Effects of Captopril on Ischemia and Dysfunction of the Left Ventricle After Myocardial Infarction

Peter Søgaard, MD; Carl-Otto Gøtzsche, MD; Jan Ravkilde, MD; and Kristian Thygesen, MD

Background. Treatment with angiotensin converting enzyme inhibitors has been shown to be beneficial in patients with heart failure and myocardial infarction. Experimental studies have shown beneficial effects on ischemic myocardium.

Methods and Results. The effects of captopril were evaluated in 64 patients with left ventricular dysfunction after myocardial infarction. The patients were randomized at day 7 to either placebo or captopril (50 mg daily) in a double-blind parallel study over a period of 6 months. The patients were followed up by means of ambulatory ECG monitoring, bicycle ergometer testing, and echocardiographic examination. The duration of ST segment depression detected during ambulatory ECG monitoring was lower in the captopril group (87 minutes) than in the placebo group (638 minutes) (p < 0.001), and the number of patients in the captopril group with exercise-induced ST segment depression (p < 0.01) was lower at the completion of the study. The working capacity increased during the study period from 540 ± 47 seconds to 738 ± 41 seconds (p < 0.01) in the captopril group and was higher than that in the placebo group (530 ± 43 seconds, p < 0.01) at the end of the study. Furthermore, a significant dilation of the left ventricular end-diastolic and end-systolic volumes was observed in the placebo group (p < 0.05); this was prevented in the captopril group where, in addition, a reduction in end-systolic volume was observed (p < 0.05).

Conclusions. Captopril has a favorable effect on the dysfunctioning myocardium after myocardial infarction inasmuch as the ischemic burden is reduced, the working capacity is increased, dilation of the left ventricle is prevented, and systolic function is improved. (Circulation 1993;87:1093-1099)

Key Words • exercise capacity • ACE inhibition • ST segment • myocardial remodeling

Ischemia and ailing function of the myocardium are key factors in the prognosis after myocardial infarction (MI). Angiotensin converting enzyme (ACE) inhibitors have a beneficial effect on severe heart failure inasmuch as mortality is reduced and left ventricular function is improved.1,2 Animal experimental studies have shown that ACE inhibitors have an advantageous effect on the remodeling of the myocardium after MI.3,4 Furthermore, clinical studies have demonstrated that captopril prevents progressive dilation of the left ventricle after MI.5-7

Experimental studies have also indicated that ACE inhibitors dilate the coronary arteries in cases of acute ischemia.8,9 In addition, it has been shown that infarct extension can be impeded and survival improved as a result of ACE inhibition during experimental MI.9-12 The effects of ACE inhibitors on the myocardium appear to be multifactorial and promising with regard to an anti-ischemic effect as well as in the treatment of acute heart failure. The present study was designed to elucidate the role played by the ACE inhibitor captopril in combating ischemia of the myocardium and the ability of captopril to improve physical performance in patients having signs of left ventricular dysfunction during the early phase after MI.

Methods

Patient Selection

Patients younger than 70 years of age suffering from MI were included provided that they had left ventricular ejection fraction (EF) < 45% as evaluated by echocardiography on day 5 after the MI. The diagnosis of MI was established using the World Health Organization criteria.13 Patients who had previously received treatment for heart failure and who required an ACE inhibitor or digoxin were excluded from the investigation. Furthermore, patients presenting with ECG signs of a previous MI, persistent ST segment deviation, and paradoxical wall movement of the left ventricle demonstrated by echocardiography were also excluded, as were patients with systolic blood pressure < 100 mm Hg, atrial fibrillation, diseases of the heart valves, bundle branch block, severe systemic disease, and liver or kidney disease.

Patients who were subjected to coronary artery bypass grafting during the follow-up period were excluded. The reason for this was that this procedure presumably has an effect on the ischemia during the remainder of the follow-up period.14

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A total of 64 patients were enrolled; this was the result of background screening of 134 consecutive MI patients. All of the patients gave their informed consent after both oral and written information was given. The study was carried out in agreement with the Helsinki II Declaration and was approved by the regional Scientific Ethical Committee as well as the Danish National Board of Health.

