Lower Loop Reentry as a Mechanism of Clockwise Right Atrial Flutter

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Background—Right atrial reentrant tachycardia resulting from lower loop reentry (LLR) around the inferior vena cava (IVC) has been described recently. However, all reported cases of LLR in the literature have negative flutter waves on the inferior surface ECG leads similar to that of counterclockwise typical atrial flutter around the tricuspid annulus (TA). Right atrial flutter with positive flutter waves in the inferior ECG leads has been assumed to rotate as a single reentrant activation wave front around the TA, and the role of LLR in those patients is not known.

Methods and Results—Twelve consecutive patients with flutter wave morphology on surface ECG consistent with clockwise atrial flutter were studied. The endocardial activation pattern recorded from conventional multipolar electrode catheters was characteristic of clockwise atrial flutter around the TA. Entrainment pacing in all 12 patients and 3D activation sequence mapping in 7 patients, however, revealed clockwise LLR involving the lower right atrium around the IVC in 7 patients, figure-of-8 double-loop reentry around both the IVC and TA in 4, and single reentrant loop around the TA in 1. Linear radiofrequency ablation in the isthmus between the TA and IVC (TI isthmus) terminated the tachycardia in all patients.

Conclusions—Surface ECG flutter wave morphology and limited recording intracardiac sites proved insufficient to delineate the precise mechanism of the TI isthmus–dependent clockwise right atrial flutters. Most right atrial flutters with positive flutter wave on surface ECG may be supported by a reentrant circuit around the IVC or a figure-of-8 double-loop reentry involving both the IVC and TA. (Circulation. 2004;109:1630-1635.)

Key Words: atrial flutter • pacing • reentry

Lower loop reentry is defined as macroreentrant tachycardia maintained by circus movement of the activation wave front around the inferior vena cava instead of around the tricuspid annulus as in typical right atrial flutter (AFL).1–3 Lower loop reentry (LLR) uses the same isthmus between the tricuspid annulus and inferior vena cava as in AFL and therefore is similarly amendable by linear ablation of that isthmus.1,2,4,5 To date, only LLR with surface ECG flutter wave morphology similar to that of counterclockwise AFL has been described.1,2 We hypothesized that LLR may also be the operative mechanism underlying right AFL with positive flutter wave morphology. In the present study, we investigated the exact reentry circuitry in patients with AFL that was associated with surface ECG flutter wave morphology and endocardial recordings from conventional catheters that are characteristic of clockwise AFL.

Methods

Patient Selection
Twelve consecutive patients with a clinical diagnosis of clockwise AFL based on ECG criteria (Figure 1A) who were referred to our laboratories for ablation were studied from November 2000 to December 2002. Nine were men, and the average age was 62.1 ± 8.0 years (range, 50 to 72 years). Two had coronary artery disease; 2 had hypertension; 0 had significant valvular disease; 11 had normal or preserved left ventricular systolic function; and 1 had ejection fraction of 40% as a result of prior myocardial infarction. All had normal thyroid function tests, and none had clinical evidence of active myocardial ischemia. Three had a history of paroxysmal atrial fibrillation. Antiarrhythmic medications were discontinued at least 5 half-lives before the procedure in all but 1 patient who was taking amiodarone. One underwent pulmonary vein and superior vena caval isolation after flutter ablation.

