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Left Atrial Chamber and Appendage Function After Internal Atrial Defibrillation: A Prospective and Serial Transesophageal Echocardiographic Study

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Objectives. **The purpose of this prospective study was to assess left atrial chamber and appendage function after internal atrial defibrillation of atrial fibrillation and to evaluate the time course of recovery.**

Background. **External cardioversion of atrial fibrillation may result in left atrial appendage dysfunction ("stunning") and may promote thrombus formation. In contrast to external cardioversion, internal atrial defibrillation utilizes lower energies; however, it is unknown whether the use of lower energies may avoid stunning of the left atrial appendage.**

Methods. **Transesophageal and transthoracic echocardiography were performed in 20 patients 24 h before and 1 and 7 days after internal atrial defibrillation to assess both left atrial chamber and appendage function. Transthoracic echocardiography was again performed 28 days after internal atrial defibrillation to assess left atrial function. The incidence and degree of spontaneous echo contrast accumulation (range 1+ to 4+) was noted, and peak emptying velocities of the left atrial appendage were measured before and after internal atrial defibrillation. To determine left atrial mechanical function, peak A wave velocities were obtained from transmitral flow velocity profiles.**

Results. **Sinus rhythm was restored in all patients. The mean ± SD peak A wave velocities increased gradually after cardioversion, from 0.47 ± 0.16 m/s at 24 h to 0.61 ± 0.13 m/s after 7 days ($p < 0.05$) and 0.63 ± 0.13 m/s after 4 weeks. Peak emptying velocities of the left atrial appendage were 0.37 ± 0.16 m/s before internal atrial defibrillation, decreased significantly after internal atrial defibrillation to 0.23 ± 0.1 m/s at 24 h ($p < 0.01$) and then recovered to 0.49 ± 0.23 m/s ($p < 0.01$) after 7 days. The corresponding values for the degree of spontaneous echo contrast were 1.2 ± 1.2 before internal atrial defibrillation versus 2.0 ± 1.0 ($p < 0.01$) and 1.1 ± 1.3 ($p < 0.01$) 1 and 7 days after cardioversion, respectively. One patient developed a new thrombus in the left atrial appendage, and another had a thromboembolic event after internal atrial defibrillation.**

Conclusions. **Internal atrial defibrillation causes depressed left atrial chamber and appendage function and may result in the subacute accumulation of spontaneous echo contrast and development of new thrombi after cardioversion. These findings have important clinical implications for anticoagulation therapy before and after low energy internal atrial defibrillation in patients with atrial fibrillation.**

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Atrial fibrillation occurs in 0.3% to 0.4% of the adult population, and its prevalence increases with age (1). One of its consequences is the loss of organized atrial contractions, which in turn causes a predisposition to thromboembolism. As a result, there is a fivefold increase in risk for stroke in patients with atrial fibrillation (2). However, cardioversion of atrial fibrillation is not without risk because the procedure itself may cause embolic events (3). It has been suggested that thromboembolism occurs after cardioversion because atrial contraction is restored, which may dislodge fresh thrombi that have formed during atrial fibrillation. Recent studies have provided additional explanations of the mechanism of postcardioversion thromboembolism: Cardioversion itself may create a thrombogenic mileu that may increase the risk of development of left atrial appendage thrombi after cardioversion. Investigators using transesophageal echocardiography during external electrical cardioversion have shown (4) that the mechanical function of the left atrial appendage is depressed immediately after external electrical cardioversion. In addition, the development of spontaneous echo contrast accumulation—an echogenic swirling pattern of blood flow—may be observed in the left atrium and left atrial appendage after external electrical cardioversion. These findings suggest that stunned left atrial appendage function after external electrical cardioversion may predispose the atria to thrombus formation despite the restoration of sinus rhythm (5).

Transvenous atrial cardioversion of atrial fibrillation has

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Abbreviations and Acronyms

INR = international normalized ratio
VTI_A = velocity time integral of atrial filling wave
VTI_E = velocity time integral of early filling wave

been introduced as an alternative approach (6). In contrast to external cardioversion, it utilizes lower energies. The use of lower energies may avoid the stunning of the left atrial appendage and left atrium or may result in an earlier recovery of left atrial appendage function than that after external cardioversion. However, left atrial chamber and appendage function after internal atrial defibrillation have not yet been described. The purpose of this prospective study was to assess left atrial appendage and left atrial function after internal atrial defibrillation and to evaluate the time course of their recovery.

