PATHOPHYSIOLOGY AND NATURAL HISTORY
CORONARY ARTERY DISEASE

Long-term prognosis after recovery from myocardial infarction: a nine year follow-up of the Perth Coronary Register

CRAIG A. MARTIN, B.SC., M.B., B.S., PETER L. THOMPSON, M.B., B.S., F.R.A.C.P.,
BRUCE K. ARMSTRONG, M.B., B.S., D.PHIL., F.R.A.C.P.,

ABSTRACT Patients registered by the 1971 Perth Coronary Register as having suffered a myocardial infarction were followed up for 9 years. The Register was a community-based study that used standard methods and criteria as part of a World Health Organization collaborative investigation. Of the 1078 patients studied, 77% survived the first 24 hr and 62% the first 28 days; 0.3% were lost to follow-up. For the 666 patients alive at 28 days, the crude 1, 5, and 9 year survival rates were 88%, 67%, and 52%, respectively. The relationship between 54 variables and the survival of patients alive 28 days after myocardial infarction was examined by life-table methods and the log rank test, and then by fitting a proportional hazards model to the data. The important prognostic factors were age, sex, past history of myocardial infarction, stroke, diabetes and hypertension, tachycardia at presentation, hypotension at presentation, and the occurrence of arrhythmias as short-term complications. The most appropriate mathematical description of the joint effects of the prognostic factors was a multiplicative model with no interaction.


ASSESSMENT of factors that affect the prognosis of individual patients after myocardial infarction (MI) is potentially useful in patient management, design of clinical trials of therapy after MI, elucidation of the mechanisms of death after MI, and stratification to permit valid comparison of cohorts of post-MI patients. However, lack of agreement among previous studies on these prognostic factors has raised doubts that have limited their usefulness. The most important potential sources of disagreement are various kinds of bias, poor statistical power, and variations in factor definition or measurement.

Current knowledge of survival after MI is largely based on results from patients treated in the hospital, patients selected for clinical trials of therapy after MI, or patients identified in health insurance studies. These groups have been particularly liable to selection bias and poor standardization of diagnostic criteria.

This article reports the 9 year survival of 666 patients registered in the 1971 Perth Coronary Register (PCR) who survived the acute phase of MI. The PCR was a community-based study that used standard methods and diagnostic criteria as part of a World Health Organization (WHO) collaborative investigation. Evidence from several sources suggested a fairly complete ascertainment of cases of MI. Furthermore, the stability of Perth’s population due to its relatively isolated location provided an opportunity for near-total follow-up of patients registered.

Methods

The PCR was part of a WHO collaborative investigation of the incidence of MI in 19 centers in Europe, Israel, and Australia. By use of a common protocol, an attempt was made to register every MI occurring between October 1, 1970, and September 30, 1971, in people 30 to 69 years old. The PCR operated in the Perth metropolitan area, which covered about 100 square miles, contained approximately 600,000 people, and had four hospital-based and two mobile coronary care units.

The PCR study population was 239,546 people 30 to 69 years old, among whom 1375 possibly relevant events were registered; of these 1138 were eventually classified as definite or possible MI. These in turn occurred among 1078 patients having one or more MIs. Each of these 1078 was followed from the date of earliest registered MI up to December 31, 1979, at which
time there were 719 dead, 336 alive, 20 who had moved from the study area, and three who could not be located. The death certificate, together with autopsy findings and medical records when available, was reviewed for each death. Fatal events consistent with either of the WHO diagnostic categories of definite or possible MI were defined for this study as ischemic heart disease (IHD) deaths.

**Analysis of prognostic factors.** Univariate analyses of the survival of persons alive at 28 days were performed by the Kaplan-Meier method, and the effects on survival of the 54 variables available in the PCR were examined by use of the log rank test. Multivariate analyses were performed with the most important variables on biological grounds or those identified in univariate analyses or past studies. The variables fitted were age, sex, smoking habits at MI, and Quetelet’s index (wt/ht²); history of previous MI, angina, stroke, diabetes, hypertension, or claudication; systolic blood pressure, heart rate (tachycardia, bradycardia), cardiac failure, or shock at presentation; and cardiac failure, arrhythmia, shock, or cardiac arrest as a short-term complication.

Missing values among the 19 variables originally selected for multivariate analyses led to the exclusion of 59 of the 666 patients alive 28 days after MI. To assess the possibility of appreciable bias as a result of these exclusions, the excluded and nonexcluded groups were compared by examining the values of the 19 variables that were present among the excluded cases. Seventeen of the 19 variables, as well as the survival times and death rates, were not significantly different between the two groups. The variables that differed significantly were history of hypertension (χ² = 4.7) and heart rate at presentation (χ² = 4.6; p < .05 in each case).

