Automatic Implantable Cardioverter/Defibrillator Discharges and Acute Myocardial Injury

Boaz Avitall, MD, PhD, Steven Port, MD, Rami Gal, MD, James McKinnie, MD, Patrick Tchou, MD, Mohammad Jazayeri, MD, Paul Troup, MD, and Masood Akhtar, MD

Multiple defibrillations by the automatic implantable cardioverter/defibrillator (AICD) have been reported to result in localized epicardial damage. No data exist, however, regarding whether this damage can be detected in the clinical setting or whether it interferes with the detection of true myocardial infarction. Forty-nine patients who received defibrillations by patch electrodes were studied prospectively. We attempted to document the presence of myocardial injury with the following three commonly used modalities for the detection of myocardial infarction: serial electrocardiographic changes, serial creatine phosphokinase (CPK) and CPK-MB release, and technetium 99m pyrophosphate scanning. Fifteen patients received defibrillations by AICD patches at the time of AICD generator replacement. Nine patients received defibrillations at the time of new AICD lead placement. The average total energy delivered was 85±29 J. None of these patients had detectable myocardial injury. Ten patients had defibrillations by the AICD patches at the time of bypass operation. One patient in this group developed acute myocardial infarction in the inferior wall after posterior descending coronary bypass operation, as detected by electrocardiogram, 99mTc pyrophosphate scanning, and CPK-MB analysis. Fifteen patients were evaluated for spontaneous AICD discharges. Thirteen had a maximum of five consecutive shocks, and cumulative energy delivered was not greater than 330 J. None of these patients had detectable injury. Two patients had CPK-MB release of 15.3% and 7.5%, respectively. One of these patients had a positive 99mTc pyrophosphate scan. These two patients received 12 and 17 rapid and consecutive AICD discharges, respectively, with cumulative delivered energy of 360 and 510 J, respectively. Twenty-one patients in this series developed nonspecific ST-T segment changes that normalized within 48–72 hours after AICD discharges. We conclude that 1) defibrillation efficacy testing limited to 85±29 J does not cause detectable myocardial injury; 2) spontaneous discharges of the AICD with a maximum cumulative energy of 330 J does not result in detectable myocardial injury when the rate of discharge for five rapid shocks is less than one shock per minute; 3) rapid consecutive (more than 12 at less than 1 minute apart) AICD discharges can result in a positive 99mTc pyrophosphate scan and CPK-MB release (however, electrocardiographic changes consistent with new myocardial infarction are rare); 4) the appearance of new Q waves or persistent (after 48–72 hours) T wave changes, together with significant release of CPK isoenzymes, is probably because of myocardial infarction caused by vascular occlusion; and 5) transient (48–72 hours) ST-T segment changes are common after AICD discharges. (Circulation 1990;81:1482–1487)

With the introduction of the automatic implantable cardioverter/defibrillator (AICD), a steadily growing population of patients is being exposed to repeated defibrillations applied directly to the heart. Laboratory animal data suggest that detectable myocardial injury can occur with directly applied single shocks of 30 J or multiple shocks of 10 J each.1 In humans, several studies have documented myocardial injury as a result of direct cardiac defibrillation.2–5 No report, however, has specifically focused on whether defibrillators using two-patch electrode configuration, implanted over

From Cardiovascular Research Laboratory and the Nuclear Cardiology Laboratory, University of Wisconsin-Milwaukee Clinical Campus, Sinai Samaritan Medical Center, Milwaukee. Address for correspondence: Boaz Avitall, MD, PhD, Sinai Samaritan Medical Center, Mount Sinai Campus, 950 North Twelfth Street, W-429, Milwaukee, WI 53201.

