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TITLE: Factors Associated with Delay in Response to Symptoms of Acute Myocardial Infarction.

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ABSTRACT:

Statement of the Problem: For patients with an acute myocardial infarction (AMI), the time from symptom onset to initiation of medical treatment is a critical determinant of outcome both for survival and for preservation of myocardial tissue. This is true because the effectiveness of thrombolytic therapy is inversely related to time between onset of symptoms and initiation of therapy. Studies have shown that the time that patients take to decide to seek help accounts for most of the delay, a factor that has not improved despite efforts in education campaigns. Thus far, only two studies on delay time have been conducted in Canada. The study objective was to describe the delay time and investigate the factors associated with delay in response to symptoms of AMI in a Canadian setting.

Methods: Medical records for 692 patients admitted to the Ottawa General Hospital emergency department with a final diagnosis of AMI between September 1994 and August 1997 were reviewed to obtain information regarding delay from onset of symptoms to arrival to hospital, demographic factors, and medical history factors. A convenience sample of 68 patients admitted between November 1996 and August 1997 was also interviewed to obtain additional information on sociodemographic, behavioral, and contextual factors. Log-transformed delay time between onset of symptoms to arrival to hospital was used as the outcome variable. Also, bivariate and multivariate linear regression analyses were conducted.

Results: The median pre-hospital delay was 2.1 hours; 69% of patients delayed less than four hours, while 23% delayed more than 6 hours before arriving at the hospital. We found medical history and demographic factors to have poor predictive value in explaining delay time. Multiple linear regression on the *Fastrak* group using only available demographic and

medical history variables showed three variables (gender, diabetes, previous MI) to be independent predictors of delay time with only 2.6% of the log-delay time variability explained. Meanwhile, the linear regression model conducted using the interview group showed that five factors were significant ($p<0.05$) in explaining the variability of log-delay time ($R^2=0.63$). In particular, patients who had a witness who acted like symptoms were serious, and those who had a spouse or other relative making the decision to seek care at the hospital were associated with shorter delay. Meanwhile, patients who tried to relieve their symptoms to a greater extent with self-medication had a longer delay. In addition, patients who experienced no pain recurrence, or who were a current smoker, were found to have a longer delay.

Conclusion: Substantial delay occurs between onset of symptoms and arrival to hospital. Our results suggest that demographic and medical history factors cannot adequately explain why patients delay in responding to their symptoms of AMI. Instead, the patient's perception of the symptoms and their coping behaviors can strongly influence pre-hospital delay time. Education efforts should be aimed at preventing patients from overly relying on self-medication when experiencing symptoms and also encourage patients to inform a spouse or a relative about their symptoms when experiencing a potential cardiac event.

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1. INTRODUCTION

1.1 Background In Cardiovascular Disease

In the last few years, considerable efforts have been placed on identifying factors that place individuals at risk of developing cardiovascular disease and stroke. However, despite these efforts, cardiovascular disease remains the major cause of death, disability and illness in Canada. In 1995, cardiovascular disease was the leading cause of death in the country accounting for 37% of all deaths with ischemic heart disease representing the greatest percentage at 21% (1).

Among those who die from ischemic heart diseases, 50% are attributed to acute myocardial infarction. While the overall age-standardized mortality rate from acute myocardial infarction has been declining over the years (2), the rate increases dramatically in the older age groups. In the coming decades as the proportion of elderly within the population increases, the age-standardized mortality rate due to acute myocardial infarction may in fact increase.

Cardiovascular disease, especially myocardial infarction, has a significant impact on Canada's health care system. On average, patients with cardiovascular disease tend to have a longer length of stay in hospital than other patients (3). In 1992-1993, among those who presented to hospital with an acute myocardial infraction, 16.8% of males and 10.3% of females underwent a revascularization procedure (3). For individuals who made visits to physicians, nearly ten percent (26 million visits) were for cardiovascular disease, and of these visits, fifteen percent were for ischemic heart disease (4). When we consider other direct and indirect costs of cardiovascular disease, there is no doubt that it consumes a

significant portion of the health care dollar and places a major burden on our health care system.

Considerable efforts have been placed on identifying major risk factors for ischemic heart disease. It is now well recognized that smoking, hypertension, hypercholesterolemia, and physical inactivity are major "modifiable" risk factors which contribute to the development of ischemic heart disease. In addition, family history of ischemic heart disease, age, and the male sex are some of the "non-modifiable" risk factors.

1.2 **Acute Myocardial Infarction**

Myocardial Infarction is a condition in which there is irreversible necrosis of heart muscle due to prolonged ischemia. As the oxygen supply to the heart is through the coronary arteries, disruption of blood flow will lead to periods of inadequate oxygen delivery. Myocardial infarction occurs when there is an abrupt decrease of flow due to a thrombotic lesion.

In most cases, patients already have coronary arteries narrowed secondary to atherosclerosis. The progression of the atherosclerotic lesions to the point of thrombus formation is a complex process, which is related to vascular injury. This process is often produced or facilitated by factors such as hypertension, cigarette smoking, and hyperlipidemia. When the atherosclerotic plaque ruptures or ulcerates, the coronary arteries may then be occluded. Rarely, other mechanisms such as coronary spasm or congenital abnormalities may also precipitate a myocardial infarction.

The distinctions between an acute myocardial infarction (AMI) and other ischemic heart diseases are the severity of the ischemic episode and the extent of heart cell damage. In

a myocardial infarction, patients suffer from ischemia for more than 30 minutes, resulting in some level of cardiac cell death. The extent of damage depends upon the territory supplied by the affected vessel, the severity of the occlusion, and the amount of blood supplied to the affected area by collateral vessels.

The World Health Organization (WHO) defines the diagnosis of myocardial infarction (MI) based on the presence of at least two of the following three criteria: (i) a clinical history of ischemic-type chest discomfort; (ii) changes on serially obtained electrocardiographic tracings; and (iii) a rise and fall in serum cardiac markers (5).

1.3 Thrombolytic Therapy

Given that the pathophysiology of myocardial infarction is one of thrombus occlusion, it seems logical that the aim of treatment should be to achieve reperfusion to the affected tissue. The treatment strategy is based on the concept that after a coronary vessel is occluded, a "wave" of myocardial cell death progresses from the endocardium outward to the epicardium, and reperfusion may therefore limit infarct size (6).

Several large-scale randomized clinical trials were conducted to investigate the effectiveness of various thrombolytic therapies in reducing mortality and morbidity (7,8,9). Compared with standard medical therapy, the use of thrombolytic agents has shown a highly significant 21% reduction in 35-day mortality (10), which is consistent with an overall reduction of 21 deaths per 1000 patients treated based on data from more than 10 large, randomized trials that enrolled more than 1000 patients each (11).

Thrombolytic drugs that are currently available include streptokinase, tissue plasminogen activator (rt-PA), and anisoylated plasminogen-streptokinase–activator complex

(APSAD). All the above drugs act thrombolytically by slightly different mechanisms: converting plasminogen to plasmin either directly or indirectly. Studies directly comparing mortality reductions between various thrombolytic agents have generally shown no significant differences in reducing infarct size and improved mortality (12,13,14,15). However, accelerated-doses of tissue plasminogen activator were more efficacious than streptokinase (9).

While thrombolytic therapies has been shown to improve left ventricular function and limit the infarct size, this effect is maximized with very early treatment (16-19). The first Gruppo Italiano per lo Studio della Steptochinasi nell'Infarto Miocardio (GISSI-1) trial provided initial support for the relation between time-to-treatment and clinical outcome. The results showed that mortality was significantly reduced if thrombolytic therapy was administered within the first hour of symptoms, while those randomized after the third hour received little benefit (20).

Similar relationships between time-to-treatment and mortality were shown in the GUSTO trial (9) and the ISIS-2 study (8) each showing that the overall odds reduction for mortality was greater if thrombolytic therapy was initiated sooner. In the Fibrinolytic Therapy Trialists' (FTT) Collaborative Group overview of more than 58,000 patients randomized to either thrombolytic therapy or other measures, significant odds reduction for mortality were 26% for the 0-3hr group, 18% for the 4-6hr group, and 14% for the 7-12hr (11). However, no significant reduction in mortality was found for those receiving thrombolytic therapy more than 12 hours after onset of symptoms.

On an average the mortality rate increased by 1.6 per 1,000 patients, for each additional hour of delay in administering thrombolytic agents (21). This effect is quite

significant with an estimated 35 lives saved per 1,000 patients when thrombolysis is started in the first hour after onset of symptoms, compared to only 16 lives saved per 1,000 treated within 7 to 12 hours of symptom onset (10).

Eighty years after Herrick (22) first introduced the concept that acute myocardial infarction was due to acute coronary thrombosis, thrombolytic therapy has now become the standard treatment for acute myocardial infarction. According to the recommendations from the 5th ACCP Consensus Conference on Antithrombotic Therapy, all patients presenting with characteristic symptoms and ECG criteria of an acute myocardial infarction should be given thrombolytic therapy within 6 hours of symptom onset (23). For those presenting between 7 to 12 hours of symptom onset, there may still be benefit from thrombolytic treatment, but the benefit vs. risk of therapy must be considered. The guidelines however recommend that patients presenting more than 12 hours after symptom onset should not receive thrombolytic therapy routinely. Furthermore, at a National Emergency Pre-hospital Care Conference held in Ottawa, it was suggested that the window of opportunity from onset of symptoms to reaching emergency is now 90 minutes (24).

1.4 Delay in Presentation

Despite the fact that effectiveness of thrombolytic therapy is inversely related to time between the onset of symptoms of acute myocardial infarction and therapy, significant delays occur between symptom onset and initiation of medical treatment (25). In many cases, patients are ineligible for thrombolytic therapy by the time they present to the hospital for treatment (26).

The median and mean delay times between onset of AMI symptoms and patient arrival at the hospital varies markedly within and across studies (Table 1). In general, median delay times range from just under 2 to 4 hours, although some have reported unusually long delays of over 6 hours (27,28). The difference is likely due to differences in the definition of time of onset. While Dracup et al. reported the duration from onset of first symptoms until hospital presentation (28), other investigators reported the time of onset of symptom which initiated action until hospital presentation (29) which does not take into consideration the duration of any preceding intermittent symptoms.

Regardless of the variability, all trials reported significantly longer mean delay times than the median times with many exceeding 6 hours (29-41) and one > 21 hr (27). The difference between the mean and median time is due to the skewing effect caused by individuals who wait many hours or even days before seeking medical attention for their AMI. Overall, approximately 25% to 50% of MI patients delay seeking medical care for cardiac symptoms for more than 6 hours (42,43).

The largest component of delay to treatment is due to patient delay in seeking care, a factor that has not improved over the past 30 years, despite educational campaigns (30,33,50). For example, an intense media campaign specifically intended to shorten this delay time conducted in Gothenburg, Sweden in 1987 (59) only managed to decrease median delay between symptom onset and hospital arrival from 3 hours to 2 hours and 35 minutes among the patients.

Table 1. Reported Pre-hospital Delay Time (in hours).

Author	Sample size	Mean	Median
Hackett et al., 1969 [57]	100 confirmed	10.6	4
Moss et al., 1969 [38]	64 confirmed	5.1	
Schroeder et al., 1978 [30]	211 suspected	7.6	3.5
Alonzo et al. 1986 [37]	1102 suspected		2.2
Turi et al., 1986 [52]	778 confirmed		2
Cooper et al., 1986 [27]	111 confirmed	20.2	6.4
Wielgosz et al., 1988 [32]	201/43 suspected	6.9 / 6.5	2.7 / 3.4
Ho et al., 1986 [33]	135 confirmed		2.6
Leitch et al., 1989 [29]	100 suspected		2
Karlson et al., 1990 [58]	908 confirmed		3.2
Schmidt et al., 1990 [53]	126 confirmed	5.9	2
Hartford et al. 1990 [66]	234 confirmed		2
Kenyon et al., 1991 [50]	103 confirmed	9.0	5.0
Goldberg et al., 1992 [51]	800 confirmed	4	2
Ridker et al., 1992 [81]	258 confirmed		1.8
Ell et al., 1994 [31]	200 suspected	9.1	2.7
Yarzebski et al. 1994 [72]	1279 confirmed	3.7	1.9
Burnett et al., 1995 [25]	453 confirmed	3.1	1
GISSI group, 1995 [43]	5301 confirmed	8.3	3.8
Newby et al., 1996 [54]	41021 confirmed		1.5
Dracup et al., 1997 [28]	317 confirmed		6.4

1.5 Components of Delay

Most studies define delay time as the amount of time that elapses from the first awareness of symptoms to the arrival of hospital. However, given the importance of time-to-treatment for thrombolytic therapy, the total delay time from the onset of symptoms to initiation of reperfusion therapy is perhaps the most important. Many investigators have divided total delay time into numerous phases, ranging from just three (34,36) to as many as eleven (35). For practical purposes, total delay time is best divided into three phases:

Patient Decision Delay. The patient delay phase comprises the time between onset of symptoms and the decision to call emergency medical services (EMS), or initiate travel to the hospital if travel is by other methods. The definition of symptom onset must be clear as approximately one-third of AMI patients do not report an abrupt onset of symptoms and often have difficulty identifying the time of onset (45). Many patients experience a prodromal period when they experience vague symptoms or symptoms that wax and wane over time, sometimes disappearing completely. These initial mild symptoms do not tend to cause individuals to seek medical care. It is the onset of more acute severe symptoms that usually leads to the decision to seek medical care.

Unfortunately, many patients experiencing acute symptoms do not initially believe they have cardiac problems, leading to prolonged delays in seeking medical care. Reilly et al. found that although 49% of patients thought their symptoms were cardiac-related, 31% believed they had indigestion, and 8% tried to ignore the symptoms (46). It was also found that patients' perception of AMI symptoms did not match the actual symptoms experienced by 74% of patients who were surveyed (47) and these patients delayed significantly longer than those whose expectations matched their experience.

When patients recognize their acute symptoms, they may still engage in a variety of behaviors that can affect the patient delay time. Patients may decide to seek medical care at once; they may decide to wait and periodically evaluate or self-treat their symptoms; or they may decide to seek consultation from friends, relatives, or medical personnel (45). In the majority of cases, patients will inform someone they are ill and this lay advice tends to have an impact on the behavior of the patient (37).

EMS/Travel Delay. The EMS/travel delay time is the interval between calling EMS and the patient's arrival at the hospital. For those who arrived at the hospital by other means, this phase refers to the period between initiating travel to the hospital (including preparation) and arrival at the hospital. In most cases, the time spent travelling to the hospital is a fundamental component of this phase. For transport by EMS, this phase is affected by access (i.e. ability to call for help), EMS response time, as well as transportation.

With the improvement of EMS services in North America, the mean delay time for this component ranges from 7 to 22 minutes (30,32,35,38), which is insignificant when viewed in relation to the average total delay time.

Hospital Delay. Between hospital arrival and initiation of thrombolytic therapy, the patient with AMI may experience delay in receiving definitive care. Delays can occur during patient registration, triage, entry to examination room, history and examination by physician, obtaining 12-lead ECG, consultation with a cardiologist, decision to order thrombolytic therapy, and delay in administration of ordered therapy.

The hospital delay has been reported to last for approximately 60-90 minutes (39-40,72). While it represents a small component of the total delay time compared to the patient delay, increasing attention has been placed on hospital delay time with attempts to streamline

the triage system (48). The most recent study to investigate delay to thrombolysis in Canada also included analysis on hospital delay times and reported a median delay of 59 minutes (49). The study also reported increased hospital delay for women, older patients, and those with a history of MI.

1.6 Factors affecting Patient Delay

Studies investigating factors affecting patient delay often have methodological differences and inherent limitations that lead to conflicting results. Researchers may include only proven AMI patients in one study, while others include both suspected and documented cases. The definition of symptom onset is also variable, thereby making comparison between studies difficult. In addition, studies were often conducted in different countries and regions that may differ in their system of medical care and culture. Nevertheless, Dracup et al. have reported a thorough overview of the results (45). Effects of various factors investigated in the past are presented in Table 2 to Table 5.

Age. While the evidence for the effect of age on delay time is conflicting, it is now generally accepted that older age is associated with an increase in patient delay time (46). While some investigators reported that age did not affect patient delay time (30,32,37,50,51), most of these studies had small sample sizes and lacked the statistical power to detect the differences. In Canada, Wielgosz et al. (32) studied 201 patients admitted for suspected AMI and found no relation between age and delay.

Recently, studies with large sample sizes have been conducted showing a direct relationship between delay and age, with older patients delaying more (41,52-54). In contrast to the smaller studies conducted earlier, Newby et al. found longer delays for elderly

Table 2. Reported Effect of Social Demographic Variables on Prehospital Delay.

Author	Sample size	↑Age	FemaleSex	Race	Low Income	Lower Education	Married
Hackett et al., 1969 [57]	100 confirmed	+	+/-		+/-		
Moss et al., 1969 [38]	64 confirmed	+	+			+/-	
Schroeder et al., 1978 [30]	211 suspected		+/-				
Matthews et al., 1983 [80]	43 confirmed	+/-	+/-	-	+/-	-	
Alonzo et al., 1986 [37]	1102 suspected	+/-	+	+			
Turi et al., 1986 [52]	778 confirmed	+	+	+/-		+/-	
Cooper et al., 1986 [27]	111 confirmed			+			
Wielgosz et al., 1988 [32]	201 (43) suspected	+/-	+/-		+/-	+/-	
Rawles et al., 1988 [79]	450 confirmed	+					
Cunningham et al., 1989 [56]	7734 suspected		+				
Maynard et al., 1989 [55]	690 confirmed	+	+/-				
Karlson et al., 1990 [58]	908 confirmed	+	+/-				
Schmidt et al., 1990 [53]	126 confirmed	+	+		+	+/-	+/-
Hartford et al., 1990 [66]	234 confirmed	+/-	+/-				
Kenyon et al., 1991 [50]	103 confirmed	+/-	+/-	+/-	+/-	+/-	+/-
Goldberg et al., 1992 [51]	800 confirmed	+/-	+/-				
Meischke et al., 1993 [41]	2947 confirmed	+	+				
Heriot et al., 1993 [77]	103 confirmed	+/-					
Yarzebski et al., 1994 [72]	1279 confirmed	+	+/-				
GISSI group., 1995 [43]	5301 confirmed	+	+			+/-	+/-
Burnett et al., 1995 [25]	453 confirmed	+/-	+/-	+/-		+/-	-
Newby et al., 1996 [54]	41021 confirmed	+	+	+/-			
Dracup et al., 1997 [28]	317 confirmed	+/-	+/-	+/-	+	+	

+/-, no effect on delay; +, increase in delay; - , decrease in delay; blank, not studied.
Race: Black race compared to white race

Table 3. Reported Effect of Medical History Variables on Pre-hospital Delay.

Author	Sample size	AMI	Angina	CAD	DM	HTN	Smoking	CHF	CABG	PTCA
Moss et al., 1969 [38]	64 confirmed	+/-	+/-			+/-		+/-		
Hackett et al., 1969 [57]	100 confirmed	+/-								
Schroeder et al., 1978 [30]	211 suspected	+/-	+	+/-						
Turi et al., 1986 [52]	778 confirmed	+/-	+/-		+	+	-	+	+/-	
Wielgosz et al., 1988 [32]	201 (43) suspected	+/-	+/-							
Rawles et al., 1988 [79]	450 confirmed			+/-						
Lietch et al., 1989 [29]	100 suspected			+/-						
Maynard et al., 1989 [55]	690 confirmed	+/-								
Hartford et al., 1990 [66]	234 confirmed	+/-	+/-		+/-	+/-				
Karlson et al., 1990 [58]	908 confirmed	-	+/-		+/-	+/-		+/-		
Schmidt et al., 1990 [53]	126 confirmed	-	+/-		+/-	+	+/-		+/-	
Kenyon et al., 1991 [50]	103 confirmed	+/-								
Goldberg et al., 1992 [51]	800 confirmed		+/-		+/-	+/-				
Meischke et al., 1993 [41]	2947 confirmed	-	+		+			+		
Yarzebski et al., 1994 [72]	1279 confirmed	+	+/-		+	+/-				
GISSI group., 1995 [43]	5301 confirmed	+/-			+					
Newby et al., 1996 [54]	41021 confirmed	-	+		+	+	+/-		+/-	+/-

AMI: Acute Myocardial Infarction; CAD: Coronary Artery Disease; DM: Diabetes; HTN: Hypertension; CHF: Congestive Heart Failure; CABG: Coronary Artery Bypass Grafting; PTCA: Angioplasty

+/-, no effect on delay; +, increase in delay; - , decrease in delay; blank, not studied.

Table 4. Reported Effect of Counterroles on Pre-hospital Delay.

Author	Sample size	Home Onset	Weekend Onset	Contact Family Doctor	Transport by Car	Severe Pain
Hackett et al., 1969 [57]	100 confirmed					-
Moss et al., 1969 [38]	64 confirmed		+			
Alonzo et al., 1986 [37]	1102 suspected	+		+		
Turi et al., 1986 [52]	778 confirmed	+				
Lietch et al., 1989 [29]	100 suspected			+		+/-
Maynard et al., 1989 [55]	690 confirmed					+/-
Hartford et al., 1990 [66]	234 confirmed	+		+	+	-
Karlson et al., 1990 [58]	908 confirmed				+	
Schmidt et al., 1990 [53]	126 confirmed	+/-		+	+/-	
Kenyon et al., 1991 [50]	103 confirmed					-
Goldberg et al., 1992 [51]	800 confirmed		+/-			-
Heriot et al., 1993 [77]	103 confirmed			+		
Yarzebski et al., 1994 [72]	1279 confirmed		+/-			
Ell et al., 1994 [31]	200 suspected			+	+	-
GISSI group., 1995 [43]	5301 confirmed	+		+	+	
Burnett et al., 1995 [25]	453 confirmed	+/-	+/-			+/-
Dracup et al., 1997 [28]	317 confirmed	+/-			+	-

+/-, no effect on delay; +, increase in delay; - , decrease in delay; blank, not studied.

Table 5. Reported Effect of Patient's Coping Behaviors On Pre-hospital Delay.

Author	Sample size	Attribute Symptoms to Heart	Consider Symptoms as Serious	Self Treat	Consult with Others
Hackett et al., 1969 [57]	100 confirmed	-			+
Moss et al., 1969 [38]	64 confirmed				+
Wielgosz et al., 1988 [32]	201 (43) suspected			+	-
Hartford et al., 1990 [66]	234 confirmed	-			- †
Schmidt et al., 1990 [53]	126 confirmed			+/-	
Kenyon et al., 1991 [50]	103 confirmed	-			
Heriot et al., 1993 [77]	103 confirmed	-			
Ell et al., 1994 [31]	200 suspected				-
GISSI group., 1995 [43]	5301 confirmed				-
Burnett et al., 1995 [25]	453 confirmed	-	-		+/-
Dracup et al., 1997 [28]	317 confirmed		-		+/- †

+/-, no effect on delay; +, increase in delay; - ,decrease in delay; blank, not studied

† if consulted with spouse

patients when they evaluated approximately 41,000 patients (54). Further evidence was provided by Meischke et al. when multiple regression analyses conducted on 2947 patients found that the prehospital delay interval was significantly greater for individuals who were older (41).

Gender. Of those studies that included gender as a factor contributing to delay in their analyses, some have reported a lack of a relationship between gender and delay (32,50,55), while others have found women delaying longer than men (37,38,41,54,56). In all studies that found a difference between the two genders, women have consistently delayed longer. Alonzo et al. (37) reported a median of 47 minutes longer for women to seek medical treatment, while another study found women delayed a mean of 3.3 hours longer than men (38).

While some investigators reported no statistically significant differences between gender in some earlier studies with smaller sample size, the differences were often clinically significant (57,58). In one of the earliest reports on delay times, Hackett and Cassem found women delayed a median of 1 hour longer than men. However, no statistically significant differences were noted due to the fact only 64 men and 24 women were compared.

Ethnic Origin. In most of the literature investigating delay, subjects were white middle- and upper-class males (45). Of those who investigated the importance of race, black and Hispanic patients had a markedly longer delay (27,60,61). Clark et al. (61) reported longer median (3 hours) and mean ± standard deviation delay times for blacks (13.1 ± 25.5 hours) and Hispanics (median, 4 hour; mean, 12.4 ± 2.9 hours) than for whites (median, 2 hours; mean, 3.3 ± 2.9 hours). This is consistent with another report which found twice as

long median delay and three times as long mean delay for black when compared to white populations (27). On the other hand, no differences were reported between white and Asians in Britain (62).

Socioeconomic Status. The few studies examining the effect of income on patient delay time found no significant association (32,50,57,80), although most of these were conducted using small sample size and among a white population. The fact that many studies conducted among African Americans point to a significant increase in delay may in fact be related to a confounding effect of ethnicity and economic status. This view was supported by Cooper (27) and Ell (31) who conducted their studies in hospitals serving populations that were poor and underprivileged. The importance of the health care delivery system must also be considered. In Canada, where all patients receive universal health care coverage, no differences in delay times were reported between those with lower and higher income (32).

Education/Knowledge of MI. Interestingly, researchers who included education level as a factor in their studies have generally found no relation between educational levels and delay times (31,32,38,52). This result may also explain why several educational campaigns aimed at increasing knowledge of symptoms of MI to reduce delay times have failed to produce a significant reduction (33,59,63). In addition, studies that investigated the effects of knowledge of AMI symptoms have found that it does not improve the patient's ability to recognize their symptoms (61) and does not lead to a reduction in patient delay time for seeking medical treatment (29).

