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Separating Non–Isthmus- From Isthmus-Dependent Atrial Flutter Using Wavefront Variability

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OBJECTIVES The aim of this study was to separate isthmus-dependent atrial flutter (IDAFL) from non–isthmus-dependent atrial flutter (NIDAFL) from the electrocardiogram (ECG) based on functional differences.

BACKGROUND The ECG analyses of F-wave shape suboptimally separate NIDAFL from IDAFL. The authors hypothesized that anatomic and functional differences may result in greater wavefront variability in NIDAFL than IDAFL, allowing their separation. The authors tested this hypothesis in patients undergoing ablation for atrial flutter using a novel ECG algorithm to detect subtle F-wave variability, validated by intracardiac measurements.

METHODS In 62 patients (23 NIDAFL, 39 IDAFL) ECG atrial wavefronts were represented as correlations of an F-wave template to the ECG over time. Correlations in orthogonal ECG lead-pairs were plotted at each time point to yield loops reflecting temporal and spatial regularity in each plane. The ECG analyses were compared with intracardiac standard deviations of: 1) atrial electrograms (temporal variability), and 2) bi-atrial activation time differences (spatial variability).

RESULTS Atrial ECG temporospatial loops were reproducible in IDAFL, but varied in NIDAFL ($p < 0.01$) suggesting greater variability that correctly classified IDAFL (39 of 39 cases) from NIDAFL (22 of 23 cases; $p < 0.001$). Intra-atrial mapping confirmed greater temporal variability for NIDAFL versus IDAFL, in lateral ($p < 0.01$) and septal ($p = 0.03$) right atrium, and proximal ($p = 0.02$) and distal ($p < 0.01$) coronary sinus. Spatial variability was greater in NIDAFL than IDAFL ($p = 0.02$).

CONCLUSIONS Greater cycle-to-cycle atrial wavefront variability separates NIDAFL from IDAFL and is detectable from the ECG using temporospatial analyses. These results have implications for guiding ablation and support the concept that IDAFL and NIDAFL lie along a spectrum of intracardiac organization. (J Am Coll Cardiol 2005;45:1269–79) © 2005 by the American College of Cardiology Foundation

Classifying atrial macro–re-entry from the electrocardiogram (ECG) is central in guiding the approach to ablation yet remains suboptimal. Typical atrial flutter is defined by atrial waveforms that are inverted in the inferior ECG leads and upright in lead V_1 (1) (sub-eustachian isthmus-dependent atrial flutter [IDAFL], counterclockwise [CCW]), or "inverted" in reverse typical atrial flutter (clockwise [CW]) (2). However, such F waves are also seen in atypical atrial macro–re-entry (non–isthmus-dependent atrial flutter [NIDAFL]) (3,4), including left atrial circuits (5). Furthermore, F waves in IDAFL may be "atypical" in patients with atrial enlargement, heart failure, factors favoring atrial fibrillation (6), or varying bi-atrial activation (7,8). These observations may explain why the absence of typical F waves does not accurately separate NIDAFL from IDAFL (2).

We hypothesized that functional differences may separate forms of stable atrial flutter. Although it is increasingly felt that IDAFL and NIDAFL lie along an organizational spectrum that interfaces with atrial fibrillation (2,9), this suggests either that NIDAFL is less organized than IDAFL or simply less stereotypical. However, these concepts may be interrelated. Certainly, macro–re-entry in IDAFL is stereotypical, yet it also occupies a large portion of the right atrium and is bounded significantly by anatomic obstacles so that passive activation of right and left (10) atria are also stereotypical, and cycle-to-cycle variability is limited. Conversely, the variable location of circuits in NIDAFL relative to anatomic obstacles, and smaller zones of conduction block (or scar), may render passive activation more susceptible to functional block and allow greater cycle-to-cycle variability. Although reports hint at greater F-wave variability in stable NIDAFL than IDAFL (5,11,12), this difference has not been quantified and is not used routinely to separate NIDAFL from IDAFL (2).

We tested the hypothesis that subtle F-wave variability between cycles would distinguish NIDAFL from IDAFL, by developing a sensitive ECG algorithm (13) then validating our findings from detailed clinical mapping in patients undergoing ablation of atrial arrhythmias.

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Abbreviations and Acronyms

A:V = atrio:ventricular
CCW = counter-clockwise
CL = cycle length
CS = coronary sinus
CW = clockwise
ECG = electrocardiogram
EPS = electrophysiologic study
FFT = Fast Fourier Transform
IDAFL = isthmus-dependent atrial flutter
LA = left atrium
NIDAFL = non–isthmus-dependent atrial flutter
IVC = inferior vena cava
RA = right atrium
TA = tricuspid annulus

METHODS

Clinical protocol. We studied 62 consecutive patients referred to the University of California (UCSD) and Veterans' Affairs Medical Centers (VAMC), San Diego, for electrophysiologic study (EPS) and ablation of stable atrial macro–re-entry, defined from prior ECGs and subsequently using entrainment. This study was approved by the joint UCSD/VAMC Institutional Review Board. Patients were excluded if they were too unstable to undergo EPS or were inadequately anticoagulated, unless they lacked atrial thrombus on transesophageal echocardiography.

