Randomized Evaluation of the Effects of Filter-Based Distal Protection on Myocardial Perfusion and Infarct Size After Primary Percutaneous Catheter Intervention in Myocardial Infarction With and Without ST-Segment Elevation

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Background—In acute myocardial infarction, distal embolization of debris during primary percutaneous catheter intervention may curtail microvascular reperfusion of the infarct region. Our randomized trial investigated whether distal protection with a filter device can improve microvascular perfusion and reduce infarct size after primary percutaneous catheter intervention.

Methods and Results—We enrolled 200 patients who had angina within 48 hours after onset of pain plus at least 1 of 3 additional criteria: ST-segment elevation, elevated myocardial marker proteins, and angiographic evidence of thrombotic occlusion. Among the patients included (83% men; mean age, 62±12 years), 100 were randomly assigned to the filter-wire group and 100 to the control group. The primary end point was the maximal adenosine-induced Doppler flow velocity in the recanalized infarct artery; the secondary end point was infarct size estimated by the volume of delayed enhancement on nuclear MRI. ST-segment elevation myocardial infarction was present in 68.5% of the patients; the median time from onset of pain was 6.9 hours. In the filter-wire group, maximal adenosine-induced flow velocity was 34±17 compared with 36±20 cm/s in the control group (P=0.46). Infarct sizes, assessed in 82 patients in the filter-wire group and 78 patients in the control group, were 11.8±9.3% of the left ventricular mass in the filter-wire group and 10.4±9.4% in the control group (P=0.33). Thirty-day mortality was 2% in filter-wire group and 3% in the control group.

Conclusions—The filter wire as an adjunct to primary percutaneous catheter intervention in myocardial infarction with and without ST-segment elevation did not improve reperfusion or reduce infarct size. (Circulation. 2005;112:1462-1469.)

Key Words: catheters ■ embolism ■ infarction ■ reperfusion ■ stents
We designed the randomized controlled Protection Devices in PCI Treatment of Myocardial Infarction for Salvage of Endangered Myocardium (PROMISE) study to evaluate the effects of the FilterWire-EX (Boston Scientific) on myocardial perfusion as determined by the maximal blood flow velocity across the recanalized infarct-related artery. We thus intended to test the pivotal mechanism for a potential clinical benefit of the filter wire as an adjunct to primary PCI in myocardial infarction with and without ST-segment elevation. In addition, we assessed infarct sizes by nuclear MRI.

**Methods**

**Study Cohort**

The study included patients with myocardial infarction with and without ST-segment elevation undergoing recanalization by stent placement within 48 hours after onset of pain. Inclusion criteria were (1) at least 1 episode of typical anginal pain lasting >30 minutes within the preceding 48 hours, (2) coronary artery lesion deemed suitable for stent placement and application of the filter wire, (3) ST-segment elevation of at least 1 mm in ≥2 contiguous leads, (4) elevation in creatine kinase to at least 3 times the upper limit of normal with a concomitant rise in MB isoenzyme, and (5) coronary artery occlusion with angiographic appearance of fresh thrombus. We recruited patients who met criteria 1 and 2 plus at least 1 of criteria 3 through 5. Major exclusion criteria were the following: presumed distal vessel size <3.0 mm; relevant coronary left main involvement; vessel anatomy interfering with a safe placement of the filter wire (eg, extreme tortuosity or heavily calcified vessel proximal to culprit lesion); culprit lesion in saphenous vein graft; contraindications to abciximab, aspirin, clopidogrel, or heparin; mechanical ventilation or inotropic support; and inability to give informed consent.

The study, carried out according to the Declaration of Helsinki, was approved by our institutional ethics review board. All patients gave written informed consent before enrollment.

**Study Protocol**

Immediately after the diagnostic angiography, we allocated eligible patients to the filter-wire or control group using a computergenerated random sequence. The randomization sequence was set in blocks of 20. The size of the block and the random sequence were selected by the statistician and were unknown to the investigators and medical staff caring for the patients.