Medical History. The importance of medical history has been investigated in many studies. Contrary to the assumption that patients who had a history of prior AMI or coronary

heart disease would seek medical help earlier when symptoms occur, studies have consistently documented that a past medical history of AMI has no effect on patient delay times (32,38,50,55,57). Factors studied include previous AMI, history of arteriosclerosis, previous bypass surgery, angina, hypertension, congestive heart failure, and diabetes mellitus. In some studies, patients with a history of angina (30,41), hypertension (52,54), and diabetes (30,41,43,54) actually had increased patient delay times.

Clinical Factors. Several investigators have examined the relationship between delay time and various clinical factors. Trent et al. reported that AMI patients with more severe deterioration of left ventricular function had a shorter delay after the onset of symptoms (64). Others found patients with large transmural infarctions (65), and who were hemodynamically unstable to have decreased delay times (52,55). These factors may in turn translate to increased severity of symptoms that prompted patients to seek medical care for their myocardial infarct.

Psychological and coping factors. Studies that investigated the importance of psychological and coping factors consistently found significantly shorter delay in patients who were more comfortable in seeking medical assistance, feeling symptoms were serious, and more anxious or upset when first noticing symptoms. Such patients were more likely to attribute their symptoms to their heart (25,32). In turn, studies found patients who attribute their symptoms to the heart had shorter delay time (25,50,57,66). Kenyon et al. further suggested that patients who had decreased emotional awareness and low levels of somatic awareness display the greatest mean delay (50). Meanwhile, Wielgosz et al. reported longer delay for patients who attempted to self-medicate when experiencing symptoms of myocardial infarction.

Counterroles. Few investigators have examined the social context and the ways witnesses play in influencing the patient's decision to seek medical treatment (45). In simple terms, counterroles refer to the role another person play in influencing the action of the patient. For example, when a patient suffers from certain symptoms, he or she may communicate this to another person, and in turn the behaviors and actions of this person may influence the action of the patient to either take medication or request for medical assistance. In most cases, patients suffering from a heart attack arrive at the decision to seek medical treatment for symptoms with the assistance of a family member (57). However, it has been reported that this has resulted in longer delays than if the patient comes to the decision alone (38,57). This is particularly disturbing since patients are often instructed to inform their spouse or family member when they notice symptoms of an acute attack. On the other hand, several studies reported the contrary and found patients who consult with others had shorter delay time (31,32,43,66). In particular, Alonzo et al. reported that the shortest delays occurred when an unrelated person (coworker, friend, or stranger) was providing advice in the decision to seek medical care (37).

The timing of the event has been the subject of investigation in a few studies. In a study by Moss, patients who came to the hospital during the weekend had a significantly longer delay than those who developed chest pain on a weekday (38). However, Weilgosz reported the opposite (32) and also found no association between the time of day symptoms begin and delay time. Other studies, on the other hand, reported shorter delay time if symptoms occur during the nighttime (37-39).

While consulting with their family physician seems to be a logical step in the decision process, those who do have been shown to take longer to get to the hospital, particularly

when compared to those who utilize EMS or turned to a friend or relative for assistance (39,66). This increase in delay times may be due to the inability of patients to contact their physician, recommendations from their doctor to self-medicate, or failure of the family physician to accurately interpret the history as cardiac in nature.

For those who develop symptoms at home, delay was found to be significantly longer than at work or in a public place (39,43,66). It was reported that those whose symptoms started at work had the shortest delay time (66). This may be related to the influence other people have on the patient's decision to seek medical attention.

Summary

In summary, patient delay times are increased in females and in the elderly. Individuals who are Black or Hispanic also have a longer delay as well as those of lower socioeconomic status. Shorter delay times occur in patients who are hemodynamically unstable, patients with large infarcts, and those with poor left ventricular function. Patients with a history of AMI or coronary heart disease do not have a shorter delay as would be expected. In some studies, a history of angina, hypertension, and diabetes are actually associated with longer delay times. In addition, knowledge about symptoms of AMI does not improve the response time of patients in seeking treatment. Finally, the social context in which the AMI occurs has been found to have an influence on the patient delay time.

1.7 Models of Health Behavior

Most of the literature that deals with patient delay has focused on the characteristics of the patient in explaining the reason for delay. While some recent studies began investigating the importance of the social context and personality type (37,50), the true interactions between

different variables in causing delay are not clearly understood. The concept of a theoretical model in explaining the causes of delay has gained interest as researchers attempt to understand the complexity of help seeking behavior in patients with AMI (45,67).

The details of the various proposed health behavior models are beyond the scope of this project, nor are they the focus of our current objectives. However, one should be aware of the common models proposed to explain the help-seeking behavior of AMI patients. According to Dracup et al., three models used in the area of patient compliance seem to hold promise for explaining AMI patient behavior: i) the health belief model; ii) self-regulation model; and iii) interactionist role theory.

Health Belief Model. The health belief model is perhaps one of the best known models applied to understand patient compliance (68). This model suggests that patients suffering from a heart attack will decide whether to seek medical help depending on the perceived threat related to the symptoms, and the value of the action being considered. Both perception of threat and the value of the action are in turn affected by demographic factors, patient attitudes and interaction between the patient and other individuals.

Perceived threat by the patient will be affected by the patients' feelings of vulnerability to the illness, previous experience of the symptoms, and the extent of the symptoms. The value of the action is based on the patient's view on whether taking the action will improve his or her health status, and the balance between cost and benefit for such action.

Self-regulation Model. The self-regulation model conceptualizes the patient as an active problem solver (69). When faced with a traumatic experience, the patient must first identify the signs and symptoms based on his or her knowledge about the disease, previous

experiences of the symptoms, and his/her sense of vulnerability. Following this first stage, the patient engages in coping mechanisms to deal with the perceived threat. Finally, in the last stage, the patient will assess the situation and develop the coping strategy.

Interactionist Role Theory. This model is thought to be useful in explaining help seeking behavior as it includes the important roles other people play in the process. The behaviors of the patient are believed to be constantly redefined, based on the interactions of other individuals. The model involves four components: i) Act of decision; ii) Self-concept; iii) Counterroles; iv) Periodic evaluation (70).

The first component involves the patient identifying the health problem and then taking some action such as self-medication, or calling his/her doctor. Self-concept refers to the individual's sociodemographic characteristics, past experiences and personality traits which may all play a role in their behavior. The counterroles, as discussed earlier, are played by any individual the patient interacts with during the traumatic episode and have a significant impact on the decision making process of the patient. Finally, the patient will evaluate symptoms, actions, and interactions with others.

1.8 The Canadian Situation

While many studies have been conducted to investigate the issue of patient delay in MI patients, the vast majority of these were conducted in the United States, which has a very different health care structure than Canada. Thus far, only two studies have been conducted in Canada (32,49) with one conducted more than ten years ago, and the other recently published after the start of this current project. In both studies, delay time between onset of symptoms and arrival to emergency department were excessive. One important aspect that

should be examine is whether delay time for patients suffering from an acute myocardial infarction has in fact decreased over the years due to better understanding of the importance of prompt presentation to the hospital.

The universal access to health care services in Canada may affect the delay time and decisions differently than in other countries since health care costs should not be a factor in seeking care in Canada. Therefore, it is important to investigate in detail the factors associated with patient delay time in response to symptoms of acute myocardial infarction in a Canadian setting. Furthermore, identifying the factors that contribute to excessive delay time can be extremely valuable in shaping the public health message aimed towards modifying any behaviors that may help in decreasing delay time to thrombolytic therapy and in turn, improve survival and cardiac function. The current recommendation from most cardiologists and the Heart and Stroke Foundation is that patients should inform someone when experiencing any symptoms that may resemble a myocardial infarction event. The utility of such message needs to be revisited.

In addition, recent cutbacks to health care and restructuring in our health care facilities may affect the in-hospital response time when the patient arrives at the emergency department, and should be examined and compare with that of other health care facilities in the country. The *Fastrak* project was initiated with these goals in mind in order to examine the current practices of treatment for myocardial infarction at various centers across Canada. The Ottawa General Hospital participated in this multi-center study and all patients recorded with a confirmed diagnosis of myocardial infarction since September 1st, 1994 were identified and included as subjects in the *Fastrak* database. Demographic and treatment

information for patients who seek cardiac care was collected by a trained person through reviewing medical charts, emergency and discharge records.

2. OBJECTIVE OF THESIS

Overall Objectives:

To identify factors that may contribute to delay in responding to signs and symptoms of acute myocardial infarction. Modifiable factors identified may then be used to formulate public health messages that may help in decreasing the amount of patient delay time among those who suffer from a myocardial infarction.

Specific Objectives:

(1) To describe the delay time in seeking treatment for symptoms of acute myocardial infarction among patients admitted to the Ottawa General Hospital, and compare and contrast with those previously reported in other studies.

(2) To examine the effects of demographics and previous medical history on delay time in seeking treatment for symptoms of acute myocardial infarction using information collected in the *Fastrak* database. Specific factors are age, sex, previous experience of cardiovascular diseases, past cardiovascular related procedures, and various cardiovascular risk factors.

(3) To examine the effects of additional demographics, medical history factors, and contextual variables in affecting patient delay time by collecting information from a sub-group of acute myocardial infarction patients admitted to the Ottawa General Hospital. These factors include education, occupation, marital status, the location where symptom onset occurred, time of day the event occur, type of transportation used to arrive at the hospital, etc.

(4) To examine the role spouses or long-term partners play in affecting delay time by comparing delay time of those who informed their spouse first concerning their acute myocardial infarction and those who informed other type of witnesses. The effects of patients' coping behaviors on delay time will also be examined by comparing delay time based on various behavioral and psychological factors.

(5) To examine the importance of spousal relationship on delay time through administration of a previously validated questionnaire (abbreviated dyadic adjustment scale) with patients who are married, and determine its significance through correlation and multivariate analysis.

3. METHODS

3.1 Study Population

***Fastrak* Database.** The *Fastrak* project is an on-going multi-center study across Canada. All patients at the Ottawa General Hospital with a recorded diagnosis of myocardial infarction since September 1st, 1994 were identified and included as subjects in the *Fastrak* database. This included patients who were admitted to the hospital for myocardial infarction or those who developed a myocardial infarct while admitted to hospital for a different diagnosis. Demographic and treatment information of patients who sought cardiac care was collected to study the trends and practices of thrombolytic therapy. For patients who developed their heart attack outside of the hospital, only those who were admitted to the hospital were included in the database, including those who died while being treated in the emergency department. However, patients who died before arriving to the hospital (dead on arrival) were not included. All patients who had a confirmed diagnosis of myocardial infarction were identified from the hospital record. A trained person reviewed the medical charts of these patients, and recorded specific information using a standardized data entry form (Appendix A). All data was then entered into a database using Microsoft Access.

The diagnosis of myocardial infarction was determined clinically by the attending cardiologist based on the World Health Organization definition with the presence of at least two of the following three criteria: (i) a clinical history of ischemic-type retrosternal chest pain with or without radiation to arm, neck, jaw, back, or shoulder, usually lasting more than 20 minutes; (ii) changes on serially obtained electrocardiographic tracings showing new Q waves or new repolarization changes such as ST segment elevation and inverted T waves;

and (iii) a rise in serum cardiac markers (total CK and/or CKMB), follow by return to baseline 3 to 4 days after the event (5).

Exclusion Criteria. For the purpose of our study, we excluded patients who were: (i) transferred to the Ottawa General Hospital from another hospital; (ii) already in the hospital for other reasons when the cardiac event occurred; (iii) had no reported time for the onset of myocardial infarction; and (iv) had onset after August 31st ,1997.

Interview Subgroup. In order to gather additional information for analysis, a convenience sample of patients admitted through the emergency department of the hospital for myocardial infarction between November 1996 and August 1997 were interviewed. The coordinator of the *Fastrak* project identified all new patients with acute myocardial infarction through hospital admission records every other day and notified the author of the new admissions.

After they were transferred from the emergency department or Intensive Care Unit to the general ward for recovery, we approached these patients for an interview if they were well enough. If the patient was unavailable or not well enough to be interviewed but agreed to participate, another suitable time was arranged to obtain the necessary information. An effort was made to interview all potential subjects and obtain the information as soon as possible to avoid reporting bias. However, some subjects refused to participate or were discharged before we had the opportunity to interview them. Therefore, inclusion into the interview sample was based on the availability of the patient and their voluntary participation. The author only conducted interviews in English, and patients who did not speak English were not included as subjects.

In order to improve the accuracy of the information reported by the patients, attempts were made to corroborate the information with a family member or witness if they were available and present at the time of the AMI. We piloted the questionnaire and the procedures on a small sample of convenience before the actual data collection began. The data were entered into Microsoft Excel by the author and linked with the corresponding information recorded in the *Fastrak* database through the hospital chart number.

3.2 Consent

Patients identified as potential subjects for the interview group were contacted by the author during their stay in the hospital. The objectives of the study were explained to the patient and they were asked if they would be willing to participate. Before the interview was conducted, all participants signed a written consent (Appendix A). All information provided was kept confidential and patients will not be identified in any reports. Only English speaking patients were recruited for the interview subgroup. The study protocol was approved by the Research Ethics committee of the Ottawa General Hospital.

3.3 Variables

***Fastrak* Database.** The *Fastrak* database included information on more than 100 variables relating to the cardiac event of each patient. A trained person obtained all the information through medical chart review (Appendix B). For the purpose of our analysis, we were mostly interested in pre-hospital factors.

For each patient, demographic and medical history information was recorded including: gender, age, weight, history of previous myocardial infarct (MI), smoking, angina,

diabetes, coronary artery bypass grafting (CABG), stroke, congestive heart failure (CHF), percutaneous coronary angioplasty (PTCA), hypertension, and hypercholesterolemia.

In addition, the date and time of: MI onset, arrival to the Ottawa General Hospital, thrombolytic IV ordered, and thrombolytic IV started were noted. The time of MI onset, defined as the time when patients first experienced the symptoms that eventually prompted them to seek medical help, was determined by the attending cardiologist based on the patient's presenting history and interpretation by the attending cardiologist. In most cases, this is based on onset of typical symptoms of retrosternal chest pain, and/or radiation to jaw and arm. For patients who did not have typical presentation of chest pain but had vague symptoms that waxed and waned, the onset symptoms were determined to be symptoms that were similar to the ones that finally prompted the patient to seek medical help. For example, if a patient had no chest pain but developed shortness of breath and eventually decided to seek medical care because of worsening shortness of breath, then the onset symptom would be considered as shortness of breath. On the other hand, patients who suffer from typical symptoms of acute myocardial infarction will often have much clearer onset of symptoms that can be easily identified. Regardless, the determination of onset symptoms is heavily dependent on the interpretation by the attending cardiologist.

Based on the information collected, we calculated the MI onset to arrival delay time for all cases. For those patients who received thrombolytic therapy, we also calculated delay times for: arrival to thrombolytic therapy ordered; thrombolytic IV ordered to administered; total in-hospital delay; and total delay from MI onset to IV administered. We were also able to determine the proportion of onset to IV administered delay that was attributable to in-hospital delay.

Interview Subgroup. Interviews were performed by the author using a standardized questionnaire designed for the study (Appendix C). The information obtained included demographics, medical history, contextual, and behavioral variables. Demographic data included age, gender, marital status, education, and occupation. Medical history variables, particularly relating to cardiovascular risk factors were also obtained along with any family history of myocardial infarction and heart disease.

The context in which the myocardial infarction occurred was carefully investigated by noting the time of onset and the time when the patient decided to seek medical assistance for the cardiac event, location at which the patient experienced the onset, and when he or she decided to seek help. Patients who contacted their family physician before coming to the hospital were identified, and the mode of transportation was also noted.

The questionnaire elicited detailed information regarding the behavior of the patient when experiencing the cardiac event. Patients were asked with whom they discussed their symptoms before the decision was made to seek treatment, and the roles these individuals played in the decision making process. Finally, the patients' perception of the cardiac symptoms and their behavior were studied through a series of questions, noting the extent to which they: thought about what their symptoms meant; thought it was serious; tried to ignore the symptoms; thought about the cause; thought about other individuals with similar symptoms; tried to relieve their symptoms with self-medication; and tried to lie down and rest.

The Abbreviated Spanier Dyadic Adjustment Scale (ADAS) questionnaire was included in our interview to evaluate the relationship of the patient with a spouse or long-term partner so that we may determine whether this will affect the delay time of the patient.

While the original Dyadic Adjustment Scale (DAS) was designed to measure the degree of satisfaction reported by individuals across 31 aspects of marital interaction plus one global item of "overall happiness", the abbreviated form of the DAS comprises 6 items plus a global item with a total score ranging from 0 to 36. It was reported that the ADAS correctly classified 92% of 95 respondents in a preliminary study. Furthermore, in a validation study based on 545 subjects by Sharpley et al., those who were separated or divorced had a significantly lower score compared to those who were married, and it was concluded that ADAS is a reliable and valid tool to rapidly screen for marital adjustment (71).

3.4 Distribution

The distributions of patient delay time in both the *Fastrak* group and the interview group were heavily skewed due to a small number of patients who had excessively long delays (Figure 1 and 2). In order to satisfy the assumptions of normal distribution for t-test and ANOVA, patient delay time used in statistical analysis was transformed to obtain a normal distribution. Furthermore, while multiple regression does not require normality of the dependent variable, it does require that for each combination of values of the independent variables, the distribution of the dependent variable to be normally distributed with constant variance. Residuals for linear regression using delay time for the *Fastrak* group was not found to be normally distributed therefore suggesting the need of mathematical transformation in order to satisfy the assumptions of regression models (Figure 3 and 4).

The natural log transformation was found to provide the best approximation to a normal distribution (Figure 5 and 6), and the normal Q-Q plot approximated closely to a straight line suggesting our natural log transformed data was close to a normal distribution (Figure 7 and

Figure 1. Distribution for onset to arrival delay time for *Fastrak* database (in hours)

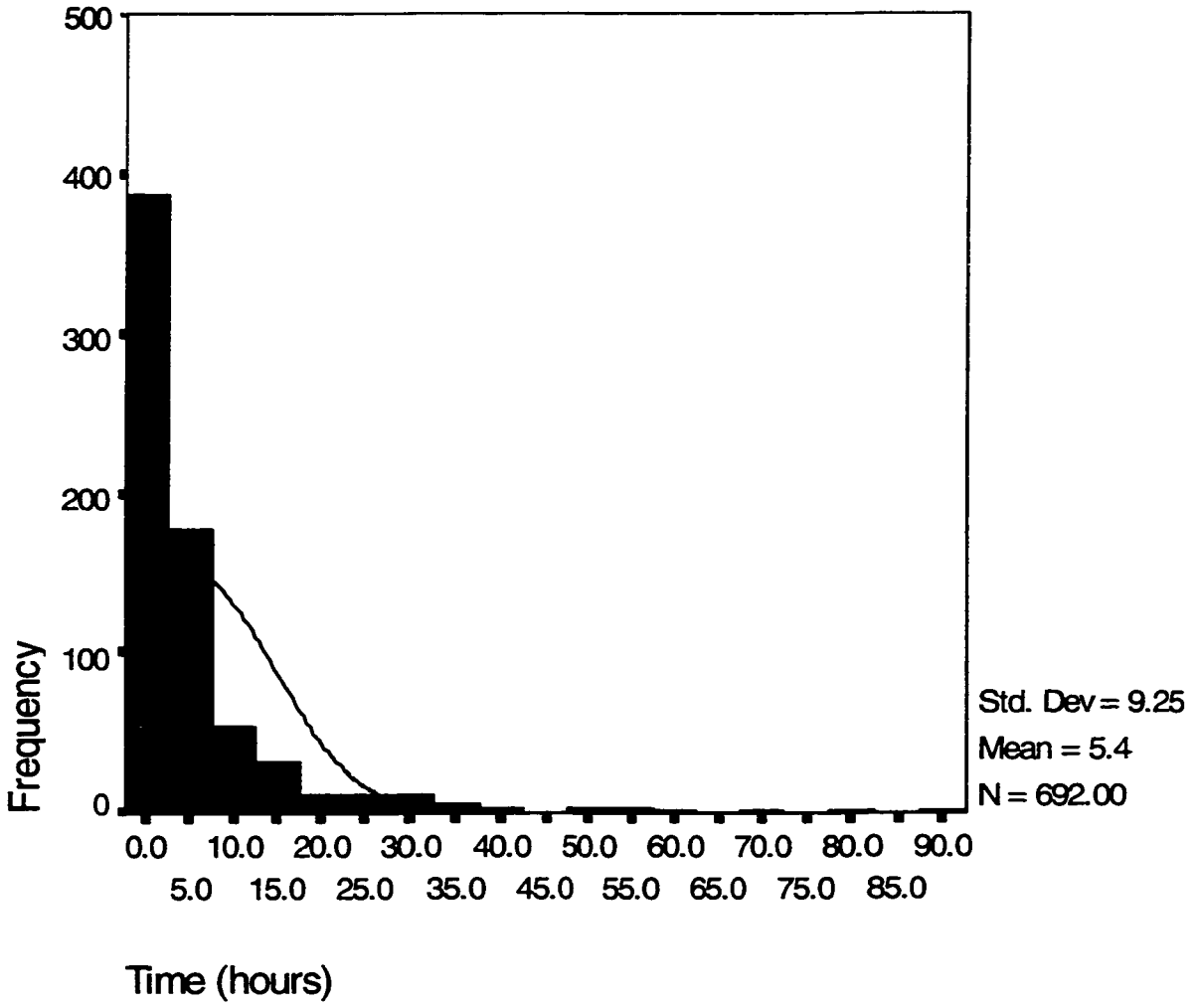


Figure 2. Distribution for onset to arrival delay time for interview group (in hours)

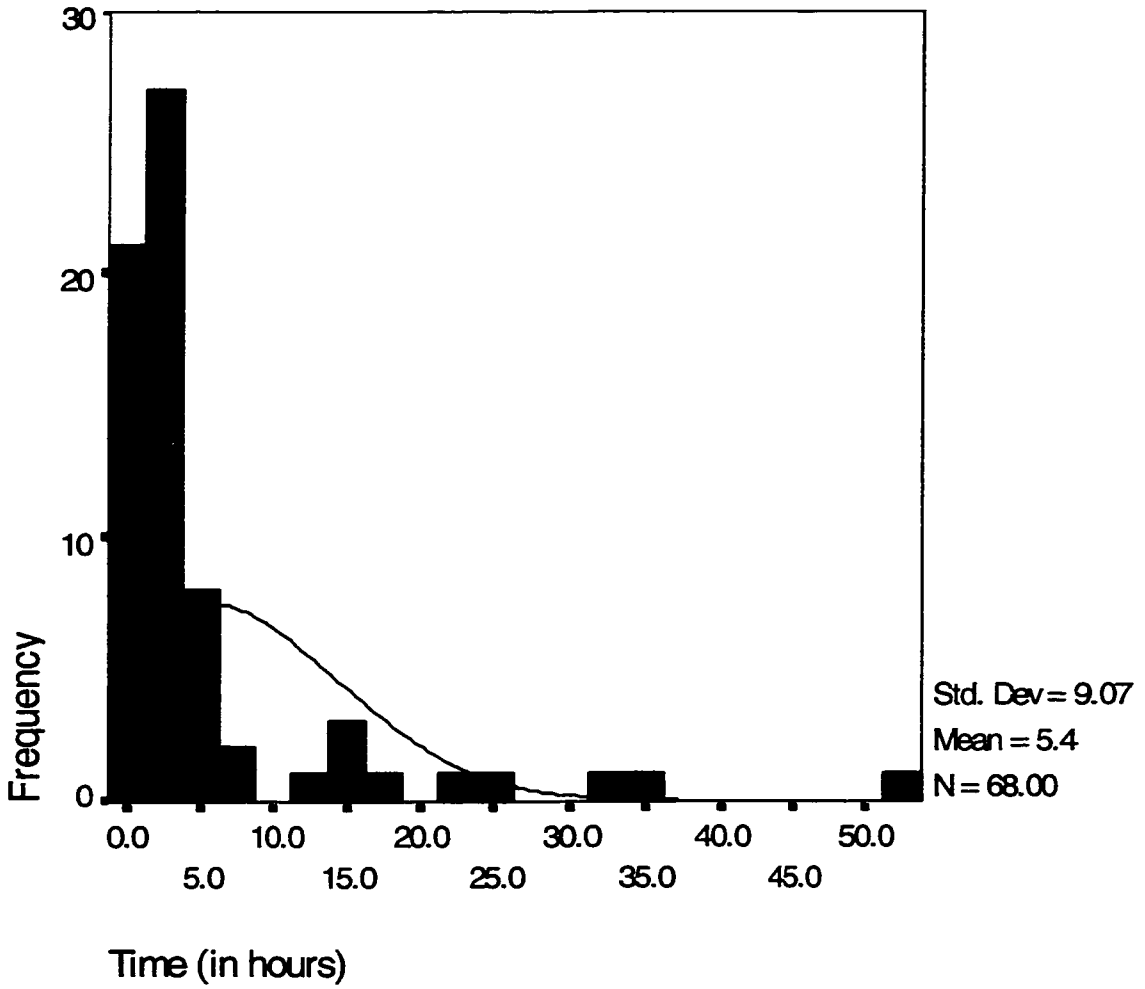


Figure 3. Distribution for residuals of linear regression analysis on delay time (hours) for Fastrak Group

