Contemporary Diagnosis and Management of Atrial Flutter

A Continuum of Atrial Fibrillation and Vice Versa?

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Abstract: Atrial flutter (AFlu) is usually a fast (>240 bpm) and regular right atrial macroreentrant tachycardia, with a constrained critical region of the reentry circuit located at the cavotricuspid isthmus (CTI; typical CTI-dependent AFlu). However, a variety of right and left atrial tachycardias, resulting from different mechanisms, can also present as AFlu (atypical non-CTI-dependent AFlu). The electrocardiogram can provide clues to its origin and location; however, additional entrainment and more sophisticated electroanatomical mapping techniques may be required to identify its mechanism, location, and target area for a successful ablation. Although atrial fibrillation and AFlu are 2 separate arrhythmias, they often coexist before and after drug and/or ablation therapies. Indeed, there appears to be a close interrelationship between these 2 arrhythmias, and one may "transform" into the other. These issues are discussed in this overview, and practical algorithms are proposed to guide AFlu localization and illustrate the AFlu and atrial fibrillation continuum.

Key Words: atrial flutter, atrial fibrillation, cavotricuspid isthmus, antiarrhythmic drugs, thromboembolism, stroke, anticoagulation, catheter ablation, tachycardiomyopathy

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Atrial flutter (AFlu) is usually a right atrial macroreentry, typically cavotricuspid isthmus (CTI)-dependent reentry, but a variety of atrial tachycardias originating from the right atrium (RA), as well as the left atrium (LA), resulting from different mechanisms, can also present as AFlu (non-CTI-dependent AFlu).[1] It is commonly ascribed to reentry around anatomic obstacles, such as the great veins or pulmonary veins (PVs), around atrial lesions and scars, and around functional obstacles. The incidence of AFlu is age-dependent, like atrial fibrillation (AF), albeit much less common, with 5/100,000 in patients <50 years and about 600/100,000 in patients >80 years of age, with an overall incidence of ≈88/100,000 person-years.[2] Concomitant structural heart disease or pulmonary disease further increases the risk of AFlu.

AFlu is a fast and regular atrial tachycardia, initially and arbitrarily defined by an atrial rate >240 bpm, except in the presence of class I or III antiarrhythmic drugs, which can considerably lower the atrial rate of AFlu. Various classifications have been proposed,[3] with the most frequent form of AFlu called *common* when negative flutter (F) waves with a sawtooth pattern and lacking isoelectric baseline are seen in the inferior leads and an apparent upright F wave is seen in lead V1 (typical or CTI-dependent AFlu); all other forms are designated as *atypical* AFlu. Thus, it is not just the rate but also the morphology of F waves that determine the presence of AFlu. The most frequent type is *counterclockwise typical CTI-dependent AFlu*, with the sawtooth pattern (Fig. 1), its mechanism being macroreentry confined in the RA, with an ascending atrial impulse along the septum and descending wavefront in the lateral wall with passive activation of the LA. In approximately 10%–30% of typical flutters, impulse propagation within the same reentrant circuit is reversed, rotating in a clockwise direction around the tricuspid valve when visualizing a left anterior oblique view, displaying positive F waves in the inferior leads and a negative and frequently bifid F wave in V1 (*clockwise CTI-dependent AFlu*). The LA is activated predominantly over the coronary sinus (CS) in counterclockwise AFlu and over the Bachmann bundle during clockwise AFlu, probably explaining the difference in morphology of the F wave on surface ECG.[4]

CTI-independent AFlu is encountered both in the RA and LA.[5,6] A CTI-independent right AFlu can be counterclockwise or clockwise and can have similar to the typical (CTI-dependent) clockwise AFlu electrocardiographic (ECG) appearance, albeit with faster atrial rate. Its circuit is often located in the upper portion of the RA, crossing the superior portion of the crista terminalis where ablation is often successful in eliminating the circuit. Other nonscar- or scar-related right AFlus have also been described. A negative F wave in V1 characterizes the RA origin of the arrhythmia. LA flutters, less frequent than right AFlus, are described later in this review.

The atrial rate of AFlu is typically between 250 and 350 bpm. The usual ventricular response in AFlu is ≈150 beats per minute (bpm) due to 2:1 atrioventricular (AV) conduction (Fig. 1). Hence, any supraventricular tachycardia with a rate ≈150 bpm may be AFlu.[7] Another clue may be offered when one looks at the undulation of the isoelectric line by ignoring or eliminating from one's visual field all the QRS complexes; then the sawtooth pattern of the F waves may become or perceived as more apparent and clear. Vagal maneuvers and/or administration of adenosine will aid in the diagnosis by disclosing apparent F waves (Fig. 1). In the presence of an antegradely conducting accessory pathway, AFlu with ensuing rapid ventricular rate may degenerate into ventricular fibrillation.[7] Also, AFlu patients receiving class IA or IC antiarrhythmic drugs without preadministration of rate control drug therapy (such as with the use of AV-node blocking agents) may develop rapid ventricular rates with 1:1 AV conduction because of the atrial rate slowing conferred by IA/IC agents, which facilitates rapid AV conduction.[8] More rarely, rapid AFlu may be observed even in patients not receiving antiarrhythmic drugs; these are usually patients with enhanced AV-nodal conduction.[9]

Patients with AFlu may present with symptoms of palpitations, fatigability, and reduced exercise capacity, chest pain, or dyspnea, presyncope, or syncope. The risk of thromboembolism is somewhat similar to that of AF[10–12]; therefore, the same antithrombotic prophylaxis is required in AFlu patients. Acutely symptomatic cases may be managed similar to that of AF, with cardioversion or pharmacologic rate control to relieve symptoms. Currently, there are no separate guidelines specific to AFlu. Prior and existing guidelines have been incorporated into the AF guidelines.[11] Investigators have also been combining these 2 akin arrhythmias as one group when presenting

