Both primary and secondary preventive trials have demonstrated that implantable cardioverter-defibrillators (ICDs) reduced mortality from sudden cardiac death because of malignant ventricular arrhythmia.\textsuperscript{1, 2} Despite this survival advantage, several studies have demonstrated that ICD shock, whether it is appropriate, is associated with increased risk of mortality among patients with reduced left ventricular (LV) systolic function.\textsuperscript{3–6} Furthermore, defibrillation threshold (DFT) testing at the time of ICD implantation sometimes invokes several critical complications, especially in patients with reduced LV contractility.\textsuperscript{7–9} These complications include transient ischemic attack or stroke, cardiopulmonary arrest because of refractory ventricular fibrillation (VF) or pulseless electrical activity, cardiogenic shock, embolic events, and death. Although ICD shock is related to short- and long-term critical complications in patients with LV systolic dysfunction, the association between electric defibrillation and cardiac function has been investigated in only a few animal experimental studies,\textsuperscript{10–12} and there are few clinical data regarding the effect of ICD shock on cardiac function and its association with tissue damage and subsequent hemodynamic change in patients with systolic heart failure.

**Background**—We investigated the acute effects of implantable cardioverter-defibrillator shock on myocardium, cardiac function, and hemodynamics in relation to left ventricular systolic function.

**Methods and Results**—We studied 50 patients who underwent implantable cardioverter-defibrillator implantation and defibrillation threshold (DFT) testing: 25 patients with left ventricular ejection fraction (LVEF) $\geq 45\%$ and 25 patients with LVEF $<45\%$. We measured cardiac biomarkers (creatine kinase, creatine kinase-MB, myoglobin, cardiac troponin T and I, and N-terminal probrain natriuretic peptide). Left ventricular relaxation was assessed by global longitudinal strain rate during the isovolumetric relaxation period using speckle-tracking echocardiography. Blood sampling and echocardiography were performed before, immediately after, and 5 minutes and 4 hours after DFT testing. Mean arterial pressure was measured directly during DFT testing. Cardiac biomarkers showed no significant changes in either group. LVEF was decreased until 5 minutes after DFT testing and had recovered to the baseline at 4 hours in the group with reduced LVEF ($P<0.001$), whereas LVEF reduction was not observed in the group with preserved LVEF ($P=0.637$). Global isovolumetric relaxation period was decreased until 5 minutes after DFT testing and had recovered to the baseline at 4 hours in both groups (preserved LVEF: $0.39\pm0.14$ versus $0.23\pm0.13^* \text{ versus } 0.23\pm0.13^* \text{ versus } 0.40\pm0.13 \text{ s}^{-1}$, $^*P<0.001$ versus baseline; reduced LVEF: $0.15\pm0.05$ versus $0.08\pm0.04^* \text{ versus } 0.09\pm0.04^* \text{ versus } 0.15\pm0.05 \text{ s}^{-1}$, $^*P<0.001$ versus baseline, repeated-measures ANOVA). Time to recovery of mean arterial pressure to the baseline was prolonged in the group with reduced LVEF ($P<0.001$).

**Conclusions**—Implantable cardioverter-defibrillator shock transiently impairs cardiac function and hemodynamics especially in patients with systolic dysfunction, although significant tissue injury is not observed. (**Circ Arrhythm Electrophysiol. 2012;5:898-905**.)

**Key Words:** echocardiography ■ hemodynamics ■ implanted cardioverter defibrillators ■ ventricular fibrillation ■ cardiac function
enabled us to quantify myocardial deformation without angle dependency,\textsuperscript{13} and global SR during the isovolumetric relaxation period (SR\textsubscript{vr})\textsuperscript{3} provides more accurate assessment of LV relaxation than conventional parameters.\textsuperscript{14} In this study, we investigated the effects and mechanisms of ICD shock on myocardial functions by echocardiography, direct central arterial pressure measurement, and measurement of cardiac biomarkers with respect to LV systolic function.

**Methods**

**Study Sample**

The study population consisted of 50 consecutive patients who were admitted to our institution to undergo transvenous ICD implantation and DFT testing between April 2008 and December 2009. The underlying heart diseases were ischemic cardiomyopathy in 13 patients, dilated cardiomyopathy in 9 patients, hypertrophic cardiomyopathy in 6 patients, cardiac sarcoidosis in 3 patients, and idiopathic dilated cardiomyopathy in 19 patients. The patients were divided into 2 groups according to the preoperative LV ejection fraction (LVEF): a group of patients with preserved LVEF (LVEF ≥45%) and a group of patients with reduced LVEF (LVEF <45%).\textsuperscript{15,16} All tests that were performed were approved by the medical ethical review committees of Okayama University Hospital. Informed consent was obtained from each patient.