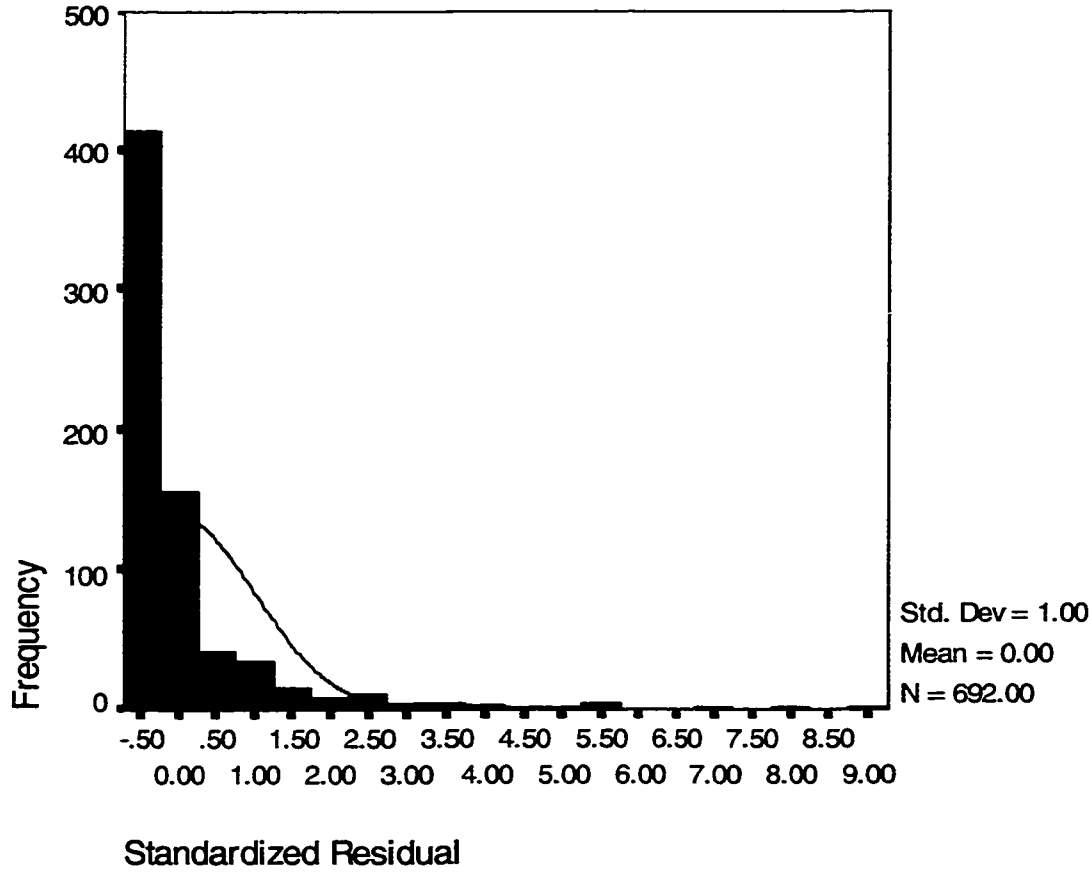


Figure 4. Normal Q-Q Plot for residuals of linear regression analysis on delay time (hours) for Fastrak Group

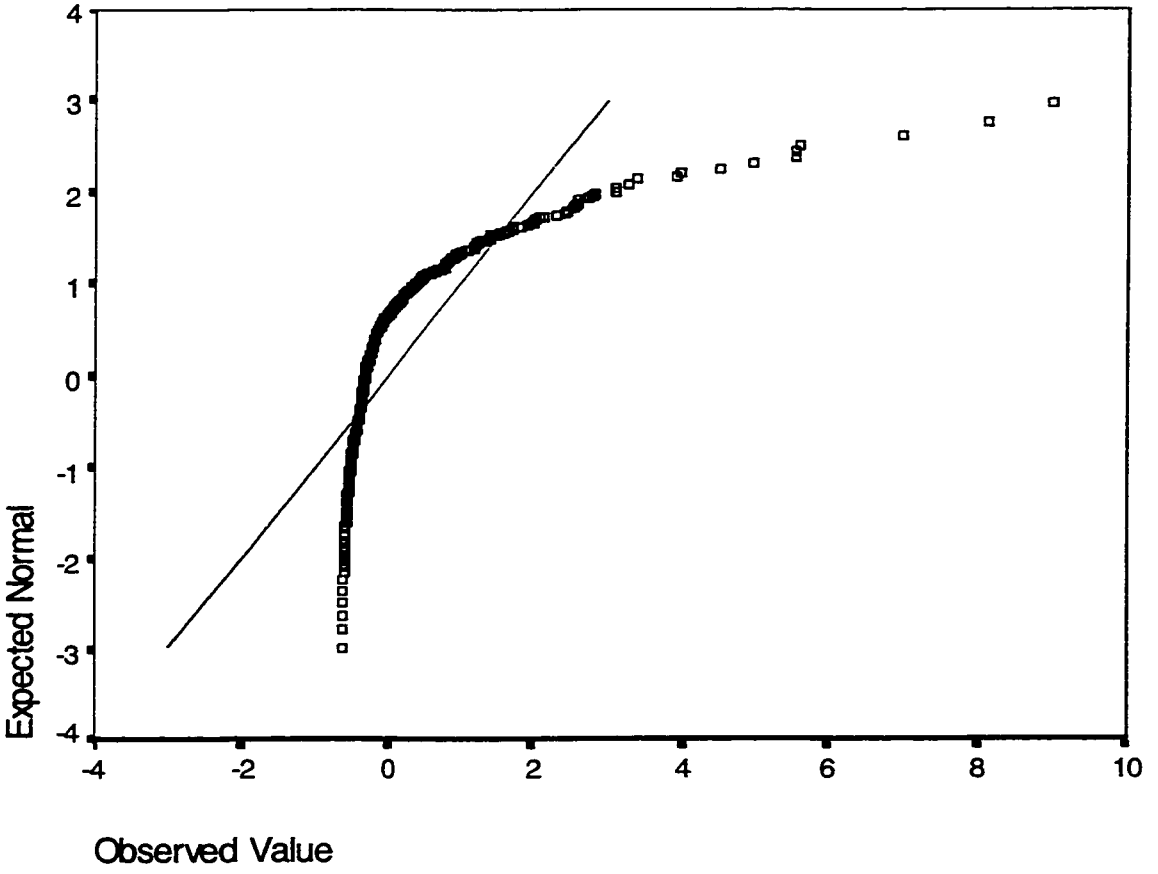


Figure 5. Distribution for log-transformed onset to arrival delay time for *Fastrak* database (in log-hours)

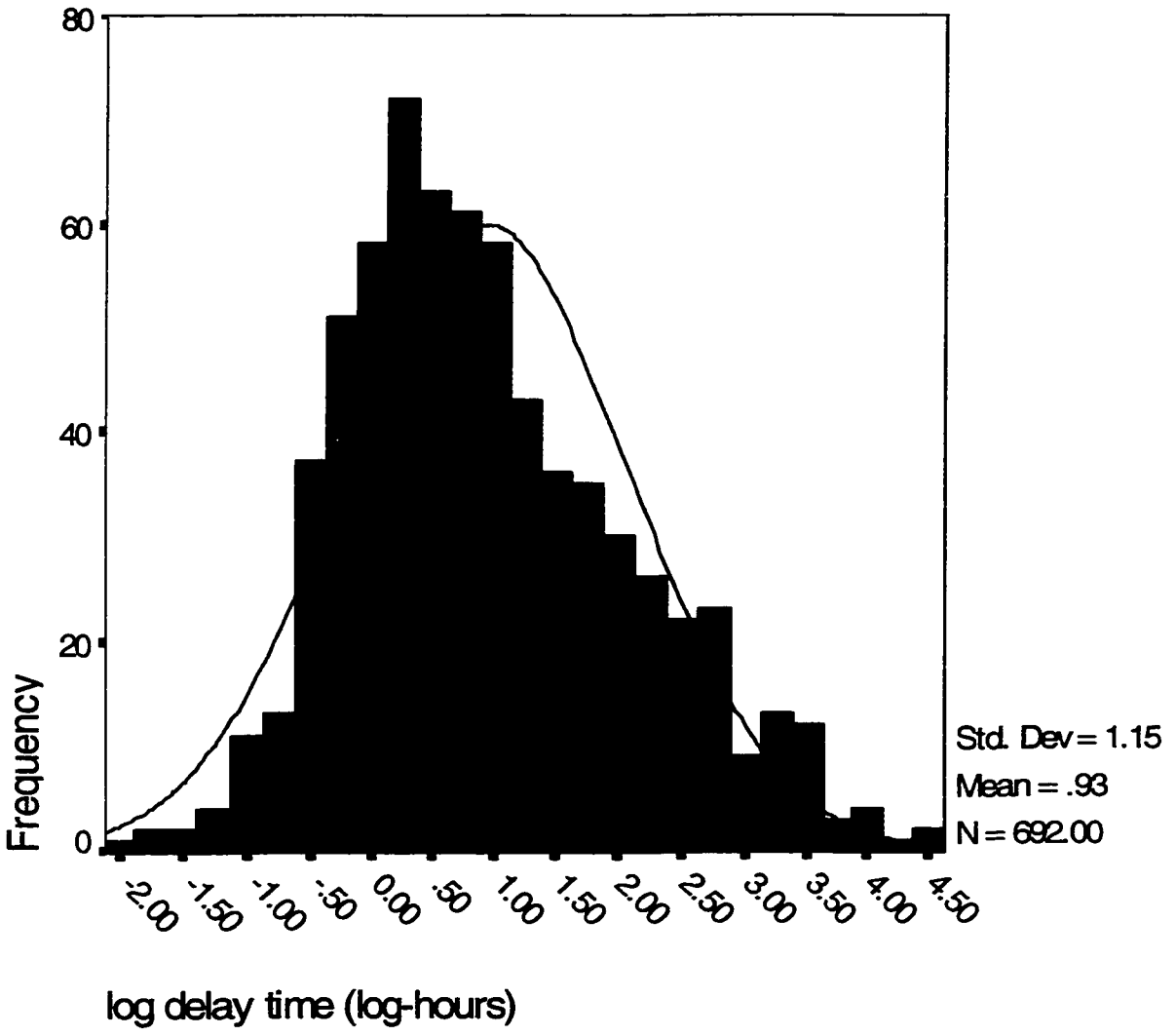


Figure 6. Distribution for log-transformed onset to arrival delay time for interview group (in log-hours)

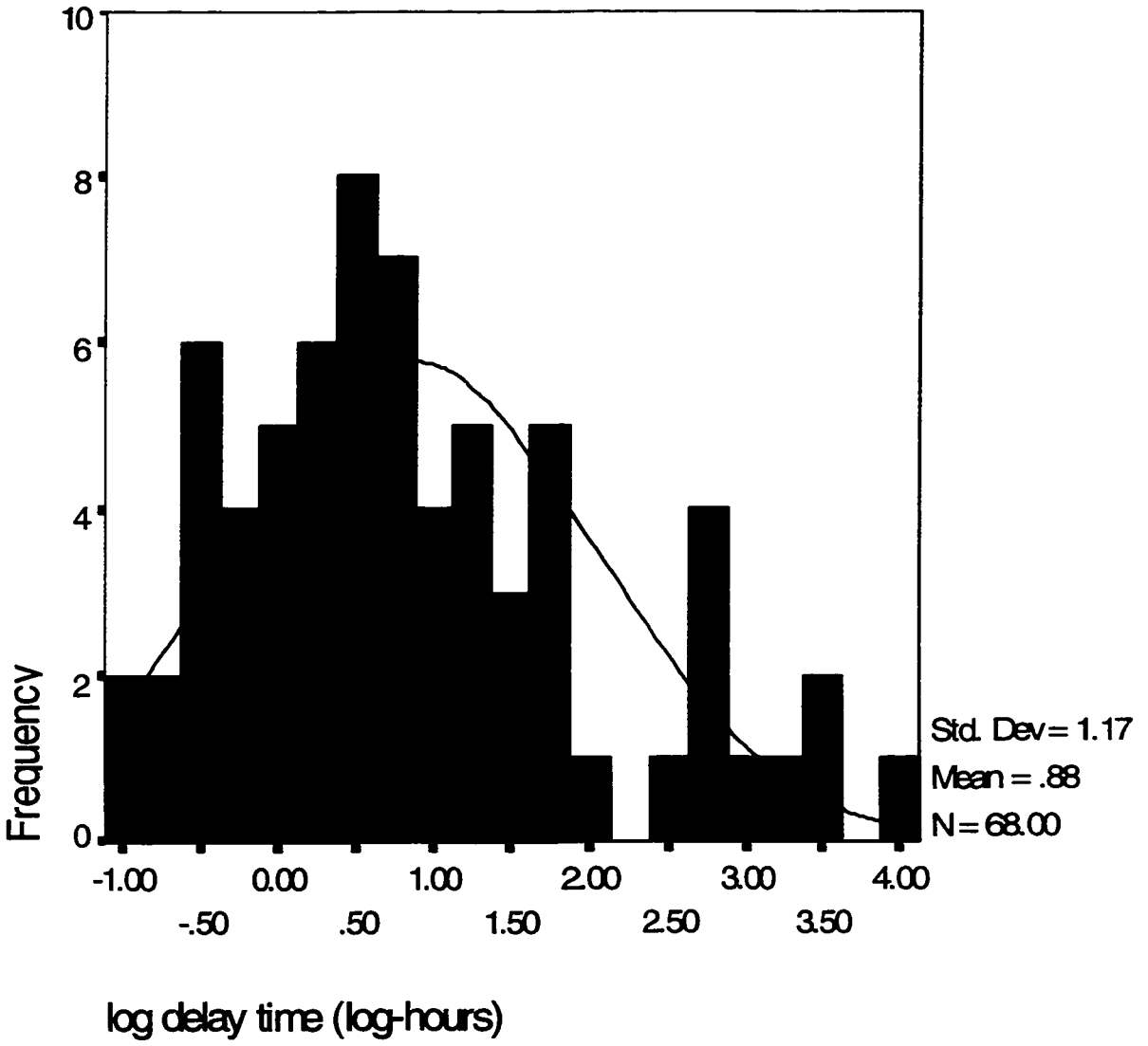


Figure 7. Normal Q-Q Plot for log-transformed patient delay in *Fastrak* group.

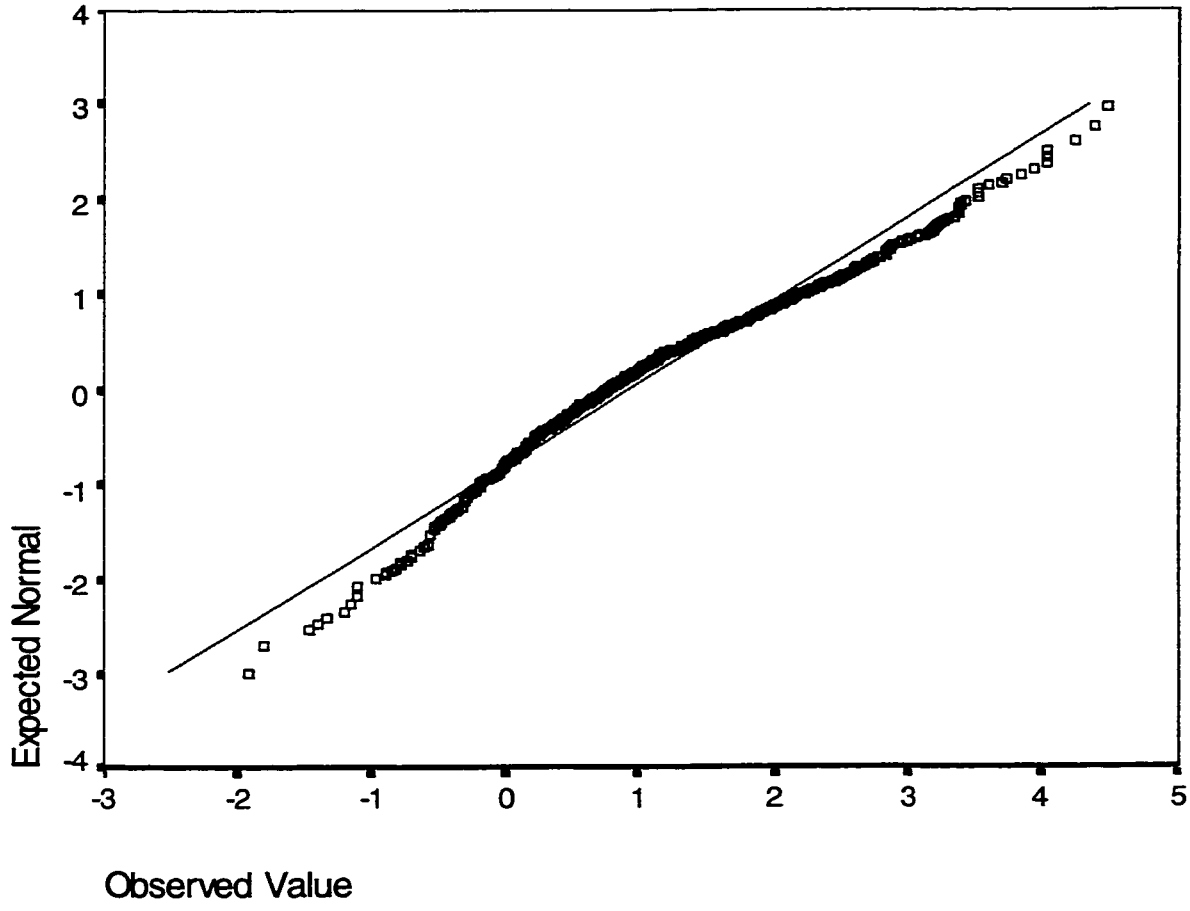


Figure 8. Normal Q-Q Plot for log-transformed patient delay in interview group.

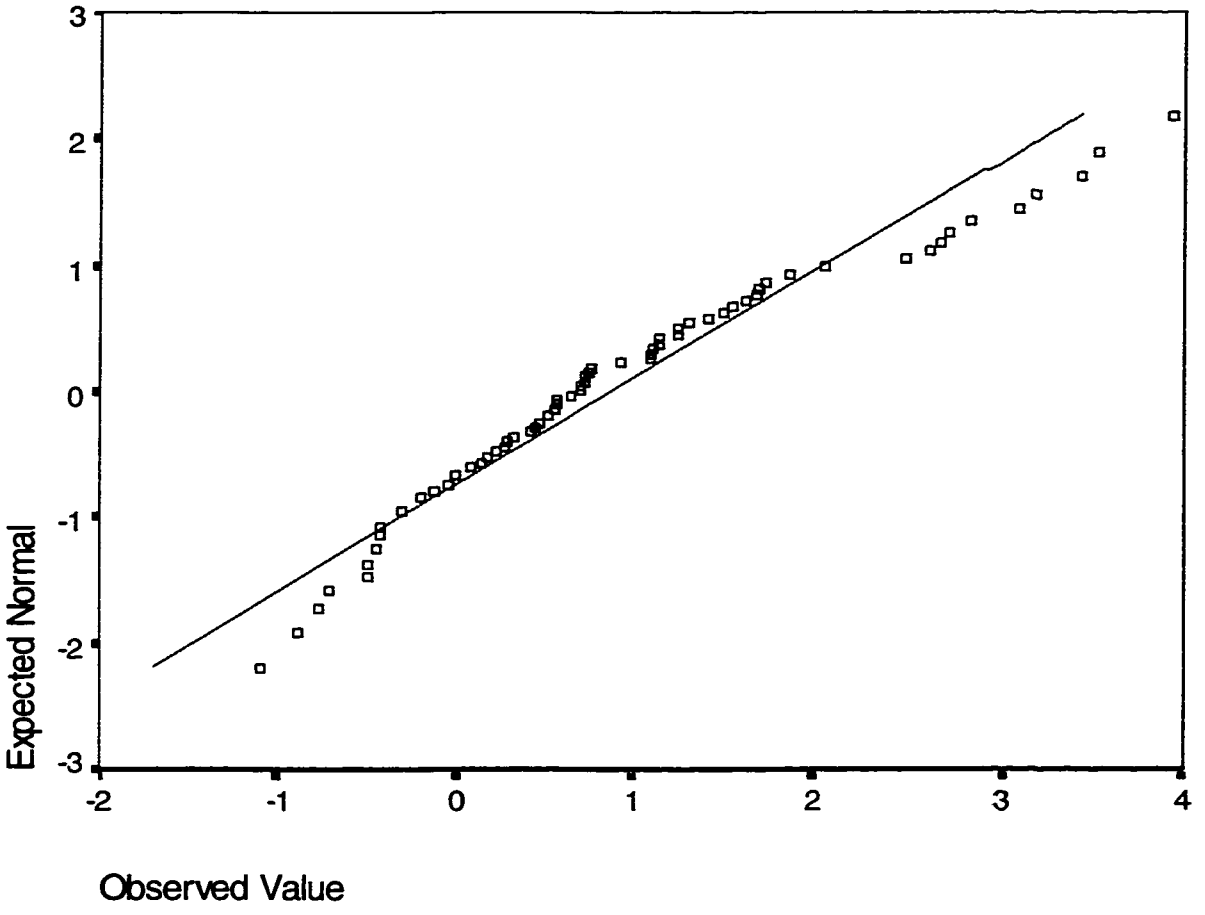


Figure 8). Our comparisons by various factors were therefore conducted based on the natural log transformed outcome and any differences between various categories of a factor should be interpreted as differences in natural log transformed delay time.

3.5 Reclassification of Categorical Variables

The majority of variables obtained from the interviews were dichotomous nominal variables. However, some categorical variables had more than two possible categories. Due to the small number of sample cases available for the interview, some of these nominal categories ended up with a very small sample size. We reclassified several of these nominal categories based on the sample sizes and clinical importance of these categories.

Marital Status. This category was reclassified as being married and non-married. The non-married category included individuals who were single, separated, divorced or widowed. Common-law relationships were considered as married.

Education. All individuals with secondary school education or less were grouped into one category, while those with higher levels of education were considered as a separate group.

Employment. Patients who were working, either full-time or part-time, were placed in a similar category. Those who were unemployed or retired were given the "not-working" designation.

Location. In order to evaluate the role of location on patient delay time, the location where the patient initially experienced their symptoms of MI was classified as home or other. The "other" category includes any location outside the home.

Transportation. Based on the fact that only 2 patients traveled to the hospital using public transportation other than taxis, we combined those who traveled in taxis and other public transportation into a single category. Those who came by ambulance or their own cars remained in 2 separate categories.

Counterroles. Patients were asked about the individuals with whom they discussed their symptoms, and the role these individuals played in their decision process. For the purpose of this study, the difference between talking to a spouse, other relatives, and non-related individuals (co-worker, friend etc) was considered important. We therefore reclassified any unrelated individuals as "others", while self, spouse, and other relatives remained as separate categories.

3.6 Rationale for Sample Size

Our study population was based on two sample groups. The *Fastrak* group consisted of patients who were admitted through the Ottawa General Hospital emergency department with a diagnosis of myocardial infarction. The number of cases in this database was predetermined based on the study period we designated. The second sample consisted of a convenience sample of patients who agreed to participate in an interview during their stay in the hospital for a myocardial infarct. Due to the limited time available to conduct our study, it was only feasible for us to conduct interviews for a maximum of nine months. We were able to interview 72 patients, although 4 patients did not turn out to have myocardial infarction after further evaluation by the attending cardiologist. Therefore, our sample size was based on convenience and the feasibility to collect data for a nine-month period.

The expected precision of our estimate for the difference in log time delay between those who were influenced by a family member in their decision to seek medical help and those who decided to seek help themselves was calculated. While Hackett et al. reported a difference of 11.6 hours between the two groups, no measure of the variability for this difference was reported. However, we were able to estimate the standard deviation to be 2.2 log hours based on the assumption that the range of the difference in log delay time between the two groups is approximately 8 standard deviations. With the assumption that among the interview group, the number of patients deciding to seek medical help themselves will be the same as those who will be influenced by a family member in their decision (i.e. 34 patients for each group), it was determined that our estimate will have a 95% confidence level of 1.5 log hours wide.

3.7 Data Analysis

Descriptive statistics (Means, standard deviation, medians, and interquartile range) were calculated to describe the various delay times in the *Fastrak* database by different patient characteristics (Appendix D). We reported the delay time (in hours) for the *Fastrak* group to allow for comparison with those reported in previous studies.

Our main outcome variable for analysis was the natural log transformed delay time between onset of symptoms and arrival. We conducted a series of t-tests on natural log transformed delay time by dichotomous variables including gender, education, marital status, medical history, and certain contextual variables such as the location where the event occurred. One-way analysis of variance (ANOVA) was performed for independent variables with more than 2 categories particularly the contextual and behavioral questions such as who

was first informed of the symptoms by the patient, mode of transportation, and the patient's behavior towards their perceived symptoms. These analyses were performed on the data from the entire *Fastrak* database using available variables, and also on the interview subgroup. The distribution of delay time was examined and the Levine test was conducted for each of the factors examined to determine if there was a violation of assumption of equal variance. Non-parametric tests were conducted for any factors where assumptions for parametric tests were violated. For factors that were found to be significant, we also examined the effect of age on the association by conducting analysis of covariance (ANCOVA), with age entered as a covariate when comparing natural log of delay time by different categories of significant factors. Possible interaction between gender and significant factors on natural log of delay time was also examined. Statistical significance was considered at $p<0.05$. All results from statistical analysis are reported in natural log transformed time (log hours).

