

Efficacy and Tolerability of Transvenous Low Energy Cardioversion of Paroxysmal Atrial Fibrillation in Humans

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Objectives. This study investigated the efficacy and tolerability of low energy shocks for termination of atrial fibrillation in patients, using an endocardial electrode configuration that embraced both atria.

Background. In animals, low energy biphasic shocks delivered between electrodes in the coronary sinus and right atrium have effectively terminated atrial fibrillation. If human defibrillation thresholds are sufficiently low, atrial defibrillation could be achieved in conscious patients using an implanted device.

Methods. Twenty-two consecutive patients with stable atrial fibrillation were studied during electrophysiologic testing. Biphasic R wave synchronous shocks were delivered between large surface area electrodes in the coronary sinus and high right atrium, using a step-up voltage protocol starting at 10 or 20 V and increasing to a maximum of 400 V. Patients were conscious at the

start of the study and were asked to report on symptoms but were sedated later if shocks were not tolerated.

Results. Cardioversion was achieved in all 19 patients who completed the study, with a mean (\pm SD) leading-edge voltage of 237 ± 55 V (range 140 to 340) and mean energy of 2.16 ± 1.02 J (range 0.7 to 4.4). The mean maximal shock delivered without sedation was 116 ± 51 V (range 60 to 180). No proarrhythmia or mechanical complications occurred.

Conclusions. The delivery of biphasic R wave synchronous shocks between the high right atrium and coronary sinus can terminate atrial fibrillation with very low energies. General anaesthesia is not required, and a minority of fully conscious patients are able to tolerate this method of cardioversion.

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Of the cardiac arrhythmias, atrial fibrillation is by far the most common and is responsible for the most hospital admissions and days in hospital (1). Although tolerated by many patients, atrial fibrillation can cause disabling symptoms and is perhaps the most important treatable cause of stroke (2,3). Both functional capacity and left ventricular function improve after the restoration and maintenance of sinus rhythm, even in patients with a previously controlled ventricular rate (4-6). Antiarrhythmic drugs are of limited efficacy (7-10), frequently cause adverse reactions and side effects and carry the risk of both atrial and ventricular proarrhythmia (8,11-13). Nonpharmacologic approaches to atrial fibrillation are, therefore, gaining attention. His bundle ablation and the corridor operation (14) do not restore atrial function or affect the thromboembolic risk, and the maze procedure (15) is a major surgical undertaking associated with significant morbidity.

An implantable atrial defibrillator could have significant

advantages over these approaches for patients with sustained symptomatic episodes of paroxysmal atrial fibrillation, but the shocks delivered would need to be of sufficiently low energy to be tolerated in full consciousness. A technique to achieve this may also have applications in the acute care of repeated atrial fibrillation episodes. Several strategies have been investigated (16-21) in experimental atrial fibrillation to deliver shocks closer to the atria and reduce the defibrillation threshold. The most efficient use biphasic shocks and include both atria in the electric field while limiting energy delivery to other tissues. The shock strength required for endocardial atrial defibrillation using this method in patients and the tolerability of such shocks are unknown.

We, therefore, investigated the clinical efficacy of biphasic low energy shocks delivered between transvenous catheters in the right atrium and coronary sinus for the cardioversion of sustained but not chronic atrial fibrillation and the degree of discomfort caused by such shocks.

Methods

Patients. The protocol was carried out in patients undergoing invasive electrophysiologic studies. Patients were considered eligible if sustained atrial fibrillation of <3 days in duration was present at the commencement of the electrophysiologic study or if atrial fibrillation was induced during the course of the study and lasted for at least 15 min. All

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Figure 1. Posteroanterior chest radiograph demonstrating defibrillation electrode configuration. CS = electrode in coronary sinus; RA = right atrial electrode; RV = right ventricular pace/sense electrode.

antiarrhythmic drugs (including digitalis) were discontinued for at least 5 half-lives before the electrophysiologic study. Patients were excluded if they had a history of stroke or thromboembolism. Our usual procedure was followed with regard to anticoagulation. Patients who develop atrial fibrillation during the electrophysiologic study do not undergo anticoagulation, but those who develop sustained atrial fibrillation beforehand (usually on discontinuation of antiarrhythmic therapy) undergo temporary anticoagulation. Anticoagulation is then discontinued before insertion of transvenous sheaths. The study protocol was approved by the District Medical Ethics Committee, and all patients gave written informed consent before the commencement of the study.