Electrophysiological Evaluation
Patients were admitted to the Electrophysiology Laboratory in a postabsorptive and unsedated state. Venous access was obtained with
Figure 1. LLR circuit in patient with clockwise right AFL. A, A 12-lead surface ECG showing positive flutter wave morphology in inferior leads. B (left), Atrial activation sequence suggested TI isthmus-dependent flutter. Concealed entrainment was demonstrated with pacing in TI isthmus. Atrial overdrive cycle length was 220 ms, and PPI recorded from ablation catheter (ABL) placed in TI isthmus was 245 ms. TCL was 240 ms. Activation sequence during pacing was identical to that during tachycardia. B (right), Involvement of posterior lower RA in reentrant circuit. Entrainment pacing from posterior lower RA just above IVC showed PPI of 245 ms, indicating that posterior lower RA was part of circuit. Pacing at multiple sites along lateral, posterior, and septal junction between RA and IVC yielded similar PPI. Insets are fluoroscopic images of catheter positions when tracings were obtained. C, Propagation maps (left) were recorded during tachycardia and show clockwise propagation of activation wave front around IVC. Top half of RA was removed along horizontal plane to expose endocardial surface of lower RA viewed in cranial-to-caudal direction. Red zone indicates propagation of activation wave front; blue zone, regions that had recovered from previous excitation. Revolution time was identical to TCL. Left lateral view of activation map (right) shows collision of wave fronts at mid-septum (dashed arrows) in area of marked conduction slowing in lower septum. Arrowed solid circle around IVC represents reentrant circuit during LLR. Tachycardia was terminated during linear radiofrequency lesion in TI isthmus, further confirming its dependency on conduction through TI isthmus. AS indicates anterior septum close to His position (HBE); RA 1-2, RA 3-4, RA 5-6, RA 7-8, and RA 9-10, bipolar electrograms recorded from decapolar catheter placed anterior to crista terminalis along lateral RA (RA 1-2 being most distal and RA 9-10 most proximal pair); CSP, electrogram recorded from proximal CS; LRA, PRA, lateral and posterior RA; SVC, superior vena cava; RAO, right anterior oblique; and LAO, left anterior oblique.
pacing traveled different routes to and from underlying reentrant circuit. PPI at that site actually reflected revolution time around TA that was significantly longer than that around IVC. This can be expected if wave front generated from TA follows the same path as that which would propagate from a pacing site in the posterior wall of the right atrium. Furthermore, this explains why PPI was comparable to that determined at the pacing site. Entrainment pacing through ablation catheter (ABL) positioned at superior TA (Figure 2B, right) revealed PPI of 425 ms that was significantly longer than TCL (right). Electrograms from the distal electrode pair, in which case pacing site was apparently moved toward septum. Therefore, circuit around IVC was identified to be a bystander in this case. Insets are fluoroscopic images of catheter positions when tracings were obtained. TA indicates tricuspid annulus; SVC, superior vena cava; CSp, CSm, and CSD, electrograms from proximal, mid, and distal CS; RAO, right anterior oblique; and LAO, left anterior oblique.

The 12-lead surface ECG and intracardiac signals were recorded with a computerized multichannel data acquisition system (either the EP LabSystem by CR Bard, Inc, or CardioLab by GE Medical Systems). Intracardiac signals were filtered with low and high cutoff frequencies of 30 and 500 Hz, respectively, with a sampling frequency of ~1000 Hz. Programmed stimulation was delivered through a programmable stimulator (Bloom Associates, Ltd).

The diagnosis of clockwise flutter was supported by a typical endocardial activation sequence and evidence of concealed entrainment from the TI isthmus and the surface ECG flutter wave morphology. The orientation of the flutter is defined to be clockwise if the activation wave front during flutter in the TI isthmus is propagating from the septal or medial to lateral aspect. In 7 of the 12 patients, 3D activation sequence mapping was performed with the CARTO mapping system by Biosense Webster, Inc (total RA mapping points, 58 ±14; range, 39 to 81). Entrainment pacing was performed with overdrive pacing at a cycle length 15 to 25 ms shorter than the tachycardia cycle length (TCL) at multiple atrial sites in all patients. Transient entrainment of the tachycardia was confirmed by acceleration of the tachycardia to the paced rate. The postspacing interval (PPI) was defined as the time interval from the last pacing artifact to the first local electrogram recorded at the pacing site. Occasionally, the electrograms were significantly distorted at the distal electrode pair when pacing from the roving ablation catheter, in which case electrograms from the proximal electrode pair were used. The site of pacing is considered to be outside the reentrant circuit if the PPI is ≥20 ms longer than the flutter TCL. For entrainment pacing along the junction between
the RA and IVC, the catheter was positioned at the most inferior site in the RA where local capture was demonstrated with maximum pacing output (10 V with pulse width of 2 ms). Entrainment pacing at the superior aspect of the TA was also performed with maximum pacing output.

**Ablation**

After the baseline study, radiofrequency ablation was performed during tachycardia with a linear drug lesion from the TA to the eustachian ridge. The temperature was set at 60°C to 65°C with maximum power of 50 W. Once tachycardia was terminated during ablation, ablation was continued during pacing from the CS at 600 ms. Bidirectional conduction block in the TI isthmus was used as the end point of ablation.

**Results**

All patients had positive flutter wave morphology in the inferior ECG leads and endocardial activation sequences recorded from multipolar electrode catheters, typical of clockwise AFL (Figure 1A and 1B). The average TCL was 243.5 ± 16.5 ms. Concealed entrainment was demonstrated during overdrive pacing from the TI isthmus (Figure 1B, left), and tachycardia was terminated during linear ablation in the TI isthmus, confirming that the tachycardia was isthmus dependent.