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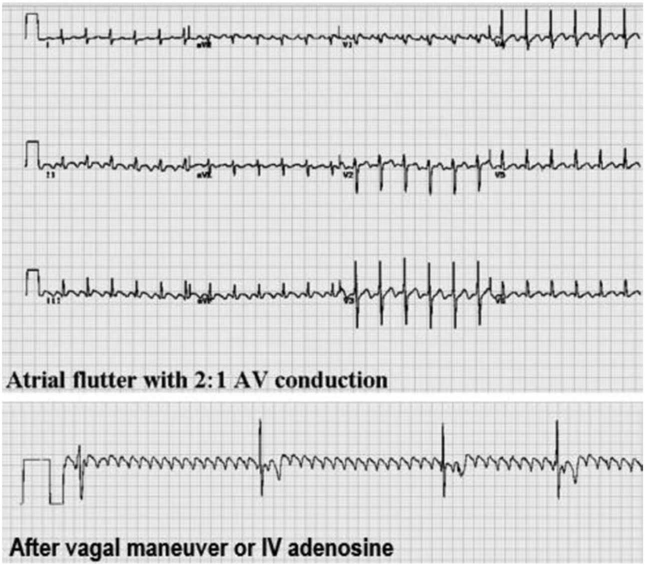

FIGURE 1. Typical atrial flutter displays negative, saw-tooth flutter (F) waves in the inferior leads (II, III, aVF); however, when the ventricular rate is rapid as in 2:1 atrioventricular (AV) conduction, one may need to perform vagal maneuvers or administer intravenous (IV) adenosine to reduce AV conduction and disclose the F waves.

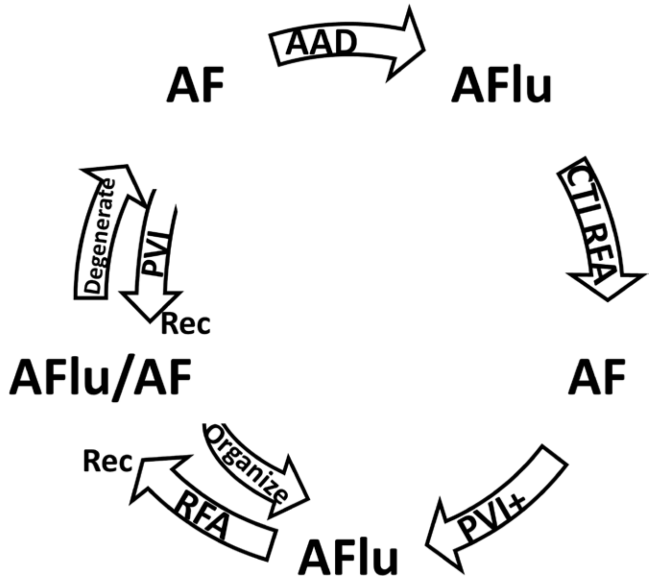

FIGURE 2. Graphic illustrates the close interrelationship between atrial fibrillation (AF) and atrial flutter (AFlu). These 2 arrhythmias often co-exist or one "transforms" into the other (see text for discussion). AAD indicates antiarrhythmic drugs; CTI, cavotricuspid isthmus; CTI-RFA, cavotricuspid isthmus radiofrequency ablation; PVI, pulmonary vein isolation; PVI+, PVI plus substrate ablation; Rec, recurrence; RFA, radiofrequency ablation.

population data.[13–15] Long-term management, however, may differ in a couple of aspects. Rate control may be more difficult than AF, whereas ablation efficacy is much higher and constitutes a first-line approach in the majority of AFlu patients.[11] A recent survey highlighted some variability in the management of AFlu but indicated a reasonable consensus regarding oral anticoagulation and the desired end points of ablation for patients with CTI-dependent AFlu.[16]

Although AF and AFlu are 2 separate arrhythmias, they often coexist before and after drug or ablation therapies (Fig. 2). Indeed, there appears to be a close interrelationship between AF and AFlu.[17] For example, patients receiving antiarrhythmic drugs (class IA, IC, and III) for AF may develop organized atrial tachyarrhythmia similar to typical CTI-dependent AFlu, amenable to CTI ablation.[18,19] On the other hand, some AF patients submitted to PV isolation may also present at a later time with typical CTI-dependent AFlu. Some investigators even believe that CTI-dependent AFlu almost always develops from antecedent AF of variable duration.[20,21] However, there have been plenty of cases where AFlu occurs in the absence of antecedent AF, whereas AFlu ablation appears to cure this arrhythmia in a significant proportion of patients without the subsequent development of new incident AF. Nevertheless, a plethora of cases show that these 2 arrhythmias co-exist and/or one "transforms" into the other.[10] There is evidence that advanced age, female gender, diabetes, and hypertension seem to predispose patients with AFlu to subsequently develop AF.[10] These inter-relationships will be further examined in this review.

TYPICAL CTI-DEPENDENT ATRIAL FLUTTER

Typical CTI-dependent AFlu is the most common atrial macroreentry tachycardia, with the reentry circuit located in the RA, as described earlier, with both counterclockwise and clockwise patterns. With regard to diagnosis, one must be cognizant of the occurrence of a pseudo-AFlu ECG appearance produced by Parkinsonian tremor and/or other artifacts.[22]

Similar to AF, albeit rarer, AFlu-related tachycardiomyopathy may occur and is a treatable and reversible cause of heart failure.[23,24] In addition to drug or electrical cardioversion, AFlu is also amenable to overdrive atrial pacing,[25,26] avoiding potential drug side effects (eg, torsade-de-pointes with the use of ibutilide) or obviating the need for deep sedation for performing electrical cardioversion.