Protocol. Two identical custom-made 6F catheters (Electro-Catheter Inc.) were used to deliver defibrillation shocks. Each

had nine 5-mm platinum segments separated by 2-mm flexible plastic spacers. These were connected so as to constitute a single electrode length of 6 cm and total surface area 2.83 cm². The catheters were positioned under fluoroscopic guidance, and their positions were confirmed radiographically (Fig. 1). The left atrial catheter was introduced through a sheath in the left subclavian or right internal jugular vein, advanced as far as it would easily pass into the coronary sinus and then withdrawn ~1 cm so that its tip would not exert pressure on the vessel wall. The right atrial catheter was introduced through a sheath in the right femoral vein and advanced into the high right atrium, with the tip in the appendage and, where possible, with the electrode section against the anterolateral atrial wall. In the first 15 patients, a pacing catheter, advanced from the right femoral vein and positioned in the right ventricular apex, was used to provide R wave synchronization and deliver backup ventricular demand pacing at a rate of 40 beats/min. In later patients, a ventricular lead was not used, and the surface electrocardiogram (ECG) was used for R wave synchronization.

The defibrillation catheters were connected (right atrium negative, left atrium positive) to a custom-made external atrial defibrillator (XAD, InControl Inc.). This delivers a truncated exponential shock with the two phases each of 3-ms duration, separated by 240 μ s, and with a leading-edge voltage programmable between 10 and 400 V. Shocks are synchronized to a trigger signal, with a delay of 20 ms. For safety reasons, the device was programmed to deliver shocks only after RR intervals between 500 and 1,000 ms (see Discussion). The external atrial defibrillator calibrates itself on power-up against an internal reference capacitor and, after each shock, gives a readout of the actual leading-edge voltage, delivered energy and impedance.

After a test shock of 10 or 20 V to verify correct R wave synchronization, a sequence of shocks was given, increasing from 20 V in 40-V steps, until atrial fibrillation was terminated or a 400-V shock was delivered. Shocks were separated by at least 1 min. The surface ECG was recorded continuously onto paper at a chart speed of 25 mm/s. Shocks were classified as follows (Fig. 2): *failure* = continuation of atrial fibrillation;

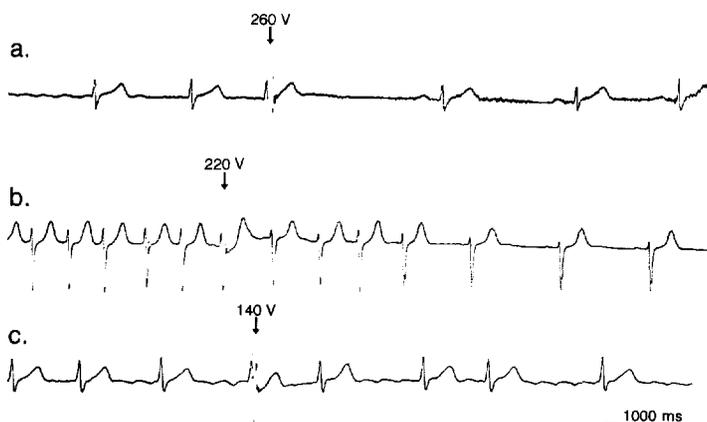


Figure 2. Electrocardiographic tracings of delivered shocks: immediate (a) and delayed (b) cessation of atrial fibrillation after shock delivery and (c) unsuccessful shock.

immediate success = no evidence of atrial fibrillation after shock delivery; *delayed success* = continued atrial fibrillation after shock delivery, terminating within seconds. In this case, the time from the shock to the first P wave was noted.

In some patients, intravenous premedication (2.5 mg of diamorphine or 2.5 mg of midazolam) was given before insertion of transvenous sheaths for the electrophysiologic study. This was always at least 2 h before the commencement of the shock protocol. To evaluate the tolerability of the defibrillation shocks, no further sedation was given before the first shock, so that all patients were fully conscious at the outset of the study and able to report any symptoms experienced, according to the following scale: 1 = shock not felt; 2 = shock felt, no discomfort; 3 = mild discomfort; 4 = moderate discomfort; 5 = severe discomfort. After each shock, patients were given the option of requesting sedation (intravenous midazolam) before the study continued. The last shock to be delivered before any such request, and all shocks given after sedation (however light), were scored as 5.

