Pre-Cardioversion
Median hs-cTnT: 12 ng/L
Median hs-cTnl: 5 ng/L

Post-Cardioversion
Median hs-cTnT: 13 ng/L
Median hs-cTnl: 7 ng/L

N = 98 patients

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Mayo Clinic Patient Education. Atrial Fibrillation and Atrial Flutter (MC2893). Rochester, MN: Mayo Clinic, 2017, p. 25. Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.
Absence of Significant Myocardial Injury following Elective Direct Current Cardioversion for Atrial Fibrillation

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Short Title: Lack of myocardial injury following cardioversion for atrial fibrillation

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Rowlens M Melduni – no conflicts
Allan S Jaffe – Dr. Jaffe presently or in the past has consulted for most of the major diagnostic companies.
ABSTRACT

Background: Direct Current (DC) cardioversion is used to terminate cardiac arrhythmias. Current guidelines list cardioversion as a cause of myocardial injury.

Objective: This study determined whether external DC cardioversion results in myocardial injury measured by serial changes in high sensitivity cardiac troponin T and I (hs-cTnT and hs-cTnI).

Methods: This was a prospective study of patients undergoing elective external DC cardioversion for atrial fibrillation. Hs-cTnT and hs-cTnI were measured pre-cardioversion and at least 6 hours post-cardioversion. Myocardial injury was present when there were significant changes in both hs-cTnT and hs-cTnI.

Results: Ninety-eight subjects were analyzed. Median (25th – 75th interquartile range) cumulative energy delivered was 121.9 J (102.2 – 302.7 J). Multiple cases 23 (23.5%) required 300J or more. Maximum cumulative energy delivered was 2455.1 J. There were small significant changes in both hs-cTnT [median pre-cardioversion 12 ng/L (7 – 19 ng/L), post-cardioversion 13 ng/L (8 – 21 ng/L), p <0.001)] and hs-cTnI [median pre-cardioversion 5 ng/L (3 – 10 ng/L), post-cardioversion 7 ng/L (3.6 – 11 ng/L), p <0.001)]. Results were similar in patients with high energy shocks and did not vary based on pre-cardioversion values. Only 2 (2%) cases met criteria for myocardial injury.

Conclusion: DC cardioversion resulted in a small but statistically significant changes in hs-cTnT and hs-cTnI in 2% of patients studied irrespective of shock energy. Patients with marked troponin elevations after elective cardioversion should be assessed for other causes of myocardial injury. It should not be assumed the myocardial injury was from the cardioversion.
KEYWORDS
Atrial fibrillation, cardioversion, troponin, high sensitivity cardiac troponin, myocardial injury

INTRODUCTION

Direct Current (DC) cardioversion with the delivery of electric shock to the heart through electrodes placed on the chest is often used to restore normal cardiac rhythm.

It has been reported that DC cardioversion results in myocardial injury\textsuperscript{1,2,3,4,5,6}. This concept was initially driven by animal studies\textsuperscript{7,8,9}. For that reason, the 4th Universal Definition of Myocardial Infarction\textsuperscript{1} lists cardioversion as a cause of myocardial injury. Present guidelines also suggest using lower energies and escalating the energy in a stepwise manner for defibrillation presumably in part to reduce the extent of myocardial injury\textsuperscript{10}. However, the observations that led to these recommendations were based on studies with monophasic defibrillators, older conductive gels and pads, and conventional (older), less sensitive troponin assays\textsuperscript{3,4,5,6}. There are mixed data in regard to frequency of myocardial injury with the newer biphasic defibrillators\textsuperscript{4,6,11}.

The aim of this study was to determine more definitively whether external DC cardioversion results in myocardial injury as measured by serial changes in high sensitivity cardiac troponin T and I (hs-cTnT and hs-cTnI).
METHODS

We prospectively recruited patients at the Mayo Clinic in Rochester, Minnesota undergoing elective DC cardioversion for atrial fibrillation / atrial flutter (AF) from July 2019 to July 2020. Written informed consent was obtained. Patients under the age of 18 years, pregnant, or who were receiving renal replacement therapy were not eligible. Patients with myocardial infarction, coronary artery bypass grafting or any invasive cardiac procedure in the previous six weeks also were excluded. Patients with transesophageal echocardiogram-guided cardioversions were included.

Cardioversion procedure

A transesophageal echocardiogram was performed if clinically indicated to exclude left atrial appendage thrombus. IV midazolam and fentanyl were given for the transesophageal portion of the procedure. For the DC cardioversion portion of the procedure, patients were sedated with IV propofol, dose adjusted according to physician preference.