Methods

Study patients. The study group included 20 adult patients who underwent successful transvenous internal atrial defibrillation of atrial fibrillation. Patient data are provided in Table 1. Except for two patients, all patients received oral digoxin before internal atrial defibrillation to achieve heart rate control.

Table 1. Patient Characteristics

Pt No./ Gender	Age (yr)	Etiology	Duration of AF (days)	Antiarrhthmic Drugs	Total Energy (J)
1/M	50	Idiopathic	3	Flecainide	19.5
2/F	81	Hypert	4	None	29.9
3/M	72	CHD	49	Amiodarone	29.9
4/F	64	IDC	60	Amiodarone	19.5
5/M	54	CHD	21	Sotalol	65
6/M	53	IDC	45	Amiodarone	45
7/M	43	IDC	350	Sotalol	4.6
8/M	56	Hypert	11	Sotalol	65
9/M	68	CHD	70	Sotalol	27.8
10/M	39	IDC	45	Sotalol	29.9
11/M	54	HCM	66	Sotalol	13.2
12/F	64	Idiopathic	4	Amiodarone	19.5
13/M	56	CHD	112	Sotalol	19.5
14/M	74	CHD	90	Sotalol	19.5
15/M	60	CHD	90	Sotalol	13.2
16/M	61	Idiopathic	360	Amiodarone	2.5
17/F	68	CHD	180	Sotalol	0.5
18/M	51	Idiopathic	42	Sotalol	3.5
19/F	64	Idiopathic	49	Flecainide	5.1
20/M	64	Hypert	260	Flecainide	5.5
Mean	59		78		22
±SD	±11		±89		±19

AF = atrial fibrillation; CHD = coronary heart disease; F = female; IDC = dilatative cardiomyopathy; HCM = hypertrophic cardiomyopathy; Hypert = hypertensive heart disease; M = male; Pt = patient; Total Energy = total amount of energy required for atrial defibrillation.

Both transthoracic and transesophageal echocardiography were performed <4 h before internal atrial defibrillation, as well as 1 and 7 days after internal atrial defibrillation. In addition, transthoracic echocardiography was undertaken 4 weeks after defibrillation. Written informed consent was obtained from all patients, and the study was approved by the Institutional Review Board of the University of Bonn.

Anticoagulation. At least 2 weeks before internal atrial defibrillation, all patients received Coumadin (warfarin sodium) to prolong the prothrombin time to an international normalized ratio level of 2.0 to 3.0. Four days before internal atrial defibrillation, Coumadin was discontinued, and intravenous weight-adjusted heparin (17 U/kg body weight per h) was administered. The dosage of heparin was adjusted in each patient to achieve an activated partial thromboplastin time ratio of 1.5 to 2.5 times the control value. Two hours before the actual procedure, heparin was discontinued to minimize bleeding complications and to allow safe puncture of the femoral and subclavian veins. After the procedure, all sheaths were removed. Intravenous heparin was reintroduced 4 h later as a bolus of 5,000 U and readjusted to the level before the procedure. On the same evening of the procedure, warfarin administration was restarted. Twenty-four hours after internal atrial defibrillation, activated partial thromboplastin time was measured to ensure that therapeutic levels were achieved in all patients (>1.5 times the control value in all patients).

Echocardiographic studies. All studies were conducted with commercially available equipment (Vingmed 800 C, Vingmed Sound, Horton, Norway). To allow off-line quantitative analysis of the echocardiographic data, studies were recorded on videotape with selected cine loops and velocity spectra digitally transferred to a Macintosh PowerPC computer for subsequent analysis.

For transthoracic echocardiography, a 3.25-MHz transducer was used, and all patients were examined in the left lateral decubitus position. A one-lead electrocardiogram was recorded continuously. The M-mode left atrial dimension was measured at end-systole in the parasternal long-axis view, and left ventricular ejection fraction was determined according to the recommendations of the North American Society of Echocardiography (7). Transmitral Doppler inflow velocities were recorded from the apical four-chamber view with the sample volume positioned between the tips of the mitral leaflets during quiet respiration.

