

Potential of an intracardiac electrogram for the rapid detection of coronary artery occlusion[☆]

Tim A. Fischell^{a,*}, David R. Fischell^b, Robert E. Fischell^b, Susan Baskerville^a, Susan Hendrick^a, Carol Moshier^a, Jonathan P. Harwood^b, Mitchell W. Krucoff^c

^aBorgess Heart Institute, Kalamazoo, MI 49048, USA

^bAngel Medical Systems, Inc., Tinton Falls, NJ 07701, USA

^cDuke University Medical Center, Durham, NC 27710, USA

Received 5 April 2005; received in revised form 10 May 2005; accepted 10 May 2005

Abstract

Background: Early identification of acute MI and prompt intervention can improve clinical outcomes. It would be valuable to identify a method that could allow the earliest possible detection of myocardial injury or ischemia.

Methods and results: This article reports one of the first clinical investigations to examine the ability of an intracardiac right ventricular (RV) electrode to identify the early onset of myocardial ischemia/injury in a cohort of patients undergoing balloon occlusion of a coronary artery during percutaneous transluminal coronary angioplasty. The primary data set for analysis included observations from 14 patients with 17 lesions, with a matched comparison of a V6 surface lead and the RV to left upper chest, “intracardiac” lead. The intracardiac lead was sensitive in detecting myocardial injury current/ischemia. There was a $36.4 \pm 5.6\%$ ST-segment shift, relative to the amplitude of the QRS complex, in the intracardiac lead at 2 min, compared with a $10.1 \pm 1.9\%$ ST shift from a surface lead ($P = .00011$). The RV to left upper chest lead detected a $>10\%$ shift in ST segment within 2 min in 17 (100%) of 17 cases vs. 8 (47%) of 17 for a V6 surface lead. The intracardiac lead provided detection of ischemia in all three major epicardial coronary distributions.

Conclusions: This study demonstrates the ability of an intracardiac (RV apex to left upper chest) lead to rapidly detect myocardial ischemia/injury during acute coronary occlusion in the setting of balloon angioplasty. The results of this study suggest that a simple implantable system resembling a ventricular pacemaker could be programmed to assist in the very early diagnosis of acute myocardial infarction. © 2005 Elsevier Inc. All rights reserved.

Keywords: Myocardial infarction; Ischemia; Detection; Implantable devices

1. Introduction

Acute myocardial infarction (MI) remains the leading cause of mortality in the western world [1]. There is little

question that early identification of acute MI 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 2.5–3.0 h [3,8–12].

Since 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 myocardial infarction [3,8–13].

[☆] Disclosures: T.A. Fischell, D.R. Fischell, and R.E. Fischell are cofounders of Angel Medical Systems. J.P. Harwood is a full-time employee of Angel Medical Systems. M. W. Krucoff is a member of the scientific advisory board for Angel Medical Systems.

* Corresponding author. Department of Medicine, Heart Institute at Borgess Medical Center, Michigan State University, 1521 Gull Road, Kalamazoo, MI 49048, USA. Tel.: +1 269 226 8362; fax: +1 269 226 8349.

E-mail address: taf1@net-link.net (T.A. Fischell).

Using this reasoning, it would be valuable to identify a method that could allow the earliest possible detection of myocardial injury or ischemia. In this study, we have performed one of the first clinical investigations to examine the ability of an intracardiac (probing) right ventricular (RV) electrode to identify the early onset of myocardial ischemia/injury in a cohort of patients undergoing balloon occlusion of a coronary artery in the setting of percutaneous transluminal coronary angioplasty (PTCA)/stenting.

The purpose of this study was to examine the recordings obtained from an intracardiac electrogram obtained from a RV (apical) temporary pacing electrode, and a reference electrode on the left upper chest, during the course of temporary balloon occlusion during PTCA and/or stenting.

2. Methods

2.1. Patient population

From October 2001 through May 2002, 17 patients were enrolled in this protocol. Patients were eligible for inclusion if they had a clinical indication for coronary artery revascularization utilizing PTCA and/or stenting in one or more major epicardial coronary artery. The exclusion criteria were (1) contraindication to placement of a temporary pacemaker via a transfemoral venous approach, (2) inability to obtain a stable RV apical electrogram using the temporary pacing electrode, (3) absence of myocardial viability distal to the intended target vessel for PTCA (prior MI in that distribution), (4) baseline resting ECG repolarization abnormalities or QRS duration of >120 ms, and/or (5) total or subtotal occlusion of vessel to be treated. The protocol was reviewed and approved by the Borgess Medical Center Institutional Review Board. All patients provided informed consent.