Cardioversion was performed using the Zoll R Series Defibrillator (Zoll Medical Corporation, Burlington, MA), which delivers biphasic rectilinear shocks. Medi-Trace Cadence Adult defibrillation electrodes (Product code: 22770R) (Cardinal Health, Dublin, OH) were used for all cardioversions. Data regarding delivered energy, delivered current and impedance was obtained from the defibrillator. These parameters are provided routinely by most defibrillators in the United States. Cardioversions were performed using a standardized protocol (supplementary material).
Laboratory analyses

High sensitivity cardiac troponin (Hs-cTn) levels were measured pre-cardioversion and at least 6 hours post-cardioversion (supplementary material). Hs-cTnT was measured using the Troponin T Gen 5 STAT assay (Roche Diagnostics, Indianapolis, IN) on the Cobas e601 analyzer. Hs-cTnI was measured using the ARCHITECT STAT High Sensitivity Troponin-I assay (Abbott Laboratories, Abbott Park, IL). The 99th percentile for the hs-cTnT assay is 15 ng/L in males and 10 ng/L in females\(^\text{12}\). The lowest reportable value / limit of quantitation (LOQ - the lowest value with a CV ≤ 20%) for hs-cTnT is 6 ng/L\(^\text{13}\). For the hs-cTnI assay, the 99th percentiles are 34 ng/L for males and 16 ng/L for females\(^\text{14}\). The LOQ is 3.5 ng/L\(^\text{14}\). We also report analyses using the lower 99th percentile values for the hs-cTnI assay from the Universal Sample Bank (USB) study of 20 ng/L for males and 13 ng/L for females\(^\text{15}\). All hs-cTn are reported as whole values, except hs-cTnI from 3.5 to 4.0 ng/L where the values are reported with 1 decimal place.

Statistical analyses

Using an alpha of 0.01, power of 90%, effect size of 1 and the change in standard deviation from a previous study\(^\text{11}\) a sample size of 88 was required for this study. To accommodate a 10% “dropout” rate, we recruited 98 patients with permission of the IRB. Statistical analysis was done using IBM SPSS Statistics for Windows, Version 28. Descriptive statistics are presented as mean and standard deviation for normally distributed continuous variables, median and range or interquartile (IQ) range for non-Gaussian distributed continuous variables, or numbers and percentages for categorical variables. The comparison between groups were done using non-parametric paired testing – Wilcoxon rank sign test. The change in values (post-procedure value
minus pre-procedure value) of hs-cTnT and hs-cTnI, which we call “delta change” throughout the manuscript was analyzed using non-parametric paired testing – Wilcoxon rank sign test.

Significant changes in hs-cTn were defined as a > 50% change which is the Reference Change Value (RCV)\textsuperscript{16,17,18} when the baseline (pre-DCCV) value was ≤ 99th percentile sex specific URL. A >20% change was used when the baseline value was > 99th percentile sex specific URL to preserve the balance between sensitivity and specificity reported for hs-cTn assays\textsuperscript{19}.

When values were < LOQ, we analyzed the data using a value just below that (5.5 ng/L for hs-cTnT and 3 ng/L for hs-cTnI). As a sensitivity analysis, we also analyzed the data where the value < LOQ was assigned a value near zero (0.1 ng/L designated as “LOQ low” as opposed to “LOQ high”). These analyses are presented separately (supplementary material). To increase specificity, we only considered myocardial injury to be present if there were significant changes in both hs-cTnT and hs-cTnI.

Ethics approval was obtained from the Mayo Clinic IRB and the study was registered in ClinicalTrials.gov (number, NCT04151966). This study was conducted in accordance with the Declaration of Helsinki guidelines.

**RESULTS**

**Demographics of Study Sample (Table 1)**

Ninety-nine subjects were recruited. One was excluded because of hemolysis (H index > 100) in the pre-cardioversion sample which alters hs-cTn values\textsuperscript{13,14}. No other interfering substances
were noted. The median time to the post-cardioversion blood sample collection was 9 hours (interquartile range: 7 – 11 hours). Echocardiographic data are presented in Table S1.