Transesophageal echocardiography was performed with a 5-MHz multiplane transducer. Topical cetacaine spray and viscous lidocaine solution were used to anesthetize the oropharynx before transesophageal echocardiography. The imaging plane and gain settings were adjusted to achieve optimal visualization of the appendage and spontaneous echo contrast. Cine loops of the left atrium and left atrial appendage were stored. The sample volume of the pulsed Doppler was placed 1 cm into the orifice of the left atrial appendage, and the profile of the velocities was recorded. Care was taken to

minimize the angle of incidence of the Doppler beam for flow assessment.

Echocardiographic data analysis. Echocardiographic evaluations were performed in single-blinded manner, with the results confirmed by two independent observers after the original examination. Precardioversion and postcardioversion data were evaluated by different observers who had no knowledge of the precardioversion results and the internal atrial defibrillation procedure itself. The data were analyzed by means of the evaluation software provided by the manufacturer (Echodisp, Vingmed Sound).

Transthoracic echocardiography. The variables derived from the transmitral velocity spectra were peak velocity of the early (E) and atrial filling (A) waves; the corresponding velocity time integrals (VTI_E and VTI_A); and the percentage of atrial contribution to total left ventricular filling. The percentage of atrial contribution to total left ventricular filling was determined by dividing VTI_A by the total diastolic velocity time integral (8). The results of five consecutive heart cycles were averaged in each patient.

Transesophageal echocardiography. The cine loops of the left atrium and left atrial appendage were examined for thrombi and spontaneous echo contrast. The degree of spontaneous echo contrast was categorized by two independent observers as being absent (0), mild (1+), mild to moderate (2+), moderate (3+) or severe (4+) on the basis of the system described by Fatkin et al. (4): 0 = absence of echogenicity; 1+ = minimal echogenicity only detectable with optimal gain settings transiently during the cardiac cycle; 2+ = transient spontaneous echo contrast detectable without increased gain settings and more dense pattern than 1+; 3+ = dense pattern of echogenicity during the entire cardiac cycle; 4+ = intense echo density with a slow swirling pattern without fluctuation in intensity during one cardiac cycle. Interobserver differences were resolved by a third observer. Left atrial appendage area was measured before cardioversion by tracing a line starting from the top of the limbus of the left upper pulmonary vein along the endocardial border of the appendage. Maximal appendage area was determined during five heart cycles and averaged. Before cardioversion, the left atrial appendage showed a fibrillatory pattern in the Doppler recordings. Peak emptying and filling wavelets were measured in seven consecutive cycles each, and maximal velocities were then averaged. After cardioversion, left atrial appendage function was organized, and late diastolic peak emptying and filling velocities were determined from the velocity spectra of the left atrial appendage. In sinus rhythm, five peak velocities were averaged.

Internal atrial defibrillation. Biphasic (3/3 ms), R wave synchronous shocks were delivered by an external defibrillator (Telectronics DTS) between large surface area electrodes (Elecath Inc.) positioned in the coronary sinus and right atrial appendage. To determine the atrial defibrillation threshold, a step-up voltage protocol starting at 60 V was applied. Voltage was increased in 40-V steps up to a leading edge voltage of 340 V. Thereafter, shocks were delivered at 440, 540 and 640 V until successful cardioversion was achieved. A third electrode was positioned in the apex of the right ventricle for proper R wave synchronization.

Statistical analysis. Analysis of variance for repeated measures was used for analysis of serial changes of continuous echocardiographic variables (size of left atrium, left atrial appendage velocities, A wave of mitral inflow) after internal atrial defibrillation. Further comparisons were subject to Bonferroni correction. In all cases, $p < 0.05$ was considered statistically significant. Categoric data (degree of spontaneous echo contrast) were compared by the Wilcoxon signed rank test for matched pairs.

Univariate regression analysis was performed to evaluate the association with peak emptying flow velocities of the left atrial appendage and the following variables: age, duration of the last episode of atrial fibrillation, left ventricular ejection fraction, left atrial chamber and appendage size, spontaneous echo contrast, peak emptying velocities of the left atrial appendage, number of shocks and amount of total energy required for atrial defibrillation. Only variables that were univariately significant at the $p < 0.05$ level were taken for stepwise backward multiple linear regression analysis (F to remove: 4.0).

