of 274 ms. In five cases, counterclockwise (CCW) endocardial activation was recorded whereas in two cases, impulse was reversed and proceeded inferiorly in the septum and superiorly in the lateral wall (Clockwise = CW). In CCW AF two patterns of activation were encountered: Ascending (n = 2) or widely spaced double spikes (DS) Pinferior to CSOs with progressive narrowing when proceeding upward with Psuperior DS fusion (n = 3): RF pulses delivered at the CSOs-TR isthmus failed to terminate AFI in the first pattern whereas RF (delivered in 2/3 cases) terminated AF in the other. In both CW AF activation proceeded inferiorly in this area. In these and in the 2 Pts with a unique ascending wavefront during CCW AF, the line of DS was further posterior in the right atrial wall. RF pulses delivered in the IVC-TR Isthmus terminated AFI in 4/4 Pts.

Conclusion: An ascending wavefront may cross the IVC-CSOs isthmus and therefore suggests a more lateral target for successful RF ablation of type I AF in this subgroup of Pts. In the other cases there seem to exist a line of block between IVC and CSOs and ablation of the CSOs-TR isthmus should be successful.

943-12 Radiofrequency Catheter Ablation of Human Type 1 Atrial Flutter — Comparison of Results with 8 mm versus 4 mm Tip Ablation Catheter

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Thirty four patients (pts) with type 1 atrial flutter (AFL) underwent radiofrequency catheter ablation (RFCA) using either a standard 4 mm tip (25 pts) or a large 8 mm tip (10 pts) electrode catheter. The pts, 26 male and 8 female, ranged in age from 21–89 yrs (57 ± 15). AFL cycle length ranged from 190–350 msec (248 ± 35). *Methods:* For RFCA with 4 mm tip, exit sites from the isthmus between the inferior vena cava (IVC) and tricuspid valve annulus (TVA) near the coronary sinus ostium were identified by an atrial activation time 20–50 msec before P wave onset, and an exact 12-lead ECG P wave map with short stimulus-to-P wave interval (≤40 msec) and identical endocardial electrogram pattern during entrainment. For RFCA with 8 mm tip, the middle or lateral isthmus was identified an atrial activation time 50–120 msec before P wave onset, and an exact 12-lead ECG P wave map with a long stimulus-to-P wave interval (≥40 msec) and identical endocardial electrogram pattern during entrainment. *Results:* RFCA with 4 mm tip, 1–23 (11 ± 5) lesions at 16–34 watts for 30–60 sec, resulted in termination and prevention of reinduction of AFL in 21 of 25 pts, no effect in 3 pts and only slowing of AFL in 1 pt. During followup, AFL recurred in 9 pts, of whom 5 underwent repeat RFCA with 4 mm tip, 1 underwent repeat RFCA with 8 mm tip, 2 are controlled on antiarrhythmic drugs and 1 had AV node ablation and pacemaker. At 21 ± 7 mos followup, 21 of 25 pts (84%) have no AFL after 1–2 RFCA attempts with 4 mm tip. RFCA with 8 mm tip, 2–12 (6 ± 3) lesions ($p < 0.01$ compared to 4 mm tip) at 45–58 watts for 30–60 sec, resulted in termination and prevention of reinduction of AFL in 10 of 10 pts. During followup, AFL recurred in 1 pt who underwent repeat RFCA. At 4 ± 3 mos followup, 10 of 10 pts (100%) have no AFL after 1–2 RFCA attempts with 8 mm tip. *Conclusions:* 1) Type 1 AFL can be cured by RFCA of the isthmus between IVC and TVA or exits from the isthmus, 2) 8 mm tip reduces the number of energy applications required for cure, 3) Trend suggests greater primary efficacy and less recurrence of AFL with 8 mm tip.

943-13 Complexities of the Dynamical Progression of 4:1 Atrioventricular Block in Atrial Flutter

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No previous studies have dealt with the dynamics of progression of 4:1 atrioventricular block (AVB). In order to do this, we analyzed Holter recordings of 6 patients (receiving digoxin and beta blockers or calcium channel blockers) in whom atrial flutter with "stable" (≥5 min) 4:1 AVB increased to higher degrees of AVB. The ratios (ra) in each episode (ep) were expressed numerically as well as M (number of atrial deflections): N (number of ventricular complexes). In stage 1, 324 ep of 4:1 AVB progressed (with letter representation in parentheses) to one (4N + 2:N) or several cycles of 6:1 AVB and to one (4N + 4:N) or several cycles of 8:1 AVB with subsequent regression to 4:1 AVB. In these ep, the ra between adjacent M:N and M':N' ra could be expressed as M + M':N + N' in the direct 4:1 to 6:1 or 4:1 to 8:1 steps of the so called devil's staircase. In the 398 ep of stage 2, these progressions of 4:1 AVB also coexisted with increases to: a) 5:1 to 6:1; b) 5:1 to ≥8:1; and c) 5:1 to 6:1 to 8:1 AVB. The higher degrees of AVB did not regress to short cycles of 4:1 AVB for several seconds. In summary: 1) 4:1 AVB in atrial flutter could progress directly to 6:1 and 8:1 AVB, bypassing intermediate steps of the devil's staircase, and 2) 8:1 AVB could also emerge directly from 5:1 and 6:1, but apparently not from 7:1 AVB.

