Diagnosis and Management of Typical Atrial Flutter

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KEYWORDS

• Atrial flutter • Cavo-tricuspid isthmus • Ablation

Type 1 atrial flutter (AFL) is a common atrial arrhythmia that may cause significant symptoms and serious adverse effects including embolic stroke, myocardial ischemia and infarction, and rarely a tachycardia-induced cardiomyopathy as a result of rapid atrioventricular conduction. The electrophysiologic substrate underlying type 1 AFL has been shown to be a combination of slow conduction velocity in the cavo-tricuspid isthmus (CTI), plus anatomic and/or functional conduction block along the crista terminalis and Eustachian ridge (**Fig. 1**). This electrophysiologic milleu allows for a long enough reentrant path length relative to the average tissue wavelength around the tricuspid valve annulus to allow for sustained reentry.

Type 1 AFL is relatively resistant to pharmacologic suppression. As a result of the well-defined anatomic substrate and the pharmacologic resistance of type 1 AFL, radiofrequency catheter ablation has emerged in the past decade as a safe and effective first-line treatment. Although several techniques have been described for ablating type 1 AFL, the most widely accepted and successful technique is an anatomically guided approach targeting the CTI. Recent technological developments, including three-dimensional electro-anatomic contact and noncontact mapping, and the use of irrigated tip and large-tip ablation electrode catheters with high-power generators, have produced nearly uniform efficacy without increased risk. This article reviews the electrophysiology of human type 1 AFL, techniques currently used for its diagnosis and management, and emerging technologies.

ATRIAL FLUTTER TERMINOLOGY

Because of the variety of terms used to describe atrial flutter in humans, including type 1 AFL and type 2 AFL, typical and atypical atrial flutter, counterclockwise and clockwise atrial flutter, and isthmus and non-isthmus dependent flutter, the Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology convened and published a consensus document in 2001 in an attempt to develop a generally accepted standardized terminology for atrial flutter.[1] The consensus terminology derived from this working group to describe CTI-dependent, right atrial macroreentry tachycardia, in the counterclockwise or clockwise direction around the tricuspid valve annulus was ''typical'' or ''reverse typical'' AFL respectively.[1] For the purposes of this article, these two arrhythmias will be referred to specifically as typical and reverse typical AFL when being individually described, but as type 1 AFL when being referred to jointly.

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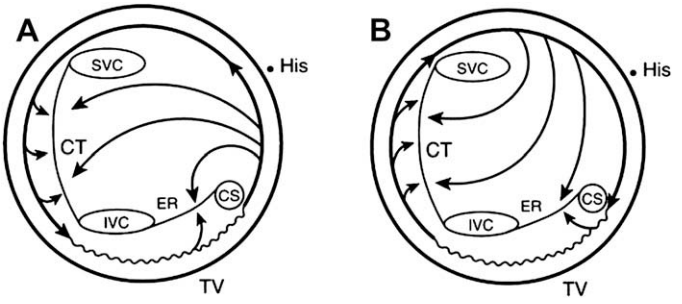

Fig.1. Schematic diagrams demonstrating the activation patterns in the typical (*A*) and reverse typical (*B*) forms of human type 1 AFL, as viewed from below the tricuspid valve annulus (TV) looking up into the right atrium. In the typical form of AFL, the reentrant wavefront rotates counterclockwise in the right atrium, whereas in the reverse typical form reentry is clockwise. Note that the Eustachian ridge (ER) and crista terminalis (CT) form lines of block, and that an area of slow conduction (*wavy line*) is present in the isthmus between the inferior vena cava (IVC) and Eustachian ridge and the tricuspid valve annulus. CS, coronary sinus ostium; His, His bundle; SVC, superior vena cava.

PATHOPHYSIOLOGIC MECHANISMS OF TYPE 1 ATRIAL FLUTTER

The development of successful radiofrequency catheter ablation techniques for human type 1 AFL was largely dependent on the delineation of its electrophysiologic mechanism. Through the use of advanced electrophysiologic techniques, including intraoperative and transcatheter activation mapping,[2–7] type 1 AFL was determined to be attributable to a macro-reentrant circuit rotating in either a counter-clockwise (typical) or clockwise (reverse typical) direction in the right atrium around the tricuspid valve annulus, with an area of relatively slow conduction velocity in the low posterior right atrium (see **Fig. 1**A, B). The predominate area of slow conduction in the AFL reentry circuit has been shown to be in the CTI, through which conduction times may reach 80 to 100 msec, accounting for one third to one half of the AFL cycle length.[8–10] The CTI is anatomically bounded by the inferior vena cava and Eustachian ridge posteriorly and the tricuspid valve annulus anteriorly (see **Fig. 1**A, B), both of which form lines of conduction block or barriers delineating a protected zone of slow conduction in the reentry circuit.[5,11–13] The presence of conduction block along the Eustachian ridge has been confirmed by demonstrating double potentials along its length during AFL. Double potentials have also been recorded along the crista terminalis suggesting that it also forms a line of block separating the smooth septal right atrium from the trabeculated right atrial free wall. Such lines of block, which may be either functional or anatomic, are necessary to create an adequate path-length for reentry to be sustained and to prevent short circuiting of the reentrant wavefront.[12–14] The medial CTI is contiguous with the interatrial septum near the coronary sinus ostium, and the lateral CTI is contiguous with the low lateral right atrium near the inferior vena cava (**Fig. 1**A, B). These areas correspond electrophysiologically to the exit and entrance to the zone of slow conduction, depending on whether the direction of reentry is counterclockwise (CCW) or clockwise (CW) in the right atrium. The path of the reentrant circuit outside the confines of the CTI consists of a broad activation wavefront in the interatrial septum and right atrial free wall around the crista terminalis and the tricuspid valve annulus.[11–14]