Results

Patients. All patients underwent successful internal atrial defibrillation. A mean (±SD) of 7.3 ± 2.4 shocks were delivered per patient (range 2 to 11). The mean atrial defibrillation threshold for successful restoration of sinus rhythm was 7.9 ± 6.3 J, corresponding to 355 ± 151 V. The average rest heart rate was 86 ± 18 beats/min before internal atrial defibrillation, 71 ± 14 beats/min at 24 h, 69 ± 13 beats/min at 7 days after and 70 ± 14 beats/min at 28 days after internal atrial defibrillation. Ejection fraction changed from 57 ± 18% before cardioversion to 56 ± 16%, 60 ± 18% and 65 ± 13% at 1, 7 and 28 days, respectively. Maximal left atrial appendage area before internal atrial defibrillation was 5.5 ± 2.2 cm^2. During the follow-up period, at 1 month, 18 of 20 patients were still in sinus rhythm. Atrial fibrillation recurred in two patients within the first week after internal atrial defibrillation. One patient had a thromboembolic event, with cerebral infarction 36 h after internal atrial defibrillation.

Pulsed Doppler transmitral recordings. Immediately after cardioversion, an A wave was clearly discernible in 19 patients. In one patient, the A wave was not detectable, although the patient was in sinus rhythm. Serial evaluation of peak A wave velocity demonstrated a significant increase after 7 days. The increase after 28 days proved not to be significant. The percentage of atrial contribution to total left ventricular filling did improve significantly within the first week, but showed no significant improvement over the next 3 weeks (Fig. 1). Peak E wave velocity did not change significantly either throughout the first week of cardioversion (0.9 ± 0.25 vs. 0.79 ± 0.2 m/s). After 28 days, peak E wave velocity was significantly decreased (0.9 ± 0.25 vs. 0.7 ± 0.2 m/s, $p = 0.02$).

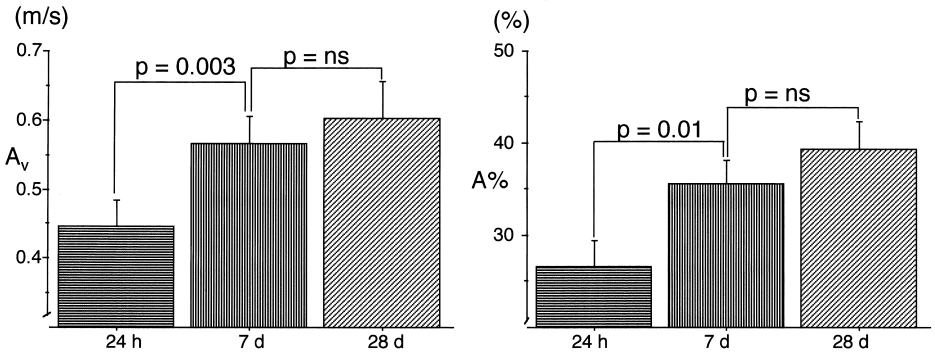


Figure 1. Peak transmitral A wave velocity and percent atrial contribution to left ventricular filling after internal atrial defibrillation. A_v = peak velocity of atrial filling wave; A% = percentage of atrial contribution to total left ventricular filling.

Left atrial size and spontaneous echo contrast. Left atrial size did not change during serial evaluation (44 ± 7 mm before vs. 45 ± 7 mm 28 days after internal atrial defibrillation). Spontaneous echo contrast was detected in the left atrium in 12 (60%) of 20 patients before cardioversion. Immediately after cardioversion, spontaneous echo contrast was found in 13 (65%) of 20 patients, and the score of the intensity of spontaneous echo contrast increased significantly, from 1.2 ± 1.2 before cardioversion to 2 ± 1 after internal atrial defibrillation. This increase in the intensity of spontaneous echo contrast disappeared after 1 week (2 ± 1 immediately vs. 1.1 ± 1.3 after 1 week). The individual changes in the intensity of spontaneous echo contrast are presented in Table 2. In one patient a new thrombus was detected 24 h after internal atrial defibrillation in the left atrial appendage (Fig. 2). This patient also exhibited dense spontaneous echo contrast. By the seventh day of the follow-up period, the thrombus had dissolved under anticoagulant therapy.

