OBJECTIVES
The purpose of this study was to evaluate feasibility of using real-time, high-fidelity, intracardiac electrogram monitoring from a permanently implantable ischemia detection system (IIDS), with long-range telemetry capability to detect ST-segment shifts associated with acute or subacute coronary occlusion in a porcine model.

BACKGROUND
Early identification of coronary occlusion with ST-segment elevation could profoundly accelerate the timing of revascularization and improve clinical outcomes.

METHODS
This paper reports the first investigation using real-time, high-fidelity, intracardiac electrogram monitoring from a permanently IIDS, with long-range telemetry capability. This IIDS was tested in an ambulatory porcine model, with acute coronary occlusion precipitated by stent thrombosis. Two overlapping copper stents were implanted in the left anterior descending (n = 3), the circumflex (n = 3), or the right coronary artery (n = 2) of juvenile farm pigs. Monitoring was carried using telemetry from the IIDS.

RESULTS
All stented pigs had acute ST-segment elevation event(s) triggering the alerting thresholds of the IIDS. All triggered events were confirmed to be caused by thrombosis of the copper stent(s), and well correlated to infarct age and location. Four of the 8 pigs died from ventricular fibrillation, recorded by the IIDS at a mean time of 70 ± 121 h after ST-segment alert. The sensitivity and specificity of alerting for ST-segment shift, associated with thrombotic coronary occlusion, were 100% and 100%, respectively.

CONCLUSIONS
This study demonstrates the ability of an implantable ischemia detection system to detect ST-segment elevation from coronary occlusion in a porcine model of ST-segment elevation myocardial infarction. ST-segment elevation was sufficient to trigger alerting thresholds in all 3 epicardial coronary distributions. Such a system, with real-time alerting capability, could advance the time frame of reperfusion therapy and potentially prevent, rather than interrupt, acute myocardial infarction in patients with coronary artery disease. (J Am Coll Cardiol 2006;48:2306–14) © 2006 by the American College of Cardiology Foundation.

Acute myocardial infarction (MI) remains the leading cause of mortality in the Western world (1). Early detection and warning of acute MI, or antecedent ischemic events, and prompt intervention can substantially improve clinical outcomes (2–9). However, despite efforts at educating the public over the past decade, the mean time from MI symptom onset to arrival at a hospital for treatment has remained, disappointingly, at 2.5 to 3.0 h (3,8–12). Continued delays in door-to-balloon time typically exceed 2 h, to lead to further myocardial necrosis and poorer clinical outcomes (13,14). Because a large proportion of irreversible myocardial injury and fatal ventricular arrhythmias occur in the first several hours after closure of an epicardial coronary artery, it may be difficult to substantially improve upon our treatment of MI unless we can make an early and reliable diagnosis of the acute MI (3,8–20).

Previous testing of this concept in patients, using an intracardiac electrogram (ICEG) obtained from a skin electrode on the left upper chest to a temporary (pacemaker) electrode in the right ventricular apex has demonstrated consistent, rapidly evolving, and dramatic ST-segment shifts during balloon occlusion during percutaneous coronary intervention (PCI) in humans (16,17,21,22). Permanently implanted leads have the advantage of being free from ST-segment artifact, muscle noise, transthoracic damping, or concerns about electrode movement over the course of electrocardiogram (ECG) monitoring.
The current device monitors the ICEG 24 h a day 7 days a week. If any new ischemic event (either heart-rate-related ST-segment depression or sudden ST-segment elevation) occurs, the device will alert the patient with a buzzing from the device. It is expected that patients with such a device would immediately call their physician, and/or 911 upon getting such an ischemic alert. This early alerting capability could be used to improve the time to treatment, and outcomes, in high-risk coronary artery disease patients with ST-segment elevation MI (STEMI).

The purpose of this study was to expand upon the early human feasibility testing that utilized temporary pacemaker leads during PCI in supine patients. In this study, we report the first findings from a fully implantable, high-fidelity ICEG monitoring system, with programmable ST-segment elevation ischemia detection and alerting thresholds, and with long-range telemetry capability. This system was tested using the tip electrode from a permanent apical right ventricular (pacemaker) lead attached to the implantable ischemia detection system (IIDS), in an ambulatory, porcine copper stent, STEMI model (23).