**Cardioversion Details (Table 2)**

In total, 92 (93.9%) patients had successful restoration of sinus rhythm; 57 (58.2%) after only 1 shock. Twenty-six (26.5%) subjects received 3 or more shocks. A total of 23 (23.5%) received cumulative energies of more than 300 J. Four (4.1%) subjects received dual simultaneous defibrillator shocks. Median impedance was 103 ohms for first shock, 103 ohms for second shock, 109 ohms for 3rd shock and 108 ohms for 4th shock. There were no statistically significant changes in impedance between consecutive shocks (Table S2).

**Hs-cTn Measurements (Table 3)**

Pre-DCCV hs-cTnT ranged from below 6 ng/L to 120 ng/L. Post-DCCV hs-cTnT ranged from below 6 ng/L to 128 ng/L. The delta hs-cTnT change ranged from -21 ng/L to +64 ng/L.

Pre-DCCV hs-cTnI ranged from below 3.5 ng/L to 211 ng/L. Post-DCCV values ranged from below 3.5 ng/L to 216 ng/L. The delta hs-cTnI change ranged from -16 ng/L to +103 ng/L.

Table 3 shows the distribution of the pre-and-post-cardioversion differences for hs-cTnT and hs-cTnI. There was a statistically significant difference between the pre-and-post DCCV values for both. This finding was also present using the ‘LOQ low’ value (Table S3). The median change however was only 1 ng/L for hs-cTnT with 25% of the study population having a change of 0
ng/L and 75% of the population having a change of up to 2 ng/L. Similarly, the median change for hs-cTnI was only 0.7 ng/L, with 25% of the study population having a change of 0 ng/L and 75% of the population having a change of up to 2 ng/L.

Figures S1 and S2 shows the distribution of the pre-DCCV versus the post-DCCV hs-cTnT and hs-cTnI values. Most values are similar.

Figures 1 and 2 shows the distribution of the change in hs-cTn values (post-cardioversion minus pre-cardioversion) plotted against the cumulative energy delivered. The differences are not related to the cumulative energy delivered.

**Cases with Significant Changes in hs-cTn Values**

Table 4 shows the patients with significant changes for hs-cTnT, hs-cTnI using standard cutoffs. Data using the USB Abbott cut off values are in Table S4. There were 2 patients (one male and one female) who fulfilled our primary end point (both hs-cTnT and hs-cTnI changes above RCV occurred).

When using the lower 99th percentile USB cutoffs for hs-cTnI, there is an increase in cases (3 males and 1 female) with significant changes where pre-DCCV values are > URL and a decrease in cases (5 males and 4 females) with significant changes where pre-DCCV values were ≤ URL (Table S4). This resulted in an additional case (1 female) who had fulfilled our primary end point.

Using ‘LOQ low’ values accentuated the number of cases with significant changes when the pre-DCCV value was ≤ URL (9 males and 8 females for hs-cTnT; 8 males and 8 females for hs-
cTnI) (Table S4). This resulted in an additional 5 cases (all females) who had fulfilled our primary end point. For hs-cTnI using USB cutoffs, there was also accentuation of the number of cases (6 males and 8 females). This resulted in an additional 4 cases (all females) who had fulfilled our primary endpoint (Table S4).

The number of cases where there is a significant change in both hs-cTnT and hs-cTnI were small. Using the USB Abbott cutoff values did not appear to have a large influence on the number of cases (1 male and 2 females) (Table S4).

Two cases met our primary endpoint when standard LOQ cutoffs (‘LOQ high’) were used (Table 5). Table S5 shows the cases that met our primary endpoint when ‘LOQ low’ cutoffs were used. There were no significant differences in age, sex, BMI, LVEF, LVMI or CHA2DS2-VaSc score in the patients with significant changes compared to those without.

Despite larger amounts of energies delivered with dual defibrillation, only 1 case (case 109) had both a substantial hs-cTnT and hs-cTnI rise, and it was not the case that received the highest amount of energy (Table 6).

**DISCUSSION**

Our data elucidate several important findings. Firstly, there was a statistically significant but small change in median hs-cTnT and hs-cTnI values (1 ng/L and 2 ng/L respectively) after DC cardioversion for AF. Because of the small magnitude of the changes, the biological importance of them is unlikely to be profound. Secondly, of the 98 cases, only 2 (2%) met our criteria for significant myocardial injury; in one a modest change occurred (case 47) and in the other, a much larger change (case 109). Our data do not suggest that the increases were related to the
amount of energy delivered or number of shocks. There also were no discernible differences in clinical characteristics in those patients with significant increases that might explain their differences from other patients. We also noted that patients with a pre-cardioversion value that was < 99th percentile (no pre-existing myocardial injury) tended to have more frequent significant changes compared to those patients with a pre-cardioversion value > 99th percentile (pre-existing myocardial injury) with hs-cTnI, but not with hs-cTnT (see supplementary material for further details).

