Ventricular Arrhythmias in the Acute and Chronic Phases After Acute Myocardial Infarction

Effect of Intervention With Captopril

Peter Søgaard, MD; Carl-Otto Gøtzsche, MD; Jan Ravkilde, MD; Aage Nørgaard, MD; Kristian Thygesen, MD, FACC

Background Ventricular arrhythmias (VAs) are independent predictors of mortality in survivors of myocardial infarction (MI), and they are more likely to be induced in dilated hearts with increased wall stress. Angiotensin-converting enzyme (ACE) inhibitors have been shown to prevent progressive dilation of the left ventricle after MI.

Methods and Results The effects of captopril were evaluated in 58 patients with left ventricular (LV) dysfunction after MI. Patients were randomized on day 7 to either placebo or captopril (50 mg daily) in a double-blind parallel study over a period of 6 months. Patients were followed up by means of ambulatory ECG monitoring and echocardiography. There was a significant increase in VA in the placebo group (P<.05) in contrast to a significant decrease in the captopril group (P<.05). As a consequence, there was a significant between-group difference after 6 months (P<.05). Furthermore, the number of patients without VA at baseline who presented with this at the completion of the study was 6% in the captopril group versus 38% in the placebo group (P<.05). At baseline as well as at the termination of the study, LV end-diastolic volume index (LVEDVI) and LV end-systolic volume index (LVESVI) were significantly increased among patients with VA (P<.01). On day 180, both myocardial ischemia and an increase in the LVEDVI were independent predictors of VA; however, progressive dilation of the left ventricle was confined to the placebo patients with significant increases in the LVEDVI compared with the captopril group: 17% versus 0%, respectively (P<.01). Furthermore, the duration of ambulatory ST-segment depression was significantly longer in this group compared with the captopril group (P<.01).

Conclusions Dilation of the left ventricle and myocardial ischemia predict VA during both the acute and chronic phases after MI. In post-MI patients with LV dysfunction, captopril has a beneficial effect on both the number of complex VAs as well as the number of patients who develop VA during the chronic phase. This is in all probability mediated through effects on both LV remodeling, LV function, and myocardial ischemia in patients who are exposed to an increased risk of undergoing progressive dilation of the left ventricle.

(Circulation. 1994;90:101-107.)

Key Words • arrhythmias • remodeling • captopril • ischemia

Experimental and clinical studies indicate that increased myocardial wall stress and loading may induce arrhythmias and that stretch-induced arrhythmias are more likely to occur after ventricular dilation.12-14 Remodeling of the heart in post-MI patients may lead to progressive dilation of the left ventricle, a process that can be prevented by captopril. As a result of this, beneficial effects on myocardial ischemia and cardiovascular morbidity and mortality have been observed.15-18 In addition, load manipulation by captopril as well as enalapril has been shown to reduce the incidence of sudden cardiac death and ventricular tachycardia (VT) in patients with severely reduced LV function.19,20 Captopril also may have an effect on impulse conduction21 and sympathetic tone.22

The aim of the present study was to examine the relation between LV dilation, LV function, and myocardial ischemia compared with the prevalence and severity of VA in post-MI patients with reduced LV function. The effect of captopril on these relations also was assessed.

Methods

Patient Selection

Patients in the present study are from a database from which reports regarding the effect of captopril on post-MI patients...
have been presented previously.17 The database comprises patients with an established diagnosis of MI included if they had an LV ejection fraction (EF) ≤45% on day 5 after MI and were below the age of 70 years.

Patients with a history of congestive heart failure and patients requiring an ACE inhibitor or digoxin were excluded, as were patients with a systolic blood pressure <100 mm Hg, atrial fibrillation, diseases of the heart valves, bundle branch block, heart aneurysm, severe systemic disease, and liver or kidney disease.

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. Patients gave their consent to participate after both oral and written information.

Study Design

The present study was a randomized, placebo-controlled, double-blind parallel study. Patients were randomized on day 7 to receive a blinded test dose of placebo or captopril 6.25 mg. This was followed by placebo or captopril 12.5 mg BID for a period of 2 weeks. After a clinical control, the daily dose of placebo or captopril was increased to 25 mg BID. This medication was continued for the remainder of the trial period, which lasted 180 days.

