

Reduced risk of death at 28 days in patients taking a β blocker before admission to hospital with myocardial infarction

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Abstract

Objective—To see whether patients taking an oral β blocker at the time of admission to hospital with myocardial infarction have a reduced risk of death at 28 days.

Design—Retrospective analysis of data collected on patients admitted over four years.

Setting—Community based study.

Patients—2430 Consecutive patients living in the Perth statistical division admitted to hospital with myocardial infarction during 1984-7.

Main outcome measure—Survival at 28 days among patients taking a β blocker at onset of myocardial infarction.

Results—Patients were grouped into those who were and were not taking a β blocker at the time of admission. Though patients taking a β blocker were older and more likely to have a history of myocardial infarction, angina, or hypertension, the overall mortality at 28 days was similar in the two groups. A logistic regression model used to adjust for factors predictive of cardiac death at 28 days confirmed that patients taking a β blocker at the time of admission had a significantly reduced risk of death (relative risk 0.50; 95% confidence interval 0.34 to 0.76). Though the incidence of fatal ventricular fibrillation was similar in the two groups, mean peak creatine kinase activity was significantly lower in the β blocker group.

Conclusions—These data support the value of long term use of β blockers in patients at risk of myocardial infarction. They suggest that patients taking these agents before admission to hospital with myocardial infarction have a significant survival advantage at 28 days, which may be due to a reduction in infarct size.

Introduction

Though patients taking an oral β blocker after acute myocardial infarction have a reduced risk of sudden out of hospital cardiac death and reinfarction,¹ it is not known whether these patients continue to derive benefit from this treatment after admission to hospital with myocardial infarction. This study therefore compared morbidity and mortality at 28 days after myocardial infarction in patients who were and were not taking an oral β blocker at the time of admission.

Detection of a measurable effect of β blockers on 28 day survival in these patients would have important implications regarding their long term use in patients at risk of myocardial infarction and also would help show whether the widespread use of these agents in the community has contributed to the decline in mortality at 28 days after myocardial infarction.^{2,3}

Patients and methods

We studied data from patients aged 25-64 living within the Perth statistical division who were admitted

to hospital with a myocardial infarction during the four years 1984-7. The data form a subset of those collected as part of the MONICA project, an international study conducted under the auspices of the World Health Organisation to monitor trends and determinants of mortality from cardiovascular disease over 10 years.⁴ Prehospital data include each patient's age, sex, smoking habits, and history of cardiovascular disease; medicines being taken at the time of onset of myocardial infarction; and symptoms and complications occurring before arrival at hospital. In hospital data include clinical features on admission, peak plasma creatine kinase activities, cardiac complications, and cardioactive drugs given in hospital. Up to five electrocardiograms for each episode are selected and coded in detail by using the Minnesota system.⁵ Deaths occurring within 28 days after the onset of symptoms of myocardial infarction are determined by review of all death registrations in Western Australia. In accordance with the MONICA protocol episodes of infarction beginning less than 28 days apart are counted only once and each is classified as a "definite" or "possible" infarction according to criteria adopted by the WHO.⁶

Included in the analysis were all patients suffering a definite myocardial infarction as well as those fatal cases diagnosed as possible (these patients had symptoms typical of myocardial infarction but died before confirmation of definite infarction by electrocardiography or enzyme analysis⁷). Comparison of peak plasma creatine kinase activities was limited to those patients surviving at least 24 hours after admission and who had not had cardiopulmonary resuscitation or DC cardioversion. This was done in order to ensure that true peak creatine kinase activities were measured and that patients with non-cardiac sources of creatine kinase were excluded.

Statistical analysis—The characteristics of patients who were and were not taking a β blocker at the time of onset of symptoms of myocardial infarction were compared by χ^2 and *t* tests. Logistic regression was used to determine characteristics predictive of death within 28 days after the onset of symptoms by using the EPIC program.⁸ Variables included in these models were those identified in past studies as having prognostic relevance and those which were biologically important or suggested by preliminary univariate analyses. History of myocardial infarction was recorded as positive if the medical records for the current event included evidence of previous admission to hospital with a clinical diagnosis of myocardial infarction. Where appropriate variables measured on a continuous scale were grouped into categories based on past studies. After including several known prognostic factors in a logistic regression model a variable indicating whether or not the patient was taking a β blocker at the time of the present infarct was added. The value of this extra term was assessed by means of a likelihood ratio test. The logistic regression model was also used to determine the expected number of deaths in the

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entire group had β blockers not been available by supposing that each patient in the β blocker group had the same risk of death as patients in the non- β blocker group.

