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### Is There a Connection Between the Right Upper Pulmonary Vein and the Superior Vena Cava in Patients With Atrial Fibrillation?

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**Background:** Ectopic atrial activity in the pulmonary veins (PV) and superior vena cava (SVC) has been recently described as a possible trigger for atrial fibrillation (AF). However, no relationship between these arrhythmogenic foci has been documented.

**Methods:** We report our experience in 179 consecutive patients (mean age 53±11 years, 79% male) undergoing catheter ablation for AF. Atrial fibrillation was paroxysmal in 99 patients, persistent in 26 and permanent in 54 patients. All patients failed 3 ± 0.9 antiarrhythmic drugs.

**Results:** Sustained atrial arrhythmias originating from the SVC were observed in 5.5% (10 out of 179 patients). In all of these patients AF appeared to originate from the right upper PV (RUPV). After isolation of the RUPV, 7 patients developed spontaneous atrial tachycardia originating from the SVC (cycle length ranging from 170 to 260 ms). In the remaining 3 patients, firing from the SVC continued to lead to AF. After successful SVC isolation all 10 patients remained arrhythmia free.

**Conclusion:** Superior vena cava foci triggering atrial arrhythmias appear to be consistently associated with the presence of right upper PV focus. This may suggest an inter-venous connection between these 2 structures, or reflect a common embryological origin. In patient showing RUPV firing ablation in the SVC may be required to maximize long-term cure.

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### Atrial Electro-Anatomical Remodeling as a Predictor of the Success of Circumferential Pulmonary Vein Ablation for Atrial Fibrillation

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**Background:** Circumferential radiofrequency (RF) ablation around pulmonary veins (PV) is a promising approach for curing atrial fibrillation (AF), but its effect on AF sources and substrate is still unclear. Identification of factors affecting success could give insight into the mechanism and aid patient selection. **Methods:** Between Dec. 1999 and July 2001, 628 patients with paroxysmal (n=409) or permanent (n=219) AF (79% male, age 59±9 yrs, AF duration 7±2.5 years; 59% with structural heart disease) underwent circumferential PV ablation using 3D-electroanatomical guidance. Patients were followed up with serial visits, Holter recordings, transthoracic and transesophageal echocardiography. **Results:** After 18.2±6.3 months, 348 patients with paroxysmal AF (85%) and 164 with permanent AF (75%) were AF-free. Patients with and without AF recurrence did not differ in age, AF duration, prevalence of heart disease and ejection fraction. By echocardiography, pre-RF left atrial (LA) diameter was significantly higher (P<0.001) in permanent AF patients with recurrence. Post-RF reduction of LA size was noted in all patients without AF recurrence, associated with preserved and/or improved LA contraction as assessed by mitral inflow Doppler tracings. Proportion of PVs with complete lesions (bipolar voltage <0.1 mV inside the circular line) was similar between patients with and without recurrence, but the latter had larger post-RF encircled low-voltage area in percent of LA surface area (31% vs 18%, P<0.001). This finding was related in part to a greater extent of the low-voltage area outside the lesions (P=0.01). By multivariate analysis, independent predictors of a higher likelihood of sinus rhythm restoration were postablation LA size reduction and amount of low-voltage encircled area. **Conclusions:** Pre-RF clinical factors or effective PV trigger isolation may not be critical to achieve AF suppression. Benefits of circumferential PV ablation appear to depend on the degree of reversal of atrial electroanatomical remodeling and related probability of AF initiation and maintenance.

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### Intraatrial Conduction Abnormalities in Patients With Brugada-Type ECG Estimated by P Wave Triggered Signal-Averaged ECG

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**Background:** Brugada syndrome is often accompanied by atrial tachyarrhythmias including paroxysmal atrial fibrillation (PAF). Underlying mechanisms for the atrial arrhythmogenicity are unknown.

**Methods:** Fifteen patients (pts) (all males, 45±5 years) with Brugada-type ECG (RBBB with ST-segment elevation in the right precordial leads) without organic heart disease were studied. All of these pts showed additional ST-segment elevation (> 0.15 mV) in response to pilsicainide application (1.0 mg/kg, i.v.). Two pts had syncopal events, and one had a family history of sudden death. Remaining 12 pts were asymptomatic, but ventricular fibrillation was induced in 4 of the 5 asymptomatic pts who underwent electrophysiologic testing. No episodes of PAF were documented in all the pts. Twenty-five age-matched male pts (42±8 years) with normal ECG were employed as controls. P wave-triggered signal averaged ECG was recorded from the standard 12 leads. Filtered P duration (FPD) and root-mean-square-voltage for the last 20 ms (RMS20) were measured in the vector magnitude obtained via a modified XYZ lead system using I, aVF, V1 leads as X, Y, Z, respectively. P wave dispersion (Pd) was estimated from the difference between the maximum and minimum duration of signal-averaged P wave (Pmax, Pmin) in the 12 leads.

