Electrical Therapy of Atrial Fibrillation

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The limitations of pharmacological therapy have led to novel electrical approaches for treatment of atrial fibrillation, including pacing, ablation and atrial defibrillation. The choice of these approaches mostly depends on the type of atrial fibrillation and specific patient subpopulation.

Atrial fibrillation is the most frequent arrhythmia that occurs in humans. Despite this, its medical control is still unsatisfactory. Recurrence of arrhythmia is common, and up to 50% of patients may experience a relapse of atrial fibrillation during a given antarrhythmic drug therapy within one year [1]. On the other hand, almost 20% of patients do not tolerate effective drugs, and proarhythmic events may occur, especially in the patients with left ventricular dysfunction [2]. In permanent atrial fibrillation, the lack of success of drugs is usually demonstrated as an inadequate ventricular rate control during exercise and the daily activity of the patients [3].

The limitations of pharmacological therapy have led to novel nonpharmacological, electrical approaches for treatment of atrial fibrillation. They can be used to prevent atrial fibrillation, to control ventricular rhythm during arrhythmia, to eliminate arrhythmogenic substrate responsible for maintenance of atrial fibrillation, or to convert atrial fibrillation to sinus rhythm. The current status of some of those options will be presented and discussed herein.

Atrial Pacing for Prevention of Atrial Fibrillation

The mechanism of atrial pacing in the prevention of atrial fibrillation is now better understood. The atrial pacing has an effect on a substrate and a trigger responsible for the development of atrial fibrillation [4, 5]. It has also some effect on the mechanical atrial function and the neurohumoral system. By acting on a substrate, the atrial pacing reduces dispersion of atrial refractoriness and interatrial conduction delay. Both of these effects result in the reduced window for atrial fibrillation to occur. In addition, there are some preliminary data that overdrive atrial pacing may suppress premature atrial beats [6].

Single-site atrial pacing

During the last ten years, several retrospective studies have found that atrial pacing in patients with sick sinus syndrome was associated with significantly lower development of atrial fibrillation and mortality, compared with ventricular pacing [7–9]. The main criticisms for these studies were bias in the mode selection and the inability to account for the effect of associated co-morbidity. The Danish study is the first one that prospectively examined the effects of atrial versus ventricular pacing in patients with sick sinus syndrome (Table 1) [10]. At the time of randomization, all patients had sinus rhythm, but about 45% of them had episodes of atrial fibrillation periodically. Over a mean follow-up period of 5.5 years, the cumulative incidence of atrial fibrillation events and chronic atrial fibrillation were significantly lower and the survival was significantly higher in the atrial than in the ventricular pacing group. Multivariate analysis identified the presence of atrial fibrillation episodes at the beginning of the study as the only independent risk factor associated with development of chronic atrial fibrillation. Recently, the Canadian Trial of Physiologic Pacing (CTOPP) showed that the annual rate of atrial fibrillation was significantly lower among the patients in a physiologic pacing group (DDD, DDDR or AAIR), than among those in a ventricular pacing group (5.3% versus 6.6%, p<0.05), with a reduction in relative risk of 18% [11]. The beneficial effect of physiologic pacing on the rate of atrial fibrillation was not apparent until two years following implantation. Subgroup analysis in CTOPP suggests younger patients without structural heart disease are more likely to benefit from physiological pacing for prevention of atrial fibrillation [12]. This is consistent with the Danish study, where the follow-up was 8 years.

