Patients were followed from 1 month to 13 years (mean 4 ± 4 years). Atrial pacing and programmed atrial stimulation with 1 and 2 extrastimuli salvos of AF (n=25), wide-QRS tachycardia suspected of atrial origin (n=6). AF (group I). Remaining 124 patients had no documented AF (group II) and sinus rhythm; 35 patients had presented one episode of documented sustained cardio or fibrillation (AF) by esophageal EPS.

The purpose of study was to evaluate the cause of palpitations in patients with negative Holter monitoring or when the cardiac event monitor is not interpretable. The sensitivity of AF and 2 with negative EPS (NS). Two group I patients (6%) with induced AF were more likely to be female (OR=1.35, CI=1.00-1.83, p=0.05), more likely to be treated with a class I AAD (45.5% vs 7.7%), isolated or associated with beta-blockers and more likely tended to be treated with Amiodarone (36.5% vs. 31.2%, p=0.08). After ablation, 260 (23.2%) patients experienced AF. In a multivariable model, AF prior to ablation (OR=1.90, CI=1.42-2.54, p<0.001) and female gender (OR=1.77, CI=1.29-2.42, p<0.001) were associated with a higher risk of AF after ablation. In patients without prior AF, Class I AAD and Amiodarone prior to AFL were independently associated with higher risk of AF after ablation (OR=2.11, CI=1.15-3.88, p=0.02 and OR=1.60, CI=1.08-2.36, p=0.02 respectively). Patients with previously diagnosed AF were more likely to be treated with Class I AAD (45.5% vs. 7.7%), isolated or associated with beta-blockers (data not shown), and more likely tended to be treated with Amiodarone (36.5% vs 31.2%, p=0.08).

Conclusions: AF occurrence after AFL ablation is frequent (>20%), especially in patients with a history of AF, in female patients, and in patients treated with Class I antiarrhythmics/Amiodarone prior to AFL. The risk was similar in patients treated with class I or III drug. In a patient referred for AFL ablation without known AF before AFL, treated with AAD, the follow-up should be careful because these patients appear at high risk of AF occurrence.

Methods: Esophageal EPS was performed in 159 patients, 72 males, 87 females, aged from 19 to 89 years (mean 56±16) with a normal ECG in sinus rhythm; 35 patients had presented one episode of documented sustained AF (group I). Remaining 124 patients had no documented AF (group II) and were studied for not documented tachycardia (n=70), not documented tachycardia associated with dizziness/syncope (n=25), unexplained stroke and salvos of AF (n=25), wide-QRS tachycardia suspected of atrial origin (n=6). Atrial pacing and programmed atrial stimulation with 1 and 2 extrastimuli were performed in control state (CS) and after infusion of isoproterenol. Patients were followed from 1 month to 13 years (mean 4±4 years).

Results: Among group I, AF was induced in 21 patients (60%). Sustained AF was induced in CS (n=50) or after isoproterenol (n=64) in all group II patients. The follow-up indicated that 7 group I patients (21%) had recurrent AF/atrial flutter requiring ablation, 5 patients with induced AF and 2 with negative EPS (NS). Two group I patients (6%) with induced AF died from a cardiac cause. Among group II, 20 patients (16%) presented documented AF/atrial flutter and 14 of them required an ablation. Five group II patients (4%) died from a cardiac cause. The sensitivity of esophageal EPS to reproduce AF was 60%. Its positive predictive value to predict the occurrence of AF in symptomatic patients without documented AF was 16%. The positive value to predict AF and cardiac death was 24%.

Conclusions: Despite an average sensitivity for the induction of AF in patients with documented AF, the risk of subsequent AF and/or cardiac death was relatively high in these patients and in symptomatic patients without documented AF but with induced AF. These patients require a careful follow-up.