**Results:** Brugada-type pts showed significantly longer FPD (143.7±10.3 versus 122.3±9.9 ms, p<0.0001) and lower RMS20 (2.0±1.0 versus 3.7±1.4 uV, p<0.001) compared with control pts. Pmax and Pmin in Brugada-type pts were significantly longer than

those in control pts (152.3±13.9 versus 133.3±10.4 ms for Pmax, p<0.0001; 126.6±15.10 versus 115.6±8.7 ms for Pmin, p<0.05). Pd in Brugada-type pts was significantly larger than that in control pts (25.7±10.4 versus 17.6±4.2 ms, p<0.005). Abnormal values (out of the 90th percentile of controls) for filtered P wave (FDP > 136 ms and RMS20 < 2.0 uV) and Pd (> 23 ms) were observed in 7 (47%) and 8 (53%) pts, respectively, in the Brugada-type group.

**Conclusion:** Intraatrial conduction in pts with Brugada-type ECG is delayed and spatially inhomogeneous. Such abnormal conduction properties could be a substrate for reentrant atrial tachyarrhythmias.

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### Revised Anatomical Description of the Bachmann Bundle and Its Relationship to the True Interatrial Septum

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**Background:** Recent basic and clinical electrophysiologic studies have outlined the critical role of Bachmann's Bundle in interatrial conduction and most likely in the pathogenesis of atrial fibrillation. However, there are no contemporary anatomic studies of Bachmann's Bundle, and although described as a bundle, it remains unclear whether it can be distinctly isolated, as well as its exact relationship to the true septum.

**Methods:** Twelve preserved human heart pathology specimens without cardiovascular disease and with the Interatrial region intact were dissected to study Bachmann's Bundle gross anatomy. Dissection was started at well identifiable Interatrial sulcus in adjacent regions; it was pursued "beneath" the bundle by following cleavage planes in connective tissue without entering myocardium. In each specimen the Bachmann's Bundle was isolated as a distinct bundle bridging the two atria.

**Results:** The bundle is a superficial trapezoidal structure wedging the convexity of the atrial walls. Cross section shows a thickness of 14.3mm (median 4) and height of 18.6mm (median 9). The lower short side is 11.2mm (median 3); its distance to Fossa Ovalis 16.2mm (median 7). Projection of right Endocardial attachment is not at the pre-caval bundle attachment but posterior and left along a vertical line bisecting the FO and within 2cm from its upper pole.

**Conclusion:** Gross anatomy of Bachmann's Bundle documents a true bundle, well documented from the fossa ovalis and true atrial septum. These results provide important anatomic data towards endocardial or epicardial approaches during catheter or surgical interventions towards modifying or eliminating conduction over Bachmann's Bundle.

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### Mechanism of Immediate Recurrence of Atrial Fibrillation in Patients With Paroxysmal Atrial Fibrillation

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**Background:** Immediate recurrence of atrial fibrillation (IRAF) may occur during isolation of pulmonary veins (PV) for atrial fibrillation (AF). Although it has been suggested that the prematurity of a single APD is critical for the initiation of AF, the mechanism by which IRAF occurs is not clear. **Objective:** To determine the mechanisms by which IRAF is initiated. **Method:** Nine men and 1 woman (mean age=51±6) underwent a PV isolation procedure for paroxysmal (n=8) or persistent AF (n=2). A decapolar ring catheter was positioned within a PV; a catheter was placed in the left atrium (LA) just outside the PV ostium. AF was induced by rapid atrial pacing in 5 patients who were in sinus rhythm before the isolation. The remaining 5 patients were in AF. After transthoracic cardioversion, electrograms from the PV, LA and CS were recorded. IRAF was defined as any occurrence of AF within 60 seconds after cardioversion. The prevalence of IRAF and the coupling interval and prematurity index (1-[coupling interval/preceding sinus cycle length]) at the onset of IRAF were determined. The onset of AF was verified by electrograms recorded from the LA catheter. **Results:** There were 44 episodes of single PV depolarizations (4.9 ± 2.8 per patient) and 25 bursts of a PV tachycardia (2.5 ± 2.0 per patient) within 1 minute of resumption of sinus rhythm. IRAF occurred 28 times in 10 patients (2.8±1.9 per patient) within 21 ± 28 secs after termination of AF. Each of the 25 bursts of PV tachycardia initiated IRAF (100%), whereas 3 of the 44 single PV depolarizations triggered an episode of IRAF (7%, p<0.001). The coupling interval and the prematurity index of APDs that did not initiate IRAF, 332±91 and 0.61±0.15, were similar to those of the first beat of a burst of a PV tachycardia that triggered IRAF, 319±123 msec and 0.62±0.14, respectively (p=NS). IRAF was initiated either by the third (62%) or the fourth beat (38%) of a PV tachycardia. **Conclusions:** IRAF is usually triggered by a burst of PV tachycardia rather than by a single APD, regardless of the coupling interval. These observations suggest that PV tachycardias may play a critical role in the initiation of IRAF. Therefore, IRAF may be a marker of patients with AF who may benefit from isolation of the PV's.