Table 1. Atrial pacing in sick sinus syndrome

<table>
<thead>
<tr>
<th></th>
<th>Danish prospective study (follow-up 8 years)</th>
<th>Atarial pacing</th>
<th>Ventricular pacing</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>110</td>
<td>115</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pts with AF episodes at randomisation</td>
<td>43</td>
<td>51</td>
<td>&lt;0.51</td>
<td></td>
</tr>
<tr>
<td>Pts with AF episodes at control</td>
<td>26</td>
<td>40</td>
<td>&lt;0.012</td>
<td></td>
</tr>
<tr>
<td>Pts with developed chronic AF</td>
<td>9</td>
<td>22</td>
<td>&lt;0.004</td>
<td></td>
</tr>
</tbody>
</table>

*Pts = patients; AF = atrial fibrillation*
with the Danish study because patients with intact atrioventricular (AV) conduction tend to be younger and healthier than the general pacemaker population. The exact mechanism that links atrial pacing with a clinical benefit is not quite clear. Ventricular pacing may predispose or atrial pacing may prevent or delay the natural evolution of sinus node disease to chronic atrial fibrillation. The explanation might be related to atrial overdrive pacing eliminating sinus bradycardia with concomitant homogenization of atrial refractory period, or to preserved AV synchrony, or both. However, it is not known if atrial pacing did really reduce atrial fibrillation or was only exhibiting less atrial fibrillation than ventricular pacing because neither of these prospective studies had a control group of patients without pacing.

The use of rate-adaptive atrial pacing in the prevention of atrial fibrillation in the absence of symptomatic bradycardia was recently evaluated in the study by Gillis et al. [13]. A Medtronic Thera DR pacemaker was implanted in 97 patients with drug refractory paroxysmal atrial fibrillation 3 months before planned AV node ablation. Patients were randomized to no pacing (n=48) or to atrial rate-adaptive pacing (n=49) group. Over the period of 10 weeks, paroxysmal atrial fibrillation burden was lower in the no-pacing group than in the atrial pacing group, while time to first episode of sustained paroxysmal atrial fibrillation was similar in both groups. These results show that atrial rate-adaptive pacing does not prevent paroxysmal atrial fibrillation over the short term in patients with drug-resistant paroxysmal atrial fibrillation without symptomatic bradycardia. For better evaluation of this mode of atrial pacing, studies with longer follow-up are needed, because a delayed beneficial effect of atrial pacing was observed in the Danish and CTOPP study [10, 11].

**Interatrial septal pacing**

The rationale for interatrial septal pacing is to achieve synchronous depolarization of both atria without concern for interatrial conduction. The theory is that such a pacing system allows an optimal timing of the left-sided atrioventricular delay [14]. The validity of this pacing system was examined by Padeletti et al. in 25 patients with paroxysmal atrial fibrillation (about 6 episodes per month) and sinus bradycardia [15]. For achieving the interatrial septal stimulation, a steroid eluting fixation lead was used in the right atrium and a passive fixation lead in the coronary sinus. In the recent study by this group, biatrial pacing was performed by simultaneous stimulation of the right high atrium and either the coronary sinus ostium or distal coronary sinus [17, 18]. Dual-site atrial pacing has been performed by simultaneous stimulation of the high right atrium and either the coronary sinus ostium or distal coronary sinus [17, 18]. Both pacing modalities significantly shortened P-wave duration due to two simultaneously originating wavefronts, fusion occurring in the region of crista terminals and the coronary sinus ostial area.

In a prospective crossover study, Default et al. examined the benefit of dual site atrial pacing in 30 patients who had atrial fibrillation refractory to drug therapy. The primary indication for cardiac pacing was sick sinus syndrome in 8 (27 %) patients, conduction system disease in 6 (20 %), drug-induced bradycardia in 11 (37 %) and neurocardiogenic syncope with bradyarrhythmia in 5 (17 %) patients. During nine months, the freedom from any recurrence of atrial fibrillation was significantly higher during dual-site than during single site right atrial pacing (89 % versus 62 %, p < 0.02). High right atrial pacing and coronary sinus ostial pacing did not differ in efficacy. Over the three year follow-up, three groups of patients could be stratified in this study. In about 10 % of patients, dual atrial pacing could not achieve even control over the follow-up. About 50 % of patients were free of atrial fibrillation suppression by dual-site pacemaker, and 40 % of patients have had infrequent relapses of atrial fibrillation, requiring adjunctive drug therapy. The benefit of dual atrial pacing has been seen in patients with and without manifest bradyarrhythmias at the time of atrial fibrillation emergence. This would suggest that this beneficial effect is rather due to direct action on the atrial arrhythmia, by changing of slow conduction in regional areas, or by reduction in dispersion of refractoriness, than mediated by bradycardia prevention.