CTI Ablation

AFlu ablation has been increasingly offered as first-line therapy[11] and has been safely performed over the last decades, with relatively low complication rates (0.5%–4%), albeit with a significant association reported between low hospital procedural volume (<50 procedures per year) and increased adverse outcomes.[27,28] All-cause mortality has been reported in ≈0.2%–0.6%. Single-procedure success has been >90%, whereas multiple-procedure success is as high as 97%.[28] Arrhythmia recurrences have been noted in ≈10%–15% of patients, with repeat ablation performed in 8%–10%. Although most studies attest to the favorable course of AFlu patients undergoing ablation, there is an observational nationwide cohort study that indicated that patients with AFlu had a significantly higher all-cause mortality rate compared with those with AF after an ablation procedure, but similar thromboembolic event rates.[29] However, these discrepant results compared to other studies have been attributed to the higher rate of comorbidities and lesser usage of oral anticoagulants in this study. It is, therefore, critical to provide AFlu patients with a similar antithrombotic prophylaxis as AF patients.

Linear radiofrequency ablation along the CTI aiming to produce *bidirectional CTI block*[30–32] is the standard ablation technique that has been adopted (Fig. 3); this was preceded by a period when limited focal ablation was performed, which though was associated with higher recurrence rates.[33] CTI ablation is performed with the use of 4-mm or 8-mm or irrigated-tip electrode radiofrequency catheters or less often with cryocatheters.[34] The use of a circular (Halo) multipolar catheter

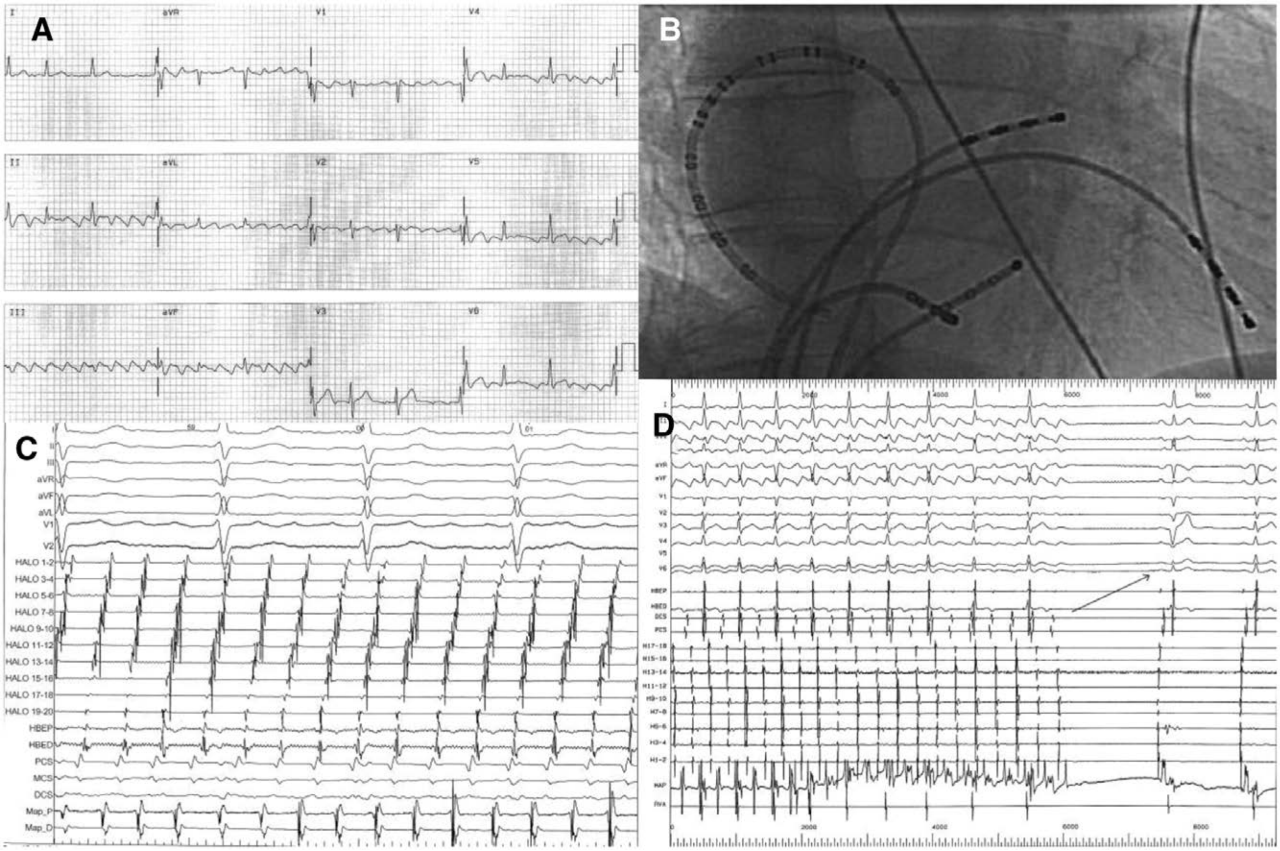

FIGURE 3. A, A 12-lead electrocardiogram of common (counterclockwise) atrial flutter (AFlu) with negative saw-tooth flutter (F) waves depicted in the inferior leads. Use of an eicosapolar (Halo) catheter (B) helped to illustrate caudocranial septal and craniocaudal right atrial activation of the AFlu, with a cycle length of 240 ms (rate 250 bpm; C). Atrial flutter ended during radiofrequency (RF) current application (D, arrow) delivered via a steerable 7F quadripolar 4-mm-tip thermistor RF ablation catheter used to make a line of RF lesions along the cavotricuspid isthmus (CTI) extending from the tricuspid annulus to the inferior vena cava until bidirectional CTI conduction block could be demonstrated (not shown).

