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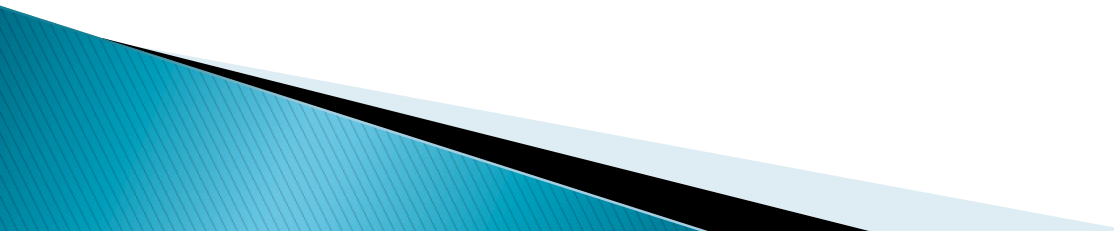
## **Dr Fabien SQUARA**

## **Dr Guillaume THEODORE**

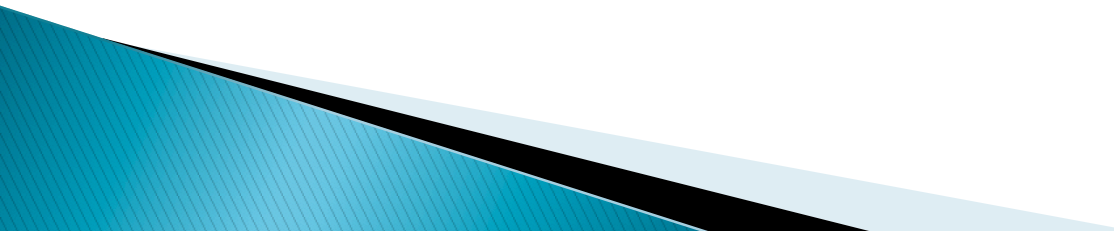


UNIVERSITÉ CÔTE D'AZUR 

« PAFIYAMA » syndrome



**P**aroxysmal  
**AF** In **Y**oung  
**A**nd **M**iddle-aged  
**A**thletes



# Définition

PAFIYAMA syndrome criteria [32,86,135,142,162–164].

## Major criteria

1. Onset as paroxysmal AF
2. Age usually  $\leq 60$  years, male sex
3. Prolonged practice of SEE ( $\geq 6$  to 8 h/week with intensity greater than 60% of maximum heart rate, for 6+ months)
4. Preserved ejection fraction ( $\geq 55\%$ )

## Minor criteria

1. ST-segment elevation at the J-point (STE)  $\geq 0.1$  mm in 2 leads
2. T-wave inversion in 2 leads
3. Increased vagal tone (sinus bradycardia, prolonged PQ time, first degree AV-block)
4. LA enlargement
5. LV hypertrophy
6. Increased LV wall thickness and LV mass
7. Normal, or even supranormal, diastolic function

## Absence of common AF-risk factors

1. No overweight (BMI  $> 25$  kg/m<sup>2</sup>) or obesity (BMI  $> 30$  kg/m<sup>2</sup>)
2. No arterial hypertension at rest
3. No smoking habits
4. No diabetes

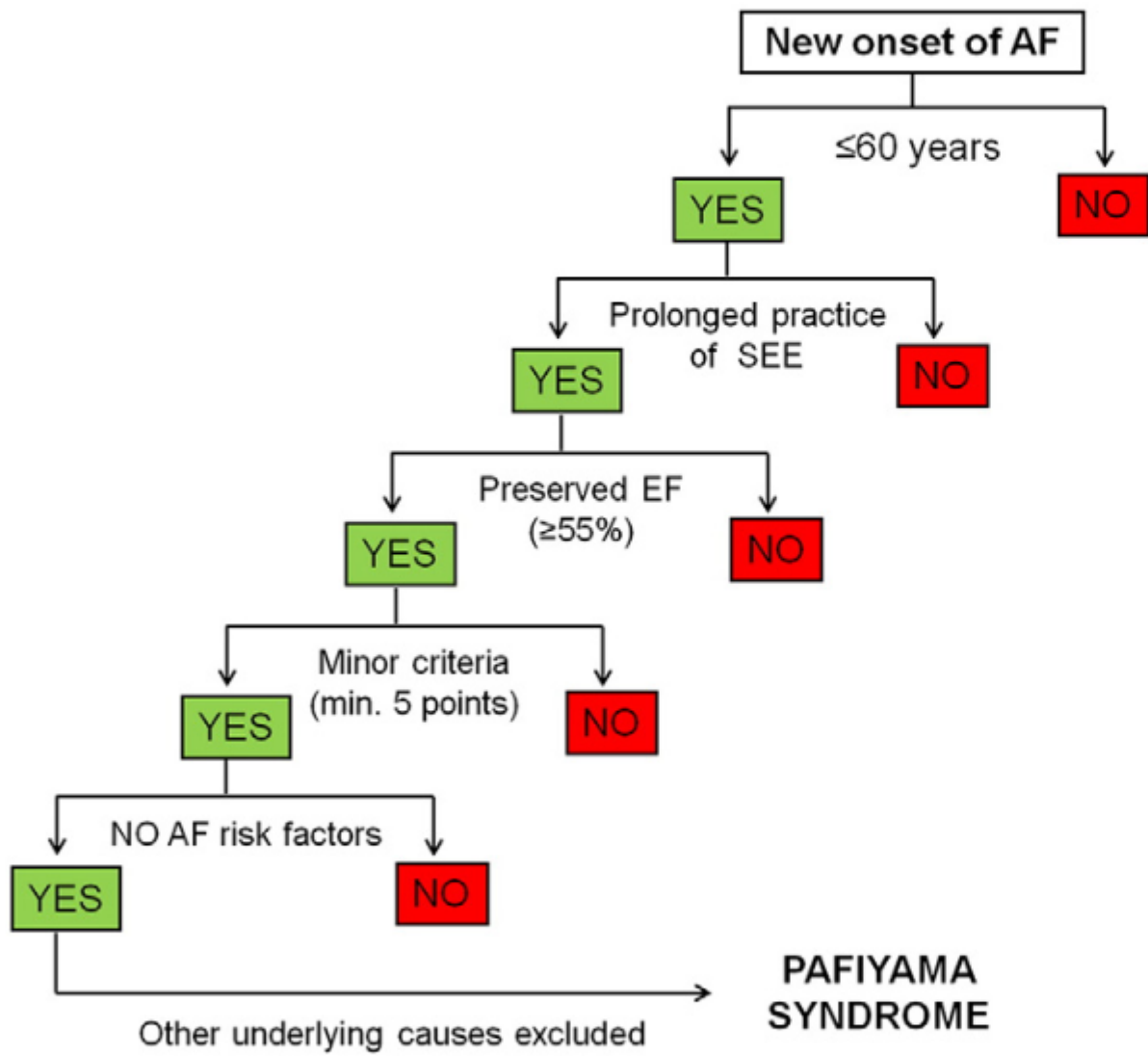
## Other underlying causes to exclude

1. Metabolic or hormonal diseases (hyperthyroidism, pheochromocytoma)
2. Dilated or hypertrophic cardiomyopathy
3. Pericarditis
4. Coronary artery disease
5. Wolff-Parkinson-White syndrome, Brugada syndrome, long QT syndrome, arrhythmogenic cardiomyopathy or catecholaminergic ventricular tachycardia
6. Performance-enhancing agents or illicit drug use
7. Obstructive sleep apnea
8. Electrolyte abnormalities

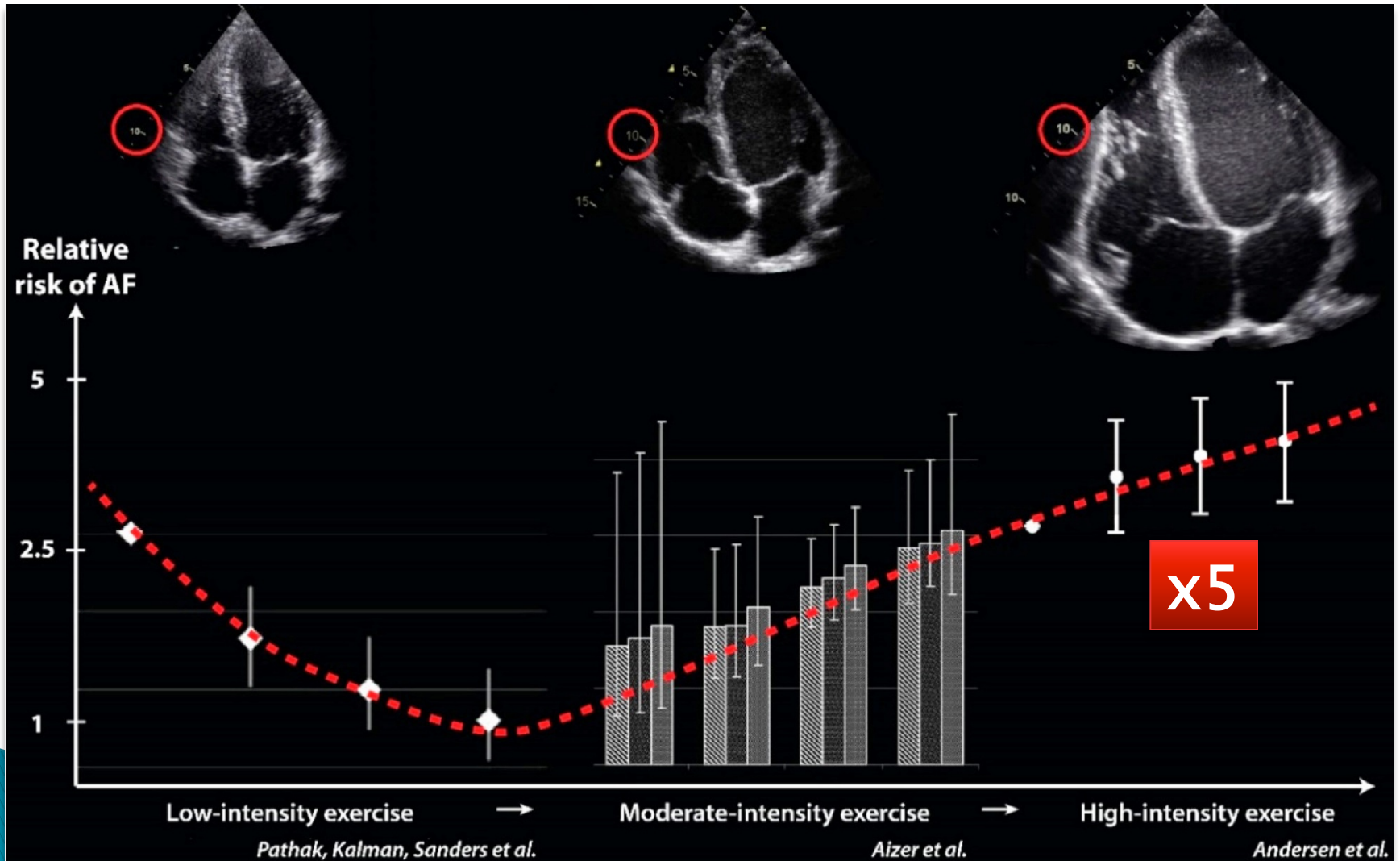
Abbreviations: AF, atrial fibrillation; AV, atrioventricular; BMI, body mass index; EF, ejection fraction LA, left atrium; LV, left ventricle; SEE, strenuous endurance exercise; STE, ST-segment elevation.