Left atrial appendage flow velocity profile. Before internal atrial defibrillation, the profile of the velocities of the left atrial appendage demonstrated a fibrillatory pattern (Fig. 3) in all patients. Twenty-four hours after conversion to sinus rhythm,

Table 2. Peak Emptying Velocities of Left Atrial Appendage and Degree of Spontaneous Echo Contrast Before and After Internal Atrial Defibrillation

Pt No.	Peak Emptying Velocity of Left Atrial Appendage			Grade of Spontaneous Echo Contrast		
	Before	24 h After	7 d After	Before	24 h After	7 d After
1	0.46	0.18	0.79	0	0	0
2	0.51	0.33	0.54	0	0	0
3	0.39	0.36	0.4	0	1	0
4	0.35	0.35	0.51	1	2	0
5	0.12	0.11	0.18	3	3	2
6	0.18	0.1	0.27	1	3	2
7	0.21	0.21	0.38	2	2	2
8	0.53	0.18	0.62	1	1	1
9	0.45	0.13	0.48	1	2	1
10*	0.15	0.18	0.19	4	4	4
11	0.29	0.13	0.36	2	3	3
12	0.73	0.29	0.74	0	0	0
13	0.56	0.29	0.91	2	3	0
14	0.35	0.28	0.36†	3	3	3
15	0.3	0.2	0.32†	0	1	
16	0.3	0.12	0.29	2	3	2
17	0.42	0.41	0.6	0	0	0
18	0.58	0.41	0.9	0	0	0
19	0.22	0.22	0.38	0	0	0
20‡	0.36	0.14	0.35	1	4	3
Mean	0.37	0.23	0.49	1.2	2	1.1
±SD	±0.16	±0.1	±0.23	±1.2	±1	±1.3
p value		<0.001	<0.001		0.003	0.006

*Patient had thromboembolic event with cerebral infarction 36 h after internal atrial defibrillation. †Patient in atrial fibrillation. ‡Patient developed a new thrombus 24 h after internal atrial defibrillation, which resolved over the next 7 days. d = days; Pt = patient.

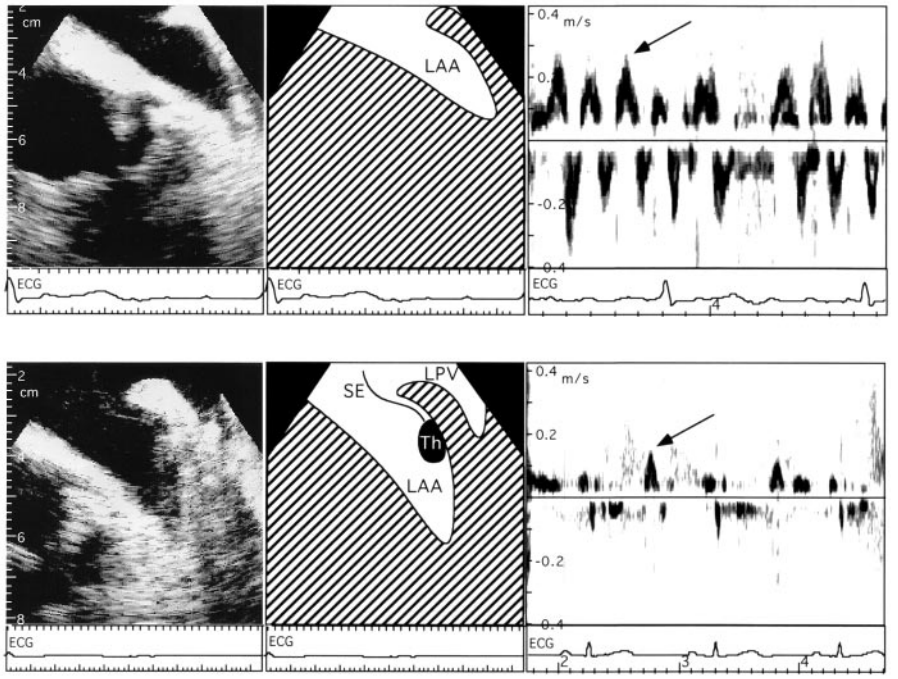
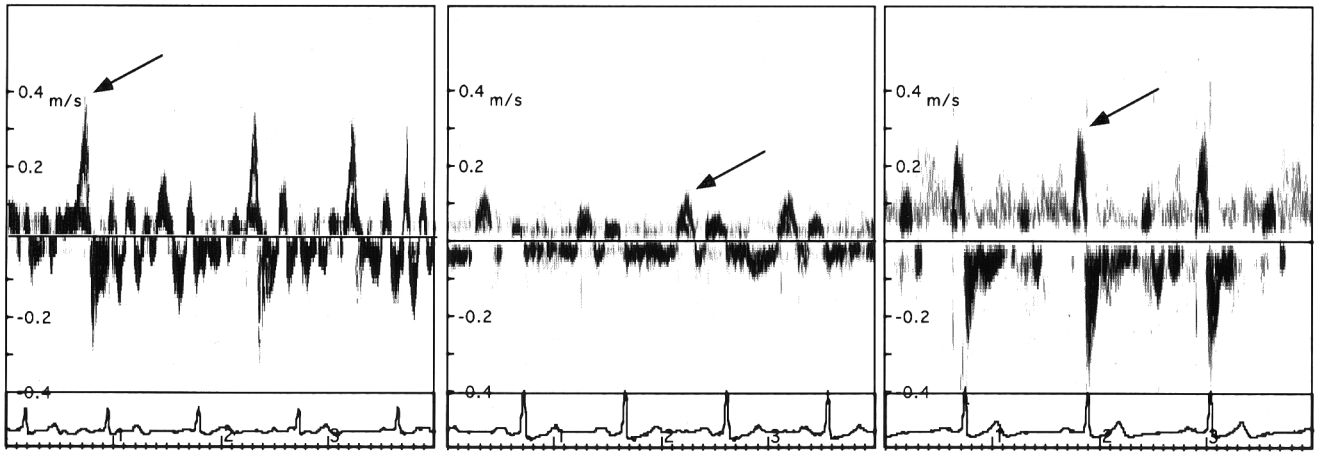