In patients allocated to the filter-wire group, the culprit lesion was crossed with a FilterWire EX (Boston Scientific), and the filter was deployed at the closest distance to the culprit lesion that allowed safe predilation and stent placement. Details of the design and the deployment technique of the filter have been described previously.16,18 If needed for safe deployment of the filter wire, the use of a 1.5-mm balloon was allowed. The filter wire was left in place until the PCI procedure was completed. After retrieval of the filter wire, we inspected the filters visually. In addition, 10 randomly selected deployed filters from the initial series were fixed in 4% formaldehyde (pH 7.0), embedded in paraffin, and processed with hematoxylin and van Gieson staining, as described previously.19

In both groups, PCI was performed with bare metal stent placement as described previously.20 Peri-interventionally, all patients received intravenous aspirin 300 mg, abciximab in a bolus of 25 mg/kg body weight followed by continuous infusion of 10 μg/min for 12 hours, unfractionated heparin 100 U/kg, and a loading dose of clopidogrel 600 mg. In addition, we gave an intracoronary bolus dose of 0.2 mg nitroglycerine in both groups immediately after reestablishing antegrade flow.

After completion of PCI and withdrawal of the filter wire, coronary flow velocities in the stented segment were measured with the Doppler FlowWire and analyzed by the FloMap system (Volcano Therapeutics Inc) as described previously.4 We positioned the tip of the Doppler wire at the proximal end of the stented segment to ensure a reproducible sampling site that is not subject to vasomotor changes. Doppler flow velocity spectra were analyzed online to determine time-averaged peak velocity (APV). We determined basal and peak coronary flow velocities after intracoronary bolus doses of adenosine 40 μg.21

**MRI** to assess infarct sizes was scheduled on day 3,22–24. As reviewed recently,25 MRI scanning is an established method to assess infarct sizes and has been validated against the previous gold standard of Tc-99m-sestamibi scanning.23 Each patient was placed supine in a 1.5-T clinical scanner (Magnetom Sonata, Siemens), and a phased-array receiver coil was positioned on the chest for imaging. All images were acquired with breathhold for 8 to 15 seconds and were gated on the ECG. Cine images (true fast imaging with steady-state free precession) were acquired in 3 long-axis and 7 to 9 short-axis views, 8 mm in thickness with a 2 mm interslice gap, which achieved full left ventricular (LV) coverage. Gadolinium-based contrast agent (Omniscan, Nycomed Amersham) was then administered intravenously at a dose of 0.2 mmol/kg body weight. After a 1.5-minute delay, contrast-enhanced images were acquired in the same views as those used for cine MRI. A segmented inversion-recovery sequence (fast low-angle shot) was used.22 The inversion time was meticulously adjusted to obtain maximal nulling of remote normal LV myocardium. Areas of increased signal intensity (“delayed enhancement”) represent myocardial scar or necrosis.22

During the hospital stay, creatine kinase was routinely checked every 8 hours until it returned to normal or at least for the first 24 hours and daily thereafter.

**Study End Points, MRI Postprocessing Analysis, Measures of Clinical Outcome, and Quantitative Angiography**

The primary end point was the maximal adenosine-induced APV in the recanalized infarct-related artery. We also measured basal APV and calculated the coronary flow reserve in the recanalized infarct-related artery. In addition, we assessed myocardial blush grade and TIMI flow grade after PCI as semiquantitative variables of coronary perfusion. Angiographically apparent distal embolization was also documented in both groups.

The secondary end point was infarct size, assessed by the volume of area with delayed enhancement on MRI and expressed as percentage of LV mass.26 We also calculated the global LV ejection fraction from the MRI scans.26 For these analyses, all MRI images were examined by 2 experienced observers who were unaware of the patient’s study group assignment. For global LV analysis, all short-axis slices from the apex to the base were assessed with a semiautomatic analysis software (ARGUS, Siemens). By planimetry of all short-axis views, we determined LV end-systolic volume (in milliliters), LV end-diastolic volume (in milliliters), ejection fraction (in percent), and LV mass (in grams). Contrast-enhanced images were analyzed in corresponding short-axis views except for the first apical view, which can be affected by partial-volume effects. The true apical segment was carefully assessed on at least 2 long-axis views. Hyperenhanced regions were quantified by planimetry on each of the short-axis images and multiplied by the slice thickness. All hyperenhanced slice volumes were added to calculate the infarct volume (delayed enhancement volume in milliliters). Infarct size was expressed as percentage of LV mass assuming myocardial density of 1.05 g/mL.

In patients with reinfarction who showed >1 distinct region of delayed hyperenhancement, we assigned 1 region to the recent infarction based on ECG and angiographic criteria. The size of this region was entered into the database for further analysis. If there was only 1 distinct region, the entire region was entered into the database.