Validity. Cohen's kappa coefficients were calculated for several demographic and medical history variables that were recorded both in the *Fastrak* database from medical charts and obtained through patient interviews. For continuous variables such as age and delay time, Pearson correlation coefficients were calculated. The validity of pre-hospital delay was determined by performing bivariate correlation analysis using pre-hospital delay time recorded in the *Fastrak* database and the sum of patient decision time and travel time as reported by the patient during interview. In addition, we calculated the time difference between the symptom onset time recorded in *Fastrak* and that reported by the patient during the interview and tested for the hypothesis that this difference is zero using a one-sample t-test.

Linear Regression. A multiple stepwise linear regression was conducted on the *Fastrak* group using the natural log-transformed delay time as the dependent variable. All available demographics and medical history variables were included in the selection pool (Appendix D). Variables were entered based on the criteria of a significant increase in R^2 ($p<0.05$).

Multiple stepwise linear regression was also conducted on the interview group. Since only patients who were married answered questions regarding their relationship with their spouse, and one of our objectives was to determine whether this variable was important in affecting delay time, two samples of those who were interviewed were therefore used in the linear regression analysis. For both models, we did not include the variable regarding whether "patients accepted suggestions from another person to seek medical help" in our pool of possible independent variables as only 50 patients had another person making suggestions to them, and would limit our sample size significantly for analysis.

Our first sample consisted of patients with data for all the variables we were studying, with the exception of ADAS, which only applied to those who were married. Among those who were excluded from the sample, 8 did not talk to anyone about their symptoms before coming to hospital. Hence they did not have responses for variables relating to the counterroles a witness played in the decision process. In addition, 2 patients did not provide responses for the extent of their pain. Therefore, our final sample included 58 cases. The ADAS measurement was not included in the analysis pool for this sample.

The second sample consisted only of those who were married and provided answers to the ADAS questionnaire. We conducted this analysis mainly to determine whether spousal relationship as measured by the abbreviated dyadic adjustment scale is a significant

factor associated with delay time. We included all other independent variables for this analysis and a total of fifty-one patients were included in this sample.

For all models, we used natural log-transformed patient delay time as the dependent variable. We treated the different categories for the behavioral questions, pain recurrence, and pain quality as continuous variables. Contrast coding was used for other nominal variables with more than 2 categories. Variables were entered with the criteria of a significant increase in R^2 ($p<0.05$). Preliminary residual analysis was performed to ensure no outliers, and violation of assumptions for the linear regression model.

Since our linear regression model was conducted using the natural log transformed delay time as the dependent variable, one may interpret the results similar to a multiplicative model as shown in the following illustration, with y representing delay time and X_i representing independent variables. Therefore, our linear regression model would be:

$$\text{Ln } y = \alpha + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \ldots. \beta_i X_i$$

Which can be mathematically rearrange as:

$$y = e^{\alpha + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \ldots. \beta_i X_i}$$

Similar as stating:

$$y = e^{\alpha} * e^{\beta_1 X_1} * e^{\beta_2 X_2} * e^{\beta_3 X_3} * \ldots\ldots e^{\beta_i X_i}$$

When $X_1 = 0$, then $e^{\beta_1 X_1} = 1$,

and when $X_1 = 1$, then $e^{\beta_1 X_1} = e^{\beta_1}$

Therefore, delay time for when variable X_i is coded as 1 can be interpreted as e^{β_1} times the delay time when X_i is coded as 0.

Logistic Regression. At the National Emergency Pre-hospital Care Conference, it was suggested that the window of opportunity from onset of MI symptoms to reaching emergency is 90 minutes in order to maximize the effectiveness of thrombolytic therapy and minimize cardiac muscle damage (24). Clinically, it seemed meaningful to dichotomize our outcome, the patient delay time, and determine what factors are associated with having a delay time of over 90 minutes. Therefore, multivariate logistic regression analysis was conducted in the *Fastrak* and interview cases using outcome as delay time more than 1.5 hours. We examined the delay time of all our *Fastrak* and interview cases, and did not find any cases that had delay time within 10 minutes of our cut point of 1.5 hours.

Due to limitation of the sample size in the interview group, we were unable to obtain a logistic regression model if we included all the available independent variables for selection using a forward conditional selection method. Therefore, we conducted chi-square analysis to determine which of the independent variables were associated with having a delay of more than 90 minutes. Behavioral variables were re-grouped into 2 categories of yes or no. Independent variables with $p<0.20$ for chi-square were then included in the selection pool for logistic regression modeling utilizing forward conditional selection method with criteria of $p<0.10$. Variables used for selection in the logistic regression analyses are listed in Appendix D. Once again, two samples were used for modeling; one without using the ADAS variable in the selection pool, and another with ADAS included. All analyses were conducted on SPSS for Windows (version 7.0).

4. RESULTS

4.1 Study Population

***Fastrak* Group.** Between September 1994 and August 1997, data were collected for 1062 patients who were admitted to the Ottawa General Hospital with a diagnosis of acute myocardial infarction. Among those included in the *Fastrak* database, 92 cases were transferred from another hospital; 152 cases suffered their attack while they were already in the hospital for other reasons; and 122 cases did not have data for onset time. Therefore, 696 patients satisfied our inclusion criteria for analysis. We further excluded 4 cases with an unusual length of onset to arrival delay (3 cases > 7 days and 1 case < 2 minutes) after checking with medical records to ensure they were not data entry errors. Our analysis for the *Fastrak* group was finally conducted on 692 cases (Table 6).

Interview Subgroup. Interviews were conducted with 74 patients who were admitted to the Ottawa General Hospital with an initial diagnosis of myocardial infarction. Among them, 4 patients did not turn out to be actual myocardial infarction cases, and 2 patients were transferred from another hospital, and hence were excluded. A total of 68 cases were used for our analysis in the interview subgroup (Table 6).

4.2 Demographics

***Fastrak* Group.** The patient characteristics are presented in Table 7. The mean age for the study population was 65 years. Females (69.5 years) were significantly older than males (62.4 years), $p<0.05$. There were approximately twice as many males than females included in the study population. Among this population, 236 patients (34.4%) received thrombolytic

Table 6. Study population

Population breakdown for the Fastrak database	# count
Patients with a final diagnosis of MI between September 1994 and August 1997	1062
# of subjects transferred to the General from another hospital (excluded)	92
# of subjects with an MI while in hospital for other medical problems (excluded)	152
# of subjects without data for symptom onset (excluded)	122
# of subjects with unusual length of pre-hospital delay time (excluded)	4
Total # of subjects used for analysis in Fastrak	692
Population breakdown for the interview subgroup	**# count**
Total # of Patients interviewed	74
# of patients transferred to the General from another hospital (excluded)	2
# of subjects whose final diagnosis was not MI	4
Total # of subjects used for analysis in interview subgroup	68

Table 7. Characteristics of MI cases who were admitted to the Ottawa General Hospital between September 1994 and August 1997

Characteristics	**% (# of patients)**
Sex	
Male	64.9 (449)
Female	35.1 (243)
Mean age ± standard deviation, yrs	64.9 ± 13.3
Mean weight ± standard error, lbs	167.8 ± 37.6 †
% (and number) of patients with history of:	
Previous MI	36.3 (251)
Angina	34.7 (240)
Diabetes	25.1 (174)
Current smoker	31.6 (219)
Coronary Artery Bypass Grafting	9.2 (64)
Congestive Heart Failure	10.7 (74)
Stroke	8.4 (58)
Angioplasty (PTCA)	4.3 (30)
Hypertension	40.3 (279)
Hypercholesterolemia	26.0 (180)
% (and number) of patients who received	
IV thrombolytic therapy	34.4 (236) *
% (and number) of in-hospital death	9.2 (64)

Total sample size is 692, except for * (687) and † (430)

therapy. As expected, the prevalence of cardiovascular risk factors was high ranging from 26% for hypercholesterolemia to 40% for hypertension. In-hospital death occurred in 64 patients (9%) following admission to the hospital.

Interview Group. The characteristics of the interview group are presented in Table 8. Of those patients who were interviewed, 51 patients (75%) were male and 17 patients (25%) were females. The mean age for this subgroup was 61.5 years, and there was no difference in age between the 2 genders (61.5 vs. 61.6 years). 55 patients (81%) whom we interviewed were married, and 24 (35%) had higher than secondary education. Over 50% of this group received thrombolytic therapy and only 1 patient died in the hospital subsequent to the interview.

Compared to the *Fastrak* group, our interview group differed significantly in certain aspects. There was a significantly higher proportion of males in our interview group (75% vs. 65%, $p<0.05$), and the average age was younger ($p<0.05$). The *Fastrak* population, on the other hand, had a higher proportion of patients with a history of angina, congestive heart failure, and in-hospital death ($p<0.05$). In addition, patients who were interviewed were more likely to have received thrombolytic therapy (54% vs. 34%, $p<0.001$).

4.3 Validity of Data

Table 9 shows the kappa coefficients for several nominal variables that were collected both in the *Fastrak* database and during patient interviews. With the exception of "history of angina", the agreement is acceptable with coefficients ranging from 0.79 to 1.00. The agreement for reporting angina was poor with a kappa value of 0.46. Age was perfectly

Table 8. Characteristics of MI cases who were interviewed.

Characteristics	**% (# patients)**
Sex	
Male	75 (51)
Female	25 (17)
Mean age ± standard deviation, yrs	61.5 ± 11.5
Mean weight ± standard deviation, lbs	177.9 ± 33.6 *
% (and number) of patients	
> Secondary education	35.3 (24)
Married	80.9 (55)
Working	39.7 (27)
% (and number) of patients with history of:	
Previous MI	29.4 (20)
Angina	22.1 (15)
Diabetes	19.1 (13)
Current smoker	32.4 (22)
Coronary Artery Bypass Grafting	13.2 (9)
Congestive Heart Failure	2.9 (2)
Stroke	4.4 (3)
Angioplasty (PTCA)	5.9 (4)
Hypertension	35.3 (24)
Hypercholesterolemia	25.0 (17)
% (and number) of patients who received	
IV thrombolytic therapy	54.4 (37)
% (and number) of in-hospital death	1.5 (1)

Total sample size is 68, except for * which was 35.

Table 9. Validity Test: Between Data Recorded in the *Fastrak* Database and those obtained from Patient Interviews.

Demographic and Medical History Variables	Kappa Coefficient
Gender	1.00
Current smoker	0.79
Previous MI	0.79
History of Angina	0.46
Previous Coronary Artery Bypass Surgery	1.00
Continuous Variable	**Pearson Correlation Coefficient**
Pre-hospital Delay	0.77
Age	1.00

correlated with a Pearson correlation coefficient of 1.00. The average time difference between the symptom onset time collected in the *Fastrak* database and that reported by the patients during interview was 8.3 minutes with a Pearson coefficient of 0.77 ($p<0.01$) and was not significantly different from zero.

4.4 Delay Time

***Fastrak* Group.** The mean delay time from onset of symptoms to arrival for the *Fastrak group* was 5 hours and 22 minutes (median 2 hours 8 minutes). Approximately 48% of the patients arrived at the hospital within 2 hrs, 21% between 2 to 4 hours, 8% between 4 to 6 hours, and 23% delayed more than 6 hours before arriving at the hospital. Substantial delay occurred in all components of in-hospital process (Table 10). On average, 73.5 minutes (median 55 minutes) elapsed between arrival to the emergency department and initiation of thrombolytic therapy to patients who were eligible (Figure 9), with the in-hospital delay accounting for 37.6% of total delay between onset of symptoms to administration of thrombolytic therapy. Once the physician ordered thrombolytic therapy, a mean delay of 17 minutes (median 14 minutes) occurred between ordering and the actual administration of the drug (Figure 10). As expected, all delay times were heavily skewed with median times much lower than mean times. Mean and median for patient delay by various characteristics are described in Table 11.

Our study group consists of patients who were admitted over a 4-year period from 1994 to 1998. We did not find any significant difference in any components of delay over the 4 years (Figure 11).

Table 10. Delay Times for Patients Admitted to Ottawa General between September 1994 and August 1997.

Delay time	**Mean ± SD**	**Median time (and IR)**
Onset to arrival, hrs (n=692)	5.38 ± 9.25	2.13 (1.08-5.25)
Arrival to IV thrombolytic ordered, minutes (n=229)	56.23 ± 56.62	40.00 (22.00-70.00)
IV thrombolytic ordered to administered, minutes (n=227)	17.15 ± 14.87	14.00 (6.00-25.00)
Total, from hospital arrival to thrombolysis, minutes (n=227)	73.50 ± 60.50	55.00 (36.00-95.00)
Total, from onset of symptoms to thrombolysis, minutes (n=227)	261.11 ± 328.30	175.00 (120.00-270.00)
Proportion of total IV delay due to in-hospital delay, % (n=227)	37.63 ± 21.14	35.79 (20.88-51.72)

IR = Interquartile Range, **SD** = Standard Deviation

Figure 9. Distribution for time delay between arrival and initiation of thrombolytic therapy (in minutes).

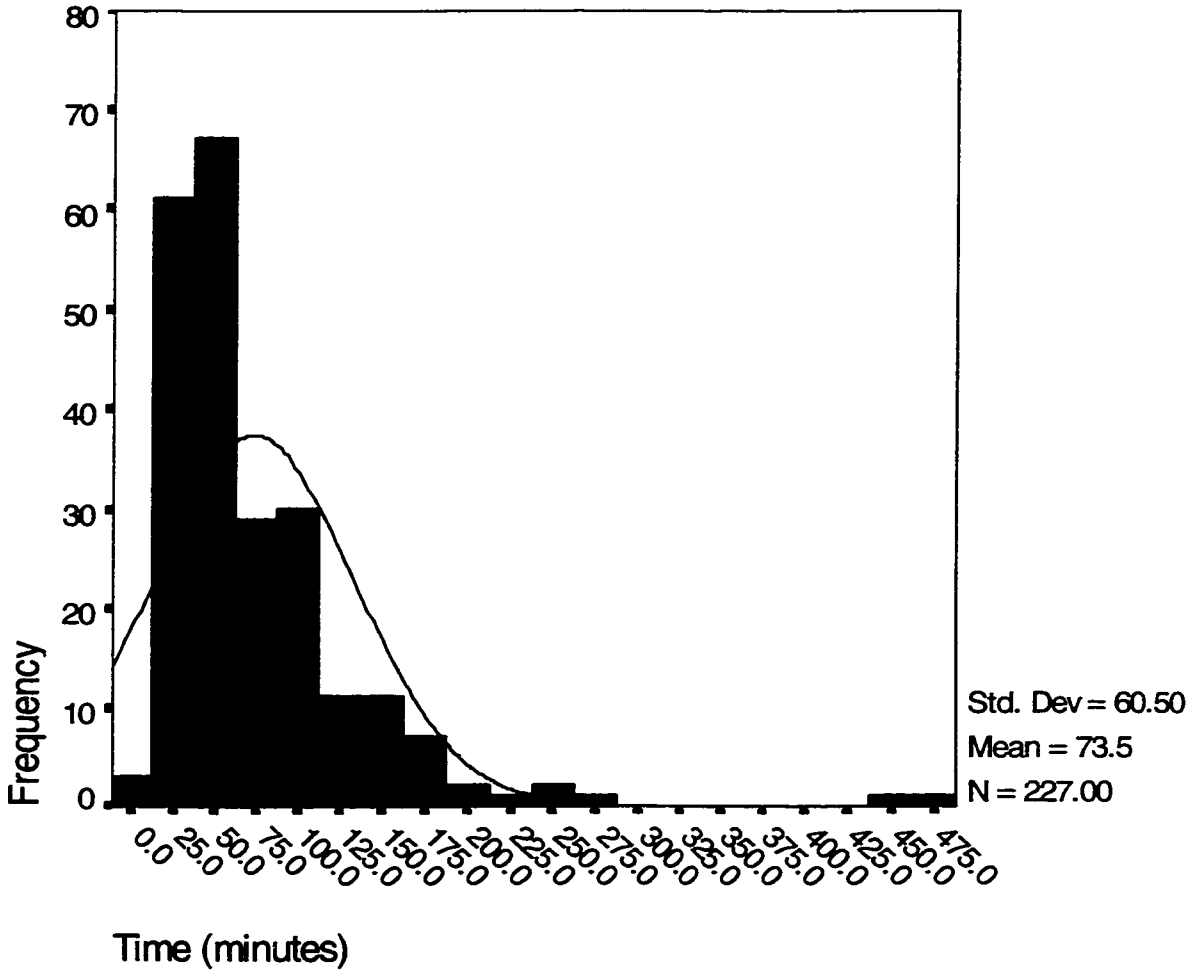


Figure 10. Distribution for time delay between ordering and administration of thrombolytic therapy (in minutes).

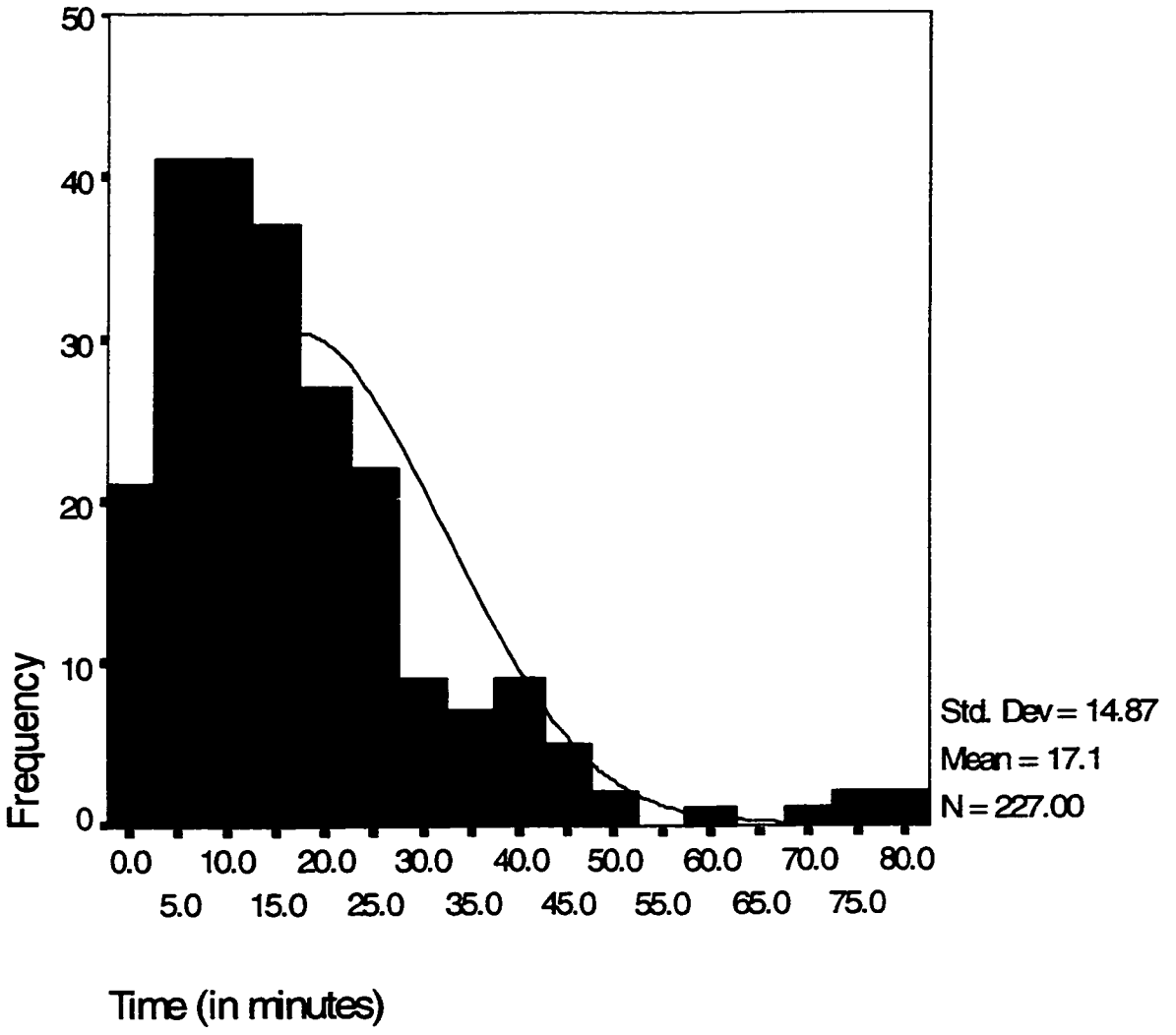
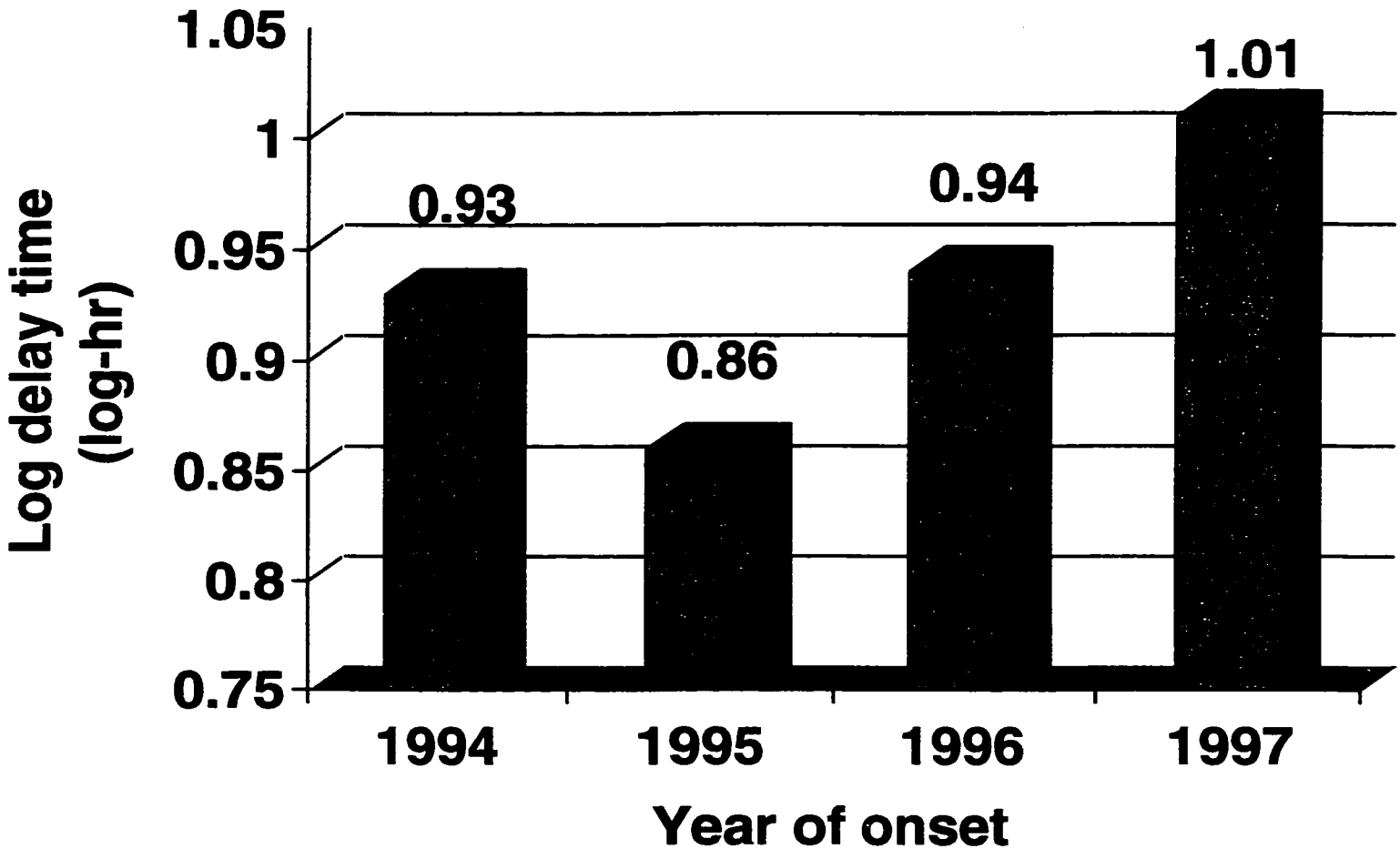


Table 11. Onset to Arrival Delay Time in hours of MI cases who were Admitted to the Ottawa General Hospital between September 1994 and August 1997, by Sociodemographic Variables and Medical History.