METHODS

IIDS. This study was designed to evaluate the feasibility of a programmable, implantable device to provide early detection and alerts for transmural ischemia (sudden ST-segment elevation). The individual components of the IIDS include a pacemaker-sized implanted medical device (IMD), a standard bipolar “screw in” pacemaker lead, a lead adaptor containing the long-range telemetry antenna, a small portable external alerting device (EXD), and a laptop computer programmer to set event detection parameters and display the electrical signals recorded by the IMD. The components of the system are shown in Figure 1. This device is implanted in a manner analogous to a single-chamber pacemaker.

The programmer utilizes the EXD to provide wireless data connectivity to the IMD. The IMD attaches to a standard bipolar pacemaker lead with a lead adaptor as shown in Figure 1. The lead adaptor includes the antenna for EXD to IMD communication. This has a transmission range of 6 to 8 feet.

The IMD monitors the ICEG signal from the heart (can-to-tip ICEG) for ischemic ST-segment shift, by measuring the level of the ST-segment versus P-Q segment (Fig. 2). The IMD analyzes 10 consecutive QRST complexes every 30 s. This 10 beats every 30 s sampling is done to provide a close to “real-time” detection of events but with intent to conserve the implant’s battery life. For each beat, the IMD computes the average value of ST-segment voltage. The algorithm then uses the subject’s own baseline ST-segment voltage relative to the P-Q segment, measured 24 h previously, as the “control” signal for ST-segment shift comparisons. An appropriate heart-rate-matched “baseline” is examined as close to 24 h as possible if the control ST-segment is in a different heart rate bin. If ST-segment shift is detected in 8 of 10 analyzable beats, in 3 consecutive 30-s periods, this will set off the appropriate (programmed) alert. The requirement for detection of programmed threshold ST-segment shift in 8 of 10 beats in 3 consecutive samples is intended to reduce the likelihood of “false-positive” alerts. If at least 6 of 6 analyzable beats have excessive ST-segment shift in 3 successive 10-s segments, then an ST-segment shift alert can be activated, either with vibration of the IMD (like a cell phone) within the IMD case, or through an EXD to provide an additional auditory alert (beeps) and/or visual alert such as flashing light-
emitting diodes. Eventually this system could also be connected to a cellular-based service network.

Once an alert is initiated, ICEG segments related to the alert are saved by the IMD and can be retrieved for review by a physician using the programmer. The IMD is also capable of capturing ICEG data without alerting, when different cardiac events such as a low or an irregular heart rate is detected. There are other sophisticated programmable features that will not be addressed in detail in the current paper. For the purposes of this report, only the ST-segment deviation alerts were tested, with a threshold of \(\frac{1}{3}\)ST-segment shift relative to QRS height programmed as a significant ST-segment shift. This 30% threshold was chosen based upon the data from our previously published clinical data with temporary electrodes during PCI (16). No reperfusion therapy was attempted in this protocol.

Animal protocol. A total of 10 juvenile farm swine (Landrace-Duroc Yorkshire cross), weighing 30 to 40 kg, were used in this protocol. The protocol was reviewed and approved by the MPI Research Institutional Animal Care and Use Committee, and was performed at a major Good Laboratory Practices certified animal research facility (MPI Research) in Mattawan, Michigan. The study conformed to the “Position of the American Heart Association on Research Animal Use” adopted by the Association in November 1994.

Acute IIDS implants. Animals were pre-treated with aspirin, 325 mg, clopidogrel, 75 mg, and nifedipine, 30 mg, before IMD implant. General anesthesia was performed, with endotracheal intubation and medications per protocol. The medications included atropine, 0.05 mg/kg, telazol, 8 mg/kg intramuscularly, isoflurane by inhalation, cefazolin, 20 mg/kg intravenously, and fentanyl patch, 50 to 75 \(\mu\)g/kg transdermally, post-operatively.

The IMD was implanted in the same manner as a single-chamber pacemaker. The IMD was attached to a St. Jude Medical (Sylmar, California) bipolar pacemaker lead. This steroid eluting, screw-in lead was inserted transvenously using standard lead implantation techniques after cut-down in the animals’ left lower neck to access the left internal jugular. The lead tip was carefully positioned at the right ventricular apex, under fluoroscopic guidance, and then secured using the standard “screw-in” lead technique. Due to the anatomy of the juvenile swine, the IMD, in the current study, was implanted in the left lower neck of the pig. In humans, the IMD would be placed in the preferred position, in the left upper chest (16). The lead was attached, using the antenna/lead adapter, to the IMD. Baseline (control) ICEG data were retrieved via telemetry to the EXD over the next 24 to 72 h.