The censored survival data were analyzed with a proportional hazards model (see Kalbfleisch and Prentice for a full explanation and derivation):

\[ \lambda(t;z) = \lambda_0(t) \exp(z \beta) \]

where λ(t;z) is the hazard function at given time t and covariate vector z; \( \lambda_0(t) \) is an arbitrary unspecified baseline hazard function, z is a row vector of the measured covariates of any type, and \( \beta \) is the column vector of the corresponding regression parameters.

The maximum likelihood estimates of \( \beta \) were calculated with the computer program "RISK." Analyses were performed for both of the outcomes: all causes of mortality and IHD mortality. Each variable was categorized and each category was treated as a separate variable. The proportional increase in the risk of dying with increasing age was linear for 10 year age groups and thus age was subsequently entered as a continuous variable. A forward-stepping procedure was used and covariates were added until the p value for inclusion was greater than .10. At this point the β value for each covariate included was estimated, adjusting for all other covariates included. The relative risks were then calculated with the following relationship: relative risk = \( e^{\beta t} \). Correlation coefficients (Kendall’s tau) were calculated for variables significant (p < .05) by either univariate or multivariate analyses to elucidate confounding effects between variables.

**Testing of assumptions.** The model was used with the assumptions that covariates did not interact, that their effects did not change with time, and that they acted multiplicatively on each other rather than additively. To test these assumptions and perhaps provide a more accurate description of the survival process, three extra models were fitted: one including all possible two-way interactions between the covariates considered for multivariate analysis, one with linear and quadratic time interactions for each variable whose main effect was significant (p < .05), and one in which the risks acted additively rather than multiplicatively. To avoid collinearity between the fitted variables when using interaction terms for continuous (or ordered categorical) variables, the means were subtracted from the variables before multiplication.

**Validation.** The goodness of fit of the model for all deaths to other data was assessed by randomly splitting the total sample into four groups and successively comparing the survival of each quarter with that predicted from the survival function estimated by the other three quarters. This comparison was tested with the limiting distribution for large sample size of the truncated Kolmgorov-Smirnov statistic [VN(\( D_n(0) \)] as described by Elandt-Johnson and Johnson.

**Prediction.** For the model finally adopted in this study, the probability of survival to time t was given by the relationship

\[ f(t) = R_1 \times R_2 \times \ldots \times R_n \]

where f(t) was the empirical baseline survival function and \( R_i \) to \( R_n \) were the relative risks for each covariate used. To derive a predictive equation, a curve was fitted to the baseline survival function for all causes of death with the method of least squares.

To examine the distribution of prognostic factors among the patients alive 28 days after MI according to their level of risk and their survival according to level of risk, the patients were stratified into quintiles of risk of death with the above model and their survival was analyzed by the Kaplan-Meier method.

**Results**

**Survival after MI.** Of the 1078 patients studied, 250 (23%) died in the first 24 hr and 412 (38%) died in the first 28 days after MI, leaving 666 survivors of the acute phase MI. For these 666, 12.3% died by the end of the first year after MI, and subsequently the annual death rate was 7.7% in the second year, 6.4% in each of years 3 to 5, and 5.7% in each of years 6 to 9. This may be compared with an annual death rate of 1.3% for Western Australians of the same age and sex in the same period. The age and sex distribution of the 28 day survivors is shown in table 1.

The survival rate of patients alive at 28 days is shown in figure 1 to the end of the follow-up period (approximately 9 years) by the lower curve. The upper curve shows their expected survival rate adjusted for age and sex, based on mortality rates for all Western Australians over the same period.

**Prognostic factors**

**Univariate analyses.** Patients alive at 28 days after MI were stratified on values of each of the 54 variables studied, and survival curves for each of the strata were compared by the log rank test. An example of this stratification is shown in figure 2 for history of myocardial infarction (lower curve) is much worse than the survival rate of those without such a history (upper curve).

Table 2 shows all variables that had a statistically significant effect on survival or were of particular in-
terest. It shows for each variable the highest risk stratum, the lowest risk stratum, the ratio of deaths divided by the total exposure to risk in the highest risk stratum to that in the lowest risk stratum (relative risk), and the p value from the log rank test for the difference between all strata.