**Study Protocol**

The study protocol is summarized in Figure 1. ICD implantation was performed using local anesthesia combined with sedation only for DFT testing. At the end of ICD implantation, we induced VF by T-wave shock after monitored anesthesia care using a bolus injection of thiopental (4 mg/kg). For minimizing change in loading condition during monitored anesthesia care, saline infusion rate was set at 0.33 mL/min. Defibrillation shock was fixed to 20J and automatically delivered from the ICD after detection of VF. We repeated the same protocol 5 minutes after the first DFT testing and did not use a step-down protocol in any of the subjects. We performed venous blood sampling and echocardiographic examination before, immediately after, and 5 minutes and 4 hours after 2 consecutive DFT testing. Vascular access was achieved through the femoral artery, and central arterial pressure was continuously monitored in the ascending aorta during DFT testing.

**Analysis of Laboratory Data**

To evaluate myocardial injury by DFT testing, we measured cardiac biomarkers: serum levels of creatine kinase (CK), CK-MB fraction (CK-MB), myoglobin, cardiac troponin T, cardiac troponin I, and N-terminal pro-brain natriuretic peptide (NT-proBNP). CK activity was measured with CicaLiquid reagents (Kanto Chemical, Tokyo, Japan) on a Bio-Majesty analyzer (Nihondenshi, Tokyo, Japan), with upper normal limits of 287 U/L for men and 163 U/L for women. The CK-MB activity was determined using a commercially available immunoinhibition assay (CicaLiquid CK-MB; Kanto Chemical, Tokyo, Japan), with an upper normal limit of 60 ng/mL. Cardiac troponin T was assessed by an electrochemiluminescence immunoassay on an Elecsys 1010 analyzer (Roche Diagnostics, Mannheim, Germany). The lower limit of detection was 0.01 ng/mL, and the discrimination level used for myocardial injury was 0.10 ng/mL. Cardiac troponin I was determined using a 2-site immunoenzymatic assay (Access AccuTnl, Beckman Coulter, Brea, CA), with an upper normal limit of 0.50 ng/mL. NT-proBNP was measured using an electrochemiluminescence immunoassay on an Elecsys 1010 analyzer (Roche Diagnostics), with an upper normal limit of 125 pg/mL.

**Analysis of Echocardiographic Data**

All echocardiographic studies were performed with Vivid 7 (GE Healthcare, Milwaukee, WI). We measured LV volume and ejection fraction according to the recommendations of the American Society of Echocardiography.\textsuperscript{11} From mitral flow velocity pattern, we measured peak mitral inflow early diastolic and atrial filling velocities and the E-wave deceleration time. Peak early diastolic mitral annular velocities were measured at septal and lateral mitral annular sites by pulsed tissue Doppler imaging, and then the average values were used for analysis (e’ velocity). The ratio of peak E velocity to e’ velocity (E/e’) was calculated as a surrogate for LV filling pressure. Longitudinal SR analysis was performed using the speckle-tracking system in an EchoPAC PC (GE Healthcare) as previously described.\textsuperscript{14} In brief, after tracing the entire LV endocardium, the displacement of speckles of the myocardium was analyzed automatically through the cardiac cycle in the speckle-tracking system. Then the SR curve of each segment was displayed and approved. LV global SR was calculated with the use of the entire length of the LV myocardium, and peak global SR during the isovolumetric relaxation period was defined as global SR\textsubscript{ivc}. The global SR\textsubscript{ivc} values from the 3 apical views were averaged and used for analysis. All echocardiographic measurements and analysis were performed offline by an experienced investigator (N.T.), with no clinical information about the patients.

The following measures were taken to obtain adequate echocardiographic images for analysis promptly and maintain operative field sterility: (1) we enrolled only patients with optimal echocardiographic images, (2) the transducer position was fixed at apical impulse for minimizing loss of time and maintaining sterility because an apical

![Image](http://circep.ahajournals.org/content/120/2/899/F1.large.jpg)

**Figure 1.** Outline of the clinical study protocol. ICD indicates implantable cardioverter-defibrillator; DFT, defibrillation threshold.
window was sufficient for acquiring all data as mentioned above, and (3) the operative field and catheter insertion site were carefully covered with sterile surgical drapes.

