Higher Energy Monophasic DC Cardioversion for Persistent Atrial Fibrillation: *Is it Time to Start at 360 Joules?*

C Boos, M.B.B.S., M.R.C.P.,* MD Thomas, M.B.B.S., M.R.C.P.,†
A Jones, M.B.B.S., M.R.C.P.,‡ E Clarke, M.B.B.S., M.R.C.P.,*
G Wilbourne, M.B.B.S., M.R.C.P.,* and RS More, M.B., Chb,*

From the *St Mary's Hospital, Milton Road, Portsmouth, PO3 6AD, UK; †Department of Cardiology, Charing Cross Hospital, London, UK; ‡Centre For Cardiovascular Medicine, University College Hospital, London, WC1E 6JJ, UK

Background: Electrical direct-current cardioversion (DCCV) has become a routine therapy for atrial fibrillation (AF), although some uncertainty remains regarding the optimal energy settings.

Aims: This study examines whether the use of a higher initial energy monophasic shock of 360 joules (J) for external DCCV, in patients with persistent AF would prove more effective, yet as safe, as the use of a lower initial energy 200 J shock.

Methods: A cohort of 107 patients with persistent AF was prospectively randomized to an initial synchronized DCCV shock of 360 J versus 200 J (n = 50 vs 57), followed by a similar shock sequence thereafter of four further shocks of 360 J for the two groups. In all patients the levels of troponin I (cTnI) were measured precardioversion and 18–20 hours later, the following day. In a subgroup of 36 patients in each group, the levels of creatine kinase (CK) and aspartate transaminase (AST) were measured pre- and 18–20 hours postcardioversion.

Results: The success rate for DCCV was significantly higher in the 360 J group compared to the 200 J group (96.0% vs 75.4%, $P = 0.003$). The mean CK IU/L levels (1137.5.0 vs 2411.8, $P = 0.014$) and AST levels (39.83 vs 52.86, $P = 0.010$) were significantly lower in the 360 J group compared to the 200 J group. There was no statistical rise in cTnI ($\mu\text{g/L}$) in either group ($P = 1.00$). The average number of shocks delivered (1.84 vs 2.56, $P = 0.006$) was significantly less in the 360 J group than in the 200 J group, although total energy requirements for DCCV were similar for the two groups (662.4 J vs 762.4 J, $P = 0.67$).

Conclusion: For patients with persistent AF the use of a higher initial-energy monophasic shock of 360 J achieves a significantly greater success rate, with less skeletal muscle damage (and no cardiac muscle damage) as compared with the traditional starting energy of a 200 J DC shock.

A.N.E. 2003;8(2):121–126

cardioversion; atrial fibrillation; monophasic; external DCCV

Atrial fibrillation (AF) is the most common sustained tachyarrhythmia found in clinical practice^{1–3} and its incidence is increasing.⁴ It is the most common cause of embolic stroke,⁵ and is associated with a doubling of overall morbidity and mortality from cardiovascular disease.⁶ Electrical direct-current cardioversion (DCCV) has become a routine therapy for AF patients since its introduction in 1962.⁷

Lately, intracardiac⁸ and transoesophageal cardioversion⁹ have provided alternatives to tra-

ditional external DCCV, often where external cardioversion has failed. However, these techniques are technically more difficult and may have a greater risk of complications. Recently, it has been shown that transthoracic biphasic shocks are superior to, and possibly safer than, monophasic shocks.¹⁰ However, the widespread application of biphasic defibrillation will take some time, and at present, the success of monophasic defibrillation needs to be maximized.

DCCV has been shown to result in skeletal muscle injury and release of creatine kinase (CK) and aspartate transaminase (AST).^{11,12} Cardiac troponin I (cTnI) and T are myofibrillator proteins that are specific to myocardial cells. Their levels do not increase after routine cardioversion^{13–15} suggesting that cardiac damage does not occur following DCCV. Changes in cTnI in combination with AST and CK levels help ascertain whether there has been cardiac or skeletal damage after cardioversion.

There has been increasing evidence that higher initial energy monophasic shock levels (200 J or higher) are safe and may lead to higher overall success rates of conversion to sinus rhythm.^{16–18} The Working Group of the European Cardiac Society currently recommends an initial minimal starting energy level of at least 200 J for DCCV.¹ This study certainly supports this view and compares the efficacy, safety, and success of starting DCCV at the higher initial energy of 360 J instead of 200 J as part of a standard external monophasic DCCV algorithm.

METHODS

Patients

Ethical approval was granted by The Portsmouth Research Development and Ethical Committee and informed consent to enter the study and to separately perform the procedure of cardioversion was obtained from each patient. The subjects of this study were 107 consecutive patients who underwent elective external cardioversion for stable persistent AF.

