Non-pharmacological treatment of supraventricular arrhythmias

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In recent years the recognition of toxicity and relative ineffectiveness of antiarrhythmic drugs in the management of arrhythmias has led to the development of non-pharmacological approaches. In the setting of supraventricular arrhythmias, such non-pharmacological approaches include preventative pacing, atrial defibrillation, surgery and radiofrequency catheter ablation. Success rates for catheter ablation exceeding 90% are expected in A-V nodal reentrant tachycardia, A-V reentry due to concealed or manifest bypass tracts, and incessant atrial tachycardias. Success rates in the management of atrial flutter are rapidly approaching this. Atrial fibrillation remains the main problem for which pharmacological therapy may still be useful. New preventative pacing modes, automatic atrial defibrillation, and perhaps surgical or radiofrequency procedures to limit the ability to sustain atrial fibrillation are on the horizon.

Key Words: Supraventricular tachycardia, electrophysiology, radiofrequency ablation.

Introduction

The ability to perform electrophysiological studies using catheter techniques, developed in the 1960s, permitted not only a better understanding of underlying mechanisms of arrhythmias, but also led to new concepts of treatment. Since then, during the last decade, different methods of non-pharmacological treatment of supraventricular arrhythmias have evolved. They all aim to prevent drug-related side effects and proarrhythmic effects as well as to avoid the haemodynamic consequences of arrhythmias.

The associated morbidity and mortality of cardiac arrhythmias originating in the atrium or requiring atrial or AV-nodal tissue for their maintenance includes palpitations and/or syncope, tachycardia-induced cardiomyopathy, induction of ventricular arrhythmias with the possibility of sudden cardiac death, and thromboembolic events associated with atrial fibrillation. At the very least these non-pharmacological therapies reduce the duration and frequency of hospitalization and reduce the cost of treatment.

Today, non-pharmacological therapies include pacing modalities, atrial defibrillation, surgery and catheter ablation using radiofrequency current (RF). While RF ablation is well established for treatment of atrioventricular-nodal reentry tachycardias (AVNRT), AV reentry tachycardias (AVRT) and atrial tachycardias, its efficacy in the treatment of atrial flutter and more importantly atrial fibrillation (AF), with its high prevalence in the population, is still not satisfactory. For this reason current investigation of the non-pharmacological treatment of atrial arrhythmias focus on these two types of supraventricular tachycardia (SVT).

Pacing for atrial tachyarrhythmias

Pacing in the setting of SVTs is used for different purposes. These include pacing to avoid combined symptomatic bradycardia, pacing to prevent episodes of tachycardia, and pace termination of SVTs.

Antibradycardia pacing in the setting of Brady-Tachy Syndrome has gained widespread use as many patients with atrial fibrillation also suffer from bradycardia-related symptoms. Bradycardia may be due to underlying sinus node dysfunction, or AV conduction disturbance each of which may be primarily or drug related. Over the last years, with the evolution in pacemaker technology, several studies comparing ventricular vs atrial based (either atrial or AV sequential) pacing have been published. Most have been retrospective, non-randomized studies; nevertheless, they indicate a marked reduction in the onset of chronic atrial fibrillation (47 vs 7%) and reduced mortality (23 vs 8%) when AV sequential pacing has been used. A study from Rosenqvist et al. further reported a reduction in congestive heart failure from 37 to 15% with atrial pacing systems. These observations found their expression in the guidelines of the British Pacing and Electrophysiology Group in 1991. They recommend the use of AV synchronized...
pacing with only one exception: chronic atrial fibrillation with AV block or slow ventricular response. A recent prospective and randomized Danish trial of 225 patients confirmed these findings[4].

Inhomogeneous slow conduction, with fractionated electrograms and short atrial refractoriness have been reported as the underlying substrate for atrial flutter and fibrillation[3], abnormalities which can be exaggerated by bradycardia and atrial premature beats allowing for initiation and maintenance of AF. Ventricular pacing may also contribute to the development of AF because of impaired haemodynamics, especially during ventriculo-atrial (VA) conduction with cannon A waves leading to atrial stretch and altered atrial refractoriness.

Atrial pacing at higher rates is believed to reduce dispersion of conduction, restore homogeneity of refractoriness and suppress ectopic activity. Attuel et al. showed in patients with paroxysmal, sustained, recurrent atrial arrhythmias and intra-atrial conduction delay that pacing paradoxically improved intra-atrial conduction[6]. In these patients, long-term suppression of AF was improved by atrial sequential pacing. The same group has also shown that atrial pacing in patients with vagally mediated atrial arrhythmias improves the suppression of AF[7]. Newer concepts to prevent AF using atrial pacing include pacing at increased rates after sensed atrial premature beats (APB), suppressing subsequent APBs as well as the prevention of post APB pauses[8] and pacing of more than one atrial site[9].

