

Atrial Intracardiac Cardioversion and Ibutilide in Patients with Persistent Atrial Fibrillation Undergoing Interventional Electrophysiological Procedures

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Summary

Radiofrequency ablation of focally initiated atrial fibrillation has been described as an effective therapy in selected patients. To identify "focal firing" in these patients, atrial fibrillation must be terminated. In this article, we describe the successful termination of atrial fibrillation by means of an interventional procedure after a failed external cardioversion in two patients with persistent atrial fibrillation. During the procedure, the internal cardioversion catheter was inserted in the distal part of the coronary sinus and shocks were delivered between the distal coil located in the coronary sinus and the proximal coil located in the right atrium or the junction of the superior vena cava and the right atrium. Shock delivery at high energies caused dislocation of the mapping catheters positioned in the pulmonary veins, but they could easily be repositioned. In one patient, internal cardioversion was only successful after IV ibutilide infusion. Atrial internal cardioversion with and without IV ibutilide is a safe and efficacious tool for achieving sinus rhythm in patients during an electrophysiological intervention.

Key Words

Internal atrial cardioversion, persistent atrial fibrillation, radiofrequency ablation, focal ablation site

Introduction

Atrial fibrillation is a very common arrhythmia. The prevalence of this condition increases with age and underlying heart disease. Its occurrence is associated with a higher mortality rate [1], a higher risk of stroke [2-3], lower stroke volume, reduced cardiac output (up to 35 % decrease [4]), and diminished exercise capacity, especially in patients with heart disease [5]. Therapeutic modalities to treat episodes of atrial fibrillation include pharmacological intervention, which has a moderate success rate depending on the type and duration of the arrhythmia. The success rate varies between 40 and 70 % in patients with persistent atrial fibrillation [4,6-8]. Another method is external DC cardioversion, which has an efficacy of approximately

85 – 90 % [9,10]. Failure of external cardioversion is related to a high body mass index [11-12] or to improper positioning of the paddles on the chest. A third method, developed for patients refractory to the described conventional treatment, is internal cardioversion using percutaneous transvenous electrodes. Animal and human experiments showed rather low defibrillation thresholds (< 5 J) when one shock coil was positioned in the coronary sinus and the other shock coil was located in the high right atrium, near the junction with the superior vena cava [13-16]. The amount of atrial tissue that is encompassed between the two shock coils is critical. This treatment is restricted to patients refractory to conventional treatment, but can also be applied in patients

during an electrophysiological procedure. When atrial fibrillation persists for hours and any drug treatment influences the substrate, such as the focus or bypass to ablate, cardioversion is the only remedy and internal cardioversion is the most elegant solution. We describe two patients with persistent atrial fibrillation and unsuccessful external cardioversion. Internal cardioversion was successful for both patients during the electrophysiology intervention procedure, i.e., during radio frequency ablation of the pulmonary veins. In one patient, the cardioversion was only successful after IV ibutilide and it is recommended to repeat internal cardioversion after IV ibutilide if the first series of shocks fail to convert the heart into sinus rhythm.

Case Presentation 1

A 45-year-old man with a history of persistent atrial fibrillation was scheduled for ablation of a diagnosed atrial fibrillation. Each recurrence of atrial fibrillation had to be terminated by external electrical cardioversion. Physical examination and echocardiography evaluation revealed no underlying heart disease and no coronary artery disease. The patient's weight and height were 80.5 kg and 179 cm, respectively. The patient was on warfarin therapy until two days before the planned intervention. External cardioversion was performed before the procedure, but it failed to convert the heart into sinus rhythm. Antiarrhythmic drugs (80 mg sotalol twice a day) were discontinued 5 half-lives before the intervention, which was recently interrupted for the ablation procedure.

Three mapping catheters were positioned in the pulmonary veins via the trans-septal route; the cardiover-

sion catheter was positioned with its tip in the distal part of the coronary sinus, and the second shock coil was placed in the high right atrium. A third shock coil was positioned in the left subclavian vein, into which the catheter was inserted. The special catheter (V207, Biotronik, Germany) is constructed as a bi-directional, steerable ablation catheter (Multicath, Biotronik), with three additional conventional shock coils that have a fractal iridium coating (Figure 1).