[\*Atrial fibrillation in highly trained endurance athletes – Description of a syndrome.\*](#)

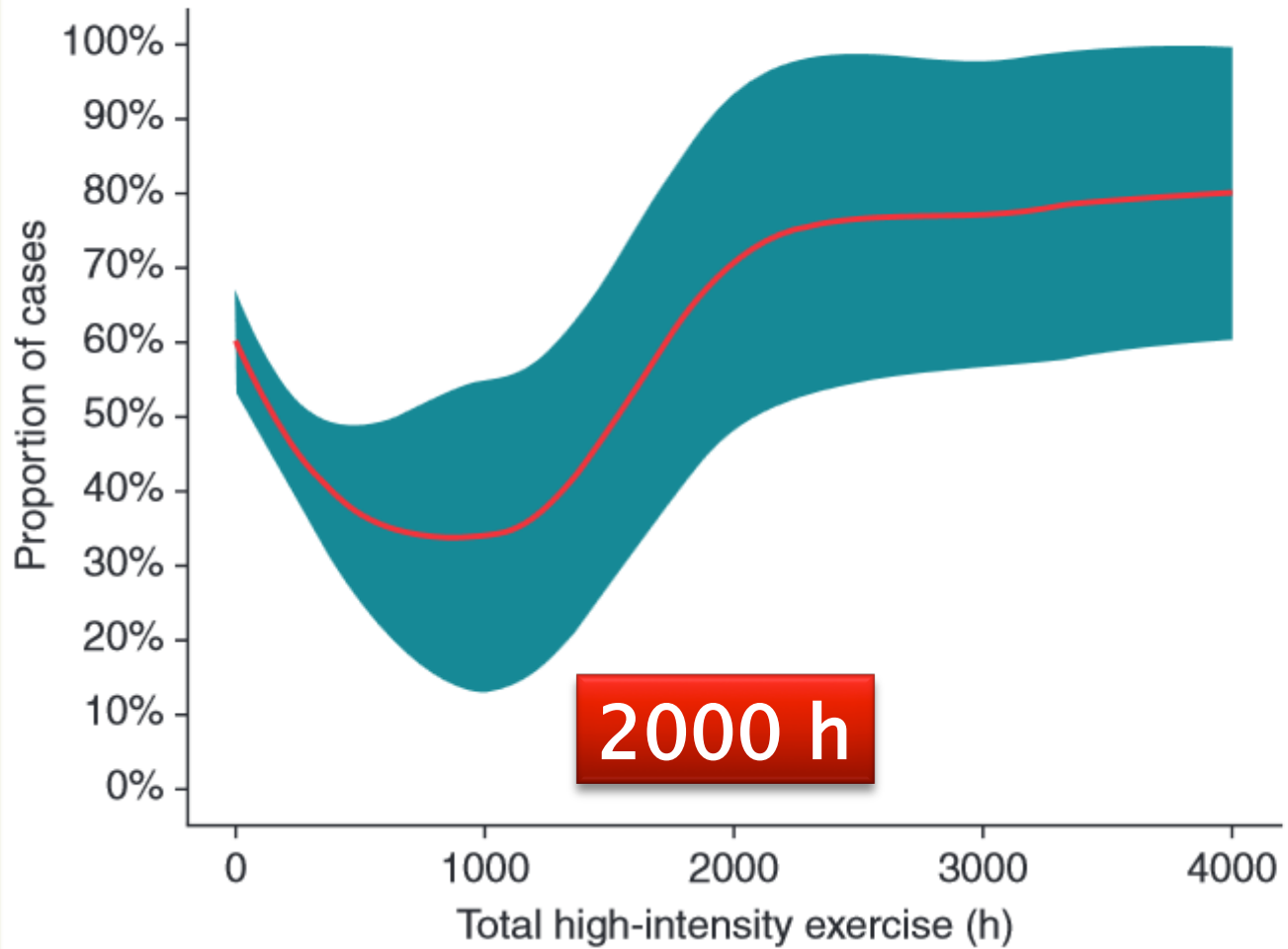
Sanchis-Gomar F and all.  
Int J Cardiol. 2017 Jan 1;226:11–20



# Incidence



# Incidence



# Incidence

Study Type	Number of subjects	Age/Gender	Type of exercise	Prevalence of AF in athletes/ controls (%)
Karjalainen et al <sup>15</sup>	795	35-39 years/ Male	Cross country Running	5.3/0.9
Baldesberger et al <sup>17</sup>	196	~66 years/ Male	Cyclists vs. golfers	10/0
Mont et al <sup>18</sup>	216	<65 years/ Male + Female	Endurance athletes	63/15
Elosua et al <sup>16</sup>	109	41-55 years/ Male	Endurance athletes	32/14
Heidbuchel et al <sup>22</sup>		60 years/83% Male, 17% Female	Cycling, running, or swimming	
Molina et al <sup>18</sup>	557	48 years	Marathon runners vs. sedentary	5/0.7
Grimsmo et al <sup>11</sup>	78	54-62 years- Group I 72-80 years- Group II 87-92 years- Group III	Cross-country runners, skiers	12.8



**FA du sportif**

**=**

**normal ?**

# ECG du sportif : Pas de FA

## Normal ECG Findings

- Increased QRS voltage for LVH or RVH
- Incomplete RBBB
- Early repolarization/ST segment elevation
- ST elevation followed by T wave inversion V1-V4 in black athletes
- T wave inversion V1-V3  $\leq$  age 16 years old
- Sinus bradycardia or arrhythmia
- Ectopic atrial or junctional rhythm
- 1° AV block
- Mobitz Type I 2° AV block

## Borderline ECG Findings

- Left axis deviation
- Left atrial enlargement
- Right axis deviation
- Right atrial enlargement
- Complete RBBB

## Abnormal ECG Findings

- T wave inversion
- ST segment depression
- Pathologic Q waves
- Complete LBBB
- QRS  $\geq$  140 ms duration
- Epsilon wave
- Ventricular pre-excitation
- Prolonged QT interval
- Brugada Type 1 pattern
- Profound sinus bradycardia  $<$  30 bpm
- PR interval  $\geq$  400 ms
- Mobitz Type II 2° AV block
- 3° AV block
- $>$  2 PVCs
- Atrial tachyarrhythmias
- Ventricular arrhythmias

**No further evaluation required** in asymptomatic athletes with no family history of inherited cardiac disease or SCD

In isolation

2 or more

**Further evaluation required** to investigate for pathologic cardiovascular disorders associated with SCD in athletes

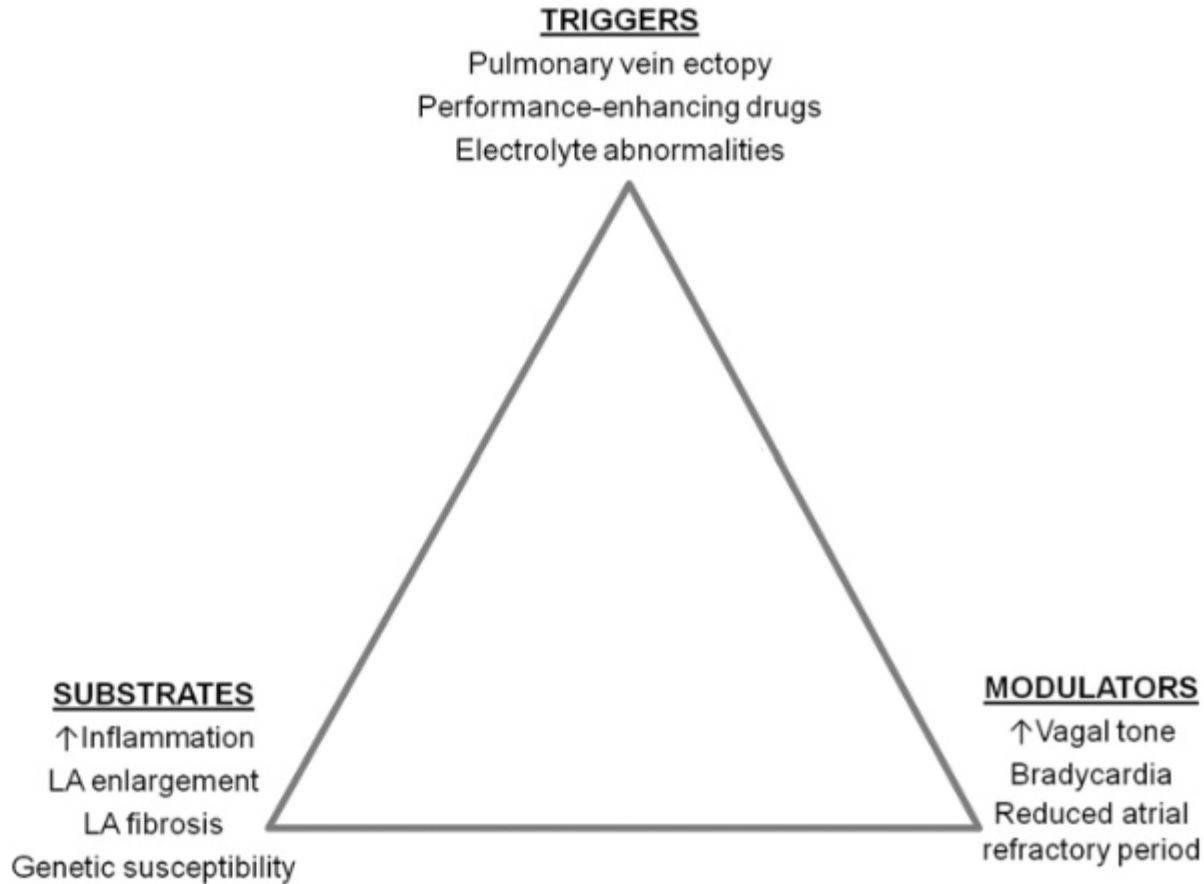
## Abnormal ECG findings in athletes

These ECG findings are **unrelated to regular training or expected physiological adaptation to exercise**, may suggest the presence of pathological cardiovascular disease and require further diagnostic investigation.

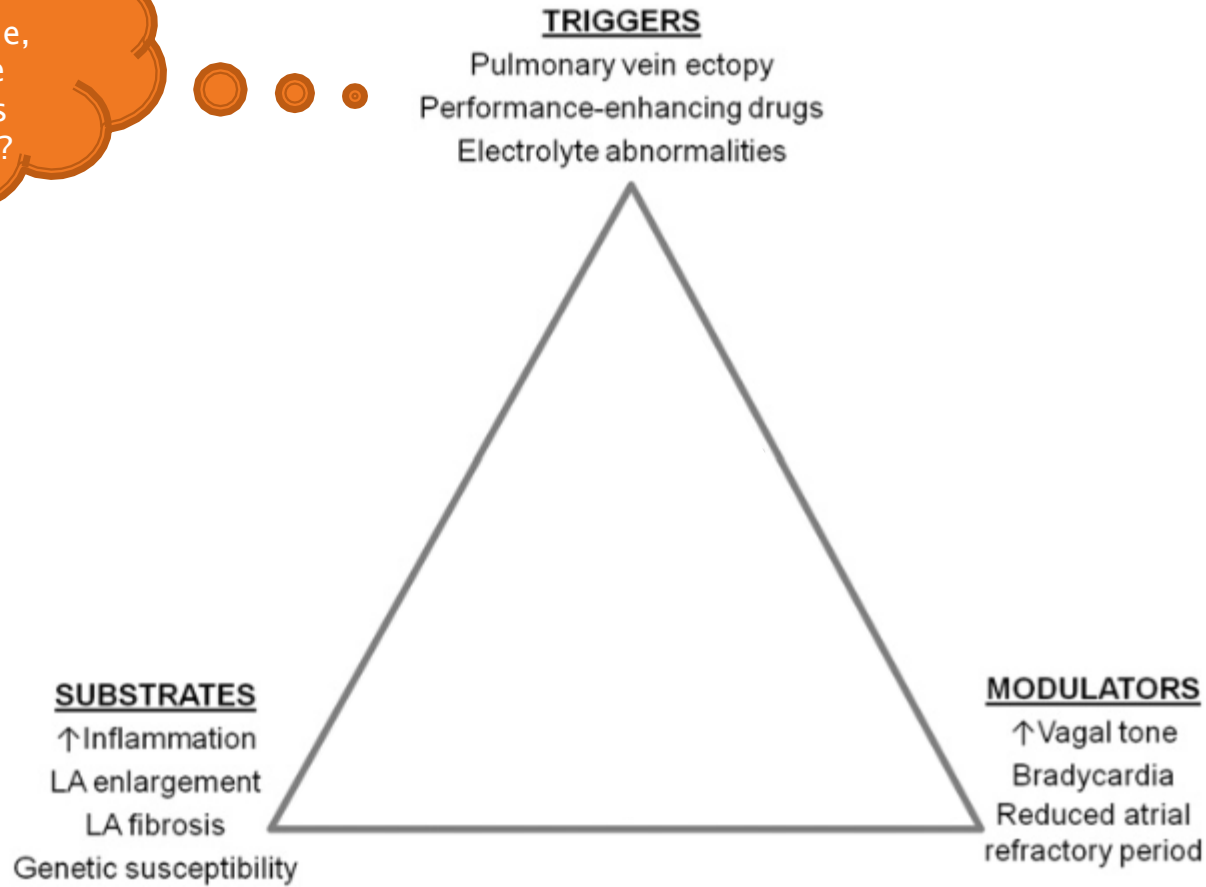
# Tous les sportifs ne font pas de la FA



# Que faut-il rechercher ???



Bilan  
biologique,  
prise de  
produits  
dopants?