Figure 2. Original image and schematic drawing of the left atrial appendage and left atrial appendage velocity spectrum before and 24 h after internal atrial defibrillation. After internal atrial defibrillation, peak emptying velocities of the left atrial appendage **(arrows)** were reduced with the formation of a new thrombus and increased spontaneous echo contrast in the left atrial appendage. LAA = left atrial appendage; SE = spontaneous echo contrast; LPV = left pulmonary vein; Th = thrombus; ECG = electrocardiogram.

the profile of the velocities of the left atrial appendage demonstrated an organized pattern: 16 patients (80%) had a pattern of both early and late diastolic emptying and filling velocities, whereas 4 (20%) had a pattern exhibiting only late diastolic emptying and filling velocities (Fig. 3). Because emptying and filling velocities did not differ significantly, only emptying velocities are reported. Late diastolic velocities were consistently higher than early diastolic velocities. Values of peak emptying velocities are shown in Table 2. Peak emptying velocities were significantly reduced 24 h after cardioversion compared with peak fibrillatory velocities before cardioversion. However, 7 days after cardioversion, peak emptying velocities had recovered. Within this period, the peak left atrial appendage velocities reached the same levels as those obtained before atrial defibrillation. This could also be demonstrated for the two patients who had recurrence of atrial fibrillation.

Age, left ventricular ejection fraction, left atrial appendage area, duration of atrial fibrillation before atrial defibrillation, number of shocks and total amount of energy required for internal atrial defibrillation were not significantly correlated with peak emptying velocities of the left atrial appendage 24 h after internal atrial defibrillation. However, left atrial size ($R = -0.55$), peak emptying velocities before internal atrial defibrillation ($R = 0.56$) and spontaneous echo contrast ($R = -0.44$) correlated significantly with peak emptying velocities of the left atrial appendage 24 h after internal atrial defibrillation.

Figure 3. Examples of the flow velocity profile in the left atrial appendage before and 24 h and 7 days after internal atrial defibrillation. Before cardioversion, the velocity profile shows a fibrillatory pattern. Twenty-four hours after cardioversion, the flow velocity profile demonstrates an organized pattern, but velocities are reduced. Seven days after cardioversion, left atrial appendage velocities have recovered.



Multiple stepwise regression analysis revealed left atrial size and peak emptying velocities of the left atrial appendage before internal atrial defibrillation as the only significant factors for determining peak emptying velocities of the left atrial appendage 24 h after internal atrial defibrillation ($R = 0.63$, $p < 0.01$).