Copper stent implants. At 24 to 72 h after IMD implantation, 8 of the animals were brought back to the animal catheterization suite for copper stent placement. The other 2 animals served as controls. For the animals receiving stents, general anesthesia and pre-medication were administered as in the preceding text. After cut-down, a 7-F sheath was placed in the right common carotid artery. Intravenous heparin was administered to achieve an activated clotting time of >150 s. The pig’s right or left coronary artery was engaged using a 7-F JR3 coronary guiding catheter. The target vessel for stenting in each pig was predetermined by random assignment. Three animals had stenting of the left anterior descending, 3 animals had stenting of the left circumflex, and 2 animals had stenting of the right coronary artery. In each case, 2 custom made, copper, 18-mm length, balloon expandable stents were implanted in tandem fashion in the target vessel, with approximately 3 to 4 mm of stent overlap. The stents were implanted at 12 atms inflation pressure, with either a 3.0- or 3.5-mm diameter balloon, with inflation time ranging from 2 to 4 min. This copper stent model has been shown to be a reproducible model for subacute stent thrombosis, and “spontaneous” STEMI (20).

In lab ST-segment recordings. Both 12-lead ECG and ICEG ST-segment recordings were obtained in 6 of the 8 animals during the acute stent implantation (2-min balloon inflations) (Table 1). In 2 animals, surface lead data had too
much artifact for interpretation. After stent implantation, the animals were recovered, and baseline ICEG data were downloaded. All antiplatelet agents were discontinued after the stent implantation.

Two juvenile swine had IMD implants, without any coronary manipulation. No stents were placed in these animals so that they could serve as controls, specifically looking for false-positive ST-segment alerts in these ambulatory animals over 28 days of follow-up. In addition, baseline ICEG data from the stented pigs were monitored for 24 to 72 h before stent placement, to gather additional “control” ICEG data.

ICEG monitoring. After recovery, the animals were observed in holding cages with the EXD mounted within 6 to 8 feet of the animal for downloading of ICEG data. Formal data downloads were performed a minimum of 2 times/day, until animal death or sacrifice. All event-related IMD data were downloaded and analyzed to get detailed assessment of the ST-segment shift events (Fig. 2), and fatal arrhythmic events. The exact time-based relationship(s) of these events were easily accessed via the download of the IMD data. Surface ECG data were not obtained during the ambulatory period because the animals do not tolerate this type of monitoring.

Four of the 8 animals with copper stents died from ventricular fibrillation before 26 days. Repeat coronary angiography was performed before animal sacrifice in 3 of the 4 surviving animals. It was not feasible to perform anesthesia during ischemic events in the ambulatory animals, and no interventions were performed to interrupt the detected STEMIs. All 8 animals had a detailed cardiac necropsy study to assess both coronary patency and MI(s).

Pathologic data collection. All 8 animals with copper stent implants underwent detailed cardiac necropsy examination. The pathologists were blinded to the time of death relative to STEMI or stent implant. The pathologists were asked to provide qualitative age estimation, and the regional localization of any MI observed at necropsy.

Before processing the hearts, digital photographs and radiographs were obtained to document gross findings and to assess device and stent placement. For assessment of myocardial infarcts/scars, the hearts were “bread-loafed” from base to apex at 1-cm intervals.

Stented vessel segments were dehydrated in a graded series of ethanol and embedded in methylmethacrylate plastic. Sections from the stents were then cut on a rotary microtome at 4 to 5 μm, mounted and stained with hematoxylin and eosin and elastic Van Gieson stains. All myocardial sections were embedded in paraffin, cut on a rotary microtome, mounted on glass slides, and stained with H&E and Masson’s trichrome stains.

Data analysis. All ICEG recordings were stored from IMD downloads for subsequent analysis. A caliper-based, manual method (16) was used to measure the ST-segment shift compared with the PR segment at each of the following time points: baseline (before stenting); after 2 min of balloon inflation for both IMD and limb lead tracings (n = 5); at the time of the first automated ST-segment alarm; and at the peak ST-segment elevation detected before death or sacrifice.