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TABLE 1. Clinical Characteristics of the Patients

<table>
<thead>
<tr>
<th>Clinical characteristics</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (male/female)</td>
<td>13/2</td>
<td>8/1</td>
<td>10/0</td>
<td>13/2</td>
</tr>
<tr>
<td>Age (mean±SD yr)</td>
<td>62.2±9</td>
<td>60±4</td>
<td>64±11</td>
<td>57±10</td>
</tr>
<tr>
<td>LVEF (mean±SD)</td>
<td>37±12</td>
<td>35±12</td>
<td>41±12</td>
<td>33±15</td>
</tr>
<tr>
<td>Clinical arrhythmias (VT/VF)</td>
<td>5/10</td>
<td>6/3</td>
<td>2/8</td>
<td>8/7</td>
</tr>
<tr>
<td>Induced arrhythmias (SMVT/PVT)</td>
<td>10/5</td>
<td>9/0</td>
<td>7/3</td>
<td>13/2</td>
</tr>
</tbody>
</table>

Group 1, AICD generator replacement only, preexisting patches; group 2, new AICD implants, screw-in leads plus patches; group 3, new AICD implants, screw-in leads plus patches, plus CABG; group 4, chronic implants, multiple AICD discharges.

Methods

Patient Population

Forty-nine patients who received AICD implants were prospectively studied during 18 months. The population data are presented in Table 1. The 49 patients comprised four groups.

Group 1 included 15 patients with previously implanted AICD generators and leads who required AICD generator replacement only (preexisting patches). In 11 of 15 patients, patches had been placed directly over the epicardial surface during coronary artery bypass graft (CABG) surgery. The remaining four patients did not have bypass surgery, and their patches had been placed over the external surface of the pericardium.

Group 2 was made up of nine patients who were evaluated at the time of initial surgery for patch placement and AICD generator implant (new AICD implants, screw-in leads plus patches). All patches in this group were applied to the external surface of the pericardium.

Group 3 consisted of 10 patients studied at the time of CABG and epicardial patch placement (new AICD implants, screw-in leads plus patches, plus CABG).

Group 4 included 15 patients who were admitted after multiple spontaneous AICD discharges (chronic implants, multiple AICD discharges). None had angina before the AICD discharges. A fifth group included 16 patients who had undergone multiple CABG without implantation of AICD electrodes. The fifth group of patients served as a reference for group 3. These two groups of patients had similar aortic cross-clamp times, and no patient in the reference group had electrocardiographic or creatine phosphokinase (CPK)-isozyme evidence of perioperative myocardial infarction.

Intraoperative Defibrillations by AICD Patches in Groups 1, 2, and 3

Ventricular fibrillation was induced by nonrectified 60 Hz current applied to the heart for 2–4 seconds by the patch electrodes. After 10 seconds of ventricular fibrillation, a fixed-tilt defibrillation test pulse, initially set to deliver 15 J, was applied to the patch electrodes from an external cardioverter defibrillator unit with an effective capacitance of 135±10 μF. If ventricular fibrillation persisted after the test shock, a 30–40 J rescue shock was immediately applied. If defibrillation with the test shock was successful, the process was repeated at 10 J and, if successful, at 5 J. To ascertain the proper function of the AICD unit after it was attached to the electrodes, the heart was fibrillated and the AICD was allowed to sense and convert the arrhythmia.

Spontaneous AICD Shocks in Group 4

In 13 patients, the number of consecutive spontaneous AICD discharges was in the range of three to 11. In these 13 patients, no more than five discharges occurred in rapid succession within a 5–10-minute period. Two of these 13 patients had AICD discharges because of atrial fibrillation with ventricular responses in excess of the AICD rate threshold. Two additional patients received 12 and 17 clinically appropriate AICD discharges, respectively, in rapid succession.

All the patients evaluated in this study received Cardiac Pacemaker Incorporated patch electrodes. In all patients, the posterior patch was 20 cm². The anterior patch was 20 cm² in 77% of the patients and 10 cm² in the remainder of the patients.

Method for Detection of Myocardial Injury

ECGs were recorded daily for at least 3 days after shocks. Criteria for myocardial injury included new Q waves or new nonreversible T wave inversion in two
electrocardiographic leads. All other transient ST-T segment changes were considered nonspecific.

Serial CPK and CPK-MB analysis was performed within 3 hours after defibrillations and every 8 hours until peak MB fractions were recorded. Myocardial injury was considered present when the total CPK increased to more than 60 IU/l in conjunction with a CPK-MB fraction more than 5% of the total CPK.6

Tc-PYP scans were acquired 24–48 hours after AICD shocks. Scans were obtained by using a portable small field-of-view gamma camera and a low-energy all-purpose collimator. Images were obtained 2 hours after intravenous administration of 20 mCi of radionuclide.7 Tc-PYP scans were blindly evaluated by three experienced observers. Final readings resulted from agreement of at least two of the three observers. A positive Tc-PYP scan was defined as focal uptake in the area of the heart that was unrelated to rib or costochondral cartilage and that cleared 2 weeks later on a follow-up scan.