Measurements

Echocardiography was carried out according to a standardized protocol on day 5 and repeated on days 30, 90, and 180 after the MI. Patients were examined in the left lateral decubitus position, and echocardiographic recordings were obtained at end expiration during normal breathing; an apical four-chamber view was used. The LV end-diastolic volume was measured at the onset of the QRS complex and the end-systolic volume at the end of the T wave using ECG-triggered recordings. The single-plane area-length method was used for calculation of volumes on the screen of the echocardiograph; the mean of three measurements was used. LV end-diastolic volume index (LVEDVI, mL/m²) and LV end-systolic volume index (LVESVI, mL/m²) were derived using the surface area estimated at each time point. The interobserver coefficient of variation (CV%) was 2% and the intraobserver CV% was 1.2% for repeated measurements of consecutive samples.18

Transmitral flow was obtained from the apical four-chamber view between the mitral leaflets by using pulsed-wave Doppler echocardiography (transducer, 2 mHz; sample length, 2 mm). The ratio between early and atrial peak flow velocities (E/PA ratio) was calculated using the mean of five measurements.

Twenty-four hours of calibrated ambulatory ECG monitoring using an amplitude-modulated monitor, Reynolds tracker, was carried out on day 6 after MI and repeated on days 30, 90, and 180. Tapes were replayed under visual observation at 60 times the recording speed on a Reynolds pathfinder 3T connected to a computerized arrhythmia unit, Laserpack. The presence of premature ventricular beats (PVCs) >10×hours⁻¹ was noted, as were couplets and VT, defined as three or more PVCs in a row with a rate >110×min⁻¹.

ST-segment analyses also were performed using the Reynolds ST scope unit. Only horizontal and downsloping ST-segment depression of at least 0.1 mV with a duration of 1 minute measured 80 milliseconds after the J-point was considered a sign of ischemia. All episodes of significant ST-segment depression were printed out for visual evaluation before final acceptance.

The intraobserver day-to-day variability was determined from a random sample of 20 Holter tapes. The intraobserver CV% was 1.2% for the detection of couplets per VT, 3.5% for the detection of PVCs, and 4% for the detection of significant ST-segment depressions.

Additional Medication

Treatment with the thrombolytic agent streptokinase as well as oral treatment during days 0 to 5 with low-dose acetylsalicylic acid, β-blockers, nitrates, calcium antagonists, and diuretics followed the standard of the department. No patient received antiarrhythmic medication other than β-blockade.

β-Blockers were used in 22 patients in both the placebo and captopril groups at baseline and 21 patients in both groups at the termination of the study.

Statistical Analyses

The baseline data 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. The Mantel-Haenszel χ² test was used to calculate rate differences and 95% confidence intervals. CV% was calculated as the relation between the standard deviation and the mean of the observations multiplied by 100%. A value of P<.05 was considered statistically significant.

Results

Captopril vs Placebo

A total of 64 patients were enrolled. Six patients dropped out during the trial period. The reasons for dropout were the following: 2 patients died, 3 patients were subjected to coronary artery bypass grafting, and 1 patient was lost to follow-up. Thus, a total of 58 patients, 29 in each parallel group, completed the trial period, and data presented include only these 58 patients. Baseline clinical and demographic profiles for the randomization groups are listed in Table 1. No significant differences between groups were noted.

A gradual reduction in the number of couplets per VT from 75 to 15 (P<.05) episodes was observed in the captopril group throughout the trial period. This was in contrast to a gradual increase in the number of couplets per VT from 23 to 118 in the placebo group (P<.05). These changes gave rise to a significant between-group difference at the completion of the study (P<.05). The prevalence of VA did not change significantly either within or between groups during the trial period. However, of 17 patients in the captopril group without VA at baseline, 1 had VA at the completion of the study. This is in contrast to 8 patients with VA out of 21 in the placebo group (P<.05) (Table 2).