The shocks for cardioversion are triggered by the R-wave from a bipolar signal that was derived from the coils S1 and S2 or from the coils S2 and S3 (Figure 1). As an alternative, the trigger signal can be derived from the R-wave of an adapted surface ECG lead. Both synchronization and shock delivery can be coordinated and programmed by a dedicated programmer and shock analyzer used for implantable cardioverter-defibrillator (ICD) implantation (TMS 1000, Biotronik). This device has been used during standard ICD implantations in many cases and is a regular and accepted tool of the intra-operative ICD-system tests. This device regulates charging and the timing relative to the triggered R-wave; furthermore, it measures and stores shock impedances. The shock energy can be regulated in small, controlled steps from 0.5 J up to 30 J. Emergency defibrillation can be performed at an energy level of 40 J.

During the shock there is an exponentially decreasing, biphasic current within a few millisecond, as can be seen in implantable ICDs. This current leads to a simultaneous depolarization in a large part of the myocardium and will then terminate the arrhythmia. For safety reasons, an external defibrillator should be

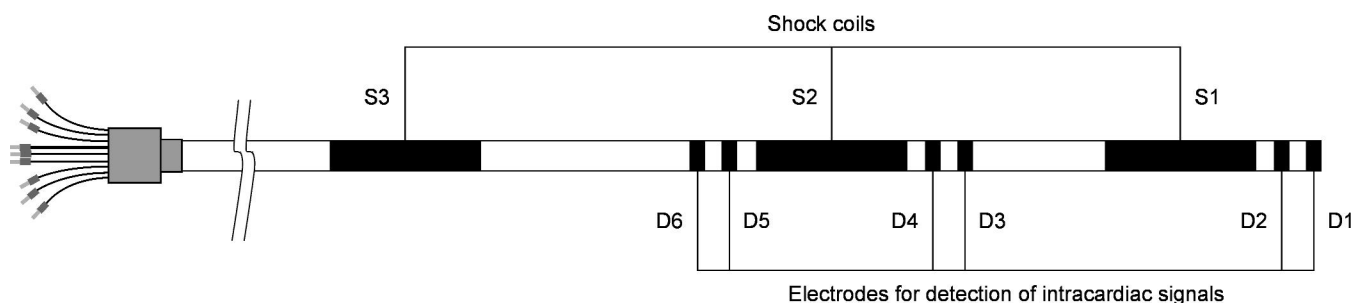


Figure 1. Catheter V207 (Biotronik, Germany) for atrial internal cardioversion. Shocks can be delivered between S1 and S2, S1 and S3, or even S2 and S3; bi-directional shocks can also be delivered. Each shock coil has a length of 60 mm and the distances from S1 to S2 and from S2 to S3 are 78 and 114 mm, respectively.

