

Effect of Direct Current Shocks on Left Atrial Mechanical Function in Patients With Structural Heart Disease

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Objectives. This study examined the effect of endocardial and transthoracic direct current (DC) shocks on left atrial and left atrial appendage function in humans with structural heart disease.

Background. DC cardioversion of atrial fibrillation (AF) to sinus rhythm is associated with transient left atrial and left atrial appendage dysfunction and the development of spontaneous echo contrast (SEC). This phenomenon has been termed atrial "stunning" and may be associated with thrombus formation and embolic stroke. To what extent the shock itself contributes to atrial stunning is unclear.

Methods. Thirteen patients in sinus rhythm undergoing implantation of a ventricular implantable cardioverter defibrillator (ICD) were prospectively evaluated. All patients had significant structural heart disease. To evaluate the effects of DC shocks on left atrial and left atrial appendage function, biphasic R wave synchronized endocardial shocks of 1, 10 and 20 J were delivered

between the right ventricular electrode and the left pectoral generator of the ICD in sinus rhythm. R wave synchronized transthoracic shocks of 360 J were also delivered between anteriorly and posteriorly positioned chest electrodes. Transesophageal echocardiography was performed to evaluate left atrial appendage velocities, mitral inflow velocities and the presence of SEC before and immediately after each DC shock.

Results. There were no significant changes in left atrial or left atrial appendage function after endocardial or transthoracic DC shocks. Left atrial SEC did not develop after endocardial or transthoracic DC shocks.

Conclusions. Endocardial and transthoracic DC shocks are not directly responsible for left atrial and left atrial appendage stunning and do not contribute to the stunning that is observed after the cardioversion of AF to sinus rhythm.

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Cardioversion of atrial fibrillation (AF) to sinus rhythm is associated with transient mechanical dysfunction of the left atrium and left atrial appendage and the development of spontaneous echo contrast (SEC) (1-3). This phenomenon has been termed "stunning" and appears to develop after both electrical and pharmacologic cardioversion of AF to sinus rhythm (4,5). There is considerable evidence to suggest that left atrial and left atrial appendage stunning is responsible for the increased risk of thromboembolic stroke after cardioversion of AF to sinus rhythm that may occur hours to weeks after cardioversion (2,6). The exact mechanism underlying the development of atrial stunning remains unknown. There is evidence to suggest that direct current (DC) shocks lead to considerable myocardial damage at both a biochemical and histologic level and that the shock itself may contribute to

atrial stunning (2-4,7-11). Other evidence suggests that it is the arrhythmia itself that is responsible for the postreversion mechanical dysfunction rather than the method of reversion (1,12).

The concept of mechanical dysfunction after cardioversion of AF is well established. To evaluate the contribution of DC shocks to the mechanism of atrial stunning, we assessed the effects of endocardial and transthoracic DC shocks on left atrial and left atrial appendage function in patients in sinus rhythm.

Methods

Patients. Thirteen patients in sinus rhythm undergoing implantation of a ventricular implantable cardioverter-defibrillator (ICD) for accepted indications were recruited. All patients gave written informed consent to participate in the study, which was approved by the Board of Medical Research of the Royal Melbourne Hospital. All patients underwent transthoracic echocardiography and gated blood pool scanning to evaluate left atrial size and left ventricular function before ICD implantation.

ICD implantation procedure. A standardized general anesthetic was given to all patients and consisted of propofol

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Abbreviations and Acronyms

AF	= atrial fibrillation
DC	= direct current
ICD	= implantable cardioverter-defibrillator
SEC	= spontaneous echo contrast
TEE	= transesophageal echocardiography (echocardiographic)
VF	= ventricular fibrillation

induction followed by anesthesia maintained with the volatile agent isoflurane and the muscle relaxant atracurium. All devices were implanted in the left prepectoral position with the ventricular endocardial leads placed at the right ventricular apex.