We monitored major adverse cardiac events, including death, nonfatal reinfarction, and target lesion revascularization. For the diagnosis of myocardial infarction, we applied the ESC/ACC consensus criteria as reinforced by the AHA.27-28 Diagnosis of recurrent infarction was based on typical chest pain, new ST-segment changes,
and an increase in creatine kinase of at least 50% over the previous trough level in at least 2 samples.

Angiographic images were stored on our clinical database and analyzed offline by the independent angiographic core laboratory at our institution.29 TIMI flow grades and myocardial blush were assessed visually by applying published definitions.13,30 As in previous studies,31,32 our core laboratory assessed myocardial blush as a dichotomized variable (0/1 and 2/3). A convex filling defect partially or completely obstructing a coronary vessel distal to the culprit lesion was taken as evidence of distal embolization. Quantitative analysis was performed as described previously.8,29,33 We obtained minimal luminal diameter, reference diameter, percent diameter stenosis, and diameter of the maximally inflated balloon from the analysis system (MEDIS Medical Imaging Systems). Acute gain was calculated as the difference between poststenting and predilatation minimal luminal diameter. As procedural time, we monitored the time that the patient spent in the catheterization laboratory.

Statistical Analysis

According to our previous experience,8 we assumed a maximal APV of 41±15 cm/s in the control group. We designed our study to have a power of β=80% to detect an improvement in maximal APV by 15% with a level of significance of α<0.05. These assumptions yielded a sample size of 95 patients in each treatment arm. To account for potential dropouts, we intended to include 100 patients in each treatment arm.

All data were analyzed on an intention-to-treat basis. Discrete variables are reported as counts and continuous variables, mean±SD, or median and interquartile range. To test differences between treatment groups, we used Fisher’s exact test or the χ² test, as appropriate, for discrete variables and 1-way ANOVA for continuous variables. Some analyses of continuous variables were corroborated by nonparametric testing with the Mann-Whitney U test. For a prespecified secondary analysis, we used the general linear model to adjust observed differences in the primary end point for age, gender, and pertinent infarct-related variables, including time from onset of pain to PCI, presence or absence of ST-segment elevation, TIMI flow grade before intervention, and location of culprit lesion (left anterior descending coronary artery versus other). The same models were used to assess the interaction of the study treatment with the time from onset of pain to PCI with respect to our primary end point. We also analyzed differences in the primary end point in subgroups defined by these infarct-related variables. For all statistical analyses, we used the SPSS software package, version 11.0. A value of P<0.05 in the 2-tailed test was regarded as significant.

Results

Study Cohort and Angiographic Outcome

The study enrolled 200 consecutive patients; 100 were assigned to the filter wire and 100 to usual care. With respect to baseline demographic, clinical, and angiographic characteristics (Tables 1 and 2), there were no significant differences between the study groups. ST-segment elevation myocardial infarction was present in 68.5% of the patients, and

| TABLE 1. Baseline Demographic, Clinical, and Angiographic Characteristics of the Study Population |
|-----------------------------------------------|------------------|------------------|
| Filter Wire (n=100) | Usual Care (n=100) | P          |
| Age, y             | 62.9±11.3        | 60.2±13.0       | 0.12 |
| Women, n           | 14               | 20              | 0.26 |
| Smoker, n          | 35               | 35              | 1.00 |
| Arterial hypertension, n | 65              | 69              | 0.55 |
| Diabetes mellitus, n | 21              | 26              | 0.40 |
| Total cholesterol, mg/dL | 206±51          | 205±39          | 0.89 |
| LDL cholesterol, mg/dL | 138±44          | 137±37          | 0.95 |
| Previous CABG, n   | 1                | 2               | 0.56 |
| Previous PCI, n    | 6                | 7               | 0.77 |
| Previous myocardial infarction, n | 12             | 17              | 0.32 |
| ST-segment–elevation myocardial infarction,* n | 66              | 71              | 0.45 |
| Door to balloon time, min | 62 (42–92)      | 67 (40–91)      | 0.83 |
| In patients presenting within 6 h, min | 57 (37–74)      | 65 (39–85)      | 0.38 |
| Pain to admission time, h | 4.9 (2.8–10.2)  | 6.6 (3.5–13.8)  | 0.20 |
| Pain to balloon time, h | 6.2 (3.5–12.1)  | 7.9 (4.6–15.6)  | 0.16 |
| Distribution of pain to balloon time, h |                    | 0.26 |
| <6                | 46               | 37              |
| 6–12              | 29               | 28              |
| >12–48            | 25               | 35              |
| Killip class       |                    | 0.50 |
| I                 | 25               | 22              |
| II                | 64               | 69              |
| III               | 9                | 9               |
| IV                | 2                | 0               |

Data are expressed as mean±SD, median (interquartile range), or number of patients.

