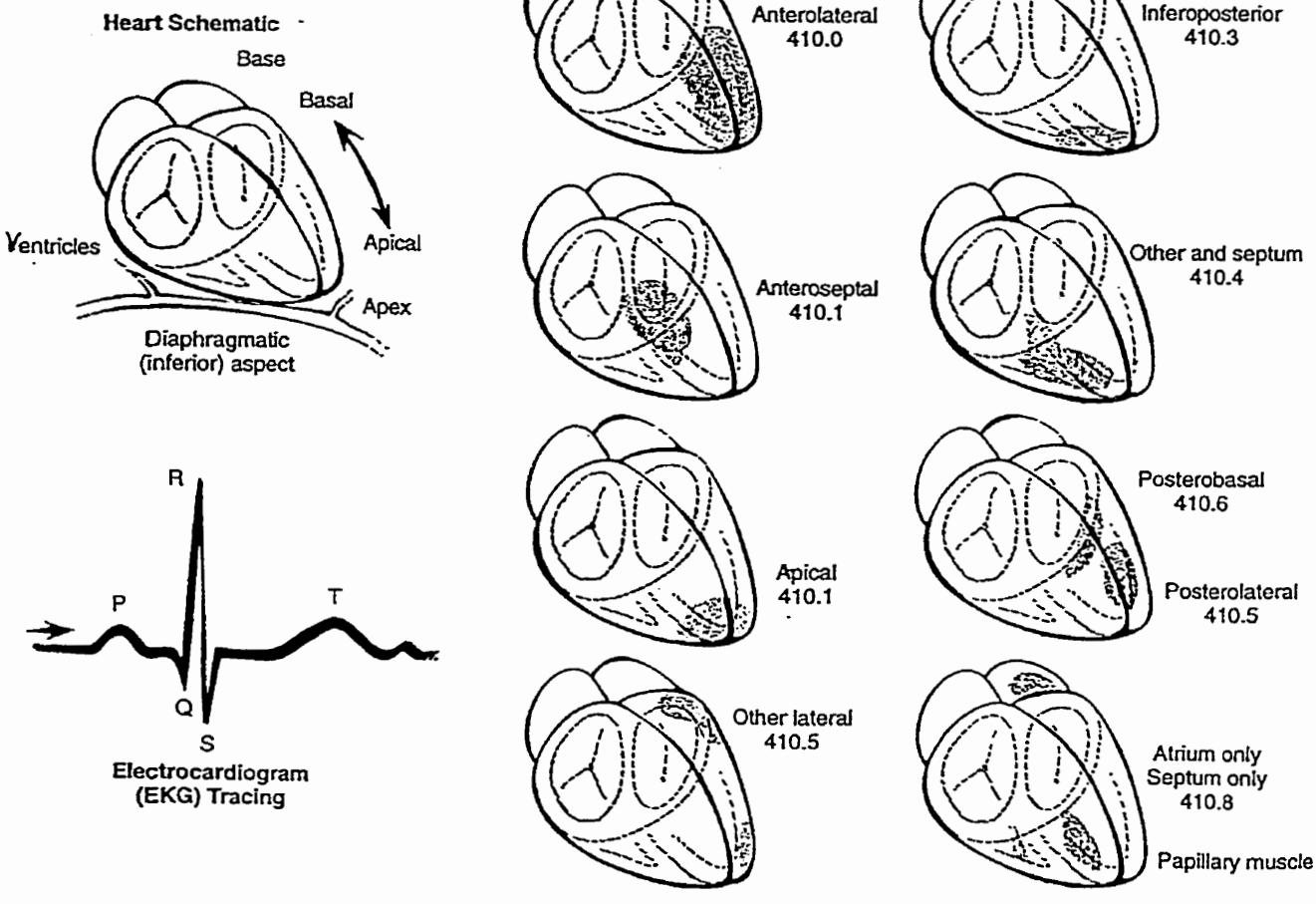
0 episode of care unspecified
Use when the source document does not contain sufficient information for the assignment of fifth digit 1 or 2.

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.

➡ AHA Coding Clinic: 3Q, '98, 15; 4Q, '97, 37; 3Q, '95, 9; 4Q, '92, 24; 1Q, '92,10; 3Q, '91, 18; 1Q, '91, 14; 3Q, '89, 3

Acute Myocardial Infarction



J. Uhanova

✓5th **410.0** **Of anterolateral wall**

➡ **CC Excl: For code 410.01:** 410.00-410.92, 459.89, 459.9

✔ **Clinical Coder: For code 410.01:** see Chapter 5

✓5th **410.1** **Of other anterior wall**

Infarction:
anterior (wall) NOS
anteroapical
anteroseptal
} (with contiguous portion of intraventricular septum)

➡ **CC Excl: For code 410.11:** see Code 410.0

▽ **DRG 121 and DRG 122: For code 410.11**

✔ **Clinical Coder: For code 410.11:** see Chapter 5

✓5th **410.2** **Of inferolateral wall**

➡ **CC Excl: For code 410.21:** see Code 410.0

✔ **Clinical Coder: For code 410.21:** see Chapter 5

✓5th **410.3** **Of inferoposterior wall**

➡ **CC Excl: For code 410.31:** see Code 410.0

✔ **Clinical Coder: For code 410.31:** see Chapter 5

✓5th **410.4** **Of other inferior wall**

Infarction:
diaphragmatic wall NOS
inferior (wall) NOS
} (with contiguous portion of intraventricular septum)

➡ **CC Excl: For code 410.41:** see Code 410.0

➡ **AHA Coding Clinic:** 1Q, '00, 7, 26; 4Q, '99, 9; 3Q, '97, 10

▽ **DRG 121 and DRG 122: For code 410.41**

✔ **Clinical Coder: For code 410.41:** see Chapter 5

✓5th **410.5** **Of other lateral wall**

Infarction:
apical-lateral
basal-lateral

Infarction:
high lateral
posterolateral

➡ **CC Excl: For code 410.51:** see Code 410.0

✔ **Clinical Coder: For code 410.51:** see Chapter 5

✓5th **410.6** **True posterior wall infarction**

Infarction:
posterobasal
strictly posterior

➡ **CC Excl: For code 410.61:** see Code 410.0

✔ **Clinical Coder: For code 410.61:** see Chapter 5

✓5th **410.7** **Subendocardial infarction**

Nontransmural infarction

➡ **CC Excl: For code 410.71:** see Code 410.0

➡ **AHA Coding Clinic:** 1Q, '00, 7

▽ **DRG 121 and DRG 122: For code 410.71**

✔ **Clinical Coder: For code 410.71:** see Chapter 5

✓5th **410.8** **Of other specified sites**

Infarction of:
atrium
papillary muscle
septum alone

➡ **CC Excl: For code 410.81:** see Code 410.0

✔ **Clinical Coder: For code 410.81:** see Chapter 5

5th **410.9** **Unspecified site**

Acute myocardial infarction NOS
Coronary occlusion NOS

➡ **CC Excl: For code 410.91:** see Code 410.0

➡ **AHA Coding Clinic:** 1Q, '92, 9

▽ **DRG 121 and DRG 122: For code 410.91**

✔ **Clinical Coder: For code 410.91:** see Chapter 5

411 **Other acute and subacute forms of ischemic heart disease** A: 15–124

➡ **AHA Coding Clinic:** 4Q, '94, 55; 3Q, '91, 24

411.0 **Postmyocardial infarction syndrome**

➡ **Def:** A complication developing several days/weeks after myocardial infarction; it is characterized by fever, leukocytosis, chest pain, evidence of pericarditis, pleurisy and pneumonitis, and has a tendency to recur.

Dressler's syndrome

➡ **CC Excl:** 411.0, 411.81, 411.89, 459.89, 459.9

✔ **Clinical Coder:** see Chapters 5, 6 & 7

411.1 **Intermediate coronary syndrome**

➡ **Def:** Intermediate Coronary Syndrome: A condition representing an intermediate stage between angina of effort and acute myocardial infarction. It is often documented by the physician as "unstable angina."

➡ **Def:** Impending infarction: An acute increase in anginal symptoms including severe spasmodic substernal pain radiating down the left arm, usually preceding imminent infarction.

➡ **Def:** Preinfarction angina: Anginal symptoms that occur at rest and usually are refractory to treatment.

➡ **Def:** Preinfarction syndrome: The array of symptoms that precede impending infarction. Symptoms include chest pain, shortness of breath, palpitations, fatigue, vomiting and paresthesis.

➡ **Def:** Unstable angina: Sudden increase in frequency, duration and severity of anginal symptoms, usually without provocation.

Impending infarction
Preinfarction angina
Preinfarction syndrome
Unstable angina

Excludes: *angina (pectoris) (413.9)*
decubitus (413.0)

➡ **CC Excl:** 410.00-410.92, 411.1-411.89, 413.0-413.9 414.8-414.9, 459.89, 459.9

➡ **AHA Coding Clinic:** 4Q, '98, 86; 2Q, '96, 10; 3Q, '91, 24; 1Q, '91, 14; 3Q, '90, 6; 4Q, '89, 10

▽ **DRG 140**

✔ **Clinical Coder:** see Chapters 5 & 7

✔ **Diagnosis Coding Advisor:** see Chapter 7

5th **411.8** **Other**

➡ **AHA Coding Clinic:** 3Q, '91, 18; 3Q, '89, 4

✔ **Clinical Coder:** see Chapters 5 & 7

APPENDIX TWO

Sample of Chart Abstract Form

MHRIS – AMI

FW318 – 685 William Avenue

Winnipeg, Canada

R3E 0Z2

Hospital_____HRN_____Study ID_____

1. Which of the ICD-9-CM codes or diagnoses for AMI is recorded on the admission/separation abstract, emergency record, or discharge summary?

☐ 410.0	Anteriolateral wall
☐ 410.1	Other anterior
☐ 410.2	Inferiolateral wall
☐ 410.3	Inferioposterior wall
☐ 410.4	Other inferior
☐ 410.5	Other lateral
☐ 410.6	True posterior infarction
☐ 410.7	Subendocardial infarction
☐ 410.8	Other specified sites
☐ 410.9	Unspecified sites
☐	Not applicable

If you check NOT APPLICABLE, stop abstracting and give chart to supervisor for review.

2. Is the admission date between 01/10/91 and 30/09/92?

☐ No ☐ Yes

If you check NO, stop abstracting and give chart to supervisor for review.

3. Were any of the following noted:

☐ No ☐ Yes 3a. Patient less than 45 years old

☐ No ☐ Yes 3b. Cerebrovascular accident (CVA) in the 48 hours prior to admission

☐ No ☐ Yes 3c. Multiple trauma (MVA, assault) with open wounds, other injuries or fractures as reason for admission.

If you check YES, stop abstracting and give chart to supervisor for review.

4. Give a brief description of i) the acute event on admission; ii) highlights of the hospital stay; iii) information necessary to understand the course of events; iv) cause of death (ER record, Admission note, Discharge summary)

_____

_____

_____

_____

_____

Section A: Transfers from another acute care facility
(see Transfer documentation, ER record, discharge summary)

A1. Was the patient transferred from another acute care facility?

☐ Yes
☐ No
(Go to Section B)

A2. Were they at that facility for 48 hours or more? ☐ No ☐ Yes
If YES, stop abstracting and give chart to supervisor for review.