Characteristics	Mean ± SD	Median time (and IR)
Sex		
Male (n=449)	4.90 ± 9.10	2.00 (1.08-4.57)
Female (n=243)	6.29 ± 9.47	2.62 (1.13-6.50)
Previous MI		
No (n=441)	6.01 ± 10.51	2.25 (1.17-5.94)
Yes (n=251)	4.29 ± 6.36	1.88 (1.00-4.55)
History of Angina		
No (n=452)	5.37 ± 9.29	2.17 (1.08-5.08)
Yes (n=240)	5.42 ± 9.19	2.03 (1.14-5.73)
History of Diabetes		
No (n=518)	5.10 ± 9.12	2.03 (1.02-4.83)
Yes (n=174)	6.22 ± 9.62	2.56 (1.28-6.20)
Current smoker		
No (n=473)	5.25 ± 9.16	2.10 (1.08-5.08)
Yes (n=219)	5.68 ± 9.46	2.25 (1.10-6.07)
Previous Coronary Artery Bypass Grafting		
No (n=628)	5.45 ± 9.39	2.13 (1.08-5.25)
Yes (n=64)	4.73 ± 7.83	2.15 (1.10-5.18)
History of Congestive Heart Failure		
No (n=618)	5.53 ± 9.66	2.13 (1.08-5.18)
Yes (n=74)	4.16 ± 4.48	2.21 (1.08-6.24
History of Stroke		
No (n=634)	5.37 ± 9.29	2.14 (1.08-5.25)
Yes (n=58)	5.51 ± 8.91	2.10 (1.16-5.82)
History of angioplasty (PTCA)		
No (n=662)	5.48 ± 9.39	2.17 (1.10-5.42)
Yes (n=30)	3.23 ± 4.97	1.23 (0.90-4.37)
History of Hypertension		
No (n=413)	5.43 ± 9.54	2.07 (1.09-4.88)
Yes (n=279)	5.31 ± 8.82	2.32 (1.08-6.08)
History of Hypercholesterolemia		
No (n=512)	5.60 ± 9.83	2.22 (1.08-5.25)
Yes (n=180)	4.78 ± 7.35	1.77 (1.08-5.40)
In-hospital death		
No (n=628)	5.43 ± 9.49	2.17 (1.10-5.15)
Yes (n=64)	4.98 ± 6.57	1.67 (0.90-7.10)

IR = Interquartile Range, **SD** = Standard Deviation

Figure 11. Log delay time by year of onset



Interview Group. Patients who were interviewed had similar delay times to those in the *Fastrak* group. The mean delay from onset to arrival was 5 hours 22 minutes, with a median of 2 hours. For the 32 patients who received thrombolytic therapy and for whom intervals to treatment were available, the mean in-hospital delay from arrival to drug administration was slightly over an hour (67 minutes) and median of 53 minutes. A similar delay of 18 minutes (median, 15 minutes) occurred between the time of drug ordering and the actual administration for those who were interviewed as for the *Fastrak group*.

4.5 Effect of Demographics and Medical History on Pre-hospital Delay

Fastrak Group. Log-transformed patient delay times by gender and various medical history factors are presented in Table 12. Among the various factors investigated using the *Fastrak* group, we found females to have a significantly longer log-delay than males ($p<0.05$), and this effect remained significant even when age was included as a covariate. Patients who had a previous myocardial infarction were found to have a significantly shorter log-delay time, while diabetics delayed longer. We did not find any difference in log delay time between non-survivors and survivors (0.93 ± 0.05 vs. 0.86 ± 0.15 log-hrs), although surprisingly, non-survivors arrived at the hospital slightly faster.

While a comparison of log-delay times between various age categories was not significant, we did find a significant exponential trend with older patients having longer delay (Figure 12). The delay time (in hours) by different age groups is presented in Table 13.

Interview Group. We compared the log-transformed pre-hospital delay times between the *Fastrak* group and the interview group for each of the categories (Table 12). No

Table 12. Onset to Arrival Delay Time (log-transformed), in log hours, of MI cases who were Admitted to the General Hospital from September 1994 to August 1997 (Fastrak group), and patients who were interviewed, by gender and medical History.

Characteristics	Mean ± SE (# of cases)	
	Fastrak Group	Interview Group
Sex		
Male	0.85 ± 0.05 (449) *	0.70 ± 0.14 (51) *
Female	1.08 ± 0.08 (243)	1.40 ± 0.35 (17)
Previous MI		
No	1.00 ± 0.06 (441) *	0.97 ± 0.17 (48)
Yes	0.78 ± 0.07 (251)	0.66 ± 0.24 (20)
History of Angina		
No	0.92 ± 0.05 (452)	0.97 ± 0.17 (53)
Yes	0.95 ± 0.07 (240)	0.54 ± 0.24 (15)
History of Diabetes		
No	0.86 ± 0.05 (518) *	0.78 ± 0.15 (55)
Yes	1.12 ± 0.08 (174)	1.26 ± 0.38 (13)
Current Smoker		
No	0.90 ± 0.05 (473)	0.72 ± 0.17 (46)
Yes	0.98 ± 0.08 (219)	1.20 ± 0.25 (22)
Previous Coronary Artery Bypass Grafting		
No	0.93 ± 0.05 (628)	0.90 ± 0.15 (59)
Yes	0.88 ± 0.13 (64)	0.69 ± 0.44 (9)
History of Congestive Heart Failure		
No	0.93 ± 0.05 (618)	0.98 ± 1.16 (66)
Yes	0.89 ± 0.12 (74)	-0.20 ± 0.20 (2)
History of Stroke		
No	0.92 ± 0.05 (634)	0.90 ± 0.15 (65)
Yes	0.98 ± 0.15 (58)	0.42 ± 0.23 (3)
History of angioplasty (PTCA)		
No	0.94 ± 0.04 (662)	0.94 ± 0.15 (64)
Yes	0.61 ± 0.18 (30)	-0.13 ± 0.16 (4) ⁸
History of Hypertension		
No	0.91 ± 0.06 (413)	0.84 ± 0.19 (44)
Yes	0.95 ± 0.07 (279)	0.95 ± 0.22 (24)
History of Hypercholesterolemia		
No	0.95 ± 0.05 (512)	0.81 ± 0.16 (51)
Yes	0.86 ± 0.08 (180)	1.08 ± 0.32 (17)

* $p<0.05$ (comparison within the *Fastrak* group or interview group)

⁸ $p < 0.05$ (comparison between *Fastrak* and interview group)

Figure 12. Log delay time by age group

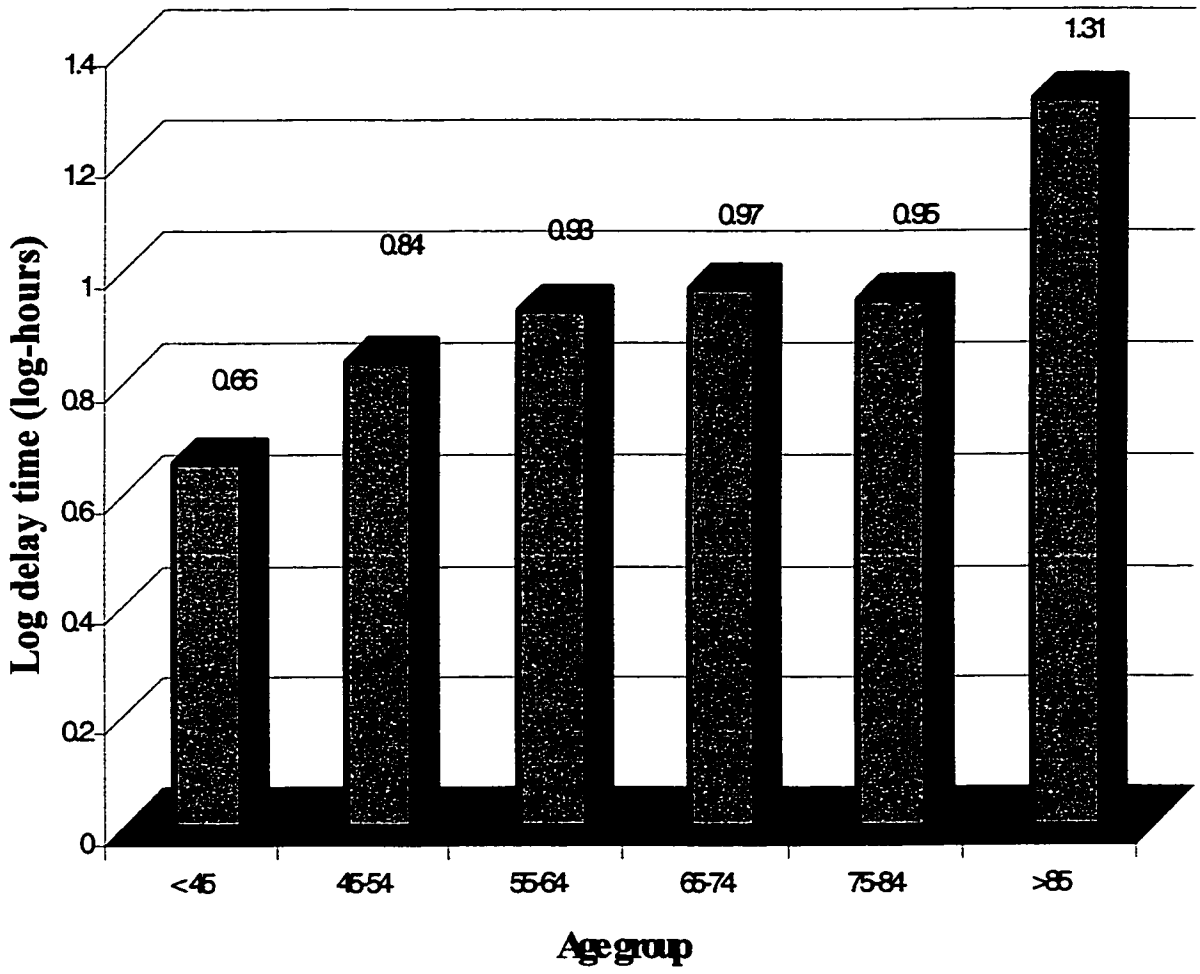


Table 13. Onset to Arrival Delay Time, in hours, of MI Cases who were Admitted to the Ottawa General Hospital between September 1994 and August 1997, by Age Group.

Characteristics	**Mean ± SD**	**Median time (and IR)**
Age, yr		
< 45 (n=59)	3.80 ± 6.19	1.38 (0.97-3.23)
45-54 (n=124)	5.78 ± 10.89	1.75 (0.97-5.47)
55-64 (n=125)	5.17 ± 9.80	2.25 (1.17-5.18)
65-74 (n=194)	5.45 ± 8.57	2.24 (1.17-5.02)
75-84 (n=162)	5.54 ± 9.71	2.22 (1.18-5.93)
>85 (n=28)	6.54 ± 5.74	5.34 (1.14-11.77)

IR = Interquartile Range, **SD** = Standard Deviation

significant difference was detected except for those who had a previous PTCA. However, there were only 4 patients from the interview subgroup in this category.

Female patients in our interview subgroup also delayed significantly longer than males, similar to those in the *Fastrak* group. Unlike those in the *Fastrak* group, diabetic patients and those who had a previous MI did not differ significantly from those without these medical conditions. However, the direction remained, with diabetics having a longer patient delay time (1.26 vs. 0.78 log-hrs), and patients with previous MI arriving at the emergency department faster (0.66 vs. 0.97 log-hrs). None of the other sociodemographic and medical history variables were found to have a significant effects on delay times (Table 12 & 14).

4.6 Effects of Time, Pain, and Transportation Variables on Pre-hospital Delay

Patient log delay time was not found to be affected by the time of day that onset of symptoms was experienced. Similarly, there was no statistically significant difference between those who experienced their symptom onset during the weekdays or during the weekend, although patients with weekend onset appear to delay less (0.48 vs. 0.98 log-hrs). Patients who experienced their symptom onset at home appeared to delay longer than those at other locations, but this was also not statistically significant (Table 15).

Individuals who believed their symptoms were due to a heart problem arrived at the hospital significantly earlier than those who attributed their symptoms to other causes (0.55 vs. 1.09 log-hours, $p<0.05$). Patients with pain that was ongoing (0.53 log-hrs) arrived faster than those with recurrence of pain (1.29 log-hrs), who in turn, arrived faster than those who didn't experience any pain recurrence (1.50 log-hrs). A statistically significant exponential

Table 14. Onset to Arrival Delay Time (log transformed), in log hours, for interview cases by Sociodemographic and Medical History Variables.

Characteristics	Mean ± SE
Marital Status	
Not Married (n=13)	1.24 ± 0.37
Married (n=55)	0.79 ± 0.15
Education	
≤ Secondary education (n=44)	0.98 ± 0.18
> Secondary education (n=24)	0.69 ± 0.24
Occupation	
Not Working (n=41)	0.93 ± 0.20
Working (n=27)	0.79 ± 0.20
Born in Canada	
No (n=16)	0.78 ± 0.30
Yes (n=52)	0.90 ± 0.16
Family history of MI	
No (n=21)	0.68 ± 0.19
Yes (n=47)	0.96 ± 0.19
Patient believe heart disease runs in family	
No (n=32)	1.09 ± 0.22
Yes (n=30)	0.68 ± 0.18
Don't Know (n=6)	0.72 ± 0.66
History of Arrhythmia	
No (n=56)	0.85 ± 0.15
Yes (n=7)	0.67 ± 0.62
Don't Know (n=5)	1.43 ± 0.55
History of Arteriosclerosis	
No (n=47)	0.95 ± 0.18
Yes (n=15)	0.47 ± 0.24
Don't Know (n=6)	1.28 ± 0.47
Previous consultation with cardiologist	
No (n=38)	0.92 ± 0.19
Yes (n=30)	0.82 ± 0.22
Previous experience of any chest pain	
No (n=21)	1.14 ± 0.30
Yes (n=47)	0.76 ± 0.16
Previous experience of the same type of pain as this event	
No (n=53)	0.94 ± 0.16
Yes (n=15)	0.66 ± 0.35

* $p<0.05$; **SE** = standard error

Table 15. Onset to Arrival Delay Time (log transformed), in log hours, for Interview Cases by Time, Pain, and Transportation Variables.

Characteristics	Mean ± SE
Onset type of day	
Weekday (n=54)	0.98 ± 0.16
Weekend (n=14)	0.48 ± 0.27
Onset time of day	
Morning (n=18)	1.04 ± 0.29
Afternoon (n=18)	0.77 ± 1.30
Evening (n=19)	0.93 ± 0.28
Late Night (n=13)	0.72 ± 0.25
Location when experienced onset	
Home (n=50)	1.00 ± 0.17
Other (n=18)	0.54 ± 0.22
Location when decided to seek medical help	
Home (n=46)	0.97 ± 0.18
Other (n=22)	0.67 ± 0.22
Patient think pain was due to heart problem when experienced onset	
No (n=43)	1.09 ± 0.19*
Yes (n=24)	0.55 ± 0.18
Pain recurred when decide to seek help	
No (n=12)	1.50 ± 0.44*
Yes (n=17)	1.29 ± 0.31
Never stopped (n=38)	0.53 ± 0.14
Pain quality	
Steady (n=57)	0.90 ± 0.15
Intermittent (n=8)	1.14 ± 0.48
One Sharp Pain (n=1)	1.75
Call family doctor about symptoms/pain	
No (n=54)	0.59 ± 0.13¥
Yes (n=14)	1.96 ± 0.36
Call for ambulance	
No (n=40)	1.23 ± 0.20§
Yes (n=28)	0.38 ± 0.14
Mode of transportation to hospital	
Self-drive (n=32)	1.14 ± 0.22*
Ambulance (n=27)	0.40 ± 0.15
Others (n=9)	1.33 ± 0.50

* $p < 0.05$; § $p < 0.01$; ¥ $p < 0.001$; SE = standard error

trend was found for the 3 levels of pain recurrence. Post-hoc Bonferroni multiple comparison test showed that the there was a statistical significant difference between those who had on-going pain and those with no pain recurrence.

Mode of transportation was found to be an important factor affecting the log-delay time. Patients arriving at the hospital by ambulance were found to have shortened their log delay time by more than half compared to those who came to the hospital using their own cars. Substantial delay was also found to occur when patients contacted their family doctor about their symptoms before coming to hospital ($p<0.001$).

4.7 Effects of Contextual Variables on Pre-hospital Delay

The log-transformed delay times by various contextual variables are presented in Table 16. For patients who talked to another person about their symptoms, having someone who acted like they thought the symptoms were serious significantly shortened the log-delay time ($p<0.001$). The person who made the decision to come to the hospital also significantly affected the patient log-delay time, with both the spouse and another relative making the decision fastest, followed by the patient, and lastly an unrelated person associated with the greatest delay. Post-hoc multiple comparison tests showed that the differences detected through ANOVA were in fact due to a significant difference between those who made the decision themselves and an unrelated person, and also between a spouse and an unrelated person.

Table 16. Onset to Arrival Delay Time (log transformed), in log hours, for Interview Cases by Contextual Variables.

Characteristics	Mean ± SE
Talk to someone about pain/symptoms before coming to hospital	
No (n=8)	0.41 ± 0.31
Yes (n=60)	0.94 ± 0.16
Another person suggested it may be cardiac in nature	
No (n=35)	0.93 ± 0.22
Yes (n=25)	0.94 ± 0.22
Another person act like they thought symptoms were serious	
No (n=14)	2.17 ± 0.32[¥]
Yes (n=46)	0.56 ± 0.14
Another person suggested to seek medical help	
No (n=16)	0.82 ± 0.33
Yes (n=44)	0.98 ± 0.18
Patient take suggestion of another person to seek medical help	
No (n=9)	0.90 ± 0.49
Yes (n=41)	0.95 ± 0.18
Anyone came with patient to the hospital	
No (n=14)	1.01 ± 0.36
Yes (n=54)	0.84 ± 0.16
Who was the first person patient told about symptoms	
Spouse (n=40)	0.84 ± 0.17
Other relatives (n=8)	1.41 ± 0.51
Others (n=12)	0.93 ± 0.42
Who suggested to come to hospital	
Self (n=27)	0.77 ± 0.20
Spouse (n=18)	0.54 ± 0.25
Other relatives (n=6)	0.68 ± 0.46
Others (n=17)	1.47 ± 0.33
Who decided to come to hospital	
Self (n=45)	0.77 ± 0.14[§]
Spouse (n=8)	0.35 ± 0.50
Other relatives (n=4)	0.23 ± 0.25
Others (n=11)	1.93 ± 0.43

[§] $p<0.01$; [¥] $p<0.001$

SE = standard error

Table 17. Onset to Arrival Delay Time (log transformed) in log-hours of MI Cases who were interviewed, by behavioral questions.

Questions:	Mean ± SE (# of response)		
	Not at all	Some	A lot
Think about what symptoms meant?	0.95 ± 0.25 (26)	1.08 ± 0.23 (24)	0.50 ± 0.24 (18)
Think about how serious they were?	1.34 ± 0.25 (30)	0.63 ± 0.19 (19)	0.38 ± 0.21$^{\aleph}$ (19)
Try to ignore symptoms?	0.61 ± 0.19 (38)	1.07 ± 0.20 (23)	1.67 ± 0.54 (7)
Think about others with similar symptoms?	0.87 ± 0.16 (59)	0.89 ± 0.29 (8)	1.10 (1)
Think about the cause of symptoms?	1.03 ± 0.31 (18)	1.05 ± 0.19 (35)	0.29 ± 0.23 (15)
Try to relieve symptoms with medication?	0.52 ± 0.19 (33)	1.22 ± 0.23 (29)	1.15 ± 0.35* (6)
Try lying down and rest?	0.20 ± 0.15 (18)	0.83 ± 0.22 (30)	1.55 ± 0.25$^{\aleph}$ (20)

SE = standard error; $^{\aleph}$ $p < 0.01$; * $p < 0.05$

4.8 Effects of Behavioral Factors on Pre-hospital Delay

We examined several symptom-related coping behaviors, and found them to have a significant effect on patient delay time (Table 17). The extent to which the patient tried to lie down or rest following onset of symptoms was found to have a highly significant effect on delay times with those engaging in the behavior having a significantly higher log-delay time ($p<0.01$, trend $p<0.01$). Patients who sought relief through self-medication or other means also had a significantly higher log-delay time ($p<0.05$). In addition, patients who thought about the seriousness of their symptoms to a greater extent had a much shorter log-delay time than those who did not consider it at all ($p<0.01$).

4.9 Multivariate Analysis for Pre-hospital Delay

Linear Regression. A stepwise linear regression was conducted on the *Fastrak* group and two samples of patients who were interviewed. For the interview group, one sample consisted of 58 interviewed cases that had values on all variables we included in the pool for the analysis, with the exception of ADAS as that would only include patients who were married. The second interview sample excluded the 7 non-married cases from the previous sample, and hence had information for the ADAS questionnaire.

We reported regression models based on entry criterion for stepwise selection of $p<0.05$ instead of a more relaxed criterion such as $p<0.10$. For the regression model on the *Fastrak* group, there was no difference in the variables that were selected for the model using either criterion. On the other hand, goodness of fit for regression models on the interview group only improved slightly using a more relaxed criterion, including an addition of three to four variables to improve the R^2 only by 0.05. Given that our sample size was quite small,

we chose to report our results using a more conservative model based on the $p<0.05$ criterion. Furthermore, variables entered in the 2 models using the $p<0.05$ criterion were quite consistent, while additional variables selected with a more relaxed criterion were different for the 2 models on the interview group suggesting it was highly dependent on the sample size and may be less valid.

For all multivariate linear regression models, we examined potential effects of interaction between significant variables entered, and also interaction between each variable, age, and sex. None of the interaction effects were significant, therefore interaction terms were not included in our final models reported. Similarly, potential confounding effects of age and sex were examined and were not found to have significant effects (changes of more than 10%) on regression coefficients of variables in the models.

Model 1. The results of the stepwise linear regression conducted on the *Fastrak* group is presented in Table 18. History of diabetes, previous myocardial infarction, and gender were found to be significant in predicting the delay time. However, these 3 variables only explained 2.6% of the variance in log-delay time. When all 3 factors are considered, we found diabetic and female patients to delay longer, while patients who had a previous myocardial infarction arrived at the hospital faster.

Model 2. For the model based on 58 interview cases (without ADAS in the variable pool), we found 5 variables to be significant in predicting the log of delay time (Table 19). The following variables accounted for approximately 63% of the variance in log delay time: the extent to which the patient sought relief (e.g. taking medication or other measures), having another person behaving as if the they felt symptoms were serious, current smoker

Table 18. Multivariate Linear Regression on Log-transformed Patient Delay Time for *Fastrak* Group (model 1)

Variables	R^2	β *coefficient* ± *S.E.*	p-value
Step 1			
History of diabetes	0.01	0.26 ± 0.10	0.01
Step 2			
History of diabetes		0.30 ± 0.10	0.003
Previous myocardial infarction	0.02	-0.25 ± 0.09	0.007
Step 3			
History of diabetes		0.28 ± 0.10	0.007
Previous myocardial infarction		-0.24 ± 0.09	0.01
Female gender	0.026	0.19 ± 0.09	0.04

Table 19. Multivariate Linear Regression on Log-transformed Patient Delay Time for 58 Patients (model 2)

Variables	R^2	β *coefficient* ± *S.E.*	p-value
Step 1			
Another person act like they believe symptoms were serious	0.32	-1.55 ± 0.30	<0.001
Step 2			
Another person act like they believe symptoms were serious		-1.33 ± 0.29	<0.001
Pain recurred after initial onset of symptoms	0.42	-0.50 ± 0.16	0.003
Step 3			
Another person act like they believe symptoms were serious		-1.33 ± 0.28	<0.001
Pain recurred after initial onset of symptoms		-0.49 ± 0.16	0.003
Relieve seeking	0.48	0.46 ± 0.18	0.015
Step 4			
Another person act like they believe symptoms were serious		-1.36 ± 0.40	<0.001
Pain recurred after initial onset of symptoms		-0.53 ± 0.14	0.001
Relieve seeking		0.48 ± 0.17	0.007
Who decide to come to hospital (quadratic effect)	0.55	0.10 ± 0.04	0.008
Step 5			
Another person act like they believe symptoms were serious		-1.41 ± 0.26	<0.001
Pain recurred after initial onset of symptoms		-0.46 ± 0.14	0.002
Relieve seeking		0.46 ± 0.17	0.008
Who decide to come to hospital (quadratic effect)		0.11 ± 0.04	0.004
Current Smoker	0.58	0.48 ± 0.23	0.04
Step 6			
Another person act like they believe symptoms were serious		-1.30 ± 0.25	<0.001
Pain recurred after initial onset of symptoms		-0.36 ± 0.15	0.02
Relieve seeking		0.52 ± 0.16	0.002
Who decide to come to hospital (quadratic effect) *		0.13 ± 0.04	0.001
Current Smoker		0.53 ± 0.22	0.021
Who decide to come to hospital (linear effect)	0.63	0.11 ± 0.05	0.035

* Variable considered as ordinal based on how closely related to the patient. (i.e. self-deciding being closest to the patient, follow by a spouse, another relative, and an unrelated person.)

the extent of pain recurrence during the cardiac event, and who made the decision to come to the hospital for medical care.

Delay time for patients who had another person behaving as if the symptoms were serious was 0.27 times of those who did not. Compared to patients who did not try to seek relief, those who did to a certain extent delayed 1.7 times longer, while patients who indicated they sought relief a great deal delayed even longer by close to 3 times. Interestingly, current smokers were found to have longer delay time compare to nonsmokers by about 70%. The extent of pain recurrence is also important, with those having recurrence arriving 30% faster than patients who didn't experience any pain recurrence. Those who had pain that did not go away came to the hospital fastest at about half the time of those who did not have any recurrence in their pain. Finally, we found that patients arrived fastest when the spouse or another relative made the decision to come to the hospital, while those decided by an unrelated person experienced the longest delay.

Model 3. The regression model conducted on 51 patients who were married and had data for all variables including the ADAS measurement was very similar to model 2 (data not reported). In this model, 66% of the variance in log delay time was accounted for by 4 variables. Similar to the previous model based on 58 patients (model 2), extent of pain recurrence, extent of relief sought, who made the decision to come to the hospital for medical care, and having another person suggesting the symptoms as serious were found to be most significant. In addition, the effects for the in both models were similar. Therefore, our subsequent discussion for multivariate analysis will focus on results obtained from the larger sample (model 2).

Through our analysis, we found ADAS score did not contribute significantly in predicting the log-delay time when compare to the other variables that entered into the regression model. Furthermore, correlation analysis between log-delay time and the ADAS score showed a non-significant Pearson R coefficient of -0.23.