ICD shock vector, waveform and DC shock delivery. All endocardial shocks were delivered between the right ventricular cathode and the ICD generator anode. After endocardial lead placement and ICD insertion, a standardized series of endocardial DC shocks were delivered with transesophageal echocardiographic (TEE) assessment of left atrial and left atrial appendage function performed before and after each shock. All shocks were delivered in sinus rhythm and were synchronized to the R wave of the intrinsic QRS complex. Shock energy, synchronization and delivery were controlled through telemetry of the ICD system. Biphasic endocardial shocks of 1, 10 and 20 J were delivered, followed by immediate echocardiographic assessment. A 5-min interval separated each shock. Formal testing of the ICD was then performed with induction of ventricular fibrillation (VF) and assessment of endocardial defibrillation thresholds. After each DC shock reversion of VF, another echocardiographic assessment of left atrial and left atrial appendage function was made.

After satisfactory testing and programming of the ICD, a transthoracic DC shock of 360 J synchronized to the R wave was delivered in sinus rhythm, and a further assessment of left atrial and left atrial appendage function was made. Transthoracic shocks were delivered through anteriorly and posteriorly positioned adhesive patches (Physio-Control Fast-Patch, Physio-Control Corporation) with a combined surface area of ~150 cm² connected to an external DC defibrillator (Physio-Control Lifepak 8).

Echocardiographic analysis. Left atrial and left atrial appendage function were assessed before the first shock delivery and immediately after each endocardial or transthoracic shock. Recordings were performed during apnea to minimize potential velocity variations caused by mechanical ventilation. TEE was performed using a 5-MHz phased array multiplane probe connected to a Hewlett-Packard Sonos 2500 ultrasound system. Images were recorded on 0.5-in. super-VHS tape and analyzed off-line using Hewlett-Packard software. All velocity measurements were performed off-line and averaged over 20 cardiac cycles.

Left atrial appendage emptying velocities were assessed using pulsed wave Doppler, with the sample volume placed 1 cm into the mouth of the left atrial appendage. A multiplane

Table 1. Clinical and Echocardiographic Characteristics of 13 Patients Undergoing Implantable Cardioverter-Defibrillator Implantation and Direct Current Shocks in Sinus Rhythm

Age (yr)	55.7 ± 11.3
Male/female	10/3
History of AF	1
Ischemic cardiomyopathy	7
Dilated cardiomyopathy	4
Mitral valve prolapse	2
LVEF (%)	28.2 ± 12.7
LA size (cm)	5.3 ± 0.4
LVEDD (cm)	5.7 ± 0.9
LVEDD (cm)	7.3 ± 0.6
LVFS (%)	22.6 ± 7.9
Drug therapy	
Amiodarone	9
Sotalol	4

Data presented are mean value ± SD or number of patients. AF = atrial fibrillation; LA = left atrial; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; LVEDD = left ventricular end-systolic diameter; LVFS = left ventricular fractional shortening.

probe was used to scan the appendage from 0° to 130° in the transverse plane to establish an angle at which maximal flow velocities could be obtained. This angle was retained for subsequent analysis. Left atrial function was assessed with TEE pulsed wave Doppler interrogation of the mitral inflow E and A waves, with the sample volume placed at the level of the mitral valve leaflet tips.

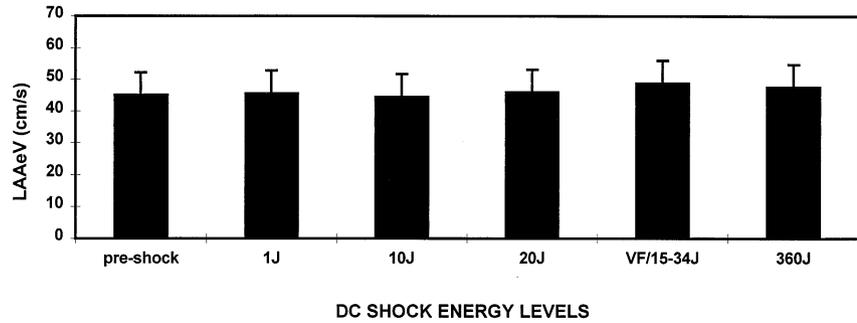
SEC was defined as swirling patterns of echogenicity in the left atrium and left atrial appendage distinct from white noise artifact. Gain settings were reduced in a sequential manner to distinguish echo contrast from noise artifact and were maintained for the duration of the study. Two experienced echocardiographers (J.K., A.Y.) analyzed the preshock and postshock images for SEC. Analysis was performed off-line, and each echocardiographer had no knowledge of other's interpretations.