The slower conduction velocity in the CTI, relative to the interatrial septum and right atrial free wall, may be caused by anisotropic fiber orientation in the CTI.[2,8–10,15,16] This may also predispose to development of unidirectional block during rapid atrial pacing, and account for the observation that typical (CCW) AFL is more likely to be induced when pacing is performed from the coronary sinus ostium. Conversely, reverse typical (CW) AFL is more likely to be induced when pacing from the low lateral right atrium.[17,18] This hypothesis is further supported by direct mapping in animal studies demonstrating that the direction of rotation of the reentrant wavefront during AFL is dependent on the direction of the paced wavefront producing unidirectional block at the time of its induction.[19] In humans, the predominate clinical presentation of type 1 AFL is the typical variety, likely because the trigger(s) for AFL commonly arise from the left atrium in the form of premature atrial contractions or nonsustained atrial fibrillation.[20] Triggers arising from the left atrium or pulmonary veins usually conduct to the right atrium via the coronary sinus or interatrial septum, thus entering the CTI from medial to lateral, which results in clockwise unidirectional block in the CTI with resultant initiation of counterclockwise typical AFL.

The development of abnormal dispersion or shortening of atrial refractoriness as a result of atrial electrical remodeling may increase the likelihood of developing regional conduction block and

abnormal shortening of tissue wavelength responsible for initiating and sustaining reentry in AFL.[21,22]

ECG DIAGNOSIS OF TYPE 1 ATRIAL FLUTTER

The surface 12-lead ECG is helpful in establishing a diagnosis of type 1 AFL, particularly the typical form (**Box 1**). In typical AFL, an inverted saw-tooth flutter (F) wave pattern is observed in the inferior ECG leads II, III, and aVF, with a low amplitude biphasic F waves in leads I and aVL, an upright F wave in precordial lead V1, and an inverted F wave in lead V6. In contrast, in reverse typical AFL, the F wave pattern on the 12-lead ECG is less specific, often with a sine wave pattern in the inferior ECG leads (**Fig. 2**A, B). The determinants of F wave pattern on ECG are largely dependent on the activation pattern of the left atrium resulting from reentry in the right atrium, with inverted F waves inscribed in the inferior ECG leads in typical AFL as a result of activation of the left atrium initially posterior near the coronary sinus, and upright F waves inscribed in the inferior ECG leads in reverse typical AFL as a result of activation of the left atrium initially anterior near Bachman's bundle[23,24] Because the typical and reverse typical forms of type 1 AFL use the same reentry circuit, but in opposite directions, their rates are usually similar.

Box 1
Diagnostic criteria for typical and reverse typical AFL

1. Demonstration of a saw-tooth F wave pattern in the inferior ECG leads (typical AFL) or a sine wave or upright F wave pattern in the inferior ECG leads (reverse typical AFL), with atrial rate between 240 and 350 beats per minute, and 2:1 or variable AV conduction
2. Demonstration of counterclockwise (typical) or clockwise (reverse typical) macroreentrant circuit around tricuspid valve annulus by standard multi-electrode catheter mapping or 3-D computerized mapping
3. Demonstration of concealed entrainment criteria during pacing from the cavotricuspid isthmus, including acceleration of the tachycardia to the paced cycle length, first post-pacing interval equal to the tachycardia cycle length, and stimulus-to-F wave interval equal to electrogram-to-F wave interval on the pacing catheter

MEDICAL THERAPY VERSUS CATHETER ABLATION

Class III antiarrhythmic drugs, by selectively lengthening the cardiac action potential, have shown efficacy in converting atrial flutter to normal sinus rhythm.[25] However, despite an 80% initial success rate with the Class III agent Ibutilide,[26] recurrence rates are extremely high (70% to 90%) despite maintenance on antiarrhythmic drugs.[27,28] Therefore, catheter ablation is considered a first-line approach for many patients with atrial flutter given the high acute and chronic efficacy of the procedure (>90%) and relatively low complication rates.[29] Prospective trials that have randomized patients to medical therapy versus first-line catheter ablation have shown that patients who received ablation as a first-line strategy had significantly better maintenance of sinus rhythm, fewer hospitalizations, better quality of life, and fewer overall complications when compared with patients who received antiarrhythmic drug therapy.[28,30]

Despite the excellent acute results and long-term outcome after radiofrequency catheter ablation for freedom from type 1 atrial flutter, one must keep in mind that development of atrial fibrillation is high in this population of patients; 30% of these patients may develop atrial fibrillation over a 5-year period, especially if there is a history of atrial fibrillation or underlying heart disease.[28,30–32] However, ablation of the CTI may reduce or in rare cases may eliminate recurrences of atrial fibrillation, and CTI ablation is also effective in patients undergoing pharmacologic treatment for atrial fibrillation with antiarrhythmic drug–induced type 1 atrial flutter (the so-called "hybrid approach"). Ablation of the CTI may also be required in patients undergoing ablation for atrial fibrillation who also have a history of type 1 atrial flutter.[33]

ELECTROPHYSIOLOGIC MAPPING OF TYPE 1 ATRIAL FLUTTER

Despite the utility of the 12-lead ECG in making a presumptive diagnosis of typical AFL, an electrophysiologic study with mapping and entrainment must be performed to confirm the underlying mechanism if radiofrequency catheter ablation is to be successfully performed (see **Box 1**). This is particularly true in the case of reverse typical AFL, which is much more difficult to diagnose on 12-lead ECG. For the electrophysiologic study of AFL, activation mapping may be performed using standard multi-electrode catheters, or one of the currently available three-dimensional computerized activation mapping systems. For standard multi-electrode catheter mapping, catheters are