Characteristics of Patients With VA

The group with ventricular ectopy at baseline consisted of 5 patients with PVC >10×hours⁻¹ and 18 patients with couplets per VT; 3 patients had both PVC >10×hours⁻¹ and couplets per VT; thus, a total of 20 patients had VA at baseline, and the total number of couplets per VT was 98 (Table 2). Throughout the trial period there was a slight increase in both the prevalence and severity of VA in the total population (Table 2). VA was present in 23 patients (40%) of the total population at the completion of the study. Six patients had PVC >10×hours⁻¹ and 22 had couplets per VT; 5 had both PVC >10×hours⁻¹ and couplets per VT, and the total number of couplets per VT was 133 (Table 2).

The demographic and clinical profiles of patients with and without VA are presented in Table 3. There was no significant difference in demographic data. Significant between-group differences were noted regarding clini-
The percentage change in LVEDVI from baseline to day 180 was 15% in patients with VA versus 3% in patients without VA ($P<.01$). The corresponding values were 0% in the captopril group and 17% in the placebo group (Fig 3). Moreover, an increase in LVESVI was observed in 29 patients, of whom 14 had VA (Table 4). Furthermore, the percentage change in LVESVI among patients with VA was 8% versus −5% in patients without VA ($P<.01$), with the corresponding values being −13% in the captopril group versus +14% in the placebo group ($P<.01$) (Fig 3).

The duration of ST-segment depression per day as detected during Holter monitoring at baseline was significantly longer in patients with VA: 40 versus 27 minutes in patients without VA ($P<.05$), as seen in

### Table 1. Demographic and Clinical Profile at Baseline in Placebo and Captopril Groups

<table>
<thead>
<tr>
<th></th>
<th>Placebo n=29</th>
<th>Captopril n=29</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>58±2</td>
<td>60±2</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>27/2</td>
<td>26/3</td>
<td>NS</td>
</tr>
<tr>
<td>Height, cm</td>
<td>176±2</td>
<td>175±2</td>
<td>NS</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>83±4</td>
<td>77±4</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>3 (10%)</td>
<td>4 (14%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>5 (17%)</td>
<td>5 (17%)</td>
<td>NS</td>
</tr>
<tr>
<td>Previous MI</td>
<td>1 (3%)</td>
<td>2 (7%)</td>
<td>NS</td>
</tr>
<tr>
<td>Streptokinase</td>
<td>24 (83%)</td>
<td>23 (79%)</td>
<td>NS</td>
</tr>
<tr>
<td>Aspirin</td>
<td>29 (100%)</td>
<td>29 (100%)</td>
<td>NS</td>
</tr>
<tr>
<td>β-Blocker therapy</td>
<td>22 (76%)</td>
<td>22 (76%)</td>
<td>NS</td>
</tr>
<tr>
<td>ST-depression, min</td>
<td>30±6</td>
<td>32±5</td>
<td>NS</td>
</tr>
<tr>
<td>MI (anterior/Inferior)</td>
<td>16/13</td>
<td>16/13</td>
<td>NS</td>
</tr>
<tr>
<td>Q-wave MI</td>
<td>21 (72%)</td>
<td>20 (69%)</td>
<td>NS</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>16 (55%)</td>
<td>20 (69%)</td>
<td>NS</td>
</tr>
<tr>
<td>PE/PA</td>
<td>1.3±0.1</td>
<td>1.2±0.1</td>
<td>NS</td>
</tr>
<tr>
<td>LVESVI, mL/m²</td>
<td>71±4</td>
<td>73±3</td>
<td>NS</td>
</tr>
<tr>
<td>LVESVI, mL/m²</td>
<td>44±3</td>
<td>45±3</td>
<td>NS</td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction; PE/PA, Doppler E- to A-wave ratio of transmitral flow; LVESVI, left ventricular end-systolic volume index; and LVESVI, left ventricular end-diastolic volume index.

Values are mean±SEM.