**Study Design**

The study was designed as a randomized, double-blind, placebo-controlled parallel study. On the seventh day after onset of MI, the patients were consecutively randomized to either placebo or captopril and given an initial blinded dose of 6.25 mg followed by placebo or captopril 12.5 mg b.i.d. for the next 14 days. The randomization schedule had a block size of 10. The dosage was increased after a clinical examination to 25 mg b.i.d. for the remainder of the study period, which lasted 180 days.

Echocardiography was carried out by two of the authors (P.S., C.O.G.) on day 5 after the onset of symptoms; this was repeated at the outpatient control examination on days 30, 90, and 180. The examination was carried out with the patient in the left lateral position. An apical four-chamber scan was used, the longitudinal axis was maximized, and the images were taken after expiration during normal breathing. Using ECG-triggered recordings, the left ventricular end-diastolic volume was recorded at the end of the T wave and the end-diastolic volume at the onset of the QRS complex. The single-plane area–length method was used for calculation of the volume of the left ventricle on the echocardiographic screen, and hard copies were recorded for documentation. An average of three measurements was used. Left ventricular end-diastolic volume index (EDVI, mL/m²) and left ventricular systolic volume index (ESVI, mL/m²) were derived using the body surface area estimated at each time point.

Interobserver and intraobserver variability was determined from a random sample of 10 study patients. The interobserver coefficient of variation (CV%) was 2% and the intraobserver CV% was 1.2% for repeated measurement of consecutive samples.

Twenty-four hours of calibrated ambulatory ECG monitoring (Reynolds-Tracker) was performed on day 6 after the onset of symptoms and was repeated at all of the outpatient examinations. Medication was continued during the monitoring. ECG mapping was carried out, and leads showing infarct-related Q waves and ST segment deviation as well as significant postural changes were discarded; modified V₁, V₅, and aVF leads were used to achieve ST segment analyses in two separate leads. All of the tape analyses were performed under visual control by one of the authors (P.S.) at 60 times the recording speed on a Reynolds Pathfinder 3 ST connected to an ST scope. ST events were considered significant when they were horizontal or descending and were ≥0.1 mV, ≥1 minute, and measured 80 msec after the J-point. All such events were printed out for visual evaluation before final acceptance. Changes in the T-vector were not accepted as a sign of ischemia unless they were accompanied by the above-mentioned shifts in the ST segment.

### Table 1. Demographic Data at Baseline

<table>
<thead>
<tr>
<th></th>
<th>Placebo (n=32)</th>
<th>Captopril (n=32)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>58 (43–70)</td>
<td>60 (35–70)</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>30/2</td>
<td>28/4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176 (152–195)</td>
<td>175 (160–189)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83 (62–105)</td>
<td>77 (59–107)</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>1/4</td>
<td>2/5</td>
</tr>
<tr>
<td>Hypertension</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Anterior/inferior myocardial infarction</td>
<td>18/14</td>
<td>17/15</td>
</tr>
<tr>
<td>Peak creatine kinase–B (units/L)</td>
<td>87 (17–276)</td>
<td>83 (17–210)</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>18</td>
<td>20</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>15</td>
<td>17</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>40 (30–45)</td>
<td>39 (25–45)</td>
</tr>
</tbody>
</table>

Values are mean (range shown in parentheses). Between-group comparisons are all nonsignificant.

A symptom-limited bicycle ergometer test was performed on days 30, 90, and 180. The initial loading was 50 W, increasing by 25 W every 3 minutes with a steady-state period. A 12-lead ECG and blood pressure were recorded every minute. The patients carried on the test until exhaustion or until a period of 18 minutes had been completed. A fall in blood pressure >10 mm Hg at two consecutive measurements and/or the presence of severe arrhythmias resulted in discontinuation of the test. Total exercise capacity, ST segment depression, heart rate–blood pressure product (RBPP), and increase in RBPP were noted.

Tapes from 24-hour ECG monitoring were surveyed, and evaluation of echocardiographic results and exercise tests was performed after the last patient had completed the study but before the code was opened.