A3. Name of transferring facility: _____

Reason for transfer: _____

A4. What documentation is available from transferring facility?

☐ 1. MD transfer summary/referral letter
☐ 2. Copies or ER record, progress notes, etc.
☐ 3. Transfer/referral form
☐ 9. No documentation available

A5. What time did the patient arrive at the transferring facility?

Date (day/month/year) ☐☐ / ☐☐ / ☐☐

Time ☐☐ : ☐☐ **24 HR Time

A6. Was the patient admitted\at the transferring facility for:

☐ No ☐ Yes Cardiac surgery or procedures (such as CABG, PTCA or valve repair)

☐ No ☐ Yes Other vascular or non-vascular surgery, procedures or tests (such as lower extremity vascular surgery or aneurysm repair)

If YES to either question, stop abstracting and give chart to a supervisor for review.

A7. Was the patient admitted\at the transferring facility for a medical condition that was definitively not cardiac in origin (e.g. fever, pneumonia):

☐ No ☐ Yes
(Go to A8)

a. Did cardiac symptoms begin in-hospital?
☐ No ☐ Yes

b. Did a physician say that the MI was either a complication or unrelated to the reason for admission?
☐ No ☐ Yes

If YES to either question, stop abstracting and give chart to a supervisor for review

(Go to Section B)

A8. When did the symptoms begin?

Date of onset (day/month/year) ☐☐ / ☐☐ / ☐☐

Time of onset ☐☐ : ☐☐ **24 HR Time

OR

Time between onset of symptoms and arrival at transferring facility:

Hours ☐☐ Days ☐☐

A9. Diagnosis/complaint at transferring facility:

- ☐ 1. Chest pain/angina/Unstable angina/Neck pain/arm pain
- ☐ 2. Dyspnea/Shortness of Breath
- ☐ 3. Nausea/stomach upset
- ☐ 4. Congestive Heart Failure
- ☐ 5. Arrythmia/cardiac arrest/syncope
- ☐ 6. MI/rule-out MI
- ☐ 8. Other

A10. First recorded physiologic variables after arrival at transferring facility:

Heart rate/min ☐☐☐

Temperature (Celsius) ☐☐.☐

Blood pressure ☐☐☐ / ☐☐☐

Respiratory rate ☐☐

A11. Were any of the following treatments/tests administered at the transferring hospital?

☐ No ☐ Yes Aspirin, ASA, ECASA, entrophen

☐ No ☐ Yes Thrombolytic Therapy

Date (day/month/year) ☐☐ / ☐☐ / ☐☐

Time ☐☐ : ☐☐ **24 HR Time

☐ No ☐ Yes Heparin

☐ No ☐ Yes EKGs adminstered

Were ST elevations or depressions noted?

☐ No ☐ Yes

☐ No ☐ Yes Patient intubated

☐ No ☐ Yes CPR/cardioversion administered

(Go to Section B)

(A11. Treatments administered at the transferring facility: continued)

☐ No (Go to Section B)

☐ Yes — Enzymes measured?

a. HIGHEST recorded LDH at transferring facility: _____

LDH1 level _____ LDH2 level _____

Date (day/month/year) ☐☐ / ☐☐ / ☐☐

Time ☐☐ : ☐☐ **24 HR Time

b. FIRST recorded CPK at transferring facility: ☐☐☐☐

Percent MB ☐☐ % or MB u/l ☐☐☐

Date (day/month/year) ☐☐ / ☐☐ / ☐☐

Time ☐☐ : ☐☐ **24 HR Time

c. HIGHEST recorded CPK at transferring facility: ☐☐☐☐

Percent MB ☐☐ % or MB u/l ☐☐☐

Date (day/month/year) ☐☐ / ☐☐ / ☐☐

Time ☐☐ : ☐☐ **24 HR Time

Section B: Admission/SeparationAbstract, Emergency Room Record, DischargeSummary, Progress Notes from First Day

B1. Arrival of patient to ER at marker hospital (Triage/Arrival time on ER record):

Date (day/month/year) ☐☐ / ☐☐ / ☐☐

Time ☐☐ : ☐☐ **24 HR Time

☐ 1. Patient admitted directly to ward

B2. Was the patient admitted\at the hospital for:

☐ No ☐ Yes Cardiac surgery or procedure such as CABG, PTCA or valve repair

☐ No ☐ Yes Other vascular or non-vascular surgery, procedure or test (such as lower extremity vascular surgery or aneurysm repair?)

If YES to either question, stop abstracting and give chart to a supervisor for review.
DO NOT RULE-OUT TRANSFER CASES HERE!!!

B3. Was the patient admitted\at the hospital for a medical condition that was definitively not cardiac origin (e.g. fever, pneumonia):

☐ No ☐ Yes

a. Did cardiac symptoms begin in-hospital?
☐ No ☐ Yes

b. Did a physician say that the MI was either a complication or unrelated to the reason for admission?
☐ No ☐ Yes

If YES to either question, stop abstracting and give chart to a supervisor for review

B4. Onset of symptoms: (If transfer patient and onset noted in A8, skip this question)

Date of onset (day/month/year) ☐☐/☐☐/☐☐
Time of onset ☐☐:☐☐ **24 HR Time

OR

Time between onset of symptoms and arrival at marker hospital:

Hours ☐☐ Days ☐☐

B5. Diagnosis/complaint at marker hospital:

☐ 1. Chest pain/angina/Unstable angina/Neck pain/arm pain
☐ 2. Dyspnea/Shortness of Breath
☐ 3. Nausea/stomach upset
☐ 4. Congestive Heart Failure
☐ 5. Arrythmia/cardiac arrest/syncope
☐ 6. MI/rule-out MI
☐ 8. Other _____

B6. Which of the following treatments were administered within 24 hours of arrival at marker hospital:

☐ No ☐ Yes Aspirin, ASA, ECASA/enteric-coated ASA, entropher

☐ No ☐ Yes Thrombolytic Therapy

Date (day/month/year) ☐☐ / ☐☐ / ☐☐
Time ☐☐ : ☐☐ **24 HR Time

☐ No ☐ Yes Heparin

☐ No ☐ Yes Cardioversion/ Defibrillation

☐ No ☐ Yes Patient intubated

B7. First recorded physiologic variables after arrival at marker hospital:
(only record if noted within 24 hours)

Heart rate/min ☐☐☐
Temperature (Celsius) ☐☐.☐
Blood pressure ☐☐☐ / ☐☐☐
Respiratory rate ☐☐☐

SECTION C: History and Physical
(see MD admission notes, ER record, RN data base/admission notes, consult notes)

C1. Did the patient have history of:

☐ No/NR ☐ Yes Previous MI

Was the most recent event within the last 8 weeks?
☐ No/NR ☐ Yes

☐ No/NR ☐ Yes History of Angina/Chest pain/other pain attributed to a cardiac origin

☐ No/NR ☐ Yes Ischemic Heart disease/IHD or Coronary Artery Disease/CAD

☐ No/NR ☐ Yes Pacemaker previously inserted (and not noted as removed)

☐ No/NR ☐ Yes Aspirin (ASA) allergy/hypersensitivity

☐ No/NR ☐ Yes Stroke/Cardiovascular Accident/CVA

☐ No/NR ☐ Yes Major surgery within last 2 weeks?

(C1- Past medical/surgical history continued)

☐ No/NR ☐ Yes	Congestive Heart Failure/ CHF
☐ No/NR ☐ Yes	Peripheral vascular disease/PVD, claudication, peripheral artery surgery
☐ No/NR ☐ Yes	Hypertension/High blood pressure/HTN
☐ No/NR ☐ Yes	Chronic Pulmonary Disease/COPD, asthma, emphysema, chronic bronchitis, restrictive airway disease
☐ No/NR ☐ Yes	Ulcer disease, include only peptic, stomach, duodenal, esophageal ulcers (whether they are on medicine or not)
☐ No/NR ☐ Yes	Chronic liver diease, chronic hepatitis, cirrhosis
☐ No/NR ☐ Yes	Genito-urinary /GU or gastrointestinal/GI bleedingwithin 1 year of arrival. (Include hemopysis or any test positive for blood in the stool)
☐ No/NR ☐ Yes	Bleeding disorder; include only chronic lymphocytic leukemia/CLL, factor VIII, myelodysplasia, esophageal varices, thrombocytopenia, hemophilia
☐ No/NR ☐ Yes	Trauma in past month; includes falls, fractures, motor vehicle accidents and head trauma
☐ No/NR ☐ Yes	Disseminated/metastatic cancer or intracranial neoplasm
☐ No/NR ☐ Yes	Other non-metastatic cancer
☐ No/NR ☐ Yes	HIV+/AIDS
☐ No/NR ☐ Yes	Renal failure, dialysis, nephritis
☐ No/NR ☐ Yes	Dementia/Alzheimer's disease/chronic confusion/senility
☐ No/NR ☐ Yes	Diabetes (any type)
☐ No/NR ☐ Yes	CABG/coronary bypass sugery
☐ No/NR ☐ Yes	PTCA (coronary angioplasty)
☐ No/NR ☐ Yes	Other cardiac surgery; includes implantable defibrillator (AICD), valve repair/replacement, aneurysmectomy, and repair of congenital or acquired heart abnormalities
☐ No/NR ☐ Yes	Terminal illness, poor prognosis, comfort measures only/CMO/CCO, do not resuscitate/DNR

C2. Was the patient's smoking status noted in the physician's history, nursing database, or MD discharge summary?

- ☐ 1. Non-smoker
- ☐ 2. Smoker
- ☐ 9. Smoking status not noted/ unclear

C3. Record all medications the patient was taking (at home) at the time of admission noted by an MD or a nurse.

_____

_____

_____

C4. Record all contraindications to thrombolysis or previous use of streptokinase noted by a physician (including consults):

_____

_____

_____

☐ 1. No contraindications to thrombolysis noted

Physician's Physical Exam notes:
(See also MD admission notes, first 24 hours of MD progress notes, consult notes in first 24 hours)

C5. Were any of the following noted:

☐ No/NR	☐ Yes	Pulmonary Edema
☐ No/NR	☐ Yes	S3
☐ No/NR	☐ Yes	Shock
☐ No/NR	☐ Yes	Rales

C6. Was the location of the current MI noted?

- ☐ 1. Anterior
- ☐ 2. Inferior
- ☐ 3. Other
- ☐ 4. MI noted but not location
- ☐ 9. No mention of an MI

C7. Was the type of the current MI noted?

- ☐ 1. Q-Wave/transmural
- ☐ 2. Non-Q/Non-transmural/Subendocardial
- ☐ 9. MI type not noted

C8. Based on the physician's reporting of EKG results, were any of the following changes noted? (DO NOT INCLUDE CHANGES NOTED AS DEFINITELY BEING OLD!)