Analysis of Hemodynamic Data
Continuous measurements of systolic and diastolic arterial pressures were performed at the ascending aorta during DFT testing. Mean arterial pressure (MAP) was obtained by direct integration of the blood pressure curve. Time to reach baseline MAP was defined as the interval between the second ICD shock and the time MAP returned again.

Statistical Analysis
Data are expressed as mean±SD. Unpaired t test was used to detect statistical differences for continuous variables with normality of data distributions between 2 groups, and categorical data and percentage frequencies were analyzed by the Fisher exact test. Serial data (before and after the procedure) were analyzed by linear mixed-effects models, and 2-way repeated-measures ANOVA was conducted. If a significant difference between 2 groups or among 4 time points was detected by a global test, ad hoc multiple comparison was performed. Central arterial pressures before and after DFT testing were compared by paired t test. Ten subjects were randomly selected from each group and analyzed blindly by 2 independent investigators (N.T. and H.O.) to assess the intraclass correlation coefficient for evaluating reproducibility of longitudinal SR measurements. P<0.05 was considered statistically significant. All analyses were performed with JMP 9 (SAS Institute, Cary, NC).

Results
Clinical Characteristics
Table 1 shows the characteristics of the study population. There were no significant differences in age, sex, and body surface area between the 2 groups. New York Heart Association functional class was higher in the group with reduced LVEF than in the group with preserved LVEF. The group with reduced LVEF more frequently included ischemic and dilated cardiomyopathies than the group with preserved LVEF. Idiopathic ventricular fibrillation was the major cause of ICD implantation in the group with preserved LVEF. Concomitant cardiovascular drug therapy was common in the group with reduced LVEF.

Serial Changes of Serum Markers Before and After DFT Testing
Serial changes of serum markers are listed in Table 2. At baseline, there were no differences in biomarkers except for NT-proBNP between the groups before DFT testing. Baseline NT-proBNP was significantly higher in the group with reduced LVEF than in the group with preserved LVEF (P<0.002).

All patients received 2 consecutive 20-J shocks with a 5-minute interval. All induced VFs were successfully terminated by the first 20-J shock, and shocks neither higher nor lower than 20 J were delivered. Although the response to DFT testing in CK-MB and NT-proBNP differed between the groups using repeated-measures ANOVA, DFT testing did not cause significant changes in CK, CK-MB, myoglobin, and NT-proBNP in either group. DFT testing slightly increased cardiac troponin T in the group with preserved LVEF and cardiac troponin I in the group with reduced LVEF, but these values did not exceed the normal ranges (Table 2).

Table 1. Baseline and Clinical Characteristics of the Study Population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preserved LVEF (n=25)</th>
<th>Reduced LVEF (n=25)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>55±13</td>
<td>57±14</td>
<td>0.661</td>
</tr>
<tr>
<td>Sex, male</td>
<td>20 (80)</td>
<td>15 (60)</td>
<td>0.217</td>
</tr>
<tr>
<td>Body surface area, m²</td>
<td>1.72±0.20</td>
<td>1.66±0.19</td>
<td>0.254</td>
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<tr>
<td>NYHA functional class</td>
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<td>&lt;0.001</td>
</tr>
<tr>
<td>I</td>
<td>19</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>6</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>0</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Cardiac disease history</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic cardiomyopathy</td>
<td>1 (4)</td>
<td>12 (48)</td>
<td>0.001</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>0 (0)</td>
<td>9 (36)</td>
<td>0.002</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>5 (20)</td>
<td>1 (4)</td>
<td>0.190</td>
</tr>
<tr>
<td>Cardiac sarcoidosis</td>
<td>0 (0)</td>
<td>3 (12)</td>
<td>0.235</td>
</tr>
<tr>
<td>Idiopathic ventricular fibrillation</td>
<td>19 (76)</td>
<td>0 (0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Concomitant cardiovascular therapies</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE inhibitors/ARBs</td>
<td>6</td>
<td>19</td>
<td>0.001</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>9</td>
<td>23</td>
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<tr>
<td>Calcium channel blockers</td>
<td>3</td>
<td>1</td>
<td>0.609</td>
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<tr>
<td>Diuretics</td>
<td>3</td>
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<td>&lt;0.001</td>
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<tr>
<td>Class III antiarrhythmic agent</td>
<td>9</td>
<td>5</td>
<td>0.345</td>
</tr>
<tr>
<td>Statins</td>
<td>3</td>
<td>5</td>
<td>0.702</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; NYHA, New York Heart Association; ACE, angiotensin-converting enzyme; ARBs, angiotensin II receptor blockers. Values are n (%) or mean±SD.
Serial Changes of Echocardiographic Parameters Before and After DFT Testing