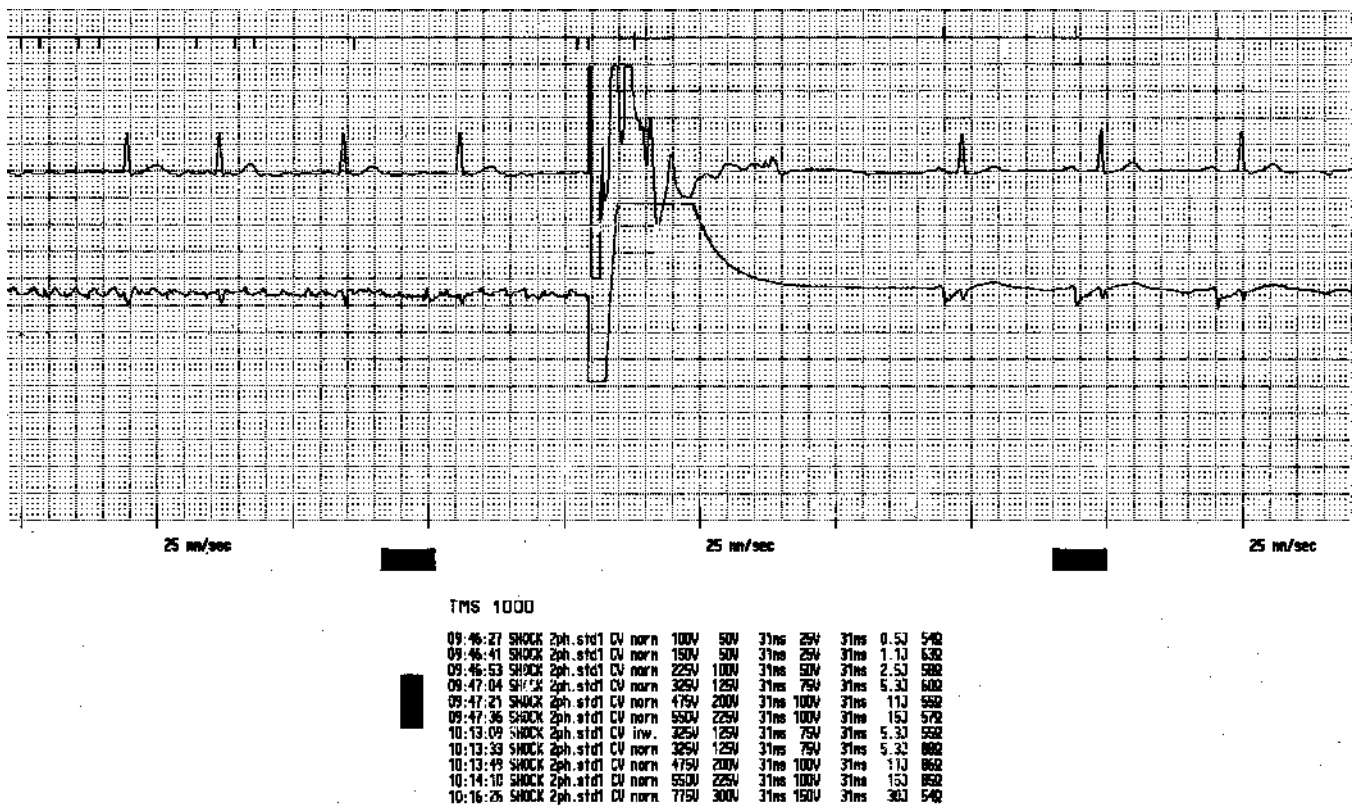


Figure 2. Successful termination of atrial fibrillation with a maximum energy of 30 J as a last resort of therapy in a patient with persistent atrial fibrillation that was refractory to external cardioversion.

available during the investigation because there is a low risk of inducing ventricular tachyarrhythmias by inadvertently timing the shock in the vulnerable phase of the ventricular activation or by delivering properly timed shocks at ventricular cycle lengths below 300 ms [17].

The patient was sedated with propofol and the defibrillation threshold was determined. Cardioversion was successful at 30 J with normal shock impedance (Figure 2). Later in the procedure, atrial fibrillation was induced by catheter manipulation, and this was terminated with a single 15-J shock accompanied by additional mild sedation.

Case Presentation 2

A second patient had a long history of recurrences of persistent atrial fibrillation that could only be converted to sinus rhythm via external cardioversion. The natural course changed a year before the ablation

procedure: atrial fibrillation was very difficult to terminate with external electrical cardioversion. The 24-hour ambulatory ECG revealed numerous atrial extrasystoles, and a focal site of atrial fibrillation was suspected. The patient was extremely obese, and had diabetes mellitus and regulated hypertension. His weight had recently gone down from 170 kg to 140 kg, and his height was 176 cm. The catheters were introduced via the femoral vein and the left subclavian vein for positioning in the coronary sinus for cardioversion, and in the pulmonary veins for mapping the focal extrasystolic beats. A subsequent increase of energy during internal cardioversion did not result in sinus rhythm, despite the use of the maximum energy of 30 J. A full dose of intravenous ibutilide (Upjohn) was given twice without results. Fifteen minutes after the last dose of ibutilide, a second attempt at internal cardioversion was made, which led to a successful conversion to sinus rhythm at 30 J.