It can be seen that age increased mortality 2.9 times for the oldest compared with the youngest stratum and that sex, occupation, and country of birth were not statistically significant (p < .05) in their effect on survival. Histories of hypertension, angina, diabetes, MI, and stroke were statistically significant and increased mortality between 1.6 and 3.4 times. Cardiac failure at presentation and sinus tachycardia (of > 104 beats/min) at presentation were both significant and increased mortality 1.6 and 1.8 times, respectively. Of the complications in the first 28 days after infarction, the occurrence of an arrhythmia, shock in the first 24 hr, and cardiac failure were significant and increased mortality between 1.4 and 1.5 times.

Multivariate analyses. The results of fitting the Cox-Breslow proportional hazards model are shown in table 3 for all causes of death and IHD deaths. Among the 607 patients in this analysis there were 276 deaths, of which 208 (75%) were caused by IHD.

For both outcomes the same nine covariates (p < .10) were included in the survival function, although the order of inclusion differed after the first three covariates. The relative risks for both outcomes were also very similar, although they tended to be a little higher for IHD deaths. Apart from age and sex, the important covariates were history of MI, stroke, diabetes, and hypertension, presenting tachycardia (at >104 beats/min), presenting hypotension (systolic blood pressure of <115 mm Hg), and the occurrence of arrhythmias as short-term complications. There were too few cases to permit each type of arrhythmia to be meaningfully entered as a separate covariate.

Confounding effects. Comparison of tables 2 and 3 showed that the relative risks were very similar overall for both the univariate and multivariate analyses.

The only covariates to be significant in the multivariate analyses that were not significant in the univariate analyses were sex and presenting systolic hypotension. Conversely, history of angina, cardiac failure at presentation and as a short-term complication, and shock in the first 24 hr were significant modifiers of survival when considered alone, but not in the multivariate analyses.

These confounding effects could be explained by the correlations between variables shown in table 4. Thus the better survival rate of women tended to be obscured because they were more likely to have histories of hypertension and diabetes and to be older than men. Similarly, the worse survival rate of patients with a systolic blood pressure less than 115 mm Hg at presen-

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Age and sex distribution of patients alive 28 days after MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Five year age groups</td>
<td>Totals</td>
</tr>
<tr>
<td></td>
<td>30-34</td>
</tr>
<tr>
<td>Men</td>
<td>7</td>
</tr>
<tr>
<td>Women</td>
<td>0</td>
</tr>
</tbody>
</table>

FIGURE 1. Crude survival curve for patients alive 28 days after MI and survival curve for a matched "normal" population.

FIGURE 2. Crude survival curves for patients alive 28 days after MI, stratified by history of MI.
tation tended to be obscured by an absence of a history of hypertension. The poor survival rate of patients with a history of angina appeared to be due to their more frequent histories of MI, stroke, and hypertension and their greater age. The poor survival rate of patients suffering cardiac failure on presentation (in the absence of shock) or in the subsequent 28 days appeared to be explained by their more frequent history of MI, stroke, and diabetes, presenting tachycardia, complicating arrhythmias, and their greater age. Finally, the poor survival rate of patients suffering shock in the first 24 hr after MI appeared to be due to their greater likelihood of a systolic blood pressure of less than 115 mm Hg at presentation and of arrhythmias in the first 28 days.

Testing of assumptions. The fit of the models that included the nine main effects shown in table 3 could not be significantly improved by any combination of

964

CIRCULATION
two-way interaction terms (thus the testing of higher-order interactions seemed unnecessary). However, there was some indication that the effects of the covariates did change with months since MI when the model with time interactions was fitted; although the only significant effect was a linear increase in the difference between men and women for IHD deaths. The relative risk for men changed by a factor of 1.3 \((\log_{10} + 3.09)\), where \(t\) is months since MI and 3.09 is the mean of the natural logarithms of the survival times. Inclusion of this interaction term significantly increased the overall likelihood ratio chi square from 98.0 on 9 degrees of freedom to 101.9 on 10 degrees of freedom, but the resultant relative risks and 95% confidence intervals for the nine main effects were virtually unchanged from those shown in table 2 for IHD deaths.

No maximum likelihood solution to the additive risks model could be found. The computational algorithm failed because a number of low-risk subjects were estimated to have a probability of death of less than zero.

**Validation.** The largest value of \(\sqrt{N} D_n(0)\) for any of the four samples was 0.891, and since 0 was between 0.40 and 0.50 for all four samples the predicted and the actual survival functions were not significantly different \((p > .1; \text{table 7.6 in ref. 24})\). There was close agreement between the estimates of the relative risks for the total sample and the average of those for the four samples.