The presence of cardiac disease was noted in 73 % of patients, proving that this technique can be successfully applied to the atrial fibrillation population with and without structural heart disease. However, intractable heart failure or severe mitral regurgitation have not been associated with atrial fibrillation suppression and should be grounds for exclusion for patient selection at this time.

Biatrial pacing was the first introduced by Daubert et al. for the treatment of severe intra- and/or interatrial conduction delay in patients with sick sinus syndrome [18]. A screw-in fixation lead was used in the right atrium and a passive fixation lead in the coronary sinus. In the recent study by this group, biatrial pacing was evaluated in a subpopulation of 86 patients with drug refractory atrial fibrillation and interatrial conduction delay of ≥ 100 ms [19]. During biatrial pacing, the mean duration of P-wave was shortened from 187 ms to 105 ms. After a mean follow-up period of 33 months, 64 % of patients remained in sinus rhythm and 33 % were free of atrial fibrillation recurrence. The only predictive factor of positive response to biatrial pacing was P-wave duration > 160 ms at baseline evaluation. It is quite obvious that in this subpopulation of patients, biatrial pacing reduces total atrial activation time and modifies or eliminates regional areas of slow conduction. Coronary sinus dislodgement seen in 20 % of patients was the major technical problem. The main limitation of this study was the absence of randomized or crossover comparison with single-site pacing. Therefore, it is not clear whether a beneficial effect was obtained by biatrial or high right atrial pacing. In the recent, randomized, crosso-
New algorithms for atrial fibrillation prevention
There are several algorithms for atrial fibrillation prevention, incorporated in new pacemaker generations, that are now in the phase of the clinical investigation.

The Premature Atrial Complexes (PAC) suppression algorithm is utilized in the AFAtherapy European study [21]. If a premature atrial beat is sensed, the pacing rate increases by 15 beats/min over the mean last 8 basic cycles. This rate is maintained for 600 beats, thereafter beginning a slow slackening of 1 beat/min every 16th cycle. Not to reach too high a rate of atrial pacing, this algorithm is applied only once after the activation.

The Pace Conditioning algorithm intends to overdrive the atrium constantly at a rate just above the spontaneous sinus rate [21]. It utilizes the same parameters as a PAC suppression algorithm without the plateau of 600 beats, and the trigger is the sense of the intrinsic sinus activity. The Consistent Atrial Pacing and Overdrive Atrial Pacing algorithms aim at the same result [6, 22]. After every sensed atrial event outside the post ventricular refractory period, the atrial escape interval is shortened by a programmable value until the programmed upper rate value is reached. After a programmable number of paced atrial events, the atrial escape interval is lengthened by a programmable value until the programmed lower rate value is reached.

The Dynamic Atrial Overdrive (DAO) algorithm is designed so that the atrial pacing rate is always just above the patient’s intrinsic atrial rate [23]. It accomplishes this by continually monitoring the atrial rate, promptly increasing the stimulation rate when the intrinsic rhythm emerges, and periodically comparing its own rate with the intrinsic one so as not to maintain the heart rate at an inappropriately high level for a longer period. In essence, the DAO algorithm reflects the circadian rhythm while delivering a high percentage of atrial pacing.

The Atrial Rate Stabilization algorithm prevents intrinsic PAC from being followed by a long pause. This algorithm measures each atrial interval delivering the next pacing escape interval at the same interval plus a programmable time (Figure 1). In the case of an early PAC, the short previous interval is measured and the subsequent cycle is only slightly longer, nevertheless much shorter than the base or sensor defined interval.