### Table 2. Prevalence of Ventricular Arrhythmia and Number of Couplets per Ventricular Tachycardia During Trial Period

<table>
<thead>
<tr>
<th>Study Group n=58</th>
<th>Captopril n=29</th>
<th>Placebo n=29</th>
</tr>
</thead>
<tbody>
<tr>
<td>Couplets per VT baseline</td>
<td>98</td>
<td>75</td>
</tr>
<tr>
<td>Couplets per VT on day 30</td>
<td>108</td>
<td>60</td>
</tr>
<tr>
<td>Couplets per VT on day 90</td>
<td>129</td>
<td>29</td>
</tr>
<tr>
<td>Couplets per VT on day 180</td>
<td>133</td>
<td>15*</td>
</tr>
<tr>
<td>No. of patients with VA at baseline</td>
<td>20 (34%)</td>
<td>12 (41%)</td>
</tr>
<tr>
<td>No. of patients with VA on day 30</td>
<td>20 (34%)</td>
<td>11 (38%)</td>
</tr>
<tr>
<td>No. of patients with VA on day 90</td>
<td>22 (38%)</td>
<td>9 (31%)</td>
</tr>
<tr>
<td>No. of patients with VA on day 180</td>
<td>23 (40%)</td>
<td>9 (31%)</td>
</tr>
<tr>
<td>No. of patients without VA at baseline who presented with VA on day 180</td>
<td>9 (24%)</td>
<td>1 (6%§)</td>
</tr>
</tbody>
</table>

VT indicates ventricular tachycardia; VA, ventricular arrhythmia.

*P<.05 in comparison with inclusion; †P<.05 between groups; §1 of 17 in the captopril group; §8 of 21 in the placebo group.
Table 3. On day 180, the duration of ambulatory ST-segment depression was 18 minutes per day in patients with VA versus 9 minutes per day in patients without VA (P<.05), with the corresponding values being 3 minutes per day in the captopril group versus 22 minutes per day in the placebo group (Fig 3). Myocardial ischemia was present in 29 patients on day 180, of whom 19 had VA (Table 4). Finally, the combination of an increase in both LVEDVI and myocardial ischemia was present in 22 patients on day 180, 18 of whom had VA (Table 4).

There was a drop in the PE/PA ratio among patients with VA from 1.2 to 0.8 (P<.05). This was significantly lower than in patients without VA, ie, 1.3 versus 0.8 (P<.05) (Table 5). The use of β-blockers at baseline was less frequent in the VA group: 11 patients (55%) versus 33 patients (87%) (P<.05) (Table 3). On day 180, β-blockers were still used less frequently, although not statistically significant, in patients with VA, ie, 13 (56%) versus 29 (83%) (P<.06). Finally, on day 180, Q-wave MIs were more frequent in patients with VA, ie, 20 (87%) versus 21 (60%) (P<.05) (Table 5).

Table 3. Demographic and Clinical Profile at Baseline of Patients With and Without Ventricular Arrhythmia

<table>
<thead>
<tr>
<th></th>
<th>VA Present n=20</th>
<th>VA Absent n=38</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>61±4</td>
<td>57±3</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>18/2</td>
<td>35/3</td>
<td>NS</td>
</tr>
<tr>
<td>Height, cm</td>
<td>179±2</td>
<td>174±2</td>
<td>NS</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>81±4</td>
<td>77±3</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2 (10%)</td>
<td>5 (13%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4 (20%)</td>
<td>6 (16%)</td>
<td>NS</td>
</tr>
<tr>
<td>Previous MI</td>
<td>1 (5%)</td>
<td>2 (5%)</td>
<td>NS</td>
</tr>
<tr>
<td>Streptokinase</td>
<td>16 (80%)</td>
<td>31 (82%)</td>
<td>NS</td>
</tr>
<tr>
<td>Aspirin</td>
<td>20 (100%)</td>
<td>38 (100%)</td>
<td>NS</td>
</tr>
<tr>
<td>β-Blocker therapy</td>
<td>11 (55%)</td>
<td>33 (87%)</td>
<td>.02</td>
</tr>
<tr>
<td>ST-depression, min</td>
<td>40±8</td>
<td>27±5</td>
<td>.03</td>
</tr>
<tr>
<td>MI (anterior/inferior)</td>
<td>11/9</td>
<td>21/17</td>
<td>NS</td>
</tr>
<tr>
<td>Q-wave MI</td>
<td>15 (75%)</td>
<td>26 (68%)</td>
<td>NS</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>13 (65%)</td>
<td>23 (61%)</td>
<td>NS</td>
</tr>
<tr>
<td>PE/PA</td>
<td>1.2±0.1</td>
<td>1.3±0.1</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDVI, ml/m²</td>
<td>80±5</td>
<td>68±3</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>LVESVI, ml/m²</td>
<td>52±3</td>
<td>39±3</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction; PE/PA, Doppler E- to A-wave ratio of transmitral flow; LVEDVI, left ventricular end-diastolic volume index; and LVESVI, left ventricular end-systolic volume index. Values are mean±SEM.