Statistical Analysis

Standard analysis of variance and t tests were used when appropriate to determine statistical differences between the groups.

Results

Patch age, defibrillation threshold, cumulative total energy delivered, energy in a single discharge, and number of discharges are shown in Table 2. In groups 2 and 3, AICD patches were freshly placed, and no age or previous defibrillation threshold is indicated. For groups 1 and 4, patch age was not significantly different. Defibrillation threshold at the time of implant did not significantly differ among the groups. The cumulative energy delivered in groups 1, 2, and 3 was not significantly different; however, the total energy and number of discharges delivered in patients in group 4 were significantly higher (p<0.001) than in patients in any of the other groups.

None of the patients who underwent AICD generator replacement (group 1) or patients undergoing new AICD system implants (group 2) developed electrocardiographic, CPK-isoenzyme, or Tc-PYP-scan abnormalities suggesting acute myocardial injury. Five of 15 (33%) patients in group 1 and seven of nine (78%) patients in group 2 exhibited transient lateral or inferolateral ST-T changes that normalized within 48–72 hours after the procedure.

Of the 10 patients who underwent bypass surgery in addition to patch placement (group 3), two developed positive Tc-PYP scans. One of these patients had new inferior wall Q waves and 5.8% CPK-MB fraction after placement of an aortocoronary vein graft to the posterior descending coronary artery. The other patient had transient anterior ST elevation with a peak CPK-MB fraction of 4%. Six patients (60%) in this group had transient ST-T changes.

Further analysis of the CPK and CPK-MB data is shown in Figure 1. The lowest total CPK and CPK-MB values among groups 1, 2, and 3 were in group 1, in which there was no cardiac manipulation. In three of these patients, peak CPK was less than 60 IU and CPK-MB was less than 1 IU/l. CPK-isoenzyme release was significantly greater in groups 2 and 3 than in group 1. The highest values were observed in group 3, which was the group in which cardiopulmonary bypass and aortic cross-clamping were used. Patients in group 3, and to a lesser extent in group 2, underwent cardiac manipulation and placement of myocardial screw-in electrodes for rate determination. Isoenzyme data for patients in group 3 were compared with data from a reference group of 16 patients who underwent only myocardial revascularization with a similar number of bypass grafts and aortic cross-clamp times. These patients exhibited no electrocardiographic or CPK-MB evidence of perioperative myocardial infarction. CPK-MB-isoenzyme release was significantly greater (p<0.01) in patients receiving AICD systems than those undergoing myocardial revascularization without AICD system implants (31±10 vs. 18.8±8), which is probably related to the insertion of the myocardial screw-in
Table 3. Cardiac Enzyme Analysis in Patients With Chronic Implants Who Received Spontaneous AICD Discharges (Group 4)

<table>
<thead>
<tr>
<th>Analysis data</th>
<th>(-)-CPK-MB</th>
<th>(+)-CPK-MB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>Total energy delivered (J)</td>
<td>198±76</td>
<td>360, 510</td>
</tr>
<tr>
<td>Discharges (n)</td>
<td>3–11</td>
<td>12, 17</td>
</tr>
<tr>
<td>Maximum single discharge (J)</td>
<td>28–32</td>
<td>28–32</td>
</tr>
<tr>
<td>Peak CPK (IU/l)</td>
<td>65±48</td>
<td>943, 376</td>
</tr>
<tr>
<td>Peak CPK-MB (IU/l)</td>
<td>2±2</td>
<td>70, 75</td>
</tr>
<tr>
<td>Peak CPK-MB (%)</td>
<td>3±2.6</td>
<td>7, 5, 15.3</td>
</tr>
<tr>
<td>Positive Tc-PYP scan</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

AICD, automatic implantable cardioverter/defibrillator; Tc-PYP, technetium 99m pyrophosphate.

electrodes. Differences in total CPK or the percentage of CPK-MB between these two groups of patients, however, were not significant. In group 4, two patients received a large number (12 and 17, respectively) of AICD discharges in rapid succession and had marked elevation of CPK isoenzymes within 8 hours after the event, with rapid CPK decay. The first patient exhibited transient mild anterior ST segment elevation and a positive Tc-PYP scan. The second patient had no negative Tc-PYP scan and no electrocardiographic changes. In the remaining 13 patients in group 4, there were three to 11 AICD discharges with no more than five successive shocks (Table 3). Two of these patients had anterolateral, and one patient had inferolateral (20%) transient ST-T flattening.