In summary, atrial pacing strategy should be considered in different subpopulations of patients with atrial fibrillation. There is no doubt that patients with sick sinus syndrome benefit from single-site atrial pacing. However, the benefit has been observed only after a relatively long follow-up period. In other subpopulations of patients with symptomatic atrial fibrillation, the new methods of pacing, like interatrial septum pacing or dual right atrial pacing can be effective and sufficient. In a subpopulation which has significant interatrial conduction delay (P-wave duration ≤ 160 ms) and drug-refractory atrial fibrillation with or without bradycardia the atrial pacing can be useful, alone or combined with adjunctive therapy like drugs or ablation. The new algorithms for atrial prevention are promising, but their efficacy needs a confirmation in ongoing trials.

Atroventricular Nodal Ablation Versus Atroventricular Nodal Modification

The therapeutic goal of AV nodal ablation and AV nodal modification is to obtain adequate ventricular rate control during paroxysmal or chronic atrial fibrillation refractory to drugs. However, the end-point of the AV nodal ablation is to induce complete AV block with subsequent pacemaker implantation, while the end-point of the AV nodal modification is to achieve an average ventricular rate lower than 120 beats per minute during isoproterenol or atropine infusion, with preserved AV conduction. The indications for AV nodal ablation and AV nodal modification are the same:

1) patients with symptomatic atrial fibrillation who have inadequately controlled ventricular rate unless primary ablation of the atrial fibrillation is possible, 2) symptomatic atrial fibrillation such as those above but when drugs are not tolerated or the patient does not wish to take them, and 3) patients resuscitated from sudden cardiac death due to atrial fibrillation/flutter with rapid ventricular response in the absence of an accessory pathway [24]. The target zone of AV nodal modification is the midseptal or posteroseptal area of the right atrium, where the AV nodal slow pathway would be expected. In patients with atrial fibrillation, an analysis of the distribution of RR intervals from Holter monitoring can be useful for the AV nodal modification. A bimodal RR interval distribution during atrial fibrillation is highly suggestive for the presence of two anatomically distinctly separated entrances to AV node, one from the interatrial septum and one from the terminal part of the crista terminals between the orium of the coronary sinus and the tricuspid valve [25]. Accordingly, patients with such finding are appropriate candidates for AV nodal modification, targeting a posteroseptal area of the right atrium.

Recently, two studies compared the benefits and limitations of AV nodal ablation and AV nodal modification (Table 2) [26, 27]. The success rate of the AV nodal ablation is almost 100 %, but the patients have lifelong pacemaker dependency [28]. The success rate of the AV nodal modification is lower, about 70 %, but the patients do not need pacemaker implantation [29, 30]. However, a complete AV block, induced by this procedure was observed in about 10–20 % of patients. The AV nodal ablation has been proven more effective than AV nodal modification in ventricular rate control, in reducing of symptoms, in improving quality of life, and in

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**Figure 1.** Atrial rate stabilization. Premature atrial beat (marked by arrow) occurs 520 ms after stimulated P-wave of DDD Jewel AF ICD pacing. Device starts immediately to stimulate atrium with the same interval (520 ms) plus programmable increment of 120 ms, augmenting the stimulating interval in every second stimulated P-wave (700 + 120 ms, 800 + 120 ms), until an interval of basal atrial stimulation is not obtained (960 ms).
improving the left ventricular ejection fraction, when it was lower than 40%. On the other hand, one third of patients with AV nodal modification continues to have palpitations, about 15% have recurrence of rapid atrial fibrillation, and some of them, with reduced left ventricular function, have a facilitation of polymorphic ventricular tachycardia [28]. Because this type of ventricular arrhythmia may be suppressed by an increase in rate and facilitated by a decrease in rate, it is likely that the relative bradycardia that ensued after the radiofrequency modification procedure is also a predisposing factor for polymorphic ventricular tachycardia [28].