Inclusion Criteria

All included patients had a serum potassium between 3.5 and 5.0 mmol/L, with normal renal and thyroid function. Prior to cardioversion the patients received a minimum of 3 weeks of anticoagulation with warfarin and an INR of 2.0–3.0 was taken to be therapeutic.² We included patients aged 16–80 years, with sustained AF duration greater than 1 month, who were considered suitable for cardioversion by the referring physician. Antiarrhythmic therapy was decided by the referring physician.

Exclusion Criteria

The following groups of patients were excluded from the study: patients with hemodynamically unstable AF in whom cardioversion had to be per-

formed urgently; LA dimensions > 60 mm measured by M Mode echocardiography; hypo- or hyperthyroidism; untreated or inadequately treated hypertension; pregnancy; patients with significant cardiac failure (NYHA III/IV); patients with prosthetic valves; and patients who had undergone a cardioversion within the previous 3 months.

Study Design

All patients were investigated prior to cardioversion with transthoracic echocardiography, and renal function, liver profile, and thyroid function test measurements were performed. All patients had their renal profile, full blood count, and INR checked 48 hours prior to cardioversion. The patients were sedated with intravenous propofol and external cardioversion was performed with two manual rectangular paddles (8 × 10 cm) delivered by the Codemaster XL⁺ (Hewlett Packard). In a subgroup of 36 patients the levels of AST and CK were checked 48 hours precardioversion and then remeasured at 18–20 hours postcardioversion. All patients had their levels of cTnI levels checked 48 hours precardioversion and at 18–20 hours postcardioversion.

Patients were prospectively randomized in a single blind fashion to one of the two initial anterior-apical (AA) shock sequences 360 J (n = 50) versus 200 J (n = 57). The shock sequence thereafter of four further shocks was similar for the two groups (Fig. 1): 1 × 360 J AA, 1 × 360 J AA, 1 × 360 J anterior-posterior (AP) and 1 × 360 J AP. For the AA position the anterior paddle was placed at the right sternal border and the lateral paddle was placed over the cardiac apex. For the anteroposterior approach the anterior paddle was placed at the left sternal border and the posterior paddle at the angle of the left scapula. New gel pads were used after the first three shocks were delivered. To avoid myocardial damage the interval between two successive shocks was greater than 1 minute.¹⁹ The cardioversion protocol was terminated by either technical success (defined in our case as sinus rhythm confirmed by a 12-lead ECG and maintained for at least 20 minutes after successful cardioversion) or the delivery of a sequence of five shocks.

Study Endpoints

1. The primary endpoint of the study was the comparative success rates for cardioversion between the two groups.

Figure 1. External DCCV algorithm showing sequence of up to five shocks with only the first shock being different between the two groups 360 J versus 200 J.

- The secondary endpoints were the comparative total energy level utilized between the two groups, the comparative number of shocks delivered, and the comparative AST, CK, and cTnI levels between the two groups at 18–20 hours postcardioversion.

Assay Selection

Serum cTnI, CK, and AST were measured using the ADVIA Centaur cTnI assay by Bayer. This is a two-site sandwich assay using direct chemiluminometric technology, which uses constant amounts of polyclonal and monoclonal antibodies. The assay temperature was 37°C. The reference ranges were as follows: cTnI < 0.15 µg/L, CK male 38–220 IU/L,

female 32–165 IU/L, and AST 12–40 IU/L, K⁺ (3.5–5.0 mmol/L), and TSH (0.35–5.5 mU/L).

Statistical Analysis

Sample size calculation: assuming a 70% success for lower energy shock cardioversion and 95% rate for higher energy shock, a sample size of 53 for each group was needed for a power of 90% and a level of significance of 0.05. All data are expressed as mean ± standard deviation (SD) for continuous variables and as frequencies for categorical variables. Continuous variables were tested by the Mann-Whitney U statistic. The Fischer's exact test was used to determine the two-tailed statistical significance of categorical variables in 2 × 2 tables. A P value of < 0.05 was considered to be statistically significant.

RESULTS

Characteristics of Patients

In total, 107 consecutive patients were enrolled in the study with 50 patients randomized to an initial shock of 360 J and 57 patients to an initial 200 J shock. Patient demographics are shown in Table 1. No clinically relevant differences with respect to age and sex of the patients were seen between the two groups. The etiology of AF was divided into the following groups (see Table 1): unknown, hypertension, valvular heart disease, and ischemic heart disease (IHD). In some cases there was more than one cause of AF and this was included in the two groups. There also was no statistically significant difference in left atrial (LA), left ventricular end-systolic (LVESD) and end-diastolic (LVEDD) dimensions. Patient groups also were well balanced with respect to the use of antiarrhythmics (some patients were on more than one antiarrhythmic) and the length of time that they were in AF. There was no difference in the etiology of AF between the two groups. Furthermore, serum potassium and thyroxine levels preprocedure were similar in the two groups.