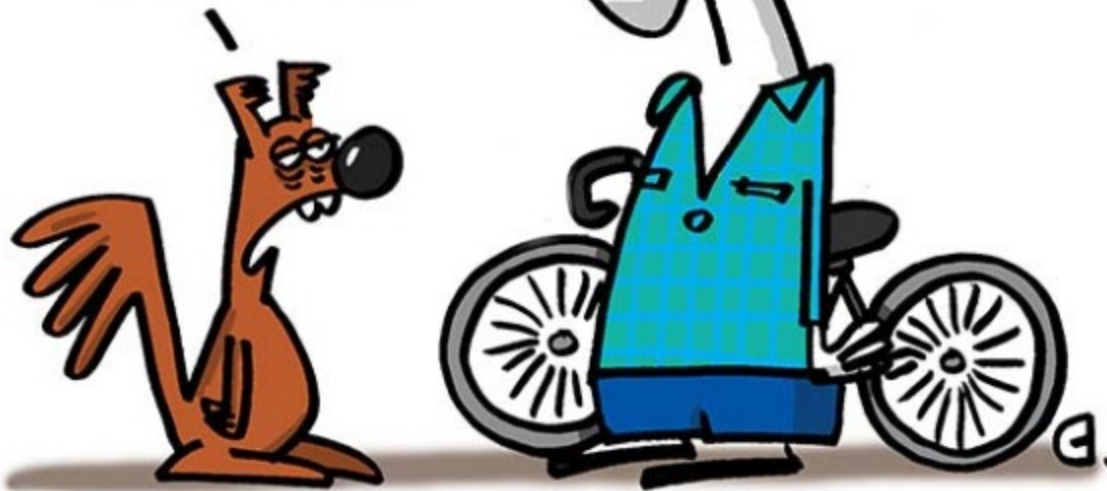
EXTRASYSTOLIE  
ATRIALE

Pas de preuves

**Table 4** Supraventricular arrhythmias

	<b>FAs (n = 62)</b>	<b>Controls (n = 62)</b>	<b>P-value</b>
<hr/>			
Atrial premature complexes per 24 h			
Median (range)	18 (0–2616)	17 (1–6135)	0.35
<hr/>			
Atrial flutter or fibrillation, n (%)	6 (10)	0	0.028

VOUS QUI AVEZ  
TRAVAILLÉ DANS  
LE CYCLISME,  
VOUS N'AURIEZ  
RIEN POUR ME  
BOOSTER?



**Table 1. Performance-Enhancing Drugs and Potential Cardiovascular Side Effects**

Substance Group	Examples	Direct Cardiovascular Side Effects
Oxygen-carrying modulators	Erythropoietin Erythropoietin-stimulating agents (AICAR, GW 15-16) Erythropoietin receptor agonists Blood doping Synthetic blood	Thromboembolic events Myocardial infarction Stroke Hypertension
Oxygen dissociation curve modulators	Cobalt RSR13	Cardiomyopathy
Anabolic agents	Human growth hormone, insulin-like growth factor-1 Endogenous anabolic steroids (eg; testosterone) and their metabolites (eg, 5-androstenedione; 7 $\beta$ -hydroxy-dehydroepiandrosterone) and exogenous steroid analogues (eg; stanozolol, nandrolone).	Dyslipidemia Hypertension Pathological cardiac Hypertrophy/cardiac fibrosis Arrhythmias
$\beta_2$ -Adrenergic receptor antagonists	Clenbuterol	Arrhythmias in animals
Phosphodiesterase type 5 inhibitors	Sildenafil*	Unknown in athletes
Selective androgen receptor modulators	Thymosin beta 4 Andarine Ostarine Multiple "designer peptides"	Largely unknown
Selective estrogen receptor modulators	Tamoxifen (counteract negative side effects of anabolic agents)	Venous thrombosis, pulmonary embolism
Hormone/metabolic modulators	Meldonium (mildronate) Corticosteroids Insulin and mimetics Thyroxine $\beta$ -Alanine* Creatine* L-Carnitine*	Hypertension, hyper- or hypoglycemia, dyslipidemia, many agents with untested safety profiles
Amphetamines/stimulants	Methylphenidate, modafinil	Unknown in athletes
Others	Glycerol trinitrate* Tramadol* Opiates* (enables athletes to suppress pain in training and racing) Iron supplementation (especially in combination with altitude or O <sub>2</sub> -carrying modulators)* Diuretics (masking agents/making weight) Epitestosterone (masking agent, normalizes testosterone to epitestosterone ratio)	Unknown in athletes

Production facilitée par l'hormone de croissance  
Efficacité augmentée par l'apport de fer

**LE DOPAGE  
JE CONNAIS PAS MOI**



**SUBSTANCES  
DOPANTES**

**5% des FA  
du sportif ?**

(2-30%)



**HYPOKALIEMIE  
HYPOMAGNESEMIE**

**Pas de preuves**

**HYPONATREMIE**



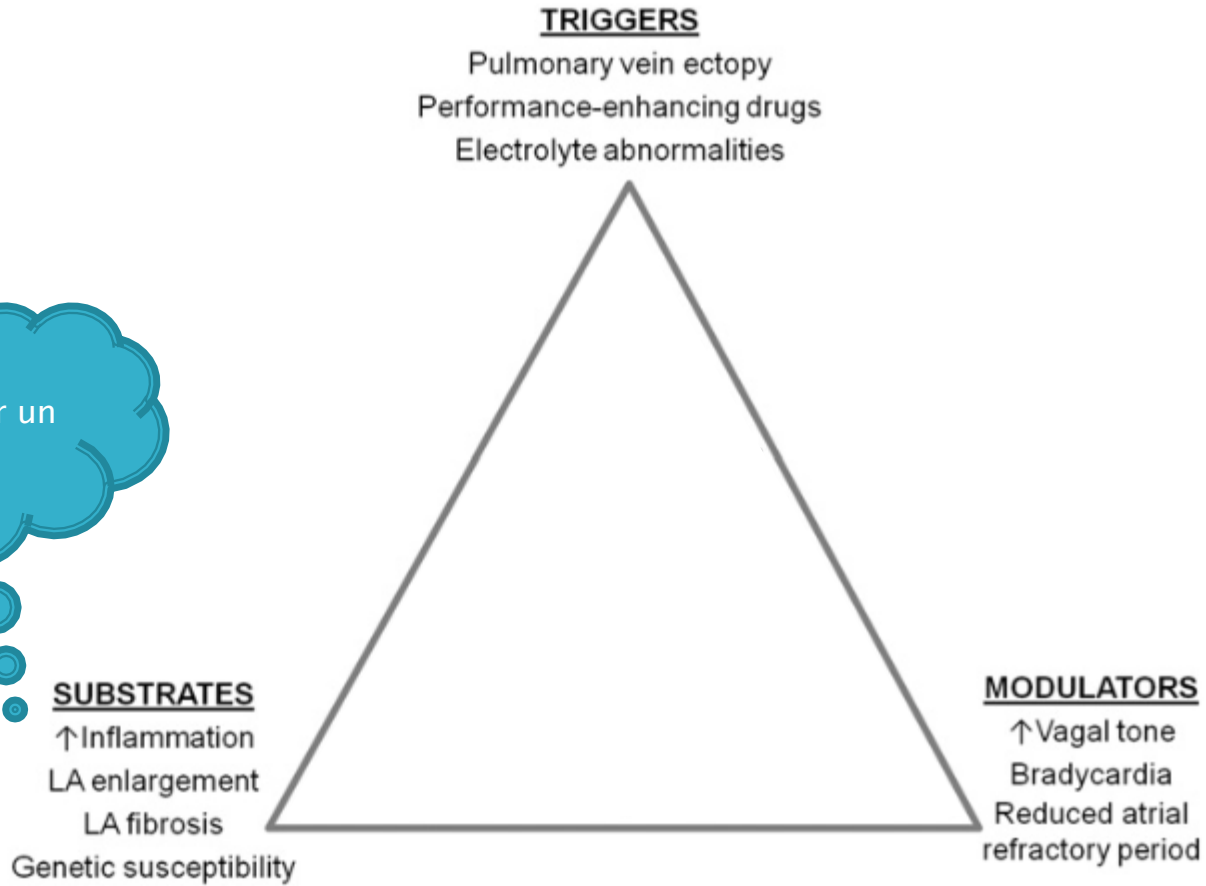
**9%**

**DYSKALIEMIE  
DYSMG**



**NS**

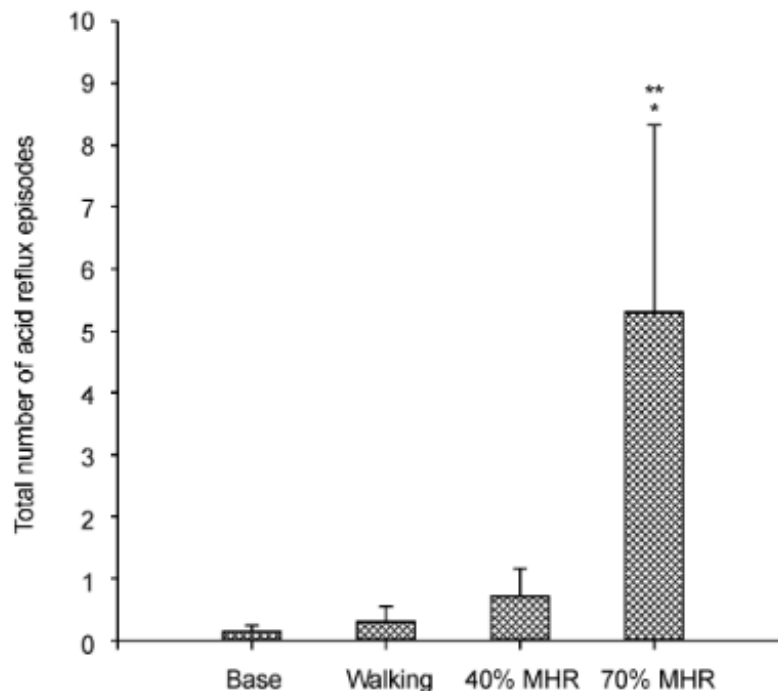
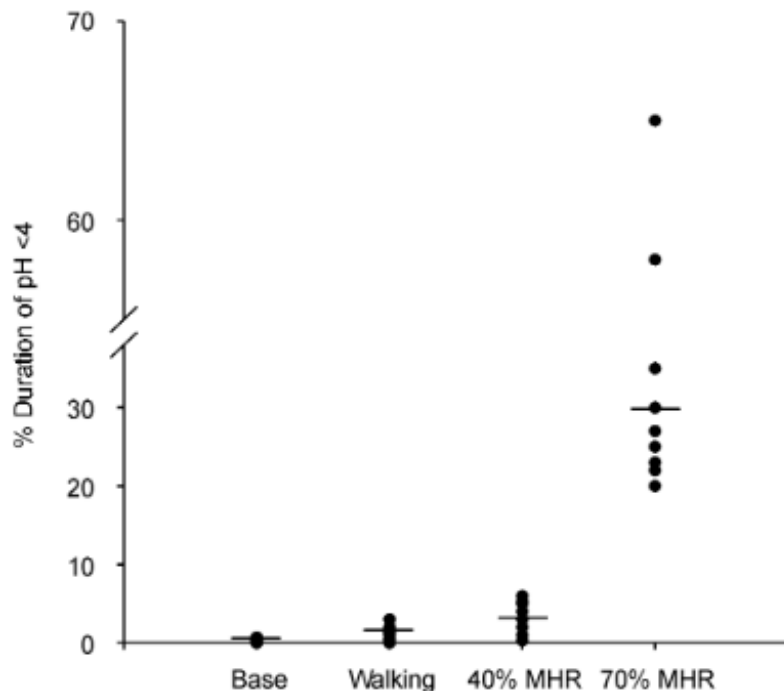
Rechercher un  
RGO