can aid in identifying the (counterclockwise or clockwise) direction of the reentry circuit of AFlu (Figs. 3 and 4) and also assist in swift confirmation of bidirectional block at the end of the procedure. Ablation can be performed during sinus rhythm, as long as a bidirectional CTI block is produced, but also during AFlu, which is terminated when CTI block is achieved (Figs. 3 and 4); nevertheless, even in this case, a bidirectional block should still be confirmed with pacing maneuvers. This is done by observing sequential activation mapping at both sides of the ablation line during pacing at the proximal CS and at the anteroinferior RA. Complete bidirectional CTI block includes craniocaudal atrial activation sequence along the Halo catheter during proximal CS pacing (clockwise block), whereby the distal bipole of the Halo catheter is depolarized later than more proximal bipoles and depolarization of the proximal CS area recorded later than that at the His bundle region during anteroinferior RA pacing (counterclockwise CTI block).[35]

Although CTI ablation is easily performed in the majority of AFlu patients, 5%–10% of cases pose a significant challenge, needing prolonged radiofrequency applications and long procedure times and/or a second ablation session at a later time, probably due to anatomic CTI variations.[36] If an initial ablation line fails to achieve complete isthmus block, an electrogram mapping technique may be used searching for single or narrowly split double atrial electrograms to be targeted along the CTI.[37] Occasionally, one may need to extend the ablation line to the CS ostium and/or inside the proximal CS by targeting fragmented atrial potentials to obtain successful ablation.[38] Also, a long or steerable sheath can provide greater catheter stability and allow proper energy delivery to areas that are difficult to reach. The use of 8-mm-tip catheters and high energy (70 watts) delivery have been associated with a high success rate and minimal ablation times.[39] CTI length, but not morphology, influences the acute success rate of ablation of CTI-dependent AFlu, irrespective of energy source.[40]

ATYPICAL ATRIAL FLUTTERS

CTI-independent AFlus are encountered both in the RA and LA.[5,6] Some types of right AFlu were described earlier. *Left AFlus* are less frequent than RA flutters and are commonly associated with significant underlying structural heart disease, including mitral valve disease, hypertension, or heart failure, but in a few cases, they may be encountered even in the absence of detectable left ventricular heart disease.[1] Other types comprise postintervention LA flutters observed after PV isolation, but especially after extensive LA substrate ablation, including linear lesions and/or ablation of areas with complex fractionated atrial electrograms. Many of these tachycardias are reentrant and related to gaps in prior ablation lines, with perimitral, roof-dependent, and septal macroreentrant circuits.[41,42] PV-related atrial tachycardias manifesting with an AFlu ECG appearance should also be considered and are easier to ablate with reisolation of the PVs[43];

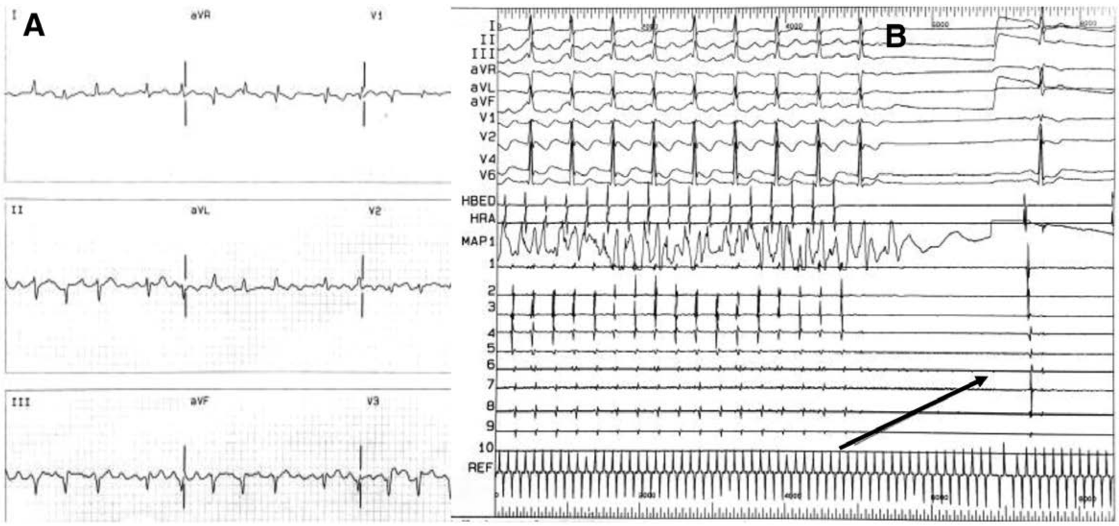

FIGURE 4. Clockwise atrial flutter (AFlu) uses the same circuit as the typical counterclockwise AFlu; however, the impulse is moving in a reversed direction, and thus, it displays positive F waves in the inferior leads and bifid F waves in V1 (A). This type of AFlu is also amenable to cavotricuspid isthmus (CTI) ablation (CTI-dependent AFlu). B, The interruption of AFlu and restoration of sinus rhythm (arrow) when the last radiofrequency energy application completed CTI block.

these tachycardias may be due to microreentry using gaps in the line surrounding the PVs or possibly to focal PV discharges.[44]