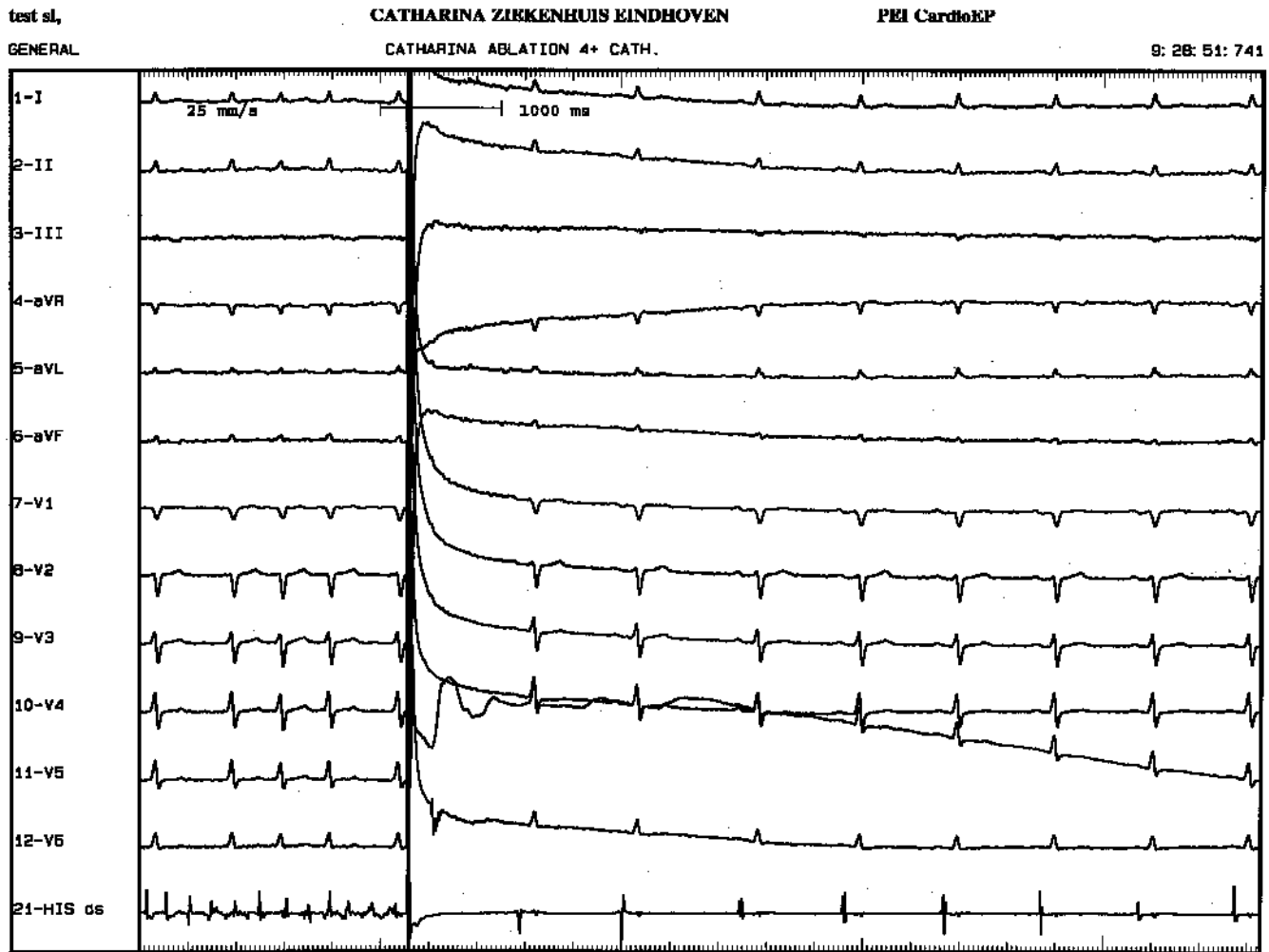


Figure 3. Persistent atrial fibrillation is converted to sinus rhythm during the EP-procedure of mapping the extrasystolic beats generated by the focus in the left pulmonary vein. The internal cardioversion was successful after a dose of IV ibutilide.

Discussion

Internal atrial cardioversion has recently been introduced as an alternative treatment to external cardioversion [11]. The success rate of external cardioversion has been further improved by simple or technical methods such as using the anteroposterior positioning for the patches [18], exercising pressure on the defibrillation patches, automated adjustments of the shock energy [19], and the use of biphasic shocks [20]. In a direct comparative study, the efficacy of internal cardioversion was significantly greater than that of external cardioversion (91 % versus 67 %, $P = 0.002$) [16]. The only variable that was associated with the outcome of cardioversion was body weight. Despite the reduc-

tion from 170 kg to a 140 kg, body weight was still a great problem in the second patient, especially with respect to the cardioversion. The goal of the entire procedure was to detect the origin of the extrasystolic beats in order to ablate the focus, which was probably located in one of the pulmonary veins. Therefore, cardioversion needed to be successful. Administering antiarrhythmic medication before internal or external cardioversion may increase the success rate, but the antiarrhythmic medication may suppress the extrasystolic beats needed for localization of the focus. However, some antiarrhythmic drugs, such as ibutilide and flecainide, lower the internal cardioversion or defibrillation threshold [21-22]. Ibutilide itself may

chemically convert the atrial fibrillation into sinus rhythm, even in cases of long-standing atrial fibrillation [23]. The success rate of flecainide diminishes with the duration of the arrhythmia. When atrial fibrillation was present for more than 24 hours, the success rate with flecainide decreased from 86 % to 40 % [24]. Because of the suppression of the atrial extra systoles and the low chance of successful cardioversion, flecainide was not administered. The effect of ibutilide on the suppression of the atrial extrasystolic beats is unknown. Early after-depolarizations in the ventricle have been described in dogs in combination with a greater dispersion of ventricular repolarization [25]. In the clinical setting, the proarrhythmic effects are comparable to those of other drugs. In a survey of 568 patients treated with ibutilide, the incidence of sustained and non-sustained polymorphic VT was 1.7 % and 2.7 %, respectively. These episodes occurred within 30 minutes after the administration of ibutilide [23].