Patients underwent EPS in the fasting state after discontinuing anti-arrhythmic and rate control medications (Table 1). Catheters were advanced transvenously to the His bundle position (quadrapolar) and coronary sinus (CS) (decapolar). A duodecapolar ("Halo") catheter was placed in the right atrium (RA) parallel to the tricuspid annulus (TA) with its proximal poles at the inter-atrial septum and its distal poles across the inferior vena cava (IVC)-TA isthmus. An ablation catheter (EP technologies, Sunnyvale, California) was used for mapping.

Table 1. Clinical Characteristics

	IDAFL (n = 39)	NIDAFL (n = 23)	p Value
Age (yrs)	58.5 ± 13.5	60.6 ± 12.8	NS
AFL CL (ms)	246.7 ± 30.8	291.2 ± 42.2	< 0.01
Ventricular CL (ms)	617.7 ± 200.6	685.3 ± 215.3	NS
Atrio:ventricular ratio	2.5 ± 0.8	2.4 ± 0.8	NS
LA diameter (mm)	38.9 ± 9.2	43.7 ± 7.8	NS
LVEF (%)	58.0 ± 16.5	51.3 ± 12.2	NS
NYHA heart failure class ≥II	0	3	NS
Prior cardiac surgery	6	11	< 0.01
Medications			
Anti-arrhythmic*	22	9	NS
Rate-slowing†	25	10	NS
Hypertension	18	8	NS
Diabetes mellitus	7	3	NS

*Prior therapy with class I or class III agents; †therapy with beta-blocker or calcium antagonists.

AFL = atrial flutter; CL = cycle length; IDAFL = isthmus-dependent atrial flutter; LA = left atrial; LVEF = left ventricular ejection fraction; NIDAFL = non–isthmus-dependent atrial flutter; NYHA = New York Heart Association.

Isthmus-dependent atrial flutter was diagnosed by sequential (CCW or CW) activation around the TA, concealed entrainment at the IVC-TA isthmus, and the inability to reinduce atrial flutter after creating bidirectional block across the IVC-TA isthmus by drag-line ablation. Non–isthmus-dependent atrial flutter was diagnosed by a distinct activation pattern from IDAFL, concealed entrainment at sites of earliest atrial activation or double potentials outside the IVC-TA isthmus, and termination and the inability to reinduce after ablation at these sites. An electroanatomic mapping system (Carto, Biosense-Webster, Diamond Bar, California) was used in NIDAFL to help localize the critical earliest site of activation and the isthmus of macro–re-entry for ablation (16 of 23 cases).

Acquisition of data. We recorded 12-lead surface ECGs (filtered at 0.05 to 100 Hz) and simultaneous bipolar intracardiac electrograms (30 to 100 Hz) of atrial flutter, digitized at 1 kHz to 16-bit resolution (Bard, Billerica, Massachusetts).

Analyses were blinded to all clinical data. Electrograms were analyzed on a personal computer using software written by the authors (S.M.N.) in Labview (National Instruments, Texas) (13). The ECG analysis focused on leads V_5, aVF, and V_1, related to orthogonal leads X, Y, and Z, respectively. Intracardiac electrograms were analyzed at 200 mm/s scale.

ECG temporal regularity. Analyses were blinded to clinical and EPS data. We quantified F-wave temporal and spatial variability as recently described (13). Briefly, ECG F-waves were represented by a 120-ms sample selected to avoid isoelectric ECG segments (Fig. 1). Each sample was cross-correlated to its ECG at successive time points using the Pearson coefficient on M pairs of data (A_{k+i}, B_{j+i}), where A_{k+i} and B_{j+i} are corresponding points of the F-wave sample and native ECG:

$$r_j = \frac{M\left(\sum_{i=1}^{M} A_{k+i}B_{j+i}\right) - \sum_{i=1}^{M} A_{k+i}\sum_{i=1}^{M} B_{j+i}}{\sqrt{\left[M\sum_{i=1}^{M} A_{k+i}^2 - \left(\sum_{i=1}^{M} A_{k+i}\right)^2\right]\left[M\sum_{i=1}^{M} B_{j+i}^2 - \left(\sum_{i=1}^{M} B_{j+i}\right)^2\right]}}$$

where r_j is the coefficient at the *jth* timepoint; j ranges from the first ECG point to Q − M, ($0 \le j \le$ Q − M), Q being the last ECG point; k ranges from L to L + M ($0 \le L \le$ Q − M). Repeating this operation for all ECG time points results in correlation-time series (Figs. 1, 2A, and 2B, lower left panels), in which F-waves (and the sampled region) have correlation r = 1, whereas non-exact matches (QRS, T-waves) give r values between −1 and +1. Because the correlation approach reduces differences in signal amplitude through scaling, the use of staggered overlapping ECG samples "extracts" F-wave components that are superimposed or partially revealed during a QRS complex or T-wave.

Regularity in F-wave timing was quantified using Fast Fourier Transforms (FFT) of 8.192 s (2^{13} ms) of correlation-

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