☐ No/NR	☐ Yes	ST elevation or depression
☐ No/NR	☐ Yes	Other ST or T-wave changes
☐ No/NR	☐ Yes	Loss of R forces/reduced anterior forces/poor R-wave progression Q waves (new, significant, large)
☐ No/NR	☐ Yes	Heart block/ A-V Block

Type:
- ☐ 1. Type 1/First degree
- ☐ 2. Type 2/Second degree
- ☐ 3. Type 3/Third degree/complete
- ☐ 9. Type not noted

☐ No/NR	☐ Yes	Bundle Branch Block/BBB
☐ No/NR	☐ Yes	Ventricular Tachycardia/V-TACH
☐ No/NR	☐ Yes	Ventricular Fibrillation/ V FIB
☐ No/NR	☐ Yes	Atrial Fibrillation/A FIB
☐ No/NR	☐ Yes	Supraventricular tachycardia /SVT
☐ No/NR	☐ Yes	Ischemic Changes

C9. Was the patient described as having a decreased level of consciousness (other than in the operating room) or a coma at any time during the initial 24 hours?

☐ Yes
☐ No

(Go to Section D)

C10. Were medications administered which could have resulted in this decreased LOC?

☐ No ☐ Yes

Specify: _____

C11. Did eyes open spontaneously or to painful/verbal stimuli?

☐ No ☐ Yes

C12. Choose the worst condition from each category during the initial 24 hours

VERBAL

☐ 1. Oriented, converses
☐ 2. Confused conversation
☐ 3. Inappropriate words/incomprehensible sounds
☐ 4. No patient response
☐ 9. No data available

MOTOR

☐ 1. Obeys verbal command
☐ 2. Localizes pain
☐ 3. Flexion withdrawal/decorticate rigidity
☐ 4. Decreased rigidity/no patient response
☐ 9. No data available

SECTION D: Physician's Progress Notes and Discharge Summary

D1. Was a pacemaker implanted at any time after admission?

☐ No/NR ☐ Yes

Was it:
- ☐ 1. Permanent
- ☐ 2. Temporary

D2. Was smoking cessation discussed with the patient?

☐ No/NR ☐ Yes

D3. Was a Swan-Ganz/right heart catheter inserted?

☐ No/NR ☐ Yes

D4. Was an Intra-Aortic Balloon Pump (IABP) inserted?

☐ No/NR ☐ Yes

D5. Complications and adverse events which developed/were noted after admission
(DO NOT include complications noted at the time of admission/in section C unless worsening)

☐ No/NR ☐ Query ☐ Yes Pneumonia

☐ No/NR ☐ Query ☐ Yes Deep venous thrombosis (DVT)

☐ No/NR ☐ Query ☐ Yes Pulmonary Embolus (PE)

☐ No/NR ☐ Query ☐ Yes Cerebrovascular accident (CVA)/ Subarachnoid Hemorrhage/Intracranial hemmorage/ Stroke

Terms used to describe stroke:

☐ No/NR ☐ Query ☐ Yes New onset or worsening congestive heart failure (CHF)/Fluid overload/Pulmonary edema

☐ No/NR ☐ Query ☐ Yes Extended/New AMI

Date noted (day/month/year) ☐☐/☐☐/☐☐

☐ No/NR ☐ Query ☐ Yes Bleeding/hemorrhage (GI, GU, wound site, catheter site, does not include CVA)

☐ No/NR ☐ Query ☐ Yes Chest pain/angina (recurrent chest pain/discomfort/tightness, heaviness, ache, pressure, shoulder/neck/jaw/arm pain or any pain attributed to cardiac origin; greater than 24 hours after arrival)

☐ No/NR ☐ Yes Shock

☐ No/NR ☐ Yes Cardiac arrest (do not note if cardiac arrest happens at time of death)

☐ No/NR ☐ Yes Death

☐ No/NR ☐ Yes Other complications not listed above:

_____

_____

SECTION E: Test Reports and Procedures
(use reports only, if test conducted more than once, pick the first one)

E1. Was Coronary Angioplasty (PTCA) conducted?

☐ No ☐ Yes

Date: (day/month/year) ☐☐/☐☐/☐☐

E2. Was Coronary Artery Bypass Surgery (CABG) conducted?

☐ No ☐ Yes

Date: (day/month/year) ☐☐/☐☐/☐☐

E3. Was echocardiography reported?

☐ No ☐ Yes

a. Date: (day/month/year) ☐☐/☐☐/☐☐

b. What was the ejection fraction? _____ %

E4. Was stress testing/exercise tolerance testing reported?

☐ No ☐ Yes

a. Date: (day/month/year) ☐☐/☐☐/☐☐

b. Was the test:

- ☐ 1. Normal/negative
- ☐ 2. Inconclusive/Non-diagnostic
- ☐ 3. Abnormal/positive

E5. Was a Thallium Scan/exercise test with Thallium reported?

☐ No ☐ Yes

Date: (day/month/year) ☐☐ / ☐☐ / ☐☐

E6. Was a MUGA scan reported?

☐ No ☐ Yes

a. Date: (day/month/year) ☐☐ / ☐☐ / ☐☐

b. What was the ejection fraction? _____ %

E7. Was Angiography reported?

☐ No ☐ Yes

Date: (day/month/year) ☐☐ / ☐☐ / ☐☐

E8. Other tests or procedures?

☐ No ☐ Yes

a. Specify:	b. Date: (day/month/year)
_____	☐☐ / ☐☐ / ☐☐
_____	☐☐ / ☐☐ / ☐☐
_____	☐☐ / ☐☐ / ☐☐
_____	☐☐ / ☐☐ / ☐☐

SECTION F: EKG Reports
(See EKG reports only.
Do not include information on EKGs from MD progress notes)

F1. Check the first EKG report after arrival

Date of report (day/month/year) ☐☐/☐☐/☐☐

Time of report ☐☐:☐☐ **24 HR Time

DO NOT REPORT FINDINGS THAT ARE CLEARLY CONSIDERED TO BE ASSOCIATED WITH AN OLD EVENT

F2. Was the location of the infarct noted?

- ☐ 1. Anterior
- ☐ 2. Inferior
- ☐ 3. Other_____
- ☐ 4. MI noted but not location
- ☐ 9. No mention of an MI on EKG report

F3. Was the type of the current MI noted?

- ☐ 1. Q-Wave/transmural
- ☐ 2. Non-Q/Non-transmural/Subendocardial
- ☐ 9. MI type not noted

F4. Were any of the following noted:

☐ No/NR ☐ Yes ST elevation or depression

☐ No/NR ☐ Yes Other ST changes or T-wave changes

☐ No/NR ☐ Yes Loss of R forces/reduction of anterior forces/ poor R wave progression, Q waves (new, significant, large)

☐ No/NR ☐ Yes Heart block/ A-V Block

Type:
- ☐ 1. Type 1/First degree
- ☐ 2. Type 2/Second degree
- ☐ 3. Type 3/Third degree/complete
- ☐ 9. Type not noted

☐ No/NR ☐ Yes Bundle Branch Block/BBB

F5. Check the last EKG report

Date of report (day/month/year) ☐☐/☐☐/☐☐

Time of report ☐☐:☐☐ **24 HR Time

F6. Was the location of the infarct noted?

- ☐ 1. Anterior
- ☐ 2. Inferior
- ☐ 3. Other _____
- ☐ 4. MI noted but not location
- ☐ 9. No mention of an MI on EKG report

F7. Was the type of the current MI noted?

- ☐ 1. Q-Wave/transmural
- ☐ 2. Non-Q/Non-transmural/Subendocardi
- ☐ 9. MI type not noted

F8. Were any of the following noted:

☐ No/NR ☐ Yes ST elevation or depression

☐ No/NR ☐ Yes Other ST changes or T-wave changes

☐ No/NR ☐ Yes Loss of R forces/reduction of anterior forces/ poor R wave progression, Q waves (new, significant, large)

☐ No/NR ☐ Yes Heart block/ A-V Block

Type:
- ☐ 1. Type 1/First degree
- ☐ 2. Type 2/Second degree
- ☐ 3. Type 3/Third degree/complete
- ☐ 9. Type not noted

☐ No/NR ☐ Yes Bundle Branch Block/BBB

SECTION G: Laboratory Test Results
(See laboratory reports only. Do NOT report lab data found in other sources)

G1. First recorded CPK measurement ☐☐☐☐

☐☐ %MB or ☐☐☐ MB u/l

Date of first measure : ☐☐/☐☐/☐☐
(day/month/year)

Time ☐☐:☐☐ **24 HR Time

Check here if first CPK was highest ☐ or only measure ☐

G2. Highest recorded CPK measurement (if multiple peaks, record first peak): ☐☐☐☐

☐☐ %MB or ☐☐☐ MB u/l

Date of high measure : ☐☐/☐☐/☐☐
(day/month/year)

Time ☐☐:☐☐ **24 HR Time

G3. Highest recorded LDH measurement (if multiple peaks, record first peak): _____

LDH_1 measure _____ LDH_2 measure _____

Date of high measure: ☐☐/☐☐/☐☐
(day/month/year)

Time ☐☐:☐☐ **24 HR Time

G4. Record the first measured value on the day of admission or the day after.

Hematology:

Hematocrit	☐.☐☐☐
WBC count	☐☐.☐
Platelet Count	☐☐☐
Prothrombin Time/PT (sec.)	☐☐.☐
Partial Thromboplastin Time/PTT (sec.)	☐☐.☐

Chemistry (mmol/L):

Creatinine	☐.☐☐☐
BUN (Urea)	☐☐.☐
Sodium	☐☐☐
Potassium	☐.☐
Calcium	☐.☐☐
Albumin	☐☐☐
Total Bilirubin	☐☐
Glucose	☐☐.☐
Total Serum CO_2	☐☐.☐

ABG:

PaO_2	☐☐☐
pH	☐.☐☐
$PaCO_2$	☐☐☐
O_2 Saturation (%)	☐☐☐
Liters per minute/LPM O_2	☐☐
	or
	☐ room air

SECTION H: Chest X-Ray results
(see reports from the day of admission/event or the day after)

H1. Were any of the following noted:

☐ 9. No xray ☐ 0. Not noted ☐ 1. Noted Cardiomegaly

☐ 9. No xray ☐ 0. Not noted ☐ 1. Noted Congestive Heart Failure/ CHF

☐ 9. No xray ☐ 0. Not noted ☐ 1. Noted Pulmonary Edema

SECTION I: Nursing Admission Notes and Nursing Data Base

From nursing admission notes:

I1. Admission date (day/month/year) ☐☐/☐☐/☐☐

I2. Admission time ☐☐:☐☐ **24 HR Time

From nursing data base:

I3. Which of the following were noted on the nursing data base/initial assessment?

☐ 1. History of present illness (entry complaint)
☐ 2. Patient's living arrangements at the time of admission
☐ 3. Past medical history
☐ 4. Medications patient taking prior to admission
☐ 5. Systems review
☐ 6. Vital Signs (B/P, Pulse, Temp)
☐ 7. Functional status prior to admission
☐ 8. Allergies
☐ 9. Smoking status
☐ 99. No nursing assessment found/not on it

I4. What explanation is provided for sections not completed?

_____

_____

I5. Prior to admission, what were the living arrangements of the patient?

- ☐ 1. Lived alone
- ☐ 2. Lived with others
- ☐ 3. Lived in residental institution/PCH
- ☐ 4. Other
- ☐ 9. Not recorded

I6. Functional status (if unavailable from nursing data base or nursing admission notes see physio and OT consults, MD history and physical, and transfer/referral form.)