2.2. Cath lab protocol

Arterial and venous access was obtained via a femoral approach in all cases. Intravenous heparin was given to achieve an ACT of >250 . A 4-French temporary pacing catheter (Bard Electrophysiology, Lowell, MA) was advanced from the femoral vein, across the tricuspid valve. The distal electrode tip was advanced to the RV apex using fluoroscopic guidance. The reference electrode for the RV electrode was chosen to be the skin electrode on the left upper chest. This lead pair was chosen to closely mimic the “tip-to-can” electrogram obtained from a ventricular pacemaker implanted in the left upper chest. For purpose of comparison to the intracardiac lead, recordings were also collected from a V6 chest lead (surface) lead. This lead was chosen as the single (surface) lead, due to its reasonable detection capability for all three major epicardial distributions. Baseline electrograms were obtained from the surface lead and from the RV apex to left upper

chest. This lead recorded the electropotential difference from the RV apex (distal electrode only) to the left arm/shoulder (skin lead). The configuration of the leads is shown in Fig. 1. The RV apical lead was chosen because the three-dimensional relationship between the RV apex and the left upper chest appears favorable for capturing electrical information from the inferior, lateral, and anterior left ventricular myocardium.

All of the cases reported utilized ($n=17$ vessels and 14 patients) were recorded using a Compaq PC with customized software, in conjunction with Pulse Biomedical QRS-Card hardware (Norristown, PA). This system was used to connect the intracardiac and surface leads into the serial port of a Windows PC. The QRS card has a frequency response of 0.5 to 100 Hz. For the purposes of these electrogram and surface electrocardiogram recordings, high-pass filtering was done at 0.5 Hz. The QRS card was set to a sampling rate of 240 samples per second. The analog to digital converter had 12 bits of resolution.

Typically, when good endocardial contact was achieved with the temporary pacemaker, a stable electrogram could be recorded. The baseline (RV) electrogram morphology was inconsistent in three patients, apparently due to systolic motion of the 4-French pacing catheters. These cases were excluded from the study. Acceptable intracardiac lead recordings were obtained in a total of 14 patients and 17 vessels.

In each of these cases simultaneous intracardiac and surface lead (stable) electrograms were obtained continuously, starting 30 s prior to balloon inflation in an epicardial coronary artery and for a minimum of 2 min following balloon deflation, using the QRS card system. Electrogram tracings were recorded during intervention in a second vessel, when two-vessel angioplasty was performed ($n=3$). Following the final balloon inflation, the temporary pacing catheter was removed, and the data were stored for off-line analysis.

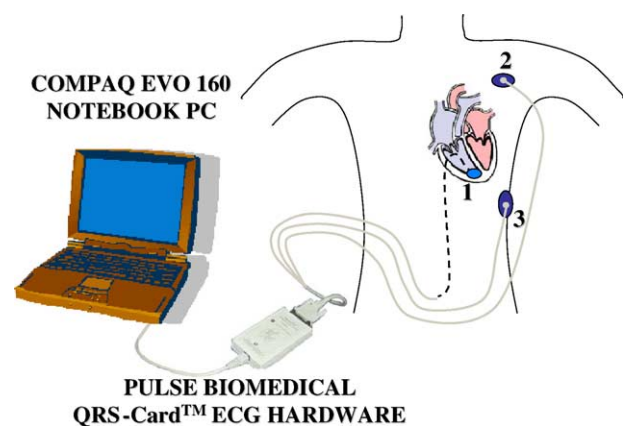


Fig. 1. Schematic illustrating the lead configuration and ECG recording system used in the study. The RV apical electrode (1) is placed using a temporary pacemaker lead. The skin electrodes are placed on the left upper chest (2) and in the left posterior axillary line (3). Electropotential differences are recorded between leads 1 and 2 (“intracardiac electrogram”) and between leads 2 and 3 (“surface lead”).