It must be noted that AV nodal ablation and AV nodal modification are not curative techniques for atrial fibrillation, since arrhythmia remains and the embolic risk may not be reduced. The ablate and pace strategy has found wider clinical acceptance, probably because it is easier to apply and is considered to be safer and more effective in the long term. However, the influence of these techniques on survival has yet to be established.

### Radiofrequency Catheter Ablation of Atrial Fibrillation

Radiofrequency catheter ablation of atrial fibrillation is primarily based on the surgical maze procedure and aims to divide the atrial anatomy in a similar way. This technique is directed against the atrial tissue, and it should be considered regarding the mechanism of atrial fibrillation (Table 3). Therefore, the atrial mapping during spontaneous or induced atrial fibrillation is an important part of this therapeutic approach. The reduction of atrial mass should be created by using linear lesions in the right and left atrium, the purpose of which is to make impossible the random reentry of atrial impulses through the atria. To achieve this, lesions need to be continuous, transmural and connected with other lesions or anatomic structures that cause blockage of atrial conduction. The area between the inferior vena cava and the inferior part of the tricuspid annulus may be critical for development and maintenance of atrial fibrillation [31]. In these cases, the selected linear lesion should be done between these anatomic structures. If an arrhythmic focus is identified during electrophysiological study, it has to be carefully mapped. The ablation should be performed targeting the earliest bipolar atrial activity relative to the P wave onset on a surface electrocardiogram.

The results of radiofrequency ablation of atrial fibrillation, imitating the maze procedure, show that the success of this therapeutic option depends on the number and site of ablated lines. In the study by Haissaguerre et al., the right atrial ablation, performed with one, three, or four linear lesions, organized local electrical activity and led to stable sinus rhythm during the procedure in 18 (40%) of 45 patients, but noninducibility of atrial fibrillation was achieved only in 5 patients [32]. Final success rates with all three types of lesions were similar, ranging from 13% without drugs to 40% with drugs. When the linear lesions were performed in the right (3 lesions) and in the left atrium (3 lesions), the results were significantly better with a success rate of 87%. These results suggest that the left atrium is more relevant for maintenance of atrial fibrillation than the right atrium. Recent data from the same group show that bilateral ablation (3 linear lesions in the right and 5 linear lesions in the left atrium) was successful in 38 (84%) of 44 patients with multidrug resistant daily atrial fibrillation, during a mean follow-up of 20 months [33]. However, the additional sessions were required for focal ablation in 29 patients with atrial fibrillation and in 27 patients with newly created atrial flutter. The major disadvantage of this procedure is a large number of radiofrequency pulses necessary for compartmentalization of the target chamber and a long fluoroscopy and procedure duration (> 10 hours). Although there is increasing evidence about safety of this rather aggressive approach, its efficacy in achieving the intended model and curing the clinical symptoms remains to be determined.

Table 2. AV nodal ablation versus AV nodal modification: benefits and limitations

<table>
<thead>
<tr>
<th>Ablation</th>
<th>Modification</th>
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</thead>
<tbody>
<tr>
<td>1) Success rate ~ 100 %</td>
<td>1) Success rate ~ 70 %</td>
</tr>
<tr>
<td>2) Life-long pacemaker</td>
<td>2) No need for pacemaker implantation</td>
</tr>
<tr>
<td>3) Better ventricular rate control</td>
<td>3) Persistence of ventricular irregularity</td>
</tr>
<tr>
<td>4) Better control of symptoms and quality of life</td>
<td>4) Palpitations are not alleviated</td>
</tr>
<tr>
<td>5) Greater improvement of LVEF, when &lt; 40 %</td>
<td>5) Recurrence of rapid AF</td>
</tr>
<tr>
<td></td>
<td>6) Facilitation of polymorphic VT in some</td>
</tr>
</tbody>
</table>

LVEF = left ventricular ejection fraction; AF = atrial fibrillation; VT = ventricular tachycardia