# RGO

SPORT  
→ ↓ pH

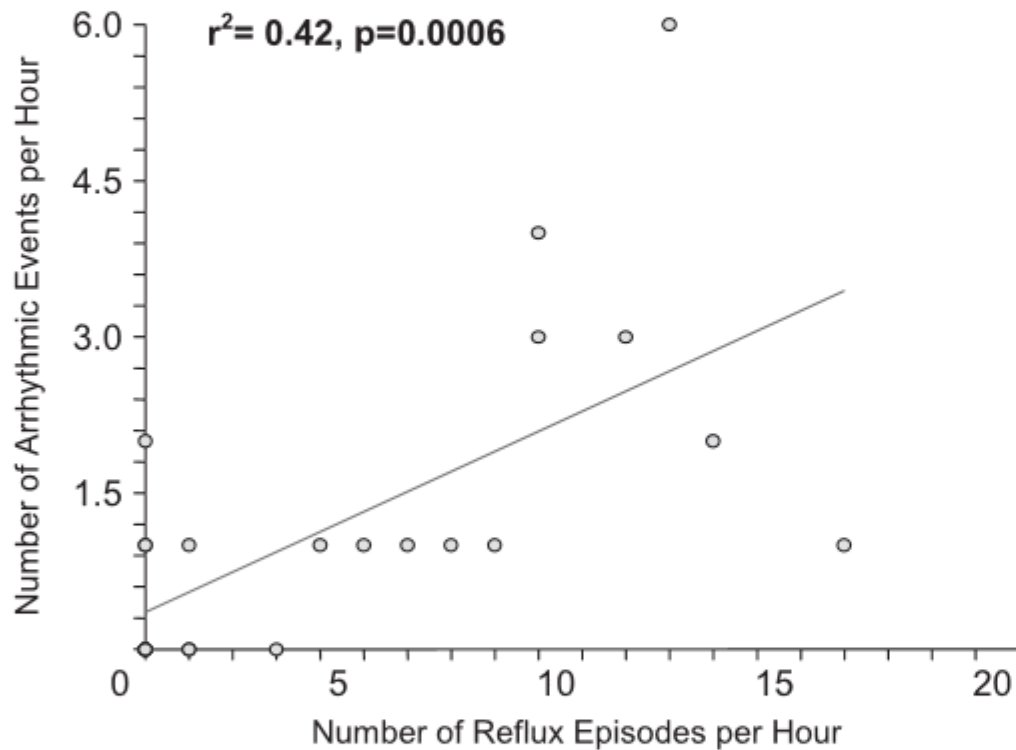
Effect of Running on Esophageal Motility and Gastroesophageal Reflux



# RGO

RGO → FA

15% des FA  
du sportif ?



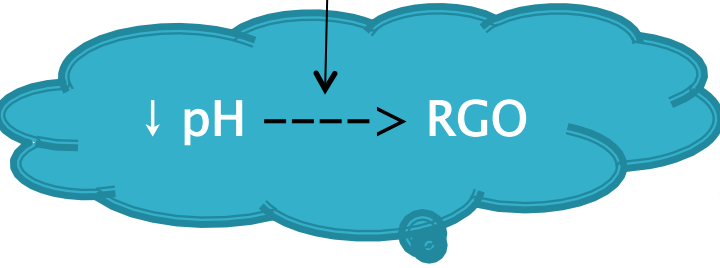


Europace  
doi:10.1093/europace/euw092

# Atrial fibrillation and gastroesophageal reflux disease: the cardiogastric interaction

Dominik Linz<sup>1\*</sup>, Mathias Hohl<sup>1</sup>, Johanna Vollmar<sup>2</sup>, Christian Ukena<sup>1</sup>, Felix Mahfoud<sup>1</sup>, and Michael Böhm<sup>1</sup>

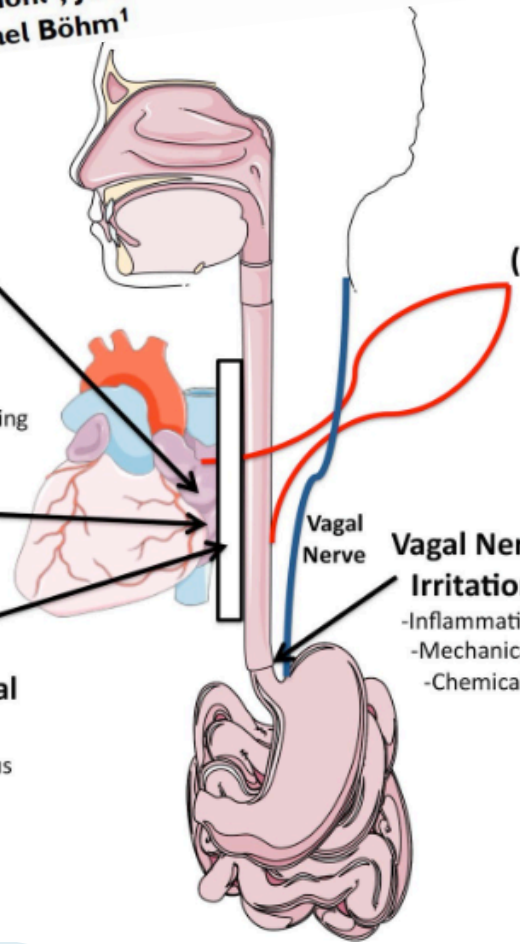
Clairance oesophagienne



**Inflammation**  
Local:  
Pericarditis/  
Atrial Myocarditis  
Systemic:  
Structural Atrial Remodeling

**Atrial Mechanical Irritation**

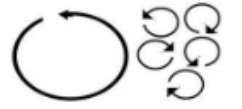
**Chemical & Mechanical Irritation Of**  
-Perioesophageal Nerve Plexus  
-Oesophageal Vessels  
-Lymph Nodes



**Neural Reflex Arcs (Eosophageal-Cardiac)**

**Vagal Nerve Irritation**  
-Inflammation  
-Mechanical  
-Chemical

Vagal Activity ↑↑  
Action Potential ↓



# RGO en pratique

## RGO

**Corrélation forte avec les épisodes de FA**

↓ **Relaxation sphincter inférieur œsophage**

↑ Pression abdominale

Modification de la jonction oesophago - gastrique

**↓RGO = ↓ épisodes de FA**

# RGO en pratique

## EN PRATIQUE

RGO atypique : Pas de brûlure gastrique  
RGO pseudo angineux : Les innervations  
oesophagienne et cardiaque se croisent.

### FOGD

- Hernie hiatale ? Favorisant le RGO
- Oesophagite ?
- EBO ? (RGO atypiques)

Si négatif : **pH métrie**

Impédancemétrie oesophagienne

# Traitement du RGO efficace sur la FA

Atrial fibrillation associated with gastroesophageal reflux disease: Data from literature

Ref.	Type of study	Patients (n)	Diagnosis of GERD and AF	Conclusion
Weigl et al[14], 2003	Retrospective	GERD and lone AF (n = 18)	Gastroscopy ECG	PPIs therapy could be beneficial in a subgroup of patients with AF, with fewer side effects
Cuomo et al[15], 2006	Prospective, mechanistic	GERD and arrhythmias (n = 32) Control group (n = 9)	Upper endoscopy Simultaneous 24-h pH-meter and Holter monitoring	PPIs therapy seems to improve GERD and cardiac symptoms in dysrhythmic patients in whom esophageal acid stimulus induces cardiac autonomic reflexes
Bunch et al[16], 2008	Prospective, epidemiologic, randomized	Olmsted County residents (n = 5288)	Self-report questionnaire Medical records ECG ICD-9 codes	There was no association between GERD and AF. Patients with esophagitis are more predisposed to develop AF
Kunz et al[17], 2009	Retrospective, epidemiologic	n = 163627	ICD-9 codes	GERD is associated with a higher risk of AF
Shimazu et al[18], 2011	Retrospective	GERD and AF (n = 86)	Questionnaire F-scale ECG, Holter monitoring	AF is an independent risk factor for GERD
Huang et al[19], 2012	Prospective, epidemiologic	GERD (n = 29688)Control group (n = 29597)	ICD-9 codes Endoscopy or 24-hs pH-meter ECG, Holter monitoring	Taken independently, GERD is associated with a higher risk of future AF in a nationwide population-based cohort
Reddy et al[21], 2013	Prospective, matched, case-control, mechanistic	GERD and AF (n = 10) Control group (n = 30)	Symptoms questionnaire	Patients with GERD are more likely to have AF and a positive vagal response during radiofrequency catheter ablation
Kubota et al[22], 2013	Cross-sectional	n = 201Control group (n = 278)	Questionnaire F-scale ECG, Holter monitoring	Significant correlation between nonvalvular AF and symptomatic GERD

AF: Atrial fibrillation; GERD: Gastroesophageal reflux disease; PPIs: Proton pump inhibitors; ECG: Electrocardiogram; ICD: International statistical classification of diseases.