Table 1
Baseline clinical angiographic and ECG data

Patient #	Location of PTCA	CASS lesion type	Prior MI yes/no	History angina	Beta blockers	Calcium Ch. blocker	# Vessels >70% DS	Diabetes (yes/no)	Hypertension (yes/no)	Baseline heart rate	Normal electrolytes	QRS duration (msec)	ST abnormalities baseline	
1	Mid RCA	B1	no	yes	yes	no	yes	1	yes	yes	76	yes	108	no
2	Proximal LCX	B2	no	yes	yes	yes	yes	2	no	no	81	yes	111	no
3	Distal RCA	B1	no	yes	yes	no	yes	1	no	yes	64	yes	100	no
4	Proximal diagonal	B2	no	no	yes	no	yes	1	no	no	68	yes	105	no
5	Mid LAD	A	no	yes	yes	no	no	1	no	no	74	yes	116	no
6	Distal RCA	B2	no	yes	no	yes	no	2	no	yes	90	yes	120	no
7	Mid RCA	B2	no	yes	yes	no	yes	1	yes	yes	61	yes	100	no
8	Distal RCA	C	no	yes	yes	no	yes	1	no	no	65	yes	98	no
9	Proximal LCX	B2	no	yes	yes	no	yes	1	no	no	72	yes	106	no
10	Obtuse marginal	B2	no	yes	no	no	yes	2	no	no	78	yes	109	no
11	Mid LCX	B1	no	yes	yes	no	yes	1	no	no	64	yes	119	no
12	Mid LAD	B1	no	yes	yes	yes	no	1	yes	yes	62	yes	111	no
13	Proximal RCA	B1	no	yes	yes	no	yes	1	no	yes	58	yes	102	no
14	Mid RCA	B1	no	yes	no	no	yes	1	no	yes	77	yes	104	no

- number, MI - myocardial infarction, % - percentage, Ch - channel, RCA - right coronary, LCX - left circumflex, LAD - left anterior descending; electrolytes include Na, K, Cl, HCO₃.

The complete data set included a total of 14 patients and 17 vessels treated with simultaneously acquired data from the surface and intracardiac leads.

2.3. Data analysis

All electrogram recordings were stored for subsequent analysis. A caliper-based manual method was used to

measure the ST-segment shift compared with the PR segment at each of the following time points: baseline (prior to balloon inflation), after 1 min of balloon inflation, and after 2 min of balloon inflation. The interobserver variability of this measurement was 0.4 ± 0.1 mV. To make uniform measurements among the cases, the ST shift was defined relative to the PR-segment height. The ST segments were measured at a point taken 120 ms after the end of the QRS complex.

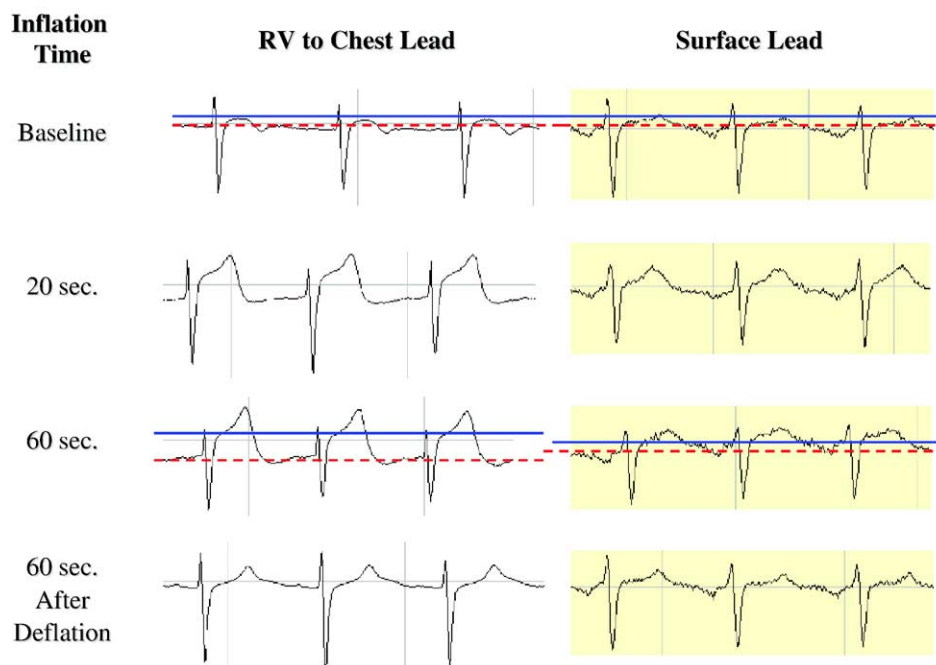


Fig. 2. Case example illustrating greater ST shift with the intracardiac lead (RV to chest lead) compared with the surface lead in a patient during balloon occlusion/PTCA of the LAD coronary artery. At baseline there is minimal ST shift. At 20 and 60 s after balloon inflation there are definite changes (ST elevation) in the intracardiac lead with minimal changes in the surface lead. Both leads normalize quickly at 1 min following balloon deflation (bottom panels).