943-14 Termination and Suppression of Experimental Atrial Flutter by Quinidine: Effects on Dispersion of Repolarization and Conduction

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Recent studies in our canine crush-injury (CI) model of atrial flutter (AFL) suggest that elimination of dispersion of repolarization is an important antiarrhythmic effect of newer class 3 antiarrhythmic drugs. However, the effects of class 1a antiarrhythmic drugs on dispersion of repolarization and its relation to termination and suppression of AFL have not been studied. Therefore, the effects of quinidine (Q) were studied in 8 dogs, using a 56-electrode plaque placed over the CI to map activation patterns during AFL and its induction and termination, and to measure effective refractory periods (ERP) at all 56 electrodes and conduction velocity (CV) parallel to the CI, before and after Q (10 mg/kg over 10 min, then 3 mg/kg/hr). *Results:* Induction of AFL at baseline was due to unidirectional block below the CI. Q terminated AFL in only 3 dogs, due to conduction block below the CI, occurring either abruptly or following premature eccentric activation of the reentry circuit. Sustained AFL was reinducible after Q in 5 dogs, and nonsustained AFL in 3. Mean overall ERP increased 14% from 112 ± 11 msec at baseline to 128 ± 15 msec after Q ($p < 0.001$). ERP was longer below CI than above (115 ± 10 ms vs 109 ± 10 ms, $p < 0.001$), and this difference was not eliminated by Q (133 ± 14 ms vs 123 ± 14 ms, $p < 0.001$). Overall dispersion of ERP (9.9 ± 2.3 msec) was unchanged by Q (10.0 ± 1.3 msec, p = NS). The number of adjacent electrodes with ERP difference ≥20 msec (14.4 ± 10.4) was also not changed by Q (12.5 ± 7.9, p = NS). CV was slower below the CI than above at baseline (0.7 ± 0.1 vs 0.9 ± 0.1 m/s, $p < 0.001$), and this difference was unchanged by Q (0.6 ± 0.1 vs 0.8 ± 0.1 m/s, $p < 0.001$), although CV was slowed overall ($p < 0.01$). Q prolonged AFL cycle length 57% from 123 ± 12 to 198 ± 37 msec ($p < 0.01$). *Conclusion:* 1) Baseline dispersion of ERP and CV is important for initiating and sustaining AFL in this model. 2) The class 1a drug quinidine is relatively ineffective in terminating and suppressing AFL in this model, because it prolongs ERP less than AFL cycle length and it does not eliminate dispersion of ERP or CV.

943-15 Site-dependent Intra-atrial Conduction Delay: Relationship to Initiation of Atrial Flutter/Fibrillation

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Atrial flutter/fibrillation (AF) are more readily inducible by atrial premature complexes (APCs) from the high right atrium (HRA) than from the coronary sinus (CS). Patients (pts) who have clinical AF have an exaggerated intra-atrial conduction delay (IACD) and prolonged relative refractory period (RRP) in response to APCs delivered from the HRA. We therefore compared the response of APCs delivered from the HRA, posterior Triangle of Koch (SP), and CS to evaluate whether the propensity of HRA stimuli to induce AF is related to site-specific anisotropic conduction abnormalities. Programmed atrial stimulation at twice threshold was caried out in five pts from the HRA, SP, and CS. Recordings were made at the HRA, SP, along the Tendon of Todaro (HBE catheter), and the CS. Pts were 3 women and 2 men, age 50 to 79, none of whom had clinical AF or organic heart disease. None had IACD during sinus rhythm. The RRP was defined as the range of intervals over which A1A2 was >S1S2 at the stimulus site, and A1A2 at distance sites was >A1A2 at stimulus sites. ΔCT denotes the maximum increase in conduction time at each site.

Recording site	ΔCT			RRP		
Stimulation site	HRA	CS*	SP*	HRA	CS*	SP*
HRA	0	23	25	53	30	52
SP	65	24	0	82	30	32
HBEd	53	28	46	82	30	58
HBE2	52	28	33	78	28	58
HBE3	53	28	38	78	28	52
CSp	62	19	34	76	28	64
CS4	67	16	34	88	26	50
CS3	71	2	39	86	24	54
CS2	65	0	46	86	10	60

* $P < 0.03$, Data are means (in msec)

AF was induced in 2 pts from the HRA but not from other sites. Our results demonstrate the greatest degree of IACD and RRP was seen with HRA stimulation and the least with CS stimulation. SP stimulation resulted in intermediate results. The marked site-specific difference in IACD supports anisotropic impulse conduction in the atria, and may explain why AF is more often initiated from the HRA than the CS.