Statistical analysis. Results are presented as mean value ± SD. A repeated measures analysis of variance was used to analyze Doppler velocities. A p value <0.05 was considered significant. The study was designed with 95% power to detect a mean fall in left atrial appendage velocity of 14 ± 13 cm/s (1). Using a two-tailed test, the required number of patients was 12 (13).

Results

Patients. The study included 13 consecutive patients undergoing implantation of an ICD for accepted indications. All patients were either survivors of resuscitated sudden cardiac death or had rapid ventricular tachycardia or VF at electrophysiologic study. All patients had significant structural heart disease with left ventricular dysfunction (mean left ventricular ejection fraction 28.2 ± 12.7%). Left atrial enlargement was present in all patients (5.3 ± 0.4 cm), and mitral regurgitation ranged from severe to trivial, with a mean grade of 1.2 ± 1.0

Figure 1. Mean left atrial appendage emptying velocities, with standard errors indicated. LAAeV = left atrial appendage emptying velocity.



(Table 1). Nine patients (69%) were taking amiodarone, and 4 (31%) were taking sotalol for ventricular arrhythmias.

Left atrial and left atrial appendage function before shock (Fig. 1 and 2). Mean left atrial appendage velocity for the patients was 45.0 ± 25.6 cm/s. Preshock transmitral A wave velocity was 42.6 ± 14.2 cm/s, and the E wave velocity was 63.0 ± 12.5 cm/s (Table 2). SEC was absent in all patients before shock delivery.

Left atrial and left atrial appendage function after shock. There were no significant changes in transmitral A wave velocities or left atrial appendage velocities compared with preshock baseline values after endocardial DC shock delivery at 1, 10 and 20 J and after external DC shock delivery at 360 J (Table 2). There was no evidence of left atrial stunning caused by endocardial or external shocks delivered in sinus rhythm. Mean sinus rates after endocardial and external shock delivery were not significantly different from baseline values (Table 2). Mean endocardial defibrillation energy delivered for VF was 20.5 ± 5.3 J (range 15 to 34 J). After induction of VF and successful endocardial DC shock reversion, left atrial and left atrial appendage function were again similar to preshock values.

Left atrial SEC. No patient developed left atrial or left atrial appendage SEC after endocardial or external DC shocks delivered in sinus rhythm. Left atrial SEC developed in all patients after induction of VF and cleared rapidly with resumption of coordinated ventricular contraction and effective left atrial emptying after successful ventricular defibrillation.

Discussion

New findings. Transient left atrial and left atrial appendage dysfunction or “stunning” is considered an important

mechanism responsible for the development of late thromboembolism occurring hours to weeks after cardioversion of AF to sinus rhythm (2,3,14). This concept is based largely on the significant changes in left atrial and left atrial appendage flow that develop after cardioversion of AF to sinus rhythm. Decreased late mitral inflow velocities and reduced left atrial appendage velocities after cardioversion correlate strongly with the development of left atrial SEC, which occurs commonly with left atrial appendage thrombosis and the development of late thromboembolic stroke (2).

The findings of the present study challenge the concept that atrial stunning is due in part to the electrical energy delivered at the time of cardioversion. We demonstrated that after both endocardial and externally delivered shocks, left atrial and left atrial appendage function were not significantly affected, and the phenomenon of stunning did not occur. Indeed, there was no alteration in mechanical function associated with the cumulative effects of these sequential shocks. This was shown in a group of patients with structural heart disease who might reasonably be considered similar to patients undergoing cardioversion of AF to sinus rhythm.