Results

During 1984-7, 2430 residents of the Perth statistical division under the age of 65 were admitted to hospital with a clinical diagnosis of myocardial infarction. Of these, 2277 had a definite myocardial infarction and 153 patients died of a possible myocardial infarction. Altogether 325 patients died within 28 days of their infarction and 2105 survived this period. A total of 574 (24%) patients with definite or fatal possible myocardial infarction were taking a β blocker at the time of onset of symptoms. At discharge 491 (85%) of these patients were still taking a β blocker and half (975) of those in the non- β blocker group had started β blockers.

Table I compares the clinical details of patients who were and were not taking a β blocker at the time of admission. Patients in the β blocker group were older, more likely to be women, and more likely to have a past history of acute myocardial infarction, angina, hypertension, and coronary artery bypass surgery. They were also more likely to be non-smokers and to be receiving other forms of treatment, including calcium channel blockers, diuretics, and nitrates. The maximum pulse rate recorded in the first 24 hours after admission was lower in patients taking a β blocker ($p < 0.001$) but blood pressure on admission was higher. The frequency of left ventricular failure and the proportion of patients with anterior infarction were almost identical in the two groups.

TABLE I—Clinical details of patients taking and not taking β blockers on admission to hospital. Except where stated otherwise figures are numbers (percentages) of patients

| | β Blocker group (n=574) | Non- β blocker group (n=1856) | p Value (χ^2 test except*) |
|---|----------------------------------|--|-------------------------------------|
| Male | 441 (76.8) | 1531 (82.5) | 0.002 |
| Mean age (years) | 56.7 | 54.5 | <0.001* |
| Past history: | | | |
| Acute myocardial infarction | 284 (49.5) | 472 (25.4) | <0.001 |
| Angina | 279 (48.6) | 666 (35.9) | <0.001 |
| Hypertension | 451 (78.6) | 575 (31.0) | <0.001 |
| Coronary artery bypass graft | 65 (11.3) | 89 (4.8) | <0.001 |
| Current smoker | 202 (35.2) | 915 (49.3) | <0.001 |
| Ex-smoker | 219 (38.2) | 497 (26.8) | <0.001 |
| Time to medical care <4 h | 385 (67.1) | 1225 (66.0) | NS |
| Drugs being taken on admission: | | | |
| Calcium channel blockers | 105 (18.3) | 244 (13.1) | 0.002 |
| Diuretics | 207 (36.1) | 326 (17.6) | <0.001 |
| Digoxin | 16 (2.8) | 113 (6.1) | 0.002 |
| Nitrates | 222 (38.7) | 296 (15.9) | <0.001 |
| Findings during current admission: | | | |
| Mean maximum pulse rate in first 24 h (beats/min) | 92 | 97 | <0.001* |
| Mean systolic blood pressure (mm Hg) | 141 | 138 | 0.033* |
| Anterior acute myocardial infarction | 248 (43.2) | 765 (41.2) | NS |
| Acute left ventricular failure | 21 (3.7) | 66 (3.6) | NS |
| Mean peak creatine kinase activity (IU/l) | 1358 | 1503 | 0.001† |

*Student's *t* test.

†Wilcoxon rank test.

There was no significant difference in the overall mortality at 28 days between the β blocker and non- β blocker groups (76/574 (13.2%) *v* 249/1856 (13.4%); $p=0.91$) (table II). Nevertheless, for patients among whom mortality was expected to be high—for example, those with a past history of myocardial infarction, bypass surgery, or use of diuretics—it was significantly lower at 28 days in those who had been taking a β blocker before the onset of the acute event. A similar trend was seen in patients with a history of hypertension.

TABLE II—Univariate analyses of 28 day mortality in the two groups. Figures are numbers (percentages) of patients

| | β Blocker group | Non- β blocker group | p Value |
|-----------------------------------|-----------------------|----------------------------|---------|
| All patients | 76/574 (13.2) | 249/1856 (13.4) | 0.91 |
| Past acute myocardial infarction | 38/284 (12.7) | 117/472 (24.8) | <0.001 |
| Patients taking diuretics | 35/207 (16.9) | 121/326 (37.1) | <0.001 |
| Past coronary artery bypass graft | 8/65 (12.3) | 24/89 (27.0) | 0.027 |
| Hypertension | 59/451 (13.1) | 95/575 (16.5) | 0.126 |

Table III gives the results from the logistic regression model used to determine the predictors of 28 day mortality. The main predictors of death were past history of myocardial infarction; female sex; age; maximum pulse rate recorded during the first 24 hours after admission; and the development of atrial fibrillation, complete heart block, ventricular fibrillation, or pulmonary oedema. Neither site of infarction nor a past history of either hypertension or angina was found to be an independent marker of 28 day mortality in this model. Smoking was associated with a survival advantage, but this was not evident among patients surviving more than 24 hours after admission. Prior use of diuretics, calcium channel blocking agents, digoxin, or nitrates was associated with an increased risk of death at 28 days.