Continuous variables were compared using the Student *t* test, and the Fisher exact test was used for discrete variables; *p* < 0.05 was considered statistically significant.

Results

Patients. Twenty-two patients entered the study and were classified into two groups. Group 1 included 18 patients in whom atrial fibrillation was induced by catheter manipulation or electrical stimulation during electrophysiologic study (mean [\pm SD] duration of atrial fibrillation 35 ± 19 min, range 15 to 75). Thirteen of these patients also had a history of documented spontaneous atrial fibrillation. Group 2 included four patients with spontaneous atrial fibrillation at the beginning of the electrophysiologic study (duration of atrial fibrillation 35 ± 25 h, range 18 to 72). The clinical characteristics of the two groups are shown in Table 1.

Outcomes. A total of 144 shocks were given (6.5 ± 1.7 shocks/patient). The protocol was not completed in three patients. In one patient, the procedure was abandoned after shocks of up to 340 V failed to restore sinus rhythm. This was one of the earliest patients to be studied, and the protocol was subsequently amended to allow shocks of up to 400 V to be delivered. In the second patient, atrial fibrillation converted to atrial flutter >1 min after the delivery of the 100-V shock; the flutter was terminated by overdrive atrial pacing. In view of the long interval between shock delivery and cessation of atrial fibrillation, this was considered to be a spontaneous change in rhythm rather than a success. The third patient regained sinus rhythm ~1 min after the delivery of the 300-V shock and immediately before the 340-V shock. Again, this was considered to have been a spontaneous cessation of arrhythmia.

The remaining 19 patients completed the protocol, and atrial fibrillation was terminated in every patient. Sinus rhythm resulted in 18 patients, with 13 immediate successes. In five patients, there was continued ECG evidence of atrial fibrillation immediately after shock delivery, and the time to the first

Table 1. Clinical Characteristics of 22 Study Patients

	Group 1: Induced AF (n = 18)	Group 2: Spontaneous AF (n = 4)	p Value
Men/women	14/4	3/1	
Age (yr)			
Mean \pm SD	52 \pm 11	64 \pm 7	0.037
Range	33-77	56-71	
LA diameter (mm)			
Mean \pm SD	3.7 \pm 0.7	4.7 \pm 0.7	0.013
Range	2.5-4.8	4.2-5.6	
AF duration (h)			
Mean \pm SD	0.58 \pm 0.32	35 \pm 25	0.0003
Range	0.25-1.25	18-72	
Documented clinical AF	13 (72%)	4 (100%)	0.54
Structural heart disease			1.00
IHD	1	1	
ASD	1	0	
DCM	1	0	
Other arrhythmias			0.11
AT	1	0	
SND	1	0	
AFL	1	0	
AVNRT	2	0	
AVRT	3	0	
VT	1	0	
"Lone" AF	9 (50%)	3 (75%)	0.59

Data presented are mean value \pm SD or number (%) of patients. AF = atrial fibrillation; AFL = atrial flutter; ASD = repaired atrial septal defect; AT = (automatic) atrial tachycardia; AVNRT = atrioventricular node reentrant tachycardia; AVRT = atrioventricular reentrant tachycardia; DCM = dilated cardiomyopathy; IHD = ischemic heart disease; LA = left atrial; "Lone" AF = no structural cardiac disease or arrhythmia other than atrial fibrillation; SND = sinus node disease; VT = ventricular tachycardia.

P wave was $2,500 \pm 1,400$ ms (range 1,600 to 4,900). Because atrial fibrillation had been present for between 15 and 60 min, this was unlikely to be a chance occurrence, and these cases were considered to be delayed successes. In the remaining patients, atrioventricular (AV) node reentrant tachycardia resumed immediately after the shock—this arrhythmia was virtually incessant in this patient and was the indication for the electrophysiologic study.