**Prediction.** A straight line was found to be a good fit to the baseline survival function \((r = 1.00)\) and gave a probability of survival of

\[
(1 - 0.0011t)^{R_i} \times R_2 \times \ldots \times R_9
\]

for all causes of death (with \(t < 109\) months). In this expression, \(R_1\) to \(R_9\) are the relative risks shown in table 3 for all causes of death.

The distribution of the nine significant prognostic variables for patients stratified by quintiles of risk of death is shown in table 5 and their survival rates are shown in figure 3. For example, of the patients in the lowest risk quintile, 95% were 34 to 63 years old, 70% were men, and most were in the low-risk category for the other seven prognostic variables. Conversely, of patients in the highest risk quintile, 95% were 49 to 69 years old, 84% were men, 20% had a history of stroke, 77% a history of MI, 17% a history of diabetes, 55% a history of hypertension, 16% a diastolic blood pressure less than 115 mm Hg at presentation, 25% a heart rate greater than 104 beats/min at presentation, and 69% an arrhythmia as a short-term complication. The nine year survival rates of those in the highest and lowest risk quintiles were 22% and 78%, respectively, compared with 52% for all patients alive 28 days after MI.

**Discussion**

**Control of selection bias.** This study has presented the long-term follow-up of patients in a community-based
TABLE 4

Kendall's tau correlation coefficients for variables significant (p < .05) by either univariate or multivariate analyses

<table>
<thead>
<tr>
<th>Covariate</th>
<th>History of MI</th>
<th>History of angina</th>
<th>History of stroke</th>
<th>History of diabetes</th>
<th>History of hypertension</th>
<th>Sex (male)</th>
<th>Systolic BP &lt;115 at presentation</th>
<th>Heart rate bpm &gt;104 at presentation</th>
<th>Arrhythmia in first 28 days</th>
<th>Cardiac failure in first 28 days</th>
<th>Cardiac failure in first 24 hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of MI</td>
<td>.46</td>
<td>.09</td>
<td>.02</td>
<td>-.02</td>
<td>.17</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of angina</td>
<td>-.02</td>
<td>.15</td>
<td>.14</td>
<td>.14</td>
<td>.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of stroke</td>
<td>-.02</td>
<td>-.01</td>
<td>.00</td>
<td>-.11</td>
<td>-.23</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of diabetes</td>
<td>.05</td>
<td>.14</td>
<td>.14</td>
<td>.08</td>
<td>.13</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP &lt;115 at presentation</td>
<td>-.00</td>
<td>.00</td>
<td>.03</td>
<td>-.03</td>
<td>-.16</td>
<td>-.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate &gt;104 bpm at presentation</td>
<td>.04</td>
<td>.03</td>
<td>.05</td>
<td>.04</td>
<td>.12</td>
<td>-.03</td>
<td>-.03</td>
<td>.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arrhythmia in first 28 days</td>
<td>.04</td>
<td>.06</td>
<td>-.00</td>
<td>-.03</td>
<td>.00</td>
<td>.08</td>
<td>.09</td>
<td>.12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac failure on presentation</td>
<td>.08</td>
<td>.06</td>
<td>.15</td>
<td>.05</td>
<td>.03</td>
<td>-.03</td>
<td>.01</td>
<td>.25</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac failure in first 28 days</td>
<td>.10</td>
<td>.06</td>
<td>.10</td>
<td>.11</td>
<td>-.02</td>
<td>.03</td>
<td>.05</td>
<td>.08</td>
<td>.28</td>
<td>.32</td>
<td></td>
</tr>
<tr>
<td>Shock in first 24 hr</td>
<td>.02</td>
<td>-.03</td>
<td>.05</td>
<td>.03</td>
<td>-.05</td>
<td>.03</td>
<td>.03</td>
<td>.20</td>
<td>.02</td>
<td>.15</td>
<td>.10</td>
</tr>
<tr>
<td>Age</td>
<td>.13</td>
<td>.16</td>
<td>.09</td>
<td>.05</td>
<td>.07</td>
<td>-.09</td>
<td>.02</td>
<td>.07</td>
<td>.01</td>
<td>.06</td>
<td>.14</td>
</tr>
</tbody>
</table>