Postsurgical Atrial Flutter

This type of atypical AFlu is a macroreentrant atrial tachycardia that develops several years after surgery. It relates to scars at sites of surgical incisions and usually occurs in the setting of prior surgical repair of a congenital heart disease, such as atrial septal defect, tetralogy of Fallot, D-transposition (Mustard or Senning repair), or single ventricle (Fontan repair).[45] However, even in these populations, CTI-dependent AFlu is still the most common type of AFlu.[46] Reentry around the atriotomy incisions may co-exist or may be the predominant arrhythmia in many of these patients, as well. After ablation of the typical AFlu, the atypical AFlu may emerge, requiring additional ablation in other areas or isthmuses of complex circuits that need electroanatomical mapping techniques for better localization.[47–49] It is, thus, crucial during ablation to monitor for changes in AFlu wave morphology and rate, which may be subtle and go unnoticed, as well as for any change in endocardial activation pattern to discern whether the circuit has relocated. Finally, atriotomies or cannulation sites during mitral valve surgery may also contribute to postoperative incisional macroreentrant atrial tachycardias (atypical left AFlus).[50]

ECG Diagnosis

A proposed ECG algorithm to help discern the different types of AFlu is presented in Figure 5. As detailed earlier, common AFlu, in its most frequent form of a counterclockwise pattern, is characterized by negative F waves in the inferior leads with a sawtooth pattern and apparent upright F waves in V1 (Figs. 1 and 3). In rapid AFlu, it may be difficult to discern the F waves, and one may need to resort to vagal maneuvers and/or administration of adenosine (Fig. 1). The less frequent clockwise form displays positive F waves in the inferior leads and a negative and frequently bifid F wave in V1 (Fig. 4). In left AFlu, the F wave usually shows a prominent positive deflection in V1 and uncommonly is flat or isoelectric (Fig. 6) and, thus, difficult to discern from the counterclockwise typical flutter. Nevertheless, a broad upright F wave in V1 with upright waves in the inferior leads or with low amplitude or isoelectric waves in the inferior and other leads is suggestive of an LA origin.[1,51] A negative component in lead I, when present, can differentiate counterclockwise perimitral flutter from left PV atrial tachycardias. Broad P waves and isoelectric lines between atrial waves help to distinguish a focal mechanism from a macroreentrant mechanism.[44]

Entrainment Mapping/Electroanatomical Mapping

Entrainment involves rapid (overdrive) pacing, whereby capture of the reentrant circuit of a tachycardia is effected (by pacing slightly faster than the tachycardia rate) without interrupting the tachycardia (ie, with cessation of pacing, the spontaneous reentrant tachycardia continues on).[52]

Entrainment mapping is a standard pacing technique that allows the electrophysiologist to establish reentry as the mechanism of a particular arrhythmia, as opposed to automaticity or triggered activity, and most importantly to identify a putative target for successful ablation. Entrainment maneuvers that can be performed to define the pathway of the arrhythmia may include the response of the return cycle upon cessation of entrainment (postpacing interval). A site within the circuit is determined when the postpacing interval is almost equal (within 30 ms) to the tachycardia cycle length; if outside the circuit, the return cycle would be longer than the tachycardia cycle length. Furthermore, the morphology of the paced ECG complex can be compared to the arrhythmia's ECG complex to determine whether the area (from which entrainment is attained) is within a critical conducting channel or isthmus. This would be relevant because such an area is typically a good target for ablation. Thus, entrainment of the AFlu achieved within the CTI with a postpacing interval close to the flutter cycle length determines a CTI-dependent AFlu. If not, other areas of non-CTI-dependent AFlu in the RA or LA should be sought for testing, a search that can be initially and grossly guided by the ECG morphology of the F waves (Fig. 5).

Entrainment mapping from the CS points to the left site of origin of an AFlu, whereas entrainment effected from the lateral RA wall readily distinguishes a right site of origin, a maneuver that can obviate the need for unnecessary transseptal puncture. As detailed earlier, in cases of co-existing typical and atypical AFlu, one has to be vigilant for subtle changes in AFlu wave morphology and rate, as well as in the endocardial activation pattern, to discern relocated circuits and isthmuses, which may further need to be confirmed

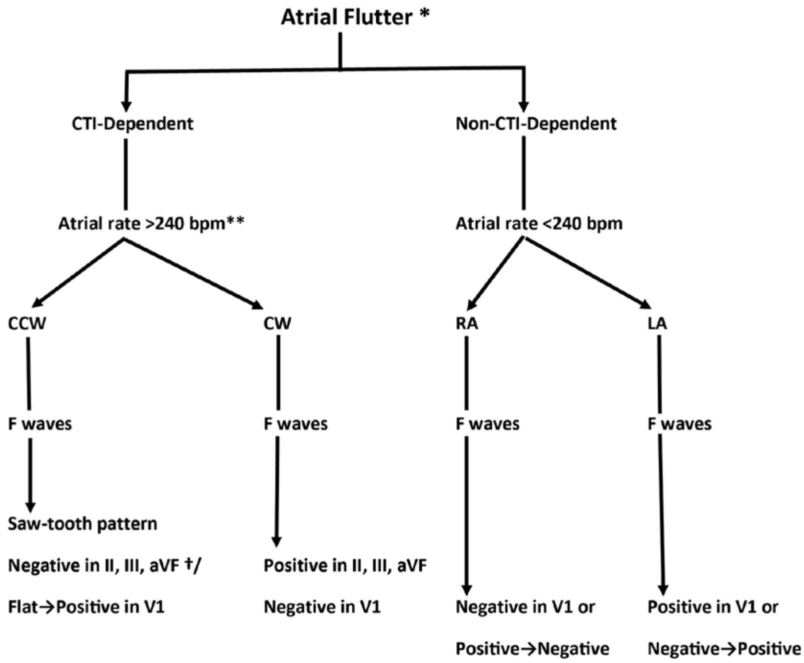

FIGURE 5. An algorithm is presented, which may assist in identifying the type and origin of atrial flutter (AFlu) by examining the atrial rate and the morphology of flutter (F) waves on surface ECG, before resorting to entrainment or electroanatomical mapping. Key ECG leads include the inferior leads and lead V1. N.B.: In patients with prior pulmonary vein isolation presenting with CTI-dependent CCW AFlu, the F waves in the inferior leads may be upright in as many as 60% of these patients, attributed to altered left atrial activation. CCW indicates counterclockwise; CL, cycle length; CTI, cavotricuspid isthmus; CW, clockwise; LA, left atrial; RA, right atrial. *CTI-dependency confirmation at electrophysiology study by entrainment mapping. **In presence of antiarrhythmic drugs, the atrial rate may be quite lower. †May be positive in patients with prior LA ablation.