Electrical variables for defibrillation. The defibrillation impedance was calculated for each patient as the mean of the impedances measured at each shock—there was very little variation between shocks (mean of standard deviations for each patient = 2.9 ohms). The impedance did not differ significantly between patients with induced and spontaneous atrial fibrillation (56.9 ± 7.5 and 56.3 ± 7.6 ohms, respectively, *p* = 0.89), nor between patients with immediate and delayed success in cardioversion (57.7 ± 8.0 and 58.3 ± 2.7 ohms, respectively, *p* = 0.87). The mean leading-edge voltage of successful shocks was 237 ± 55 V (range 140 to 340), and their mean delivered energy was 2.16 ± 1.02 J (range 0.7 to 4.4). There was no significant difference between induced and spontaneous atrial fibrillation in the leading-edge voltages (230 ± 55 and 260 ± 57 V, respectively, *p* = 0.36, 95%

confidence limits [CL] for difference $-95, +37$) or energies (2.05 ± 0.97 and 2.55 ± 1.28 J, respectively, $p = 0.40$, CL $-1.72, +0.73$) at which defibrillation was achieved (Fig. 3). These measures were lower in shocks achieving delayed success than in those achieving immediate success, but the differences were of borderline significance (196 ± 54 vs. 251 ± 50 V, $p = 0.05$, CL $-1, +111$; 1.50 ± 0.78 vs. 2.39 ± 1.02 J, $p = 0.09$, CL $-0.17, 1.96$, respectively) (Fig. 4).

Safety and tolerability. No adverse events, including ventricular arrhythmias, occurred during any of the procedures. Immediately after the larger shocks, there was some evidence of sinus and AV node slowing. After successful shocks, the longest sinus pause was 2.52 s, and the slowest heart rate was 43.1 beats/min (measured over the first 5 beats). After unsuccessful shocks, the longest RR interval was 1.32 s, and the lowest heart rate was 50.3 beats/min. Paced beats were delivered in 2 of the 15 patients with temporary ventricular pacing. In both patients, this was due to a sinus pause immediately after successful shocks. A single paced beat was seen in one patient and two paced beats in the other. No shock caused sustained bradycardia.

The symptoms caused by shocks are shown in Figure 5. All but three patients were able to sense the smallest shock delivered (10 or 20 V), and the degree of discomfort always increased with shock strength. The largest shock delivered in full consciousness was 116 ± 51 V and varied considerably between 60 V (0.1 J) in seven patients and 180 V (1.1 to 1.2 J) in six. In 17 patients, this was less than the voltage required for cardioversion, so that some degree of sedation was required. In two patients, no sedation was given. In one of these patients, the successful shock was the first to cause severe discomfort, and in the other, it caused only moderate discomfort. In the remaining three patients, the defibrillation threshold was not established because of discontinuation of the protocol. No patient reported any residual discomfort after the procedure, and there was no echocardiographic evidence of cardiac trauma after the procedure.

Figure 3. Leading-edge voltages and delivered energies in patients with induced and spontaneous (spont.) atrial fibrillation. **Vertical bars and circles** = mean value \pm SD.

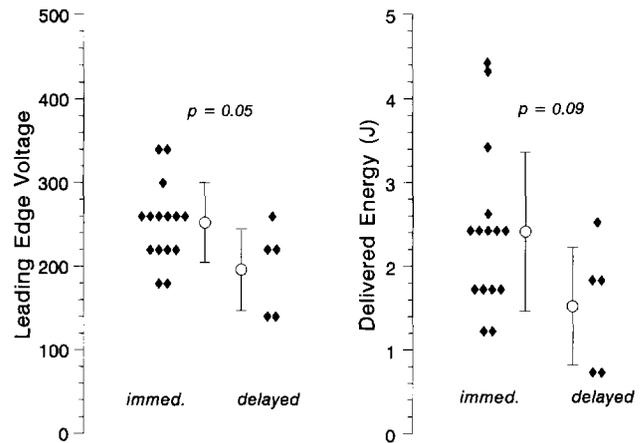
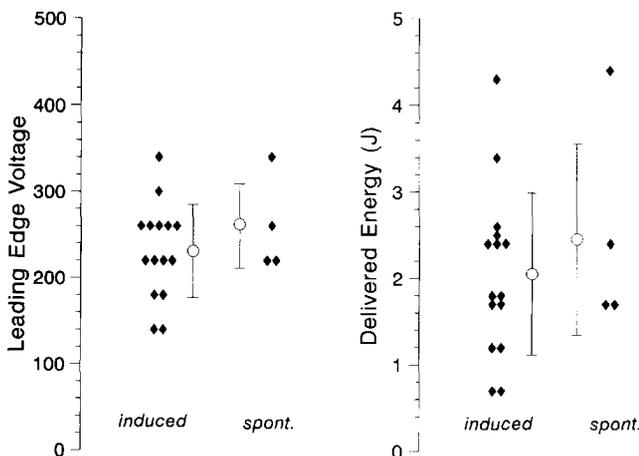