Table 2
Summary of matched data from surface lead

Patient	Vessel	Location	Inflation <i>T</i> (min)	R height (mV)	ST baseline	ST 1 min	ST change 1 min (mV)	% ST shift. (1 min)	ST 2 min	ST change 2 min (mV)	% ST shift (2 min)
1	1	RCA	2.0	18.0	-0.5	-0.3	0.2	1.1	-0.3	0.2	1.1
2	2	LCX	2.0	13.0	0.0	2.8	2.8	21.5	3.0	3.0	23.1
3	3	LAD	2.0	13.0	0.0	1.5	1.5	11.5	2.0	2.0	15.4
3	4	RCA	2.0	32.0	-2.0	0.5	2.5	7.8	1.0	3.0	9.4
4	5	Diag	2.0	15.0	0.0	0.5	0.5	3.3	1.0	1.0	6.7
5	6	LAD	1.0	11.0	1.0	2.5	1.5	13.6	-	-	-
6	7	Distal RCA	2.0	12.5	0.0	3.0	3.0	24.0	3.5	3.0	24.0
7	8	RCA	2.0	41.0	1.0	1.5	0.5	1.2	1.5	0.5	1.2
8	9	LCX	2.0	32.0	0.0	3.0	3.0	9.4	3.5	3.5	10.9
8	10	Distal RCA	2.0	31.0	0.5	2.0	1.5	4.8	2.0	1.5	4.8
9	11	LCX	3.5	24.0	0.5	2.2	1.7	7.1	2.5	2.0	8.3
10	12	LCX	2.0	26.0	1.0	3.0	2.0	7.7	3.0	2.0	7.7
10	13	LAD	2.0	27.0	-2.0	-4.0	2.0	7.4	-4.0	2.0	7.4
11	14	LCX	2.5	20.0	-1.0	-0.5	0.5	2.5	0.0	1.0	5.0
12	15	LAD	2.0	22.0	1.5	2.5	1.0	4.5	3.0	1.5	6.8
13	16	RCA	2.0	25.0	0.0	2.0	2.0	8.0	3.5	3.5	14.0
14	17	RCA	2.0	14.0	0.0	2.5	2.5	17.9	3.0	3.0	21.4
Mean			2.1±0.1	22.2±2.1	0±0.2	1.5±0.4	1.7±0.2	9.1±1.6	1.7±0.5	2.0±0.3	10.1±1.9

R height=QRS height, *T*=time.

To normalize the ST-segment shift for the differing amplitude of surface and intracardiac electrogram recordings, the data for ST shift were plotted as the absolute percentage change of the ST segment, relative to the QRS complex amplitude (i.e., change in ST (mV)/QRS amplitude (mV)×100%). These data served as the primary data for analysis and are referred to in the text and the tables as the percent change in ST at each time point (e.g., % ST change 1 min). We plotted the relative, time-related ST-segment shift from the surface and intracardiac leads for the left anterior descending (LAD), left circumflex (LCX), and the right coronary arteries (RCA).

2.4. Statistics

The ST-segment and QRS-amplitude data were recorded in mm (mV) using Excel spreadsheets (Microsoft, Bellevue, WA). This software was used to calculate % ST change at each time point. The statistical package from Excel (Microsoft Corp., Bellevue, WA) was used for statistical comparisons. Absolute ST shift at each time and % ST change at each time point were compared using ANOVA for repeated measures. This methodology was used to compare the % ST change between the surface (modified chest) lead and the intracardiac lead at each time point. All data in tables and in