Base answers on the patient's functional status PRIOR TO THE INJURY

	Normal or no significant impairment	Mild or moderate impairment	Serious or severe impairment	No information available
Feeding	☐ 0	☐ 1	☐ 2	☐ 9/NR
Bathing	☐ 0	☐ 1	☐ 2	☐ 9/NR
Dressing	☐ 0	☐ 1	☐ 2	☐ 9/NR
Transferring	☐ 0	☐ 1	☐ 2	☐ 9/NR
Ambulation	☐ 0	☐ 1	☐ 2	☐ 9/NR
Fecal (bowel control)	☐ 0	☐ 1	☐ 2	☐ 9/NR
Urinary (bladder control)	☐ 0	☐ 1	☐ 2	☐ 9/NR
Mental status	☐ 0	☐ 1	☐ 2	☐ 9/NR

SECTION J: Nursing Notes and Flow Sheets

J1. Was the patient hospitalized in Winnipeg?

- ☐ Yes
- ☐ No

a. Date admitted to CCU ☐☐/☐☐/☐☐
(day/month/year)

b. Date discharged from CCU ☐☐/☐☐/☐☐
(day/month/year)

J2. Was a teaching plan discussed with the patient?

☐ No/NR ☐ Yes

SECTION K: Nursing Discharge Notes, D/C Sheet

K1. Was the patient discharged alive or dead?

☐ 1. Alive
☐ 2. Dead

K2. Was the patient discharge against medical advice (AMA)?

☐ No/NR ☐ Yes

K3. Where was the patient discharged to?

☐ 1. Own home
☐ 2. Home of friend or relative
☐ 3. Institution name _____
☐ 4. Other _____
☐ 9. Not recorded

K4. List all medications the patient was on at the time of discharge.

_____
_____
_____
_____
_____
_____
_____
_____

APPENDIX THREE

Sample of Field Manual

MHRIS

Comprehensive Manual:

Acute Myocardial Infarction

[AMI]

1MHRIS CODING MANUAL - AMI oct24.94 revised feb01.95, ... mar06/95,mar16/95, mar22/95, apr19/95,may04/95,may25/95, jul14/95, aug8/95, sept27/95, mar12/96codeman.ami

GENERAL INSTRUCTIONS

PM will code in green ink, other coders shall use red ink.
Coding must be complete prior to data entry.
All 'stopped abstracting' reviews should be forwarded to PM.

Verify completeness of all items:
YES/NO questions must have either YES or NO checked;
Multiple options questions must have at least one choice checked.
If items are missing with no note of 'Not recorded/NR', FLAG FOR REVIEWER.
Verify proper skips have been followed, ie are there questions completed that should not have been completed (skip patterns not followed)? If yes, FLAG FOR PM. Appropriately skipped data will not be coded or entered.

Where errors have been corrected, the circled response prevails, unless the reviewer or coder has clearly indicated a different response should be recorded.

Notes in margin - FLAG FOR PM (even if responses/boxes completed) re instructions on how to code. Keep notes on coding clarifications, items which do and items which do not count. All notes in blue ink which are NOT crossed out or checked (✓) by PM or coder should be noted in a WP file. If part of the note is circled by coder, enter only this data. Green ink comments should not be entered unless circled in green.

Open-ended questions (except for medications) - create WP file ordered by question #, response and study ID #. If part of the note is circled by coder, enter only this part of the note on the WP file.

FLAGS - list questionable items on post-it note (specify questions for PM and questions for reviewer). At the questions you have indicated, write a note on the review form explaining why you are returning the review.

FACE SHEET

Date of abstraction - dd/mm/yy.
Time start/time finish - if any breaks are noted, adjust the finish time accordingly. If a start time is noted along with times stopped and restarted, but the final finish time is not noted, then record NR (re data entry - leave blank).

Hospital name - code the number as per hospital coding list.

Variable list - be sure all items have been checked. (Study Team note: this will allow us to quickly identify cases where the selected hospital stay is not the stay abstracted.) **410 codes ending in '2' - FLAG FOR PM**
If no 410 code written on adm/sep abstr (noted by reviewer) enter code printed on the facesheet. At #1 - WP "no 410 recorded and coded as per F/S".

Final Status
- Completed cases should be NO for excluded cases
- Excluded cases should have exclusion Question # entered.
- Flagged cases should be passed to PM following coding.

** Reliability cases should be entered on the reliability data file.

QUESTIONS 1 - 4

1. More than one item may be checked.-
If 'not applicable' is checked, **FLAG FOR PM.** BGH, Concordia and Thompson Hosp.- 410 codes followed by (A) - adm. dx. should be excluded here IF MD specifically notes "no MI'.

2. If NO is checked, has PM checked this review?

3. If any item is checked YES, has PM checked this review? OOP cases are to be ruled out at 3d. - ie stay of interest is OOP.

4. Must be completed for all reviews, even if abstracting stopped at 1, 2, or 3. DO NOT RECORD these notes in the WP file. If narrative conflicts with responses to other questions, **FLAG FOR REVIEWER.** Special studies ie. Gusto Program at Grace and 96 hour protocol at HSC should be underlined and WP. Silent or missed MIs should be reviewed if not elsewhere excluded.

SECTION A

A1. If NO is checked, no other A questions should be completed.

A2. If YES is checked, 'name of transferring facility' in A3 should be completed, but no other A questions should be completed.

A3. Code the hospital number as per the coding list:
1. NO ICU BEDS
2. Any other reason given for transfer
3. N/R.

A4. At least one box must be checked. Use Box 2 for copies of EKG or Lab results.

A5. Dates must be recorded dd/mm/yy. Any month greater than 12 is not valid; Times must be 24 hr clock. Verify accuracy of hrs recorded as 0000 or 2400.

A6. If YES checked for either question, FLAG FOR PM.

A7. If NO is checked, questions a & b should not be completed. If YES checked and YES checked for either Question a or b, FLAG FOR PM.

** A8. Expect either <u>date and time</u> OR <u>days</u> OR <u>hours</u> to be completed - only one entry is accepted in D.E. If date is recorded, time N/R and hours ie. 2 - accept 2 hours and cross off the date.
Couple = 2, few = 3, several = 4.
Check: Is onset of symptoms prior to arrival? If not, FLAG FOR PM.

CODE: **11-PNEUMONIA**
12-ABDOMINAL PAIN
13-CORONARY-MYOCARDIALINSUFFICIENCY/ISCHEMIA
14-COPD, ASTHMA, RESPIRATORY ARREST
15-BACK PAIN NYD

A10. If NR is noted for any items, '9' fill these values.
ie Heart rate 999
Temp 99.9
BP 999/999
RR 999

If pt. noted to pulseless and arrests at adm., then code "0" for pulse and BP. If pt. intubated or bagged and no rate indicated, code "997". Do not code modifiers ie wet, irregular, etc. If (R) and (L) BP recorded: use (L) BP if no significant difference between (R) and (L). If significant difference - D.E. Lt. and WP both Rt and Lt. 03/03/95

A11. If YES is checked for Thrombolytic therapy (TT), EKGs, or enzymes (next page) ensure items in boxes are completed by reviewer. If not FLAG FOR REVIEWER.

Check: Was TT administered prior to admission? If YES, FLAG FOR REVIEWER.
Were enzymes measured prior to admission? If YES, FOR REVIEWER.

If NR is noted for any enzymes '9' fill these values.
ie highest LDH 9999 highest LDH1 99.99

If tests or reports are missing or not done - Coded 15/09/91, Time - N/R

1st recorded CPK and highest are the same if only one done. Do no '9' fill highest if only one done.

CPR/cardioversion should be checked if administered at the transferring hospital.

SECTION B

B1. If ADMITTED DIRECTLY TO WARD checked, date and time should be missing.
if both these items are completed with no explanation, FLAG FOR PM.
Check: Arrival date and time should be reasonably later than arrival at transferring hosp where applicable.

B2. If YES checked for either question, FLAG FOR PM.

B3. If YES checked for either question, FLAG FOR PM.

** B4. Expect either <u>date and time</u> OR <u>days</u> OR <u>hours</u> to be completed - only one entry is accepted in D.E. If date is recorded, time N/R and hours ie. 2 - accept 2 hours and cross off the date. If transfer case, expect B4 to be skipped.
Couple = 2, few = 3, several = 4.

Check: Is onset of symptoms prior to arrival? If not, FLAG FOR PM. Verify accuracy of hrs noted as either 0000 or 2400.

Time assignment: If onset is noted as:
'early this am' - 0700
'this am/morning' and time of arrival is 1300 hours or later, assign 1000;
'this am/morning' and time of arrival is prior to 1300 hrs, assign a time of 2 hrs prior to arrival;
'yesterday morning', assign 1000 hrs and be sure date is day prior to arrival;
'this afternoon' and time of arrival is 1900 hours or later, assign 1600;
'this afternoon' and time of arrival is prior to 1900 hrs, assign a time of 2 hrs prior to arrival, or 1200 (it must be a pm time);
'yesterday' assign date as day prior to arrival and leave time blank;
'yesterday afternoon' - date and 14:30;
'this evening' and time of arrival is 2300 hours or later, assign 2200;
'this evening' and time of arrival is prior to 2300 hrs, assign a time of 2 hours prior to arrival, or 1800 (it must be an evening time);
'last evening/yesterday evening' assign 2030 hrs;
'today' assign date of arrival and leave time blank;
'last night/tonight - if time of arrival is 0700 or later, then assign date of arrival as date and time as 0200. If time of arrival is between 0000 and 0700, assign time of 2 hrs. prior to arrival; if time of arrival is prior to midnight assign time as for evening. BE CAREFUL - Evening hours as for 24 hr. clock, also date/day prior as necessary.
'sudden onset' - Code - "96" hours.

If any time assignment by these rules does not seem appropriate, FLAG FOR PM.

B5. At least one item should be checked. Notes beside OTHER which cannot be recoded into an option provided should be noted in WP file.

NOT CODED:
- Conditions which are actually medical history will not be coded here (eg - COPD/COLD, asthma, DM), unless there is an indication that there is a problem on admission (eg - COPD exacerbation, diabetic coma, etc.
- IHD, dizzy, weak headache, allergic dermatitis, hyperlipodemia...
- Delete diaphoresis and weakness unless no other box is checked.

RECODED:
- Code as Chest Pain
  - Chest pressure/tightness
- Code as CHF
  - Pulmonary Edema
  - Cardiogentic Shock (if nothing else coded)
- Code as Arrythmia/Cardiac Arrest/Syncope
  - any arrythmia, ie A-fib, heart block, rapid heart rate...
  - post arrest, ie - pt arrested PTA
- - collapse, blackout (unless noted with resp. arrest - coded resp. arrest)
- Code as Abd. Pain

- gastritis, esosphagitis, pancreatitis, ? stress ulcer, gastric in origin, ? GI, heartburn

CODE: **11-PNEUMONIA**
12-ABDOMINAL PAIN
13-CORONARY-MYOCARDIALINSUFFICIENCY/ISCHEMIA
14-COPD, Asthma, Respiratory Arrest
15-Back Pain NYD

B6. If YES is checked for Thrombolytic therapy (TT) ensure items in boxes are completed by reviewer. If not FLAG FOR REVIEWER.

Check: Was TT administered prior admission? If YES, FLAG FOR REVIEWER.

Cardioversion/defib should be checked if CPR or cardioversion administered within 24 hrs. of arrival. If Cardioversion PTA or pt. intubated at transferring hosp or in ambulance - Code YES.

