Marked Reduction in Atrial Defibrillation Thresholds With Repeated Internal Cardioversion

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OBJECTIVES
This study was performed to assess the atrial defibrillation threshold in patients with recurrent atrial fibrillation (AF) using repeated internal cardioversion.

BACKGROUND
Previous studies in patients with chronic AF undergoing internal cardioversion have shown this method to be effective and safe. However, current energy requirements might preclude patients with longer-lasting AF from being eligible for an implantable atrial defibrillator.

METHODS
Internal shocks were delivered via defibrillation electrodes placed in the right atrium (cathode) and the coronary sinus (anode) or the right atrium (cathode) and the left pulmonary artery. After cardioversion, patients were orally treated with sotalol (mean 189 ± 63 mg/day). Eighty consecutive patients with chronic AF (mean duration 291 ± 237 days) underwent internal cardioversion, and sinus rhythm was restored in 74 patients. Eighteen patients underwent repeated internal cardioversion using the same electrode position and shock configuration after recurrence of AF (mean duration 34 ± 25 days).

RESULTS
In these 18 patients, the overall mean defibrillation threshold was 6.67 ± 3.09 J for the first cardioversion and 3.83 ± 2.62 J for the second (p = 0.003). Mean lead impedance was 55.6 ± 5.1 V and 57.1 ± 3.7 V, respectively (not significant). For sedation, 6.7 ± 2.9 mg and 3.9 ± 2.2 mg midazolam were administered intravenously (p = 0.003), and the pain score (0 = not felt, 10 = intolerable) was 5.1 ± 1.9 and 2.7 ± 1.8 (p = 0.001). Uni- and multivariate analyses revealed only the duration of AF before cardioversion to be of relevance, lasting 175 ± 113 days before the first and 34 ± 25 days before the second cardioversion in these 18 patients (p = 0.002).

CONCLUSIONS
If the duration of AF is reduced, a significant reduction in defibrillation energy requirements for internal cardioversion ensues. This might extend the group of patients eligible for an implantable atrial defibrillator despite relatively high initial defibrillation thresholds. (J Am Coll Cardiol 1999;34:1569–76) © 1999 by the American College of Cardiology

Atrial fibrillation (AF) has been shown to deteriorate hemodynamics (1–3), to lead to systemic embolism including stroke (1,2) and to a twofold risk of cardiac mortality (1,4). Associated structural changes of the atria foster the arrhythmia (2). Therefore, prompt restoration of sinus rhythm is desirable in principle. Several therapeutic options have been devised for restoration and maintenance of sinus rhythm, including pharmacological, external and internal conversion, surgical treatment, atrial pacemaker implantation, high-frequency ablation of foci and, most recently, an implantable atrial defibrillator (1,2,5–13). Despite recent technical advances, such as optimized shock morphology and electrode position in internal cardioversion (11,14–18), some concerns still exist with this new mode of treatment.

Relatively high energy requirements (10,16,19–21) and consequent pain perception (12,17,22–24) currently limit the use of an automatic atrial defibrillator to patients with low initial atrial defibrillation thresholds (1,2,5,12,20,25).

Thus, it was the aim of the present study to assess defibrillation thresholds for repeated internal cardioversion with respect to patients’ parameters. By proving the hypothesis that shortened AF duration lowers defibrillation thresholds for internal cardioversion, more patients might be considered for an implantable atrial defibrillator (2,16,20,24–26).

METHODS
Patients. In the present study, patients who underwent repeated internal cardioversion were included. Of 80 patients to undergo this procedure for chronic AF, sinus rhythm could be successfully restored in 74, as previously reported (27).

All 74 patients were on sotalol (189 ± 63 mg/day); 35
had a relapse of AF after a mean follow-up of 14.2 ± 6.9
(range 12.5 to 36) months. Eighteen of these patients
requested a second internal cardioversion, and they formed
the basis of this study.

The 18 patients (58 ± 6 years of age; 4 women, 14 men,
body weight 75 ± 11 kg, body height 170 ± 12 cm; left
atrial size 58 ± 4 mm) had suffered chronic AF for 175 ±
113 days (range 30 to 500 days) before their first cardiover-
sion. The underlying heart disease was diagnosed as coro-
nary artery disease in 5 patients, hypertension in 4, valvular
disease in 4, lone or idiopathic AF in 3 and dilated
cardiomyopathy in 2 patients. Pharmacological conversion
attempts had been performed in 14 patients (72%) before
initial internal cardioversion without success in eight pa-
tients (57%); antiarrhythmic medication did not prevent
recurrent AF in the other six patients.