Statistics. The ST-segment data and the QRS amplitude data were recorded in mm (mV) using Excel spreadsheets (Microsoft Corp., Bellevue, Washington). After raw data entry, this software was used to calculate % ST-segment change relative to baseline QRS amplitude at each time point. The statistical package from Excel (Microsoft Corp.) was used for statistical comparisons. The % ST-segment change at each time point was compared to baseline measurements using analysis of variance for repeated measures. This methodology was also used to compare the % ST-segment change between the surface (limb) lead and the intracardiac lead in the 5 animals with simultaneous measurement during stent implantation. All data in tables and figures are shown as mean ± SD. Sensitivity was defined as the number of animals with ST-segment shift causing emergency alert from the IMD with timing consistent with angiographic or pathologic evidence of occlusion and/or infarction divided by documented coronary occlusions. Specificity was defined by the incidence/probability of negative monitoring (i.e., no alerting ST-segment shifts).
among animals without coronary occlusion. A p value of <0.05 was considered statistically significant.

RESULTS

Electrogram data and STEMI in copper-stented animals. All 10 animals had stable intracardiac ICEGs at 24 h after IMD implant. There was minor ST-segment shift immediately after the right ventricle lead implants in 4 of 10 animals. This local “injury current” effect resolved by 24 h after permanent right ventricle lead implants in this model. There were no false positive ST-segment shift events from the IMD monitoring before stent implantation.

In 2 control animals, no coronary stenting was performed after IMD implantation. There were no false-positive ST-segment shift events in these animals over the 30 days of monitoring.

In the 6 animals with simultaneous recording of ICEGs and surface leads during stent implantation, there was a more prominent ST-segment shift after 2 min of balloon inflation from the intracardiac recording (33 ± 7%) compared with greatest ST-segment shift observed in any of the limb leads (22 ± 8%; p < 0.05 vs. intracardiac ST-segment shift) (Table 1). The mean ST-segment shift from spontaneous coronary occlusion, at the time that the emergency alert was activated, was 48% for the left anterior descending coronary artery, 38% for the left circumflex coronary artery, and 37% for the right coronary artery (all p values = NS).

All 8 stented animals demonstrated spontaneous and rapidly evolving ST-segment shift events between 3 and 77 h after copper stent implant. All of these events were consistent with subacute stent thrombosis and an associated STEMI (Table 1). The mean time from copper stent implant to the first ST-segment elevation emergency alert was 34 ± 19 h. The % ST-segment shift relative to baseline QRS amplitude, at the time of the first emergency ST-segment shift alert was 39 ± 6% (p < 0.001 vs. baseline ST-segment shift). It should also be noted that there was continued evolution of ST-segment shift observed in all 8 animals after the initial alerting detection. The

---

Figure 3. Illustration of acute ST-segment changes and spontaneous ST-segment elevation myocardial infarction as recorded by implanted medical device. Tracings were obtained from telemetry from implantable cardiac device to external alerting device. (A) Shows stable normal sinus rhythm without ST-segment shift at baseline 30 h after medical device implant. (B) Heart rate-related (heart rate increase from 119 at baseline to 148 beats/min) ST-segment depression sets off a “see your physician” alert 13 h after copper stent implantation in the left anterior descending coronary artery. (C) ST-segment elevation is noted at 59 h after copper stent implantation, resulting in an “emergency” alert. (D and E) There are more frequent ventricular premature beats, leading to ventricular fibrillation 1 h after the initial ST-segment emergency alert (F). The animal passed away with an agonal rhythm 15 min after the initial ventricular fibrillation (G).
peak ST-segment shift observed via IMD telemetry was 67 ± 15% (p < 0.0001 vs. baseline ST-segment shift) before sacrifice or death (n = 8).

Four of the 8 pigs died from ventricular fibrillation, all recorded by the IMD, before the scheduled 26- to 28-day sacrifice. The mean time from ST-segment elevation emergency alert to ventricular fibrillation/death was 70 ± 121 h. Two examples of ST-segment elevation, followed by ventricular fibrillation, are shown from the downloaded ICEGs in Figures 3 and 4.

Table 2 provides a summary of the time from automated ST-segment shift detection to death or sacrifice, and correlates these observations with the necropsy findings. There was a good correlation between the estimated age of MI and the vessel status by pathology, and by timing from first STEMI emergency alerting event via the IMD telemetry data.