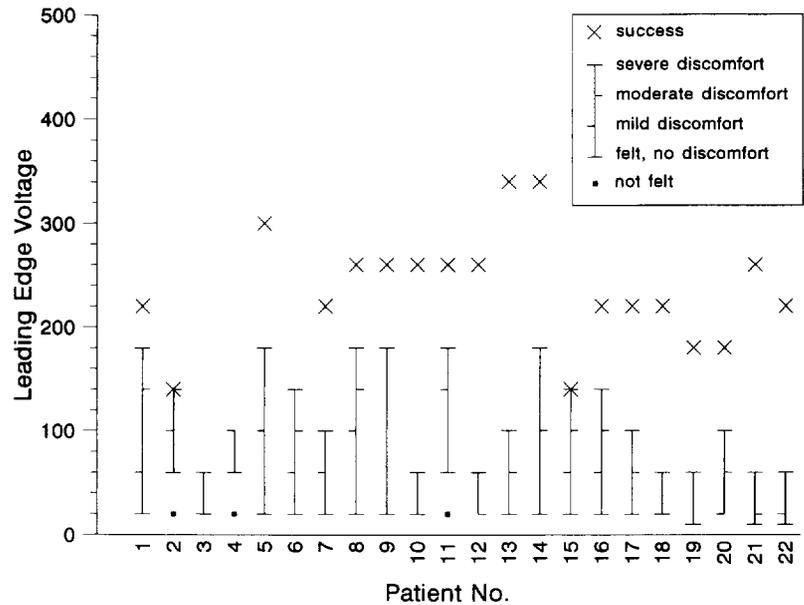
Figure 4. Leading-edge voltages and delivered energies in patients in whom immediate (immed.) and delayed success was observed for atrial defibrillation. **Vertical bars and circles** = mean value \pm SD.

Discussion

Background. Electrical cardioversion by transthoracic capacitor discharge has been a standard treatment for atrial fibrillation since its description by Lown et al. (22). However, general anesthesia is an almost universal prerequisite because energies of >100 J are generally needed, even with adjunctive drug therapy (23). Less energy is required if the electric field is closer to, or within, the atria. In dogs, defibrillation thresholds of <1.5 J were obtained by placing a catheter in the esophagus (16), but transesophageal cardioversion in humans requires up to 100 to 200 J to achieve 80% success (24). Cardioversion has also been achieved in a canine model using endocavitary shocks with energies of <5 J (17,18). In sheep (whose heart size is similar to that of humans), 80% success was achieved with 2.5-J shocks delivered between the right atrium and a skin patch (19). A systematic comparison of several electrode configurations and shock waveforms was performed in a sheep model of paroxysmal atrial fibrillation by Cooper et al. (20). The lowest thresholds were obtained using configurations embracing both atria. Biphasic shocks were delivered between the right atrial appendage and the coronary sinus, and cardioversion was achieved with thresholds of 1.3 ± 0.4 J. In patients, right atrial unipolar shocks can be used to cardiovert chronic atrial fibrillation that is resistant to transthoracic shocks, but energies of 200 to 300 J are needed (25), and early attempts at low energy cardioversion in humans were uniformly unsuccessful (26). However, Keane et al. (21) recently reported atrial defibrillation thresholds of <1 J using biphasic shocks delivered between paddle electrodes on the left and right atrial epicardial surfaces in patients with atrial fibrillation induced artificially during cardiopulmonary bypass.