Le dopage (5%)  
et RGO (15%)  
n'explique pas  
toutes les FA

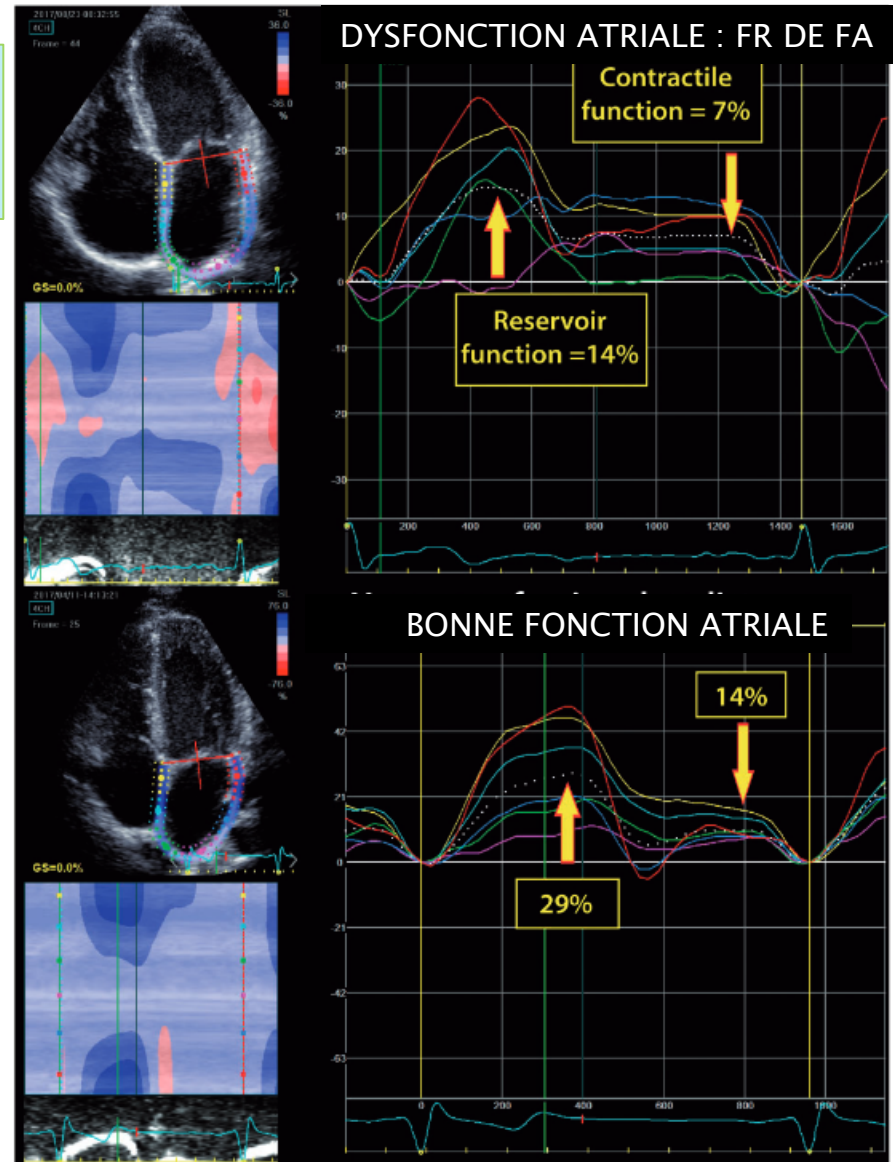
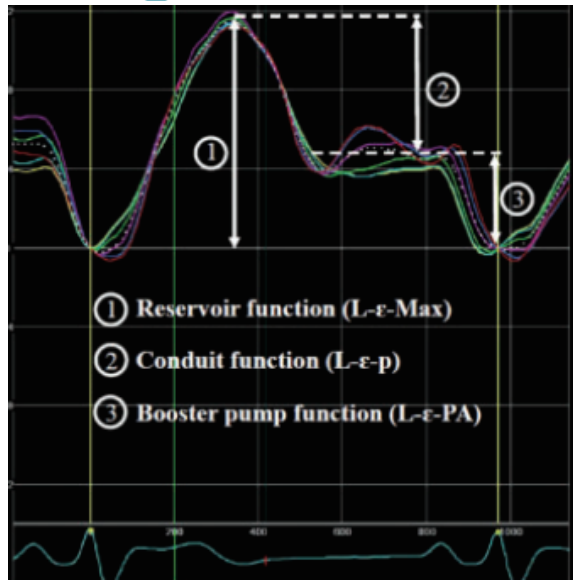
Comment  
expliquer les  
80% restants ???



# Dysfonction atriale

Rechercher un substrat

Remodelage atrial



# Remodelage atrial

Characteristics	PAF group (n = 27)	Control group (n = 30)	P-value
Left and right atrial reservoir function			
LAEF (%)	59.3 ± 12.9	67.6 ± 10.0	0.010
LA L-ε-Max (%)	29.3 ± 7.9	49.1 ± 7.8	<0.0001
RAEF (%)	51.9 ± 16.5	59.6 ± 11.4	0.044
RA L-ε-Max (%)	36.5 ± 7.0	50.6 ± 10.2	<0.0001
Left and right atrial conduit function			
LA passive emptying fraction (%)	36.0 ± 16.2	37.9 ± 10.1	0.591
LA L-ε-p (%)	17.0 ± 6.2	27.0 ± 6.7	<0.0001
RA passive emptying fraction (%)	24.9 ± 13.6	25.7 ± 13.9	0.822
RA L-ε-p (%)	21.6 ± 6.9	28.5 ± 9.3	0.003
Left and right atrial pump function			
LA active emptying fraction (%)	23.4 ± 9.6	29.6 ± 9.0	0.014
LA L-ε-PA (%)	12.3 ± 6.4	22.0 ± 5.6	<0.0001
RA active emptying fraction (%)	26.0 ± 11.3	33.8 ± 10.8	0.010
RA L-ε-PA (%)	14.9 ± 5.9	22.1 ± 5.6	<0.0001

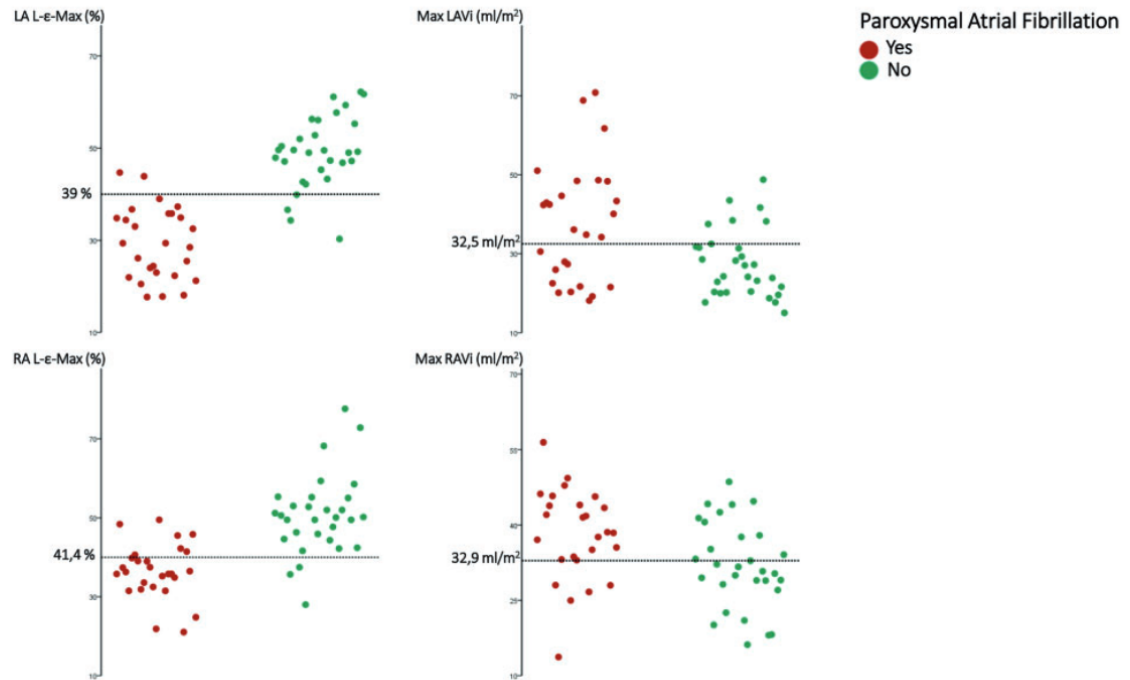
**55% des FA  
du sportif !!!**

# Remodelage atrial

**Vétérans**

**Mauvais  
remodelage atrial**

**cause de la FA du  
vétérans !**

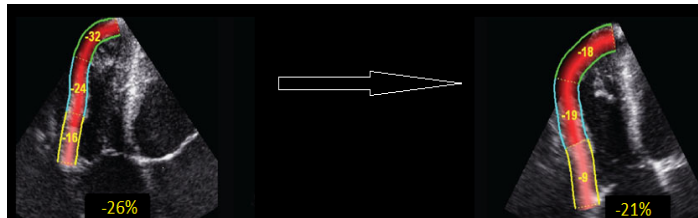
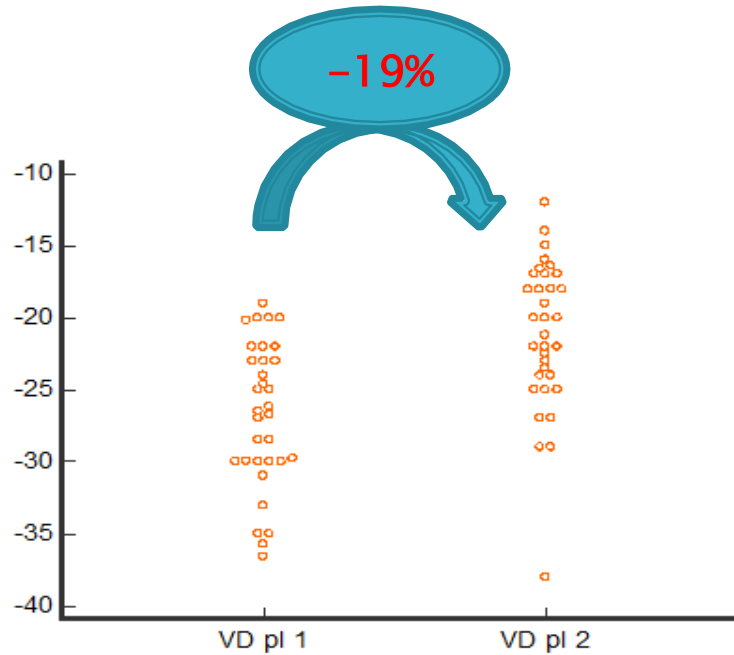


# Physiopathologie

## Conclusions

LA and RA dilation in male veteran endurance athletes can be as well an adaptative or a mal-adaptative remodelling, but decrease of atrial function appears to be always a sign of mal-adaptation which can promote AF.

# Une autre histoire qui finit pareil

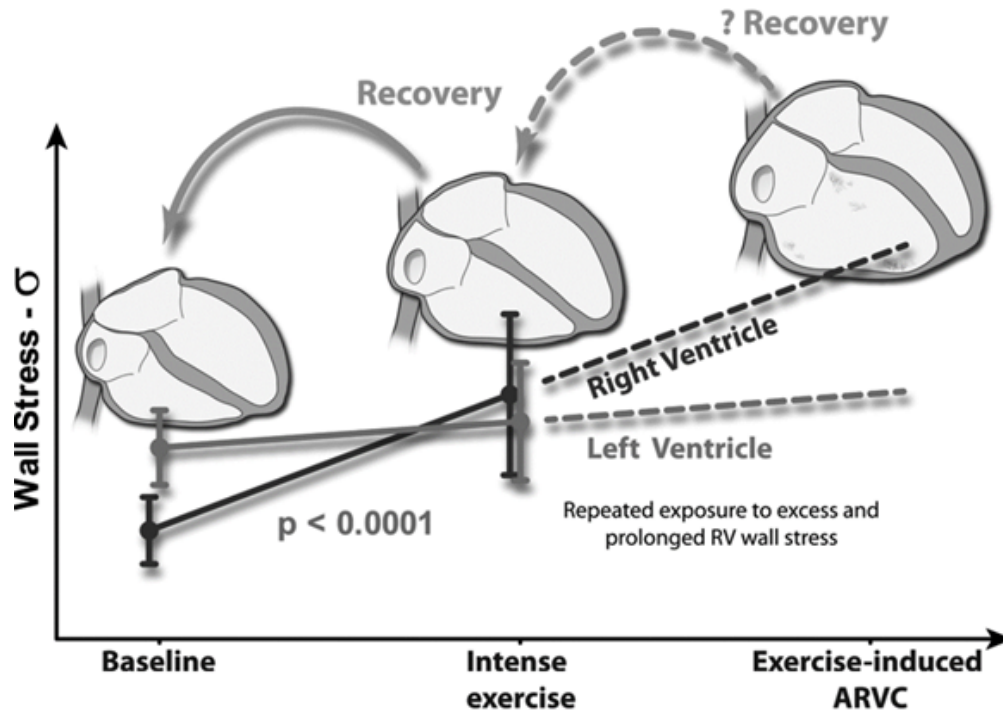


Baisse transitoire de la fonction  $VD > VG$  à l'exercice

Dilatation VD et VG

Persistance quelques heures après effort

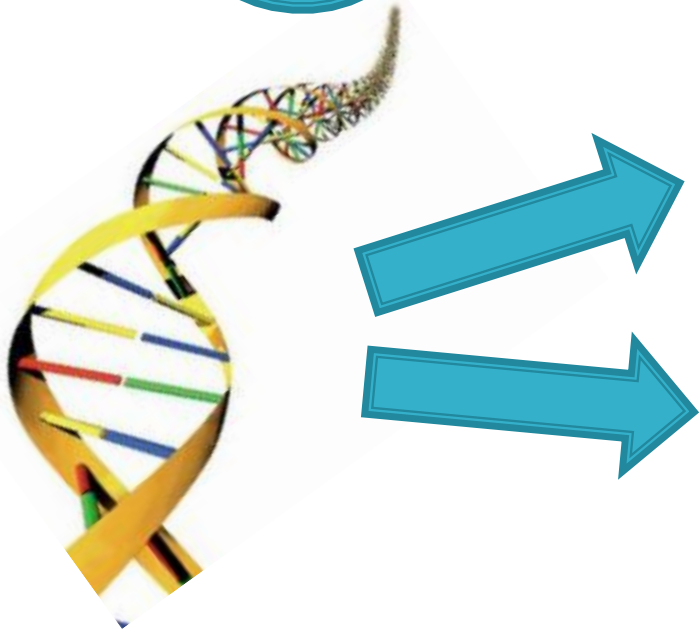
# Le VD du sportif c'est pareil !!!