Figure 4. Illustration of acute ST-segment changes and spontaneous ST-segment elevation myocardial infarction as recorded by implanted medical device. Tracings were obtained from telemetry from implantable cardiac device to external alerting device. (A) Shows stable normal sinus rhythm without ST-segment shift at baseline 26 h after medical device implant. (B) ST-segment elevation is noted after 90 s of balloon inflation, during copper stent implantation in the left anterior descending coronary artery. (C) The animal developed ventricular fibrillation at ~120 s into the left anterior descending coronary artery stent implant. External cardioversion/defibrillation is performed with 300 J (square wave in C), without deleterious effect on the implanted medical device. After 4 min of recovery, the intracardiac electrogram returns to baseline (D). At 3 h after copper stent implant, the electrogram shows rapid evolution to marked ST-segment elevation (72% vs. QRS amplitude, E) with emergency alert. Within 30 min from onset of ST-segment elevation myocardial infarction, the animal develops spontaneous ventricular fibrillation (F) and then expires, with a flat line tracing (G).
Figure 5 shows an illustration of ST-segment elevation recorded from the IMD during a (presumed) left circumflex occlusion 77 h after copper stent implantation. In this case, the animal recovered, with restabilization of the intracardiac ICEG. At angiography and necropsy 23 days later, the animal was demonstrated to have total thrombotic occlusion of the circumflex stents with a transmural lateral MI, estimated to be between 15 and 30 days old.

**DISCUSSION**

This study demonstrates the feasibility of using an intracardiac, right ventricular apical lead to detect ST-segment shift in the setting of sudden epicardial coronary artery occlusion in a copper stent, porcine model.

It is important to recognize that the only documented cause of rapidly progressive ST-segment elevation in humans is total and abrupt coronary occlusion (e.g., by balloon, thrombus, spasm, or other). Thus, the current model closely mimics the pathophysiology of coronary occlusion preceding and leading to acute MI in patients with coronary artery disease.

The clinical relevance of the observations made in this study may be viewed in the context of our current failure to achieve early diagnosis and treatment of acute MI. Despite advances in pharmacologic and mechanical means of coronary revascularization (4–7,13), the average time between onset of symptoms during acute MI and the arrival at a medical facility capable of either pharmacologic or mechanical revascularization is 2.5 to 3 h. This patient-related delay is problematic, and most often related to denial of symptoms, embarrassment, and/or misinterpretation of atypical symptoms (3,8,9–12). Additional major delays are com-

<table>
<thead>
<tr>
<th>Pig #</th>
<th>Vessel Stented</th>
<th>Survival to 28 Days</th>
<th>Time STEMI to VF/Death (h)</th>
<th>Time From STEMI Detection To Death or Sacrifice (dd:hh)</th>
<th>Angiographic and/or Pathology of Stented Vessel</th>
<th>Cardiac Pathology at Autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LAD No</td>
<td>1.1</td>
<td>00:01</td>
<td>No Angio 75% occluded; layers of thrombus</td>
<td>Focal areas of early ischemia anterior wall; myocyte hyperesinophilia, nuclear chromatin condensation</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>LAD No</td>
<td>0.6</td>
<td>00:00.6</td>
<td>No Angio 35% occluded; unorganized thrombus</td>
<td>Early ischemia; mild interstitial lymphocytic infiltrate anterior wall</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>LAD No</td>
<td>53</td>
<td>02:05</td>
<td>No Angio 91% occluded; organized thrombus</td>
<td>Transmural anterior and anteroseptal MI “4–6 days old”</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>LCX Yes</td>
<td>Survived MI</td>
<td>23:21</td>
<td>Occluded with organized thrombus</td>
<td>Transmural posterior, lateral wall MI at least 15–30 days old</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>LCX Yes</td>
<td>Survived MI</td>
<td>27:17</td>
<td>Occluded with organized thrombus</td>
<td>Transmural posterior, lateral wall MI at least 15–30 days old</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>LCX Yes</td>
<td>Survived MI</td>
<td>26:21</td>
<td>Occluded with organized thrombus</td>
<td>Transmural posterior, lateral wall MI at least 15–30 days old</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>RCA No</td>
<td>264</td>
<td>13:03</td>
<td>No Angio occluded with organized thrombus</td>
<td>Transmural posterior MI at least 10–15 days old</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>RCA Yes</td>
<td>Survived MI</td>
<td>26:19</td>
<td>Occluded with organized thrombus</td>
<td>Transmural posterior-septal, and right ventricular MI at least 15–30 days old</td>
<td></td>
</tr>
</tbody>
</table>