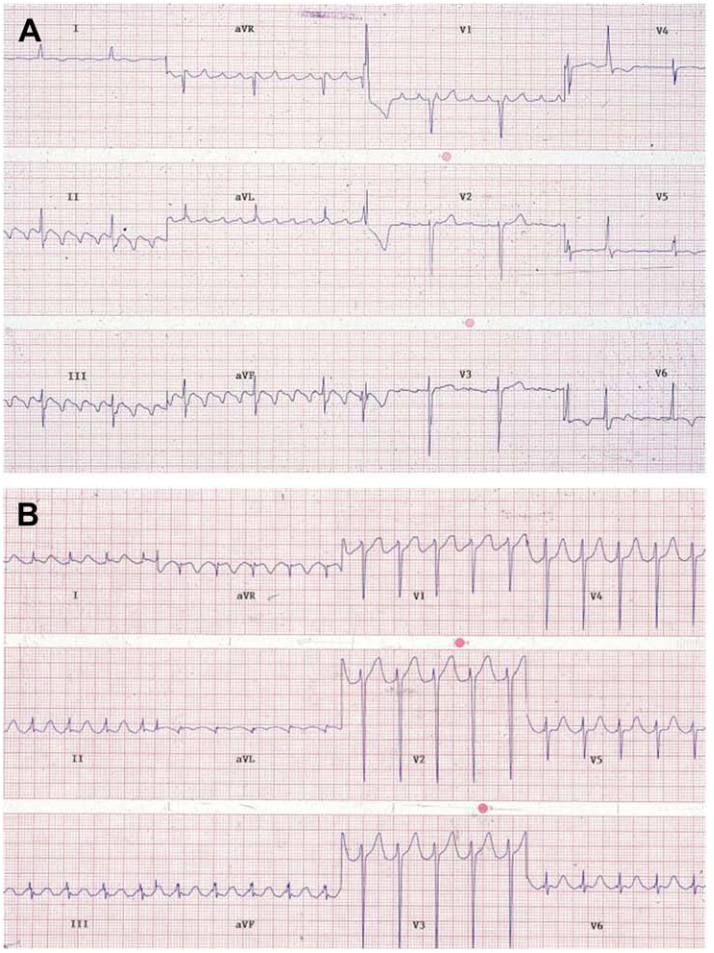

Fig. 2. (*A*) A 12-lead electrocardiogram recorded from a patient with typical AFL. Note the typical saw-toothed pattern of inverted F waves in the inferior leads II, III, aVF. Typical AFL is also characterized by flat to biphasic F waves in I and aVL respectively, an upright F wave in V1 and an inverted F wave in V6. (*B*) A 12-lead electrocardiogram recorded from a patient with the reverse typical AFL. The F wave in the reverse typical form of AFL has a less distinct sine wave pattern in the inferior leads. In this case, the F waves are upright in the inferior leads II, III, and aVF; biphasic in leads I, aVL, and V1; and upright in V6.

positioned in the right atrium, His bundle region, and coronary sinus. To most precisely elucidate the endocardial activation sequence, a Halo 20-electrode mapping catheter (Cordis-Webster, Inc., Diamond Bar, CA) is most commonly used in the right atrium positioned around the tricuspid valve annulus (**Fig. 3**). Recordings obtained during AFL from all electrodes are then analyzed to determine the right atrial activation sequence. In patients presenting to the laboratory in sinus rhythm it is necessary to induce AFL to confirm its mechanism. Induction of AFL is accomplished by atrial programmed stimulation or burst pacing. Preferred pacing sites are the coronary sinus ostium or low lateral right atrium. Burst pacing is the preferred method to induce AFL, with pacing cycle lengths between 180 and 240 msec typically effective in producing unidirectional CTI block and inducing AFL. Induction of atrial flutter typically occurs immediately following the onset of unidirectional CTI isthmus block.[17,18]

During electrophysiologic study, a diagnosis of either typical or reverse typical AFL is suggested by observing a counterclockwise or clockwise activation pattern in the right atrium and around the tricuspid valve annulus. For example, as seen in **Fig. 4**A in a patient with typical AFL, the atrial electrogram recorded at the coronary sinus ostium is timed with the initial down stroke of the F wave in the inferior surface ECG leads, followed by caudal-to-cranial activation in the interatrial septum to the His bundle atrial electrogram, and then cranial-to-caudal activation in the right atrial free wall from proximal to distal on the Halo catheter, and finally to the ablation catheter in the CTI, indicating that the underlying mechanism is a counter-clockwise macro-reentry circuit with electrical activity encompassing the entire tachycardia cycle length. In a patient with reverse typical AFL, the mirror image of this activation pattern is seen, as shown in **Fig. 4**B.

RADIOFREQUENCY CATHETER ABLATION OF TYPE 1 ATRIAL FLUTTER

Radiofrequency catheter ablation of type 1 AFL is performed with a steerable mapping/ablation