B7. If NR is noted for any items, '9' fill these values.

ie Heart rate 999
Temp 99.9
BP 999/999
RR 999

If pt. noted to pulseless and arrests at adm., then code "0" for pulse and BP. If pt. intubated or bagged and no rate noter, code "997". Do not code modifiers ie wet, irregular, etc. If (R) and (L) BP recorded: use (L) BP if no significant difference between (R) and (L). If significant difference - WP both BP. 03/03/95

SECTION C

C1. Ensure all items are checked either YES or NO. If not FLAG FOR REVIEWER. 'QUERY' is coded NO in this section. If CHF or GI Bleed is noted at B5, they should also be noted here.
If YES is checked for PREVIOUS MI, ensure item in box is completed by reviewer. If not FLAG FOR REVIEWER.

NOT CODED:
acoustic neuroma, arrythmias (except VTACH), blindness, cataracts, cholelithiasis, deafness, DVT, glaucoma, gout, hiatus hernia, hypercalcemia, hyperlipodemia, kidney stones, osteoarthritis, pulmonary embolism, rheumatism, thyroid problems, and any surgeries more than 3 weeks previous.

WP: anemia, personality disorder, schizophrenia, epilepsy, seizures and cardiac anomalies NEC.

CODED:
Organic Brain Syndrome - code as DEMENTIA, 'borderline' modifier - WP
Pulmonary Fibrosis code as COPD and WP.
Pneumonia noted as present on admission - code at B5.
Brain tumor - code as Metastatic Ca even if not stated as cancerous

C2. Non-smoker - must be non-smoking for at least 30 days, otherwise code as smoker. If noted nurse says smoker and doctor says non-smoker - CODE - Status unclear.

C3. Ensure distinction between 'not recorded/NR' and 'nil' or 'no meds' is clear.

C4. Either comments should be noted or box should be checked. If 'no contraindications...' box is not checked and contraindications are not recorded, FLAG FOR REVIEWER. Discharge summary is an acceptable source.

C6. CODED: possible, recent, evolving, query, R/O, may have been, age uncertain.

NOT CODED: Cannot be excluded, previous and old.

C8. QUERY may be used for this section. If YES is checked for HEART BLOCK, ensure item in box is completed by reviewer. If not FLAG FOR REVIEWER. 2:1 & 4:1 block is 2nd degree.

C9. If yes is checked at C9 - FLAG FOR PM (Jan04.95).

C11. If NR is noted, check YES. NOTE that this is the ONLY time you check YES if NR .

SECTION D

D1. If YES checked, ensure item in box is completed by reviewer. If not FLAG FOR REVIEWER. Demand pacemaker - leave box blank and WP.

D3. Cordis insertion - delete and check NO.

D5. CVA/stroke - If YES checked, ensure item in box is completed by reviewer. If not FLAG FOR REVIEWER.

Code the terms describing stroke as either:

1. **Thrombotic (cerebral)**
2. **Hemorrhagic**
3. **Brain anoxia, anoxic encephalopathy, Other**
9. **N/R**

Cardiac arrest should be checked only if it occurs after initial 24 hours and the patient is successfully resuscitated.

Extended / New AMI - If YES checked, ensure item in box is completed by reviewer. If not FLAG FOR REVIEWER.

Check: date should follow arrival at the marker hospital.If not FLAG FOR REVIEWER. Verify accuracy of hrs recorded as 0000 or 2400.

Other complications - any items which can be clearly coded as one of the preceding items should be changed accordingly. Pyrexia, increased temp, UTI, hypotension, arrhythmias other than <**VTACH**> need not be noted. Remaining items should be noted for open-ended entry. Dx after admission - diabetes, COPD, CRF, cancer and other conditions that were probably present before admission should be crossed off and coded YES at C1 with WP "dx during admission".

NOT CODED:

Gout, bigeminy, hypoglycemic episodes when the pt. is diabetic (ketoacidosis would be coded), hypercalcemia.

CODED:

OTHER INFECTIONS: Incision and wound infections, central line septicemia, IV site infection, chest infection, etc. If query infection - code YES and WP.

SECTION E

If YES is checked for any item, ensure items in boxes are completed by reviewer. If not FLAG FOR REVIEWER.

Check: all dates should follow arrival at the marker hospital and precede discharge date. If not FLAG FOR REVIEWER. Verify accuracy of hrs recorded as 0000 or 2400.

E3. Echo results recorded as found in doctor's notes or discharge summary should be WP. Discharge summary sometimes lists ejection fraction as a different amount than the report. If this is noted WP the comment. No ejection fraction - Code '99'. Normal EF - Coded '98'.

E4. Stress test result - Can code '9' for N/R.

E7. No WP if noted that test was performed at a different hosp.

E8. NOT CODED: Holter tests (except Camiat Holter, which should be coded for notation of study), pulmonary function tests, skeletal X-rays and CXR.

SECTION F

F1. Check: date should follow arrival at the marker hospital.
If time is earlier than arrival time and is noted as correct or is not excessively earlier, it can be changed to 'arrival time'. If more than 5 hours and not noted as to why - FLAG FOR PM. Stripped or culled charts should be left blank and WP unless MD progress notes results. If they meet the requirements, these values can be coded in the appropriate place (this applies through Section H1).

NOTE: If reports are <u>missing or not done</u> - Code - Date: 15/09/91, time: N/R and n/r or not noted for the rest of the questions (this applies through H1). This does not apply to stripped or culled charts.

F2. Only one box should be checked, unless pt. had 2 MIs. If two boxes are checked CODE the first one mentioned ie. written description of EKG or first one mentioned in #4. If anterolateral or anterior/inferior MI is noted, then code anterior only.

CODED: Possible/probable MI, recent, ? MI, R/O MI, may have had, evolving and age uncertain - Code - YES.

NOT CODED: 'Old MI's, "injury" or "ischemia' and MI can not be excluded are coded 'NO MENTION OF AN MI'. If the report states unchanged from EKG ie 3 months previous, then consider this "old".

F4. If any comments are noted, FLAG FOR PM.

ST elev or depres - Modifiers suct as mild or slight note for WP file. Modifiers such as persistent and marked - delete.

Other ST - T-Wave changes - WP modifiers ie. flipped, sagging, etc.

Loss of R and Q waves - Q's (unless noted as small) - code YES. If small - code NO and WP. R wave modifiers - WP and check yes even it says "indicative of old MI.

Heart block - If YES is checked ensure items in boxes are completed by reviewer. If not FLAG FOR REVIEWER. 2:1 & 4:1 block are 2nd degree block. Complete A-V dissociation is complete heart block (if not specified as complete, check heart block and leave type blank.

DO NOT CODE: Hemiblock, fascicular blk, bifascicular blk.

BBB - **DO NOT CODE** incomplete bbb.

Do not WP comments regarding arrythmias (other than VTACH), bradycardia, A-fib, or ischemia. All other comments, FLAG FOR PM.

F5. Check: date should follow date of first EKG report (F1). If not FLAG FOR REVIEWER. If only one EKG done and this is noted here - follow the rules for missing or not done reports at F1.

F6-8. As for F2-4

SECTION G

If NR is noted for any enzymes '9' fill these values.
ie highest LDH 9999 highest LDH1 999

G1 - G3. Were enzymes measured prior to admission? If YES, FLAG FOR REVIEWER. If no dates or time recorded - leave n/r (cannot use time reported as the samples may not have been analyzed till the day after being drawn).

G1. If neither % MB nor MB u/l is completed, '9' fill % MB only. If one or the other is completed, do not '9' fill the other.
MB % - decimal values may be entered.
If first peak is in normal range and a second peak is abnormal, the second peak will be coded.

**** MB values recorded in ng/ml - multiply by three and record in MB box. WP ID numbers for all hospitals.**

If 'first CPK was highest' box is checked, record these values in next section for highest CPK.
If 'only measure' box is checked, the next section (highest CPK) should be '9' filled. If not, FLAG FOR PM.

If CK is less than 20 - code 15. Do not WP "slightly hemolized".
If CK is greater than 10,000 - Code 9997.

'No MB band detected - Code 0000.

RURAL LABS: (G1 & G2)
If 2 measures on the same sample are reported for CK and normal ranges are somewhere between 0-200 use the CK with a MB value except in cases where the difference in CK between the two reported values is greater than 10% ie if difference is 15%, use the local report.

G2. Was highest CPK measured later or at the same time as first CPK? If not, FLAG FOR REVIEWER.

If neither % MB nor MB u/l is completed, '9' fill these values. If one or the

other is completed, do not '9' fill the other.
MB % - decimal values may be entered.

** MB values recorded in ng/ml - **multiply by three** and record in MB box. WP ID numbers for all hospitals.

If a lower CK with a higher CKMB than highest CK is recorded - WP only if the highest CK is in the normal range.

G3. If LDH value from Brandon with a LDH1/LDH2 ratio is noted - use this amount. WP ID number and local value.

G4. If NR is noted for any results '9' fill these values. Albumin and total bilirubin can now be entered in boxes however, WP if other unit noted than g/l for alb or umol for bili.

If values too large for box - Code - 999.7. If stated 'greater than' or 'less than' Code amount and WP comment.

WP: "I.V. off only 2 min. with uncertain effects on lab results" and"Slightly hemolized".

If creatinine is recorded in umol in the margin, divide by 1000 and record in the boxes provided.

LPM O_2 should only be noted where ABG values are noted.

SECTION H

If 'no x-ray' is checked for one item, it must be checked for all 3 items.

CHF - Do not code modifiers such as mild or acute.

Cardiomegaly = enlarged heart

DO NOT CODE: borderline enlarged, generous, slightly enlarged (slightly, mild cardiomegaly - WP)

Pulmonary edema

CODED: Pulmonary venous redistribution/congestion.
CODED & WP: Pulmonary vascular engorgement, pulmonary engorgement

NOT CODED: Pulmonary venous hypertension, pleural effusion.

SECTION I

I1-2. If date and/or time are missing and NR is not noted, FLAG FOR REVIEWER. If pt. never admitted ie, remained in ER or Obs - Code - Date: 15/09/91, time: N/R

Check: date should follow arrival in ER. If not FLAG FOR REVIEWER.

I3. If any items (except '99') are not checked, I4 must be answered. If not, FLAG FOR REVIEWER. "Lives in a house, apt. etc." is considered living arrangement prior to admission. Code YES if "partially completed" noted for any items.

I4. CODE: 1 - Decreased LOC/pt. ventilated/poor condition/pt. died
2 - Confusion/senility
3 - Language barrier, hard of hearing
4 - 'See old chart'/'See prev. adm'
8 - I3 is filled out correctly - N/A
9 - No explanation/not on form/not recorded
Blank - if I3 - Coded - 99 (no nursing assessment found)

I5. If brought in by wife, or D/C'd with wife and no indication pt. living in residential institution, code as LIVING WITH OTHERS (#2).

I6. Only one box should be checked for each horizontal row. If any rows are missed, FLAG FOR REVIEWER.

SECTION J

J1. If dates are missing and NR is not noted, FLAG FOR REVIEWER.
If NO ICU/CCU is noted circle the comments and check NO ICU. WP any note stating differences in transfer orders and actual transfer from ICU.

Check: date should follow or equal admission date.
If not FLAG FOR REVIEWER. Verify accuracy of hrs noted as 0000 or 2400.