According to the study protocol (27), approved by the
Ethics Committee of the Technical University of Munich,
patients 21 to 75 years old were enrolled if the following
selection criteria were fulfilled:

1. chronic atrial fibrillation of at least 14 days duration,
documented by serial electrocardiogram (ECG); and
2. effective anticoagulation with warfarin for at least 14 days
(INR [international normalized ratio] 2.5 to 4.2).

Patients were excluded from the study because of:

1. evidence of digitalis toxicity;
2. abnormal electrolyte concentrations;
3. hyperthyroidism;
4. an acute myocardial infarction within the past six weeks;
or
5. a prior history of embolism.

Clinical examination was performed and medical history
was taken. In all patients, routine 12-lead ECG, 24-h
Holter monitoring, chest roentgenogram, routine laboratory
including thyroid parameters and M-mode and Doppler
echocardiography were performed. A transthoracic echo-
cardiography was obtained if clinically indicated. Benefits
and risks of the study were discussed with the patients and
informed consent was obtained.

Protocol for internal cardioversion. The protocol was
carried out as in previous studies (27–29). Two temporary
6 F catheters (Elecath; Electro-Catheter Corp., Rahway,
New Jersey), with an active surface area of 2.62 cm²
consisting of 10 parallel stainless steel rings, were inserted
into the femoral vein and positioned in the right atrium and
either the distal coronary sinus or the left pulmonary artery.

Lead position was randomized: seven patients underwent
cardioversion and had the catheters in the right atrium and
coronary sinus, and 11 patients had the catheters in the right
atrium and pulmonary artery.

Internal cardioversion was performed either in the inte-
censive care unit or in the electrophysiology laboratory. The
patients were sedated orally with 2.5 to 5 mg diazepam.
Immediately before cardioversion, 1 mg midazolam was
administered intravenously; in two anxious patients, 5 mg
midazolam were given. No antiarrhythmic drug was admin-
istered within 48 h before or during the procedure.

The defibrillation threshold was defined as the lowest
shock energy that converted AF into sinus rhythm. Biphas-
ic, two-capacitor shocks of 3 ms/3 ms with phases sepa-
rated by a delay of 0.2 ms were used. The shocks were
delivered by an external defibrillator (Ventritex HVS-02;
Ventritex, Sunnyvale, California) and synchronized to the
R-wave. In order to ensure R-wave synchronization, the
ECG was amplified and filtered by a custom-built, variable-
gain ECG amplifier. Special circuitry prevented shock
delivery when the cycle length was shorter than 500 ms (30).
Oxygen saturation was monitored using a pulse oxymeter,
and the blood pressure was measured every 10 min. Begin-
ning with a test shock of 60-V intensity, the voltage was
increased in 40-V steps until cardioversion was achieved.
Criteria for discontinuation were patient discomfort, com-
lications such as induction of proarrhythmia or major
bleedings, or achieving the maximum voltage of 580 V.
Between unsuccessful defibrillation attempts, at least 1 min
before the next shock was applied. During the study, the
12-lead ECG and intraatrial signals were recorded and
stored (EP Lab, version 6.0; Quinton Electrophysiology,
Inc., Ontario, Canada).

The delivered voltage, current, and morphology for each
shock were recorded using a Macintosh computer and
customized LabVIEW software (National Instruments,
Austin, Texas). The delivered voltage and current wave-
forms were integrated to obtain energy, and impedance was
calculated from the peak voltage and current. Patients were
asked to quantify their level of perceived pain by means of a
scale ranging from 0 (not felt) to 10 (intolerable) after each
shock applied.

Follow-up evaluation. Upon experiencing symptoms sug-
gestive of recurrent AF, patients were asked to return for a
12-lead ECG. Otherwise, follow-ups were performed at 1,
3, 6, 9 and 12 months after cardioversion. All patients were
treated with at least 80 mg bid sotalol (mean daily dose for
these 18 patients 175 ± 55 mg, range 160 to 400 mg) after
effective cardioversion to sinus rhythm. Angiotensin
converting enzyme inhibitors, diuretics and digitalis were con-
tinued based on the clinical status of the patients. Antico-
agulation was continued after cardioversion and was stopped
only in patients who remained in sinus rhythm for 4 weeks.