Logistic Regression. Logistic regression analysis was performed due to the clinical significance of such results. We feel that identifying factors that may associate with a delay of more than 1.5 hours will perhaps be more useful in shaping our public health message to shorten patient delay time. The result of our logistic regression model based on the *Fastrak* group was similar to the linear regression model (model 1). However, the validity of our results for the interview group was limited due to the small sample size. In particular, the results based on 67 interviewed cases were completely different than the model based on only 55 cases (i.e. all married with ADAS variable), with a completely different set of variables identified as significant variables. We were also not able to include all independent variables in the selection pool as we did for the linear regression model, and could not examine any possible significant association in a multivariate model for variables not available for selection. Furthermore, the odds ratio obtained was quite extreme (e.g. 0.06 when compare to the reference group) or had very wide confidence intervals. Therefore, we opted not to report our results for the logistic regression analysis, however, the clinical significance of such analysis should be kept in mind for future studies.

Residual Analysis. Preliminary residual analysis was performed to examine any violation of assumptions for our multiple linear regression models. The distribution of standardized residuals was examined and found to approximate a normal distribution. Furthermore, Q-Q plot of standardized residuals showed data points to fall closely to a

straight line suggesting normality. In addition, residuals were plotted against the predicted values of log-delay time and appear to be in a random pattern. Finally, we did not detect any outliers in our analysis.

5. DISCUSSION

Over the past decade, several large-scale clinical trials have reported the efficacy of thrombolytic therapy in reducing mortality of an acute myocardial infarction (8,9,20). However, the benefit decreases substantially as time increases between onset of symptoms and drug administration. This has resulted in a large number of studies attempting to identify factors that are associated with pre-hospital delay, in hopes to better educate patients with a myocardial infarction to present promptly to the hospital. Of those studies, two were conducted in Canada (32, 49).

Almost 10 years have passed since the first Canadian study on patient delay was conducted by Wielgosz et al (32). Our current study is a follow-up to that initial report and one of the most extensive studies on patient delay ever conducted in Canada. In particular, we hoped to examine whether delay time has improved since the previous report and also to investigate factors associated with patient delay so we may validate previous findings and the current health message.

Our results suggested 4 factors to be most important in explaining patient delay time. When all the 4 factors were considered together, having a witness act like he/she felt symptoms were serious reduced delay. Meanwhile, patients who tried to self-medicate to a great extent had a longer delay. Interestingly, we found the person who made the decision to seek medical help played a role in affecting delay time. Furthermore, individuals who suffered pain recurrence or ongoing pain seemed to have a longer delay.

While numerous studies have attempted to identify factors that may associate with patient delay time, most studies collected data retrospectively through chart review, thereby limiting the number of variables that may be study. In particular, very few studies addressed

the importance of patient behavior and role of witnesses in affecting delay time. The data for our interview group were collected prospectively as new patients were identified after the start of the study and allowed for collection of detailed information regarding numerous factors relating to the myocardial infarction. In addition, unlike most studies that only utilized bivariate analysis, we included multivariate statistical methods in analyzing our data to determine the most important independent factors in predicting delay time, taking into account possible inter-correlations among various factors.

In order to minimize any error in recalling information by patients who were interviewed, we collected data within 2 to 4 days following admission to the hospital. Furthermore, we evaluated the agreement between data collected from hospital records in the *Fastrak* database and those that were interviewed to examine validity of our data. Finally, our study only included patients who had a confirmed diagnosis of myocardial infarction thereby truly reflecting the delay time and behaviors of those who suffered a heart attack rather than some other illnesses mistaken to be a myocardial infarction.

Despite improvements over some previous studies, our study has several weaknesses and limitation. Most importantly, our small sample size for the interview group may limit our power to detect possible effects of some independent factors. The fact that our interview group was a convenience sample may also introduce bias and limit our ability to generalize the findings to the actual population of MI patients. Another major weakness of our study is the potential inaccuracies of delay time, particularly relating to time of onset, as this was heavily dependent on patient history and interpretation by the attending physician. Other potential biases include recall and interview bias, which may also affect the validity of our findings. We shall discuss these in detail in a later section.

5.1 Validity

The kappa coefficients for nominal variables were acceptable with the exception of "history of angina". This is likely related to the lack of knowledge patients have regarding angina, and the difficulty in understanding the definition of angina. In addition, many patients may consider previous angina as a heart attack, thereby under reporting the condition. Cases reported with angina in *Fastrak* should be more accurate since information was obtained both from patients, previous medical records, and in some cases, contacting the family physician. As expected, sex and age had perfect correlation between data recorded in *Fastrak* and those recorded during interviews.

It is difficult to measure the validity of delay time since pre-hospital delay was determined by calculating time difference between onset and arrival in the *Fastrak* group, while for interview cases, our estimated pre-hospital time was determined by summing the patient decision time and the travel time. Therefore, the pre-hospital delay time determined from the *Fastrak* data should be longer since it included additional time required to wait for ambulance, or prepare to travel to the hospital. The Pearson correlation coefficient was 0.77. Perhaps a better measure of validity was to look at the difference between the onset time recorded in *Fastrak* and that obtained from the interview. If it was 100% valid, we would expect a difference of 0 minutes. Our data show a mean difference of 8 minutes and no significant difference from 0 minutes, which is quite acceptable.

5.2 Delay Time

Our findings indicate that substantial delays occur in response to symptoms of a myocardial infarction. The 692 patients admitted to the Ottawa General Hospital delayed on average 5

hours and 23 minutes (median 2 hours and 8 minutes) before arriving at the emergency department. This is an improvement over the 6 hours and 51 minutes (median 2 hours 40 minutes) reported by Wielgosz et al. in 1988. In addition, only 31% of our patients delayed more than 4 hours, compare to 42% a decade ago.

While our study population of patients consisted only of those who were admitted to the Ottawa General Hospital, Wielgosz et al. studied 201 patients who were admitted to one of four hospitals in the Ottawa region (Ottawa Civic, Ottawa General, Queensway-Carleton or Winchester Memorial). Despite this difference, it is unlikely that the improvement in delay time found in our study is simply due to different group of patients who seek medical care at different hospitals in the region, especially when the demographics of our patients are similar to the previous study. In addition, the majority of patients included by Wielgosz et al. were in fact from the Ottawa General Hospital and a similar method of identifying subjects as the *Fastrak* study was used.

Interestingly, our patient delay time is very similar to those in the United States (51,53,66,72). One might have expected that Canadians would have shorter delay time due to universal access to health care. The importance of health care access have rarely been investigated with only Yarzebski et al. (72) reporting shorter delay for patients with Medicare-Medicaid compare to those with private insurance or self-pay. Cooper (27) and Ell (31) deduced that longer delay observed among African Americans points to confounding effect of economic status, which often associates with ethnicity, and directly affects the ability to access medical care in a private health care system in the United States. However, despite free access to health care in Canada, our study and previous Canadian studies (32,49) reported AMI patients to have patient delay times similar to those in the United States,

suggesting free access to health care does not necessarily ensure improvement in patient delay.

The median time from onset of symptoms to arrival to the emergency department for 1357 Quebec patients (49) was shorter than that for our patients (1.5 hours), but they only included patients who were eligible for thrombolytic therapy, hence we expected them to have a shorter delay. In fact, when we examine only patients who received thrombolytic therapy in the *Fastrak* database, their onset to arrival delay time was indeed quite similar to the Quebec study with a median of 1.7 hours. Among those who received thrombolytic therapy in our study, more than 50% received it within 1 hour of hospital presentation. This is a significant improvement over the results from Canadian hospitals in the 1991-1992 GUSTO-I trial, which reported 75% of patients waiting more than 1 hour before treatment (73). However, that may be attributed to additional delay for enrolment and research protocol. Our median door-to-needle time of 55 minutes is comparable to that reported in Quebec (59 minutes), but is significantly longer than the ideal 30-minute period currently recommended (74).

The drug preparation time was significantly shorter in our study (median 14 minutes) than those reported in Quebec (median 22 minutes), and may point to greater efficiency at our hospital although even shorter delay should be targeted. For more than 50% of our patients, 15 minutes elapsed upon arrival before an initial ECG was obtained. This points to the need to improve our triage mechanism.

5.3 *Fastrak* Group vs. Interview Sample

The characteristics of patients interviewed differ in several aspects with those in the *Fastrak* group. There was a higher proportion of male patients in our interview sample (75% vs. 65%), likely due to the fact that we only had 68 patients in the interview group. In such a small sample size, a slight change in patient characteristics may result in significant changes in proportions. In fact, if there were only 6 fewer males in our interview sample, the proportion will change to be similar to that in the *Fastrak* group.

The mean age of our interview group was younger than in the *Fastrak* group by 4 years. Although the difference is statistically significant, the clinical significance of such a difference as relating to delay time is questionable. The difference in age is likely related to the difference in the gender balance of our sample. It is known that females generally suffer from cardiovascular diseases at a later age than males. Therefore, with a smaller proportion of females in our interview subgroup, one would expect the mean age to be younger. However, females in our interview group were younger compared to the *Fastrak* group, and in fact, females were not significantly older than males in the interview group as one would expect, raising the possibility of potential bias from our sampling method.

In addition, the *Fastrak* group had a higher proportion of patients with a history of angina and congestive heart failure. This might suggest that patients in the interview sample were healthier with fewer associated cardiovascular problems. The fact that we did not capture 100% of all potential subjects also raises concerns about the possibility that those who refused to participate or were not available may have had more serious disease, preferring not to be bothered during their stay in hospital. However, despite these differences in patient characteristics between the *Fastrak* group and those who were

interviewed, no difference was found in delay time between the two groups. One would have expected delay time for the interview group to be shorter given that older age and female gender have both been reported to associate with longer delay time. This suggests that our convenience sample may be biased and may not be representative of the *Fastrak* group population, hence, limiting our ability to generalize our results.

5.4 Effects of Demographics and Medical History on Pre-hospital Delay

Analyses conducted on 692 patients in the *Fastrak* database revealed a significantly longer delay in patients who were female and diabetic. Meanwhile, those who had experienced a previous episode of myocardial infarction had a shorter delay time. This is different from the earlier results reported by Wielgosz et al., who found no demographic or medical history factors with significant effects on delay time. However, that analysis was only conducted on 42 patients and likely lacked statistical power to detect significant differences. In fact, when we conducted a similar analysis on our interview subgroup of 68 patients, previous myocardial infarction and history of diabetes were no longer significant.

Recent studies with sample sizes larger than 500 patients have consistently reported longer delay for female and diabetic patients (41,43,52,54,56). In general, heart attacks occur nearly as frequently in men as in women, but there is a widespread misconception that acute myocardial infarction is a "male" disease, making women less apt to interpret their chest pain as a heart attack. Also, women who suffer from myocardial infarction often have atypical presentations that may be more difficult to recognize, causing a longer delay. Diabetic patients on the other hand, often have associated neuropathies that lessen their ability to notice changes in the body (75,76) leading to longer delay.

An alarming finding in previous studies was the lack of shortening of delay time among patients with previous episodes of MI (30,32,43,50,53,55). One would expect a previous attack to make a person more aware of any signals of myocardial infarction. However, the lack of such a finding may relate to the sample size or the statistical methods used. Most of the studies that reported no effect conducted their analyses by categorizing the time variable, thereby losing valuable information (32,43,53,55). Our current findings suggest that patients with previous myocardial infarction arrived at the hospital faster. Perhaps patient education after an acute myocardial infarction has improved thus improving early recognition of symptoms, or perhaps our patients with previous heart attack were more likely to believe they were at risk for suffering another episode. This finding is consistent with those reported by Meiskchke (41), Newby (54), and Karlson (58).

We found a lack of association between age group and time, which may result from the age categories we chose. Studies that found longer delay for older patients often dichotomized the age into 2 groups (e.g. < 65 vs. ≥ 65). Older patients may have longer delay for several reasons. One explanation may be that older individuals perceive their symptoms as less severe or believe their symptoms are due to other health problems. In addition, older patients may have difficulties with mobility that cause a longer delay.

Demographic and medical history variables on pre-hospital delay are likely to be less important than other contextual or behavioral variables. When we performed a stepwise linear regression on the *Fastrak* group, females, diabetics, and patients with previous MI were the only significant variables. However, this model only explained 2.6% of the variance in log-delay time.

5.5 Effects of Pain and Transportation on Pre-hospital Delay

All studies that investigated the effect of patients contacting their family physician for their chest pain have shown a significant association with longer delay (27,31,43,53,66,77). In our study group, we also found a significantly longer delay for those patients who contacted their family doctor about their symptoms. This observation was expected since substantial additional delay can occur if patients fail to contact their family doctor immediately, wait at the doctor's office for an appointment, or if the family doctor fails to recognize the symptoms. Such a phenomenon may pose a problem for MI patients since the trend in our health care system is to de-emphasize the use of emergency department as a primary site for patient care and encourage patients to visit their family doctors first. However, one must realize such a trend is mainly aim towards patients with minor medical problems such as colds or flu, and the seriousness of a potential fatal cardiac event should not simply be view in the same manner.

Encouraging patients to seek medical care directly at the emergency department may result in an increased number of patients with non-cardiac chest pain appearing at the emergency department, placing additional strain on limited facilities. Yet, unless we have highly sensitive and specific ways for patients to correctly diagnose their symptoms as a heart attack themselves, we should not be placing patients at risk for increased mortality from possible myocardial infarction simply as a trade off to keep false positive cases away from the emergency department. The Heart and Stroke Foundation supports this view: their current message in the "Signals and Actions" campaign is for individuals to seek emergency help right away if experiencing typical symptoms of a heart attack. Since our study only studied patients who truly suffered a heart attack, the behaviors and presenting symptoms of

patients who did not have a myocardial infarction could not be investigated. Future studies should perhaps focus on these patients to determine if their psychological thought processes and symptoms are significantly different than true cases. Such results could then be used in public education campaigns to decrease the number of false positive cases presenting to the emergency.

Patients who arrived by ambulance were found to have a shortest delay, while those who arrived by taxi or public transport had the longest delay. However, the difference in delay time cannot be accounted for by the travel time of the various transportation modes alone. In fact, travel time for various transportation methods were quite similar. Instead, patients who called for an ambulance may have had more pain or considered their symptoms more serious. This may explain why mode of transportation was no longer significant when we conducted a multivariate analysis taking into account the contributions of other variables to delay time.

Patients who attribute their symptoms to a heart problem have been reported to arrive at the hospital sooner (25,50,57,66,77). The sample of patients we interviewed displayed a similar behavior, suggesting the importance of the ability to recognize the pain or symptoms as cardiac related. Some studies found patients who experienced intermittent symptoms delayed longer (28,31). While we did not detect a significant difference between those who experienced steady pain and those with intermittent symptoms, the difference of 45 minutes for median time cannot be ignored.

In addition, we found patients who had pain recurrence arrived at the hospital faster than those without pain recurrence, and fastest when the pain never stopped. Goldberg et al. reported a similar observation with pain recurrence associated with greater than 2 hours

delay in a logistic regression analysis (51). The interpretation of pain recurrence is a difficult one though, and may in fact be biased as it may be related to how quickly a patient reacted to the symptoms. For example, an individual who claimed to have ongoing pain until arriving at the hospital could have been classified as having pain recurrence if he waited longer and the symptoms subsided and developed again. Similarly, this same person could have been classified as having no pain recurrence at all if he delayed so significantly to the point that pain completely subsided. Thus, reporting of this factor could actually be dependent on the outcome (i.e. delay time) leading to biased interpretation.

5.6 Effects of Contextual and Coping Behavior on Pre-hospital Delay

While the potential of family intervention is recognized in the literature (78), most studies fail to consider the influence of family, relatives, or co-workers on the impact of pre-hospital delay. Hackett et al. first suggested that consultation with a family member about the decision to seek treatment significantly increased delay. This was further supported by a subsequent study that found the influence of a family member caused increased delay (37). Alonzo postulated that family members cause longer delay as they may have been motivated to share the denial of the patient or may have been less willing to confront the patient's delay. Contrary to those findings, we found that a spouse or relatives making the decision led to earlier arrival, while patients who allow an unrelated person to make the decision delayed significantly longer.

Moss (38) and Hartford (66) have both reported a similar finding that patients turning to their spouse for assistance resulted in a shorter delay. The difference in results of various studies may relate to the fact that some studies reported the effect of the first person who

became aware of the illness (37), while others focused on the person who made the decision to seek medical help (38,66).

Perhaps more important than determining the difference between family members and others in influencing the patient pre-hospital delay is to determine whether the witness thought the symptoms were serious enough to seek immediately medical attention. We found patients arrived significantly faster when the witness felt the symptoms were serious ($p<0.001$) regardless of whether the witness was the spouse, relative, or an unrelated person. No other studies have considered this as a possible effect for investigation. Rather, they focused on the impact different witnesses have on delay time. Our findings suggest that conflicting results may be related to the perception various witness groups have on the seriousness of symptoms rather than on their ability to influence the patient as suggested by some investigators (37).

The importance of coping behaviors by the patient on pre-hospital delay has rarely been investigated in the past. Wielgosz et al. reported patients who sought relief (e.g. taking medication and rest) to a greater extent had significantly longer delay. Similar findings were reported by Alonzo who found variables relating to self-treatment or relief seeking behaviors to be one of the best predictors of increased delay in a multivariate analysis. We also found the same trend in our study with those who tried to rest and self-medicate delaying longer. While most individuals who attend to their symptoms will attempt to relieve the symptoms either with medication or rest, such behaviors are very time consuming because they include an "effect interval" during which the patient must wait to evaluate the effectiveness of the treatment resource. This is true especially for patients with a history of coronary artery disease as they may attempt to relieve symptoms of a myocardial infarction with

nitroglycerine. This may explain why some investigators have reported longer delay for MI patients who had experienced a previous MI (72).

While the witness' perception of symptom seriousness was found to be important, so was the perception by the patient. We found a significant trend with patients who considered the symptoms to be serious delaying less. Given that the patient's delay time is influenced by the perception of those who witness the episode, we will discuss the importance of this variable when we discuss our multivariate analysis.

5.7 Multivariate Analysis

A great number of studies have been conducted over the years to examine factors that are associated with delay in response to symptoms of acute myocardial infarction. However, most of these studies relied on bivariate correlations or analyses of variance as the primary method of analysis, without consideration of any important inter-correlation among different factors. The results of our bivariate analyses were discussed extensively above to allow for comparison with the majority of published studies.

For the few studies that utilized multivariate analytic tools, behavioral factors have been shown to be independent predictors of delay time (32,37,50). However, comparison between different studies is difficult as different investigators chose to investigate different types of behavioral factors. In addition, some studies only included demographic or medical history variables for selection in regression analysis leading to poor predictive values. When we conducted a multiple linear regression on the *Fastrak* group using available demographic and medical history variables, we only found 3 variables (gender, diabetes, previous MI) to be independent predictors of delay time with only 2.6% of the log-delay time variability

explained. This suggests that medical history and demographic factors cannot adequately explain why patients delay in responding to their symptoms of AMI. Rather, additional factors must be investigated such as those obtained through our interview group.

Our multiple linear regression analyses for the interview group found 5 variables to have strong predictive value for log-delay time with 63% of the variability explained. The 5 factors were: witness acted like they believe symptoms were serious; pain recurred after initial onset of symptoms; extent of relief seeking by medication; who decided to come to hospital; and current smoker. This is a marked improvement over the model that only included demographic and medical history variables (model 1), which required 3 variables to explain just 2.6% of the variance.

Patients who reported "having a witness acting like they believe symptoms were serious" arrived at the hospital much sooner. As discussed before, no other studies have investigated the importance of this variable. It would seem logical for the witness to influence the patient's perception of symptoms leading to earlier arrival. Some studies in the past have reported earlier arrival when patients considered their symptoms as serious, but failed to investigate whether this was influenced by the witness' perception. In addition, if the witness believed the symptoms were serious, he or she may have discouraged the patient from engaging in any behaviors which were found to be associated with longer delay in the bivariate analysis (e.g. call family doctor, resting). We wonder if the different types of witnesses (family, relatives, or unrelated person) may differ in their perception of symptom seriousness. If there is any difference, it may explain some of the results reported in the past. When we examined the different group of witnesses however, we did not find differences in the perception of symptom seriousness.

On-going pain or recurrence was also found to be independently predictive of delay time. Several reports in the past suggested patients arrived earlier if the symptoms were more severe (51,52,55,55,57). While we did not take into account the degree of severity of the symptoms in our study, the character of the symptoms may be an indicator of the severity. Patients who experience on-going pain will no doubt consider the episode as more severe, thereby being less likely to ignore the symptoms and less likely to delay in seeking help. In addition, if patients have ongoing pain or recurrence of pain, they will likely take early action rather than endure their symptoms. However, as discussed earlier, the importance of this variable may be biased due to the fact that the experience of pain recurrence can actually be dependent on the length of delay time. Therefore, one must be cautious when considering the importance of this factor.

The extent of relief sought was determined to be a strong predictor of delay time by Wielgosz and Alonzo. We found a similar result in our analysis with patients seeking relief with medication delaying as much as three times longer. The explanation for such a finding was discussed earlier, and suggests the importance of establishing strategies to prevent patients from excessively engaging in such behavior. Interestingly, patients who had a history of angina or previous myocardial infarction are often told to relieve their symptoms with nitroglycerin, and only if this fails, to seek medical help at the hospital. The fact that some studies have reported patients with history of myocardial infarction delayed longer may be related to patients attempting to self-medicate excessively before seeking help at the hospital. The extent to which patients try to relieve their symptoms needs to be investigated further so a clearer public health message can be formulated to discourage over dependence

on self-medication in dealing with symptoms of an acute myocardial infarction, thereby preventing excessive delay in seeking medical care at the hospital.

The individual who made the decision to come to the hospital was found to have a significant role in explaining patient delay in our regression model. The presence of a quadratic effect suggests that having a spouse or another relative deciding to come to the hospital helped in shortening delay time, while those who decided themselves or had an unrelated witness made the decision delayed the longest. This is consistent with other studies that focused on examining the effects of witness making the decision to seek medical help (38,66). Several studies only examined the role of the first witness who became aware of the illness and reported contrary results (37,57). However, this should not be considered in the same manner as the role of the person who actually made the decision to come to the hospital. In fact, when we examine our interview group, the first person who was told about the patient's symptoms was not found to affect delay time both in the bivariate and multivariate analyses.

Interestingly our analysis suggested current smokers to have an increased delay time. However, this was not consistent in both models. Few studies in the past have included smoking as a variable; Schmidt et al. reported no effect, while Turi et al. observed a decrease in delay time. The importance of smoking is questionable as it may in fact represent other factors that were not investigated in our study. Indicators of low socioeconomic status have been shown to be associated with longer delay in some studies (28,53). Furthermore, investigators who found significantly longer delay in African Americans have suggested possible confounding by socioeconomic status. It has been found that smoking is associated with lower socioeconomic status, and our results raise the possibility of such a link. On the

other hand, smokers may also be individuals who are less concerned about their health, especially when it is already well known that smoking is a risk factor for cardiovascular disease. This group of patients may pay less attention to their health status and may be more inclined to deny having any potential serious illness, hence delaying longer even when symptoms occur. Regardless of the explanation, one must investigate this factor further before any conclusions can be drawn.

While we performed logistic regression modeling with our interview sample using 1.5 hours as an outcome, the validity of the result is questionable due to our relatively small sample size. However, the importance of utilizing such statistical method should be emphasized in future studies due to the clinical importance of identifying factors associated with delay of more than 1.5 hours.

5.8 Clinical Implications

Based on our analyses, we were able to identify several important factors that were associated with delay time. From the *Fastrak* database, gender, history of diabetes, and previous myocardial infarction were significantly associated with delay time, however, all of these factors were non-modifiable, unrelated to the behavior of the patient. In addition, we found these demographic and medical history factors had poor predictive value in explaining log-delay time when compared to other behavioral variables examined with the interview group. Hence, from the public health message point of view, the clinical importance of the factors identified from the *Fastrak* database is minimal except suggesting that studies should focus on identifying behavioral factors that may associate with delay time.

On the other hand, when we examined the data from the interview group, we found several variables to have significant predictive value for log-delay time. Encouraging is the fact that several of these factors are related to modifiable behaviors that may be used in educating patients. In particular, we found patients who had a witness who felt the symptoms were serious and those who had a spouse or relative deciding they should go to the hospital, were associated with a shorter delay time.

While we did not study how witness behaviors were determined, our findings suggest that patients should inform their spouse or a relative about their symptoms because unless patients take this first step, they will not be influenced by a witness which may help in shortening delay time. This does not mean that we encourage every single person experiencing chest discomfort to seek care at the hospital, but rather to have the symptoms evaluated by another person, especially someone who will be quite concerned about the well-being of the patient such as the spouse or a relative. Our message is similar to that of the Heart and Stroke Foundation *Signals and Actions* campaign in which the public is told to "Tell someone RIGHT AWAY" when experiencing signs that are commonly encountered during a heart attack. Such message will not necessarily create more visits to the emergency department since the witness may feel the symptoms were not serious and encourage the patient to self-medicate, rest, or perhaps visit the family doctor instead. On the other hand, if symptoms were significant and the witness felt they were serious, this will help to bring the patient to the emergency department within a more reasonable time frame. In fact, from the experience of the Ottawa General Hospital, we have not noticed a flood of patients to the emergency department with non-cardiac chest pain since the initiation of the *Signals and Actions* campaign. Certainly, the sensitivity and specificity of witness's perception in not

known and cannot be examined in our study since we only included true cases of myocardial infarction, but the issue should be addressed in future investigations to determine the value of witness perception.