*Including 1 patient with new left bundle-branch block in each group.
Coronary Perfusion

The filter wire was placed successfully in 95 patients. Predilatation with an undersized balloon was needed in 42 patients. Despite this, we could not advance the device in 5 patients because of vessel tortuosity and/or calcification. We obtained flow velocity measurements in all patients. The primary end point of the study, maximal APV in the recanalized infarct-related artery (Figure 1), was not significantly different between the 2 study groups (P=0.46); it reached 34±17 cm/s in the filter-wire group and 36±20 cm/s in the control group. The mean difference in maximal APV was −1.9 cm/s (95% CI, −7.2 to 3.3; P=0.46) without adjustment and −1.3 cm/s (95% CI, −10.0 to 7.5; P=0.78) with adjustment for pertinent factors and covariables. By nonparametric analysis, we found median maximal APV of 30 cm/s (interquartile range, 23 to 42 cm/s) in the filter-wire group and of 32 cm/s (interquartile range, 21 to 45 cm/s) in the control group (P=0.76). We did not find any significant difference in maximal APV between the filter-wire group and control groups in either ST-segment elevation myocardial infarction (mean APV, 33±16 versus 35±20 cm/s; P=0.53; median APV, 29 cm/s [interquartile range, 20 to 41 cm/s] versus 28 cm/s [interquartile range, 21 to 44 cm/s]; P=0.94) or non–ST-segment elevation myocardial infarction (mean APV, 37±20 versus 38±19 cm/s; P=0.79; median APV, 31 cm/s [interquartile range, 25 to 45 cm/s] versus 35 cm/s [interquartile range, 25 to 50 cm/s]; P=0.70).

As shown in Table 3, differences in maximal APV between the treatment strategies were consistent in the entire cohort.
Causes for missing MRI scans did not differ significantly in 4, patient refusal in 19, and logistical problems in 15. The MRI scans were death in 2 patients, hemodynamic instability in the control group, underwent MRI. Reasons for missing

One hundred sixty patients, 78 in the filter-wire group and 82

Within 12 hours after onset of pain, maximal APV was 33±14 cm/s in the 56 patients in the filter-wire group and 37±20 cm/s in the 44 patients in the control group (P=0.23). The interaction between time from onset of pain to PCI and the study treatment with respect to maximal APV was insignificant (P=0.94).

Similar to maximal APV, the point estimate for coronary flow reserve (Figure 1) was slightly lower in the filter-wire group than in the control group (1.85±0.64 versus 1.94±0.70), but this difference also did not reach statistical significance (P=0.37). Although the filter wire reduced the incidence of angiographically visible distal embolization by trend compared with usual care (3 versus 8; P=0.12), we did not find any benefit of the filter wire with respect to TIMI flow grade or myocardial blush grade (Table 4). Visual inspection revealed captured debris in one third of the filters, and we found plaque material in 6 of 10 filters examined histologically.

Infarct Sizes

One hundred sixty patients, 78 in the filter-wire group and 82

In our randomized controlled trial of patients undergoing primary PCI for myocardial infarction with and without

Consistent results were obtained by distinct analysis of ST-segment elevation myocardial infarction (mean infarct size, 13.3±9.2% versus 12.2±9.9%; P=0.53; median infarct size, 14.5% [interquartile range, 5.8% to 14.4%] versus 11.0% [interquartile range, 3.6% to 20.2%]; P=0.48) and non–ST-segment elevation myocardial infarction (mean infarct size, 7.3±8.3% versus 6.0±6.3%; P=0.58; median infarct size, 5.1% [interquartile range, 0.0% to 12.4%] versus 3.5% [interquartile range, 0.6% to 10.4%]; P=1.0).

In both groups, global ejection fraction was slightly depressed on average, with no significant differences between the 2 groups (Figure 2). Consistent with the findings on MRI, peak creatine kinase was elevated to 11±10 times the upper limit of normal in the filter-wire group and to 9±8 times the upper limit of normal in the control group (P=0.13).