Statistical comparisons (p values are for the correlation coefficients being significantly less than or greater than zero): \( ^a p < .05; ^b p < .01; ^c p < .001; ^d p < .0001. \)

coronary register in which total ascertainment was attempted for symptomatic MI in patients 30 to 69 years old, including those treated at home. A 99.7% follow-up of subjects was achieved over approximately 9 years. Pohjola et al.\(^{25}\) reported a 5 year follow-up of the Helsinki Coronary Register (part of the same WHO collaborative study as the PCR). There have been few other population-based studies of prognosis after MI\(^{26-29}\) and those described as such have usually involved appreciable selection of subjects. For example, Goldberg et al.\(^{26}\) missed an estimated 10% to 15% of subjects because four hospitals refused to participate in their study. Moreover, they included only patients with a discharge diagnosis of acute MI (code 410, International Classification of Diseases, 8th revision), whereas it is necessary to review hospital discharge diagnoses for all IHD (410 to 414) plus chest pain (783.7) to get near total ascertainment of cases of MI\(^{30}\).

TABLE 5

Distribution of prognostic variables for quintiles of risk of death

<table>
<thead>
<tr>
<th>Covariate</th>
<th>1 (34–63)</th>
<th>2 (41–68)</th>
<th>3 (44–68)</th>
<th>4 (45–69)</th>
<th>5 (49–69)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age (5th–95th centiles)</td>
<td>46</td>
<td>54</td>
<td>59</td>
<td>60</td>
<td>62</td>
</tr>
<tr>
<td>Sex (% male)</td>
<td>70</td>
<td>75</td>
<td>75</td>
<td>72</td>
<td>84</td>
</tr>
<tr>
<td>History of stroke (%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>History of MI (%)</td>
<td>0</td>
<td>2</td>
<td>11</td>
<td>36</td>
<td>77</td>
</tr>
<tr>
<td>History of diabetes (%)</td>
<td>0</td>
<td>2</td>
<td>3</td>
<td>10</td>
<td>17</td>
</tr>
<tr>
<td>History of hypertension (%)</td>
<td>6</td>
<td>24</td>
<td>34</td>
<td>43</td>
<td>55</td>
</tr>
<tr>
<td>Systolic blood pressure (%)</td>
<td>0</td>
<td>6</td>
<td>11</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>&lt;115 mm Hg</td>
<td>3</td>
<td>6</td>
<td>10</td>
<td>20</td>
<td>25</td>
</tr>
<tr>
<td>Heart rate &gt;104 bpm (%)</td>
<td>27</td>
<td>46</td>
<td>59</td>
<td>57</td>
<td>69</td>
</tr>
<tr>
<td>Arrhythmia as a (%) complication</td>
<td>125</td>
<td>123</td>
<td>119</td>
<td>126</td>
<td>121</td>
</tr>
<tr>
<td>No. in quintile</td>
<td>78</td>
<td>64</td>
<td>56</td>
<td>42</td>
<td>22</td>
</tr>
<tr>
<td>Nine year survival (%)</td>
<td>78</td>
<td>64</td>
<td>56</td>
<td>42</td>
<td>22</td>
</tr>
</tbody>
</table>
Vedin missed only vital were (also S. P. Fortmann 1981, personal communication). Vedin et al. reported the follow-up of a register that missed only an estimated 10% of cases (among which were all those treated at home). However, their analyses excluded women, reinfections, and a random sample of 30% of patients 57 to 67 years old. The remaining 327 patients were analyzed only in terms of their vital status at the end of 2 years. Nevertheless, the survival rates after recovery from MI reported in past studies, 85% to 90% at 1 year, 65% to 70% at 5 years, and 50% to 55% at 9 years, are in close agreement with our findings.

Control of other sources of bias. In identifying the major factors that affect prognosis, multivariate analyses have commonly been used to control for confounding variables. However, variables whose effects on survival were not statistically significant in univariate analyses have generally been excluded from further study. In this study, sex and hypotension on presentation were statistically significant on multivariate analyses but not when considered alone.

Past studies have commonly used discriminant function analysis and multiple linear regression even though most factors studied were not continuously, let alone normally, distributed. These problems were avoided in this study by the use of nonparametric methods. Factor stratification methods have frequently been used on the assumption that the variables being analyzed interact; however, this study suggests that prognostic factors act approximately independently. In the estimation of primary cardiovascular risk factors, models in which the risks act multiplicatively rather than additively are now established as superior. Despite this, additive models have commonly been used in the analysis of prognostic factors after MI. A model in which risks act multiplicatively seems to make more sense biologically, particularly as estimates of probability of survival must fall between zero and one. In addition, the estimation of probabilities of death as less than one prevented a solution when we attempted to fit an additive model to our data.