Table 3
Summary of data from simultaneous recordings: intracardiac (RV) lead

Patient	Vessel	Location	Inflation <i>T</i> (min)	R height (mV)	ST base	ST 1 min	ST change 1 min (mV)	% ST shift (1 min)	ST 2 min	ST change 2 min (mV)	% ST shift (2 min)
1	1	RCA	2.0	41.0	-5.0	3.0	8.0	19.5	5.0	10.0	24.4
2	2	LCX	2.0	10.0	-9.0	-5.0	4.0	40.0	-3.0	6.0	60.0
3	3	LAD	2.0	16.0	-10.0	-7.0	3.0	18.8	7.0	3.0	18.8
3	4	RCA	2.0	22.0	-14.0	-11.0	3.0	13.6	-9.0	5.0	22.7
4	5	Diag	2.0	17.0	-16.0	-14.0	2.0	11.8	-13.0	3.0	17.6
5	6	LAD	1.0	12.0	2.0	7.0	5.0	41.7	-	-	-
6	7	Distal RCA	2.0	12.0	-11.0	-7.0	4.0	33.3	-7.0	4.0	33.3
7	8	RCA	2.0	17.0	-24.0	-19.0	5.0	29.4	-19.0	5.0	29.4
7	9	LCX	2.0	21.0	-13.0	-9.0	4.0	19.0	-9.0	4.0	19.0
8	10	Distal RCA	2.0	7.0	-5.0	-4.0	1.0	14.3	-4.0	1.0	14.3
9	11	LCX	3.5	7.0	-15.0	-9.0	6.0	85.7	-8.0	7.0	100.0
10	12	LCX	2.0	9.0	-9.0	-11.0	2.0	22.2	-12.0	3.0	33.3
10	13	LAD	2.0	12.0	-6.0	-2.0	4.0	33.3	0.0	6.0	50.0
11	14	LCX	2.5	24.0	-17.0	-9.0	8.0	33.3	-6.0	11.0	45.8
12	15	LAD	2.0	27.0	-6.0	-2.0	4.0	14.8	-1.0	5.0	18.5
13	16	RCA	2.0	14.0	-21.0	-17.0	4.0	28.6	-13.0	8.0	57.1
14	17	RCA	2.0	21.0	-12.0	-10.0	2.0	9.5	-4.0	8.0	38.1
Mean			2.1±0.1	17±2.0	-11.2±1.5	-7.4±1.6	4.1±0.5	27.6±4.3	-6.9±1.6	5.7±0.7	36.4±5.6

R height=QRS height, *T*=time.

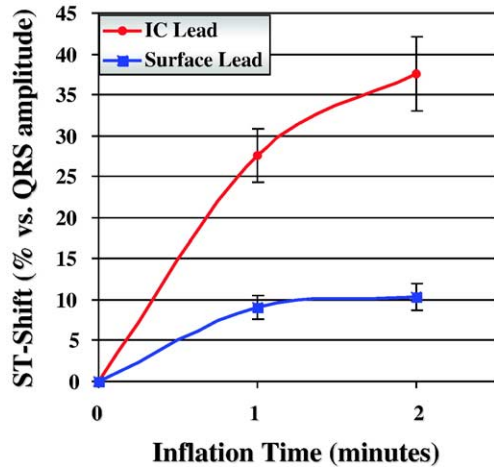


Fig. 3. Line graph depicting the ST-segment shift vs. baseline, normalized for QRS amplitude, at baseline (time=0), and at 1 and 2 min following balloon occlusion. Data depicted represent the mean±S.E. for the surface lead compared with the intracardiac lead (IC Lead) for the matched cohort (14 patients, 17 lesions). *P* values comparing these changes are $P=.0003$ and $P=.0001$, respectively, for the 1- and 2-min time points comparing the two leads (i.e., greater ST shift for intracardiac lead).

figures are shown as mean±S.E. A *P* value of $\leq .05$ was considered statistically significant.

3. Results

3.1. Patient demographics

As above, a total of 14 patients with 17 coronary vessels were studied using a temporary pacing electrode. Three patients had two-vessel intervention. The remainder of the patients had a single vessel intervention. There were 3 women and 11 men with a mean age of 62 ± 5.9 years. There were three diabetics among the 14 patients. The distribution of

vessels studied in the simultaneously recorded cohort was 5 LAD or diagonal branch, 5 LCX, and 7 RCA. The details of past medical history, lesion site and type, medications, baseline ECG, and electrolyte abnormalities are summarized in Table 1.