SECTION K

K3. If institution is checked, code the category:
1. Acute care (AC)
2. Personal Care Home (PCH)
3. Other - Riverside (Municipal), Deer Lodge, geriatric, mental and rehab.
9. Not recorded - institution checked but name not given and reviewer noted NR.

APPENDIX FOUR: List of Variables

The SAS System 15:42 Wednesday, November 15, 2000 25

CONTENTS PROCEDURE

#	Variable	Type	Len	Pos	Informat	Label
406	ASPAIDL	Num	8	2009		Ideal candidate for aspirin during
412	ASPAINT	Num	8	2057		Asp dur: (Ideal * received drug)
410	ASPDIDL	Num	8	2041		Ideal candidate for aspirin at disch
415	ASPDINT	Num	8	2081		Asp at disch: (Ideal * received drug)
352	ASPEXA	Num	8	1645		Appropriateness: aspirin dur. (0=ideal)
409	ASPEXB	Num	8	2033		
353	ASPEXD	Num	8	1653		
345	ASPIRA	Num	8	1589		Aspirin therapy during hosp
346	ASPIRD	Num	8	1597		Aspirin therapy (disch.; 1=yes)
101	ASPIRIN	Num	4	416	16.13	B6.Aspirin Administered At Marker Hosp.
355	BETAEX	Num	8	1669		Appropriateness: beta blockers (0=ideal)
411	BETAIDL	Num	8	2049		Ideal candidate for beta bl at disch
416	BETAINT	Num	8	2089		Beta at disch: (Ideal * received drug)
347	BETATHER	Num	8	1605		Beta blocker therapy (1=yes)
206	BILIRUBI	Num	4	836	3.	G4.Chemistry (mmol/L) - Total Bilirubin
82	BLEED	Num	4	340	7.4	D5.Complications - Bleeding
241	BUN	Num	8	966	4.1	G4.Chemistry (mmol/L) - BUN (Urea)
99	CABGCOND	Num	4	408	7.4	E2.CABG Conducted
260	CABGDAT	Num	6	1108	8.	E2.Date CABG Conducted
244	CALCIUM	Num	8	990	5.2	G4.Chemistry (mmol/L) - - Calcium
85	CARDARR	Num	4	352	19.15	D5.Complications - Cardiac Arrest
428	CARDDYSP	Num	8	2170		Cardiac dysrythmia from hospital claim
405	CCI	Num	8	2001		Charlson Index (0-3)
272	CCUADMDT	Num	6	1182	8.	J1a.Date Admitted to CCU
273	CCUDISDT	Num	6	1188	8.	J1b.Date Discharged From CCU
382	CENSOR1	Num	8	1823		
380	CENSOR5	Num	8	1807		
404	CHARL1P	Num	8	1993		
37	CHARYES	Num	3	121		
79	CHF	Num	4	328	7.4	D5.Complications - Congestive Hrt Fail.
425	CHFP	Num	8	2146		CHF from hospital claim
341	CHFX	Num	8	1557		CHF (adm, pulmed, exsJ, xray, rales)
342	CHFXX	Num	8	1565		CHF (same as CHFX but excl. rales)
359	COVERED	Num	8	1694		
81	CPAIN	Num	4	336	7.4	D5.Complications - Chest Pain/Angina
263	CPKF	Num	8	1124	4.	G1.First Reported CPK
325	CPKFDT	Num	8	1432		G1.CPKF DATETIME
264	CPKH	Num	8	1132	4.	G2.Highest Reported CPK
326	CPKHDT	Num	8	1440		G2.CPKH DATETIME
103	CPR	Num	4	424	16.13	B6.CPR At Marker Hospital <24 hours adm
240	CREATINI	Num	8	958	6.3	G4.Chemistry (mmol/L) - Creatinine
78	CVA	Num	4	324	7.4	D5.Complications - Cerebrovascular Acc.

The SAS System 15:42 Wednesday, November 15, 2000 26

CONTENTS PROCEDURE

#	Variable	Type	Len	Pos	Informat	Label
427	CVDP	Num	8	2162		CVD from hospital claim
235	DBP	Num	8	918	3.	B7.First Recorded DBP at Marker Hosp.
83	DEATH	Num	4	344	19.15	D5.Complications - Death
384	DEATH30	Num	8	1839		
385	DEATH1YR	Num	8	1847		
386	DEATH5YR	Num	8	1855		
432	DIABCMP	Num	8	2202		Diabetes with complications from hospita
433	DIABNCMP	Num	8	2210		Diabetes without complications from hosp
92	DISAMA	Num	4	380	9.6	K2.Discharged Against Medical Advice
91	DISCHTO	Num	4	376	19.15	K3.Discharged To
276	DISDAT	Num	6	1206	8.	FACE.Separation Date
90	DISDEAD	Num	4	372	9.6	K1.Discharged Alive or Dead?
190	DISINST	Num	4	772	19.15	K3.Discharged To Institution Type
181	DLOC	Num	4	736	7.4	C9.Decreased Level of Consciousness
74	DLOCEYE	Num	4	308	10.7	C11.Decreased Eye Open Spontaneously
73	DLOCMED	Num	4	304	6.3	C10.Decreased LOC due to Medications
219	DMED1	Char	2	864	2.	K4.Meds At Discharge, 1 of 12
220	DMED2	Char	2	866	2.	K4.Meds At Discharge, 2 of 12
221	DMED3	Char	2	868	2.	K4.Meds At Discharge, 3 of 12
222	DMED4	Char	2	870	2.	K4.Meds At Discharge, 4 of 12
223	DMED5	Char	2	872	2.	K4.Meds At Discharge, 5 of 12
224	DMED6	Char	2	874	2.	K4.Meds At Discharge, 6 of 12
225	DMED7	Char	2	876	2.	K4.Meds At Discharge, 7 of 12
226	DMED8	Char	2	878	2.	K4.Meds At Discharge, 8 of 12
227	DMED9	Char	2	880	2.	K4.Meds At Discharge, 9 of 12
228	DMED10	Char	2	882	1.	K4.Meds At Discharge, 10 of 12
229	DMED11	Char	2	884	1.	K4.Meds At Discharge, 11 of 12
230	DMED12	Char	2	886	1.	K4.Meds At Discharge, 12 of 12
274	DOB	Num	6	1194	8.	FACE.Date of Birth, Day=01 For All
360	DTHFL	Num	8	1702		
76	DVT	Num	4	316	6.3	D5.Complications-Deep Venous Thrombosis
3	DX01	Char	5	13		DIAG # 1 - ICD9CM-PRIMARY DX
4	DX02	Char	5	18		DIAGNOSIS # 2 - ICD9CM
5	DX03	Char	5	23		DIAGNOSIS # 3 - ICD9CM
6	DX04	Char	5	28		DIAGNOSIS # 4 - ICD9CM
7	DX05	Char	5	33		DIAGNOSIS # 5 - ICD9CM
8	DX06	Char	5	38		DIAGNOSIS # 6 - ICD9CM
9	DX07	Char	5	43		DIAGNOSIS # 7 - ICD9CM
10	DX08	Char	5	48		DIAGNOSIS # 8 - ICD9CM
11	DX09	Char	5	53		DIAGNOSIS # 9 - ICD9CM
12	DX10	Char	5	58		DIAGNOSIS #10 - ICD9CM
13	DX11	Char	5	63		DIAGNOSIS #11 - ICD9CM

#	Variable	Type	Len	Pos	Informat	Label
14	DX12	Char	5	68		DIAGNOSIS #12 - ICD9CM
15	DX13	Char	5	73		DIAGNOSIS #13 - ICD9CM
16	DX14	Char	5	78		DIAGNOSIS #14 - ICD9CM
17	DX15	Char	5	83		DIAGNOSIS #15 - ICD9CM
18	DX16	Char	5	88		DIAGNOSIS #16 - ICD9CM
20	DXTYPE01	Char	1	102		DIAGNOSIS TYPE OF DX01
21	DXTYPE02	Char	1	103		DIAGNOSIS TYPE OF DX02
22	DXTYPE03	Char	1	104		DIAGNOSIS TYPE OF DX03
23	DXTYPE04	Char	1	105		DIAGNOSIS TYPE OF DX04
24	DXTYPE05	Char	1	106		DIAGNOSIS TYPE OF DX05
25	DXTYPE06	Char	1	107		DIAGNOSIS TYPE OF DX06
26	DXTYPE07	Char	1	108		DIAGNOSIS TYPE OF DX07
27	DXTYPE08	Char	1	109		DIAGNOSIS TYPE OF DX08
28	DXTYPE09	Char	1	110		DIAGNOSIS TYPE OF DX09
29	DXTYPE10	Char	1	111		DIAGNOSIS TYPE OF DX10
30	DXTYPE11	Char	1	112		DIAGNOSIS TYPE OF DX11
31	DXTYPE12	Char	1	113		DIAGNOSIS TYPE OF DX12
32	DXTYPE13	Char	1	114		DIAGNOSIS TYPE OF DX13
33	DXTYPE14	Char	1	115		DIAGNOSIS TYPE OF DX14
34	DXTYPE15	Char	1	116		DIAGNOSIS TYPE OF DX15
35	DXTYPE16	Char	1	117		DIAGNOSIS TYPE OF DX16
258	ECHODAT	Num	6	1094	8.	E3.Date Echocardiography Reported
259	ECHOEF	Num	8	1100	4.1	E3.ECG Ejection Fraction
93	ECHOREP	Num	4	384	7.4	E3.Echocardiography Reported
170	EKG1BBB	Num	4	692	18.15	F4.First EKG - Bundle Branch Block
327	EKG1DT	Num	8	1448		F1.EKG1 DATETIME
172	EKG1HB	Num	4	700	18.15	F4.First EKG - Heart Block
178	EKG1HBTP	Num	4	724	19.15	F4.First EKG - Type of Heart Block
107	EKG1LOC	Num	4	440	19.15	F2.First EKG - Location of Infarct
174	EKG1LOR	Num	4	708	18.15	F4.First EKG - Loss of R Forces
279	EKG1Q	Num	4	1224		F3.First EKG - Type of Current MI
109	EKG1STCH	Num	4	448	18.15	F4.First EKG - Other ST Changes
176	EKG1STEL	Num	4	716	18.15	F4.First EKG - ST Elevation/Depression
171	EKG2BBB	Num	4	696	18.15	F8.Last EKG - Bundle Branch Block
328	EKG2DT	Num	8	1456		F5.EKG2 DATETIME
173	EKG2HB	Num	4	704	18.15	F8.Last EKG - Heart Block
179	EKG2HBTP	Num	4	728	19.15	F8.Last EKG - Type of Heart Block
108	EKG2LOC	Num	4	444	19.15	F6.Last EKG - Location of Infarct
175	EKG2LOR	Num	4	712	18.15	F8.Last EKG - Loss of R Forces
280	EKG2Q	Num	4	1228		F7.Last EKG - Type of Current MI
110	EKG2STCH	Num	4	452	18.15	F8.Last EKG - Other ST Changes
177	EKG2STEL	Num	4	720	18.15	F8.Last EKG -ST Elevation or Depression