Protocol for repeated internal cardioversion. Before the
second internal cardioversion, no patient had an attempt at

Abbreviations and Acronyms

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>AF</td>
<td>atrial fibrillation</td>
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<td>ECG</td>
<td>electrocardiogram</td>
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uni- and multivariate analysis focused on the influence of the defibrillation attempt (first vs. second) was performed. A coronary sinus vs. right atrium-left pulmonary artery) and Statistical analyses.

A two-way analysis of variance of the 12-lead ECG at four weeks. Patients who remained in sinus rhythm confirmed by status of the patients. Anticoagulation was stopped in diuretics and digitalis were continued based on the clinical cardioversion. Angiotensin converting enzyme inhibitors, perceived pain by means of a scale ranging from 0 (intravenously. Patients were asked to quantify their level of immediately before cardioversion, 1 mg midazolam was administered intravenously. Patients were sedated orally with 2.5 to 5 mg diazepam. Immediately after each shock applied. any other type of conversion. Upon renewed informed consent, the 18 patients with a mean AF duration of 34 ± 25 days (range 7 to 66 days) underwent the same protocol as for their first cardioversion; the same electrode configuration was used (Fig. 1). Sotalol was discontinued 48 h before the procedure. No other antiarrhythmic drugs were given.

Follow-up evaluation after repeated internal cardioversion. On an outpatient basis, a 12-lead ECG was obtained at 1, 3, 6, 9 and 12 months after cardioversion, or if the patient had experienced symptoms consistent with recurrent AF. All patients were treated with at least 80 mg bid sotalol (mean daily dose 194 ± 63 mg, range 160 to 320 mg) after cardioversion. Angiotensin converting enzyme inhibitors, diuretics and digitalis were continued based on the clinical status of the patients. Anticoagulation was stopped in patients who remained in sinus rhythm confirmed by 12-lead ECG at four weeks.

Statistical analyses. A two-way analysis of variance of the main effects of both the defibrillation vector (right atrium-coronary sinus vs. right atrium-left pulmonary artery) and the defibrillation attempt (first vs. second) was performed. A uni- and multivariate analysis focused on the influence of patients’ parameters (age, left atrial diameter, body mass index, duration of AF and underlying heart disease) on the defibrillation threshold for internal cardioversion. Continuous variables are expressed as mean ± standard deviation. For continuous variables, the Mann Whitney U test for unpaired groups was used. A predictive value of < 0.05 was considered to indicate statistical significance.

RESULTS

Short-term efficacy of cardioversion. Of the 80 patients initially to be treated, internal cardioversion was successful in 74 patients, with a mean defibrillation threshold of 5.81 ± 3.20 J. Of the 35 patients who relapsed into AF, 18 patients underwent repeated internal cardioversion. The initial cardioversion in these 18 patients was conducted with a mean defibrillation threshold of 6.67 ± 3.09 J, insignificantly higher compared with the 5.81 ± 3.20 J for the 74 patients initially converted (NS) and to the threshold of 5.22 ± 3.32 J for the 39 patients who did not relapse (NS). At the second cardioversion, energy requirements were 3.83 ± 2.62 J and were approximately 40% lower (p = 0.003), despite comparable lead impedance at first and second intervention (55.6 ± 5.1 Ω vs. 57.1 ± 3.7 Ω, NS). In patients using catheters positioned in the right atrium and the coronary sinus (n = 7), mean energy for cardioversion was 4.26 ± 1.71 J for the first and 2.83 ± 0.86 J for the second (p = 0.069). With the catheters in the right atrium and left pulmonary artery (n = 11), the defibrillation threshold was 8.21 ± 2.79 and 4.47 ± 3.17 J, respectively (p = 0.011; Table 1). In the first procedure, the defibrillation threshold for the right atrium-coronary sinus vector was lower than for the right atrium-pulmonary artery vector (for the first cardioversion, p = 0.018; for the second cardioversion, NS).

Patient characteristics. Of the 18 patients undergoing repeated internal cardioversion of recurrent AF, body mass index remained stable throughout the study (26 ± 0.8 kg/m²). The mean echocardiographically determined longitudinal left atrial diameter was 58 ± 4 mm (range 53 to 60 mm) at the first and 57 ± 3 mm (range 51 to 60 mm) at the second cardioversion (NS). The duration of atrial fibrillation (175 ± 113 vs. 34 ± 25 days, p = 0.002) was considerably longer at the time of the first cardioversion than at the second (Table 1, Fig. 2).