3.2. ST-segment shift

Among the intracardiac electrograms, there was significant ST-segment depression in the baseline and the recovery electrograms in 12 of 14 patients. This baseline stable ST-segment shift was attributed to a local injury current at the temporary pacemaker electrode tip/endocardial interface. This is a well-known phenomenon from an electrogram during the acute placement of a temporary pacing electrode. The downward vector of the “local injury current” ST shift is explained by the endocardial/intracardiac positioning of the lead tip. In two cases, the baseline electrograms were nearly isoelectric to the PR segment. In these cases, the electrogram more closely resembles the electrograms recorded from healed permanent pacemaker lead tips (Fig. 2).

Despite the stable, baseline ST depression (local injury current), ST-segment shift during coronary balloon occlusion was detected in the intracardiac electrogram in 14 of 14 patients and 17 of 17 vessels during balloon occlusion. In 16 (94%) of 17 cases, there was ST elevation, during balloon inflation, compared with baseline. In 1 (6%) of 17 cases, there was ST segment depression during balloon inflation.

In the intracardiac electrogram, the mean baseline ST segment demonstrated a -11.2 ± 1.5 mV depression. At 1 min following balloon occlusion, the mean ST-segment shift was -7.4 ± 1.6 mV ($P=.0002$ vs. baseline). At 2 min (16/17 cases) following balloon occlusion, the mean ST-segment shift was -6.9 ± 1.6 mV ($P=.0002$ vs. baseline). Thus, there was an absolute ST-segment shift upward of 3.8 ± 1.5 mV at 1 min and 5.3 ± 1.4 mV at 2 min after balloon inflation.

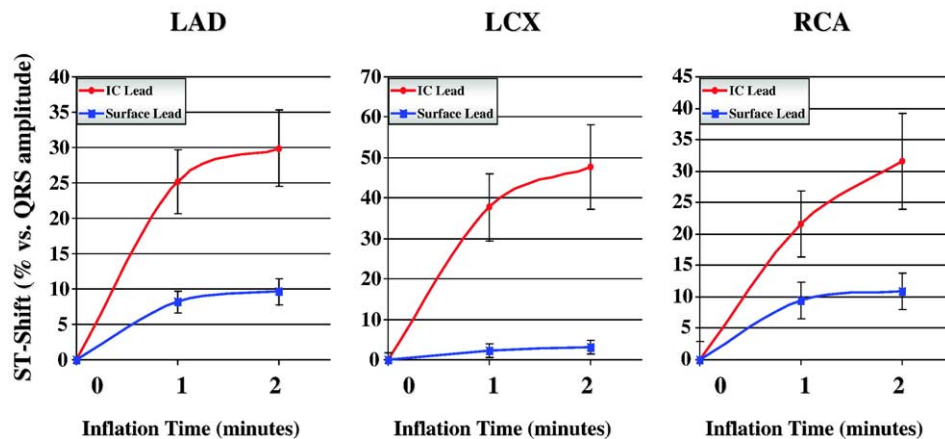


Fig. 4. Line graphs depicting the ST-segment shift vs. baseline, normalized for QRS amplitude, at baseline (time=0), and at 1 and 2 min following balloon occlusion in the LAD ($n=5$), LCX ($n=5$), and RCA ($n=7$) arteries. Data depicted represent the mean±S.E. for the surface lead compared with the intracardiac lead (IC lead) for the entire cohort. *P* values comparing these changes are $<.01$ at the 1- and 2-min time points comparing the two leads for all three vessels (i.e., greater ST shift for intracardiac lead).

The mean time of balloon inflation was 2.1 ± 0.1 min (range 60–210 s). One patient could only tolerate 1-min balloon inflation. The longest balloon inflation time was 3.5 min. In this case, there was modest ST shift at 2 min, followed by a more dramatic ST shift after 3.5 min of balloon inflation. This suggests some potentially important, time-dependent development of myocardial injury current when using such a sensing system.

A summary of the baseline and “ischemic” ST measurements for the surface and intracardiac leads are shown in Tables 2 and 3, respectively. Fig. 3 is a line graph comparing the time course and magnitude of relative % ST shift between the intracardiac and the surface lead from the cohort with simultaneous recording of the surface and intracardiac lead. Fig. 4 shows the data from the entire cohort, comparing the intracardiac ST changes with the surface lead categorized according to the vessel occluded (i.e., LAD, LCX, and RCA).