Dysfonctions VD et arythmies VD sur le long terme (**sujets prédisposés**) « Dysplasie like »

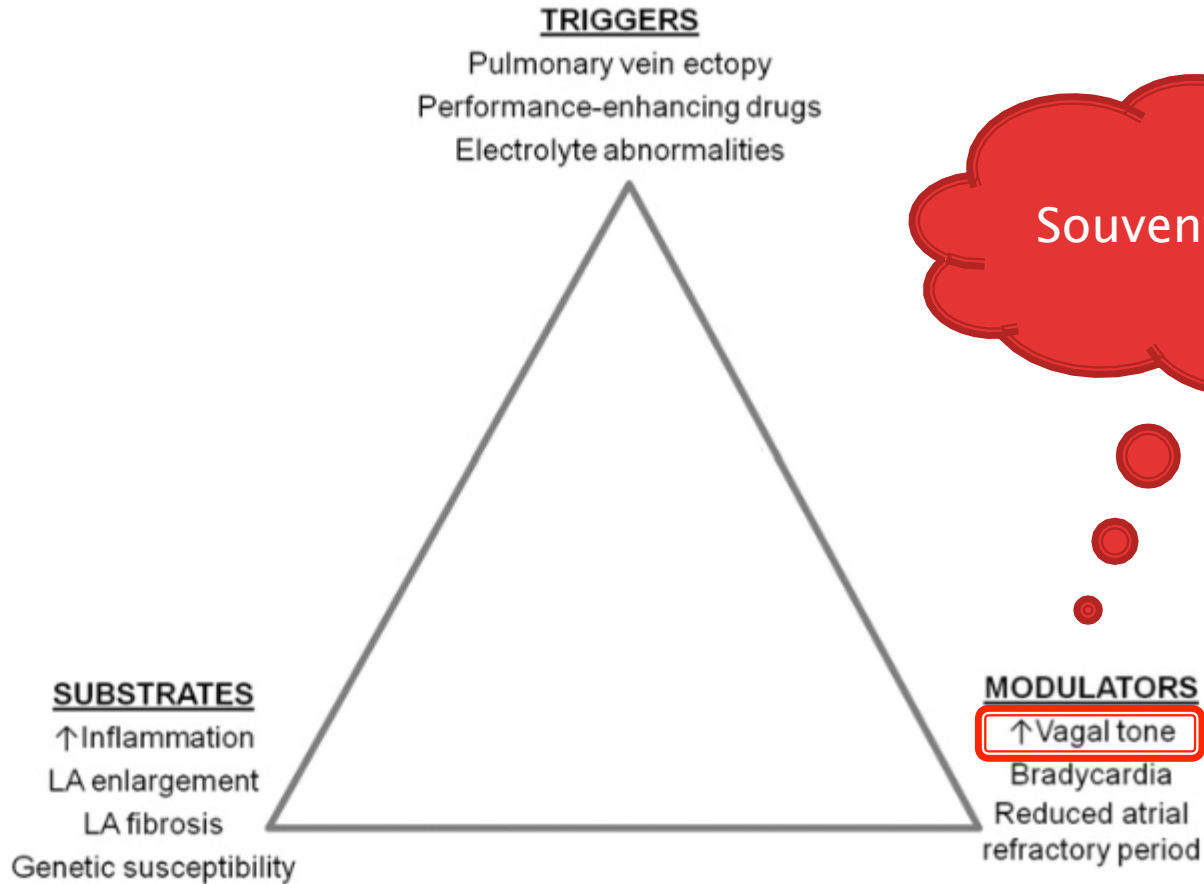
# Capital génétique : déterminant des adaptations

Rechercher  
une  
prédisposition  
génétique ?

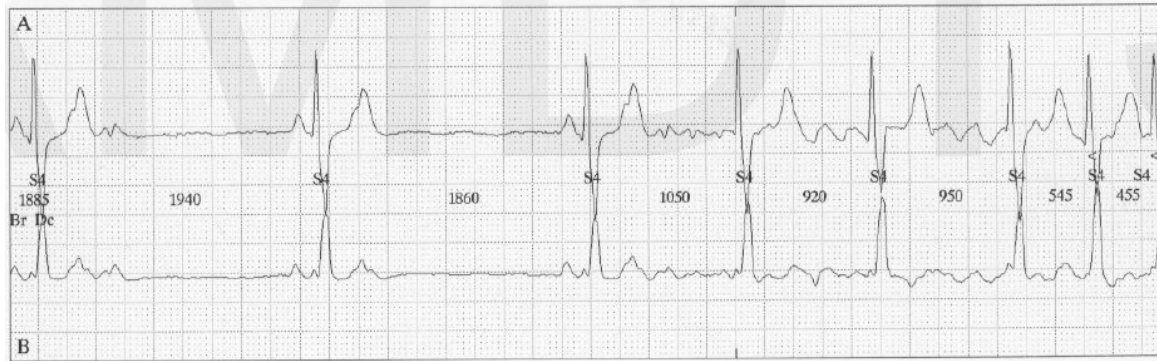
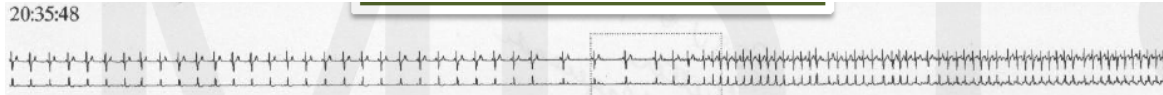




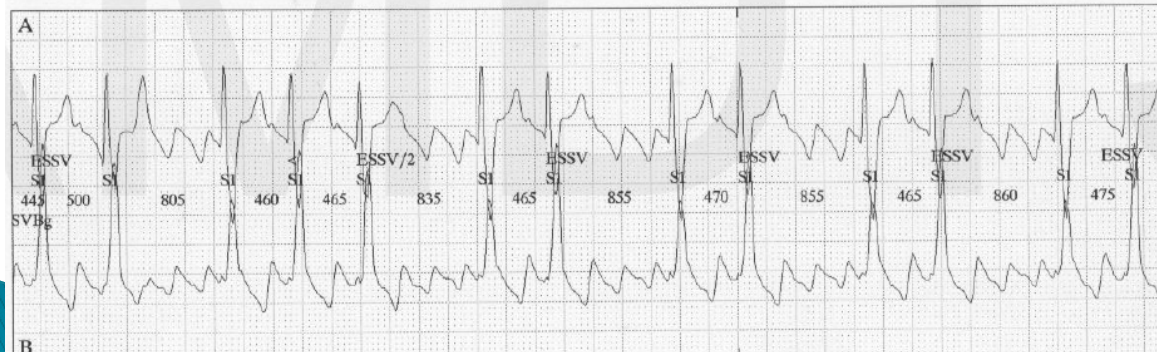
# QUE RECHERCHER ???