The SAS System 15:42 Wednesday, November 15, 2000 28

CONTENTS PROCEDURE

#	Variable	Type	Len	Pos	Informat	Label
119	EKGMAF	Num	4	488	18.15	C8.Atrial Fibrillation - MD Reports
143	EKGMBBB	Num	4	584	18.15	C8.Bundle Branch Block - MD Reports
116	EKGMHB	Num	4	476	18.15	C8.Heart Block - MD Reports Of EKG
121	EKGMHBTP	Num	4	496	19.15	C8.Type of Heart Block - MD Reports
122	EKGMISC	Num	4	500	18.15	C8.Ischemic Changes - MD Reports Of
120	EKGMLOC	Num	4	492	19.15	C6.Location of Current MI (MD Exam)
169	EKGMLOR	Num	4	688	18.15	C8.Loss Of R Forces - MD Reports
278	EKGMQ	Num	4	1220		C7.Type of the Current MI - MD Report
115	EKGMSTCH	Num	4	472	18.15	C8.Oth ST or T-Wave Changes - MD Report
114	EKGMSTEL	Num	4	468	18.15	C8.ST Elevation/Depression - MD Report
168	EKGMSVT	Num	4	684	18.15	C8.Supraventr.Tachycardia - MD Report
118	EKGMVFIB	Num	4	484	18.15	C8.Ventricular Fibrillation - MD Report
117	EKGMVTAC	Num	4	480	18.15	C8.Ventricular Tachycardia - MD Report
344	EXCLDTB	Num	8	1581		
111	EXPULMED	Num	4	456	18.15	C5.Pulmonary Edema - MD Phy Exam
113	EXRALES	Num	4	464	18.15	C5.Rales Noted on MD Phy Exam
112	EXS3	Num	4	460	18.15	C5.S3 Noted on MD Phy Exam
166	EXSHOCK	Num	4	676	18.15	C5.Shock Noted on MD Phy Exam
277	FILE	Num	8	1212		For Data Entry Purposes Only
246	GLUCOSE	Num	8	1006	4.1	G4.Chemistry (mmol/L) - Glucose
238	HEMATOCR	Num	8	942	6.3	G4.Hematology - Hematocrit
100	HEPARIN	Num	4	412	16.13	B6.Heparin Administered at Marker Hosp.
354	HEPEX	Num	8	1661		Appropriateness: heparin (0=ideal)
348	HEPH	Num	8	1613		Heparin therapy (1=yes)
407	HEPIDL	Num	8	2017		Ideal candidate for heparin during
413	HEPINT	Num	8	2065		Hep dur: (Ideal * received drug)
1	HOSP	Num	4	0		HOSPITAL CODE #
262	HOSPITAL	Char	4	1120	4.	FACE.Hospital Number
144	HOSPWPG	Num	4	588	7.4	J1.Patient Hospitalized in Winnipeg
233	HRATE	Num	8	902	3.	B7.1st Recorded Heart Rate-Marker Hosp.
19	HRN	Char	9	93	9.	HOSPITAL RECORD NUMBER
157	HXALZDZ	Num	4	640	9.6	C1.History - Demential/Altheimer's Dis.
136	HXANGIN	Num	4	556	9.6	C1.History - Angina/Chest Pain
67	HXASASEN	Num	4	280	9.6	C1.History - Aspirin Allergy/Hypersens.
152	HXBLEED	Num	4	620	9.6	C1.History - Bleeding Disorder
183	HXCA	Num	4	744	9.6	C1.History - Oth Non-Metastatic Cancer
65	HXCABG	Num	4	272	15.12	C1.History - CABG
159	HXCARDSU	Num	4	648	9.6	C1.History - Other Cardiac Surgery
145	HXCHF	Num	4	592	9.6	C1.History - Congestive Heart Failure
148	HXCOPD	Num	4	604	9.6	C1.History - Chronic Pulmonary Disease
137	HXCVA	Num	4	560	9.6	C1.History - Stroke/Cardiovacular Acc.
158	HXDIAB	Num	4	644	9.6	C1.History - Diabetes (any type)

#	Variable	Type	Len	Pos	Informat	Label
160	HXDNR	Num	4	652	9.6	C1.History - Terminal Illness, DNR
151	HXGIB	Num	4	616	9.6	C1.History - Genito-Urinary or Gastro.
155	HXHIV	Num	4	632	9.6	C1.History - HIV+/AIDS
147	HXHTN	Num	4	600	9.6	C1.History - Hypertension/High Blood Pr
184	HXIHD	Num	4	748	9.6	C1.History - Ischemic Heart Dis.or CAD
150	HXLIVER	Num	4	612	9.6	C1.History - Chronic Liver Disease
154	HXMETCA	Num	4	628	9.6	C1.History - Metastatic Cancer
135	HXMI	Num	4	552	9.6	C1.History - Previous MI
180	HXMI8W	Num	4	732	9.6	C1.History - Most Recent MI <=8 wks
185	HXPACEM	Num	4	752	9.6	C1.History - Pacemaker Prev. Insert
66	HXPTCA	Num	4	276	9.6	C1.History - PTCA
146	HXPVD	Num	4	596	9.6	C1.History - Peripheral Vascular Dis.
156	HXRENAL	Num	4	636	9.6	C1.History - Renal Failure
281	HXSMOK	Num	8	1232		C2.History - Smoking Status
138	HXSURG	Num	4	564	9.6	C1.History - Major Surgery <=2 wks.
153	HXTRAUMA	Num	4	624	9.6	C1.History - Trauma In Past Month
149	HXULCER	Num	4	608	9.6	C1.History - Ulcer Disease
436	I	Num	8	2234		
142	IABP	Num	4	580	9.6	D4.Intra-Aortic Balloon Pump Inserted
232	ICDCODE	Num	8	894	19.15	1.ICD-9-CM DX For AMI
403	INCRUR1	Num	8	1985		Income quintile 1 - rural
402	INCURB1	Num	8	1977		Income quintile 1 - urban
361	INDEXADM	Num	8	1710		Index admit date, incl. revised (N=2,222
362	INDEXSEP	Num	8	1718		Index separation date,incl.revis.(N=2,22
191	INICU	Num	4	776	7.4	J1.Patient Treated In ICU
72	INTUB	Num	4	300	17.14	B6.Patient Intubated At Marker Hospital
265	LDHH	Num	8	1140	4.	G3.Highest Recorded LDH
329	LDHHDT	Num	8	1464		G3.LDHH DATETIME
357	LFCANRSX	Char	1	1685		LFCANRSN, with code revisions for n=5
356	LFDTCAN	Char	8	1677		Coverage cancel date (to Mar.31/98)
358	LFDTDTHX	Char	8	1686		LFDTDTH with one correction
203	LMPO2	Num	4	824	3.	G4.ABG - Liters Per Minute/LPM O2
419	LOS	Num	8	2106		
423	LOSICU	Num	8	2130		
426	MALIGP	Num	8	2154		Malignancy from hospital claim
418	MARCHNG	Char	8	2098		Date of last marital status change
417	MARST	Char	1	2097		Marital status as of index AMI admission
266	MBF	Num	8	1148	4.1	G1.First Reported CPK - MB u/l
267	MBH	Num	8	1156	3.	G2.Highest Reported CPK - MB u/l
335	MBPCTF	Num	8	1512		G1.First CPK - % MB
336	MBPCTH	Num	8	1520		G2.Highest CPK - % MB
163	MEDIC	Num	4	664	6.3	B3.Admitted For Other Medical Reasons

CONTENTS PROCEDURE

#	Variable	Type	Len	Pos	Informat	Label
71	MOTOR	Num	4	296	19.15	C12.Worst Condition Initial 24 Hours
255	MUGADAT	Num	6	1076	8.	E6.Date MUGA Scan Reported
204	MUGAEF	Num	4	828	3.	E6.MUGA Ejection Fraction
95	MUGAREP	Num	4	392	7.4	E6.MUGA Scan Reported
363	MUNCODE	Char	3	1726		MUNICIPAL CODE
324	NADMITDT	Num	8	1424		I1,12.ADMIT DATETIME
192	NDB1	Num	4	780	3.	I4.Reason 1 Why Nursing Data Not Compl
193	NDB2	Num	4	784	1.	I4.Reason 2 Why Nursing Data Not Compl
316	NDBALRG	Num	4	1376		I3.ND : Allergies
315	NDBFSTAT	Num	4	1372		I3.ND : Functional status prior to adm.
309	NDBHXILL	Num	4	1348		I3.NDB : HX of present illness
312	NDBMED	Num	4	1360		I3.NDB : Medications taken prior to adm
318	NDBNO	Num	4	1384		I3.NDB : No Nursing Assessment found
311	NDBPMH	Num	4	1356		I3.NDB : Past medical history
310	NDBPTLV	Num	4	1352		I3.NDB : Living arrange.at time of adm.
317	NDBSMOK	Num	4	1380		I3.NDB : Smoking status
313	NDBSYSR	Num	4	1364		I3.NDB : Systems review
314	NDBVS	Num	4	1368		I3.NDB : Vital signs
378	NDTCAN	Num	8	1791		
377	NDTDTH	Num	8	1783		
186	NOCONTT	Num	4	756	19.15	C3.No Contraind.to Thrombolytic Therapy
124	NPATLIV	Num	4	508	19.15	I5.Living Arrangements Prior to Adm.
251	O2SAT	Num	8	1046	4.1	G4.ABG - O2 Saturation (%)
322	ONSETDT	Num	8	1408		B4.ONSET DATETIME
365	OP01	Char	4	1735		SURGICAL PROCEDURE # 1
366	OP02	Char	4	1739		SURGICAL PROCEDURE # 2
367	OP03	Char	4	1743		SURGICAL PROCEDURE # 3
368	OP04	Char	4	1747		SURGICAL PROCEDURE # 4
369	OP05	Char	4	1751		SURGICAL PROCEDURE # 5
370	OP06	Char	4	1755		SURGICAL PROCEDURE # 6
371	OP07	Char	4	1759		SURGICAL PROCEDURE # 7
372	OP08	Char	4	1763		SURGICAL PROCEDURE # 8
373	OP09	Char	4	1767		SURGICAL PROCEDURE # 9
374	OP10	Char	4	1771		SURGICAL PROCEDURE #11
375	OP11	Char	4	1775		SURGICAL PROCEDURE #11
376	OP12	Char	4	1779		SURGICAL PROCEDURE #12
86	OTHER	Num	4	356	19.15	D5.Complications - Other
194	OTHINF	Num	4	788	9.6	D5.Complications - Other Infection
182	OTHRTST	Num	4	740	7.4	E8.Other Tests Conducted
199	OTHTEST1	Num	4	808	3.	E8.Other Test 1
200	OTHTEST2	Num	4	812	3.	E8.Other Test 2
201	OTHTEST3	Num	4	816	1.	E8.Other Test 3