Correlation of defibrillation thresholds and patient parameters. There was a significant correlation of energy requirements and duration of AF before the first cardioversion with the right atrium-coronary sinus vector (p = 0.033, r² = 0.629; Fig. 3) and with the right atrium-pulmonary artery vector (p = 0.050, r² = 0.210; Fig. 3). For the second cardioversion with overall significantly shorter durations, no such correlation was observed (p = 0.230, r² = 0.272 and p = 0.668, r² = 0.004, respectively). There was no correlation between energy requirements and either left atrial size or body mass index (NS). In terms of symptoms, the pain
scores expressed by patients correlated with defibrillation thresholds for first and repeated cardioversion for both vectors \( (p = 0.051, r^2 = 0.566 \text{ and } p = 0.813, r^2 = 0.012 \) for the right atrium-coronary sinus vector vs. \( p = 0.014, r^2 = 0.504 \text{ and } p = 0.002, r^2 = 0.677 \) for the right atrium-pulmonary artery vector; Fig. 4).

**Uni- and multivariate analysis of patient parameters and defibrillation threshold.** Only the duration of AF before the first and second cardioversion was significant \( (p = 0.0007) \). Age, body mass index, left atrial diameter and underlying heart disease were not statistically significant.

**Long-term clinical outcome.** On an intention-to-treat basis, 11 of 18 patients successfully treated with repeated internal cardioversion remained in sinus rhythm after a mean follow-up of 11.4 ± 4 months (range 11 to 18 months; Table 1). In three patients, AF recurred after 7, 14 and 30 days. After their first internal cardioversion, AF had recurred after 3, 1 and 30 days, respectively. In another three patients, AF recurred after 3, 14 and 30 days after the first cardioversion, but 60 days after the second one. One patient remained in sinus rhythm for 30 days after the first and for 45 days after the second cardioversion.

**Complications.** There were no complications observed in the 18 patients of this present study.

**DISCUSSION**

The present study demonstrates that compared with the first intervention, despite nearly identical impedance, less energy is required when internal cardioversion of AF is performed a second time. In addition, sinus rhythm persists for a longer period afterwards (Table 1). Furthermore, AF duration before cardioversion might be a critical parameter, which is in keeping with the majority of literature reports available on this issue \( (2,10,25,31) \). In this study, a significant correlation was seen between defibrillation thresholds and duration of chronic AF (range from 14 to 500 days) in patients undergoing internal cardioversion for the first time. In patients undergoing cardioversion a second time shortly after the onset of AF (7 to 66 days, mean 34 ± 25 days) and with no other measured patient parameter having changed, no such correlation is seen. This indicates that in chronic AF, partially reversible changes over time may lead to increases in energy requirements.