### Statistical Analyses

Baseline characteristics were compared using the χ² test for categorical variables and the unpaired t test for continuous variables. The effects of treatment within and between groups were tested using the Kruskall-Wallis test. Fisher’s exact test was used for comparison of numbers of patients. Correlation analyses were performed using Spearman’s test. CV% was calculated as the relation between the standard deviation and the mean of the observations multiplied by 100%. A value of p<0.05 was considered statistically significant.

### Results

#### Baseline Evaluation

There were 32 patients in each group, and both groups were comparable at baseline with regard to demographic and clinical data, e.g., infarct site and size as well as left ventricular function. Eighteen patients in the placebo group and 20 in the captopril group showed signs of congestive heart failure during the first 5 days (Table 1). Treatment on admission to the coronary care unit with the thrombolytic agent streptokinase as well as oral treatment during days 0–5 with low-dose acetylsalicylic acid and metoprolol was similar in the two groups. The same applied to adjuvant treatment with isosorbide
mononitrate, diltiazem, and furosemide (Table 2). No diuretic other than furosemide was used. Only 11 patients (34%) in each group needed diuretic therapy at the time of randomization.

Follow-up Evaluation

A total of 58 patients (29 in each group) completed the study with all control examinations throughout the 180 days. Six patients dropped out for the following reasons: Two died (one in each group), three were subjected to coronary artery bypass grafting (two in the placebo group and one in the captopril group), and one was lost to follow-up (captopril group).

The patients with spontaneous ST depression during the 24 hours of ambulatory ECG monitoring were distributed as shown in Table 3. Both groups were comparable at baseline, with 22 in the captopril group and 21 in the placebo group having spontaneous ST depression. A gradual reduction in the number of patients having spontaneous ST depression was observed during the study period in the captopril group: from an initial 22 to a final seven ($p<0.01$); whereas the number of patients with spontaneous ST depression in the placebo group remained unchanged: 21 versus 22 (NS). Accordingly, there was a significant difference between the groups at both days 90 and 180 ($p<0.01$). The mean duration of the spontaneous ST depressions did not differ between the groups at baseline. A significant reduction in the duration of ST depression was observed in both groups up to day 30 ($p<0.05$). This tendency continued in the captopril group, in which a further significant reduction was observed from day 30 to day 180 ($p=0.02$), whereas in the placebo group there was a rise, although not significant ($p=0.07$), in the mean duration of ST depression during the same period. Thus, from day 90, there was a significant difference between the groups ($p<0.01$) as well as at day 180 ($p<0.001$), as shown in Table 3.

The same pattern could be seen with regard to exercise-triggered ST depression inasmuch as the captopril group, compared with the placebo group, demonstrated a significantly lower number of patients with ST segment depression during the exercise test ($p<0.01$) at the end of the study (Table 4). Of the original 12 patients in the captopril group with significant ST depression during exercise testing, six still had this at completion of the study. However, the time to depression increased in the captopril group from 300±60 seconds to 480±60 seconds ($p=0.55$), and RBPP for the same group rose at that time from 152±8 beats×min⁻¹×mm Hg×10⁻² to 209±8 beats×min⁻¹×mm Hg×10⁻² ($p<0.01$). The placebo group demonstrated no change in the time to ST depression or the RBPP during the same period. These data as well as the

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**Table 2.** Frequency of Adjuvant Therapy Used During Days 0–5

<table>
<thead>
<tr>
<th>Drug</th>
<th>Placebo (n=32)</th>
<th>Captopril (n=32)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoprolol</td>
<td>23 (72%)</td>
<td>24 (75%)</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>8 (25%)</td>
<td>6 (19%)</td>
</tr>
<tr>
<td>Isosorbide mononitrate</td>
<td>5 (16%)</td>
<td>5 (16%)</td>
</tr>
<tr>
<td>Streptokinase</td>
<td>26 (81%)</td>
<td>25 (78%)</td>
</tr>
<tr>
<td>Acetylsalicylic acid</td>
<td>32 (100%)</td>
<td>32 (100%)</td>
</tr>
<tr>
<td>Furosemide</td>
<td>18 (56%)</td>
<td>20 (63%)</td>
</tr>
</tbody>
</table>

Between-group comparisons nonsignificant.