Discovery of the rapidly firing atrial foci as a possible trigger of atrial fibrillation has enabled the development of techniques for their ablation [34]. Radiofrequency pulses can be successfully delivered to discrete sites presenting the earliest activation during spontaneous extra beats or at the time of onset of atrial fibrillation. In the study by Haissaguerre et al., 69 ectopic foci were identified as a trigger for atrial fibrillation, and ablated in 45 patients with frequent paroxysmal atrial fibrillation resistant to multiple drugs [34]. Sixty-five (94%) foci originated from the pulmonary veins, and 4 foci from the atrial tissue. The accuracy of mapping was confirmed by abrupt disappearance of triggering atrial beats after ablation with local radio-frequency energy. During a mean follow-up period of nine months, atrial fibrillation was eliminated completely in 28 (62%) patients without the use of drug therapy. The patients with one or two foci had a significantly higher success rate from the ablation than patients with more foci. It is important to note that significant pulmonary vein stenosis was induced in 10% of patients. The incidence of this complication tends to be lower by decreasing radiofrequency power limit for ablation from 50 to 30 watts [35].

Recently, ablation limited to the right atrium (3–4 linear lesions) was proposed as a therapeutic approach for patients with idiopathic atrial fibrillation [36, 37]. The rationale for this approach is the probability that critical area necessary for perpetuation of atrial fibrillation may be located in the right atrium, and to avoid risk of left atrium ablation in patients with this relatively benign arrhythmia.

The long-term results of this ablative approach were rather modest with efficacy between 28% and 61% [36–38]. In addition, Ernst et al. observed 100% recurrence rate of
idiopathic atrial fibrillation after subsequent right atrial ablation, using nonfluoroscopic mapping and 3 radiofrequency linear lesions [39].

Although the above findings are encouraging, radiofrequency ablation of atrial fibrillation is still considered to be an experimental procedure. The limitations of atrial ablation are related to the inability to accurately assess the precise anatomical location and extent of lesion formation. The risk/benefit ratio in the case of extensive ablation of the left atrium is unfavorable. With existing technology, map-guided ablation of a rapidly firing atrial focus seems the most likely solution.

### The Implantable Atrial Defibrillator

The success of the implantable cardioverter-defibrillator in the management of sudden cardiac death and recurring ventricular tachyarrhythmias has stimulated the investigators to create a similar device for atrial fibrillation. At present, two devices are commercially available (Table 4). The Metrix allows only defibrillation of the atrium, while the Jewel AF is able to treat atrial arrhythmias, including a shock to convert atrial fibrillation to sinus rhythm, and has the capacity to terminate life-threatening ventricular tachyarrhythmias.

The Metrix uses right atrial and coronary sinus lead configuration for atrial defibrillation and sensing and a bipolar right ventricular pacing lead for R-wave synchronization and pacing. The Model 3020 is able to deliver shocks up to 6 joules with bifasic waveform of 6 ms/6 ms duration. To avoid the potential ventricular proarrhythmic risk of atrial defibrillation shocks, appropriate R-wave synchronization needs to be performed and shocks should be delivered only after RR intervals above 500 ms.

The Jewel AF 7250 (Figure 2) is a dual chamber pacemaker as well as a dual cardioverter-defibrillator. The pacing and shock therapies for termination of tachyarrhythmias can be delivered both to atrial and ventricular electrode configurations. This dual defibrillator consists of an active can with one atrial and one ventricular lead, although an additional output may be used to accommodate a coronary sinus lead for lowering atrial defibrillation threshold. The primary goal of the Jewel AF is to treat promptly life-threatening ventricular tachyarrhythmias. For the treatment of atrial fibrillation, this device has an algorithm for prevention of atrial arrhythmias, and it is designed for a tiered approach to delivering atrial therapies, including antitachycardia pacing (Figure 3), burst high frequency pacing and shock therapy with energy between 0.1 and 27 joules.