# Une histoire qui finit bien

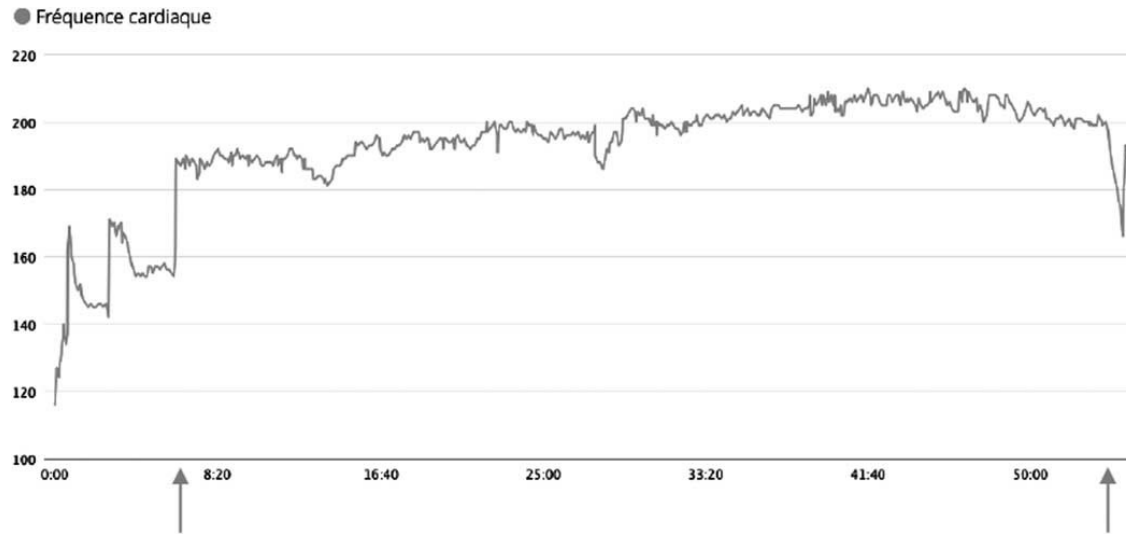


Après les repas  
Nocturne  
En récupération



Fréquence  
ventriculaire  
modérée

# Une autre histoire qui finit moins bien

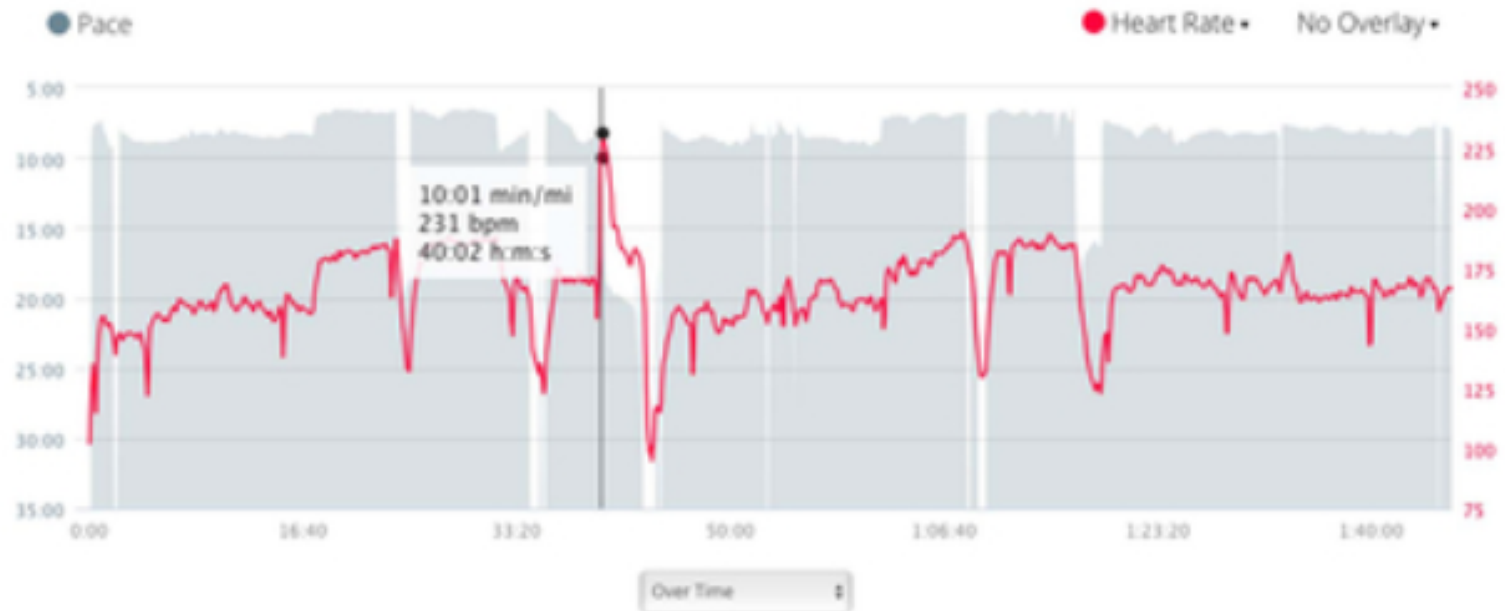


Pendant l'effort

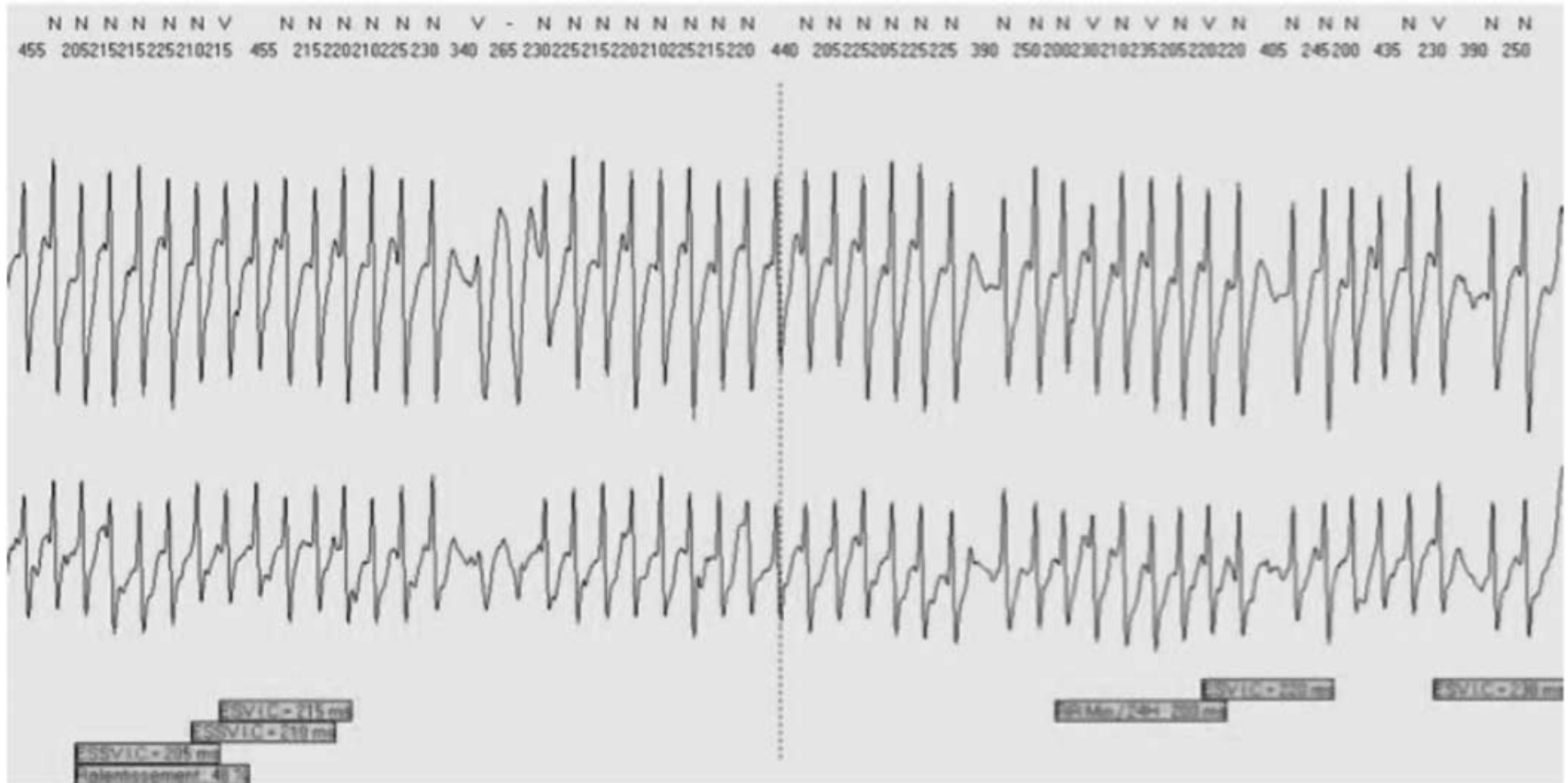
Fréquence  
ventriculaire très  
rapide

Syncope

# Une autre histoire



# Une autre histoire qui finit moins bien

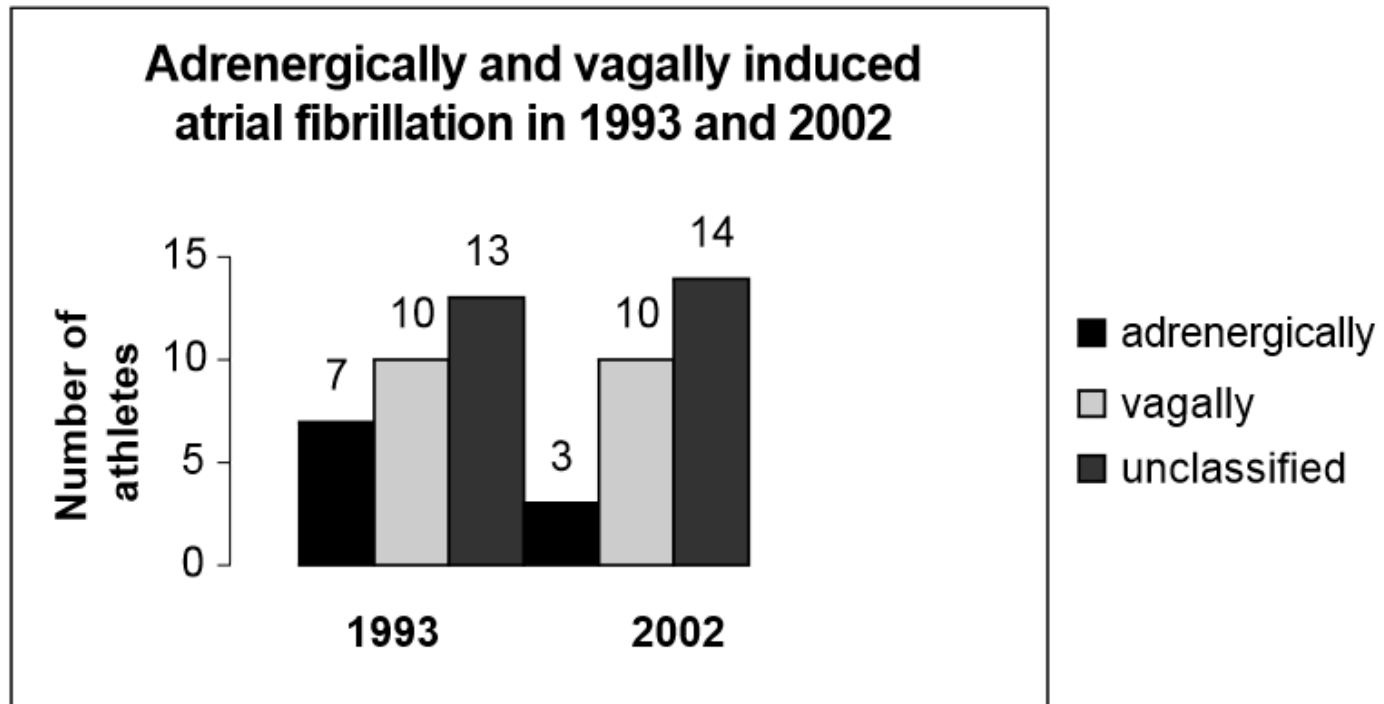


## Syncope During Competitive Events: Interrogating Heart Rate Monitor Watches May Be Useful!

Oscar Thabouillot, MD;<sup>1</sup> Kevin Bostanci, MD;<sup>2</sup> Francois Bouvier, MD;<sup>2</sup> Nicolae Dumitrescu, MD;<sup>2</sup>  
Maria Stéfuriac, MD;<sup>2</sup> Philippe Paule, MD, PhD;<sup>2</sup> Nicolas-Charles Roche, MD<sup>2</sup>

*Prehosp Disaster Med.* 2017;32(6):691-693.

# Modulateur adrénérgique ou vagal



**Figure 3** Distribution of vagally, adrenergically and mixed triggers of atrial fibrillation.

Paroxysmal atrial fibrillation in male endurance athletes. A 9-year follow up

Jan Hoogsteen<sup>a,\*</sup>, Goof Schep<sup>b</sup>, Norbert M. van Hemel<sup>c</sup>, Ernst E. van der Wall<sup>d</sup>

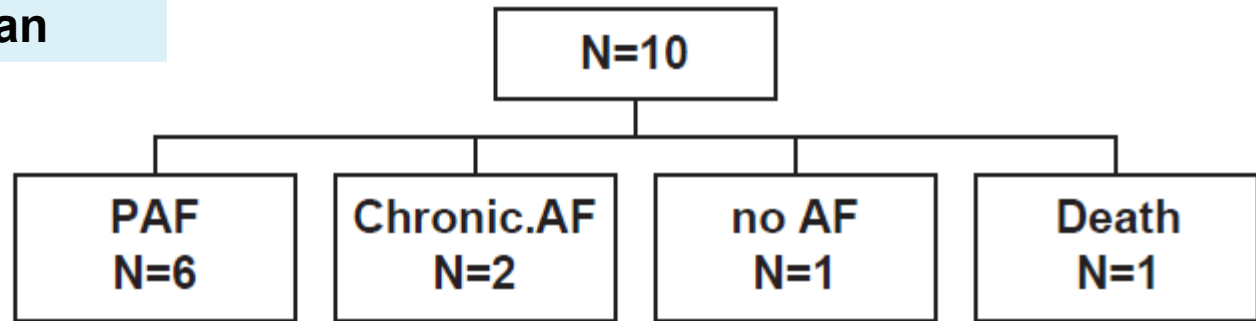
2004 The European Society of Cardiology.

# Modulateur adrénérgique ou vagal

## Vagally induced atrial fibrillation

### Profil vétéran

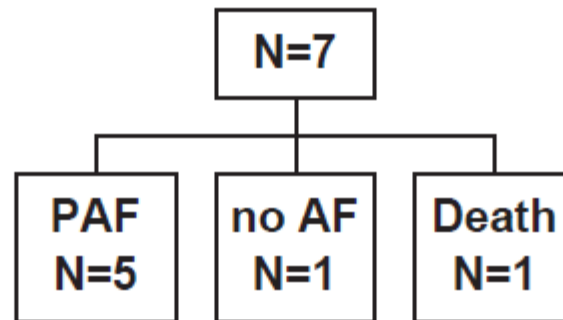
2002



## Adrenergically induced atrial fibrillation

### Profil jeune

2002



Paroxysmal atrial fibrillation in male endurance athletes. A 9-year follow up

Jan Hoogsteen<sup>a,\*</sup>, Goof Schep<sup>b</sup>, Norbert M. van Hemel<sup>c</sup>, Ernst E. van der Wall<sup>d</sup>

2004 The European Society of Cardiology.

# Traitement

- ❖ **Modulation of physical exercise** seems the best approach for significantly limiting the number and the intensity of the crises, particularly in those subjects with recent diagnosis of PAFIYAMA syndrome with atrial dilation;
- ❖ Regular exercise may be safe in patients with PAFIYAMA syndrome, although it depends of individual circumstances, i.e., frequency, duration, precipitating factors, symptoms associated, modes of termination of AF, among others (cardiologist with sports medicine expertise should be consulted);
- ❖ Discussion with a cardiologist may be advisable about the **“pill-in-the-pocket” strategy** while exercising. Importantly, following the ESC Guidelines on AF (12), it should be kept in mind that patients should refrain from exercise while AF episode persist and/or resting for at least **6–8 hours after having taken the drug** (i.e., two half-lives of the antiarrhythmic drug), either flecainide or propafenone;
- ❖ Light to moderate intensity endurance exercise has been shown to be even protective for chronic AF (13). Accordingly, a minimum of 150 min/wk of light to moderate-intensity aerobic exercise is beneficial, and hence, recommended;

❖ These recommendations may be obviously challenging and improbably (if not impossible) to be followed by professional athletes. In such cases, antiarrhythmic drug and/or ablation may be the first line therapy counseled.