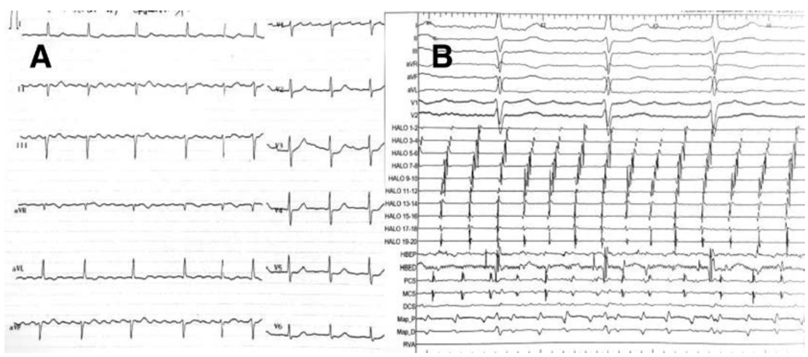

FIGURE 6. An atypical left atrial flutter is displayed with positive F waves in the inferior leads and positive F waves in V1 (A), which is difficult to discern by ECG alone and is confirmed by intracardiac recordings (noted earlier in the coronary sinus leads; B) and electrophysiological maneuvers (entrainment) or electroanatomical mapping (not shown).

by repeating entrainment techniques. Also, periodically repeating entrainment if an AFlu has not terminated despite completion of a CTI line of ablation will help determine whether such a change has already occurred and has gone unnoticed. Alternatively, one may interrupt AFlu by overdrive pacing and determine if bidirectional CTI block has already been accomplished, in which case, an alternate critical isthmus site will need to be sought.

Finally, due to limitations of the surface ECG and even of the entrainment mapping techniques,[53] the development of high-resolution electroanatomical mapping systems (Fig. 7) has greatly facilitated more precise anatomic localization of the reentrant circuit or site of tachycardia origin and can guide successful ablation, albeit at an increased cost.[1,54,55] Such an approach appears more useful in cases of atypical, non-isthmus-dependent or scar-related atrial tachycardias and AFlu.[56,57] An added advantage of these systems is their ability to significantly curtail radiation exposure during the ablation procedure.[54] It has even been suggested that it may be feasible to achieve a zero fluoroscopy approach to CTI ablation using the newer generation electroanatomical mapping systems.[58,59]

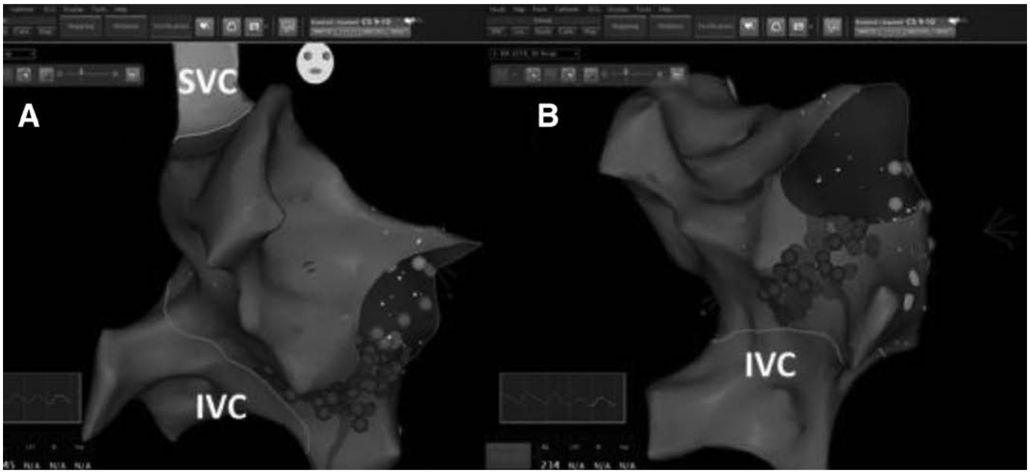

FIGURE 7. An example of electroanatomical mapping (postablation) is displayed in right (A) and left (B) anterior oblique views, a technique that can facilitate localization of the reentrant circuit and guide ablation. In this case, it was used in a typical AFlu case; however, it is usually more necessary and helpful in atypical atrial flutter cases (see text for discussion). Cluster of dots above the IVC indicate the radiofrequency current applications delivered at the cavotricuspid isthmus (CTI) with successful ablation of the CTI by creating bidirectional block in this region. Dots at the top indicate the location of the His bundle. Dots just below the His were used to identify the tricuspid annulus. IVC indicates inferior vena cava; SVC, superior vena cava. Adapted with permission from Rhythmos. 2017;121:12–14.