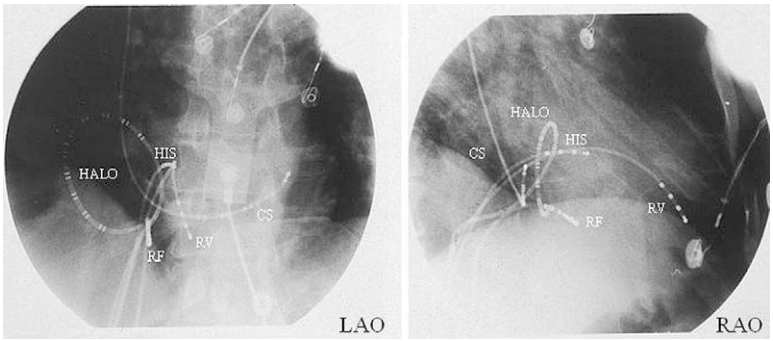

Fig. 3. Left anterior oblique (LAO) and right anterior oblique (RAO) fluoroscopic projections showing the intracardiac positions of the right ventricular (RV), His bundle (HIS), coronary sinus (CS), Halo (HALO), and mapping/ablation catheter (RF). Note that the Halo catheter is positioned around the tricuspid valve annulus, with the proximal electrode pair at 1 o'clock and the distal electrode pair at 7 o'clock in the LAO view. The mapping/ablation catheter is positioned in the sub-Eustachian isthmus, midway between the interatrial septum and low lateral right atrium, with the distal 8-mm ablation electrode near the tricuspid valve annulus.

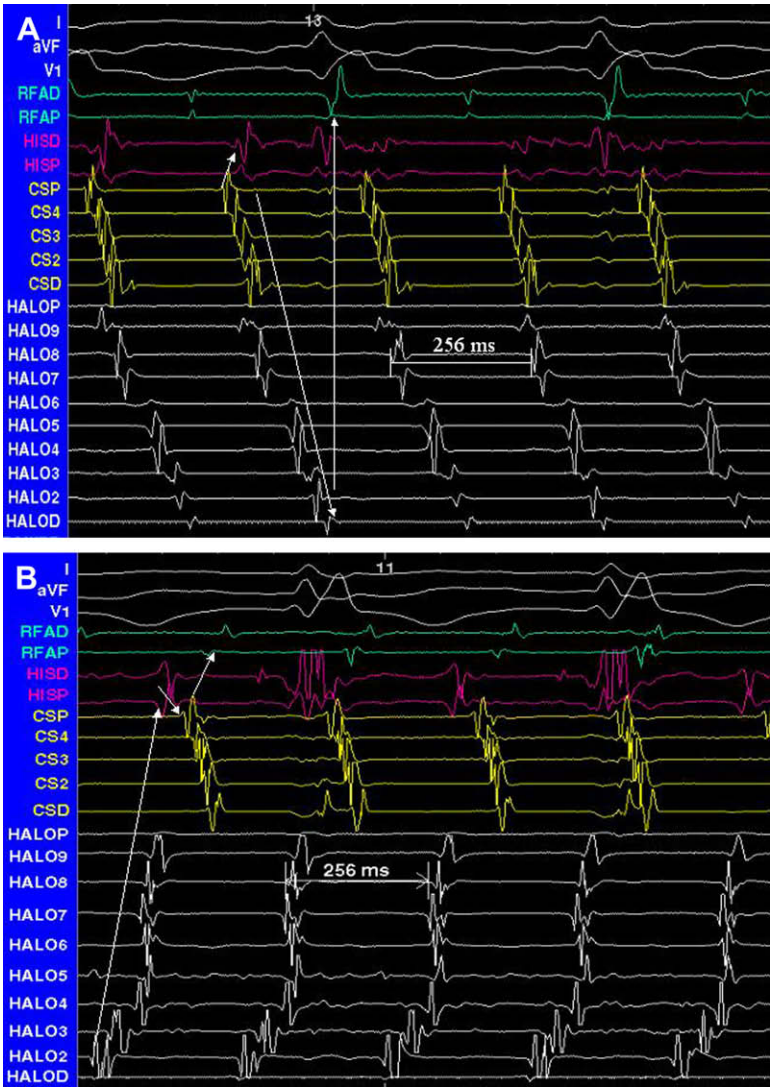

Fig. 4. Endocardial electrograms from the mapping/ablation, Halo, CS, and His bundle catheters and surface ECG leads I, aVF, and V1 demonstrating a counterclockwise (CCW) rotation of activation in the right atrium in a patient with typical AFL (*A*) and a clockwise (CW) rotation of activation in the right atrium in a patient with reverse typical AFL (*B*). The AFL cycle length was 256 msec for both CCW and CW forms. Arrows demonstrate activation sequence. Halo D - Halo P tracings are 10 bipolar electrograms recorded from the distal (low lateral right atrium) to proximal (high right atrium) poles of the 20-pole Halo catheter positioned around the tricuspid valve annulus with the proximal electrode pair at 1 o'clock and the distal electrode pair at 7 o'clock. CSP, electrograms recorded from the coronary sinus catheter proximal electrode pair positioned at the ostium of the coronary sinus; HISP, electrograms recorded from the proximal electrode pair of the His bundle catheter; RF, electrograms recorded from the mapping/ablation catheter positioned with the distal electrode pair in the cavo-tricuspid isthmus.

catheter with a large distal ablation electrode positioned in the right atrium via the femoral vein.[3,5–7,34–36] The typical radiofrequency generator used by most laboratories is capable of automatically adjusting applied power to achieve an operator programmable tissue-electrode interface temperature. Tissue temperature is monitored via a thermistor or thermocouple embedded in the distal ablation electrode. Programmable temperature with automatic power control is important because successful ablation requires a stable temperature of at least 50 to 60°C and occasionally 70°C. Temperatures in excess of 70°C may cause tissue vaporization (steam pops), tissue charring, and formation of blood coagulum on the ablation electrode resulting in a rise in impedance, which limits energy delivery and lesion formation, and may lead to complications such as cardiac perforation or embolization. A variety of mapping/ablation catheters with different shapes and curve lengths are currently available from several commercial manufacturers. We prefer to use a larger curve catheter (K2 or mid-distal large curve, EP Technologies, San Jose, CA), with or without a preshaped guiding sheath such as an SR 0, SL1, or ramp sheath (Daig, Minnetonka, MN), to ensure that the ablation electrode will reach the tricuspid valve annulus.