Procedural Outcome

This is summarized in Table 2. The success rate for DCCV was significantly higher in the 360 J group compared to the 200 J group (48/50 = 96.0% vs 43/57 = 75.4%, P = 0.003). The mean postprocedural CK IU/L (1137.5 vs 2411.8, P = 0.019) and

Table 1. Patient Demographics

Characteristic	360 J Group (n = 50)	200 J Group (n = 57)	P Value	Significance
Male, n (%)	37 (74%)	45 (79%)	0.65	ns
Female, n (%)	13 (26%)	12 (21%)	0.65	ns
Age (years)	64.4 ± 10.5	67.7 ± 9.6	0.07	ns
Age range (years)	(35–80)	(30–79)		
Etiology				
Unknown, n (%)	15 (30%)	19 (33%)	1.0	ns
Hypertension, n (%)	20 (40%)	28 (49%)	0.58	ns
Valvular, n (%)	19 (38%)	23 (40%)	0.25	ns
IHD, n (%)	12 (24%)	14 (25%)	0.37	ns
AF duration				
<3/12, n (%)	6 (12%)	2 (4%)	0.14	ns
3–6/12, n (%)	8 (16%)	14 (24%)	0.34	ns
6–12/12, n (%)	9 (18%)	13 (23%)	0.64	ns
>12/12, n (%)	27 (54%)	28 (49%)	0.31	ns
LA Dimensions (cm)	4.15 ± 0.6	4.11 ± 0.6	0.50	ns
LVEDD (cm)	4.9 ± 0.7	5.0 ± 0.8	0.73	ns
LVESD (cm)	3.6 ± 0.8	3.7 ± 0.8	0.39	ns
K ⁺	4.38 ± 0.6	4.49 ± 0.5	0.40	ns
TSH	2.11 ± 0.3	2.44 ± 0.3	0.43	ns
Total no of patients on antiarrhythmics (%)	40/50 (80%)	46/57 (81%)	1.0	ns
Digoxin, n (%)	13 (26)	12 (21)	0.64	ns
Amiodarone, n (%)	13 (26)	17 (30)	0.70	ns
Beta-Blocker, n (%)	19 (38)	23 (40)	0.84	ns
Verapamil, n (%)	0	3 (5)	0.25	ns
Flecainide, n (%)	0	1 (2)	1.0	ns

Values expressed as mean ± SD. P < 0.05 considered significant. IHD = ischemic heart disease, LA = left atrial, LVEDD = left ventricle end-diastolic dimension, LVESD = left ventricle end-systolic dimension.

AST levels IU/L (39.83 vs 52.86, P = 0.010) levels were significantly lower in the 360 J group compared to the 200 J group. There was no statistical rise in cTnI in either group (P = 1.00). The average number of shocks delivered (1.84 vs 2.56, P = 0.006) was significantly less in the 360 J group than in the 200 J group. The median number of shocks utilized was 1.0 versus 2.0 (P = 0.006). There was no difference in total energy requirements for DCCV for the two groups (662.4 J vs 762.4 J, P = 0.67). The cumulative success rates for

the two groups for each shock sequence are graphically illustrated in Figure 2.

DISCUSSION

Efficacy

This randomized trial demonstrates that for patients in persistent AF, the use of a higher initial energy shock of 360 J versus the traditional starting energy level of 200 J at DC cardioversion achieves

Table 2. Results of DCCV Study

Characteristic	360 J Group	200 J Group	P Value
Success rates, n (%)	48/50 (96%)	43/57 (75%)	0.003
Total number of shocks	1.84 ± 1.25	2.56 ± 1.41	0.006
Mean total energy used (J)	662.4 ± 450.4	762.4 ± 509.2	0.67
Mean peak AST level IU/L	39.83 ± 24.6	52.9 ± 31.8	0.010
Mean peak CK level IU/L	1137.5 ± 2016.4	2411.8 ± 2619.7	0.014

Values expressed as mean ± SD. P < 0.05 considered significant. AST = aspartate transaminase, CK = creatine kinase.