During electrophysiological studies in our laboratory we have demonstrated that simultaneous pacing of the high right atrium (HRA) and coronary sinus (CS) or CS pacing alone could prevent the induction of atrial flutter/fibrillation by HRA extrastimuli (Fig. 1). However, in our opinion the use of dual atrial or CS pacing in typical atrial flutter may be of less importance for chronic therapy because ablation with RF current has met with increasing success (see below).

The use of overdrive pacing for termination of supraventricular tachycardias such as AVNRT, AVRT, typical atrial flutter and atrial tachycardias is commonly used during electrophysiological studies. The long-term use of this pacing modality in antitachycardial pacing devices, however, has become antiquated because RF ablation has proven to be a safe, reliable and highly successful (>90%) technique to treat these arrhythmias (see below). Theoretically, overdrive pacing during AF may reduce the fibrillating mass and terminate AF. This implies that an excitable gap during AF is present. Although Allessie et al. have been able to capture areas of up to 4 cm² during AF, termination never occurred[9]. Pacing of multiple sites during electrophysiological studies of AF in our laboratory have shown the ability to capture multiple areas in the atrium during AF, but termination could never be achieved[10]. Further studies are needed to evaluate this concept.

**Atrial cardioversion and defibrillation**

Today, non-pharmacological treatment to terminate AF is limited to transthoracic electrical cardioversion. This method is highly successful in patients with AF of recent onset (<3 month) and for acute onset of AF with poor haemodynamic tolerance, reaching a 90% success rate[11]. Conversion to normal sinus rhythm (SR) and maintenance of SR is related to the duration of AF, atrial size, underlying heart disease and appropriate antiarrhythmic drug therapy. Early cardioversion is thought to be able to prevent structural and electrophysiological changes[12]. Based on this experience and the success with the implantable cardioverter/defibrillator (ICD) in the treatment of ventricular tachycardia (VT) and ventricular fibrillation (VF), interest in the development of an implantable atrial defibrillator has grown.

The first reports of transvenous atrial defibrillation by Mirowski and Mower appeared in 1974. Since then several studies have evaluated the ability to defibrillate the atrium in animals using transvenous[13,14] and transvenous combined with subcutaneous[15] electrode configurations. All demonstrated effective termination of AF with low energy shocks. However, in humans with atrial fibrillation, one frequently observes enlarged atria related to different underlying heart diseases. Thus the relevance of animal studies to human atrial fibrillation is uncertain. Keane et al. recently reported their experience with monophasic vs biphasic waveforms for atrial defibrillation intra-operatively[16] as well as their experience with endocardial catheter defibrillation using biphasic waveforms between the right atrium and coronary sinus[17]. Biphasic waveforms were superior with atrial defibrillation thresholds ranging from 3–8 joules. Current limitations for the use of this technique include: pain, detection of AF and ventricular proarrhythmia.

As known from ICD discharges during ventricular arrhythmias, severe pain is perceived at energy levels less than 2 joules. The acceptance of such a device for non-life threatening episodes of atrial fibrillation therefore makes it necessary to achieve effective atrial defibrillation with less energy. Small and variable atrial electrograms require sophisticated sensing algorithms to minimize electromechanical interference and oversensing of far-field ventricular electrograms. In animal studies the induction of VF as a proarrhythmogenic effect of atrial defibrillation reached 2.5%[18], making reliable synchronization to ventricular depolarizations essential. Careful patient selection to avoid frequent discharges due to asymptomatic short runs of AF with unnecessary painful shocks and fast battery depletion will also influence the acceptance of such a device. It is likely that a back-up ventricular defibrillator will be incorporated into such systems, particularly in patients with significant cardiac disease.

**Catheter ablation of supraventricular arrhythmias**

Over the past decade, catheter ablation evolved to become the major non-pharmacological therapy for...
Figure 1  Prevention of induced atrial fibrillation by left atrial pacing. Starting from the top, both panels are arranged with surface leads II and V₁ and recordings were of high right atrium (HRA), distal to the proximal His bundle (HBE d-p) coronary sinus proximal to distal (CS p-d), posterior Triangle of Koch (SP d-p), and right ventricular apex (RVA). In the top panel, an APD from the HRA during HRA pacing initiated atrial fibrillation. In the bottom panel, an HRA APD delivered at the same HRA coupling interval during CS pacing did not produce atrial fibrillation (see text).

supraventricular arrhythmias. It was first introduced for His bundle ablation in 1982[18,19] and direct current (DC) was used to modify or eliminate arrhythmia substrates through the 1980s. However, DC ablation was accompanied by major side effects including perforation of the myocardium and death thought to be due to proarrhythmogenic effects[20]. Recognizing these effects, the search for alternative energy sources resulted in the development of radiofrequency catheter ablation[21,22]. Producing well circumscribed and homogeneous
regions of coagulative necrosis, this technique has reduced undesired side effects dramatically\textsuperscript{24}, and for this reason DC ablation today has been essentially abandoned.