TABLE III—Relative risks of mortality at 28 days (from multiple logistic regression analysis)

| | No of patients with each risk factor | Relative risk | 95% Confidence interval |
|--|--------------------------------------|---------------|-------------------------|
| History of acute myocardial infarction | 756 | 1.50 | 1.00 to 2.30 |
| Female | 458 | 1.50 | 1.00 to 2.20 |
| Age >55 | 1031 | 2.00 | 1.40 to 2.80 |
| Pulse >100 beats/min | 747 | 2.90 | 2.00 to 4.00 |
| Atrial fibrillation | 240 | 1.80 | 1.20 to 2.80 |
| Complete heart block | 134 | 3.10 | 1.90 to 5.30 |
| Ventricular fibrillation | 225 | 8.70 | 5.80 to 13.00 |
| Pulmonary oedema | 225 | 5.70 | 3.90 to 8.50 |
| Current smoker | 1117 | 0.50 | 0.40 to 0.80 |
| Drugs being taken on admission: | | | |
| Diuretics | 533 | 1.90 | 1.30 to 2.70 |
| Calcium channel blockers | 349 | 1.40 | 0.90 to 2.20 |
| Digoxin | 129 | 1.70 | 1.00 to 2.90 |
| Nitrates | 518 | 1.50 | 1.00 to 2.30 |
| β Blockers | 574 | 0.50 | 0.34 to 0.76 |

When the prior use of a β blocker was added to the model it was found to be associated with an independent beneficial effect on mortality (relative risk 0.50; 95% confidence interval 0.34 to 0.76), which was most evident in patients surviving the first 24 hours after admission (relative risk 0.31; 95% confidence interval 0.18 to 0.54) (table IV). The expected overall mortality among the patients at 28 days had no patient been taking a β blocker was 14.6% (354 deaths) as opposed to the observed figure of 13.4% (325 deaths).

TABLE IV—Relative risks associated with prior use of β blockers for all patients and for three subgroups based on survival time. (Figures determined by fitting same multiple logistic model as used in table III)

| | Relative risk | 95% Confidence interval |
|--|---------------|-------------------------|
| All patients (patients who died at any time within first 28 days after acute myocardial infarction: n=2430; deaths=325) | 0.50 | 0.34 to 0.76 |
| “State at one hour” group (patients who died within first hour after acute myocardial infarction: n=2430; deaths=57) | 1.16 | 0.58 to 2.32 |
| “State at 24 hours” group (patients who died between one and 24 hours after acute myocardial infarction: n=2373; deaths=89) | 1.05 | 0.59 to 1.87 |
| “24 Hour survivors” group (patients who died between 24 hours and 28 days after acute myocardial infarction: n=2284; deaths=179) | 0.31 | 0.18 to 0.54 |

TABLE V—Numbers (percentages) of patients suffering complications after arrival in hospital

| Complication | β Blocker group (n=574) | Non- β blocker group (n=1856) | p Value |
|--------------------------|----------------------------------|--|---------|
| Atrial fibrillation | 58 (10.1) | 182 (9.8) | NS |
| Complete heart block | 41 (7.1) | 93 (5.0) | 0.055 |
| Ventricular tachycardia | 172 (30.0) | 671 (36.2) | 0.004 |
| Ventricular fibrillation | 72 (12.5) | 153 (8.2) | <0.001 |
| Fatal | 26 (4.5) | 70 (3.8) | NS |
| Non-fatal | 46 (8.0) | 83 (4.5) | <0.001 |
| Pulmonary oedema | 68 (11.8) | 157 (8.5) | 0.016 |
| Cardiogenic shock | 46 (8.0) | 140 (7.5) | NS |
| Reinfarction | 25 (4.4) | 59 (3.2) | 0.174 |

Table V lists the complications affecting the two groups of patients. The incidence of atrial fibrillation was similar in each group and complete heart block tended to be more frequent ($p=0.055$) in the β blocker group. Though ventricular tachycardia (three or more consecutive ventricular beats) was less common in the β blocker group ($p=0.004$), the overall incidence of ventricular fibrillation was higher in these patients ($p<0.001$). Pulmonary oedema was more frequent in the β blocker group but there was no significant difference in the incidence of cardiogenic shock between the groups. Peak creatine kinase activities in patients surviving the first 24 hours of admission were lower among those taking a β blocker ($p=0.001$; Wilcoxon rank test) (table I).