dd:hh = # days:# hours; ICEG = intracardiac electrogram; IIDS = implantable ischemia detection system; LAD = left anterior descending coronary artery; LCX = left circumflex artery; MI = myocardial infarction; NA = not available; RCA = right coronary artery; STEMI = ST-segment elevation myocardial infarction; VF = ventricular fibrillation.
mon, even after arrival at a tertiary cardiac care facility (2,12–14). Thus, the mean time from symptom onset to revascularization often exceeds 5 h. Delays in treatment are even more frequent, and potentially deadly for patients who experience atypical symptoms or no cardiac symptoms (“silent MI”) (2,12,15). This presentation is most common in diabetic patients, women, and the elderly. These “subgroups” comprise a substantial proportion of our aging population who are at risk for acute MI (1,2). Finally, a number of studies have clearly demonstrated the important relationship between the time from vessel closure to revascularization, and the clinical outcome after acute MI (2–9). Given these challenges, it may be difficult to substantially improve upon our treatment of acute MI unless we can make an earlier and reliable diagnosis. The observations from this study suggest that a simple implantable device with a configuration similar to today’s VVI pacemakers could be capable of such “early” ischemia detection.

Study limitations. This is an animal study intended to examine the feasibility of using an implantable, programmable, ischemia detection and alerting system to detect, record, and alert upon the recognition of a significant ST-segment shift during spontaneous coronary occlusion. These data were collected after implantation of copper lead during PCI, as reported by Varriale and Niznik (17), Fischell et al. (16), and others (19,21,22).

In the current study, we observed a substantial ST-segment shift (34%, relative to the QRS amplitude) within 2 min of balloon occlusion in an epicardial coronary artery of animals undergoing stent implantation. The magnitude of ST-segment shift relative to the QRS amplitude was greater for the intracardiac lead than for the surface leads, recorded during stent implantation. These data are similar to the recent human studies by Theres et al. (22), and our group (16), suggesting that intracardiac ICEG recordings may be superior to surface leads in detecting myocardial ischemia. Based upon our prior human experience (16), we believe that the detection of left circumflex coronary artery and right coronary artery events may be further enhanced with implantation of the second electrode (“can”) in the left upper chest, as opposed to the left neck, as was required in these porcine experiments.
stents to cause subacute stent thrombosis, and acute STEMI. The results obtained using this methodology may not exactly replicate the ST-segment changes that would be observed in patients with STEMI from thrombosis in the setting of chronic, atherosclerotic coronary heart disease. Although first-in-man clinical testing is planned, we do not have data from permanent implants, in humans, to evaluate the stability of the intracardiac ICEG or its sensitivity or specificity in detecting coronary occlusion. However, the 100% sensitivity and specificity of ST-segment shift alerts correlating with coronary artery thrombotic occlusion, in this model, is encouraging.

Conclusions. This study confirms the feasibility of using an implantable, programmable, ischemia detection and alerting system (IIDS) to assist in the diagnosis of an STEMI caused by subacute stent thrombosis. These results, combined with prior clinical observations (16,17,21,22), suggest that a relatively simple, implantable system, resembling a VVI pacemaker (16,24), could be programmed to assist in the early diagnosis of acute STEMI and/or antecedent ischemic events. The potential to identify human coronary occlusion within seconds of its onset, and provide immediate mechanical alerting to the patient, could shift the entire paradigm of care from (current) interruption of MI, to the prevention of myocardial necrosis.

Although a number of technical and clinical questions remain to be addressed by future work, this report suggests that intracardiac ST-segment monitoring, with an alerting capability, could be used to improve the time to treatment, and outcomes, in high-risk coronary artery disease patients with STEMI.

Acknowledgments
The authors would like to acknowledge the substantial contributions of A. Jill Schweiger, BS, Steven R. Johnson, MS, Jonathan P. Harwood, BS, Andrew J. Carter, DO, and Arnie Sippens, MD, David Moddrelle, SRS, and Paul B. Jennings, Jr., VMD, DACVS, to this project and paper.

Reprint requests and correspondence: Dr. Tim A. Fischell, Director, Heart Institute at Borgess Medical Center, Professor of Medicine, Michigan State University, 1521 Gull Road, Kalamazoo, Michigan 49048. E-mail: taf1@net-link.net.

REFERENCES