Among the cohort, there was a greater % ST shift in the intracardiac electrogram compared with the surface lead at both 1 min of inflation ($P=.00037$) and after 2 min ($P=.00011$). Fig. 2 shows an example illustrating superior sensitivity of the intracardiac as compared with the simultaneous recordings from a surface lead. This differential sensitivity in the detection of “injury” current was most evident in the LCX cases. At 2 min following balloon occlusion of the LCX, there was only a $3.1 \pm 1.6\%$ ST shift in the surface lead, compared with a $48.8 \pm 11.1\%$ ST shift in the intracardiac lead ($P<.01$). In nearly all cases, ST shift returned to baseline levels within 120–240 s following balloon deflation (Fig. 2).

4. Discussion

This study demonstrates the feasibility of using an intracardiac, RV apex to left upper chest (“intracardiac”) lead to detect very early ST-segment shift in the setting of sudden epicardial coronary artery occlusion. We believe that this model mimics the pathophysiology of acute myocardial infarction in humans. The configuration of the intracardiac lead system in this study is analogous to the tip-to-can electrogram that can be obtained from most contemporary permanent ventricular pacemakers and automatic implantable cardiac defibrillators (ICDs). These observations are consistent with preliminary observations from Siegel et al. [14] and Stark et al. [15].

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 myocardial infarction. Despite advances in pharmacologic and mechanical means of coronary revascularization [4–7,16], the average time between onset of symptoms during acute myocardial infarction and the arrival at a medical facility capable of either pharmacologic or mechanical revascularization is 2.5–3 h. This patient-related delay is problematic and most

often related to denial of symptoms, embarrassment, and/or misinterpretation of atypical symptoms [3,8–12]. Additional delays are common, even after arrival at a tertiary cardiac care facility [2,12,13]. Thus, the mean time from symptom onset to revascularization often exceeds 4 h. Delays in treatment are even more frequent and potentially deadly for patients who experience atypical symptoms or no cardiac symptoms (“silent MI”) [2,12,13]. This presentation is most common in diabetics, women, and the elderly. These “subgroups” actually comprise a substantial proportion of our aging population who are at risk for acute myocardial infarction [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 following acute myocardial infarction [2–9]. Thus, it may be difficult to substantially improve upon our treatment of acute myocardial infarction unless we can make an earlier and reliable diagnosis.

In the current study, we observed a mean ST-segment shift of 36.4%, relative to the QRS amplitude, within 2 min of balloon occlusion in an epicardial coronary artery in patients with coronary artery disease undergoing PTCA. The magnitude of ST shift from the intracardiac electrogram during balloon occlusion was similar for all three epicardial coronary distributions (LAD, LCX, and RCA). The magnitude of ST shift relative to the QRS amplitude was greater for the intracardiac to left upper chest lead than for a chest surface lead (mean change of 10.1% at 2 min). This was particularly evident in cases of PTCA or stent placement in the LCX distribution, a territory notorious for “false-negative” surface ECGs. The observations from this study suggest that a simple implantable device with a configuration similar to today’s VVI pacemakers and ICDs could be capable of such “early” MI detection.

An implantable detection system could be combined with a patient communication device to allow the patient to get EMS care within a short time after closure of an epicardial coronary artery. There is a potential for such a system to save lives and “rescue” myocardial tissue. The completed, stand-alone, ischemia-detection device is currently being evaluated in animal models.

4.1. Limitations

This is a pilot study intended to examine the feasibility of using a relatively simple intracardiac electrogram recording for the detection of ST-segment shift during balloon occlusion in the setting of PTCA. There are limitations of this early study that will have to be addressed with further investigation.

These data were collected following the acute placement of a temporary pacemaker lead, and using a skin electrode on the left chest. The results obtained using this methodology may not exactly replicate the ST changes that would be observed with a permanent RV lead and an implanted reference electrode in the left upper chest. In two patients outside of this

reported series, we have observed similar ST-segment shift when recorded from the tip-to-can electrode of a permanently implanted pacemaker (Pacesetter, Sylmar, CA). In addition, recent testing of the completed stand-alone device has demonstrated stable intracardiac electrograms in an ambulatory, porcine implant model. However, further studies will be required to validate the present observations in a chronic lead implant setting, in both animal model(s) and patients.

Although the changes observed during balloon angioplasty in our cohort were striking at 1–2 min, we do not have enough observations to examine the relationship of occlusion duration to ST shift significantly beyond 2 min. This issue will also require further investigation.