**Indications for catheter atrial fibrillation ablation**  
It is reasonable to offer high-level athletes **AF as first-line therapy** due to the negative effects of medications on athletic performance.



# Proposition

## PAFIYAMA

Profil  $\Psi$  POUR limiter le sport

Profil  $\Psi$  CONTRE la limitation sportive

**FLECAINE**  
« pill in pocket »  
Limitation AP

**Bi(Tri)thérapie**  
Betabloquants/Flécaine/  
(IPP)  
Limitation AP

**Ablation par RF**  
Reprise après 3  
semaines  
Anti arythmiques > 6  
semaines



# Les études

**Flécaïne**



**40%**

# Attention

## PAFIYAMA

**Anti arythmiques au long cours**



**\* FLECAINE → Flutter 1/1**  
**\* BÉTABLOQUANTS → Bradycardie, limitation capacité aérobie**  
**\* AMIODARONE !!! (QTc)**

**Plongée, alpinisme**



**Autorisations spécifiques**  
**Médecin fédéral**

**IPP ???**



**Rechercher un RGO**

# Histoire de Mr A...

Marathonien

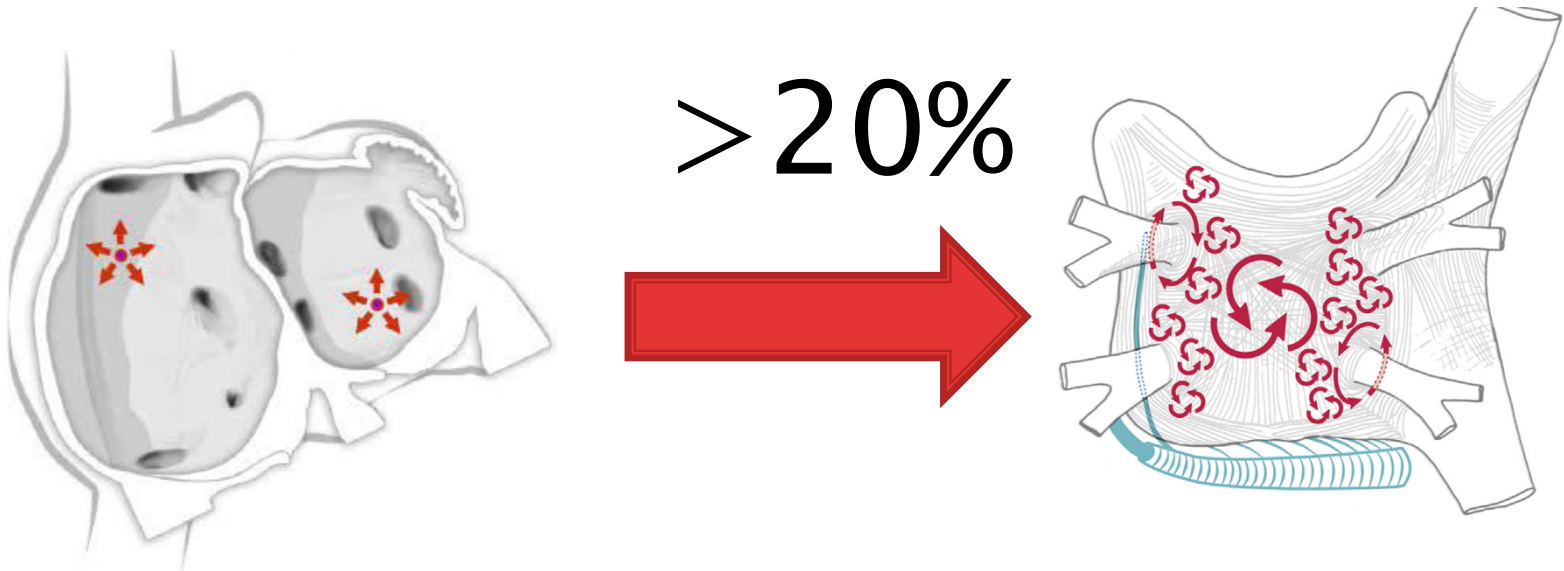
Compétiteur professionnel

FA paroxystique → persistante

Sous Flécaïne + Bétabloquants

Arrêt de la compétition

# Evolution naturelle vers une forme persistante



# 3 procédures

10/2015

FA non arrêtée (rare 5%)

## CONCLUSION

**1/ Succès d'ablation par encerclement large antral des massifs veineux pulmonaire** : déconnexions des 4 veines confirmée au pentaray, réalisée pour fibrillation atriale. Pas de modification du cycle de la FA.

**2/ Traitement du substrat : Défragmentation ciblée mais assez large de l'OG** après mapping haute densité au pentaray : base de l'auricule antérieure, toit, septum, sinus coronaire endocardique, plancher : allongement du cycle de la FA sans **organisation stable**. Cycle Final= 172ms. (+40ms/début de procédure). Cee efficace à 200l.  
**Absence de complication.**

## CONCLUSION

**1/ Contrôle des déconnexions veineuses** : reconnection des veines gauches. → reprise de l'ablation antrale gauche et nouvelle déconnection large.

**2/ CARTOGRAPHIE HD de la FA de l'OG au pentaray : Défragmentation ciblée de l'OG** : tirs sur le bord antérieur de la VPIG permettant l'arrêt de la FA pendant la RF et l'organisation stable en flutter gauche de cycle= 200ms.

**3/ Cartographie de flutter gauche de cycle = 240ms.. Succès d'ablation avec allongement du cycle et retour en rythme sinusal lors de la RF** sur la paroi antéro inférieure de la VPIG.

**4/ Absence d'arythmie inducible** par stimulation atriale décrémenteille (jusqu'à 230ms).

## CONCLUSION

**1/ Contrôle des déconnexions veineuses** : absence de reconnection des 2 massifs veineux pulmonaires

**2/ Cartographie de l'arythmie de cycle=240ms** : micro-réentrée paroi antérieure, jonction avec l'anneau mitra → arrêt de la tachycardie ~~et retour sinusal~~ pendant le tir.

**3/ Déclenchement lors de la stimulation atriale très rapide d'une** **Cartographie de la FA en faveur d'une origine DROITE** : défragmentation ciblée de la base de l'auricule droit paroi latérale et septum haut permettant l'arrêt de la FA et la conversion en un nouveau flutter de cycle= 270ms.

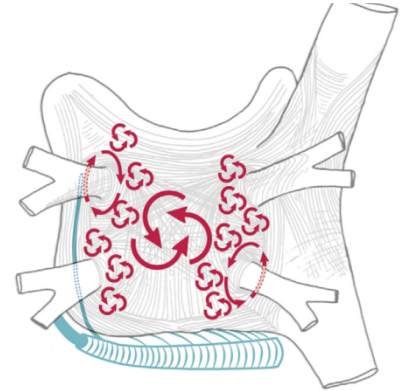
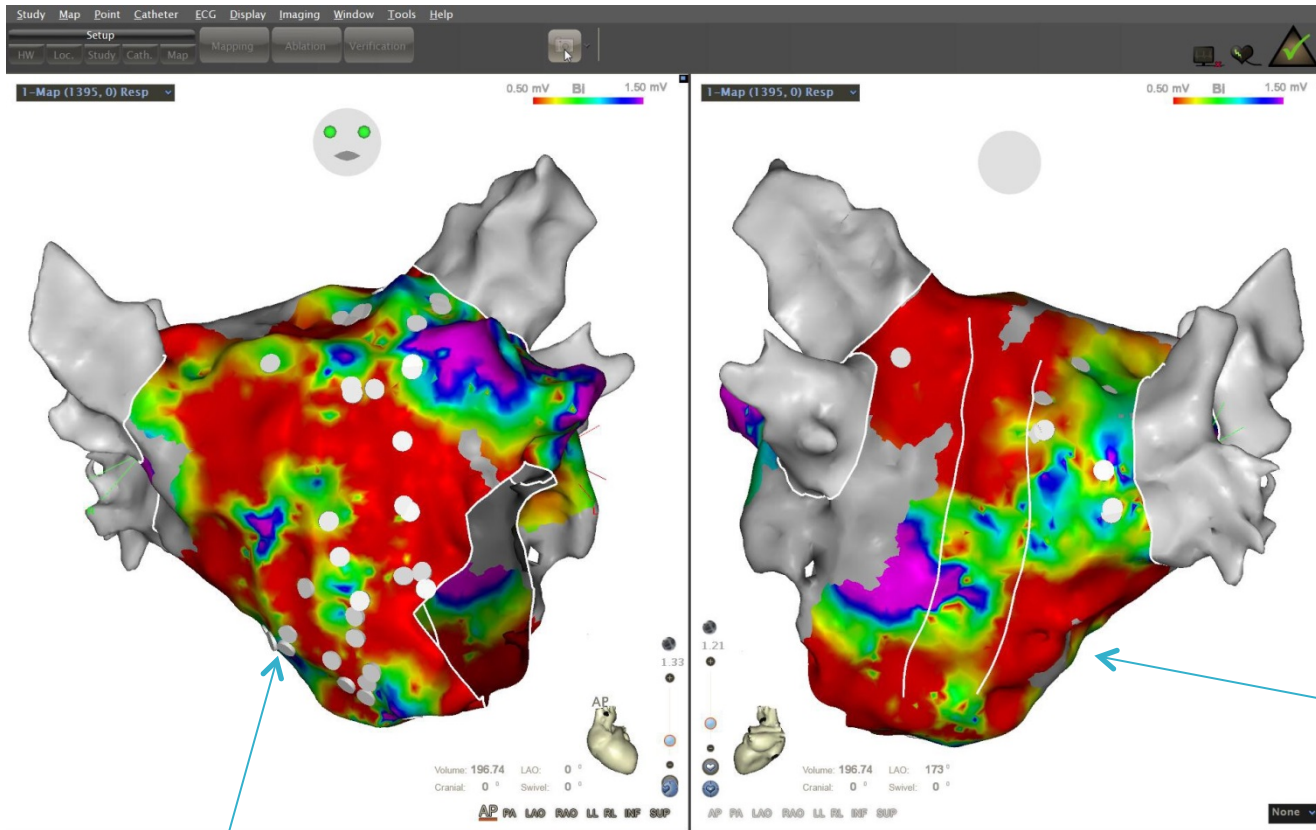
**4/ Remap** : foyer focal à la jonction toit auricule : succès d'ablation avec arrêt de la tachycardie ~~et retour de la RF sur la septum haut~~  
Absence de complication.

03/2016

Arrêt de la FA on se rapproche de la rémission

10/2016

# 2015 : Ablation de la FA



Zones rouges = Fibrose

Dispersion temporospatiale



# Suivi Mr V...

Après 3 procédures (ablation complexe, ciblée)

Arrêt des AA

Reprise compétition

Absence de récurrences



# TAKE HOME MESSAGE

- **Tous les cœurs ne sont pas capables de supporter une activité physique intense...**
- Dépistage de **substances dopantes**
- Rechercher et traiter un **RGO +++**
- Sinon **Mal adaptation de l'oreillette** avec fibrose (Intérêt du Strain OG/OD ???)
- Symptômes en récupération **et** en intensité maximale
- **Limiter l'activité sportive**
- Eviter les anti arythmiques au long cours surtout AA1c et si course intense/longue (IronMan)





**Merci de votre attention**