Relation between pulsed Doppler transmitral recordings, left atrial size, left atrial appendage function and spontaneous echo contrast. Patients with and without spontaneous echo contrast did not differ in age (58 ± 11 vs. 64 ± 10 years) or grade of mitral regurgitation (0.7 ± 0.7 vs. 0.4 ± 0.5). However, patients without spontaneous echo contrast exhibited a shorter duration of the most recent episode of atrial fibrillation (52 ± 59 vs. 96 ± 103 days, $p < 0.01$), higher peak emptying velocities of the left atrial appendage (0.45 ± 0.16 vs. 0.31 ± 0.15 m/s, $p < 0.01$) and smaller left atria (43 ± 4 vs. 47 ± 7 mm, $p = 0.02$) than patients with spontaneous echo contrast in the left atrium. Peak A wave velocities tended to be lower in patients with than in those without spontaneous echo contrast (0.42 ± 0.19 vs. 0.47 ± 0.2); however, this difference was not statistically significant. The reduction in left atrial appendage velocities did not differ significantly between both groups, although there was a tendency for patients with spontaneous echo contrast to have lower left atrial appendage velocities after cardioversion than patients without spontaneous echo contrast. Likewise the percentage of atrial contribution to left ventricular filling and peak velocity of the A wave improved more rapidly in patients without than in those with spontaneous echo contrast. However, these patients also exhibited a shorter duration of atrial fibrillation.

Discussion

Transvenous internal atrial defibrillation is an alternative approach for cardioversion of atrial fibrillation. It allows the successful use of markedly lower energies than those used in external electrical cardioversion (9). Internal atrial defibrillation has recently been shown (6) to be both safe and effective. To our knowledge, this is the first study to report on the effects of internal atrial defibrillation on left atrial chamber and appendage function.

Left atrial function. The results of the present study demonstrate that despite the use of low energies, left atrial mechanical function is impaired after internal atrial defibrillation, and recovery is delayed. Furthermore, recovery time is related to the duration of atrial fibrillation before cardioversion. These findings are in accordance with previous reports (10–12) of depressed and delayed recovery of left atrial mechanical function after external electrical cardioversion and suggest that the amount of energy has only limited impact on the degree of impairment of atrial mechanical function. However, endocavitary shocks create an electric field within the atria, which may account for the finding that even low energies appear to injure the left atrium as much as the higher, yet less locally concentrated energies during external cardioversion. A different explanation for our observations may be that the left atrium is susceptible to minimal energies, resulting in the impairment of its function even with the low energies used for internal atrial defibrillation. Irrespective of the influence of energies on the atria, it may also be suggested that atrial fibrillation itself causes persistent left atrial dysfunction even after restoration of sinus rhythm. This concept is supported by the finding that the duration of atrial fibrillation before cardioversion and its recovery time after the procedure correlate.

Left atrial appendage function. Spontaneous echo contrast and depressed left atrial appendage velocities observed in transesophageal echocardiography have been associated with an increased risk of thrombi development in the left atrial appendage in patients with atrial fibrillation (13–16). The influence of external electrical cardioversion on left atrial appendage function during and after the procedure has recently been described (4,5). The investigators found that restoration of sinus rhythm caused a return of organized left atrial appendage function in most patients. However, spontaneous echo contrast may appear or its degree may increase in up to 38% of patients. In addition, left atrial appendage velocities were reduced. In one study (5), average emptying velocities of the left atrial appendage showed a 53% reduction, from 0.32 ± 0.15 m/s before to 0.15 ± 0.08 m/s immediately after cardioversion.

Our study demonstrated that internal atrial defibrillation has similar effects on left atrial appendage function throughout the initial 24 h after the procedure. In our study group, spontaneous echo contrast appeared or increased in intensity in 45% of patients, and there was a 38% reduction of peak emptying velocities of the left atrial appendage 24 h after internal atrial defibrillation. Therefore, the effects of external and internal cardioversion seem to be similar with respect to both the generation of spontaneous echo contrast and the reduction of left atrial appendage velocities.

It has been suggested (5,17) that "stunned" left atrial appendage function after cardioversion may produce a thrombogenic milieu that could be the basis for systemic embolization after external electrical cardioversion. This assumption is supported by the observation of the formation of a new thrombus and the occurrence of thromboembolism after external electrical cardioversion (17). However, to the best of our knowledge there have been no reports on actual developments of new thrombi after internal atrial defibrillation. We believe that this is the first study to report on the formation of a new thrombus and subsequent stroke after internal atrial defibrillation.

Although our study patients had therapeutic anticoagulation levels at the time of internal atrial defibrillation, the anticoagulation level was supposingly subtherapeutic within the first 24 h after the procedure because intravenous heparin was discontinued for 4 h after the internal atrial defibrillation procedure to avoid bleeding complications. Thus, the formation of a new thrombus in one patient within 24 h of internal atrial defibrillation may have been promoted by nontherapeutic anticoagulation levels.