Considering important subgroups, mean infarct size was 13.7±9.2% of LV mass (median, 14.7%; interquartile range, 6.4% to 19.1%) in the 47 patients of filter-wire group presenting with ST-elevation myocardial infarction within 12 hours after onset of pain and 12.1±9.5% of LV mass (median, 11.2%; interquartile range, 3.7% to 20.5%) in the corresponding 40 patients in the control group (P=0.41; P=0.45). There were no appreciable differences in infarct size between the 2 groups in any of the other subgroups examined (Table 5).

Clinical Outcome

Thirty-day follow-up data were available for all patients. By 30 days, 2 patients in the filter-wire group and 3 patients in the control group were dead (P=1.0). This includes 1 patient in the control group who underwent CABG on day 2 for extensive coronary artery disease. There were no further revascularization procedures, and none of the patients experienced recurrent myocardial infarction, stroke, or stent thromboses within 30 days.

Discussion

In our randomized controlled trial of patients undergoing primary PCI for myocardial infarction with and without
ST-segment elevation, distal protection with the filter wire did not improve reperfusion, nor did it reduce infarct size. Failure to improve reperfusion was evidenced both by quantitative flow velocity measurements in the recanalized infarct-related artery and by the more qualitative variables such as myocardial blush grade and TIMI flow grade. Consistent with the findings on flow, the area of delayed enhancement on MRI, global LV ejection fraction, and peak CK release did not reveal any beneficial effect of the filter wire on infarct size.

Contrary to TIMI flow grade, myocardial blush grade, corrected TIMI frame counts and ST-segment resolution, coronary flow velocity under maximal vasodilation, our primary end point, is an objective quantitative measure of perfusion that, in the absence of coronary artery stenoses, reflects the integrity of the distal vascular bed. In a previous study, coronary flow velocity under maximal vasodilation proved to be a sensitive parameter for the microcirculatory effects of abciximab that positively affected recovery of contractile function. Even with this sensitive parameter of myocardial perfusion, we were unable to find a beneficial effect of the filter wire on coronary circulation during reperfusion. Our study also did not reveal any reduction in infarct size or improvement in global LV function with the filter wire compared with usual care. This finding is not surprising because of earlier studies that independently showed a close relation of coronary Doppler flow velocity patterns to functional recovery of reperfused myocardium.

The capability of the filter wire to capture emboli has been demonstrated repeatedly from the early experience up to the present study. In a large randomized study on distal protection during PCI in saphenous vein grafts, the filter wire was as effective in preventing distal embolization and perointerventional infarction as the established reference treatment, distal balloon occlusion and aspiration (GuardWire, Metronic). The safety and feasibility of the filter wire in acute myocardial infarction were recently confirmed by Limbruno and coworkers. When a filter device is used in acute myocardial infarction, the possibility has to be considered that, in a vessel with a number of side branches, the device may simply shift the path of embolic material down the branches despite adequate use of the filter. Moreover, material may be embolized during crossing of the device or predilatation, when needed. Nevertheless, in the study of Limbruno and coworkers, captured debris was found in every filter that was examined microscopically, and macroscopic particles were present in 34% of the filters. Angiographically visible distal embolization occurred in only 2% of patients with filter-wire protection but in 15% of a matched case-control group ($P=0.03$). In our study, the filter wire was similarly effective in the prevention of distal embolization.

Therefore, the ineffectiveness of the filter device with respect to the prevention of distal embolization is not a plausible explanation for the findings of our study. It appears more likely that the distal embolization during primary PCI that can be prevented by protection devices exerts only minor effects compared with the consequences of ischemic microvascular damage or spontaneous distal embolization arising from ruptured or eroded plaques during the natural course of an acute coronary syndrome. This inference is supported by the Enhanced Myocardial Efficacy and Recovery by Aspiration of Liberalized Debris (EMERALD) study on an alternative distal protection system, distal balloon occlusion and aspiration (GuardWire). EMERALD randomized 501 patients to distal protection with balloon occlusion and aspiration or usual care. Visible debris that otherwise would have entered the distal circulation could be removed in 73% of the patients of the study group. Nevertheless, neither the primary end point of ST-segment resolution nor any of the secondary end points, including myocardial blush or adverse cardiac events, showed any significant benefit of distal protection compared with usual care. Although we cannot definitely exclude that novel devices might still offer benefit during primary PCI, the consistent results of EMERALD and PROMISE suggest that, regardless of the technology, distal protection does not improve reperfusion after primary PCI in myocardial infarction.