Ablation

PV flutters emanating from reconnected PVs, either from local foci within the PVs or reentry around the gaps of prior PV isolation, are easily ablatable by reisolating the PVs. Other AFlu types require ablation lines in the slow-conduction zones or critical isthmuses, which are not that easily identified with the assistance of various techniques, including entrainment and electroanatomical mapping.[53] A specific type of such a postinterventional atrial tachycardia is perimitral AFlu, which is a challenging arrhythmia, most often occurring after ablation of AF, and considered a form of proarrhythmia of extensive ablation.[60] As this type of AFlu may resolve spontaneously, it has been suggested to defer ablation for several months after the index procedure. Ablation of perimitral AFlu is a demanding procedure, often requiring an epicardial approach via the CS to achieve mitral isthmus block.[61] Even when such a block is attained, recurrences are frequent (≈40%), and a mean of 2 procedures per patient will usually be needed, a far more difficult situation than with CTI-dependent AFlu. Furthermore, arrhythmia recurrences in patients developing perimitral AFlu after ablation for longstanding persistent AF may be related to PV and non-PV triggers of arrhythmia, and targeting these foci is more effective than mitral isthmus ablation. In general, for non-CTI-dependent atypical AFlu, catheter ablation aims at transecting a critical part of the macroreentrant circuit that will render the tachycardia noninducible. Typically, 2 scar or fibrotic areas are connected using a linear lesion that traverses through the critical isthmus.

ATRIAL FIBRILLATION AFTER ATRIAL FLUTTER ABLATION

New-onset AF develops in a significant percentage (20%–40%) of patients undergoing CTI ablation for AFlu, which should be factored in when deciding to withhold anticoagulation therapy. Furthermore, PV isolation may be considered in conjunction with CTI ablation, particularly in patients with previously documented AF. Among 364 consecutive patients undergoing successful CTI ablation, 230 patients had AFlu only (AFlu group) and 134 patients had AFlu and a prior documented AF (AFlu/AF group).[62] Over a mean follow-up of 22 ± 20 months, 71% of patients in the AFlu group and 50% patients in the AFlu/AF group remained free of recurrent arrhythmia ($P < 0.001$). AF developed in 22% of patients in the AFlu group and in 43% in the AFlu/AF group ($P < 0.001$).

According to a review of 10 studies comprising 1299 patients undergoing ablation of CTI-dependent AFlu, 31% of patients developed AF during AFlu ablation.[63] Over a period of 23 ± 7.6 months, 29% of patients had new-onset AF; 47% of patients in the group had inducible AF during the ablation procedure versus 21% in the noninducible group. The odds ratio for developing AF after AFlu ablation in patients with AF inducibility was 3.72.

A recent systematic review and meta-analysis of 48 studies comprising 8257 AFlu patients undergoing ablation found an incidence of new-onset AF of 29% over a mean of 2.5 years, correlated with follow-up duration.[64] In another study among 80 AFlu patients with no history of prior AF undergoing successful ablation, 22 patients (27.5%) developed new-onset AF over 4.1 ± 2.5 years.[65] Prolonged (>120 ms) intra-atrial conduction time predicted new-onset AF after ablation in this group. Similarly, in another study, advanced inter-atrial block (P wave ≥120 ms with biphasic morphology in the inferior leads) was a key predictor for high risk of new-onset AF after a successful CTI ablation in patients with typical AFlu.[66] In a cohort of 96 AFlu patients having CTI ablation, early AF occurred in 17%, mainly in those with prior AF, low ejection fraction, or significant mitral regurgitation.[34]

In other studies where patients with both AFlu and paroxysmal AF underwent either sequential procedures (CTI ablation and subsequent PV isolation after 6 weeks) or PV isolation alone, AF recurrences occurred more frequently in the combined procedure group.[67] The authors considered AFlu as a sign that non-PV triggers may be the culprit behind AF or that sufficient electrical remodeling has already occurred in both atria, and thus, a strategy that includes substrate modification may be required in patients with both arrhythmias.

Thus, despite a low AFlu recurrence rate, patients with AFlu who undergo successful ablation are by no means free from embolic complications during long-term follow-up, mainly due to a high rate of AF development. Oral anticoagulation will need to be continued, especially in those patients with underlying stroke risk factors (high CHA_2DS_2-VASc score).[68] Hence, an approach similar to that for AF is recommended for AFlu patients in this matter, albeit for a different reason.

Some studies have tested the hypothesis that preventive PV isolation at the time of AFlu ablation in patients who have not had AF will reduce a future incidence of AF[69,70]; however, long-term data showing any benefit of a combined ablation are lacking. Furthermore, such an approach may incur both higher risk and cost[71] to which many patients who may never develop subsequent AF will be exposed unnecessarily. In AFlu patients with prior history of AF, PV isolation may be performed either during the same ablation session or at a subsequent procedure; however, some prefer the sequential approach as AFlu may constitute the trigger for AF development, and when this trigger is eliminated, AF may not recur in a considerable proportion (≈50%) of patients.[62] Indeed, the sequential approach to ablation in patients with both arrhythmias may be a more prudent strategy as many patients after initial AFlu ablation may not develop AF again, thus, obviating the need for a more complex and demanding PV isolation procedure with its attendant risks and cost. During a follow-up of ≈1.4 years of 343 AFlu patients having a successful CTI ablation, AF occurrence was significantly reduced after AFlu ablation (33%) as compared with the occurrence of AF before ablation (55%; $P < 0.001$).[72] Some have even suggested that PV triggers play an important role in AFlu occurrence and have performed PV isolation in a small group of AFlu patients, observing a reduced recurrence of AFlu, even without CTI ablation.[73]

Thus, prophylactic PV isolation should not be performed in patients with AFlu alone whether or not they are undergoing CTI ablation, and neither should prophylactic CTI ablation be performed in patients with AF alone undergoing PV isolation. Ablation of the CTI during the PV isolation procedure may be recommended in patients with a history of typical AFlu or inducible CTI-dependent AFlu.[74]

In our series of 45 patients with AFlu undergoing ablation (CTI ablation in 43), 9 (20%) subsequently developed AF that required PV isolation (Table 1). In these patients, AFlu was the clinical arrhythmia before the index procedure.