This assumption is in accordance with the finding that the common denominator for thromboembolism and the new formation of a thrombus after external electrical cardioversion was inadequate anticoagulation (17). However, in the one patient who developed a cerebral embolism 36 h after internal atrial defibrillation, the anticoagulation level was therapeutic, and the precardioversion and 24-h postcardioversion transesophageal echocardiographic examinations results were negative insofar as thrombi were concerned. In this patient, left atrial appendage velocities were severely depressed, and dense spontaneous echo contrast was observed on precardioversion and postcardioversion transesophageal echocardiographic examinations. It may be assumed that in this case the anticoagulation level was not sufficient for preventing new thrombus formation because of the low flow state in the left atrium. However, a very small thrombus with similar echogenicity of the spontaneous echo contrast may have been overlooked on the precardioversion and postcardioversion transesophageal echocardiogram.

Thus, the findings of our study show that internal electrical cardioversion, even with low energies, may cause like external electrical cardioversion left atrial appendage stunning, may result in thrombus formation despite sinus rhythm and may cause subsequent thromboembolism.

Another important finding of this study is that patients with reduced left atrial appendage velocities, large left atria and precardioversion spontaneous echo contrast are prone to a pattern of stunned left atrial appendage function after internal atrial defibrillation. In contrast, patients without precardioversion spontaneous echo contrast develop only mild or no spontaneous echo contrast 24 h after internal atrial defibrillation. These findings indicate that assessing left atrial appendage function, left atrial size and the presence of spontaneous echo contrast before internal atrial defibrillation may help to identify a group of patients with a high risk of stunned left atrial appendage function and, thus, thromboembolism after internal atrial defibrillation.

To determine the long-term effect of internal atrial defibrillation on left atrial appendage function, we performed transesophageal echocardiography 1 week after the procedure. This follow-up examination showed that left atrial appendage velocities return to precardioversion levels within 1 week of internal atrial defibrillation and that spontaneous echo contrast also disappears. Furthermore, the newly developed thrombus found in one of our patients subsequently dissolved under continued anticoagulant therapy within 1 week.

Patients with low precardioversion left atrial appendage velocities did not reach higher levels after the recovery period. Depressed left atrial appendage function in this patient group may be due to previous and unrelated left atrial mechanical dysfunction.

Study limitations. The use of Doppler echocardiography for assessing left atrial mechanical function after cardioversion is widely recognized as a reliable method. Nevertheless, changes in transmitral flow patterns depend on ventricular filling pressure, heart rate, age, preload and Doppler sample position (18,10), which limits the accuracy of this technique. Although care was taken to keep gain settings constant before and after cardioversion for analyzing spontaneous echo contrast, gain settings may vary between studies, and thus comparison of the degree of spontaneous echo contrast is limited. Antiarrhythmic drugs may influence left atrial chamber and appendage function after internal atrial defibrillation. Because these drugs are known to reduce the recurrence of atrial fibrillation, the study design precluded the comparison between the effects of internal atrial defibrillation on left atrial function with and without antiarrhythmic drugs. Whether the results of our study are applicable to recipients of an automatic implantable cardioverter-defibrillator capable of delivering a maximal energy of 3 J should be defined in future studies.

Clinical implications. The results of this study potentially have clinical implications for anticoagulant therapy after internal atrial defibrillation. Twenty-four hours after internal atrial defibrillation, atrial appendage function is depressed, as shown by spontaneous echo contrast, and new thrombi in the left atrial appendage may develop with inadequate anticoagulation. Thus, our findings show that there is a thrombogenic milieu immediately after internal atrial defibrillation. To avoid thromboembolic complications, patients with atrial fibrillation should receive adequate anticoagulation therapy before and immediately after internal atrial defibrillation. It is currently recommended that anticoagulation therapy be continued for 4 weeks after successful cardioversion of atrial fibrillation in patients who have been in atrial fibrillation >2 days (19). However, our results indicate that left atrial appendage function recovers within 1 week of internal atrial defibrillation. Prospective studies should be conducted to demonstrate whether anticoagulant therapy may be discontinued as early as 1 week after the intervention without increasing the risk of thromboembolism.

This study identified a patient group that is prone to a pattern of stunned left atrial appendage function after internal atrial defibrillation as characterized by depressed left atrial appendage velocities and spontaneous echo contrast before cardioversion. Therefore, transesophageal echocardiography may be used for assessing left atrial appendage function before internal atrial defibrillation to exclude patients at high risk of thromboembolism.

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