Our study included an unselected cohort of patients presenting with myocardial infarction. Whereas the presence or absence of ST-segment elevation and the time from onset of pain are critical to the eligibility for thrombolysis, these characteristics have less impact on clinical decision making with primary PCI. Moreover, regardless of these characteristics, patients undergoing primary PCI can expect similar cardiac events, showed any significant benefit of distal protection compared with usual care. Although we cannot definitely exclude that novel devices might still offer benefit during primary PCI, the consistent results of EMERALD and PROMISE suggest that, regardless of the type of ST-segment displacement, time of presentation, and infarct location. The effect of the filter wire remained insignificant after adjustment for pertinent infarct characteristics. None of the subgroups analyzed showed a noteworthy trend toward a beneficial effect of the filter wire.

Our findings on myocardial perfusion are at conflict with those of the earlier study by Limbruno and coworkers. In that study, the filter wire was used in 53 patients. Compared with a matched case-control group, the filter wire improved ST-segment resolution and corrected TIMI frame count significantly. These findings, however, have to be interpreted cautiously because of the nonrandomized design of the
Study with a limited sample size, the use of surrogate markers of perfusion, and an unusually low rate of postinterventional TIMI grade 3 flow (85%) in the control group.16

Study Limitations

The fact that physicians could not be blinded to the assignment of treatment is a limitation of our study. Consequently, bias on the part of the investigators cannot be fully excluded as a factor influencing clinical treatment. However, flow velocities were ascertained by automated computerized evaluation, and the MRI evaluations were performed by blinded operators. Bias in the assessment of the primary and secondary end points can thus be excluded.

In some patients, we could not obtain MRI scans. This problem, noted in previous studies,8 affected both study groups to the same extent. Moreover, the distribution of the causes for missing studies was similar in both groups. Therefore, we cannot assume a relevant distortion of the secondary end point by missing studies.

Given the sample size of the trial, we had low power to look at important subgroups, including those presenting very early and those presenting with ST-segment elevation and non–ST-segment elevation myocardial infarction. For the entire cohort, we had an 80% power to detect a 15% difference in maximal APV, our primary end point. In our earlier study with essentially the same selection criteria as used in the present trial, this magnitude of difference in APV induced by abciximab was associated with a significant difference in reconvalescent wall motion and global LV ejection fraction.

Concerning our secondary end point, the power to detect a 30% difference in infarct size was 75%. The study was not designed to assess clinical end points. Therefore, a clinical benefit of distal protection cannot be excluded. On the other hand, the present findings do not give plausible reason to expect such an effect, given that the improvement in reperfusion is the presumed underlying mechanism for the intended clinical benefit by distal protection.

In patients presenting within 6 hours after the onset of pain, the results are statistically consistent with a benefit of the filter wire, but that finding needs confirmation in a larger cohort of patients. Although in our multivariable analyses neither time from onset of pain nor the interaction term (time from onset of pain by treatment group) was significantly associated with outcome, we cannot exclude that time to treatment is influencing the main findings of this study. With longer time from the onset of pain, more thrombus material that might have already been embolized distally is going to affect the microvasculature.

Utility of a filter-based device in the absence of angiographically visible thrombus, as in some patients with non–ST-segment elevation myocardial infarction, has to be discussed. Angiography, however, is a poor method to visualize mural thrombi that are an integral part of activated plaques, as in non–ST-segment elevation myocardial infarction. Moreover, dislodgement of brittle plaque material accounts for a substantial amount of the captured debris, as previously shown by Limbruno and coworkers16 and confirmed by our study. We therefore expected a beneficial effect of the filter wire even in the absence of visible thrombus.

Implications for Clinical Practice

In myocardial infarction with and without ST-segment elevation, reduction in the embolic burden by the filter wire does not improve myocardial reperfusion or reduce infarct size to a perceivable extent. The study does not provide a rationale for the routine use of the filter wire as an adjunct to primary PCI in myocardial infarction.

Acknowledgments

The study was funded in part by Boston Scientific, Waterloo, Belgium. The sponsor did not interfere with the design and the conduct of the trial, analysis of the results, or drafting of the manuscript. We thank Kristina Stahr for her excellent work in analyzing the MRI scans and the entire staff of our catheterization laboratory for their enthusiasm in the conduct of the study.

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Circulation. 2005;112:1462-1469; originally published online August 29, 2005;
doi: 10.1161/CIRCULATIONAHA.105.545178
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2005 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/112/10/1462

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