Figure 2. Cumulative success rates for each shock in the 360 J versus 200 J group.

a significantly greater success rate. Our procedural success rate for the 200 J group was in keeping with previous reports that quote an average success rate varying from 70 to 90%.^{2,20–22} The high success rates for cardioversion in the 360 J group of 96% was impressive and higher than most published series,^{2,20–22} especially considering the significant average length of time that our patients were in AF. This study provides further support to data from other studies^{17,23} to suggest that the starting energy level for routine external DC cardioversion for persistent AF should be increased to 360 J. It can be seen from Figure 2. These data suggest that a high success rate of cardioversion can be achieved with monophasic DC cardioversion.

Safety and Adverse Effects

Excessive energy delivery with cardioversion can induce myocardial injury.²⁴ Most of the published data were obtained from open-chest animals and shocks were delivered in sinus rhythm. It has been postulated that the mechanism of such damage is mediated through the generation of free radicals, which are toxic to the myocardium.²⁵ In our study there was evidence of less skeletal muscle damage in the 360 J group as evidenced by a lower rise in the comparative AST and CK levels in this group compared with the 200 J group. There also was no evidence of cardiac muscle damage in either the higher energy 360 J group or the 200 J group, as supported by an absence of cTnI rise in either group studied. Mild first-degree skin burns are a complication of external cardioversion that can be more severe at higher peak energies. The cumulative shock energies, which were lower in the 360 J group in this study, may also affect the severity of skin burn.²⁶ There was no evidence of noticeable increased burn frequency in either group. The procedural side-effect rate in this study was low. In

the 200 J group one patient went into idioventricular VT that spontaneously terminated back to AF.

Supportive Data

In a recent small study, 64 patients were randomly assigned to an initial monophasic waveform energy of 100, 200, or 360 J.¹⁷ A higher initial energy shock was significantly more effective than lower levels (immediate success rates were 14% with 100, 39% with 200, and 95% with 360 J, respectively), resulting in fewer shocks and less cumulative energy when 360 J was delivered initially. In a recent study²³ that analyzed retrospectively the efficacy of 5,152 shocks delivered to patients with AF, the probability of success on the first shock in AF of more than 30 days duration was 5.5% at <200 J, 35% at 200 J, and 56% at 360 J. In patients with AF of more than 180 days duration, the initial use of a 360 J shock was associated with the eventual use of less electrical energy than with an initial shock of 100 J or less. These data are further supported by our study that suggests that the procedural time for cardioversion might be reduced by starting at a higher starting energy of 360 J. In our study in the 360 J group 29 patients (58%) experienced sinus rhythm with the first shock versus 17 (30%) in the 200 J group.

The safety and efficacy of higher energy monophasic cardioversion are further supported by two other small studies. In one study, Saliba et al.¹⁶ published data on 55 patients with persistent AF who had failed in at least two attempts at routine cardioversion. They showed that patients could be safely, and in 84% of cases effectively cardioverted to sinus rhythm with an initial starting monophasic energy of 720 J. In another study, Bjerregaard et al.¹⁸ also showed that double external cardioversion delivering 720 J restored sinus rhythm safely in 67% of patients in whom conventional cardioversion failed with one external shock sequence.

Limitations

This study has several limitations. Patient weights prior to cardioversion were not routinely measured although it is known to affect procedural outcome in cardioversion.¹⁵ Furthermore, this was a single blind study where the paddle operator knew what energy level setting was chosen for the cardioversion. Detailed patient symptom scores and detailed skin burn assessments were not

performed. Finally, biphasic external defibrillation rather than monophasic defibrillation is likely to become the norm in the future.

CONCLUSIONS

For patients with persistent AF the use of a higher initial energy monophasic shock of 360 J achieves a significantly greater success rate, with less skeletal muscle damage (with no cardiac muscle damage) as compared with the traditional starting energy of a 200 J DC shock. A starting monophasic energy shock of 360 J is safe and effective for cardioversion of AF and should be used as the routine.

Acknowledgments: *The authors thank Kay Hughes and Joy Baker for their assistance with patient administration. We gratefully thank Roger Hoke and the nurses of our cardiac unit for their technical support.*