Efficacy. In the present study, biphasic shocks were delivered between defibrillation electrodes in the coronary sinus and high right atrium of patients with paroxysmal atrial fibrillation lasting between 15 min and 3 days. The electrodes used were of simple design and low surface area compared

Figure 5. Tolerability of shocks. For each patient, the lowest leading-edge voltage responsible for each level of discomfort is shown, along with the voltage at which cardioversion was obtained. Atrial fibrillation in Patient 3 was not cardioverted by shocks up to 340 V, and Patients 4 and 6 did not complete the study because of spontaneous return of sinus rhythm (see text for details).



with coil electrodes currently used in implantable cardioverter-defibrillator systems. Nevertheless, termination of atrial fibrillation was achieved using energies generally between 1 and 3 J—two orders of magnitude lower than those required with a transthoracic approach. Cardioversion failed in only one patient with shocks of up to 340 V. The protocol was subsequently amended to allow a maximal output of 400 V, and although this voltage has not been required since, it is not known whether it may have been effective in this patient.

Most of the patients in whom the arrhythmia was artificially induced on the occasion of this study had a history of documented, spontaneously occurring atrial fibrillation. Furthermore, the patients investigated during spontaneous atrial fibrillation did not require significantly higher voltage shocks than those with induced atrial fibrillation, despite their greater age and left atrial diameter and longer duration of their arrhythmia. Although the numbers studied were small, this would indicate that induced atrial fibrillation in susceptible patients provides an acceptable model of the spontaneous arrhythmia for the purpose of further research into internal cardioversion.

In five patients, a delay (up to 4.9 s) was observed between shock delivery and cessation of atrial fibrillation. A long period of stable atrial fibrillation was required before the delivery of the first shock, and coincidental spontaneous reversion to sinus rhythm can almost certainly be excluded as an explanation of the observation. A similar delay in response is frequently observed when ventricular fibrillation is terminated by implantable devices. With external direct current shocks, the occurrence may not be recognized because the ECG signal tends to be saturated immediately after shock delivery. The likeliest explanation for the phenomenon is that the electric current is not quite sufficient to defibrillate a critical mass of atrial tissue, so that the fibrillating wavelets are reduced in number but not

abolished. Depending on the local conduction properties and refractory state of the atrial myocardium that they encounter, the surviving wavelets may either reinitiate stable atrial fibrillation or be extinguished. This explanation is supported by the observation that in the present study, shocks leading to delayed cardioversion tended to be of lower voltage than those achieving immediate success. An alternative explanation is that in these cases cardioversion is not a purely electrical phenomenon but is instead mediated by local neuroendocrine mechanisms.

Safety. Mechanical complications of high energy endocavitary shock delivery (especially in the region of the coronary sinus) are generally related to barotrauma. This occurs when the current density is sufficient to give rise to arcing and the formation of a gas bubble, and can cause coronary sinus rupture and cardiac tamponade (27). Barotrauma does not occur with transvenous implantable cardioverter-defibrillator systems, which use lower energies and large surface area electrodes; and the use of coronary sinus defibrillation leads in such systems has been found to be safe (28). In the present study, high surface area electrodes and shocks of even lower energy were used. No mechanical complications occurred, and although the number of patients was limited, the risk of cardiac trauma using this technique appears to be minimal. The other significant potential risk with low energy atrial defibrillation is that of inducing malignant ventricular arrhythmias. The electric field generated between the right atrial and coronary sinus electrodes is largely confined to the atria, and this is probably the reason for the success of this configuration. However, the base of the ventricles is inevitably included in the field to an extent, and shocks could, therefore, be proarrhythmic if delivered during the ventricular relative refractory period. Such a risk is highest with unsynchronized shocks but theoretically possible at rapid ventricular rates even with QRS synchroni-

zation because of inhomogeneity of ventricular repolarization. The risk of ventricular proarrhythmia from synchronized low energy biatrial shocks was recently investigated in sheep by Ayers et al. (29). Over a range of paced and spontaneous cycle lengths and energies, the overall incidence of ventricular fibrillation was 0.6% and was confined entirely to shocks delivered after RR intervals of <300 ms. In the present study, all shocks were synchronized to the surface or endocardial QRS complex and were delivered after a ventricular cycle length of at least 500 ms. Such an interval always occurred within seconds of the decision to shock. In contrast to ventricular defibrillation, a delay in the cardioversion of atrial fibrillation would not be harmful. However, there may be a long delay before a suitable RR interval occurs in patients with a sustained rapid ventricular rate, and a requirement for a minimal RR interval before a shock may limit the extension of the technique to patients with atrial flutter.