Recently, radiofrequency ablation catheters with either saline-cooled ablation electrodes or large distal ablation electrodes (ie, 8–10 mm) have been approved by the Food and Drug Administration (FDA) for ablation of type 1 atrial flutter (EP Technologies, Inc., Biosense-Webster, Inc., Medtronic, Inc.). During ablation with saline-cooled catheters, the use of lower power and temperature settings is recommended to avoid steam pops, because higher intramyocardial tissue temperatures are produced than measured at the tissue-electrode interface owing to the electrode cooling effect of saline perfusion.[37–39] Although studies have reported use of up to 50 W and 60°C for ablation of AFL without higher than expected complication rates, a maximum power of 35 to 40 watts and temperature of 43 to 45°C should be used initially.[37–40] In contrast, the large-tip (8- to 10-mm) ablation catheters require a higher power, up to 100 watts, to achieve target temperatures of 50 to 70°C owing to the greater energy dispersive effects of the larger ablation electrode. This also requires the use of two grounding pads applied to the patient's skin to avoid skin burns.[29,39,41,42]

The preferred target for type 1 AFL ablation is the CTI, which when using standard multipolar electrode catheters for mapping and ablation, is localized with a combined fluoroscopically and electrophysiologically guided approach.[3,5–7,29,34–40,42] Initially, a steerable mapping/ablation catheter is positioned fluoroscopically (see **Fig. 3**) in the CTI with the distal ablation electrode on or near the TV annulus in the right anterior oblique (RAO) view, and midway between the septumand low right atrial free wall (6 or 7 o'clock position) in the left anterior oblique view (LAO). The distal ablation electrode position is then adjusted toward or away from the TV annulus based on the ratio of atrial and ventricular electrogram amplitude recorded by the bipolar ablation electrode. An optimal AV ratio is typically 1:2 or 1:4 at the tricuspid valve annulus as seen in **Fig. 4**A on the ablation electrode (RFAD). After positioning the ablation catheter on or near the tricuspid valve annulus, it is very slowly withdrawn a few millimeters at a time (usually the length of the distal ablation electrode) pausing for 30 to 60 seconds at each location during a continuous or interrupted energy application. Electrogram recordings may be used in addition to fluoroscopy to ensure that the ablation electrode is in contact with viable tissue in the CTI throughout each energy application. Ablation of the entire CTI may require several sequential 30- to 60-second energy applications during a stepwise catheter pullback, or a prolonged energy application of up to 120 seconds, or more during a continuous catheter pullback. The catheter should be gradually withdrawn until the distal ablation electrode records no atrial electrogram indicating it has reached the inferior vena cava or until the ablation electrode is noted to abruptly slip off the Eustachian ridge fluoroscopically. Radiofrequency energy application should be immediately interrupted when the catheter has reached the inferior vena cava, because ablation in the venous structures is known to cause significant pain to the patient.

PROCEDURE END POINTS FOR RADIOFREQUENCY CATHETER ABLATION OF TYPE 1 ATRIAL FLUTTER

Ablation may be performed during sustained AFL or during sinus rhythm. If performed during AFL, the first end point is its termination during energy application. Despite termination of AFL, it is common to find that CTI conduction persists. After the entire CTI ablation is completed, electrophysiologic testing should then be performed. Pacing should be done at a cycle length of 600 msec (or greater depending on sinus cycle length) to determine if there is bidirectional conduction block in the CTI (**Fig. 5**A, B and **Fig. 6**A, B). Bidirectional conduction block in the CTI is confirmed by demonstrating a change from a bidirectional wavefront with collision in the right atrial free wall or interatrial septum before ablation to a strictly cranial to caudal activation sequence following ablation during