Influence of Patch Location

Four patients in group 1 had patches located over the pericardium, whereas the remaining 11 patients had patches located intrapericardially. Peak CPK release was significantly lower with extrapericardial patches; however, the peak CPK-MB and percentage of CPK-MB were not significantly different (Table 4).

Table 4. Epicardial Versus Pericardial Patch Placement in Group 1

<table>
<thead>
<tr>
<th>Group data</th>
<th>Epicardial</th>
<th>Pericardial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n)</td>
<td>11</td>
<td>4</td>
</tr>
<tr>
<td>Patch age (mo)</td>
<td>20.3±10</td>
<td>29.7±34</td>
</tr>
<tr>
<td>Previous DFT (J)</td>
<td>8.9±2.1</td>
<td>8.3±4.7</td>
</tr>
<tr>
<td>Current DFT (J)</td>
<td>7.8±3.4</td>
<td>7.7±2.4</td>
</tr>
<tr>
<td>Total energy delivered (J)</td>
<td>86.5±26*</td>
<td>60±0</td>
</tr>
<tr>
<td>Shocks (n)</td>
<td>4±1</td>
<td>4±0</td>
</tr>
<tr>
<td>Peak CPK (IU/l)</td>
<td>123.1±81*</td>
<td>70±31</td>
</tr>
<tr>
<td>Peak CPK-MB (IU/l)</td>
<td>3.7±3</td>
<td>2.9±1.9</td>
</tr>
<tr>
<td>Peak CPK-MB (%)</td>
<td>2.05±2</td>
<td>2.1±1.5</td>
</tr>
</tbody>
</table>

Values are mean±SD. DFT, defibrillation threshold. *p<0.05, epicardial vs. pericardial.

Discussion

Direct current defibrillation has the potential to cause myocardial damage.1–5,8–11 Previous work by Doherty et al9 in dogs has shown that graded intensity defibrillations of 10–90 J result in progressive myocardial injury. With an energy level of less than 40 J, histological damage was limited to the epicardial surface. An energy level of 30 J or greater resulted in depletion of creatinine kinase. Thirty to 50 J was found to be the threshold for a significant reduction in thallium 201 uptake. As the energy level increased to 90 J, injury extended into the myocardial wall as detected by the distribution of 99mTc pyrophosphate accumulation. More importantly, when four successive 10-J defibrillations were applied less than 1 minute apart, more extensive and deeper myocardial injury was detected than when defibrillations were applied at a slower rate. Twenty repeated discharges of 50 J or greater resulted in extensive epicardial myocyte damage. Measurements of epicardial temperature showed a steep increase in temperature with multiple successive discharges of 30 J. If the discharges were applied at a rate slower than one per minute, the temperature returned to baseline between discharges.1 This increase in temperature as well as the accumulation of intracellular electron-dense particles, excess calcium, and disintegration of the plasma membrane5,12 can participate in the formation of myocardial injury as a result of rapid high-energy multiple defibrillations. In patients who had multiple defibrillations, pathological changes in the myocardium beneath AICD-patch electrodes were found to consist of contraction-band necrosis and loss of myocytes confined to the area under the patches. In that pathological study, it was estimated that less than 2% of the total myocardial mass was affected.10 No determination, however, was made regarding whether myocardial damage was a cumulative process after each defibrillation or whether it was established after the first series of defibrillations. Furthermore, many of the patients received multiple external defibrillations just before death. Mechanical injury can inflict additional damage on the epicardium as noted in humans10 and the dog model.11 With 240-J external defibrillation in dogs, myocardial necrosis was significantly greater when multiple defibrillations were applied 15 seconds apart. This damage was significantly reduced if the frequency of defibrillations was less than one every 3 minutes and if the energy was delivered into larger sized electrodes.8 Similarly, the two patients presented with myocardial injury after spontaneous discharges in this study received 30-J shocks in rapid succession. It can be concluded that the intensity, rapidity of discharges, and electrode size are the primary factors
determining myocardial injury. It seems, however, that the threshold for such injury might be higher in humans with chronically implanted AICDs. Although the animal model has been used extensively to evaluate myocardial injury induced by external or internal defibrillation, the extrapolation of these data to humans might not be accurate. Unlike the dog heart, the human heart, with AICD patches attached, often has an anterior fat pad that provides a buffer between the patches and the myocardium itself. Attachment of the patches to the parietal pericardium provides another layer of protection, whereas the fibrosis induced by the mechanical irritation of the AICD patches11 can create an additional tissue buffer between the patches and the myocardium.