Finally, we used only a single chest lead to compare with the intracardiac electrogram. Bush et al. [17] found that one could obtain up to a relatively high sensitivity for ST shift detection during PTCA balloon occlusion if one used a 12-lead ECG recording. However, the use of surface leads may not be a practical means for real-time 24-h monitoring.

4.2. Conclusions

This study demonstrates the ability of a single intracardiac RV apical lead (reference electrode on the left upper chest) to rapidly detect myocardial ischemia/injury during acute coronary occlusion in the setting of balloon angioplasty. In many ways, this model mimics the first few minutes of acute myocardial infarction. The results of this study suggest that a simple, implantable system resembling a ventricular pacemaker could be programmed to assist in the very early diagnosis of acute myocardial infarction. Further studies will be required to refine and extend these preliminary observations.

Acknowledgments

We thank Angel Medical Systems for their partial funding of this study.

References

- [1] Heart and Stroke statistical Update. Dallas (Tex): American Heart Association, 1999.
- [2] Canto JG, Zalenski RJ, Ornato JP, et al. Use of emergency medical services in acute myocardial infarction and subsequent quality of care observations from the national registry of myocardial infarction 2. *Circulation* 2002;106:3018–23.
- [3] Ryan TJ, Anderson JL, Antman EM, et al. The physician's role in minimizing pre-hospital delay in patients at high risk for acute myocardial infarction: recommendations from the National Heart Attach Alert Program. Working Group on Educational Strategies to Prevent Prehospital Delay in Patients at High Risk for Acute Myocardial Infarction. *Ann Intern Med* 1997;126:645–51.
- [4] GISSI (Gruppo Italiano per lo Studio della Streptochinasi nell'Infarcto Miocardico). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986;1:397–402.
- [5] Second International Study of Infarct Survival (ISIS-2) Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both or neither among 17, 187 cases of suspected acute myocardial infarction. *Lancet* 1988;2:349–60.
- [6] Grines CL, Browne KF, Marco J, et al. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. *N Engl J Med* 1993;328:673–9.
- [7] Gibbons RJ, Holmes DR, Reeder GS, et al. Immediate angioplasty compared with the administration of a thrombolytic agent followed by conservative treatment for myocardial infarction. *N Engl J Med* 1993; 328:685–91.
- [8] Faxon D, Lenfant C. Timing is everything: motivating patients to call 9-1-1 at onset of acute myocardial infarction. *Circulation* 2001;104: 1210–1.
- [9] DeLuca G, Suryapranata H, Ottervanger JP, Antman EM. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction Every minute counts. *Circulation* 2004;109:1223–5.
- [10] Brown AL, Mann C, Daya M, et al. Demographic, belief and situational factors influencing the decision to utilize emergency medical services among chest pain patients. *Circulation* 2000;102: 173–8.
- [11] Meischke H, Ho MT, Eisenbert MS, et al. Reasons patients with chest pain delay or do not call 911. *Ann Emerg Med* 1995;25:193–7.
- [12] Chew DP, Moliterno DJ, Herrmann HC. Present and potential future paradigms for the treatment of ST-segment elevation acute myocardial infarction. *J Invas Cardiol* 2002;14(Suppl A):3A–20A.
- [13] Luepker RV, Raczynski JM, Osganian S, et al. Effort of a community intervention on patient delay and emergency medical service use in acute coronary heart disease: the Rapid Early Action for Coronary Treatment (REACT) trial. *JAMA* 2000;284:60–7.
- [14] Siegel S, Brodman R, Fischer J, et al. Intracardiac electrode detection of early or subendocardial ischemia. *Pacing Clin Electrophysiol* 1982;892–902.
- [15] Stark KS, Krucoff MW, Schryver B, Kent KM. Quantification of ST-segment changes during coronary angioplasty in patients with left bundle branch block. *Am J Cardiol* 1991;67:1219–22.
- [16] Herrmann HC, Moliterno DJ, Ohman EM, et al. Facilitation of early percutaneous coronary intervention after reteplase with or without abciximab in acute myocardial infarction. Results from the SPEED (GUSTO-4) Pilot trial. *J Am Coll Cardiol* 2000;36: 1489–96.
- [17] Bush HS, Gerguson JJ, Angelini P, Willerson JT. Twelve-lead electrocardiographic evaluation of ischemia during percutaneous transluminal coronary angioplasty and its correlation with acute reocclusion. *Am Heart J* 1991;121:1591–9.