Fig 1. Bar graph: Left ventricular end-diastolic volume index (LVEDVI, ml/m²) in relation to ventricular arrhythmia during follow-up. Hatched bars represent patients with ventricular arrhythmias; open bars, patients without ventricular arrhythmias. *P<.01 between groups. **P<.05 compared with inclusion.

Fig 2. Bar graph: Left ventricular end-systolic volume index (LVESVI, ml/m²) in relation to ventricular arrhythmia during follow-up. Hatched bars represent patients with ventricular arrhythmias; open bars, patients without ventricular arrhythmias. *P<.01 between groups.
TABLE 4. Predictors of Ventricular Arrhythmia on Day 180

<table>
<thead>
<tr>
<th>Patients With Ventricular Arrhythmia</th>
<th>Rate Difference (95% Confidence Intervals)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase in LVESVI (n=29)</td>
<td>14</td>
<td>0.17 (-0.08 to 0.42)</td>
</tr>
<tr>
<td>Increase in LVEDVI (n=37)</td>
<td>21</td>
<td>0.47 (0.27 to 0.68)</td>
</tr>
<tr>
<td>ST-segment depression (n=29)</td>
<td>19</td>
<td>0.52 (0.3 to 0.73)</td>
</tr>
<tr>
<td>Increase in LVEDVI and ST-segment depression (n=22)</td>
<td>18</td>
<td>0.68 (0.48 to 0.88)</td>
</tr>
</tbody>
</table>

LVESVI indicates left ventricular end-systolic volume index; LVEDVI, left ventricular end-diastolic volume index.

Discussion

VAs in the Post-MI Patient

Frequent and complex VAs are an independent risk factor for cardiac mortality after MI, and their impact on risk increases when combined with reduced LV function and/or myocardial ischemia. There is growing evidence that increased myocardial wall stress and ventricular loading play an important role in the genesis of arrhythmias associated with reduced LV function, especially in combination with increased LV volume. The great majority of VAs in hearts with healed myocardial infarction are due to reentry, with the anatomic substrate being isolated bundles of surviving myocardial fibers within the infarct zone. Several possible mechanisms could underlie this arrhythmogenesis, including increased cardiac sympathetic nerve stimulation leading to arrhythmic events and mortality in post-MI patients.

Holter monitoring discloses VA in 50% to 80% of post-MI survivors just before hospital discharge. The varying prevalence reflects differences in definition criteria as well as different durations of Holter monitoring, with the prevalence increasing after longer duration of Holter monitoring. Complex VAs are independent predictors of both sudden and total cardiac death in all of the cited studies. However, all of these results stem from the prethrombolytic era. Recent reports suggest that thrombolytic therapy markedly reduces the prevalence of VA. Furthermore, our study confirms previously reported beneficial acute and long-term effects of β-blockade because the use of β-blockers in the present study was more frequent among patients without VA. The frequent use of streptokinase (82% of patients) and β-blockers (76% of patients) in our patient population may explain the low prevalence of VA during both the acute and follow-up phases.