At the early stage of catheter ablation, DC current was directed to the AV junction to achieve ventricular rate control in patients with AF, atrial flutter and atrial tachycardia by destroying the AV node or His bundle. In AVNRT and AVRT this approach was used in selected patients to destroy a necessary component of the tachycardia circuit\textsuperscript{20}. With the exception of Wolf–Parkinson–White patients in which the accessory pathway (AP) is capable of AV conduction, all other patients required antibradycardia pacing.

With growing experience, a better understanding of tachycardia mechanisms, and the use of RF current producing well circumscribed lesions, has enabled AVNRT, AVRT, typical atrial flutter and atrial tachycardias to be cured.

**AV junctional ablation and modification**

AV junctional ablation is now reserved for treatment in drug-refractory AF with insufficient rate control or intolerable drug side effects and for treatment of symptomatic patients with atrial flutter and atrial tachycardia that have failed curative ablation. After initially positioning the ablation catheter across the tricuspid valve, the catheter is withdrawn and RF energy is directed to a site with a small His bundle recording and accompanying large atrial electrogram. In the rare instances when this approach fails, a left ventricular approach, positioning the ablation catheter along the posterior–superior interventricular septum often results in successful ablation of the His bundle. With these approaches, a success rate of >95% can be achieved\textsuperscript{25}.

Following AVN ablation, a permanent pacemaker is required. In paroxysmal AF, DDDR systems with mode switching algorithms are optimal, and in chronic AF, VVIR pacemaker remains state of the art. Because of the requirement for pacemakers, efforts have been undertaken to achieve sufficient rate control without producing complete heart block. In patients with AF, the persistence of this arrhythmia requires anticoagulation to minimize the risk of embolization. For this reason and because of the side effects following surgical treatment of AF (maze procedure), efforts have been made to improve our knowledge about mechanisms of AF and to develop catheter techniques aimed at achieving results similar to the maze procedure. A recent publication reports the first success of RF ablation in a more focal type of AF with organized left atrial activation: a net of RF lesions were created in the right atrium\textsuperscript{26}.

However, merely controlling the heart rate by AVN ablation has been shown to improve quality of life\textsuperscript{27,28} and exercise tolerance, and to reduce hospital admissions\textsuperscript{28}. This, together with eliminating drugs for rate control, is highly cost effective.

**Atrial tachycardia**

There are two types of atrial tachycardia. Patients with automatic atrial tachycardias most commonly have no underlying heart disease, but tachycardia-related cardiomyopathy may occur. The atrial foci are often located near the pulmonary veins, between the cavae, or at the base of the atrial appendages. Activation mapping with atrial activation preceding the onset of the P-wave by 20–60 ms is used to locate the zone of origin. RF application at this site often results in successful ablation\textsuperscript{29}.

Atrial reentrant tachycardia is normally related to structural heart disease with prior atrial surgery. Locating the protected zone (isthmus) in diastole requires the identification of early atrial activation, entrainment of the tachycardia to verify the relationship of the atrial activation to the subsequent P-wave and concealed entrainment to prove the site to be a critical portion of the reentrant circuit. The same approach is also used to verify a critical zone of slow conduction during typical atrial flutter, which has been shown to be located between the posterolateral IVC/RA junction and the tricuspid ring, extending to the isthmus between the os of the coronary sinus (CS) and the tricuspid ring\textsuperscript{30}.

Ablation in this region results in acute cure of typical atrial flutter in >90%\textsuperscript{31} (Fig. 2).

**AV nodal re-entry tachycardia**

AVNRT is characterized by two distinct patterns of AV conduction, referred to as 'slow pathway' (SP) and 'fast pathway' (FP). The SP is characterized by slow conduction velocity and a short refractory period and has been suggested to be at the posterior–inferior portion of the tricuspid annulus near the os of the CS and serves as the anterograde limb of the reentry circuit during typical AVNRT (90% of AVNRT). The FP exhibit rapid conduction, a relatively long refractory period and is usually located at the anterior–superior portion of the triangle of Koch, above the site of the His bundle recording, and serves as the retrograde limb during typical AVNRT. In atypical AVNRT the pathways are assumed to be used in the opposite direction.