Serial changes of echocardiographic parameters in both groups are demonstrated in Table 3. In baseline echocardiographic data, LV end-diastolic volume and end-systolic volume were significantly greater in the group with reduced LVEF (both $P<0.001$). Parameters of transmitral flow showed no significant differences between the 2 groups. The $e'$ velocity was significantly lower, and $E/e'$ was greater in the group with reduced LVEF than in the group with preserved LVEF ($P<0.001$ and $P=0.042$, respectively). Global SRIVR was less in the group with reduced LVEF than in the group with preserved LVEF ($P<0.001$).

By repeated-measures ANOVA, the response to DFT testing differed between the groups in all echocardiographic parameters listed in Table 3. In the group with reduced LVEF, LVEF decreased immediately after DFT testing and had recovered to the baseline level 4 hours after the test, whereas it showed no significant changes after DFT testing in the group with preserved LVEF ($P<0.001$ and $P=0.042$, respectively). Global $SR_{IVR}$ was less in the group with reduced LVEF than in the group with preserved LVEF ($P<0.001$).

Changes of Central Arterial Pressure Before and After DFT Testing

After monitored anesthesia care, although systolic and diastolic blood pressures were significantly decreased in both groups (group with preserved LVEF: 127±14 versus 121±13 mm Hg, $P=0.004$; 74±12 versus 67±8 mm Hg, $P=0.016$, respectively; reduced LVEF group: 112±18 versus 103±18 mm Hg, $P=0.013$; 66±8 versus 58±9 mm Hg, $P=0.005$, respectively), decrease in heart rate was not significant (group with preserved LVEF: 63±9 versus 61±8 beats per minute, $P=0.092$; group with reduced LVEF: 70±10 versus 68±13 beats per minute, $P=0.337$).

Central arterial pressures before and after DFT testing are shown in Table 4. The group with reduced LVEF had lower systolic and diastolic arterial pressures and MAP than the group with preserved LVEF before DFT testing. DFT testing caused transient, yet significant, decreases in systolic and diastolic arterial pressures and MAP in both groups. Time to recovery of MAP to the baseline level was more prolonged in the group with reduced LVEF than in the group with preserved LVEF ($43±24$ versus $12±10$ s; $P<0.001$).

Discussion

In the present study, we first found that ICD shock caused LV systolic dysfunction in patients with reduced LVEF as well as LV diastolic dysfunction, irrespective of baseline LVEF in the clinical setting. Impaired ventricular relaxation lasted at least 5 minutes after ICD shock in both groups, as demonstrated by sustained reduction of global $SR_{IVR}$. However, serum cardiac
markers were unaffected or did not exceed normal values at any time point in either group, suggesting that transient ventricular dysfunction was not a result of myocardial injury. Furthermore, time to recovery of central arterial pressure to the baseline level was significantly longer in patients with reduced LVEF than in patients with preserved LVEF.

Effect of ICD Shock on Cardiac Function

The impact of internal cardioversion on LV systolic function remains controversial. Some previous echocardiographic studies showed that LV systolic function was unaffected after internal cardioversion during ICD implantation, but LV systolic function was assessed by LVEF from the apical 4-chamber view only or the LV fractional area change from a single-plane, transgastric, short-axis view using transesophageal echocardiography. In contrast, a previous animal study demonstrated that contractile dysfunction was provoked after defibrillator shock given directly to the myocardium. In the present study, LV systolic dysfunction after DFT testing was limited in patients with reduced LVEF, and this result does not contradict previous observations that cardiac output was deteriorated only in patients with low LVEF after inductions of ICD shock.

In contrast to the effect of DFT testing on systolic function, DFT testing promoted transient diastolic dysfunction in all patients, irrespective of preoperative LVEF in the present study. Experimental studies revealed that the time constant of LV relaxation was prolonged, and LV end-diastolic pressure was increased after direct current shock even in normal hearts. These results indicated that electric defibrillation impaired LV relaxation and deteriorated LV diastolic function. This is the first study demonstrating that defibrillation shock induced transient LV diastolic dysfunction in humans. We confirmed that reduced global SRIVR, which is a new surrogate of LV relaxation, was sustained for at least 5 minutes after DFT testing and had recovered to the baseline level 4 hours after DFT testing in both groups, indicating that ICD shock impaired LV relaxation but that it was temporal in the clinical setting. Transient impairment of both systolic and diastolic LV dysfunctions by DFT testing in patients with reduced LVEF is associated with hemodynamic instability. Prolonged recovery of central arterial pressure may have a pivotal role in the occurrence of DFT testing–related critical complications.