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**Table 3.** Twenty-Four-Hour Ambulatory ECG Monitoring After Myocardial Infarction: Duration of ST Segment Depression

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Day 30</th>
<th>Day 90</th>
<th>Day 180</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration (minutes)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>32 (0–84)</td>
<td>13 (0–66)†</td>
<td>21 (0–74)</td>
<td></td>
</tr>
<tr>
<td>Captopril</td>
<td>35 (0–86)</td>
<td>13 (0–85)‡</td>
<td>6 (0–60)</td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>21</td>
<td>18</td>
<td>21</td>
<td>22</td>
</tr>
<tr>
<td>Captopril</td>
<td>22</td>
<td>14</td>
<td>10</td>
<td>7</td>
</tr>
</tbody>
</table>

Mean duration of significant ST segment depressions are shown with ranges in parentheses.

* $p<0.01$, †$p<0.001$ between groups.
‡$p<0.05$ compared with baseline.
§$p=0.02$ compared with day 30.
||$p<0.01$ compared with inclusion.

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**Table 4.** Exercise Testing Up to Day 180 After Myocardial Infarction

<table>
<thead>
<tr>
<th></th>
<th>Day 30</th>
<th>Day 90</th>
<th>Day 180</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal RBPP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>216±10</td>
<td>224±11</td>
<td>226±10</td>
</tr>
<tr>
<td>Captopril</td>
<td>220±9</td>
<td>240±8</td>
<td>255±9</td>
</tr>
<tr>
<td>Increase in RBPP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>2.8±0.05</td>
<td>2.6±0.04</td>
<td>2.5±0.05</td>
</tr>
<tr>
<td>Captopril</td>
<td>2.9±0.06</td>
<td>3.2±0.05</td>
<td>3.4±0.04</td>
</tr>
<tr>
<td>Time to ST depression (0.1 mV) (seconds)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>420±60</td>
<td>420±60</td>
<td>420±60</td>
</tr>
<tr>
<td>Captopril</td>
<td>300±60</td>
<td>360±60</td>
<td>480±60*</td>
</tr>
<tr>
<td>RBPP at onset of 0.1 mV ST depression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>165±8</td>
<td>171±8</td>
<td>178±9</td>
</tr>
<tr>
<td>Captopril</td>
<td>152±8</td>
<td>179±9</td>
<td>209±8†</td>
</tr>
</tbody>
</table>

Values are mean±SEM. RBPP, heart rate–blood pressure product (beats×min⁻¹×mm Hg×10⁻²).

* $p=0.055$, †$p<0.01$ compared with baseline.
‡$p<0.01$, between-group comparison.
maximum RBPP and the increase in RBPP from the resting values are shown in Table 4.

The systolic blood pressure rose in the placebo group from 111±2 mm Hg at baseline to 128±3 mm Hg at the completion of the study (p<0.01). In the captopril group, the systolic blood pressure remained unchanged at 112±2 mm Hg without any reflexory increase in heart rate being observed (Table 5).

The working capacity after 30 days was almost identical in the groups, as can be seen in Figure 1. Expressed as endurance, the average working capacity in the captopril group increased from 540±47 seconds to 738±41 seconds (p<0.01), whereas the placebo group showed a slight but insignificant fall in working capacity from 596±43 seconds to 530±43 seconds. At the end of the study, there was a significant between-group difference in favor of captopril (p<0.01). In the captopril group, four patients completed the maximum exercise time of 18 minutes eight times (9.2% of total exercise tests) compared with only one patient in the placebo group completing two times (2.3% of total exercise tests). Of the remaining exercise tests in the captopril group, the reasons for stopping prematurely were the following: exhaustion, 85%; chest pain, 10%; and a combination, 5%. In the placebo group, exhaustion was the reason for stopping prematurely in 65% of the cases, chest pain in 20%, and a combination in 15%.