Tolerability. The determinants of discomfort caused by intracardiac electric discharges are poorly understood. Early experiments with low energy intracardiac shocks failed to find a correlation between energy delivered and discomfort (26). Conversely, in the present study, the degree of discomfort increased steadily with shock strength in all patients. However, the inpatient reproducibility of symptoms was not formally assessed. Although the maximal voltage delivered without sedation varied greatly between patients, it lay in most cases between 100 and 180 V, equivalent to ~0.5 to 1.0 J. As shown in Figure 5, the shock strengths required for cardioversion were not usually sufficiently low to be tolerated without sedation but were of the same order of magnitude. The leading-edge voltage of shocks is unlikely to be the only determinant of symptoms. Pain from shocks may result from stimulation of nerve fibers or direct excitation of skeletal muscle, and it is possible that lower defibrillation thresholds or greater tolerability, or both, may be achieved by fine modifications to shock waveforms and the design and locations of defibrillation electrodes (e.g., to avoid stimulating the phrenic nerve and fibers passing near the coronary sinus ostium). Posture, psychologic conditioning and circumstance may also affect symptoms and explain why some patients with implanted cardioverter-defibrillators are able to tolerate shocks of several joules in full consciousness. In the present study, sedation was requested by patients in the anticipation of an unknown number of further shocks of steadily increasing strength in an unfamiliar environment. A verbal warning was given before each shock. With an implanted atrial defibrillator, it is unclear whether the perception of shocks might be improved by familiarity and the expectation of a therapeutic benefit (termination of a prolonged symptomatic episode of atrial fibrillation) and whether a warning feature or patient control of the device would be desirable.

Applications. Low energy transvenous cardioversion has several potential applications. Atrial fibrillation frequently occurs during electrophysiologic study and catheter ablation procedures. This can be terminated repeatedly without antiarrhythmic drugs (which can influence the study) or the need for

general anesthesia. Similarly, indwelling temporary defibrillation electrodes could be used to terminate recurrent attacks of atrial fibrillation occurring in coronary care units or after cardiac surgery. Atrial fibrillation occurs frequently in patients with implanted cardioverter-defibrillators and is a common cause of hemodynamic deterioration and inappropriate device therapy (30). The ability to recognize and terminate atrial fibrillation with low energy shocks would, therefore, be a valuable asset, requiring only the addition of a coronary sinus electrode, which is already featured in some systems. Finally, patients with recurrent, sustained and symptomatic atrial fibrillation unresponsive to conventional therapy are frequently treated by AV node ablation. An implantable atrial defibrillator may be a superior treatment for these patients, preserving the benefits of sinus rhythm and avoiding the commitment to lifelong pacemaker therapy. Such devices have recently been tested in sheep (31) and, because of the low stored energy requirement, can be made sufficiently small for routine prepectoral implantation in humans. The cost of this strategy may not exceed that of AV node ablation and pacemaker implantation.

Study limitations. The present study was intended to evaluate the feasibility of transvenous low energy atrial defibrillation. Although complications, including ventricular proarrhythmia, were not observed, no firm conclusions can be drawn regarding the issue of safety because of the small numbers involved. Clearly, large-scale cooperative efforts are required to examine this critical issue. The "step-up" shock protocol was used so that patients could be conscious at the start of the study and thus be able to report symptoms. Repeated reinduction of atrial fibrillation, with shocks in a randomized order, would be necessary to determine the precise relation between delivered energy and the probability of success. It is likely that this follows a sigmoidal dose-response curve, as in animal models of atrial and ventricular fibrillation (32,33). If this curve has a steep slope, a genuine defibrillation threshold can be said to exist, and a single shock just above this level would have a very high probability of success. However, if the slope is shallow, it would be possible to achieve cardioversion using a series of small shocks, each with a low individual likelihood of success but with a cumulatively increasing probability of cardioversion. The delayed cardioversions sometimes observed at relatively low energies support this likelihood. Finally, the study size was inadequate to determine the effects of clinical variables, such as etiology, atrial diameter and drug therapy, on the defibrillation threshold.

Conclusions. Biphasic shocks delivered between the coronary sinus and high right atrium are able to terminate paroxysmal atrial fibrillation. The energies required are very low, and no complications have been observed. Although cardioversion cannot usually be achieved without any sedation, general anesthesia is not required. This technique has several potential applications, including use in implantable devices.

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