Different approaches to either ablate the FP or the SP have been developed\textsuperscript{32–36}. The FP may be ablated either using an anatomical approach\textsuperscript{39} or by recording the earliest activation. FP ablation results in the elimination or at least marked attenuation of VA conduction and an increase in the AH interval. The HV interval, the AV block cycle length and the AV effective refractory period do not change significantly. Anatomically guided SP ablation may be safely performed in the region between the os of the CS and the tricuspid ring (SP region)\textsuperscript{33}. Other approaches use the morphology of atrial electrograms with so called SP-potentials\textsuperscript{34,35} or the earliest site of retrograde atrial activation during retrograde conduction over the SP. After ablation of the SP an increase in the anterograde AV block cycle length

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and an increase in the AV nodal effective refractory period occurs. When dual AV nodal physiology and single AVN echoes without inducible AVNRT persists (~50%), there is a recurrence rate of ~10%.

Both FP and SP ablation have proven successful in >95% of patients with a higher incidence of total AVN block after FP ablation\textsuperscript{132-361}. For this reason SP ablation should be the approach of first choice.

### Accessory bypass traits

In patients with either overt or concealed accessory bypass tracts (BT), ablation may be performed at the atrial or ventricular insertion site. To determine the ablation site, earliest atrial activation during orthodromic AVRT or V pacing is used. Early ventricular activation in relation to the onset of the delta wave in patients with overt BTs during sinus rhythm, antidromic AVRT or atrial pacing is used to guide RF ablation. Early retrograde activation during orthodromic SVT is used when concealed AV bypass tracts are operative.

The recording of accessory bypass tract potentials as sharp deflections between atrial and ventricular activation with a fixed relationship may be useful\textsuperscript{37,38} but proving these deflections are truly BT potentials is difficult and time consuming. In unfiltered unipolar electrograms, a QS-wave indicates the proximity of the ventricular activation origin and therefore provides additional information in patients with overt BT. Overall success rates in the ablation of BT also exceeds 90-95\%\textsuperscript{39} (Figs. 3(a) and (b)).

Because of the success rate and the low complication rate during RF ablation in atrial arrhythmias, this therapy is the treatment of choice in patients with symptomatic episodes and in selected patients without symptoms (e.g. airline pilots, professional athletes).

### Surgical approaches for atrial arrhythmias

Indications for surgical intervention in atrial arrhythmias have changed rapidly during the last 5 years. This is
due to the development and widespread use of catheter ablation techniques, resulting in successful ablation of AVNRTs, AVRTs, atrial tachycardias and typical atrial flutter in more than 90% of all cases. For this reason and because of its lower morbidity and mortality, RF ablation rendered surgical ablation a non-pharmacological therapy of second choice. Nevertheless, surgical treatment is necessary for drug-refractory atrial arrhythmias when RF ablation fails.

Surgical interruption of an AV bypass tract was first performed by Sealy et al. in 1969\[40\]. Since then two different approaches, the endocardial\[41\] and the

\[40\] Sealy et al., 1969

\[41\] Endocardial
single thromboembolic event during anticoagulation is rarely observed, although the morbidity is increased. Whether a patient is "clinically refractory" to AF in patients who cannot tolerate the presence of AF for a variety of reasons. It may also be important to consider the use of anticoagulation in these interventions has been reported.

For atrial tachycardias, guided by detailed mapping, excision or isolation of large areas of atrial tissue must be performed to achieve an acceptable success rate. Long-term success is uncertain and the development of other atrial foci has been observed after surgery.

Surgical ablation of the bundle of His, first introduced by Sealy et al., to control the ventricular response to atrial arrhythmias, is now obsolete because RF ablation of the AV node or His bundle is successful in nearly 100% of patients.

In 1985 Guiraudon et al. presented the first surgical technique for treatment of atrial fibrillation. This operation, called the corridor procedure, created a band of muscle including the sinus node and the AVN. This left the ventricular response controlled by the sinus node. The isolated right and left ventricular atrium, however, remain in atrial fibrillation, rendering anticoagulation necessary. For this reason, this procedure is infrequently used and has been replaced by AV nodal ablation and the use of pacemakers.

In 1987 Cox and Boineau introduced the 'maze procedure', based on the multiple wavelet hypothesis of AF developed by Moe and Abildskov, with each wavelet requiring a critical mass of atrial tissue. Using multiple atriotomies creating multiple dead end pathways, they were able to eliminate the possibility of impulses reentering an area of atrial tissue previously activated. The goal of this technique is to prevent AF and to maintain sinus rhythm. With a reported 98% success rate in selected patients, this procedure appears highly effective in experienced hands although several postoperative problems occur. Transient atrial flutter/fibrillation occurred postoperatively in nearly 50% of patients, which has been suggested to result from temporary shortening of atrial refractoriness by catecholamines, allowing smaller reentrant wavefronts to occur between two atrial incisions. Furthermore, massive fluid retention occurred frequently, which has been attributed to a loss of atrial natriuretic factor secondary to the atriotomies. Diuretic therapy is needed in these patients, as the atriotomy wound is an important factor in the morbidity and mortality. Am Heart J 1988; 116: 16-22.


References


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