In the placebo group, a significant increase was found in the EDVI of 16.7% (from 71±3.6 mL/m² to 83±4.5 mL/m², p<0.05) and in the ESVI of 13.7% (from 43.4±2.6 mL/m² to 49.1±3.3 mL/m², p<0.05). In the captopril group, the EDVI remained unchanged (72.9±3.4 mL/m² versus 73.0±3.2 mL/m²), whereas the ESVI in this group was reduced by 13.1% (from 44.9±2.8 mL/m² to 39.0±2.6 mL/m², p<0.05) (Table 5). The changes in ESVI gave rise to a significant between-group difference at days 90 and 180 (p<0.05), whereas the changes in EDVI were only borderline with respect to between-group differences (p=0.07).

Left ventricular volumes and exercise capacity as well as left ventricular volumes and changes in duration of ambulatory ST segment depressions were correlated. The percentage changes in EDVI in all patients from day 30 to day 180 were correlated with the changes in the duration of ST segment depressions detected during the same period (p=0.63, p<0.001, n=58), as can be seen in Figure 2. There was no correlation between left ventricular volumes and working capacity in the two groups during the same period.

In the captopril group, the need for adjuvant diuretic therapy was reduced. At the time of randomization, 34% of the patients in this group required diuretic therapy; this was reduced to 10% by the time the study was completed (p<0.05). The need of furosemide in the placebo group remained unchanged: 34% versus 28% (NS).

Concomitant anti-ischemic therapy remained, on the whole, unchanged during the study period. At day 180, metoprolol was given to 21 patients in both groups, and diltiazem was given to four captopril-treated patients versus seven patients given placebo. The use of isosorbide mononitrate was of the same order of magnitude during the whole of the study period.
The number of cardiac-related events comprising death, reinfarction, or coronary artery bypass graft (CABG) were also in favor of captopril treatment. A total of nine such events (one cardiac death, two CABGs, and six reinfarctions) occurred in the placebo group versus only two (one cardiac death and one CABG) in the captopril group (p<0.05). Side effects leading to a reduction in dosage of the study drug were seen in three patients in the captopril group. These were cough in one and slight dizziness in two.

**Discussion**

Patients having suffered a recent MI are exposed to progressive deterioration in or new manifestations of their coronary disease. Therapy of such patients must take several pathological processes into consideration, of which residual ischemia and left ventricular dysfunction are probably the most important. To date, no single medical treatment has had a positive effect on both factors. Therefore, the object of the present investigation was to determine whether ACE inhibitor intervention has a role in the treatment of these conditions after MI.

The patient material was selected in such a manner that all of the MI patients had left ventricular dysfunction. We observed\(^6\) (as did Pfeffer et al and Sharp et al\(^5\)) a progressive dilation of the left ventricle in patients given placebo. Although no significant between-group difference in EDVI was observed (probably because of the short observation period), this aspect of the remodeling process was prevented in patients treated with captopril. Furthermore, the systolic volume was reduced, leading to an increased stroke volume, indicating an improved contractility and improved systolic function of the left ventricle.

Myocardial ischemia is the result of an imbalance between the oxygen requirement and the supply. As a result of a reduction in the reserve capacity of vasodilation as well as extrinsic compression during the systole combined with an overall higher metabolic rate, the subendocardium is more prone to ischemia than the subepicardium. Therefore, high metabolic demand and/or maldistribution of the transmural flow are responsible for subendocardial ischemia with subsequent ST segment depression.\(^17\),\(^18\) This can be detected by exercise testing and ambulatory ECG monitoring, both of which have a high sensitivity in patients with known coronary artery disease, and can be used for evaluation of the effect of treatment as well as prognosis in post-MI patients.\(^19\)-\(^22\)

ST segment depression (symptomatic and asymptomatic) is relatively frequent during the first days after MI and is observed in 35–82% of patients.\(^23\)-\(^25\) These ST segment depressions within or outside the infarct zone may reflect ischemic episodes indicating stenosis of the coronary arteries or infarct extension after reocclusion.\(^26\) We were not particularly interested in the ischemic changes within the infarct zone itself but only in the residual ischemia in the noninfarcted myocardium. ECG mapping was therefore carried out so that leads showing infarct-related ST segment changes and/or Q waves could be avoided. To enhance the sensitivity of ambulatory ECG monitoring, two separate leads were placed in the area that was presumably unaffected by the infarction. The same procedure was used during exercise testing.