Discussion

Treatment with β blockers given intravenously during the early hours after acute myocardial infarction has been shown to reduce early cardiac mortality,⁹ possibly by reducing infarct size.¹⁰ Furthermore, when given to patients surviving myocardial infarction oral β blockers reduce the incidence of both out of hospital sudden cardiac death and recurrent myocardial infarction.¹¹⁻¹⁴ In this study we tested the hypothesis that patients taking a β blocker at the time of onset of acute myocardial infarction would receive a benefit from this treatment and that this would be detectable as a reduction in mortality at 28 days after the acute event.

In this community based study 574 (24%) of 2430 patients admitted to hospital with acute myocardial infarction were taking a β blocker. This high proportion reflects the high prevalence of hypertension and angina among patients developing myocardial infarction and also highlights the widespread use of β blockers in this community.¹⁵ Though these patients were at high risk of early cardiac death by virtue of being older and having a higher prevalence of past angina, myocardial infarction, hypertension, or bypass surgery, 28 day mortality was similar to that of patients who were not taking a β blocker at the time of infarction. After correction for these and other recognised adverse influences on survival it was confirmed that patients taking a β blocker before the onset of myocardial infarction had a significantly reduced risk of death at 28 days, their overall relative risk being 0.50.

A possible interpretation of these data is that patients taking a β blocker at the time of myocardial infarction were more likely to die suddenly before they could be admitted to hospital, so that those who reached hospital alive formed a group selected for better survival at 28 days. This hypothesis, however, contradicts the large body of evidence that oral β blockade reduces the risk of sudden out of hospital cardiac death.¹ In addition, the patients in the β blocker group in this study were clearly at a high risk of death (table I). Another possible interpretation is that the apparent benefit of a β blocker was not dependent on its prior use but rather reflected the fact that a

higher proportion of patients in the β blocker group (491/574 (85%) v 975/1856 (53%) in the non- β blocker group) received a β blocker in hospital. This explanation also seems unlikely, however, as no published study evaluating the effect of oral β blockers begun in hospital after myocardial infarction has been able to show a statistically significant survival advantage by 28 days.¹¹⁻¹⁴

A more likely explanation for the observed benefit is that it was due to the prior use of oral β blockers. Though there was no evidence of suppression of ventricular fibrillation by the prior use of these agents, our finding that the mean peak creatine kinase activity was lower among patients in the β blocker group suggests that established β blockade may have conferred a cardioprotective effect by acting to reduce infarct size. This observation is consistent with several studies in which infarct size estimated by measuring total creatine kinase release was found to have been reduced after treatment with intravenous β blockers given in the early hours after infarction.^{10, 16-18} Also in keeping with this suggestion is the observation that patients in the β blocker group were no more likely to develop cardiogenic shock despite their higher risk profile. Furthermore, that the protective effect was particularly evident among high risk patients (table II) is consistent with the results of both the ISIS-1 trial (first international study of infarct survival)¹ and the MIAMI trial (metoprolol in acute myocardial infarction).¹¹

In view of the relative benefit associated with prior use of β blockers and the high proportion of patients (24%) receiving this treatment at the time of their infarction it seems that β blockers have contributed to the decline in 28 day mortality in patients admitted to hospital after myocardial infarction in this community.¹⁵ We estimate that 29 more deaths would have occurred among these patients had none of them been taking β blockers. This represents one death prevented for every 19 patients taking a β blocker at the time of onset of symptoms. In contrast, the ISIS-1 study concluded that one life would be saved for every 200 patients treated early with intravenous β blocker after myocardial infarction. Taken together, these results are consistent with the hypothesis that the efficacy of β blockers in myocardial infarction is dependent on when they are given.

In conclusion, this community based study suggests that prior treatment with oral β blockers may confer a "cardioprotective effect" in patients admitted to hospital with acute myocardial infarction. Though this benefit does not seem to be due to a reduction in serious arrhythmias, it may be due to a reduction in infarct size. An important consequence of these data is that they support the long term use of β blockers in patients at risk of myocardial infarction. They also suggest that the widespread use of β blockers in the community has contributed to the decline in 28 day mortality of patients admitted to hospital with myocardial infarction.