In 7 patients, entrainment mapping indicated that the lower portion of the posterior RA was part of the reentrant circuit because PPI was within 20 ms of the TCL. This was demonstrated in the posterior and septal regions of the lower RA along the IVC, as shown in Figure 1B (right), Figure 2A (left), and Figure 3B. The 3D activation sequence mapping performed in 4 of these 7 patients showed a reentrant tachycardia around the IVC with the wave front propagating from the septal to the lateral aspect of the TI isthmus (clockwise LLR; Figure 1C). Wave fronts collided at the septum where a substantial conduction delay was noted (Figure 1C). In the other 3 patients, 3D activation sequence mapping was not performed. However, entrainment pacing similarly revealed that the circuit was confined to the lower portion of the RA around the IVC (Figure 2).

Figure-of-8 double-loop reentry was evidenced by entrainment mapping in 4 patients (Figure 3) in whom entrainment pacing at the superior TA and lower posterior RA demonstrated PPIs ≤ TCL plus 20 ms. The 3D activation mapping performed in 3 of these 4 patients revealed identical revolution times around the IVC and TA (Figure 3). Typical clockwise AFL with single reentrant loop around the TA was confirmed in 1 patient.

In 11 of 12 patients in whom the posterior RA was found to be in the reentrant circuit, there were often subtle changes in the activation sequence, recorded mainly from the CS sites, during entrainment pacing in the posterior RA compared with that during tachycardia (left tracings in Figures 2A and 3B).

**Discussion**

The major finding of our study is that most TI isthmus–dependent flutter with positive flutter wave in the inferior ECG leads involves a reentrant circuit around the IVC. In all but 1 of the 12 patients, entrainment pacing in the lateral, posterior, and septal RA along the RA-IVC junction revealed PPIs that were comparable to the TCL during flutter, indicating that lower RA constitutes an integral part of the reentrant circuit. In 7 of the 11 patients, the superior portion of the TA was shown not to be involved in the reentrant circuit (single-loop LLR). The 3D activation sequence mapping revealed an activation wave front circulating around the IVC with evidence of slow conduction and wave-front collision at the interatrial septum (Figure 1C). Rodriguez et al. described the global biatrial activation during both counterclockwise (7 patients) and clockwise (2 patients) AFL. Their data suggested that clockwise right AFL could be supported by a reentrant circuit around the TA. However, activation around the RA-IVC junction was not described in detail. The propagation maps from their study also showed evidence comparable to a propagation wave front around the IVC during clockwise AFL. Conduction slowing was also noted along the interatrial septum in their study. Because entrainment pacing was not performed, one cannot exclude the possibility of a figure-of-8 double-loop reentry as the operative mechanism.

Our study provides the first evidence that figure-of-8 double-loop reentry may occur around the IVC and TA and mimic typical clockwise AFL in patients without atriotomy (Figure 3). Figure-of-8 double-loop reentry has been reported in patients with atypical AFL patterns and in those with a history of atriotomy. Because the TI isthmus constitutes the common pathway between both reentrant loops, this double-loop reentry is also amendable by linear ablation that results in bidirectional conduction block in the TI isthmus.

From our observation, we speculate that the revolution or conduction times around the IVC and TA determine the actual reentrant circuit in patients with clockwise right AFL. This may provide a unifying hypothesis that the circus movement of activation wave front during TI isthmus–dependent clockwise right AFL is dictated by the revolution times around the 2 competing potential reentrant loops, 1 around the IVC and 1 around the TA. A shorter revolution time around the IVC compared with that around the TA would lead to LRR, whereas a shorter revolution time around the TA compared with that around the IVC would lead to typical clockwise AFL. A comparable revolution time around both the IVC and TA would result in figure-of-8 double-loop reentry.

We have also noted that the values of PPI determined at the superior aspect of the TA during single LLR were often much less than would be expected otherwise, considering the distance to the reentrant circuit (Figure 2A, right). Classically, PPI is expected to be the sum of the times required for the paced wave front to travel to and from the reentrant circuit plus the TCL. However, if the pacing site is within or near a bystander reentrant loop that shares a common segment or isthmus with the true reentrant circuit, it is possible that the paced wave front takes different propagation pathways to and from the true reentrant circuit. Therefore, the return cycle at that site after the last paced beat of entrainment pacing may not include the entire TCL but instead represents the revolution time around the bystander reentrant loop itself.