One of the drawbacks of internal cardioversion may be the need for expertise in handling and positioning the specialized catheter in the heart. Using internal cardioversion during an electrophysiologic (EP)-study eliminates this disadvantage. Another disadvantage is the displacement of the other catheters in the heart, but this will also occur with external cardioversion. The defibrillation thresholds were very high in these patients. This may be due to the long duration of the arrhythmia, and, in one case, severe obesity. It must be stressed that a second attempt at internal cardioversion showed immediate success. The applied energy was set at 15 J, but it could have been successful at a lower energy level. Earlier reports with a similar catheter showed low defibrillation thresholds with a mean threshold of 2.6 ± 2.3 J (range 0.6 – 7.3 J) [26].

The patient was sedated during the procedure in order to eliminate the sensation of pain. Depending on the patient, pain sensation or perception begins at values from less than 0.5 J up to 4 J. In general, perception of pain is dependent on the wave-form of the shock [27] and the shock energy [28]; a second shock arouses more discomfort than the first shock [29], which comes often by surprise and is very short-lived. This last consideration is a strong argument for performing the internal cardioversion in one step, but at a level of energy that is high enough to convert the rhythm: the shock sensation is over before the patient is aware of it. Most cardioversion was described by patients without sedation as a "heavy thump" in the chest that lasted for

Atrial fibrillation		
Step	< 48 h	> 48 h
1	Chemical CV	ECV standard
2	ECV standard/oblique	ECV AP/biphasic/patches
3	ECV AP/biphasic/patches	Ibutilide + ECV
4	Ibutilide + ECV	ICV
5	ICV	Ibutilide + ICV
6	Ibutilide + ICV	Amiodarone + ICV

Table 1. Tiered therapy for cardioversion of atrial fibrillation. CV = cardioversion; ECV = external cardioversion; AICV = internal cardioversion; AP = anteroposterior positioning.

a few seconds [28]. By reducing the shock energy, the physician is then able to limit the degree of anesthesia to conscious sedation with midazolam or diazepam or even to no sedation, which would further simplify the procedure [27-30]. Performing atrial internal cardioversion rather than conventional external cardioversion may be considered at the request of the patient or if sedation is contraindicated.

Outside the EP-lab, in the general treatment of atrial fibrillation the proper role of internal cardioversion is under debate. Internal cardioversion should not replace external cardioversion because it requires expertise in the handling of catheters. In addition, catheters are expensive compared to the patches used for external cardioversion. Furthermore, fluoroscopy is needed. However, internal cardioversion may be viewed as an expansion of the therapeutic arsenal and can be considered as a last resort of therapy. For the clinical practice in general, the therapeutic role of ibutilide infusion and internal cardioversion is given in Table 1. The patients should be differentiated by the duration of atrial fibrillation, and for each category of patients a six-step protocol can be followed. For example, after failure of conversion using flecainide or other Class Ia or Ic anti-arrhythmic drugs, external cardioversion is attempted. If these attempts are made with the standard patch positions or monophasic shocks, the next attempts can be made with oblique positions of the patches or biphasic shocks. As a fourth step, ibutilide can be administered followed by external cardioversion. Internal cardioversion is the fifth step; if unsuccessful, it may be directly

followed by ibutilide infusion and renewed attempts at internal cardioversion. Overall, a similar pattern can be applied in patients with long-standing atrial fibrillation, which requires full antithrombotic therapy. The last step may be the administration of amiodarone, possibly combined with internal cardioversion.

Conclusion

Internal atrial cardioversion is a useful tool in the EP laboratory when termination of atrial fibrillation is indicated or necessary and Class I or III anti-arrhythmic medication is strictly contra-indicated. Ibutilide is a potent drug that enhances the success of cardioversion, but the serious side effects of the drug should be monitored. During an EP study, these side effects can be monitored easily and do not hamper the progress of the intended ablation. Therefore, we recommend the use of ibutilide when subsequent internal and external cardioversion fail. A second attempt at internal cardioversion after administration of ibutilide can be performed safely and easily.

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