As mentioned previously, the initial perception that external DC cardioversion caused myocardial injury came from animal studies\textsuperscript{7,8,9}. These studies measured hemodynamic parameters post-cardioversion rather than measuring biomarkers of myocardial injury. The reduction in hemodynamic function may be simply a marker of post-resuscitation myocardial dysfunction, which is a temporary phenomenon\textsuperscript{20}, rather than myocardial injury. It is also unclear whether the energy levels used in these animal studies correspond to the energy levels used in humans (not just in terms of weight and body composition but also of anatomy).

The current study challenges the accepted norm that elective DC cardioversion results in myocardial injury. Thus, patients in this setting who have marked troponin increases in hs-cTnT or hs-cTnI should be assessed for other causes of myocardial injury. It may be that some of these differences are related at least in part to the modern-day use of biphasic defibrillators. Most studies have suggested they result in the lower risk of post-shock myocardial injury because the energy requirements are lower\textsuperscript{4,21}. Monophasic defibrillators deliver higher peak voltages and higher peak current\textsuperscript{22}. These data are consistent with earlier studies with biphasic devices albeit with less sensitive cardiac troponin assays that suggested exactly this. Biphasic waveforms have greater shock efficacy likely secondary to the ability of the second phase to achieve
cardioversion more easily by creating a homogenous distribution of postshock transmembrane voltage and the removal of excess charge left on the myocardial cell membranes at the end of the first phase (charge balancing). These effects may be contributory to the lack of injury with biphasic shocks. Importantly, there was no evidence that energy levels were determinative.

Our experience mirrors the findings described by Neal et al and have potential clinical implications. Even though we had a 93.9% success rate in restoring sinus rhythm, 41.8% of patients required more than 1 shock. Our practice is to use 120 J in AF for the first biphasic shock and increase if needed to 150 J and 200 J. Whether the second shock using higher energy is successful due to the increased energy is unclear. However, if that were to be the case one could potentially start cardioversions with higher energy levels since they do not appear to increase the extent of myocardial injury. This might reduce the energy required and the time spent under sedation, allowing procedures to be done more rapidly. There are multiple older reports that using higher initial energy levels result in higher conversion rates. Similarly, the recent CHESS trial showed higher success rates when a maximum-fixed (360-360-360J) biphasic shock protocol was used compared to a low-escalating (125-150-200J) protocol (88% vs. 66% respectively). While it should be noted that both the studies used a defibrillator that delivered biphasic truncated exponential (BTE) waveform shocks as opposed to biphasic rectilinear (BR) waveform which we used in our study, the first shock success rate was comparable to our first shock success rate of 58.2%. The CHESS group reported their cardiac troponin data in the supplement, and it appeared as if there also was not a signal indicative of myocardial injury. However, each waveform has its own unique operating characteristics. Assuming the results with one waveform will mirror those of another approach may not be reality. In addition, there may be differences in both the pads and gels used and their relative
impedance. These interesting issues require additional studies. There are no data that would allow our observations to be extrapolated to the circumstances where defibrillation is used which is, by definition, an ischemically mediated milieu, but perhaps future studies might wish to develop protocols that would allow such questions to be answered.

It is thought that sequential shocks (monophasic or biphasic) results in reduced chest wall impedance (due to tissue electrical injury resulting in edema and hyperemia) and thus subsequent increased current flow to the myocardium. We did not observe that phenomenon (Table S2). Our data questions the validity of those prior data with the device we used. It could be related to the different biphasic waveforms used (BR vs. BTE) although a previously published randomized trial comparing them showed a progressive reduction in transthoracic impedance in both groups.

This study has several limitations. It is a single center study and thus subject to the limitations of a single center study including the population undergoing cardioversion and the specific techniques deployed in terms not only of the procedure but of the support such as anesthesia. Secondly, because of logistical difficulties in obtaining the 2nd blood sample, we obtained samples 6-24 hours after the procedure (median time = 9 hours). Perhaps we missed some additional late rises. It is possible that some patients with underlying coronary artery disease may not have as marked hs-cTn rises in samples obtained early post-DCCV because of the “slower wash out” of hs-cTn than those with normal coronary arteries. Thirdly, the echocardiographic data were obtained from TTE that were done over a wide time period, from 6 months before to 6 months after the cardioversion, and not all echo parameters were measured. Thus, we did not use the echocardiography data as a primary endpoint of our study. Finally, while increases in hs-cTn are associated with increased mortality from procedures such as non-
cardiac surgery as in the VISION study, this may not necessarily translate to cardioversions where the changes were extremely small and though present may not be of biological importance. We doubt that we will be able to see differences during follow up.