REFERENCES

1. Fuster V, Ryden LE, Asinger R, et al. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to develop guidelines for the management of patients with atrial fibrillation) developed in collaboration with the North American Society of Pacing and Electrophysiology. *Eur Heart J* 2001;20:1852-1923.
2. Prystowsky EN, Benson DW Jr, Fuster V, et al. Management of patients with atrial fibrillation. A statement for healthcare professionals. From the Subcommittee on Electrocardiography and Electrophysiology. American Heart Association. *Circulation* 1996;93:262-1277.
3. Prystowsky EN, Katz A. Atrial fibrillation. In: Topol EJ (ed.): *Textbook of Cardiovascular Medicine*. Philadelphia, Lippincott-Raven Publishers, 1998, pp. 1661-1693.
4. Donahue TP, Conti JB. Atrial fibrillation: Rate control versus maintenance of sinus rhythm. *Curr Opin Cardiol* 2001;16:46-53.
5. Wolf PA, Dawber TR, Thomas HE Jr, et al. Epidemiological assessment of chronic atrial fibrillation and the risk of stroke. The Framingham study. *Neurology* 1978;28:973-977.
6. Kannel WB, Abbott RD, Savage DD, et al. Epidemiological features of chronic atrial fibrillation and the risk of stroke. The Framingham Study. *N Engl J Med* 1982;306:1018-1022.
7. Lown B, Amarasingham R, Neuman J. New method for terminating cardiac arrhythmias: Use of synchronized capacitor discharge. *JAMA* 1962;182:548-555.
8. Schmitt C, Alt E, Plewan A, et al. Low energy intracardiac cardioversion after failed conventional external cardioversion of atrial fibrillation. *J Am Coll Cardiol* 1996;28:994-999.
9. Cochrane DJ, McEneaney DJ, Anderson JM, et al. Transthoracic versus transchest DC cardioversion. *Q J Med* 1993;86:507-511.
10. Mittal S, Ayati S, Stein KM, et al. Transthoracic cardioversion of atrial fibrillation: Comparison of rectilinear biphasic versus damped sine wave monophasic shocks. *Circulation* 2000;101:1282-1287.
11. Metcalfe MJ, Smith F, Jennings K, et al. Does cardioversion for atrial fibrillation result in myocardial damage. *Br Med J* 1988;296:1364.
12. Jakobsson J, Odmansson I, Nordlander R. Enzyme release after elective cardioversion. *Eur Heart J* 1990;11:749-752.
13. Greaves K, Crake T. Cardiac troponin T does not increase after electrical cardioversion for atrial fibrillation or atrial flutter. *Heart* 1998;80:226-228.
14. Rao AC, Naeem N, John C, et al. Direct current cardioversion does not cause cardiac damage: Evidence from cardiac troponin T estimation. *Heart* 1998;80:229-230.
15. Bonnefoy E, Chevalier P, Kirkorian G, et al. Cardiac troponin I does not increase after cardioversion. *Chest* 1997;111:15-18.
16. Saliba W, Juratli N, Chung MK. Higher energy synchronized external direct current cardioversion for refractory atrial fibrillation. *J Am Coll Cardiol* 1999;34(7):2031-2034.
17. Joglar JA, Hamdan MH, Ramaswamy K, et al. Initial energy for elective external cardioversion of persistent atrial fibrillation. *Am J Cardiol*. 2000;86:348-350.
18. Bjerregaard P, El-Shafei A, Janosik DL, et al. Double external shocks for refractory atrial fibrillation. *Am J Cardiol*. 1999;83:972-974.
19. Dahl CF, Ewy GA, Warner ED, et al. Myocardial necrosis from direct current countershock: Effect of paddle electrode size and time interval between discharges. *Circulation* 1974;50:956-961.
20. Van Gelder IC, Crijns HJ, Van Gilst WH, et al. Prediction of uneventful cardioversion and maintenance of sinus rhythm from direct-current electrical cardioversion of chronic atrial fibrillation and flutter. *Am J Cardiol* 1991;68:41-46.
21. Sodermark T, Jonsson B, Olsson A, et al. Effect of quinidine on maintaining sinus rhythm after cardioversion of atrial fibrillation or flutter: A multicenter study from Stockholm. *Br Heart J* 1975;37:486-492.
22. Lundstrom T, Ryden L. Chronic atrial fibrillation: Long-term results of direct current cardioversion. *Acta Med Scand* 1988;223:53-59.
23. Gallagher MM, Guo XH, Poloniecki JD, et al. Initial energy setting, outcome and efficiency in direct current cardioversion of atrial fibrillation and flutter. *J Am Coll Cardiol* 2001;38(5):1498-1504.
24. Van Vleet JF, Tacker WA. Cardiac Damage from transchest and ICD defibrillator shocks. In Tacker WA (ed.): *Defibrillation of the Heart*. St. Louis (MO), Mosby, 1994, pp. 259-298.
25. Trouton PG, Allen JD, Young LK, et al. Metabolic changes and mitochondrial dysfunction early following transthoracic countershocks in dogs. *PACE* 1989;12:1827-1834.
26. Dalzell GW, Anderson J, Adgey AA. Factors determining success and energy requirements for atrial fibrillation. *Q J Med* 1990;76:903-913.