Results: 4885 patients were included. 56% were men and the mean age was 53. After a mean follow up of 8.6 years, 129 deaths were recorded. In multivariate analysis, independent RF were age (Hazard Ratio (HR)=1.06, p<0.001, 95% CI [1.04-1.08]), gender (HR=5.95, p<0.001, 95% CI [3.48-10.19]) , diabetes (HR=2.49, p<0.001, 95% CI [1.49-4.16]), hypertension (HR=1.44, p=0.05, 95% CI [1.00-2.08]), LDL-cholesterol>4 mmol/L (HR=2.04, p<0.001, 95% CI [1.40-2.96]), smoking (HR=2.19, p<0.001, 95% CI [1.49-3.20]), lower educational level (HR=1.81, p=0.01, 95% CI [1.18-2.79]) and resting heart rate> 65 bpm (HR=1.54, p=0.02, 95% CI [1.06-2.24]). A good calibration was obtained (p value NS for Hosmer-Lemeshow χ^2 test). The median predicted risk of mortality was 6.52% and was not significantly different to the observed risk of all cause mortality (6.60%; 95%CI [5.22%-8.34%]).

Conclusions: RHR can be used to predict all-cause mortality in primary prevention and might be evaluated as a simple predictive tool in current practice.

0019

Significance of atrial fibrillation/tachycardia induced by esophageal stimulation

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Esophageal electrophysiological study (EPS) is an easy means to evaluate the cause of palpitations in patients with negative Holter monitoring or when cardiac event monitor is not interpretable. The purpose of study was to evaluate the clinical significance and the diagnosis value of inducible atrial tachycardia or fibrillation (AF) by esophageal EPS.

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=23), 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.

0029

Does the class of antiarrhythmic drug change the risk of atrial flutter in patients with atrial fibrillation or the risk of atrial fibrillation after atrial flutter ablation?

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Atrial fibrillation (AF) and atrial flutter (AFL) are frequently associated. The purpose of the study was to look for the effect of antiarrhythmic drugs (AAD) on the risk of AF occurrence after radiofrequency ablation of AFL.

Methods: 1121 patients, mean age 64±12 years, were referred for AFL ablation. History, data of echocardiography, antiarrhythmic drug (AAD), were collected. Patients were followed from 3 months to 10 years (mean 2.1±2.7 years). AAD was stopped after ablation except in patients with previous AF before ablation or continued otherwise.

Results: 857 patients received an AAD (n=637) or a betablocker (n=221). 356 patients (31.7%) had a history of AF prior to AFL ablation. Patients with AF prior to ablation 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 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 ablation 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 a 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.

0034

Preexcitation syndrome and atrioventricular nodal reentrant tachycardia: coincidence or not?

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Background: Reciprocating tachycardia which occurs in patients with a preexcitation syndrome (PS) generally is directly related to the presence of the accessory pathway (AP) and is called atrioventricular re-entrant tachycardia (AVRT). The purpose of the study was to evaluate the incidence of re-entrant tachycardia of other nature among patients with a PS.

Methods: 785 patients with paroxysmal tachycardia were admitted AP ablation, 294 patients with a concealed AP (group I) and 491 patients with a Wolff-Parkinson-White syndrome (WPW) (group II). Programmed atrial stimulation was performed in the control state and if necessary after isoproterenol to induce the clinical tachycardia and determine its mechanism.

Results: AVRT was induced in 760 patients (97%), 282 of group I (96%) and 478 of group II (97%) (NS). Atrioventricular nodal re-entrant tachycardia (AVNRT) was induced in 13 group I patients (4.6%) and 12 group II patients (2.5%) (NS; 0.11). In 9 group I patients (3%) and in 4 group II patients (1%) (p<0.015), both AVRT and AVNRT were induced. In patients with only induced AVNRT, slow pathway ablation was performed and accessory pathway was respected because there was no inducible tachycardia using AP and the conduction over AP was poor. These patients remained free of symptoms after ablation of AV node slow pathway. Among this population 3 families were identified as having either AVRT or AVNRT.

Conclusions: In patients with concealed or patent accessory pathway and complaining of paroxysmal tachycardia, a careful evaluation of the mechanism of tachycardia is required before ablation. Patients with concealed conduction over an AP have more frequently an association of AVRT and AVNRT than patients with a patent preexcitation syndrome. Rarely AVNRT can be the only mechanism of symptoms.