The mechanisms of cardiac dysfunction after ICD shock remain uncertain. Serum cardiac biomarkers were not increased by DFT testing, and it was likely that significant myocardial injury did not occur. One possible explanation is abnormal Ca²⁺ transient induced by defibrillation. It has been reported that electric shock prolonged the time decay of the Ca²⁺ transient and elevated diastolic intracellular calcium concentration even in normal myocytes and that abnormal Ca²⁺ handling leads to impairment of LV relaxation. Additionally, excessive intracellular Ca²⁺ overload results in contractile

Table 3. Serial Changes of Echocardiographic Parameters Before and After DFT testing

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>Immediately After</th>
<th>5 Minutes After</th>
<th>4 Hours After</th>
<th>Preserved LVEF vs Reduced LVEF (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preserved LVEF</td>
<td>61±6</td>
<td>61±7</td>
<td>61±7</td>
<td>62±6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reduced LVEF</td>
<td>27±9</td>
<td>23±9*</td>
<td>22±8*</td>
<td>27±9</td>
<td></td>
</tr>
<tr>
<td>E/A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preserved LVEF</td>
<td>1.1±0.4</td>
<td>1.1±0.4</td>
<td>1.1±0.4</td>
<td>1.1±0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reduced LVEF</td>
<td>0.9±0.8</td>
<td>0.8±0.6</td>
<td>0.8±0.6</td>
<td>0.8±0.6</td>
<td></td>
</tr>
<tr>
<td>E-wave deceleration time, ms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preserved LVEF</td>
<td>246±54</td>
<td>230±48</td>
<td>238±50</td>
<td>244±53</td>
<td>0.030</td>
</tr>
<tr>
<td>Reduced LVEF</td>
<td>272±82</td>
<td>244±71</td>
<td>259±66</td>
<td>268±85</td>
<td></td>
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<tr>
<td>Peak e’ velocity, cm/s</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Preserved LVEF</td>
<td>7.3±3.8</td>
<td>5.1±3.4</td>
<td>6.6±4.6</td>
<td>6.7±4.5</td>
<td>&lt;0.001</td>
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<tr>
<td>Reduced LVEF</td>
<td>3.4±1.9</td>
<td>2.8±1.6</td>
<td>3.2±2.0</td>
<td>3.6±2.5</td>
<td></td>
</tr>
<tr>
<td>E/e’</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preserved LVEF</td>
<td>9.6±5.2</td>
<td>13.1±8.0</td>
<td>10.2±6.8</td>
<td>9.2±4.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reduced LVEF</td>
<td>14.0±8.5</td>
<td>16.8±9.4</td>
<td>14.5±10.0</td>
<td>13.0±8.9</td>
<td></td>
</tr>
<tr>
<td>Global SRIVR</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preserved LVEF</td>
<td>0.39±0.14</td>
<td>0.23±0.13†</td>
<td>0.23±0.13†</td>
<td>0.40±0.13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reduced LVEF</td>
<td>0.15±0.05</td>
<td>0.08±0.04†</td>
<td>0.09±0.04†</td>
<td>0.15±0.05</td>
<td></td>
</tr>
</tbody>
</table>

DFT indicates defibrillation threshold; LVEF, left ventricular ejection fraction; E/A, early diastolic and atrial filling; e’, early diastolic mitral annular velocity; SRIVR, strain rate during the isovolumetric relaxation period.

Values are expressed as mean±SD.

*P<0.05 vs variables at baseline (repeated-measures ANOVA, post hoc analysis).
†P<0.01 vs variables at baseline (repeated-measures ANOVA, post hoc analysis).
dysfunction.26 Because intracellular Ca2+ handling alters and diastolic intracellular Ca2+ concentration elevates in the failing heart,25,27 defibrillation shock could transiently induce both diastolic and systolic dysfunctions in patients with reduced LVEF. Another possible mechanism is the occurrence of myocardial interstitial edema after defibrillation shock. Myocardial interstitial edema is a characteristic morphological change after ICD shock12 and is associated with reduced LV distensibility and impaired relaxation.28 However, myocardial edema is thought to be a result of thermal myocardial

**Figure 2.** Serial changes of global strain rate during the isovolumetric relaxation period (SRIVR) before and after defibrillation threshold (DFT) testing in patients with preserved left ventricular ejection fraction (LVEF). A representative case of preserved LVEF. A, Before DFT testing, global SRIVR (yellow arrow) was 0.38 s⁻¹ and LVEF was 71%. B, Immediately after DFT testing, global SRIVR was 0.20 s⁻¹ and LVEF was 71%. C, At 5 minutes after DFT testing, global SRIVR was 0.12 s⁻¹ and LVEF was 73%. D, At 4 hours after DFT testing, global SRIVR was 0.36 s⁻¹ and LVEF was 70%. AVC indicates aortic valve closure; MVO, mitral valve opening.