The efficacy and safety of the Metrix were evaluated in prospective multicenter study including 51 patients with recurrent symptomatic atrial fibrillation [40]. The patients enrolled in this study had either failed or had intolerable side effects to a mean of 3.9 antarrhythmic drugs. Most of these patients had no structural heart disease and a normal left ventricular function. Forty-one of them used an atrial defibrillator during the study. In those patients, 96% of 222 spontaneous episodes of atrial fibrillation were converted to sinus rhythm by the atrial defibrillator. Shocks did not induce ventricular arrhythmias or embolic events in any patient. However, atrial fibrillation defibrillation threshold increased from 1.3 to 2.5 during a mean follow-up of eight months.

The initial clinical evaluation of the Jewel AF was focused on patients with an accepted indication for an implantable cardioverter-defibrillator, who in addition suffered from atrial fibrillation and flutter, or who had specific indication for dual chamber pacing. At the present time, the Jewel AF has been implanted in 303 patients [41]. During a mean follow-up of eight months, about 61% of spontaneous atrial tachycardia episodes were terminated by painless therapy, with antitachycardia pacing or high frequency burst pacing, and about 72% of 133 atrial fibrillation episodes were terminated by shock. In the same time, therapy success rate for ventricular tachycardia episodes was 97%, and for ventricular fibrillation episode was 100%. However, early recurrence of atrial fibrillation after successful shock therapy was observed in 22% of atrial fibrillation episodes [42].

From the above, two different groups of patients with atrial fibrillation may be selected for an implantable atrial defibrillator. The patients at low risk for ventricular tachyarrhythmias are candidates for the atrial defibrillator only, while patients with atrial fibrillation, who have an indication for cardioverter-defibrillator have an indication for Jewel AF. There are several problems that must be overcome before these devices can find wide clinical acceptance. First is

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**Table 4. Electrical devices for the management of atrial fibrillation**

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Weight/Volume</td>
<td>82 g / 53 cc</td>
<td>93 g / 56 cc</td>
</tr>
<tr>
<td>Lead system</td>
<td>3</td>
<td>2 or 3</td>
</tr>
<tr>
<td>Pacing support</td>
<td>VVI</td>
<td>DDD</td>
</tr>
<tr>
<td>Prevention</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>ATP/50 Hz</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Max. shock energy</td>
<td>6 J</td>
<td>27 J</td>
</tr>
<tr>
<td>VT/VF support</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Electrogram</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

ATP = antitachycardia pacing; VT = ventricular tachycardia; VF = ventricular fibrillation; J = Joules.
a pain perception during delivery of atrial shock that is too strong and has a negative influence on the quality of life. Early reinitiation of atrial fibrillation after successful shock therapy may cause premature depletion of the device and reduce its long-term efficacy. Thus, the most important issue is related to the selection of the patients who really need the implantable atrial defibrillator only.

**Conclusion**

The spectrum of therapeutic electrical approaches is large, but each has its advantages and limitations, in part depending on the type of atrial fibrillation being treated and in part based on the specific patient population. Regarding the complex pathophysiology of underlying atrial fibrillation it is unlikely that one therapeutic modality will adequately treat the majority of atrial fibrillation. Therefore, it is reasonable to presume that therapy combining the advantages of different therapy approaches will offer optimal care to patients in the future.

**References**


5. Saksena S, Delfaut P, Prakash A, Kuhashik RR, Krol RB. Multisite electrode related to the selection of the patients who really need the implantable atrial defibrillator only.

Focus Figure 3. Termination of atrial tachycardia by anti-tachycardia pacing. In the upper panel, the tachycardia with a mean cycle length of 440 ms is starting. After detection of tachycardia, the Jewel AF ICD starts with an atrial burst pacing (bottom panel) and terminates tachycardia. AS = atrial sensing, TS = tachycardia sensing, VP = ventricular pacing, TD = tachycardia detection, AP = atrial pacing.
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