**Influence of AF duration on defibrillation threshold.** These findings do not point to the quality of the electrical field as the decisive aspect for cardioversion, as has been noted until recently \( (1,11,12,14,21,22,24,27,32) \) but rather to cellular and subcellular aspects brought about over time by the arrhythmia itself \( (2,5,33–36) \). Atrial atrophy, fibrosis and dilation are some of the consequences observed in chronic AF \( (2) \), which had been formerly viewed as both its cause and result \( (1,2,5,34–36) \). Apparently, both electrical changes, such as shortened fibrillatory cycle length and shortened effective refractory period, and organic lesions, as already mentioned, impact on the defibrillation threshold. Electrical phenomena, starting with a shortening of the atrial refractory period, become full-blown within a few days.
### Table 1. Acute and Long-Term Outcome of First and Second Internal Cardioversion (n = 18)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>1° IC (n = 18)</th>
<th>2° IC (n = 18)</th>
<th>1° vs. 2° IC p Value</th>
<th>1° IC RA-CS* (n = 7)</th>
<th>2° IC RA-CS* (n = 7)</th>
<th>1° vs. 2° IC p Value</th>
<th>1° IC RA-PA† (n = 11)</th>
<th>2° IC RA-PA† (n = 11)</th>
<th>1° vs. 2° IC RA-CS* vs. RA-PA† p Value</th>
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<tbody>
<tr>
<td><strong>Acute outcome</strong></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
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<tr>
<td>AF duration (days)</td>
<td>175 ± 113</td>
<td>34 ± 25</td>
<td>0.002</td>
<td>162 ± 79</td>
<td>42 ± 20</td>
<td>0.002</td>
<td>182 ± 130</td>
<td>29 ± 26</td>
<td>0.001</td>
</tr>
<tr>
<td>Success (n/n) (%)</td>
<td>18/18 (100%)</td>
<td>18/18 (100%)</td>
<td>NS</td>
<td>7/7 (100%)</td>
<td>7/7 (100%)</td>
<td>NS</td>
<td>11/11 (100%)</td>
<td>11/11 (100%)</td>
<td>NS</td>
</tr>
<tr>
<td>Defibrillation threshold (J)</td>
<td>6.67 ± 3.09</td>
<td>3.83 ± 2.62</td>
<td>0.003</td>
<td>4.26 ± 1.71</td>
<td>2.83 ± 0.86</td>
<td>0.069</td>
<td>8.21 ± 2.79</td>
<td>4.47 ± 3.17</td>
<td>0.011</td>
</tr>
<tr>
<td>Impedance (Ω)</td>
<td>55.6 ± 5.1</td>
<td>57.1 ± 3.7</td>
<td>NS</td>
<td>53.6 ± 6.2</td>
<td>54.3 ± 4.1</td>
<td>NS</td>
<td>57.6 ± 5.9</td>
<td>57.0 ± 4.7</td>
<td>NS</td>
</tr>
<tr>
<td>Time for catheter placement (min)</td>
<td>15.2 ± 9.0</td>
<td>12.0 ± 7.0</td>
<td>NS</td>
<td>20.5 ± 11.0</td>
<td>16.2 ± 8.0</td>
<td>NS</td>
<td>11.5 ± 9.0</td>
<td>9.0 ± 6.0</td>
<td>NS</td>
</tr>
<tr>
<td>Pain score (0–10)</td>
<td>5.1 ± 1.9</td>
<td>2.7 ± 1.8</td>
<td>0.001</td>
<td>4.0 ± 1.5</td>
<td>2.2 ± 0.9</td>
<td>0.044</td>
<td>5.9 ± 1.8</td>
<td>3.0 ± 2.1</td>
<td>0.009</td>
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<td>Sedation w/midazolam (mg)</td>
<td>6.7 ± 2.9</td>
<td>3.9 ± 2.2</td>
<td>0.003</td>
<td>5.7 ± 3.6</td>
<td>3.6 ± 1.5</td>
<td>NS</td>
<td>7.4 ± 2.2</td>
<td>4.0 ± 2.6</td>
<td>0.009</td>
</tr>
<tr>
<td>CK pre/6 h post-IC (U/liters)</td>
<td>50 ± 15/</td>
<td>45 ± 16/</td>
<td>NS</td>
<td>50 ± 15/</td>
<td>47 ± 18/</td>
<td>NS</td>
<td>50 ± 15/</td>
<td>43 ± 15/</td>
<td>NS</td>
</tr>
<tr>
<td>Ck-Mb pre/6 h post-IC (%)</td>
<td>3.1%/3.3%</td>
<td>3.1%/3.2%</td>
<td>NS</td>
<td>3.0%/3.2%</td>
<td>3.1%/3.2%</td>
<td>NS</td>
<td>3.1%/3.3%</td>
<td>3.2%/3.3%</td>
<td>NS</td>
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<td><strong>Long-term outcome</strong></td>
<td></td>
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<tr>
<td>Follow-up (months) (range)</td>
<td>16.0 ± 9.0</td>
<td>11.4 ± 4.0</td>
<td>NS</td>
<td>16.0 ± 9.0</td>
<td>11.6 ± 2.0</td>
<td>NS</td>
<td>16.0 ± 9.0</td>
<td>11.9 ± 6.0</td>
<td>NS</td>
</tr>
<tr>
<td>Sotalol (mg)</td>
<td>(11.5–38)</td>
<td>(11.0–18)</td>
<td>NS</td>
<td>(12.5–38)</td>
<td>(11.5–16)</td>
<td>NS</td>
<td>(11.5–36)</td>
<td>(11.0–18)</td>
<td>NS</td>
</tr>
<tr>
<td>SR (intention-to-treat) (n/n) (%)</td>
<td>0/18 (0%)</td>
<td>11/18 (61%)</td>
<td>0.0001</td>
<td>0/7 (0%)</td>
<td>4/7 (57%)</td>
<td>0.0001</td>
<td>0/11 (0%)</td>
<td>7/11 (64%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Recurrence of AF (days) (range)</td>
<td>29 ± 40</td>
<td>39 ± 23</td>
<td>NS</td>
<td>29 ± 46</td>
<td>32 ± 21</td>
<td>NS</td>
<td>28 ± 35</td>
<td>45 ± 22</td>
<td>NS</td>
</tr>
</tbody>
</table>

Statistical analysis between subgroup values based on Mann-Whitney U test comparing first and second cardioversion with respect to defibrillation vector. *RA-CS defibrillation vector right atrium-coronary sinus (n = 7); †RA-PA defibrillation vector right atrium-pulmonary artery (n = 11). Values represent mean ± standard deviation and (range) or number and percent of patients.