Studies that have performed a series of multivariate analyses for different periods after MI have generally found different patterns of prognostic factors. Conversely, there are similarities in the pattern of prognostic factors within the same periods when taken across studies. This implies a time dependence of the prognostic factors; however, to our knowledge no previous studies have tried the inclusion of terms for time in their predictive function. The finding of a significant increase in the difference in IHD mortality between men and women supported the concept of time dependence of prognostic factors. However, although some other interactions with time since MI were substantial, none apart from the interaction of sex with time was statistically significant.

Statistical power. In recent years statistical techniques and computer technology have been developed that allow much more efficient analyses of survival data. As Peto et al. pointed out in their influential article, methods involving a simple count of the numbers dead in each group being compared are inefficient because they waste information as to exactly when each death occurred. They also state that the log rank test is more likely to detect a difference than any other valid assumption-free test when the survival rates of different groups are compared. This study, unlike most previous studies, used "life table" methods and the log rank test for both the univariate and multivariate analyses of risk factors after MI. Furthermore, many previous studies have lacked statistical power because of their small numbers of deaths.

Prognostic factors. The significance of age, sex, past history of hypertension, and past history of diabetes indicates that primary cardiovascular risk factors remain important after MI. Previous studies have also found age, sex, hypertension, and diabetes, together with serum lipid disturbances, smoking habit, and psychological stress, to be associated with an increased risk of death after MI, although other studies have found these variables not to be significant. Although smoking habits after MI have been found to have a significant effect on prognosis, initial smoking status was found to have no effect in this study, confirming the observations of Mulcahy et al. The effects of serum lipids and psychological stress could not be assessed, since they were not recorded in the original data.
The adverse effect of previous MI and history of stroke probably reflected the extent and severity of atherosclerosis. Previous MI has been found to have prognostic significance in numerous studies, and a number of studies have also found coronary angiographic results to be important. A poorer prognosis in stroke patients with IHD has also been found.

The variables found significant in this study that reflect infarction size and site were hypotension and tachycardia on presentation and arrhythmias (ventricular and supraventricular) occurring as short-term complications. These factors have been found to be significant by others and support the importance of the concept of limiting infarction size in the management of MI.

Effects of treatment and clinical implications. In patient assessment after recovery from MI, the only prognostic factors identified in this study that may lead to useful therapeutic intervention were history of hypertension and diabetes. Kannel et al. also found a twofold to threefold excess of deaths for patients who suffered from hypertension before their MIs.

The other factors found to be prognostically significant in this study are generally not amenable to therapy after recovery from MI, except in some cases through coronary artery surgery. Other measures of demonstrated benefit in patient management after MI, β-blocking drugs, and smoking cessation, like coronary artery surgery, only became popular in Australia toward the end of our patient follow-up in 1979. Thus treatments proven to be effective are likely to have had little influence on survival during the period of this study. Consistent with this, recent American studies have shown no improvement in the survival of patients discharged from the hospital after MI between 1961 and 1978.

The impact of individual patient management after MI could not be addressed in this study, since no relevant data were collected. The management of these patients, however, included the full spectrum of medical care available in Perth and was probably typical of other western countries at that time. To this extent, therefore, our results may be taken as a guide to the outcome of MI in similar populations in that era. Given the increased use of β-blocking drugs and coronary artery surgery, and perhaps a greater number of patients who stop smoking, it is doubtful that our results represent the situation found today.

We thank Sister Margaret Cook for her contribution to this project, and Denham Pole, Ralph Reader, and Michael McCall as originators of the Perth Coronary Register.

References
20. Buckley JD: Program "SURVIVAL", Clinical Research Unit, Walter and Eliza Hall Institute of Medical Research, Parkville 3050, 1979
23. Thomas DC: Program "RISK," Department of Epidemiology and Health, McGill University, Montreal, Quebec, Canada, 1980
30. Hobbs MST, Martin CA, Armstrong BK: A method for estimating the incidence of acute myocardial infarction from hospital records and death certificates. NH & MRC Research Unit in Epidemiology and Preventive Medicine, Department of Medicine, University of Western Australia, Nedlands, 6009, 1981
Long-term prognosis after recovery from myocardial infarction: a nine year follow-up of the Perth Coronary Register.
C A Martin, P L Thompson, B K Armstrong, M S Hobbs and N de Klerk

Circulation. 1983;68:961-969
doi: 10.1161/01.CIR.68.5.961

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/68/5/961