The ambulatory ST segment depression, evaluated as the average duration of the spontaneous ST depression as well as the number of patients with ST depression, was of the same order of magnitude in both groups at baseline. The frequency of ischemia (67%) in our studies was relatively high although within the limits that have been reported earlier\(^23\)-\(^25\) a factor that can be explained by the selection criteria (e.g., left ventricular dysfunction). We observed a significant fall in the duration of ambulatory ischemia in both groups within the first month. The ambulatory ischemia was further reduced in the group treated with captopril in contrast to the placebo group, giving rise to a significant difference after 3 months. As no major changes were made in the adjuvant anti-ischemic treatment, this effect must be ascribed to captopril. ACE inhibitors interfere with the formation of angiotensin II, a vasconstrictor of both systemic and coronary arteries.\(^27\) The effect on the coronary circulation has been shown in experimental studies in which ACE inhibition produced a reduction in infarct size explained by increased coronary perfusion, especially of the ischemic area.\(^28\) The effect on the peripheral circulation causes a reduction in both afterload and preload and thus decreases the myocardial work load. This produces a lessening of the myocardial oxygen demand as well as a fall in blood pressure without a reflexory increase in heart rate, leading to reduced myocardial oxygen consumption.\(^28\) We did not observe a direct blood pressure–lowering effect of captopril, but the expected rise in blood pressure caused by increased stroke volume as seen in the placebo group was prevented. This could be mediated via an increase in arterial compliance.\(^29\) There are a number of mechanisms by which ACE inhibitors influence the myocardial oxygen requirement and supply via their hemodynamic effects.

It appears that the process of myocardial remodeling also contributes to the myocardial oxygen demand inasmuch as we were able to demonstrate a positive correlation between the changes in left ventricular EDVI and duration of ambulatory ST segment depressions from day 30 to day 180. The process of remodeling
comprises dilation of the infarcted segment and hypertrophy of the noninfarcted segment, both of which lead to increased myocardial oxygen demand via increased myocardial work load. Therefore, the fact that patients given placebo developed progressive dilation of the left ventricle can explain why we observed a deterioration in the ambulatory ischemic burden as well as in the exercise-triggered ischemia in this group after the temporary improvement in the early stages after MI. The fact that we observed the effects of captopril treatment gradually over the study period is in agreement with the assumption that the tissue renin–angiotensin system regulates long-term organ function in contrast to the short-term effect of the circulating renin–angiotensin system.

The improvement in working capacity was also in favor of captopril treatment, and this, too, was observed as a gradual improvement. It has been shown that this effect on exercise capacity is correlated to the configuration of the left ventricle. We have been unable to demonstrate this correlation in our study, perhaps because of an asynchronism in the phases of development. However, from our observations, we are able to conclude that as both of these effects are influenced by the treatment, factors other than these such as peripheral hemodynamic changes must also be present. The beneficial effect of captopril on myocardial ischemia and dysfunction after MI is also reflected in clinical improvement inasmuch as the occurrence of cardiac-related events was significantly lower in the captopril group, based on the frequency of reinfarctions. Results from the SAVE trial confirm this protective effect of captopril with a 25% reduction in number of reinfarcts and a cardiovascular mortality reduction of 21%. The reason for this difference could be the beneficial effect on the myocardial oxygen requirement and supply. In addition, captopril has been shown to inhibit platelet aggregation and thereby reduce the release of vasoconstrictors (thromboxane A2) and stimulators of smooth muscle proliferation (platelet-derived growth factor). The area of vascular remodeling, antiatherosclerotic effects, and effects on platelet function and fibrinolytic activity of ACE inhibitors require further elucidation. The need for adjunctive diuretic treatment in the captopril group was significantly reduced; this effect is presumably mediated via an improvement in the function of the left ventricle and/or via a reduction in ischemia-induced dysfunction.

Conclusions

Treatment with captopril of patients with dysfunction of the left ventricle after MI appears advantageous inasmuch as captopril has a positive effect on myocardial ischemia, left ventricular function, and physical performance.

References

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