Comparison with previous studies. Previous studies of atrial stunning have been performed in patients with AF and atrial flutter and have demonstrated that both low energy internal and higher energy external defibrillation may be associated with depression of left atrial function (1-3,15). Whether the shock itself is of importance in producing atrial stunning has not been studied in detail.

Fatkin et al. (2) demonstrated an association between the number and energy level of DC shocks delivered in AF and the development of SEC with reversion to sinus rhythm. These investigators suggested that the DC shock contributed to the mechanism of atrial stunning. An alternative explanation may

Figure 2. Mean mitral A wave velocities, with standard errors indicated. MVA = mitral valve A wave.

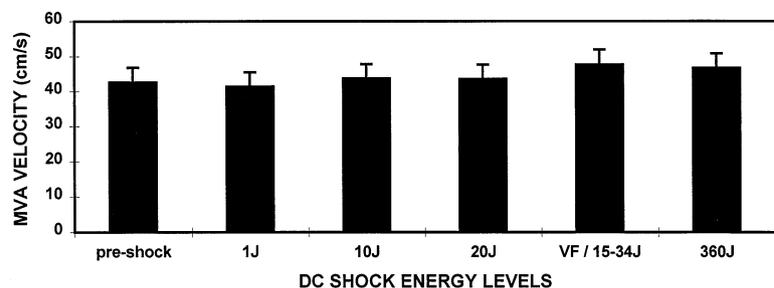


Table 2. Doppler Blood Flow Velocity Measurements and Sinus Rate Before and After Endocardial Direct Current Shocks in Sinus Rhythm and Ventricular Fibrillation and Transthoracic 360-J Shocks in Sinus Rhythm in 13 Patients

	Before Shock	1 J	10 J	20 J	VF 15-34 J	360 J	p Value
LAAeV (cm/s)	45.0 ± 26.6	45.5 ± 25.6	44.4 ± 23.7	45.9 ± 24.7	48.8 ± 25.6	47.6 ± 22.0	0.99
MVA (cm/s)	42.6 ± 14.2	41.3 ± 16.7	43.7 ± 13.6	43.5 ± 14.6	47.7 ± 17.4	46.9 ± 17.0	0.90
MVE (cm/s)	63.0 ± 12.5	62.6 ± 14.2	67.6 ± 10.2	65.3 ± 10.8	68.7 ± 11.3	66.7 ± 11.7	0.70
HR (beats/min)	63 ± 14	61 ± 14	64 ± 13	66 ± 14	67 ± 13	68 ± 13	0.90

Data presented are mean value ± SD. HR = heart rate; LAAeV = left atrial appendage emptying velocity; MVA = mitral valve A wave velocity; MVE = mitral valve E wave velocity; VF = ventricular fibrillation.

be that patients requiring more and higher energy shocks for cardioversion have longer duration AF and more advanced atrial disease, thus predisposing them to the atrial stunning that occurs on termination of the arrhythmia.

Conversely, Falcone et al. (16) examined the effect of ineffective external shocks on left atrial appendage function in patients with AF during attempts at cardioversion to sinus rhythm. Left atrial appendage function in AF did not change appreciably after noncardioverting external DC shocks of 50 to 100 J. The effects of more powerful external shocks on left atrial function and the effects of internally and externally applied energy on left atrial function in sinus rhythm were not examined.

In contrast to studies in which cardioverting DC shocks were delivered during AF, all patients in the present study were in sinus rhythm at the time of DC shock delivery. This approach permitted independent evaluation of the effect of DC current on atrial function in the absence of an effect of cardioversion itself. To enhance the possibility for atrial stunning, we specifically selected a group of patients with significant structural heart disease and atrial dilation (14,17). In addition, all patients were taking antiarrhythmic drugs, such as sotalol, which also exacerbates atrial stunning after DC cardioversion (5). Despite this potent combination of factors, stunning was not observed after DC shocks delivered in sinus rhythm.

The present study also demonstrated that left atrial and left atrial appendage SEC develops during VF but clears almost immediately after resumption of coordinated ventricular contraction after successful ventricular defibrillation. SEC in this circumstance is not a consequence of left atrial stunning but a consequence of ineffective left atrial emptying into the fibrillating ventricle.