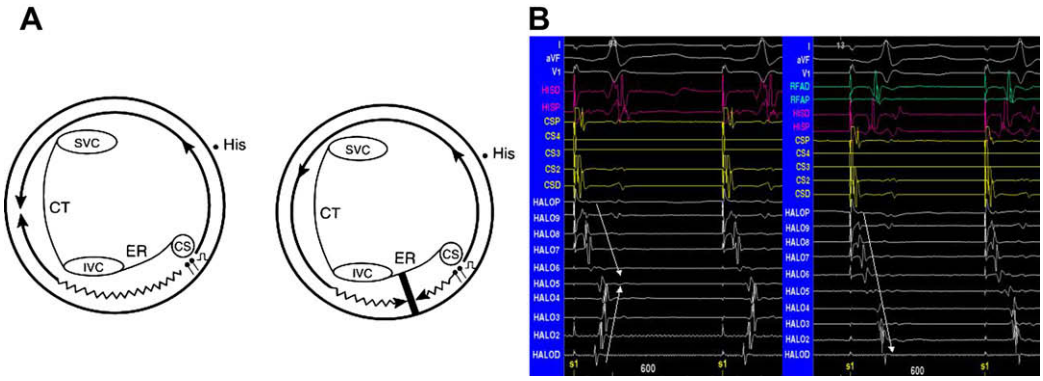

Fig. 5. (*A*) A schematic diagram of the expected right atrial activation sequence during pacing in sinus rhythm from the coronary sinus (CS) ostium before (*left panel*) and after (*right panel*) ablation of the cavo-tricuspid isthmus (CTI). Before ablation the activation pattern during coronary sinus pacing is caudal to cranial in the interatrial septum and low right atrium, with collision of the septal and right atrial wavefronts in the mid-lateral right atrium. Following ablation, the activation pattern during coronary sinus pacing is still caudal to cranial in the interatrial septum, but the lateral right atrium is now activated in a strictly cranial to caudal pattern (ie, counterclockwise), indicating complete clockwise conduction block in the CTI. CT, crista terminalis; ER, Eustachian ridge; His, His bundle; IVC, inferior vena cava; SVC, superior vena cava. (*B*) Surface ECG and right atrial endocardial electrograms recorded during pacing in sinus rhythm from the coronary sinus (CS) ostium before (*left panel*) and after (*right panel*) ablation of the cavo-tricuspid isthmus (CTI). Tracings include surface ECG leads I, aVF, and V1, and endocardial electrograms from the proximal coronary sinus (CSP), His bundle (HIS), tricuspid valve annulus at 1 o'clock (HaloP) to 7 o'clock (HaloD), and high right atrium (HRA or RFA). Before ablation, during coronary sinus pacing, there is collision of the cranial and caudal right atrial wavefronts in the mid-lateral right atrium (HALO5). Following ablation, the lateral right atrium is activated in a strictly cranial to caudal pattern (ie, counterclockwise), indicating complete medial to lateral conduction block in the CTI.

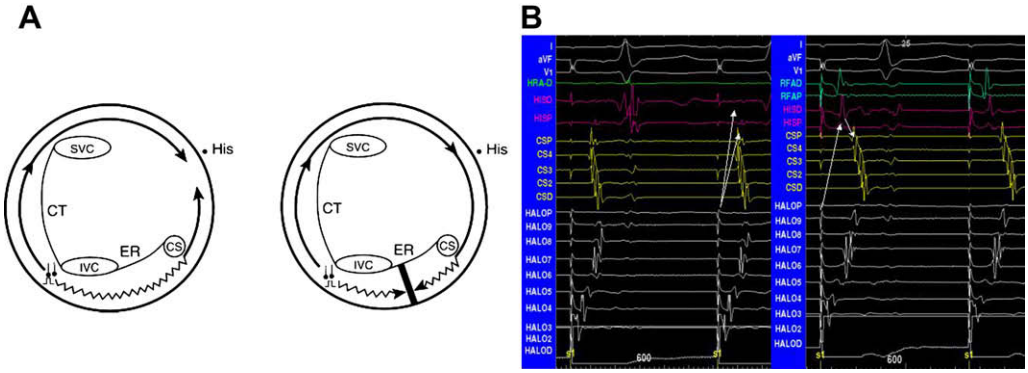

Fig. 6. (*A*) Schematic diagrams of the expected right atrial activation sequence during pacing in sinus rhythm from the low lateral right atrium before (*left panel*) and after (*right panel*) ablation of the cavo-tricuspid isthmus (CTI). Before ablation the activation pattern during coronary sinus pacing is caudal to cranial in the right atrial free wall, with collision of the cranial and caudal wavefronts in the mid-septum, with simultaneous activation at the His bundle (HISP) and proximal coronary sinus (CSP). Following ablation, the activation pattern during low lateral right atrial sinus pacing is still caudal to cranial in the right atrial free wall, but the septum is now activated in a strictly cranial to caudal pattern (ie, clockwise), indicating complete lateral to medial conduction block in the CTI. CT, crista terminalis; ER, Eustachian ridge; His, His bundle; SVC, superior vena cava; IVC, inferior vena cava. (*B*) Surface ECG and right atrial endocardial electrograms during pacing in sinus rhythm from the low lateral right atrium before (*left panel*) and after (*right panel*) ablation of the CTI. Tracings include surface ECG leads I, aVF, and V1, and endocardial electrograms from the proximal coronary sinus (CSP), His bundle (HIS), tricuspid valve annulus at 1 o'clock (HaloP) to 7 o'clock (HaloD), and high right atrium (HRA or RFA). Before ablation, during low lateral right atrial pacing, there is collision of the cranial and caudal right atrial wavefronts in the mid-septum (HIS and CSP). Following ablation, the septum is activated in a strictly cranial to caudal pattern (ie, clockwise), indicating complete lateral to medial conduction block in the CTI.

pacing from the coronary sinus ostium or low lateral right atrium respectively.[43–45] The presence of bidirectional conduction block in the CTI is also strongly supported by recording widely spaced double potentials at the site of linear ablation during pacing from the low lateral right atrium or coronary sinus ostium.[46,47] If ablation is done during sinus rhythm, pacing can be also done during energy application to monitor for the development of conduction block in the CTI. The use of this end point for ablation may be associated with a significantly lower recurrence rate of type 1 AFL during long-term follow-up.[43–45,48] Programmed stimulation and burst pacing should be repeated over the course of at least 30 minutes to ensure that bidirectional CTI block has been achieved, and that neither typical nor reverse typical AFL can be reinduced.[3,5–7,29,34–38,40–42,49]

If AFL is not terminated during the first attempt at CTI ablation, the activation sequence and isthmus dependence of the AFL should be reconfirmed, and then ablation should be repeated. During repeat ablation, it may be necessary to use a slightly higher power and/or ablation temperature, or to rotate the ablation catheter away from the initial line of energy application, either medially or laterally in the CTI, to create new or additional lines of block. In addition, if ablation is initially attempted using a standard 4- to 5-mm tip electrode and is not successful, repeat ablation with a larger-tip 8- to 10-mm electrode catheter or cooled-tip ablation catheter may produce better result.[29,37–42]