The need for a long-lasting Holter monitoring has been emphasized because of the considerable day-to-day variation in ventricular ectopy. Only 24-hour Holter monitoring was performed in the present study. However, Kennedy et al demonstrated that during 48 hours of monitoring in patients with a recent MI, the maximum Lown grading occurred within the first 24 hours in approximately 80% of the patients. Thus, it appears that 24 hours of monitoring is satisfactory to detect VA in individual monitorings in long-term studies. Moreover, frequent monitorings were performed, i.e., four times during 180 days in the present study. This would further strengthen observations regarding the development in prevalence and severity of VA during the course of the trial period.
ACE Inhibitors in Post-MI Patients

With LV Dysfunction

ACE inhibition has been shown to be beneficial in post-MI patients with reduced LV function. Progressive dilation of the left ventricle and diastolic dysfunction are prevented, and LV systolic function is improved. These changes might prevent mechanically induced VA. Furthermore, ACE inhibition has been shown to reduce myocardial ischemia in patients with LV dysfunction, in part mediated by prevention of myocardial remodeling and LV dilation. This anti-ischemic effect might be beneficial in reducing the prevalence of VA inasmuch as different anti-ischemic drugs have been reported to reduce the prevalence of VA in post-MI patients.

LV Volume and Myocardial Ischemia

in Relation to VA

The development in the number of complex VAs throughout the trial period supports the overall conclusion reached that addition of captopril had a beneficial effect on the occurrence of VA. Although the use of β-blockade was frequent, with a presumably lower prevalence of VA, it appeared that the addition of captopril had a beneficial effect on the number of complex VAs during the course of the follow-up period. In the placebo group, in which β-blockade was used to the same extent, this could not prevent a deterioration, as expressed in the significant increase in complex VAs observed in this group. The latter was primarily the result of a significantly higher number of patients in the placebo group who developed VA during the trial period as compared with the captopril group. Several possible mechanisms could be responsible for this, including changes in both LV volume and myocardial ischemia.

The present study shows that VAs are more easily induced in patients with a dilated left ventricle, as demonstrated by the significant difference in LVEDVI throughout the trial period in the VA group. In addition, changes in LV wall stress, as indicated by percent change in LVEDVI during follow-up, were a predictor of VA at the termination of the study. Finally, diastolic dysfunction as reflected by PE/PA ratio also was present in patients with VA. The progressive dilation of the left ventricle observed in the placebo group could have been responsible for the increasing number and severity of VAs observed in this group compared with the captopril group, in which LV dilation was prevented. The fact that VAs were more frequent among patients with Q-wave MI is not surprising because remodeling of the left ventricle is known to be restricted to Q-wave MI survivors.

Myocardial ischemia was a predictor of VA in the present study. The prediction of VA is even more powerful if one combines both increases in LVEDVI and myocardial ischemia. Given the previously demonstrated relation between myocardial remodeling and duration of myocardial ischemia, it is foreseeable that captopril had an effect on VA in this post-MI population.

An increase in LVESVI was not a predictor of VA during follow-up in the present study. We cannot explain this. However, there was a considerable between-group difference, with a significant reduction in LVESVI in the captopril group, and correspondingly, LVESVI was reduced in patients without VA on day 180 in contrast to an increase in LVESVI in patients who presented with VA on day 180. Thus, part of the effect of captopril could be mediated by a beneficial effect on systolic function. The effect on LVESVI is of particular interest because this variable has been shown to be a powerful predictor of prognosis in post-MI patients. It is possible that LVESVI reflects both the extent of dilation as well as systolic function of the left ventricle.

Conclusions

Dilation of the left ventricle and myocardial ischemia predict VA during both the acute and chronic phases after MI. In post-MI patients with LV dysfunction, captopril has a beneficial effect on both the number of complex VAs as well as the number of patients who develop VA during the chronic phase. This is in all probability mediated through effects on LV remodeling, LV function, and myocardial ischemia in patients who are exposed to an increased risk of undergoing progressive dilation of the left ventricle.

Acknowledgments

Peter Søgaard is a senior research fellow supported by a grant from the Danish Heart Foundation.

References


Ventricular arrhythmias in the acute and chronic phases after acute myocardial infarction. Effect of intervention with captopril.

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Circulation. 1994;90:101-107
doi: 10.1161/01.CIR.90.1.101

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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