TABLE 1. Types of Atrial Flutter in 2 Groups of Patients Undergoing Ablation

	Atrial Flutter	AF
Patients	45	67
Men/women	32/13	44/23
Age (years)	58.4 ± 11.5	56.1 ± 10.9
CTI RFA	43	5
PVI	9	67*
CCW	36 (80%)	
CW	1	
Both (CCW + CW)	6	
Atypical	2	
PAF/Pers AF		58/9
AF after CTI RFA	9 (20%)	
AFlu after PVI		6 (9%)
CCW		5
LAFlu		1

AF indicates atrial fibrillation; AFlu, atrial flutter; CCW, counterclockwise; CTI, cavotricuspid isthmus; CW, clockwise; LAFlu, left atrial flutter; PAF, paroxysmal atrial fibrillation; Pers AF, persistent atrial fibrillation; PVI, pulmonary vein isolation; RFA, radiofrequency ablation.

*Twenty-two with standard point-by-point technique/18 with use of a circular catheter/27 with cryoballoon ablation.

ATRIAL FLUTTER AFTER ATRIAL FIBRILLATION ABLATION

If AFlu was present before AF ablation, one should expect AFlu to recur after ablation, unless CTI ablation has been concurrently performed. Hence, there is a recommendation for performance of both procedures (PV isolation and CTI ablation) in this group of patients during the same session, although the sequential approach with CTI ablation first, followed by PV isolation if AF recurs, seems a more plausible strategy, as detailed earlier. However, there are still cases whereby AFlu presents in patients who have not had AFlu before, and this arrhythmia presents for the first time after AF ablation. Whether AFlu occurrence after AF ablation is also associated with the recurrence of AF remains a moot point. Some investigators considered that the occurrence of AFlu after PV isolation may be more frequent in patients with extrapulmonary triggers for AF; however, this remains speculative.

In cases of newly emerging AFlu after PV isolation, one has to be cognizant of PV-related LA flutters,[43] whereas in those with more extensive LA substrate ablation, LA tachycardias mimicking AFlu may be the underlying mechanism of the apparent AFlu.[41] The ECG may be limited in rendering a more precise diagnosis, which can only be confirmed via an electrophysiology study and high-resolution electroanatomical mapping. Nevertheless, some have suggested certain ECG characteristics to aid in differential diagnosis, for example, between focal PV atrial tachycardia due to reconnected PVs and perimitral AFlu.[75] Patients with perimitral flutter have a faster atrial rate than those with focal atrial tachycardias; counterclockwise mitral flutter has positive F waves in the inferior and precordial leads and a negative component in leads I and aVL, whereas clockwise mitral flutter demonstrates the converse limb-lead morphology with a significant negative F wave in the inferior leads and positive F wave in leads I and aVL. A negative component in lead I, when present, may be best at differentiating counterclockwise flutter from left PV-atrial tachycardias, whereas a positive F wave in lead I is best at differentiating clockwise left AFlu from typical counterclockwise right AFlu.

The reported incidence of subsequent atrial tachycardias varies according to the type of LA ablation. The less is the extent of LA ablation, the lower is the incidence of LA tachycardia. When ablation is limited to PV isolation alone, a low occurrence of atrial tachycardias of 1% to 2.9% has been reported, whereas with extensive substrate ablation, the incidence of atrial tachycardias is dramatically higher, reaching 10%–24%.[76] In patients with post-PV isolation atrial tachycardias, recovered PV conduction has been found as a dominant mechanism, which can be successfully eliminated by reisolation.[43]

Typical CTI-dependent AFlu is responsible for 7%–10% of all post-AF ablation atrial tachycardias.[77] It bears characteristics of counterclockwise rotation in the majority (80%) of patients. However, some atypical (altered) ECG characteristics have been identified in this particular subgroup in comparison to patients with typical CTI-dependent AFlu who have not undergone prior PV isolation.[78] Specifically, the F waves in the inferior leads may be upright in as many as 60% of patients with prior LA ablation compared with none of the control patients without prior PV isolation, corresponding to craniocaudal activation of the RA-free wall. This atypical feature may be attributed to altered LA activation because of a significant reduction in bipolar LA voltage. Thus, in patients presenting with AFlu after LA ablation, entrainment mapping should be performed at the CTI even if the ECG is uncharacteristic of CTI-dependent flutter.

In our series of 67 patients undergoing PV isolation, 6 (9%) patients subsequently developed AFlu. Typical CTI-dependent counterclockwise AFlu occurred in 5 of these 6 patients who subsequently underwent additional CTI ablation (Table 1). In 1 patient with left AFlu, reisolation of the PVs abolished the arrhythmia.

CONCLUSIONS

AFlu and AF often coexist before and after drug or ablation therapies (Fig. 2). There appears to be a close interrelationship between these 2 arrhythmias where one may "transform" into the other. Discerning CTI-dependent AFlu from other atypical types of AFlu is crucial in deciding the next step in ablation therapy. New-onset AF may develop in a significant percentage (20%–40%) of patients undergoing CTI ablation for AFlu that will necessitate PV isolation at a subsequent session, whereas PV isolation may be considered in conjunction with CTI ablation during the same session in patients with previously documented AF. On the other hand, there are several cases of newly emerging AFlu after PV isolation; these may be typical CTI-dependent AFlus; however, one has to be cognizant of PV-related LA flutters. In those with more extensive LA substrate ablation, LA tachycardias mimicking AFlu may be the underlying mechanism of the apparent AFlu, requiring more demanding ablation procedures. The surface ECG can provide important initial clues as to the origin and location of AFlu; however, additional entrainment and occasionally more sophisticated 3-dimensional electroanatomical mapping techniques may be required to identify its mechanism, location, and target area for a successful ablation.

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