OUTCOMES AND COMPLICATIONS OF CATHETER ABLATION OF TYPE 1 ATRIAL FLUTTER

Early reports[3–6] of radiofrequency catheter ablation of AFL revealed high initial success rates but with recurrence rates up to 20% to 45% (**Table 1**). However, as experience with radiofrequency catheter ablation of AFL has increased, both acute success rates, defined as termination of AFL and bidirectional isthmus block, and chronic success rates, defined as no recurrence of type 1 atrial flutter, have risen to 85% to 95%. Contributing in large degree to these improved results has been the introduction of bidirectional conduction block in the CTI as an end point for successful radiofrequency catheter ablation of AFL.[29,34–42] In the most recent studies using either large-tip (8- to 10-mm) electrode ablation catheters with high-power radiofrequency generators, or cooled-tip electrode ablation catheters with standard radiofrequency generators, acute success rates as high as 100% and chronic success rates as high as 98% have been reported.[29,39,42] Randomized comparisons of internally cooled, externally cooled, and large-tip ablation catheters suggest a slightly better acute and chronic success rate with the externally cooled ablation catheters, compared with internally cooled ablation catheters or large-tip ablation catheters.[37,38,40,42,49]

In nearly all the large-scale studies where CTI ablation has successfully eliminated recurrence of type 1 AFL, and where quality-of-life scores (QOL) have been assessed, there have been statistically significant improvements in QOL as a result of reduced symptoms and antiarrhythmic medication use.[28,29,49]

Radiofrequency catheter ablation of the CTI for type 1 AFL is relatively safe, but serious complications can occur including heart block, cardiac perforation and tamponade, and thromboembolic events, which include pulmonary embolism and stroke. In recent large-scale studies, major complications have been observed in approximately 2.5% to 3.0% of patients.[29,42,49] In the studies of large-tip ablation electrode catheters there did not appear to be any relationship between complication rates and the use of higher power (ie, >50 W) for ablation of the CTI. Anticoagulation with warfarin before ablation must be considered in patients with chronic type 1 AFL to help decrease the risk of thromboembolic complications such as stroke.[50] This may be particularly important in those patients with depressed left ventricular function, mitral valve disease, and left atrial enlargement with spontaneous contrast (ie, smoke) on echocardiography. As an alternative, the use of transesophageal echocardiography to rule out left atrial clot before ablation may be acceptable, but subsequent anticoagulation with warfarin is still recommended, as atrial stunning may occur after conversion of AFL, as it does with atrial fibrillation.[50]

ROLE OF COMPUTERIZED THREE-DIMENSIONAL MAPPING IN DIAGNOSIS AND ABLATION OF TYPE 1 ATRIAL FLUTTER

While not required for successful ablation of type I atrial flutter, the three-dimensional (3-D) electroanatomical Carto (BioSense-Webster, Baldwin Park, CA) or noncontact Ensite (Endocardial Solutions, St. Paul, MN) activation mapping systems have specific advantages that have made them a widely used and accepted technology. Although it is not within the scope of this article to describe the technological basis of these systems in detail, there are unique characteristics of each system that make them more or less suitable for mapping and ablation of atrial flutter.

Table 1
Success rates for radiofrequency catheter ablation of atrial flutter

Author, Year, Reference No.	N	Electrode Length	% Acute Success	Follow-up, Mo	% Chronic Success
Feld 1992[5]	16	4	100	4 ± 2	83
Cosio1993[6]	9	4	100	2–18	56
Kirkorian 1994[35]	22	4	86	8 ± 13	84
Fischer 1995[34]	80	4	73	20 ± 8	81
Poty 1995[44]	12	6/8	100	9 ± 3	92
Schwartzman 1996[45]	35	8	100	1–21	92
Chauchemez 1996[48]	20	4	100	8 ± 2	80
Tsai 1999[41]	50	8	92	10 ± 5	100
Atiga2002[40]	59	4 versus cooled	88	13 ± 4	93
Scavee 2004[38]	80	8 versus cooled	80	15	98
Feld 2004[29]	169	8 or 10	93	6	97
Calkins 2004[49]	150	8	88	6	87
Ventura 2004[42]	130	8 versus cooled	100	14 ± 2	98
Feld 2008[53]	160	Cryoablation	87.5	6	80.3

Acute and chronic success rates are reported as overall results in randomized or comparison studies.
Abbreviations: N, number of patients studied, % acute success, termination of atrial flutter during ablation and/or demonstration of isthmus block following ablation; % chronic success, % of patients in whom type 1 atrial flutter did not recur during follow-up.

The Ensite system uses a saline inflated balloon catheter on which is mounted a wire mesh containing electrodes that are capable of sensing the voltage potential of surrounding atrial endocardium, without actual electrode-tissue contact, from which the computerized mapping system can generate up to 3000 virtual endocardial electrograms and create a propagation map of the macro-reentrant circuit. In addition, a low-amplitude high-frequency electrical current emitted from the ablation catheter can be sensed and tracked in 3-D space by the mapping balloon. A 3-D anatomy can be created by roving the mapping catheter around the right atrial endocardium, upon which the propagation map demonstrating the atrial flutter reentrant circuit is superimposed. The appropriate ablation target can then be localized, and the ablation catheter can be positioned and tracked while ablation is performed. Following ablation, the mapping system can then be used to assess for bidirectional CTI conduction block during pacing from the low lateral right atrium and coronary sinus ostium. The advantages of the Ensite system include the ability to map the entire AFL activation sequence in one beat, precise anatomic representation of the right atrium including the CTI and adjacent structures, precise localization of the ablation catheter within the right atrium, and propagation maps of endocardial activation during atrial flutter and pacing after ablation to assess for CTI conduction block. In addition, any ablation catheter system can be used with the Ensite system. The major disadvantages of the Ensite system are the need to use the balloon mapping catheter, with its large 10-Fr introducer sheath, and the need for full anticoagulation during the mapping procedure.