**CONCLUSION**

In conclusion, elective external DC cardioversion in stable patients with AF results in small, but statistically significant changes in median hs-cTnT and hs-cTnI values (1 ng/L and 2 ng/L respectively). The changes did not appear related to shock energy and did not vary based on pre-cardioversion baseline values. Patients who have marked troponin elevations after elective cardioversion should be assessed for other causes of myocardial injury. They should not be assumed to have myocardial injury from the cardioversion alone.

**ACKNOWLEDGEMENTS**

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REFERENCES


Table 1. Baseline Characteristics*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n=98</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>69 (63 – 77)</td>
</tr>
<tr>
<td>Men (n=69)</td>
<td>69 (70.4%)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>32 (27.4 – 37.7)</td>
</tr>
<tr>
<td>History of CAD</td>
<td>15 (15.3%)</td>
</tr>
<tr>
<td>CHA₂DS₂-VaSc Score</td>
<td>3 (2 – 4)</td>
</tr>
<tr>
<td>Rhythm</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>61 (62.2%)</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>37 (37.8%)</td>
</tr>
<tr>
<td>TEE-guided</td>
<td>34 (34.7%)</td>
</tr>
<tr>
<td>Successful</td>
<td>92 (93.9%)</td>
</tr>
<tr>
<td>Propofol dose (mg)</td>
<td>100 (68 – 130)</td>
</tr>
<tr>
<td>Midazolam dose (n=34)</td>
<td>4 (3 – 5)</td>
</tr>
<tr>
<td>Fentanyl dose (n=34)</td>
<td>75 (50 – 100)</td>
</tr>
</tbody>
</table>

*Values are presented as numbers with percentages for categorical data and median (upper and lower quartiles) for continuous data. Abbreviations: BMI, body mass index; CAD, coronary artery disease; TEE, transesophageal echocardiogram.

Table 2. Cardioversion procedure details including delivered energy, current and impedance values presented as median (upper and lower quartiles) as well as minimum to maximum values

<table>
<thead>
<tr>
<th>No. of Shocks</th>
<th>Cumulative Energy Delivered (J)</th>
<th>Highest Energy Delivered per Shock (J)</th>
<th>Cumulative Current Delivered (A)</th>
<th>Highest Current Delivered per Shock (A)</th>
<th>Highest Impedance (Ohms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median (25th – 75th quartiles)</td>
<td>1 (1 – 3)</td>
<td>121.9 (102.2 – 302.7)</td>
<td>121.1 (62.3 – 154.8)</td>
<td>14.3 (10.3 – 29.7)</td>
<td>12.9 (9.9 – 15)</td>
</tr>
<tr>
<td>Minimum – Maximum</td>
<td>1 – 8</td>
<td>59.1 – 2455.1</td>
<td>59.1 – 509.2</td>
<td>6.7 – 202.2</td>
<td>6.7 – 42.3</td>
</tr>
</tbody>
</table>

Table 3. High sensitivity cardiac troponin values before and after cardioversion. P-values calculated using the Wilcoxon signed rank test*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Pre-Cardioversion</th>
<th>Post-Cardioversion</th>
<th>Median Difference (post-pre)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>hs-cTnT</td>
<td>12 (7 – 19)</td>
<td>13 (8 – 21)</td>
<td>1 (0 – 2)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>hs-cTnl</td>
<td>5 (3 – 10)</td>
<td>7 (3.6 – 11)</td>
<td>0.7 (0 – 2)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

*Abbreviations: hs-cTnT, high-sensitivity cardiac troponin T; hs-cTnl, high-sensitivity cardiac troponin I.
Figure 1. Scatter plot showing the change in hs-cTnT values (post-cardioversion – pre-cardioversion) plotted against the cumulative energy delivered.

Figure 2. Scatter plot showing the change in hs-cTnI values (post-cardioversion – pre-cardioversion) plotted against the cumulative energy delivered.
Table 4. Distribution of changes for hs-cTnT, hs-cTnI using standard cutoffs and hs-cTnI using Universal Sample Bank cutoffs.