Defibrillation injury in surgical patients can complicate the detection of perioperative myocardial infarction because cardiac surgical manipulation before and during AICD electrode placement causes cardiac injury. The surgical injury must be quantified before the assessment of defibrillation injury. In group 1, which had no cardiac manipulation, the CPK-MB release was the lowest. In groups 2 and 3, however, CPK-MB release was significantly greater, suggesting that both groups sustained mild myocardial injury caused by AICD screw-in leads and patch placement. In group 3, CPK-MB release was higher than in group 2 and probably occurred because of the cardiac revascularization surgery. Because the contribution of the screw-in sensing lead patch placement, defibrillations, and bypass surgery to CPK-MB release could not be distinguished, we compared the CPK-MB release in patients in group 3 with patients in a reference group that had undergone similar uncomplicated bypass surgery. Peak CPK-MB release was significantly greater in patients who received both CABG and AICD leads than in patients who had cardiac revascularization alone. Patients in group 3 also differed from patients in group 2 because the pericardium was opened in the former patients and the patches placed on the visceral pericardium. In patients in group 2, the patches were placed on the parietal pericardium. Whether that difference was in any way responsible for the difference in CPK release between the two groups is unknown.

Only three of the 49 patients evaluated in this study had CPK-MB of more than 5%. One of these patients sustained myocardial infarction perioperatively with new inferior Q waves and a positive Tc-PYP scan. The concordance of all three modalities as noted has been shown to be more specific for perioperative myocardial infarction after cardiac revascularization surgery.13 Twenty-one of 49 patients evaluated in this study had ST-T changes that normalized within 48–72 hours after AICD discharges. Dahl et al12 reported that ST segment elevation after external defibrillation in the dog was correlated with myocardial fibrosis. One of our patients received 12 spontaneous defibrillations from the AICD and presented with transient mild anterior ST segment elevation, as well as a positive Tc-PYP scan and positive CPK-MB release. That patient had previous inferior myocardial infarction. The patient who received 17 defibrillations from the AICD had previous anterior myocardial infarction. Neither of these patients had angina immediately before the AICD discharges.

Influence of AICD Patch Location

In this study, patches were placed inside the pericardial space if the patient underwent concomitant cardiac revascularization surgery. In patients who underwent patch insertion and AICD insertion alone, the patches were placed on the parietal pericardium. No significant difference in defibrillation threshold was found between these two groups of patients either immediately after the patch placement or later. Additionally, no significant change in defibrillation threshold occurred during the average 23 months of follow-up. This is an indication that the patches were in a steady-state condition before discharges.

We conclude the following: 1) Defibrillation-threshold evaluation, as described in this study, does not cause detectable myocardial injury. 2) Spontaneous defibrillation by the AICD with two-patch electrodes does not result in detectable myocardial injury with maximum cumulative energy of 330 J. The rate of discharge cannot exceed five rapid shocks with less than 1 minute between shocks. 3) In the two patients who received 12–17 rapid consecutive AICD discharges, myocardial injury caused by defibrillation is probable. The number and frequency of defibrillations sets these two patients apart from the rest of the patients who received spontaneous defibrillation. 4) No new Q waves were present after defibrillation in patients with new or preexisting AICD patches or after spontaneous AICD discharges. The appearance of new Q waves or nonreversible T wave changes, together with significant release of CPK isoenzymes, is most likely to represent myocardial infarction caused by vascular occlusion. 5) Transient (48–72 hours) ST-T segment changes are common after AICD discharges.

Acknowledgments

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