Clinical implications. The present study demonstrated that DC shocks do not contribute to atrial stunning. This finding suggests that the stunning observed after cardioversion of AF is mediated by the preceding arrhythmia itself. Although the major aim of the study related to the mechanism of stunning, if our observations also hold true for the fibrillating atrium, there may be several clinical implications:

1. Spontaneous or chemical reversion of AF to sinus rhythm is unlikely to be associated with less atrial stunning than that seen after DC cardioversion. All patients reverting to sinus rhythm from AF should therefore be considered for

prophylactic anticoagulation regardless of the mode of reversion. The decision for anticoagulation may also depend on the chronicity of AF and the existing atrial pathologic features.

2. The efficacy of low energy endocardial defibrillation for AF has been demonstrated, and a multicenter study examining the feasibility of an implanted atrial defibrillator is in progress. Data demonstrate that cardioversion with <5 J can be successful in the majority of patients with paroxysmal forms of AF (18). Our study demonstrates that patients with an implanted atrial defibrillator are not at risk for developing atrial mechanical dysfunction (and hence thromboembolism) as a result of the shock itself. Whether short-duration AF is sufficient to produce atrial stunning is still uncertain.

Implications for ICD recipients. Recent evidence (19) suggests a significant incidence of stroke after ICD discharge for ventricular arrhythmias in patients with chronic AF. Reversion of AF in these circumstances presumably results in atrial stunning. The current study suggests that endocardial shocks delivered during sinus rhythm do not cause atrial stunning and that ventricular ICD recipients in sinus rhythm are not at an increased risk for stroke after defibrillation therapy.

Limitations of the study. All shocks were delivered to atria in sinus rhythm rather than during fibrillation, and our observations may therefore not necessarily be applicable to patients with AF. It is conceivable that a fibrillating atrium is more sensitive to the effects of DC current than an atrium in sinus rhythm. Nevertheless, all patients in our study had structural heart disease, atrial dilation and baseline appendage velocities lower than previously reported normal values and were similar to patients with AF who might be considered candidates for DC cardioversion (20). No further depression of function after repeated sequential shocks to these atria is strong evidence that DC shocks do not contribute to stunning.

The endocardial shock vector differs from the coronary sinus-right atrium vector that is commonly used for internal defibrillation of AF (18). However, the active pectoral device cathode-right ventricular anode configuration is effective in reverting AF to sinus rhythm (19,21). High energy external shocks were also delivered without causing atrial stunning. It is unlikely that ineffective, nondefibrillating energy levels were delivered and that the lack of stunning observed was simply a function of low incident energy or a poorly positioned shock vector.

Conclusions. The present study provides additional information regarding the mechanisms responsible for atrial stunning after cardioversion of AF to sinus rhythm. Endocardial and external DC shocks did not cause atrial stunning, suggesting that DC shocks may not be a contributing mechanism to the stunning that is observed after cardioversion of AF to sinus rhythm. This finding has potentially important implications for recipients of an implanted atrial defibrillator. The present study has added support to the notion that atrial stunning after reversion from AF to sinus rhythm is a function of the preceding arrhythmia itself rather than the mode of reversion.