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Individual variation between general practitioners in labelling of hypertension

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Abstract

Variation in labelling of hypertension by individual general practitioners was studied during a continuous opportunistic screening programme for hypertension in a single general practice with 12 principals. All the general practitioners agreed to label as hypertensive patients with systolic pressures of ≥ 200 mm Hg or diastolic pressures of ≥ 110 mm Hg on three consecutive readings. The overall number of patients labelled hypertensive at the beginning of the screening programme was 505 and this rose to 801 after five years. There was a large variation in the numbers of patients recorded as hypertensive at the start of the screening period, with numbers ranging from eight to 112 for individual practitioners. The variation persisted during the screening period, with the numbers of patients detected by individual general practitioners ranging from four to 46. The average systolic and diastolic pressures recorded among these patients also varied between doctors, and only 24 out of 187 patients had their high pressures recorded on three occasions and so fully met the criteria for diagnosing hypertension.

Clearly, general practitioners are following their own individual criteria in defining hypertension and taking into account factors other than just the measured blood pressure.

Introduction

During the course of a computerised blood pressure screening programme in a large general practice we

studied the variation among individual general practitioners in their labelling of hypertension.

Methods

A continuous computerised blood pressure screening programme was undertaken in a large group practice of 12 principals with a list size of about 32 000. Comparison between the individual general practitioners at the start of the screening and after five years was possible as the practice adhered strictly to an individual list system: as far as possible a patient attended the general practitioner he or she was registered with.

All patients aged 40 to 65 years were identified from the age-sex register, and if a normal blood pressure (systolic ≤ 160 mm Hg and diastolic ≤ 100 mm Hg) had not been recorded in their notes within the preceding five years the notes were tagged to alert the general practitioner to measure blood pressure when the patient next attended.¹

Although there was disagreement over whether to treat mild to moderate levels of raised blood pressure, all the general practitioners agreed that a patient should be labelled hypertensive when three readings of systolic pressure ≥ 200 mm Hg or diastolic pressure ≥ 110 mm Hg were recorded.^{2,3}

Results

From 1983 to 1988 the population to be screened increased by 1856 people through people moving in and out of the practice and age group. The table shows the numbers of patients labelled hypertensive by each general practitioner at the start of the screening programme and after five years of screening.

At the start of screening there were 8800 people aged 40-65 years. Of these 505 (5.7%) were labelled hypertensive, although the numbers of such patients varied from eight to 112 for individual general practitioners. After five years of screening the total population was 10 655, of whom 801 (7.5%) were labelled hypertensive, with numbers for each general practitioner ranging from 28 to 160. The number of patients labelled hypertensive during the screening period ranged from four to 46.

None of the general practitioners adhered fully to the criteria for labelling hypertension. Of the 187 patients labelled hypertensive during screening only 24 (12.8%) had had three readings. Overall only 78 (42%) patients had had two or three readings that fulfilled the

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Variation among individual general practitioners in their labelling of hypertension during screening period 1983-8: numbers of patients detected and their average blood pressures

| Doctor | No (%) hypertensive in: | | No (%) detected by screening | No (and No treated where different) with systolic ≥ 200 mm Hg and/or diastolic ≥ 110 mm Hg on: | | | Average systolic and diastolic pressures of all patients detected during screening (mm Hg) |
|--------|-------------------------|----------|------------------------------------|--|------------|-----------|---|
| | 1983 | 1988 | | 3 Readings | 2 Readings | 1 Reading | |
| 1 | 112 (22) | 126 (16) | 22 (12) | 3 | 6 | 13 | 197/107 |
| 2 | 107 (21) | 160 (20) | 46 (25) | 2 (1) | 12 | 32 (28) | 185/103 |
| 3 | 53 (11) | 86 (11) | 27 (15) | 6 | 2 (1) | 19 (13) | 199/110 |
| 4 | 53 (11) | 72 (9) | 12 (6) | 2 | 3 | 7 | 191/109 |
| 5 | 42 (8) | 59 (7) | 22 (12) | 2 | 3 | 17 (14) | 178/105 |
| 6 | 31 (6) | 46 (6) | 9 (5) | 0 | 8 | 1 (0) | 194/108 |
| 7 | 27 (5) | 54 (7) | 15 (8) | 0 | 6 | 9 (7) | 200/105 |
| 8 | 26 (5) | 32 (4) | 6 (3) | 0 | 2 | 4 (3) | 173/109 |
| 9 | 20 (4) | 34 (4) | 4 (2) | 3 | 0 | 1 | 206/110 |
| 10 | 14 (3) | 28 (4) | 7 (4) | 2 | 3 | 2 (1) | 177/116 |
| 11 | 12 (2) | 51 (6) | 13 (7) | 3 | 7 | 3 | 193/112 |
| 12 | 8 (2) | 53 (7) | 4 (2) | 1 | 2 | 1 | 195/126 |
| Total | 505 | 801 | 187 | 24 (33) | 54 (53) | 109 (91) | |