1° = first; 2° = second; AF = atrial fibrillation; CK(-Mb) = creatinine kinase (muscle-brain type); IC = internal cardioversion; NS = not significant (p > 0.05); Ω = Ohm(s); SR = sinus rhythm.
of chronic AF and can be assumed to be reversible up to one week after restoration of sinus rhythm (33). With chronic persistence of AF, however, these phenomena can impinge on the type of AF (2,33,34) and thus influence its reversibility (10,21,25,27,31,33). By contrast, organic alterations, such as insulating collagenous septa between atrial muscle bundles (8), develop over months and years (34), but once present, they are irreversible, rendering restoration and maintenance of sinus rhythm more difficult (1,7).

According to both the pathophysiological aspects and the clinical data of the current study as well as others (10,22,24,25,31,33), and taking all the possible sequelae of AF into consideration (1–5), an early cardioversion of AF should be generally strived for. Comparably low energy requirements caused by a lower defibrillation threshold after a comparably short AF duration, and the aim of preventing occurrences or worsening of the above-mentioned AF-related phenomena, support this view.

**Influence of defibrillation threshold on pain perception.** Recent studies have shown variable response to intracardiac shocks on pain perception (12,16,20,22,37). In our population, there seems to be a strong correlation between energy applied and pain perceived (Fig. 4). Our previous findings that energy might determine shock effectiveness but peak voltage determines pain perception (18) are supported in this study. Thus, expanding the shock width may afford a reduction in leading edge voltage, in order to improve tolerability and to maintain efficacy of internal cardioversion (18,37).

**Influence of defibrillation vector on defibrillation threshold.** The selection of the catheter site in the right atrium and coronary sinus or in the right atrium and left pulmonary artery determining the shock vector seems to have an effect on efficacy and on tolerability of internal cardioversion. As reported previously, the right atrium–coronary sinus vector requires lower energy thresholds compared with the right
atrium-pulmonary artery vector (27,38,39). This beneficial effect on energy implies a favorable effect on tolerability of internal cardioversion, which can be seen in our group of patients undergoing cardioversion for the first time (p = 0.073). In the second cardioversion, a trend favoring the right atrium-coronary sinus vector can be appreciated (p = 0.102, NS).

**Influence of antiarrhythmic medication on defibrillation threshold.** In order to maintain sinus rhythm, patients were treated with oral sotalol (mean daily dose 175 ± 55 mg, range 160 to 400 mg) based on prior experience (37,39). In this study, we tried to eliminate the effect of sotalol on the defibrillation threshold by discontinuing medication at least 48 h before cardioversion, which equals four times the elimination half-life of sotalol of 12 h (40,41). None of the 18 patients had renal insufficiency. So far, sotalol has been shown to be effective in lowering the defibrillation threshold in animals (42) and patients with acute atrial fibrillation (43) only when given concurrently with a low-energy shock. By contrast to our study, sotalol was given intravenously (5 mg/kg in canines, 1.5 mg/kg in humans) and did not reduce the threshold in patients with chronic AF, but ventricular pacing was required. None of our 18 patients required ventricular pacing during or after the cardioversion, consistent with the prior termination of sotalol medication. However, plasma levels of sotalol were not obtained.

**Clinical implications and conclusions.** Initially high defibrillation thresholds could be reduced upon early renewed shock delivery, presumably due to shorter duration of AF episodes (24,25). The reported favorable alterations in the atrial defibrillation threshold for repeated internal cardioversion could potentially improve the pain perception associated with intracardiac shock delivery; consequently, the number of patients could increase in whom an implantable device might be considered (2,11,12,18,20,25).

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**Figure 4.** Correlation between defibrillation thresholds (in J) for first (open symbols) and second (filled symbols) internal cardioversion and pain scores (measured by means of a scale ranging from 0 = not felt to 10 = intolerable) at successful internal cardioversion of chronic and recurrent AF with respect to defibrillation vectors. (Top) Right atrium-coronary sinus (triangles). (Bottom) Right atrium-pulmonary artery (circles).
An implantable atrial defibrillator devised for an extended circle of patients with recurrent AF should incorporate all qualities required for optimal effectiveness of internal cardioversion already mentioned and proper detection of AF in order to keep episodes of recurrent AF short, thus preventing electrical remodeling of the atria (1,25).

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REFERENCES