**Figure 3.** Serial changes of global strain rate during the isovolumetric relaxation period (SRIVR) before and after defibrillation threshold (DFT) testing in patients with reduced left ventricular ejection fraction (LVEF). A representative case of reduced LVEF. A, Before DFT testing, global SRIVR (red arrow) was 0.23 s⁻¹ and LVEF was 39%. B, Immediately after DFT testing, global SRIVR was 0.08 s⁻¹ and LVEF was 34%. C, At 5 minutes after DFT testing, global SRIVR was 0.08 s⁻¹ and LVEF was 33%. D, At 4 hours after DFT testing, global SRIVR was 0.23 s⁻¹ and LVEF was 37%. AVC indicates aortic valve closure; MVO, mitral valve opening.
Implications of Echocardiographic Parameters

Although both global SR\textsubscript{IVR} and e' velocity reflect the property of LV relaxation, statistically significant reduction of e' velocity was not observed after DFT testing, and decreased global SR\textsubscript{IVR} was sustained for 5 minutes after DFT testing. This discrepancy may result from the fact that global SR\textsubscript{IVR} is a measurement of whole heart motion, whereas e' velocity is a localized measurement of mitral annular movement. The present results also support the superiority of global SR\textsubscript{IVR} to e' velocity for assessing LV relaxation.

Study Limitations

First, because the number of subjects in this study was limited, further research is needed to obtain a definitive conclusion regarding the association of ICD shock and subsequent cardiac dysfunction. Second, we cannot exclude the possibility of an effect of VF itself on cardiac dysfunction. Even though the duration of VF is short, VF causes cardiac dysfunction as a result of reduced blood flow and tissue perfusion. However, previous experimental studies have demonstrated that electric defibrillation itself also impaired intracellular Ca\textsuperscript{2+} dynamics and that it was associated with cardiac dysfunction.\textsuperscript{22–24} and a previous clinical study has proved that ICD shock strength, not VF, was most relevant to reduction in cardiac index.\textsuperscript{29} Thus, we believe that DFT testing after induced VF played a crucial role in cardiac dysfunction observed in this study. Third, all patients were receiving monitored anesthesia care during DFT testing and awakened during postprocedural investigation. However, the effect of anesthesia on the results might be small because echocardiographic parameters before DFT testing were comparable to those at 4 hours after DFT testing even though these data were acquired during sedated and waking periods, respectively. Central arterial pressure measurements were performed during the sedated period in all subjects. Fundamentally, it is impossible to deliver appropriate ICD shock during the waking period of patients. Fourth, because DFT testing is required in all patients undergoing ICD implantation at our institution, we were not able to include a control group with monitored anesthesia care and without DFT testing in this study, although the inclusion of such a control group would be helpful for assessing the impact of anesthesia on cardiac function and hemodynamics. Fifth, we cannot foreclose the possibility that the high prevalence of antihypertensive agent usage was associated with prolonged recovery of central arterial pressure in patients with reduced LVEF. Last, it is uncertain whether the current results can properly explain the mechanism of the adverse effect on long-term outcome after ICD shock. However, these results showed that ICD shock caused cardiac dysfunction at least temporarily and that subsequent hemodynamic instability, especially in patients with reduced LVEF, has the potential for worsening the clinical outcome after ICD shock in patients with heart failure.

Conclusions

ICD shock caused LV systolic dysfunction in patients with reduced LVEF and LV diastolic dysfunction irrespective of LVEF, although tissue injury determined by serum cardiac biomarkers was not observed. Furthermore, in patients with reduced LVEF, hemodynamic instability was prolonged. Therefore, even though the effects of ICD shock on cardiac function and hemodynamics are transient, clinicians should select an optimal medical therapy for avoiding ICD shock, and the necessity of DFT testing should be reconsidered, especially in patients with reduced LVEF.

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Disclosures

None.

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