The Carto uses a magnetic sensor in the ablation catheter, a magnetic field generated by a grid placed under the patient, and a reference pad on the skin to track the ablation catheter in 3-D space. The computer system sequentially records anatomic location and electrograms for online analysis of activation time and computation of isochronal patterns that are then superimposed on the endocardial geometry (**Fig. 7**A). A propagation map can also be produced. The advantages of the Carto include precise anatomic representation of the right atrium including the CTI and adjacent structures, precise localization of the ablation catheter within the right atrium, and static activation and propagation maps of endocardial activation can be constructed during atrial flutter and during pacing after ablation to assess for CTI conduction block (**Fig. 7**B). The disadvantages of the Carto system include the need to use the proprietary catheters and ablation generator and the need for sustained tachycardia to map the entire endocardial activation sequence.

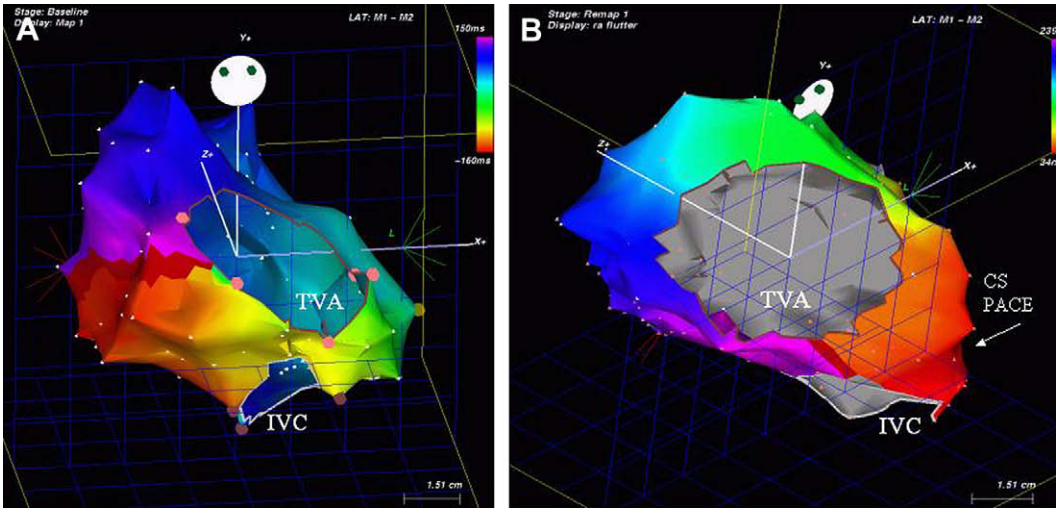

Fig. 7. A 3-D electroanatomical (Carto, Biosense Webster) map of the right atrium in a patient with typical AFL, before (*A*) and after (*B*) CTI ablation. Note the counterclockwise activation pattern around the tricuspid valve during AFL (*A*), which is based on color scheme indicating activation time from orange (early) to purple (late). Following ablation of the CTI (*B*), during pacing from the coronary sinus ostium, there is evidence of medial to lateral isthmus block as indicated by juxtaposition of orange and purple color in the CTI, indicating early and late activation, respectively. A 3-D propagation map can also be produced using the Carto system, which in some cases allows better visualization of the atrial activation sequence during AFL. IVC, inferior vena cava; TVA, tricuspid valve annulus.

The 3-D computerized mapping systems may be particularly useful in difficult cases such as those where prior ablation has failed, or in those where complex anatomy may be involved including idiopathic or postoperative scarring, or unoperated or surgically corrected congenital heart disease.

ALTERNATIVE ENERGY SOURCES FOR ABLATION OF TYPE 1 ATRIAL FLUTTER

The development of new energy sources for ablation of cardiac arrhythmias is an ongoing effort because of the disadvantages of radiofrequency energy for ablation, including the risk of coagulum formation, tissue charring, subendocardial steam pops, embolization, failure to achieve transmural ablation, and long procedure and fluoroscopy times required to ablate large areas of myocardium. Many of these disadvantages have been overcome in the case of ablation of type 1 AFL in the past decade. Nonetheless, several clinical and preclinical studies have recently been published on the use of catheter cryoablation and microwave ablation for treatment of atrial flutter and other arrhythmias.[51–57] Recent studies have been reported demonstrating that catheter cryoablation of type 1 AFL can be achieved with similar results to that achieved with radiofrequency ablation.[51–53] The potential advantages of cryoablation include the lack of pain associated with ablation, the ability to produce a large transmural ablation lesion, and the lack of tissue charring or coagulum formation. In addition, early work has begun on the use of a linear microwave ablation catheter system (Medwaves, San Diego, CA) with antenna lengths up to 4 cm.[54–57] These studies have shown the feasibility of linear microwave ablation, which may have the advantage of very rapid ablation of the CTI with a single energy application over the entire length of the ablation electrode.

SUMMARY

Radiofrequency catheter ablation has become a first-line treatment for type 1 AFL with nearly uniform acute and chronic success and low complication rates. The most effective approach preferred by most laboratories is combined anatomically and electrophysiologically guided ablation of the CTI, with procedure end points of arrhythmia noninducibility and bidirectional CTI conduction block. Currently, the use of a large-tip 8- to 10-mm ablation catheter with a high output radiofrequency generator (ie, up to 100 W) or a cooled-tip ablation catheter is recommended for optimal success rates. Computerized 3-D activation mapping is an adjunctive method, which while not mandatory, may have significant advantages in some cases resulting in improved overall

success rates. New alternate energy sources including cryoablation and microwave ablation are under investigation with the hope of further improving procedure times and success rates and potentially reducing the risk of complications during AFL ablation.

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