202	OTHTEST4	Num	4	820	1.	E8.Other Test 4
104	PACEM	Num	4	428	9.6	D1.Pacemaker Implanted After Admission
105	PACETYPE	Num	4	432	19.15	D1.Type of Pacemaker Impl. After Adm.
250	PACO2	Num	8	1038	4.1	G4.ABG - PaCO2
248	PAO2	Num	8	1022	5.1	G4.ABG - PaO2
77	PE	Num	4	320	6.3	D5.Complicacions - Pulmonary Embolus
249	PH	Num	8	1030	6.3	G4.ABG - pH
2	PHIN91	Char	9	4		MHSC SCRAMBLED PHIN
205	PLATELET	Num	4	832	3.	G4.Hematology - Platelet Count
75	PNEUM	Num	4	312	7.4	D5.Complications - Pneumonia
364	POSTAL	Char	6	1729		POSTAL CODE
420	POSTCODE	Char	6	2114		
243	POTASSIU	Num	8	982	5.2	G4.Chemistry (mmol/L) - Potassium
252	PT	Num	8	1054	4.1	G4.Hematology-Prothrombin Time/PT (sec)
98	PTCACOND	Num	4	404	7.4	E1.PTCA Conducted
261	PTCADAT	Num	6	1114	8.	E1.Date PTCA Conducted
253	PTT	Num	8	1062	4.1	G4.Hematology-Prtl Thromboplastin Time
429	PULMONP	Num	8	2178		Pulmonary edema from hospital claim
68	RACE	Num	4	284	18.15	FACE.Race
430	RENAL1P	Num	8	2186		Acute renal disease from hospital claim
431	RENAL2P	Num	8	2194		Chronic renal disease from hospital clai
236	RESP	Num	8	926	3.	B7.1st Rec.Respiratory Rate at Marker H
339	RESPID	Num	5	1344		Respondent number
401	RESRUR	Num	8	1969		Rural resident
400	RESURB	Num	8	1961		Urban resident
421	RHA	Char	2	2120		
161	ROADCAB	Num	4	656	6.3	B2.Ruled Out - Initial Admit For CABG
69	ROADMDAT	Num	4	288	7.4	2.Ruled Out - Adm. Date not in study
162	ROADSURG	Num	4	660	6.3	B2.Ruled Out-Initial Admit For Surgery
139	ROAGE	Num	4	568	6.3	3a.Ruled Out - Patient <45 Years old
134	ROCVA	Num	4	548	6.3	3b.Ruled Out-CVA <= 48 Hours before Adm
165	ROMICOM	Num	4	672	1.	B3b.Ruled Out - MI Compl. Other
164	ROMIINH	Num	4	668	1.	B3a.Ruled Out - MI Happened in Hospital
133	ROMVA	Num	4	544	6.3	3c.Multiple Trauma Open Wounds.Oth Inj
187	ROOMAIR	Num	4	760	12.9	G4.ABG - Room Air
50	ROTADCAB	Num	4	172	6.3	A6.Transfer: R/O - Initial Adm.CABS
49	ROTADSUR	Num	4	168	6.3	A6.Transfer: R/O - Initial Adm.oth srg
46	ROTLOS	Num	4	156	6.3	A2.Transfer: R/O - LOS >= 2 Days
52	ROTMICOM	Num	4	180	1.	A7b.Transfer: R/O - MI Compl. Or unrel.
51	ROTMIINH	Num	4	176	1.	A7a.Transfer: R/O - MI Happened in Hosp
234	SBP	Num	8	910	3.	B7.First Recorded SBP at Marker Hosp
383	SEP2DTH	Num	8	1831		

The SAS System 15:42 Wednesday, November 15, 2000 32

CONTENTS PROCEDURE

#	Variable	Type	Len	Pos	Informat	Label
247	SERUMCO2	Num	8	1014	4.1	G4.Chemistry (mmol/L) - Total Serum CO2
188	SEX	Num	4	764	10.7	FACE.Sex
398	SEXM	Num	8	1945		Gender (1=M; 0=F)
84	SHOCK	Num	4	348	19.15	D5.Complications - Shock
424	SHOCKP	Num	8	2138		Shock from hospital claim
343	SHOCKX	Num	8	1573		Shock (PE notes or as compl.of stay)
106	SMCESS	Num	4	436	9.6	D2.Smoking Cessation Discussed with Pat
399	SMOKER	Num	8	1953		Smoker
242	SODIUM	Num	8	974	3.	G4.Chemistry (mmol/L) - Sodium
381	SRVDAYS1	Num	8	1815		
379	SRVDAYS5	Num	8	1799		
257	STESTDAT	Num	6	1088	8.	E4.Date of Stress Test
94	STRESTRE	Num	4	388	7.4	E4.Stress Test Reported
189	STROKDEF	Num	4	768	12.9	D5.Types Of Stroke As Complication
140	STSTRES	Num	4	572	19.15	E4.Stress Test Results
141	SWANG	Num	4	576	9.6	D3.Swan-Ganz/Right Heart Cath Inserted
294	TADABDOP	Num	4	1288		A9.Transfer: Diagnosis - Abdominal Pain
290	TADCARD	Num	4	1272		A9.Transfer: Diagnosis - Cardiac Arrest
289	TADCHF	Num	4	1268		A9.Transfer: Diagnosis - CHF
296	TADCOPD	Num	4	1296		A9.Transfer: Diagnosis - COPD/asthma
295	TADCORON	Num	4	1292		A9.Transfer: Diagnosis - Coronary/Myo.
286	TADCPAIN	Num	4	1256		A9.Transfer: Diagnosis - Chest pain
287	TADDYSPN	Num	4	1260		A9.Transfer: Diagnosis - Dyspnea
291	TADMI	Num	4	1276		A9.Transfer: Diagnosis - MI/rule-out MI
288	TADNAUSE	Num	4	1264		A9.Transfer: Diagnosis - Nausea
292	TADOTH	Num	4	1280		A9.Transfer: Diagnosis - Other
293	TADPNEUM	Num	4	1284		A9.Transfer: Diagnosis - Pneumonia
321	TARRDT	Num	8	1400		A9.TARR DATETIME
45	TASPIRIN	Num	4	152	16.13	A11.Transfer : Aspirin Administered
60	TCPKF	Num	8	232	4.	A11b.Transfer : First Recorded CPK
331	TCPKFDT	Num	8	1480		A11b.TCPKF DATETIME
62	TCPKH	Num	8	248	4.	A11c.Transfer: Highest Recorded CPK
332	TCPKHDT	Num	8	1488		A11c.TCPKH DATETIME
40	TCPR	Num	4	132	16.13	A11.Transfer: CPR Administered
57	TDBP	Num	8	208	3.	A10.Transfer: First Recorded DBP
283	TDOCER	Num	4	1244		A4.Transfer doc. - ER record
282	TDOCMD	Num	4	1240		A4.Transfer doc. - MD letter
285	TDOCNO	Num	4	1252		A4.Transfer doc. - none available
284	TDOCTRAN	Num	4	1248		A4.Transfer doc. - Transfer form
123	TEACHPLA	Num	4	504	9.6	J2.Teaching Plan Discussed
41	TEKG	Num	4	136	16.13	A11.Transfer: EKGs Administered
48	TEKGMST	Num	4	164	7.4	A11.Transfer: ST Elevation/Depression

The SAS System 15:42 Wednesday, November 15, 2000 33

CONTENTS PROCEDURE

#	Variable	Type	Len	Pos	Informat	Label
237	TEMP	Num	8	934	4.1	B7.1st Recorded Temp.Marker Hosp
42	TENZYME	Num	4	140	16.13	A11.Transfer: Enzymes Measured
256	THALLDAT	Num	6	1082	8.	E5.Date Thallium Scan Reported
96	THALLREP	Num	4	396	7.4	E5.Thallium Scan Reported
44	THEPARIN	Num	4	148	16.13	A11.Transfer: Heparin Administered
55	THRATE	Num	8	192	3.	A10.Transfer: 1st Recorded Heart Rate
350	THRMBDEL	Num	8	1629		
408	THRMIDL	Num	8	2025		Ideal candidate for thromb.during
414	THRMINT	Num	8	2073		Throm dur: (Ideal * received drug)
349	THRCMBA	Num	8	1621		Thrombolytic therapy (1=yes)
351	THRGMBEX	Num	8	1637		Appropriateness: thrombolytics (0=ideal)
39	TINTUB	Num	4	128	16.13	A11.Transfer: Patient Intubated
64	TLDHH	Num	8	264	4.	A11a.Transfer: Highest Recorded LDH
333	TLDHHDT	Num	8	1496		A11a.TLDHH DATETIME
61	TMBP	Num	8	240	1.	A11b.Transfer: 1st Recorded CPK-MB u/l
63	TMBH	Num	8	256	1.	A11c.Transfer: Highest Rec.CPK-MB u/l
337	TMBPCTF	Num	8	1528		A11b.Transfer First CPK - % MB
338	TMBPCTH	Num	8	1536		A11c.Transfer Highest CPK - % MB
47	TMEDIC	Num	4	160	6.3	A7.Transfer: Adm. for oth Medical Reas
323	TONSETDT	Num	8	1416		A8.TONSET DATETIME
54	TRANFAC	Char	4	188	4.	A3.Transfer:Name Transferring Facility
38	TRANSFER	Num	4	124	7.4	A1.Transferred fr.oth Acute Care Facil
53	TREAS	Num	4	184	19.15	A3.Transfer: Reason For Transfer
58	TRESP	Num	8	216	3.	A10.Transfer: 1st Rec.Respiratory Rate
56	TSBP	Num	8	200	3.	A10.Transfer: First Recorded SBP
196	TTCONTR1	Num	4	796	3.	C4.Contraind. Thrombolytic Therapy
197	TTCONTR2	Num	4	800	3.	C4.Contraind. Thrombolytic Therapy
198	TTCONTR3	Num	4	804	1.	C4.Contraind. Thrombolytic Therapy
59	TTEMP	Num	8	224	4.1	A10.Transfer: 1st Recorded Temperature
102	TTHER	Num	4	420	16.13	B6.Thromb.therapy <=24 hrs.marker hosp
330	TTHERDT	Num	8	1472		B6.TTHER DATETIME
43	TTTHER	Num	4	144	16.13	A11.Transfer: Thromb.ther.<=24 hrs
334	TTTHERDT	Num	8	1504		A11.TTTHER DATETIME
70	VERBAL	Num	4	292	19.15	C12.Worst Condition Initial 24 Hours
239	WBC	Num	8	950	4.1	G4.Hematology - WBC Count
422	WPG	Num	8	2122		
36	XCHRLSCN	Num	3	118		Charlson Index
87	XRAYCHF	Num	4	360	13.10	H1.CHF Noted On Chest X-Ray
89	XRAYCMEG	Num	4	368	13.10	H1.Cardiomegaly - Chest X-Ray
88	XRAYPULX	Num	4	364	13.10	H1.Pulmonary Edema - Chest X-Ray
387	_RANK_	Char	2	1863		

APPENDIX FIVE

Weighted Index of Comorbidity (Charlson et al., 1987).

Conditions	**Assigned Weights for Diseases**
Myocardial Infarction	1
Congestive Heart Failure	1
Peripheral Vascular Disease	1
Cerebrovascular Disease	1
Dementia	1
Chronic Pulmonary Disease	1
Connective Tissue Disease	1
Peptic Ulcer Disease	1
Mild Liver Disease	1
Diabetes	1
Hemiplegia	2
Moderate/Severe Renal Disease	2
Diabetes with End Organ Damage	2
Any Tumor	2
Leukemia	2
Lymphoma	2
Moderate/Severe Liver Disease	3
Metastatic Solid Tumor	6
AIDS	6

Assigned weights for each condition that a patient has. The total equals the score. Example: chronic pulmonary (1) and lymphoma (2) = total score (3).