TOPIC 05 – Rythmic disease and stimulation

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Cinical impact of the implantable loop recorder on patients with isolated syncope, bundle branch block and a negative work-up including an electrophysiological study. A prospective randomized study.

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Background: Little is known in patients with isolated syncope, bundle branch block[BBB], and a negative work-up including an electrophysiological study [EPS]. Norandomized study exists in this subset of patients comparing a strategy of waiting and the implantable loop recorder (ILR).

Objectives: The aim of this multicenterprospective study was to evaluate in a population with BBB and a negative EPS: [1]the prognosis impact of an ILR [group I] compared with the waiting clinical follow-up approach [group II]; [2] the incidence of relevant electrical events.

Methods and Results: From January 2005 to December 2009, 78 pts were included. The mean follow-up was 19±12 months. Population characteristics were: mean age of 76±8 years old; 30 female [38.5%]; 18 cardiomyopathy [23%]; previous AFib [15.4%]; LVEF [56.5±11%]; ECG abnormalities [34 left BBB;11 right BB and 33 bifascicular block] and a negative EPS [mean HV interval of 55±6 ms]. Seventeen pts [22%] developed a significant arrhythmic event: 1 ventricular tachycardia [1.3%], 1 sudden death [1.3%], 2 AV block II [2.6%], 8 AV block III [10.25%] and 5 sick sinus syndrome [6.4%]. The number of events detected was significantly different between ILR group [n=13; 16.6%] and clinical follow-up [n=4; 5.1%]; (p=0.02). The Kaplan-Meier estimates of the probability of remaining free of arrhythmia events and the Logrank test [Figure]. In accordance with the final diagnosis, 15 patients received a pacemaker therapy.

Conclusions: This randomized prospective study found that in patients with isolated syncope, BBB and a negative EPS, the % of arrhythmic events is close to 22% at 2 years. A strategy with an ILR is superior to the clinical follow-up for detecting recurrent events and may influence the prognosis of these patients.

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Assessment of electro-anatomical activation using non invasive electrocardiographic imaging in patients with repaired tetralogy of Fallot and biventricular pacing.

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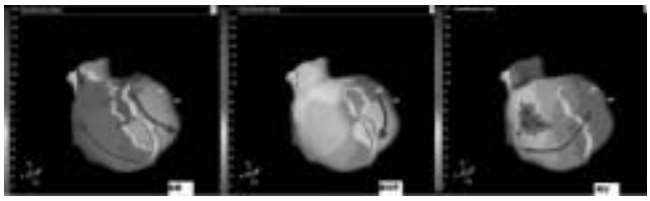
Introduction: Right heart failure is a common feature in patients with repaired tetralogy of Fallot (TOF), right ventricular (RV) dysfunction and right bundle branch block (RBBB). Biventricular pacing has been described as a potentially useful therapeutic in some cases. We aimed to investigate the changes in electrical epicardic activation using non invasive electrocardiographic imaging (ECGI) in different pacing configurations.

Methods: eight adults with TOF, clinical signs of RV dysfunction and RBBB underwent implantation of a BVP device. Electrocardiographic imaging (ECGI) was applied during intrinsic activation (IA), right ventricular (RV) stimulation and BVP.

Results: 1) In spontaneous rhythm, the activation maps were compatible with a RBBB aspect with major dispersion of the activation of the right ventricle (RV activation time 114 ± 38 ms).
2) In RV pacing, right ventricular activation time is shorter but left ventricular activation is significantly delayed (110 ± 27 vs 72 ± 22 ms, p<0,05). During BVP, the activation maps demonstrated reduction in the activation of the right ventricle without alteration of the activation of the left ventricle.
3) Global activation times in BVP are significantly reduced compared to sinus rhythm and RV pacing (122 ± 20 vs 147 ± 28 ms, p<0,05).

Conclusion: ECGI can be useful to assess non invasively the synchrony and electrophysiological substrate of patients with complex congenital heart disease.



Activation maps, anterior view.

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Does atrial differences in endothelium damage, leukocyte and platelet activation contribute to chamber specific thrombogenic status in patients with atrial fibrillation ?

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Background: In atrial fibrillation (AF), the reasons why most of the thrombi form in the left atrium are mainly unknown. In the vasculature, endothelial damage together with platelet activation and inflammation contribute to initiation of blood coagulation and thrombus growth.

Objective: The purpose of this study was to investigate whether atrial-specific differences in endothelial damage, leukocyte activation, platelet stimulation occur in patients with AF.

Methods: Twenty patients (15 men, 5 women; age 55 ± 8 years, 15 paroxystic AF, 5 persistent AF) with AF undergoing ablation were investigated. Blood samples from the left and right atrium were obtained at the start of the procedure. Procoagulant microparticles (MPs), reliable markers of vascular damage were measured by capture assays. Their procoagulant abilities were quantified by functional prothrombinase assay and their cellular origin were determined (endothelium, platelet, leukocyte). In addition, platelet reactivity was evaluated by whole blood flow cytometry for expression of platelet P-selectin (CD62P), active glycoprotein IIbIIIa receptor (PAC-1). Platelet aggregation was evaluated using Arachidonic acid (AA), ADP, TRAP and collagen-induced whole blood aggregometry.

Results: No atrial-specific differences in the levels of total procoagulant MP, leukocyte-derived-MP and platelet-derived MP could be evidenced. Conversely, endothelial-derived MPs (CD105+) were slightly elevated in the right atrium (RA 0.96 ± 0.53 vs. LA 0.80 ± 0.45 nm PhtdSer Eq.; p = 0.041) . Likewise, collagen-induced platelet aggregation was evidenced in the right atrium (Collagen 1 mg/l RA: 48 ± 33 % vs LA 37 ± 29%; p 0.035; collagen 2,5 mg/l RA: 76 ± 25 % vs LA: 60 ± 29%; p = 0.001).

Conclusions: In patients with AF, endothelial damage and collagen-induced platelet aggregation appear slightly more pronounced in the right atrium. Our data did not substantiate the view that chamber specific enhanced thrombogenic status could be a reliable explanation for the increased propensity for thrombus formation observed in the left atrium in AF patients.