Previous descriptions of LLR were always associated with negative flutter waves in the inferior ECG leads, consistent with a counterclockwise endocardial activation sequence. In these patients with counterclockwise LLR, an early breakthrough occurs along the crista terminalis and results in collision of wave fronts along the lateral wall of the RA. Therefore, counterclockwise LLR can be readily recognized with conventional recording technique. However, identification of a clockwise LLR is not as...
Figure 3. Determination of reentrant circuit in patient with figure-of-8 double-loop reentry. As in cases presented in Figures 1 and 2, endocardial activation sequence recorded from conventional catheter positions suggests clockwise typical AFL around TA with concealed entrainment during pacing in TI isthmus. Results of entrainment pacing from superior TA (RA 9-10) showed PPI identical to TCL [left tracing of A with ablation catheter (ABL) placed at TA more septal to RA-10]. Entrainment pacing from lateral RA (RA 5-6) close to TA (B, right tracings) showed similar value of PPI, indicating that TA was “in circuit.” Entrainment pacing from posterior lower RA (pacing from ablation catheter positioned at posterior lower RA (B with left tracings recorded during entrainment pacing at more lateral location and right tracings during entrainment pacing at more septal location) revealed values of PPI comparable to TCL. Interestingly, there was only subtle evidence of manifest entrainment or altered activation sequence during pacing in posterolateral lower RA vs that during tachycardia (apparent “concealed” entrainment), except polarity of atrial electrogram recorded at mid CS was reversed. As we moved pacing site to more septal location of lower RA, changes in activation sequence became more obvious because proximal CS was more “pre-excited” during pacing (B, right). During tachycardia, there was collision of 2 wave fronts in posterior LA along posterior vestibule, 1 propagating from RA-CS connection and 1 presumably from Bachmann’s bundle. During pacing at posterior lower RA, RA-CS connection was more pre-excited as pacing site was moved more septally, leading to dominantly proximal-to-distal activation pattern in CS. In addition, 3D activation sequence mapping revealed revolution times around IVC and TA that were identical to TCL (C). Color bar decodes local activation times. Cranial view of lower RA is shown on left. Top portion of RA was removed to show activation sequence around IVC. Left anterior oblique view of RA is shown on right. Note in both views that arbitrarily determined earliest site was at same location (medial aspect of common TI isthmus). Revolution times around IVC and TA were identical to TCL (250 ms) as evidenced by adjoining of sites with “earliest” (red) and “latest” (purple) local activation, with both activation wave fronts converging at entrance to TI isthmus simultaneously. LAO indicates left anterior oblique.
apparent and requires more detailed entrainment pacing and/or 3D activation sequence mapping.

We explain the possible flutter wave pattern in the inferior ECG leads during clockwise LLR by hypothesizing that the activation wave front exited laterally from the TI isthmus with marked conduction slowing in the lower septum, as shown in Figure 1C. This allows the bulk of the intra-atrial septum to be activated in the craniocaudal direction and the left atrium to be activated in a manner similar to that in clockwise typical AFL.9,11,12 Further studies are needed to define the role of slow conduction in determining the exact reentrant circuitry during clockwise right AFL.

Study Limitations
We always tried to position the pacing catheter as close to the annulus as possible during entrainment pacing around the TA. However, to the exact extent that pacing might have been performed away from the annulus, we might have underestimated the incidence of figure-of-8 double-loop reentry.

The use of high pacing output (10 V at a pulse width of 2 ms) and bipolar pacing could theoretically capture a larger area and even alter the local activation path around the site of pacing, leading to an “artifactually” shortened PPI. However, this effect is limited quantitatively and could not be responsible for the consistent values of PPI with entrainment pacing at the posterior lower RA that were comparable to the TCL, considering the substantial distance between the pacing site and the tricuspid annulus.

In addition, 1 patient was on amiodarone therapy, which may also affect the response of local myocardial tissues to pacing and prolong PPI if rapid pacing had encroached on the refractory period. However, we were pacing within the limits of the fully excitable gap, as evidenced by the values of PPI that were comparable to TCL at multiple sites in this patient, and most patients were studied without antiarrhythmic agents.

Because 3D activation mapping was not performed in all patients and we had only limited intracardiac recording sites in the remaining 5 patients, it is difficult to conclude that the site of wave-front collision was consistently located along the intraseptal isthmus in those patients. We did, however, exclude the involvement of the superior parts of the RA by entrainment pacing, and collision did not occur over the lateral RA wall. In addition, the exact location where the activation wave front crosses the crista terminalis is not precisely defined in our study. However, it is clear that such transverse conduction across the crista terminalis occurred in the lower portion of the RA on the basis of data from entrainment pacing during tachycardia and 3D activation mapping (Figures 1C and 3C).

We were not able to evaluate the relationship between the pacing stimulus at various sites and the surface flutter wave, which would be helpful in identifying the area(s) of slow conduction, because all patients had 2:1 AV conduction during flutter and identification of flutter wave onset from the surface ECG was difficult.

Conclusions
We have demonstrated that clockwise right AFL can be supported by a single LLR or figure-of-8 dual-loop reentry around both the IVC and TA. Our study further illustrates that surface ECG and endocardial activation sequence recorded from a limited number of electrodes may not adequately reflect the precise mechanism and boundaries of the flutter circuit.

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