<table>
<thead>
<tr>
<th>hs-cTnT</th>
<th>Males (n=69)</th>
<th>Females (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-DCCV &gt; URL that increased by &gt; 20% post-DCCV</td>
<td>5 (7.2%)</td>
<td>2 (6.9%)</td>
</tr>
<tr>
<td>Pre-DCCV ≤ URL that increased by &gt; 50% post-DCCV</td>
<td>1 (1.4%)</td>
<td>4 (13.8%)</td>
</tr>
<tr>
<td>Patients that had changes above RCV (20% OR 50%)</td>
<td>6 (8.7%)</td>
<td>6 (20.7%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>hs-cTnI (standard cutoff: 16 ng/L for females and 34 ng/L for males)</th>
<th>Males (n=69)</th>
<th>Females (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-DCCV &gt; URL that increased by &gt; 20% post-DCCV</td>
<td>1 (1.4%)</td>
<td>0</td>
</tr>
<tr>
<td>Pre-DCCV ≤ URL that increased by &gt; 50% post-DCCV</td>
<td>6 (8.7%)</td>
<td>4 (13.8%)</td>
</tr>
<tr>
<td>Patients that had changed above RCV (20% OR 50%)</td>
<td>7 (10.1%)</td>
<td>4 (13.8%)</td>
</tr>
<tr>
<td>Patients that had change above RCV for both hs-cTnT AND hs-cTnI</td>
<td>1 (1.4%)</td>
<td>1 (3.4%)</td>
</tr>
</tbody>
</table>

*Abbreviations: hs-cTnT, high-sensitivity cardiac troponin T; hs-cTnI, high-sensitivity cardiac troponin I; LOQ, limit of quantification; RCV, reference change value; DCCV, DC cardioversion.

Table 5. Cases where subjects had a significant rise in both hs-cTnT and hs-cTnI using standard LOQ cutoffs*

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>Sex</th>
<th>LVEF (%)</th>
<th>Number of Shocks</th>
<th>Cumulative Energy Delivered (J)</th>
<th>Maximum Energy Delivered per Shock (J)</th>
<th>Pre-DCCV hs-cTnT</th>
<th>Post-DCCV hs-cTnT</th>
<th>Pre-DCCV hs-cTnI</th>
<th>Post-DCCV hs-cTnI</th>
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<tbody>
<tr>
<td>47</td>
<td>F</td>
<td>31</td>
<td>1</td>
<td>123</td>
<td>123</td>
<td>10</td>
<td>16</td>
<td>&lt; 3.5</td>
<td>6</td>
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<tr>
<td>109</td>
<td>M</td>
<td>65</td>
<td>6</td>
<td>1460.2</td>
<td>509.2</td>
<td>30</td>
<td>59</td>
<td>33</td>
<td>136</td>
</tr>
</tbody>
</table>

*Abbreviations: hs-cTnT, high-sensitivity cardiac troponin T; hs-cTnI, high-sensitivity cardiac troponin I; DCCV, DC cardioversion.

Table 6. Cases where subjects were delivered dual defibrillator simultaneous shocks*

<table>
<thead>
<tr>
<th>Patient ID</th>
<th>Sex</th>
<th>LVEF (%)</th>
<th>Number of dual simultaneous shocks</th>
<th>Cumulative Energy Delivered (J)</th>
<th>Maximum Energy Delivered per Shock (J)</th>
<th>Pre-DCCV V hs-cTnT</th>
<th>Post-DCCV V hs-cTnT</th>
<th>Pre-DCCV V hs-cTnI</th>
<th>Post-DCCV V hs-cTnI</th>
</tr>
</thead>
<tbody>
<tr>
<td>98</td>
<td>M</td>
<td>62</td>
<td>8</td>
<td>2</td>
<td>2075</td>
<td>507.2</td>
<td>10</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>101</td>
<td>F</td>
<td>58</td>
<td>8</td>
<td>3</td>
<td>2455.1</td>
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*Abbreviations: hs-cTnT, high-sensitivity cardiac troponin T; hs-cTnI, high-sensitivity cardiac troponin I; DCCV, DC cardioversion.
Elective external cardioversion in stable patients with atrial fibrillation results in a small, statistically significant but unlikely clinically significant change in median high sensitivity cardiac troponin T and troponin I values (1 ng/L and 2 ng/L respectively). The changes do not appear related to shock energy and do not vary based on pre-cardioversion baseline values. Patients who have marked troponin elevations after elective cardioversion should be assessed for other causes of myocardial injury. They should not be assumed to have myocardial injury from the cardioversion alone.