References

1. Grimm RA, Stewart WJ, Arheart KL, Thomas JD, Klein AL. Left atrial appendage "stunning" after electrical cardioversion of atrial flutter: an attenuated response compared with atrial fibrillation as the mechanism for lower susceptibility to thromboembolic events. *J Am Coll Cardiol* 1997;29:582-9.
2. Fatkin D, Kuchar DL, Thorburn CW, Feneley MP. Transesophageal echocardiography before and during direct current cardioversion of atrial fibrillation: evidence for "atrial stunning" as a mechanism of thromboembolic complications. *J Am Coll Cardiol* 1994;23:307-16.
3. Grimm RA, Stewart WJ, Maloney JD, et al. Impact of electrical cardioversion for atrial fibrillation on left atrial appendage function and spontaneous echo contrast: characterization by simultaneous transesophageal echocardiography. *J Am Coll Cardiol* 1993;22:1359-66.
4. Manning WJ, Silverman DI, Katz SE et al. Temporal dependence of the return of atrial mechanical function on the mode of cardioversion of atrial fibrillation to sinus rhythm. *Am J Cardiol* 1995;75:624-6.
5. Pollak A, Falk RH. Aggravation of postcardioversion atrial dysfunction by sotalol. *J Am Coll Cardiol* 1995;25:665-71.
6. Arnold AZ, Mick MJ, Mazurek RP, Loop FD, Trohman RG. Role of prophylactic anticoagulation for direct current cardioversion in patients with atrial fibrillation or atrial flutter. *J Am Coll Cardiol* 1992;19:851-5.
7. Yarbrough R, Ussery G, Whitley J. A comparison of the effects of AC and DC countershock on ventricular function in thoracotomized dogs. *Am J Cardiol* 1964;14:504-12.
8. Trouton PG, Allen JD, Young LK, Rooney JJ, Adgey AAJ. Metabolic changes and mitochondrial dysfunction early following transthoracic counter-shocks in dogs. *PACE* 1989;12:1827-34.
9. Van Vleet JF, Tacker WA. Cardiac damage from transthoracic and ICD defibrillator shocks. In: Tacker WA, editor. *Defibrillation of the Heart*. St. Louis: Mosby Year Book, 1994:259-98.
10. Aranda JM, Tauth J, Brewington J, Lockeby M, Fontanet H. Atrial stunning occurs after electrical cardioversion, but not after chemical cardioversion [abstract]. *J Am Coll Cardiol* 1997;29 Suppl A:292A.
11. Mattioli AV, Mecenero A, Bastia E, Mattioli G. Atrial ejection force in patients with atrial fibrillation: a comparison between DC shock and pharmacological cardioversion [abstract]. *PACE* 1997;20:1120.
12. Grimm RA, Leung DY, Black IW, Stewart WJ, Thomas JD, Klein AL. Left atrial appendage "stunning" after spontaneous conversion of atrial fibrillation demonstrated by transesophageal Doppler echocardiography. *Am Heart J* 1995;130:174-6.
13. Bland M. *An Introduction to Medical Statistics*. 2nd ed. Oxford: Oxford University Press, 1995:336.
14. Manning WJ, Silverman DI, Katz SE, et al. Impaired left atrial mechanical function after cardioversion: relation to the duration of atrial fibrillation. *J Am Coll Cardiol* 1994;23:1535-40.
15. Omran H, Jung W, Rabahieh R, et al. Left atrial chamber and appendage function after internal atrial defibrillation: a prospective and serial transesophageal echocardiographic study. *J Am Coll Cardiol* 1997;29:131-8.
16. Falcone RA, Morady F, Armstrong WF. Transesophageal echocardiographic evaluation of left atrial appendage function and spontaneous contrast formation after chemical or electrical cardioversion of atrial fibrillation. *Am J Cardiol* 1996;78:435-9.
17. Mattioli AV, Castellani ET, Vivoli D, Molinari R, Molinari G. Restoration of atrial function after atrial fibrillation of different etiological origins. *Cardiology* 1996;87:205-11.
18. Levy S, Ricard P, Lau C-P et al. Multicenter low energy transvenous atrial defibrillation (XAD) trial results in different subsets of atrial fibrillation. *J Am Coll Cardiol* 1997;29:750-5.
19. Shenasa M, Shenasa H. Conversion of atrial fibrillation to sinus rhythm during implantable cardioverter-defibrillator discharges: incidence and implications [abstract]. *PACE* 1997;20:1127.
20. Fatkin D, Kelly RP, Feneley MP. Relations between left atrial appendage flow velocity, spontaneous echocardiographic contrast and thromboembolic risk in vivo. *J Am Coll Cardiol* 1994;23:961-9.
21. Poole JE, Kudenchuk PJ, Dolack GL et al. Atrial defibrillation using a unipolar, single lead right ventricular to pectoral can system [abstract]. *J Am Coll Cardiol* 1995;25 Suppl A:110A.