In addition, we found the extent of self-medication to relieve symptoms associated with longer delay. This is to be expected and patients should be discouraged from being overly dependent on self-medication when experiencing symptoms. We do not suggest that patients should completely refrain from using nitroglycerin when they experience chest pain and just come to the hospital, but if after 2 to 3 doses of nitroglycerin and their symptoms still persist, patients should seek medical care at the emergency department instead of further self-medicating.

The fact that our study focused on bringing AMI patients to the emergency department may concern some in the health care sector who feel that patients should be encouraged to seek medical help from their family doctors first for their medical problems. However, as mentioned earlier, the significance of early treatment for an acute myocardial infarction should not be overlooked in an attempt to lower health care costs in the emergency department. While patients with minor aches and colds should avoid using the emergency department as a primary site for medical care, the same could not be said for patients who experience chest pain. There is no doubt that symptoms of chest pain may be related to gastrointestinal reflux, but it is unreasonable to expect patients to make this distinction while experiencing symptoms and to encourage patients with symptoms of a possible MI to visit a family doctor first could worsen the outcome. Hence, from the cardiac care point of view, unless we have highly sensitive and specific tools which patients can use to determine the cause of their symptoms, we cannot simply provide sub-standard level of care and jeopardize

the possibility of increased cardiac damage or mortality for the sake of lowering health care costs.

Our findings validate the current message used in the Heart and Stroke Foundation *Signals and Actions* campaign. Perhaps the new message should be "Tell your spouse or a relative RIGHT AWAY" instead of just informing "someone". For individuals who have known cardiovascular diseases and risk factors, it will be important to discourage over reliance on self-medication when experiencing symptoms. Primary care physicians and cardiologists can achieve this goal through health education when prescribing medications such as nitroglycerin.

5.9 Limitations and Potential Biases

There are several limitations in this present study that must be addressed. Our study group consisted of patients who survived their acute episode long enough to be admitted to the hospital. Patients who suffered a fatal attack before arriving to the hospital were not included, and the factors associating with such cases or their delay time cannot be studied easily. Since the aim of shortening delay time is to decrease the mortality of a MI, it would seem more appropriate to also include patients who died prior to arrival in hospital, especially if these cases proved to have the longest delay. However, the difficulty in obtaining detailed information for these cases prevents investigators from doing so. The feasibility of such a study is questionable given that the information would have to be obtained soon following the event from a witness or family member, whose recollection and interest to participate may be affected by the grieving process. In addition, coping behaviors of the patient seem to be one of the most important factors in affecting delay time as

indicated in our study and those reported in the past. Such information will be extremely difficult to obtain since interviews cannot be conducted with the patient to determine how they coped with their symptoms of MI. Furthermore, based on the experience of the cardiology department at the Ottawa General Hospital, it was felt that only approximately 25 to 30 patients will be "dead-on-arrival" cases each year, and it would therefore take a considerable amount of time to collect an adequate sample to conduct any meaningful analysis.

While the *Fastrak* database contained data for all patients who were diagnosed with myocardial infarction at the Ottawa General Hospital, we excluded 92 patients who were transferred from another hospital, in particular, community hospitals outside the city. This may introduce bias as patients in surrounding areas without direct access to a major medical center may behave differently and have different level of delay than individuals who live in the city allowing them to come directly to the emergency department at the Ottawa General Hospital. In addition, 122 cases were excluded because they did not provide data for symptom onset. These cases could be additional patients who developed MI while they were in hospital for other illnesses or they could be patients who presented to the emergency department with MI but could not provide the information for time of onset. Unfortunately, the information available from the *Fastrak* database does not allow us to make such distinction. However, if they consist of the latter group, then potential bias may have been introduced. In particular, patients who could not provide information regarding their symptom onset may had such a long delay that they could not remember the onset of the episode when history was taken. Excluding these patients may lead to underestimation of the actual delay time for MI patients.

The patients studied were admitted into a single tertiary care teaching hospital located in a medium size city, and may not be truly representative of the general population who suffer a heart attack. However, we believe patients are just as likely to seek medical care at the Ottawa General Hospital emergency department as at other hospitals in the city especially since the Ottawa General Hospital is not a particular specialized centre for cardiac care. In addition, the selection of subjects only from the hospital should not introduce any Neyman-type bias in terms of our research goals since the aim of this study is to examine factors that may be associated with delay for those who come to the hospital for their myocardial infarct. Therefore, the patients we included in our study were chosen from the population of patients that we hope to generalize our results on, although this may be limited to a population of MI patients in a medium-sized Canadian city.

The sample of patients who were interviewed was a convenience sample of patients whom the investigator was able to see while they were in hospital. Since the investigator could not wait for all subjects to arrive at the hospital, many of the potential candidates were unavailable at the time the investigator visited their room. This may have introduced bias as we ended up interviewing patients who were not critically ill and were more willing to participate in the study. In addition, the fact that we excluded patients who spoke only French may limit our ability to generalize the results to the greater Ottawa-Carleton region. The fact that certain patient characteristics in the *Fastrak* group differ from the interview group further points to the possibility of bias in our sample selection. In particular, the interview sample was younger and had lower proportions of cardiovascular risk factors suggesting perhaps a healthier group compare to those in the *Fastrak* database. Such bias may lead to underestimation of the actual delay time and affect the validity of association

between various factors and delay time. The ability to generalize our results to a different population may also be limited given the fact that our results were based on a small convenience sample that may not be representative of the actual population of patients with myocardial infarction.

The accuracy of the recorded delay time is also of concern. We chose to use the pre-hospital delay time recorded in the *Fastrak* database for our analysis in the expectation that the medical records and ambulance record will provide more accurate information regarding the delay time. Fortunately, we did find a good correlation between the pre-hospital delay time recorded from medical charts and that obtained through interviews. However, this was not a perfect correlation. We can only determine the patient decision time (onset to deciding to seek medical help) and travel time from the interview data. Therefore the pre-hospital delay time determined from the interview data will likely be shorter since it did not take into consideration the time it took for the patient to wait for an ambulance or prepare to leave for the hospital. This limits our ability to truly test the validity of the delay time reported. However, it is encouraging to see that the mean difference for symptom onset time recorded with the two methods to be only 8 minutes. Regardless, the true onset time depends on history given by the patients, and the interpretation by the attending cardiologists as to what symptoms constitute the actual onset. Such a limitation was inherent in all studies conducted in the past including ours.

In addition to the validity of delay time, other information collected was based on patient self-reports with all the inaccuracies therein. The questioning of patients were performed by only one interviewer using a standardized questionnaire, thereby lessening the variability in obtaining information from patients. While many of the questions asked

required discrete responses with just a yes or no answer, many of the contextual and behavior questions depended heavily on the perception of the patient, and the possibility of recall bias and interpretation is inherent. The possibility that patients with longer delay may recall information differently than those with shorter delay exist and could contribute to potential bias leading to over estimation of the strength of association between certain factors and delay time. Furthermore, the potential for interview bias, however small, should not be ignore. Although the subjects were not classified into any outcome groups, the interviewer did obtain information regarding delay time while probing for other potential factors associated with delay, and may thus introduce interview bias into the study.

Finally, perhaps the biggest limitation in our study is the relatively small sample size of the interview group. The advantage of a prospective cross-sectional survey allows us to obtain more detailed information from the patient, which would be limited if data were collected retrospectively. However, such an advantage comes at a price for it is extremely time consuming to conduct a large number of interviews. Our current study interviewed 74 patients, with each interview lasting over one hour. With the large number of potential factors affecting delay, one would require a large number of cases to conduct a meaningful logistic regression analysis to prevent any category with empty cells. Furthermore, in order to capture a consecutive series of patients, an interviewer would have to be waiting in the hospital full-time to ensure an interview within the 4 to 5 days of hospital stay. Such a project would require financial resources that were not available for this current study.

Based on our experience from this study, several recommendations can be made for future studies. Given the potential for bias from sample selection, it would be useful to conduct multi-center studies to ensure we capture a more general population of patients in

order to improve our ability to generalize findings. It would also be important to interview a larger sample of patients providing enough power for various statistical analyses. In addition, every attempt should be made to capture all potential interview subjects to limit the potential for biases. Those who refuse to participate should be documented to examine possible differences in patient characteristics between participants and non-participants.

While our study focused on delay time of confirmed MI cases and took on the perspective of cardiologists, future studies should include patients on the basis of presenting symptoms resembling an MI (i.e. chest pain) rather than only including those diagnosed with an MI. Such an inclusion criteria will allow us to truly examine how patients behave when they suffer symptoms that may resemble an MI and its effect on delay time. In addition, studying all patients who presented with chest pain will also allow us to determine any differences in behavior and delay time between those who actually suffered from a myocardial infarction and those who had chest pain for other medical reasons.

The determination of symptom onset in future studies must be more systematic, perhaps utilizing a standardized criteria form which attending cardiologists may utilize when determining the true onset of symptoms. One possibility is to have attending physicians record information regarding the symptoms experienced by the patient on a standardized form, and then have a trained individual determine the onset of symptoms and time based on the information recorded, thereby reducing the variability in interpretation of symptom onset.

Studies that conduct interviews should also keep in mind the potential for interview bias during the design stage. Future studies conducting interviews with patients should obtain information regarding the patient's behaviors separately from obtaining information concerning the delay time. This can be achieved by having 2 separate interviewers, one

obtaining information for delay time only, while another interviewer obtains information regarding the context and coping behaviors of the patient during the episode. Finally, information should be collected as soon as possible following the event to prevent any error in recalling information by the patient.

6. CONCLUSION

This study is one of the few that studied the factors associated with delay in response to symptoms of an acute myocardial infarction in a Canadian setting. Similar to those reported in the past, we found substantial delay between onset of symptoms and arrival to hospital. The fact that the Canadian universal health care system provides access to health care to all Canadians regardless of socioeconomic status did not seem to lessen the pre-hospital delay as compare to delay times reported from the United States. However, compared to a similar study more than a decade ago, it is encouraging to observe some improvement in patient delay time which may point to better patient education on the importance of prompt arrival to the hospital for treatment.

The importance of various factors in affecting pre-hospital delay time were investigated through bivariate and multivariate analyses. Our results suggest that demographic and medical history factors cannot adequately explain why certain patients delay significantly. Instead, the patient's perception of the symptoms and their coping behaviors can strongly influence pre-hospital delay time. In particular, education efforts should be aimed at preventing patients from overly relying on self-medication when experiencing symptoms as this was one of the modifiable behaviors that significantly associated with delay time.

The finding that suggests having a witness acting like they believe symptoms are serious will shorten delay time is important. Previous studies have neglected to investigate how various witnesses perceive the symptoms of a patient, but instead focused on who the witness was. Further investigations are needed to determine whether different witnesses perceive symptoms of a patient differently (i.e. an unrelated witness may more likely to

perceive the symptoms as serious or vice versa). In addition, the fact that delay time seem to be influenced by the person who made the decision further points to the importance of informing someone when experiencing symptoms of an acute myocardial infarction. It seems that patients will indeed benefit from informing someone else when they suffer a heart attack especially a spouse or a relative and should continue to be emphasized in education campaigns.

The influence of smoking on delay time is interesting and requires additional validation. Particularly, one must investigate further whether the effect of smoking on delay time is confounded by socioeconomic status, or is it truly an independent predictor of delay time. Future research should be focused on modifiable factors that can be changed through education and also examine how behaviors of witnesses are determined. Furthermore, results of our study should be validated with a larger sample of patients along with the numerous improvements suggested in the previous section to minimize the potential for bias.

Reference List

1. Heart and Stroke Foundation of Canada: Heart Disease and Stroke in Canada Ottawa, Canada, 1997.

2. Nair C, Nicholls E. Cardiovascular Disease in Canada. Ottawa, ON: Canadian Centre for Health Information, Statistics Canada, 1990.

3. Johansen H, Nair C, Taylor G. Person-based information for acute myocardial infarction (AMI) from 1992/93 Canadian hospital discharge data with projections to the year 2016. Unpublished manuscript.

4. Intercontinental Medical Statistics Canada. Unpublished data.

5. Gillum RF, Fortmann SP, Prineus RJ. International diagnosis criteria of acute myocardial infarction and stroke. *Am Heart J.* 1984; 108:150-158.

6. Reimer KA, Lowe JE,Rasmussen MM, Jennings RB. The wave-front phenomenon of ischemic cell death: myocardial infarct size versus duration of coronary occlusion in dogs. *Circulation* 1977; 56:786-794.

7. Gruppo Italiano per lo studio della Streptokinase in Nell infarcto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986; 1:397-402.

8. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected acute myocardial infarction. *Lancet* 1988; 2:349-360.

9. The GUSTO Investigators. An International randomized trial comparing four thrombolytic strategies for acute myocardial infarction. *N Engl J Med* 1993; 329:672-682.

10. Ryan TJ. Management of acute myocardial infarction. *Postgraduate Medicine* 1997; 102(5):84-96.

11. Fibrinolytic Therapy Trialist's (FTT) Collaborative Group: Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1000 patients. *Lancet* 1994; 343(8893):311-22.

12. The International Study Group. In-hospital mortality and clinical course of 20,891 patients with suspected acute myocardial infarction randomized between alteplase and streptokinase with or without heparin. *Lancet* 1990; 336:71-75.

13. ISIS-3 Collaborative Group. ISIS-3: a randomized comparison of streptokinase vs. tissue plasminogen activator vs. antistreplase and of aspirin plus heparin vs. aspirin alone among 41299 cases of suspected acute myocardial infarction. *Lancet* 1992; 339:753-770.

14. PRIMI Trial Study Group. Randomized double-blind trial of recombinant pro-urokinase against streptokinase in acute myocardial infarction. *Lancet* 1989; 1:863-868.

15. Neuhaus KL, Tebbe U, GotttwikM, et al. Intravenous recombinant tissue plasminogen activator (rtPA) and urokinase in acute myocardial infarction: results of the German Activator Urokinase Study (GAUS). *J Am Coll Cardiol* 1988; 12:581-587.

16. Weaver WD, Cerqueira M, Hallstrom AP, et al. Prehospital-initiated vs. hospital-initiated thrombolytic therapy. *JAMA* 1993; 270:1211-1216.

17. Koren G, Weiss AT, Hasin Y, et al. Prevention of myocardial damage in acute myocardial ischemia by early treatment with intravenous streptokinase. *N Eng J Med* 1985; 313:1384-1389.

18. Mathey DG, Sheehan FH, Schofer J, et al. Time from onset of symptoms to thrombolytic therapy: a major determinant of myocardial salvage in patients with acute transmural infarction. *J Am Coll Cardiol* 1985; 6:518-525.

19. Simoons ML, Serruys PW, Van den Brand M, et al. Early thrombolysis in acute myocardial Infarction: limitation of infarct size and improved survival. *J Am Coll Cardiol* 1986; 7:717-728.

20. Lehmann KG, Doria RJ, Feuer JM, et al. Paradoxical increase in re-stenosis rate with chronic heparin use : final results of a randomized trial. *J Am Coll Cardiol* 1991; 17:181A.

21. Califf RM, Newby KL. How much do we gain by reducing time to reperfusion therapy? *Am J Cardiol* 1996; 78(suppl 12A):8-15.

22. Herrick JB. Clinical features of sudden obstruction of the coronary arteries. *JAMA* 1912; 59: 2015-2020.

23. Cairns JA, Kennedy JW, Fuster V. Coronary Thrombolysis. *Chest* 1998; 114:634S-657S.

24. Rukholm E. Editorial. CJCN 1992(2):2.

25. Burnett RE, Blumenthal JA, Mark DB, et al. Distinguishing between early and late responders to symptoms of acute myocardial infarction. *Am J Cardiol* 1995; 75:1019-1022.

26. Grines CL, DeMaria AN. Optimal utilization of thrombolytic therapy for acute myocardial infarction: concepts and controversies. *J Am Coll Cardiol* 1990; 16:223-231.

27. Cooper RS, Simmons B, Castaner A, et al, Survival rates sand pre-hospital delay during myocardial infarction among black persons. *Am J Cardiol* 1986; 57:208-11

28. Dracup K, McKinley SM, Moser DK. Australian patients' delay in response to heart attack symptoms. *Med J Aust* 1997; 166(5):233-236.

29. Leitch JW, Birbara T, Freedman B et al. Factors influencing the time from onset of chest pain to arrival at hospital. *Med J Aust* 1989; 150(1):6-10.

30. Schroeder JS, Lamb IH, Hu M. The prehospital course of patients with chest pain: analysis of the prodromal, symptomatic, decision-making, transportation and emergency room periods. *Am J Med* 1978; 64:742-748.

31. Ell K, Haywood LJ, Sobel E, et al. Acute chest pain in African Americans: factors in the delay in seeking emergency care. *Am J Pub Hlth* 1994; 84:965-970.

32. Wielgosz AT, Nolan RP, Earp JA, et al. Reasons for patients; delay in response to symptoms of acute myocardial infarction. *Can Med Assoc J* 1988; 139:853-857.

33. Ho MT, Eisenberg S, Litwin PE, et al. Delay between onset of chest pain and seeking medical care: the effect of public education. *Ann Emerg Med* 1989; 18:727-731.

34. Safer MA, Tharps Q, Jackson TC, et al, Determinants of three stags of delay in seeking care at a medical clinic. Med Care 1979;17:11-29.

35. Gillum RF, Feinleib M, Margolis JR, et al. Delay in the prehospital phase of acute myocardial infarction; lack of influence on incidence of sudden death. *Archives of Internal Medicine* 1976; 136(6):649-654.

36. Alonzo AA. Acute illness behavior: a conceptual exploration and specification. *Soc Sci Med* 1980; 14A:515-526.

37. Alonzo AA. The impact of the family and lay others on care-seeking during life-threatening episodes of suspected coronary artery disease. *Soc Sci Med* 1986; 22:1297-1311.

38. Moss AJ, Wynar B, Goldstein S. Delay in hospitalization during the acute coronary period. *Am J Cardiol* 1969; 24:659-65.

39. Kereiakes DJ, Weaver WD, Anderson JJ, et al. Time Delays in the diagnosis and treatment of acute myocardial infarction: a tale of eight cities; Report from the Pre-hospital Study Group and the Cincinnati Heart Project. *Am Heart J* 1990; 120:773-780.

40. Moses HW, Bartolozzi JJ Jr, Koester DL, et al. Reducing delay in the emergency room in administration of thrombolytic therapy for myocardial infarction associated with ST elevation. *Am J Cardiol* 1991; 68:251-253.

41. Meischke H, Eisenberg MS, Larsen MP. Prehospital delay interval for patients who use emergency medical services: The effect of heart-related medical condictions and demographic variables. Ann Emerg Med 1993; 22:1597-1601.

42. Dracup K, Moser D, Treatment seeking delay behavior among those with symptoms and signs of acute myocardial infarction. *Heart Lung* 1991; 20:570-575

43. GISSI-Avoidable Delay Study Group. Epidemiology of avoidable delay in the care of patients with acute myocardial infarction in Italy. *Arch Intern Med* 1995; 155:1481-1488.

44. Herlitz J, Elmfeldt D, Hjalmarson A, et al. Effects of metoprolol on indirect signs of the size and severity of acute myocardial infarction. *Am J Cardiol* 1983; 51:1282-8.

45. Dracup K, Moser DK, Eisenberg M, et al. Causes of delay in seeking treatment for heart attack symptoms. *Soc Sci Med* 1995; 40(3):379-392.

46. Reilly A, Dracup K, Dattolo J. Factors influencing pre-hospital delay in patients experiencing chest pain. *Am J Critical Care* 1994; 3(4):300-306.

47. Johnson JA, King KB. Influence of expectations about symptoms on delay in seeking treatment during myocardial infarction. *Am J Critical Care* 1995; 4(1):29-35.

48. Maynard C, Weaver WD. Streamlining the triage system for acute myocardial infarction. *Cardiology Clinics* 1995; 13(3):311-320.

49. Brophy JM, Diodati JG, Bogaty P, et al. The delay to thrombolysis: an analysis of hospital and patient characteristics: Quebec acute coronary care working group. *CMAJ* 1998; 158(4):475-480.

50. Kenyon LW, Ketterer MW, Gheorghiade M, et al. Psychological factors related to prehospital delay during acute myocardial infarction. *Circulation* 1991; 84:1969-1976.

51. Goldberg RJ, Gurwitz J, Yarzebski J, et al. Patient delay and receipt of thrombolytic therapy among patients with acute myocardial infarction from a community-wide perspective. *Am J Cardio* 1992; 70(4):421-425.

52. Turi ZG, Stone PH, Muller JE, et al. Implication for acute intervention related to time of hospital arrival in acute myocardial infarction. *Am J Cardio* 1986; 58:203-209.

53. Schmidt SB, Borsch MA. The prehospital phase of acute myocardial infarction in the era of thrombolysis. Am J Cardiol. 1990; 65:1411-1415.

54. Newby LK, Rutsch WR, Califf RM, et al. Time from symptom onset to treatment and outcome after thrombolytic therapy. *J Am Coll Cardiol* 1996; 27:1646-55.

55. Maynard C, Althouse R, Olsufka M, et al. Early versus late hospital arrival for acute myocardial infarction in the Western Washington thrombolytic therapy trials. *Am J Cardiol* 1989; 63:1296-300.

56. Cunningham MA, Lee TH, Cook EF, et al. The effect of gender on the probability of myocardial infarction among emergency department patients with acute chest pain: a report from the Multicenter Chest Pain Study Group. *J Gen Intern Med.* 1989; 4:392-398.

57. Hackett TP, Cassem NH. Factors contributing to delay in responding to the signs and symptoms of acute myocardial infarction. *Am J Cardiol.* 1969; 24:651-658.

58. Karlson BW, Herlitz J, Sjolin M, et al, Clinical factors associated with delay time in suspected acute myocardial infarction. *Am Heart J* 1990; 120:1213-1215.

59. Herlitz J, Hartford M, Blohm M, et al. Effect of a media campaign on delay times and ambulance use in suspected acute myocardial infarction. Am J Cardiol 1989; 64:90-93.

60. Lee HO. Typical and atypical clinical signs and symptoms of myocardial infarction and delayed seeking of professional care among blacks. *Am J Critical Care 1997;* 6(1):7-13.

61. Clark LT, Bellam SV, Shah AH, et al. Analysis of prehospital delay among inner-city patients with symptoms of myocardial infarction: implications for therapeutic intervention. *J Natl Med Assoc.* 1992; 84:931-937.

62. Lawrence RE, Littler WA. Acute myocardial infarction in Asians and Whites in Birmingham. *BMJ.* 1985; 290:1472.

63. Bett N, Aroney G, Thompson P. Impact of a national educational campaign to reduce patient delay in possible heart attack. *Aust J Med* 1993; 23(2):157-161.

64. Trent RJ, Rose EL, Adams JN, et al. Delay between the onset of symptoms of acute myocardial infarction and seeking medical assistance is influenced by left ventricular function at presentation. *Brit Hrt J* 1995; 73(2):125-128.

65. Hofgren K, Bondestam E, Johansson FG, et al. Initial pain course and delay to hospital admission in relation to myocardial infarct size. *Heart Lung* 1998;17(3):274-280.

66. Hartford M, Herlitz J, Karlson BW, et al. Components of delay time in suspected acute myocardial infarction with particular emphasis on patient delay. *J Int Med* 1990; 228(5):519-523.

67. Alonzo AA, Reynolds NR. The structure of emotions during acute myocardial infarction: A model of coping. *Soc Sci Med.* 1998; 46(9):1099-1110.

68. Becker MH, Drachman RH, Kirscht JP. Motivations as predictors of health behaviors. *Hlth Serv Rep.* 1972; 87:852-862.

69. Leventhal H, Cameron L. Behavioral theories and the problem of compliance. *Patient Ed. Counseling.* 10:117-138.

70. Lindesmith AR, Strauss L. *Social Psychology.* Holt, Rinehart and Winston, New York, 1968.

71. Sharpley CF, Rogers HJ. Preliminary validation of the abbreviated spanier dyadic adjustment scale: some psychometric data regarding a screening test of martial adjustment. *Educational Psychological Measurement.* 1984; 44:1045-1050.

72. Yarzebski J, Goldberg RJ, Gore JM et al. Temporal trends and factors associated with extent of delay to hospital arrival in patients with acute myocardial infarction: The Worcester Heart Attack Study. *Am Hrt J.* 1994; 128:255-263.

73. Cox JL, Lee E, Langer A, et al. Time to treatment with thrombolytic therapy: determinants and effect on short-term nonfatal outcomes of acute myocardial infarction. *CMAJ.* 1997; 156:497-505.

74. Letovsky E, Allen T. Initiating thrombolytic therapy for acute myocardial infarction: Whose job is it anyway? *CMAJ.* 1996; 154:509-511.

75. Chipkin SR, Frid D, Alpert JS, et al. Frequency of painless myocardial ischemia during exercise tolerance testing in patients with and without diabetes mellitus. *Am J Cardiol.* 1987; 59:61-65.

76. Nesto RW, Phillips RT. Asymptomatic myocardial ischemia in diabetic patients. *Am J Med.* 1988; 80:40-47.

77. Heriot AG, Brecker SJ, Coltart DJ. Delay in presentation after myocardial infarction. *J Roy Soc Med.* 1993; 86:642-644.

78. Yu PN. Prehospital care of acute myocardial infarction. *Circulation.* 1972; 45:189-214.

79. Rawles JM, Haites NE. Patient and general practitioner delays in acute myocardial infarction. *BMJ.* 1988; 296:882-884.

80. Matthews KA, Siegel JM, Kuller LH et al. Determinants of decisions to seek medical treatment by patients with acute myocardial infarction symptoms. J Pers Soc Psych. 1983; 44:1144-1156.

81. Ridker PM, Manson JE, Goldhaber SZ et al. Comparison of delay times to hospital presentation for physicians and nonphysicians with acute myocardial infarction. *Am J Cardiol.* 1992; 70:10-13.

Appendix A. Consent Form

Appendix A

HÔPITAL GÉNÉRAL D'OTTAWA OTTAWA GENERAL HOSPITAL

Dear Sir/Madam,

We are currently conducting a research study on factors associated with delay in response to symptoms of a heart attack. Our hospital records indicate that you are eligible to participate in this study and we would like to invite you to participate. Involvement in this study is completely voluntary and involves a 30 minute questionnaire about your health and other information concerning the events leading up to your arrival at the Ottawa General Hospital for treatment of your heart attack. This questionnaire will be administered by the interviewer.

In order to ensure we do not intrude on your privacy, you may want to reschedule the time or the location for this interview at your convenience. Participating in this interview will not cause any harm or discomfort, and you have the right to terminate the interview at any point should you wish to do so. You may also skip over any questions that you feel uncomfortable in providing information about.

The information collected from the interview will be kept strictly confidential. The principal investigator of this study will see the information pertaining to you. All data will be presented as an aggregate sample. You will not be identified by name in any publication or scientific literature. Participation in this study does not require any further obligations.

Results of this study may suggest new directions for educating cardiac patients about responding to their symptoms. Thank you very much for your kind attention, and please address any further questions to the interviewer or the principal investigator, Roderick Chew, at (613)-241-1420.

Yours sincerely,

Roderick Chew B. Sc.
Principal Investigator
Department of Epidemiology
University of Ottawa

A. T. Wielgosz, MD, Ph.D., FRCPC
Principal Supervisor
Division of Cardiology
Ottawa General Hospital

501 Smyth Ottawa Ontario K1H 8L6 (613) 737 7777

Monsieur/Madame,

Nous menons présentement une étude de recherche sur les facteurs reliés au délai dans la réaction éprouvée par rapport aux symptômes d'une crise cardiaque. Les dossiers de l'hôpital indiquent que vous êtes éligible pour participer à cette étude, et nous vous invitons à y participer. Votre participation à cette étude est entièrement volontaire et demande que vous répondiez à un questionnaire d'une durée de 30 minutes au sujet de votre santé et d'autres informations concernant les évènements qui vous ont mené à votre arrivée à l'Hôpital Général d'Ottawa pour le traitement de votre crise cardiaque. Ce questionnaire sera dirigé par l'enquêteur.

Afin d'assurer que nous ne nous ingérons pas dans votre vie privée, vous pouvez demander que le temps alloué pour cette entrevue ou le site soit choisi afin de répondre à vos besoins. Votre participation à cette entrevue ne vous causera aucun malaise et ne vous sera pas nuisible, et vous avez le droit de mettre fin à l'entrevue à n'importe quel moment si vous le désirez. Vous pouvez aussi passer toutes questions dont vous n'êtes pas à l'aise à y répondre.

L'information recueillie pendant l'entrevue demeurera strictement confidentielle et seulement l'enquêteur principal de cette étude verra l'information pertinente à vous. Toute information sera présentée comme un échantillon collectif, et votre nom n'apparaîtra pas dans aucune publication ou littérature scientifique. Votre participation à cette étude ne nécessite aucune autre obligation.

Les résultats de cette étude pourraient suggérer des nouvelles directions pour l'éducation des patients cardiaques au sujet de la réaction à leurs symptômes. Nous vous remercions de votre attention et nous vous prions d'adresser toutes questions à l'enquêteur, Roderick Chew, au (613) 241-1420.

Sincèrement vôtre,

Roderick Chew B. Sc.
Enquêteur principal
Département d'épidémiologie
Université d'Ottawa

A. T. Wielgosz, MD, Ph.D., FRCPC
Surveillant principal
Division de cardiologie
Hôpital Général d'Ottawa

501 Smyth Ottawa Ontario K1H 8L6 (613) 737-7777

HÔPITAL GÉNÉRAL D'OTTAWA
OTTAWA GENERAL HOSPITAL
501 Smyth, Ottawa, Ontario K1H 8L6

CONSENTEMENT DU PATIENT/ PROCURATION À DES RECHERCHES MÉDICALES

PATIENT/PROXY CONSENT FOR MEDICAL RESEARCH

Nom de l'étude

Name of research project

Factors Associated with Delay in Reporting the Symptom

Je confirme que j'ai expliqué la nature ainsi que les complications connues de ce projet de recherche/procuration du patient.

I confirm that I have explained the nature of and known complications of the research project to the patient/proxy.

Roderick Chen

(chercheur) (researcher)

Je consens à la participation de : Nom du patient	Nº de projet	I consent to the participation of : Patient's name	Research no.

à l'étude précitée et j'autorise par la présente le(s) médecin(s) et le(s) chercheurs(s) à procéder aux examens et/ou à dispenser les traitements suivants :

in the above study and I hereby authorize the physician(s) and investigator(s) to proceed with the following questionnaire, examinations and/or to administer these treatments:

J'ai lu la feuille d'explication détaillée approuvée par le Conseil d'éthique en recherches et je suis au courant des effets secondaires et des risques connus ayant trait aux examens et aux traitements.

I have read the detailed information sheet approved by the Research Ethics Board and am aware of the known side effects and risks related to the examinations and treatments.

J'ai également reçu une description de tous les avantages à attendre de ces examens et de ces traitements. On m'a fait connaître d'autres formes d'examen et de traitement.

I have also received a description of any benefits that may be expected from these examinations and treatments. As well, other forms of treatment and exams have been disclosed to me.

J'ai eu l'occasion de poser des questions au sujet de ces examens et de ces traitements et on y a bien répondu.

I have been given an opportunity to ask questions concerning the examinations and treatments involved and the questions which I have asked have been adequately answered.

On m'a dit que je pouvais retirer le consentement et suspendre la participation à l'étude à n'importe quel moment et pour quelque motif que ce soit et que cette action n'affectera pas la qualité des soins en cours et futurs.

I have been told that I can withdraw consent and stop participation in the study at any time and for any reason, and that such action will not affect the quality of ongoing and future care.

Je reconnais qu'en agissant, comme procurateur, ce sera dans le meilleur intérêt du patient.

I acknowledge that if I am acting as a proxy that I am acting in the best interest of the patient.

En toute connaissance de cause, je consens volontairement à ce que _____ participe à cette étude.

With full knowledge of this, I voluntarily consent to the participation of Subject in the study.

Ce protocole a été approuvé par le Conseil d'éthique en recherches de l'Hôpital général d'Ottawa. Ce conseil étudie les aspects éthiques de tous les projets de recherche sur les humains. Si je le désire, je peux contacter le président de ce comité.

This protocol has been approved by the Research Ethics Board of the Ottawa General Hospital. This Board considers the ethical aspects of all hospital research projects using human subjects. If I wish, I may contact the Chairperson of the Board with questions.

Nom (lettres moulées) - Name (print)	Signature	Lien de parenté - Relationship

PATIENT OU PERSONNE LÉGALEMENT RESPONSABLE (PROCURATION) - PATIENT OR PERSON LEGALLY RESPONSIBLE(PROXY)

Nom du témoin (lettres moulées) - Name of witness (print)	Signature	Date (j/d-m/m-a/y)

1911 ▸ (10/95) DOSSIER-CHART

Appendix B. *Fastrak* Date Entry Form

FASTRAK

Hospital ID# ___ ___ ___ ___ ___
Optional Field ___ ___ ___ ___ ___
Patient Initials ___ ___ ___ (First Middle Last)
Sex ○ M ○ F
Birth Date __/__/__ (M D Y)
Weight ___ ○ lb ○ kg

Demographics/Medical History

(check all that apply)

- ○ Previous MI
- ○ Angina
- ○ Diabetes
- ○ Current Smoker
- ○ CABG
- ○ CHF
- ○ Stroke
- ○ PTCA
- ○ Hypertension
- ○ Hypercholesterolemia

Presentation Profile

First Recorded
Use ___ ___ ___
? ___/___ (S D)

Admission Diagnosis
○ MI ○ Unstable Angina
○ R/O MI ○ Other ___

Clinical Presentation

- ○ No CHF
- ○ Rales
- ○ Pulmonary Edema
- ○ Hypotension
- ○ Cardiogenic Shock
- ○ Rx Dysrhythmia

Interhospital Transfer

Was patient transferred into this hospital ○ Yes ○ No
Referring Hospital ID# ___ ___ ___ ___ ___
If no ID#, Hospital Name ___

First ECG Results (check all that apply):

- ○ ST elevation ≥ 1mm
  ___ # of affected leads
- ○ ST depression
- ○ RBBB
- ○ LBBB
- ○ Nonspecific ST/T wave changes
- ○ Normal ECG
- ○ Other ___

If initial ECG nondiagnostic, MI diagnosis based on following (check all that apply):

- ○ Subsequent diagnostic ECG
- ○ Echocardiography
- ○ Coronary angiography
- ○ Cardiac enzymes
- ○ Other ___
- ○ None, patient transferred out

Timeline

Event	Date (M D Y)	Time (24-hr clock)
MI Sx onset	__/__/__	__:__
First hospital arrival	__/__/__	__:__
This hospital arrival	__/__/__	__:__
First ECG	__/__/__	__:__
First diagnostic ECG	__/__/__	__:__
IV lytic ordered	__/__/__	__:__
IV lytic started	__/__/__	__:__
First coronary angiogram	__/__/__	__:__
1° PTCA started	__/__/__	__:__
CABG started	__/__/__	__:__
Discharged/death/ transferred out	__/__/__	__:__

Initial Reperfusion Strategy

Was IV thrombolysis initial reperfusion strategy ○ Yes ○ No

If Yes

Drug ○ tPA ○ SK
Weight adjusted ○ Yes ○ No
Ordered by (check one):
○ Emergency MD ○ Cardiologist ○ Internist
Consult obtained prior to initiation of thrombolytic ○ Yes ○ No
First 3 PTTs therapeutic ○ Yes ○ No

Location
○ This hospital
○ Prehospital
○ Referring hospital

Site
○ ED ○ Cath lab
○ ICU/CCU ○ Other

If tPA complete the following:
Bolus dose ___ ___ ___ . ___ mgs.
Total dose ___ ___ ___ . ___ mgs.
Total dose duration ___ ___ ___ min.

If No

Reason(s) IV thrombolysis not utilized:

- ○ Too late • ___ ___ hours from symptom onset
- ○ Nondiagnostic ECG
- ○ Reason not documented
- ○ Contraindication (specify) ___
- ○ Other (specify) ___

Check alternative initial reperfusion strategy(ies):

- ○ Primary PTCA
- ○ IC thrombolysis • Drug: ___
  Dose: ___
- ○ Immediate CABG
- ○ No initial reperfusion strategy

Concomitant Medications Within 24 Hours of MI Diagnosis (check all that apply)

- ○ IV heparin
- ○ SQ heparin
- ○ ASA
- ○ IV NTG
- ○ IV beta blocker
- ○ Oral beta blocker
- ○ Lidocaine
- ○ IV magnesium
- ○ ACE inhibitor
- ○ Calcium blocker
- ○ Other antithrombin
- ○ Other antiplatelet

Other Procedures Performed at This Hospital Prior to Discharge (check all that apply)

- ○ Coronary angiography
- ○ Atherectomy/stent/laser
- ○ CABG (exclude immediate)
- ○ IABP
- ○ Repeat dose IV thrombolytic
- ○ PTCA (exclude 1° PTCA)
- ○ Rescue PTCA for failed thrombolysis
- ○ Pacemaker
- ○ Stress test

Clinical Events

Check the appropriate box(es) for clinical events at this hospital. If deceased, indicate cause(s) of death.

Event	Cause of Death
○ Allergic/anaphylactic reaction	○
○ Hypotension requiring intervention	○
○ Recurrent ischemia/angina	○
○ Recurrent MI (ECG+/CK-MB+)	○
○ CHF requiring drug therapy	○
○ Pericarditis	○
○ Rupture/EMD	○
○ Cardiogenic shock	○
○ Sustained V-tach/V-fib	○
○ Unexpected cardiac arrest	○
○ Other major event (specify) ___	○

Stroke/ICB

Onset of Neurological Symptoms

Date __/__/__ (M D Y) Time ___ ___ 24 hr clock

Type of Stroke (check one):

- ○ Hemorrhagic
- ○ Thromboembolic
- ○ Thromboembolic with hemorrhagic conversion
- ○ Unknown
- ○ Other (specify) ___

Outcomes Attributed to Stroke/ICB (check all that apply)

- ○ Required procedure/ surgery
- ○ Residual deficit at discharge
- ○ Death
- ○ Prolonged hospitalization
- ○ Life-threatening

CT scan or MRI obtained ○ Yes ○ No

If yes:

Date __/__/__ (M D Y) Time ___ ___ 24 hr clock

Major Bleeding

Episodes other than stroke/ICB where bleeding resulted in substantial hemodynamic compromise requiring treatment or transfusion

Specify Site(s)	Date (M D Y)	Procedure/Surgery? Yes	No	Prolonged Hospitalization? Yes	No	Resulted in Death? Yes	No
___	__/__/__	○	○	○	○	○	○
___	__/__/__	○	○	○	○	○	○

Outcome Status

MI Location/Type

- ○ Inferior
- ○ Anterior
- ○ Anterior/septal
- ○ Lateral
- ○ RV involvement
- ○ Posterior
- ○ Q wave
- ○ Non-Q wave

Total days in ICU/CCU bed (this hospital) ___ ___ ___
Peak CPK ___ IU

Complete one of the following dispositions:

- ○ **Transferred** (acute care facility)
  Receiving hospital ID# ___
  If no ID#, name ___
  Reason transferred
  - ○ Additional treatment/intervention
  - ○ Patient request
  - ○ Other
- ○ **Discharged**
  - ○ To home ○ LTC ○ AMA

  Discharge meds.
  - ○ ASA
  - ○ Beta blocker
  - ○ ACE inhibitor
  - ○ Calcium blocker
  - ○ Diuretic
  - ○ Digoxin
  - ○ Warfarin
  - ○ Nitrates
- ○ **Deceased**
  Autopsy ○ Yes ○ No

Form Completed By:

Print ___
Signature ___
Date __/__/__ (M D Y)

Appendix C. Patient Interview Questionnaire

Appendix C

Patient Questionnaire

Last Name _____ First Name _____ Hospital Chart No. _____

1. DOB (dd/mm/yy) ____/____/____ 2. Age _____ 3. Sex: M / F

4. Marital Status: Single / Married / Separated / Divorced / Widowed

5. Were you born in Canada? Yes / No If No, 6. how long have you lived in Canada? ____ yrs

7. What is the highest level of education you have completed?

   1) No Schooling
   2) Elementary
   3) Secondary
   4) College
   5) Undergraduate university
   6) Graduate or Professional Programs

8. Are you currently employed? 1. Full-time 2. Part-time 3. Temporary 4. Unemployed

9. What is your current occupation? _____

10. How long have you held this job? _____ mths

11. Do you hold more than one job? Yes / No 12. How many? ____

13. How long? _____ mths

14. Do you work at home? Yes / No 15. Do you work outside? Yes / No

16. Do you consider your job stressful?
   1. Very stressful
   2. Somewhat stressful
   3. About average
   4. Mildly stressful
   5. Not stressful at all

17. Have you smoked more than a 100 cigarettes in your lifetime? Yes / No

If Yes, 18. Do you smoke cigarettes now? Yes / No

If No, 19. How long ago did you stop? ____ wks _____ mths _____ yrs

20. Do you have hypertension? Yes / No / Don't know

21. Do you suffer from arrhythmia? Yes / No / Don't know

22. Do you suffer from arteriosclerosis? Yes / No / Don't know

23. Do you have angina? Yes / No / Don't know

24. Have you ever had a heart attack? Yes / No 25.When was the last attack? ___ yrs/mths ago

26. Have you ever seen a heart specialist before this visit to the hospital? Yes / No

If yes, 27. Was it for chest pain? Yes / No

28. Do you see the cardiologist regularly? Yes / No

29. Have you ever had a cardiac catheterization? Yes / No 30. How many? ____

31. Have you ever had a coronary artery bypass operation? Yes / No 32. How many? ____

33. How many years ago was the last one? _____ yrs ago

34. Have any of your close relatives (mother, father, brothers, sisters) ever had a heart attack?

1 = none 2 = at least one 35. How many of them? _____

36. Do you think heart disease runs in your family? Yes / No / Don't know

37. When did the pain first begin? ___/___/___ _____ _____am/pm
dd mth yr day of week time

38. Where were you exactly at the time that you felt the first pain or symptom?

1. Home
2. Office
3. Others _____

39. What were you doing when you felt the pain? _____

40. How long did the pain last? _____mins _____hrs

41. Did you first think the pain was due to a heart problem? Yes / No

42. Did you decide to go seek medical help with this initial symptom? Yes / No

43. Was this pain: 1. Steady 2. Intermittent 3. One sharp pain

44. When did you decide to seek medical help?

___/____/____ _____ _____am/pm
dd mth yr day of week time

45. When you decide to seek medical help, did the pain recur? Yes / No

46. Where were you when you decide to seek medical help?

1. Home
2. Office
3. Other _____

47. Did you call your family physician about your symptoms/pain? Yes / No

48. Did you call for an ambulance? Yes / No

49. What mode of transportation was used?
1. Self Driving
2. Ambulance
3. Cab
4. Other Public Transport
5. Others _____

50. Before you came to the hospital, did you talk to anyone about the pain or symptoms you were feeling?

1. No one
2. Only one person
3. More than one person

51. Who was the first person you told you were having pain and weren't feeling well?

1. Spouse
2. Child
3. Business colleague / Coworker
4. Medical personnel
5. Others _____

52. Did this person act like they thought the symptoms was/were serious? Yes / No

53. Did he/she suggest the symptoms may be due to heart problem? Yes / No

54. Did he/she suggest you to seek medical assistance? Yes / No

55. Did you take any of the suggestions people gave you? Yes / No

56. Who first suggested that you should go to the hospital? 1. Self
2. Spouse
3. Other relative
4. Friend
5. Others _____

57. Who would you say actually decided that you should go to the hospital?

1. Self
2. Spouse
3. Other relative
4. Friend
5. Others _____

58. Did you have to make any arrangements, like arranging child care, things at work, so you could come to the hospital? Yes / No

59. Did anyone come with you to the hospital? Yes / No

60. How long did it take from the time you left for the hospital to the time you actually got there?

_____ minutes/hours Don't know

The next few questions are about things you did when you started to feel the pain or other symptoms.

Did you	a lot	some	not at all
61. Think about what the symptoms meant?	1	2	3
62. Think about how serious they were?	1	2	3
63. Try to ignore the symptoms?	1	2	3
64. Think about other people who had had symptoms like yours?	1	2	3
65. Think about what might be causing them?	1	2	3
66. Try to relieve the symptoms by taking something?	1	2	3
67. Try lying down and resting?	1	2	3

68. Have you ever felt the type of pain you had right before coming to the hospital before?

Yes / No

If yes, 69. How long ago did you feel that type of pain? _____hrs/days/mth/yrs ago

70. Had you ever before felt any type of chest pain? Yes / No

The following questions ask you to comment on how you see your relationship with someone with whom you are coupled (e.g., spouse, long-term partner, etc.)

1. *Most persons have disagreements in their relationships. Please indicate below the approximate extent of the agreement or disagreement between you and your partner for each of the following three items.*

	ALWAYS AGREE	ALMOST ALWAYS AGREE	OCCASIONALLY DISAGREE	FREQUENTLY DISAGREE	ALMOST ALWAYS DISAGREE	DISAGREE
a) Philosophy of Life	5	4	3	2	1	0
b) Aims, goals, and things believed to be important	5	4	3	2	1	0
c) Amount of time spent together	5	4	3	2	1	0

2. *How often would you say the following events occur between you and your mate?*

	NEVER	LESS THAN ONCE A MONTH	ONCE OR TWICE A MONTH	ONCE OR TWICE A WEEK	ONCE A DAY	MORE OFTEN
a) Have a stimulating exchange of ideas	0	1	2	3	4	5
b) Calmly discuss something?	0	1	2	3	4	5
c) Work together on a project	0	1	2	3	4	5

3. *The brackets on the following line represent different degrees of happiness in your relationship. Please put an "x" in the bracket that best describes the degree of happiness, all things considered, of your relationship.*

( )	( )	( )	( )	( )	( )	( )
Extremely Unhappy	Fairly Unhappy	A little Unhappy	Happy	Very Happy	Extremely Happy	Perfect

* *Place an "x" here if you are not in a relationship at present: ( )*

Appendix D. List of Variables

List of variables used in multivariate analysis

* Variables in selection pool for model 1, 2, and 3; † Variables in selection pool for model 2 and 3.
§ Variables in selection pool for model 3 and 5 only; ¥ Variables in selection pool for model 4 and 5.

Variables	Definition	Coding
Sex *	Gender	0=male 1=female
Age * ¥	Age of patient at symptom onset	(continous variable)
Previous MI *	Previous episode of previous myocardial infarction	0=No 1=Yes
Angina *	History of angina pectoris	0=No 1=Yes
Diabetes *	History of diabetes mellitus	0=No 1=Yes
Current smoker * ¥	Patient smokes within past 6 months	0=No 1=Yes
CABG *	Previous coronary artery bypass operation	0=No 1=Yes
Congestive Heart Failure *¥	History of congestive heart failure	0=No 1=Yes
Stroke *	History of stroke	0=No 1=Yes
PTCA * ¥	Previous percutaneous transmural coronary angioplasty	0=No 1=Yes
Hypertension *	History of high blood pressure	0=No 1=Yes
Hypercholesterolemia *	History of high cholesterol	0=No 1=Yes
Arrhythmia † ¥	History of arthythmias	0=No 1=Yes 2=don't know
Arteriosclerosis †	History of arteriosclerosis	0=No 1=Yes 2=don't know
IV thrombolytic	Patient received thrombolytic therapy for this admission	0=No 1=Yes
In-hospital death	Patient did not survive to be discharged	0=No 1=Yes
Marital Status † ¥	Current martial status	0=not married 1=married

Education †	Level of formal education completed	0=≤ Secondary education 1=> Secondary education
Occupation †	Current employment status	0=not working 1=working
Born in Canada †	Patient born in Canada	0=No 1=Yes
Family history of MI †	Other family members who had myocardial infarction	0=No 1=Yes
Family History of heart disease †¥	Patient believe heart disease run in his/her family	0=No 1=Yes 2=don't know
Consult cardiologist †	Previous consultation with a cardiologist	0=No 1=Yes
Previous chest pain †¥	Previous experience of any chest pain	0=No 1=Yes
Previous same pain †	Previous experience of the same type of pain as this event	0=No 1=Yes
Onset day †	Day of week when symptom onset	0=weekday 1=weekend
Onset time †	Time of day when symptom onset	0=late night (12am-5:59am) 1=morning (6am-11:59am) 2=afternoon (12pm-5:59pm) 3=evening (6pm-11:59pm)
Location onset †¥	Location patient at when experienced onset	0=home 1=other
Location help †¥	Location patient at when decided to seek medical help	0=home 1=other
Initial heart †	Patient believe initial symptoms were due to heart problem	0=No 1=Yes
Pain recur †¥	Pain recur when patient decide to seek help	0=No 1=Yes 2=Never stopped
Pain quality †	Quality of the pain	0=One sharp pain 1=Intermittent 2=Steady
Family doctor †	Call family doctor about symptoms/pain	0=No 1=Yes
Call ambulance †¥	Patient call for an ambulance	0=No 1=Yes

Mode of transport †	Mode of transport used to travel to hospital	0=Self drive 1=Ambulanc e 2=Others
Talk to someone †	Talk to someone else about the pain/symptoms before coming to hospital	0=No 1=Yes
Person suggest heart †	Another person suggested symptoms may be cardiac in nature	0=No 1=Yes
Person serious †	Another person act like they thought symptoms were serious	0=No 1=Yes
Person suggest help †	Another person suggested to seek medical help	0=No 1=Yes
Take suggestion	Patient took suggestion of another person to seek medical help	0=No 1=Yes
Accompany hospital †	Someone came to the hospital with the patient	0=No 1=Yes
First person told †	The first person the patient told about his/her symptoms	0=spouse 1=other relatives 2=others
Suggested hospital †	The first person who suggested patient should come to hospital	0=self 1=spouse 2=other relatives 3=others
Decided hospital †	The first person who decided to come to hospital	0=self 1=spouse 2=other relatives 3=others
Symptoms †	Extent patient thought about what symptoms meant	0=not at all 1=some 2=a lot
Serious †¥	Extent patient thought how serious the symptoms were	0=not at all 1=some 2=a lot
Ignore †¥	Extent patient tried to ignore symptoms	0=not at all 1=some 2=a lot
Think others †	Extent patient thought about others with similar symptoms	0=not at all 1=some 2=a lot

Think cause †¥	Extent patient thought about the cause of symptoms	0=not at all 1=some 2=a lot
Relieve †	Extent patient tried to relieve symptoms with medication	0=not at all 1=some 2=a lot
Rest †¥	Extent patient lied down or rest after experienced symptoms	0=not at all 1=some 2=a lot
ADAS §	ADAS score for spousal relationship	(continuous)