

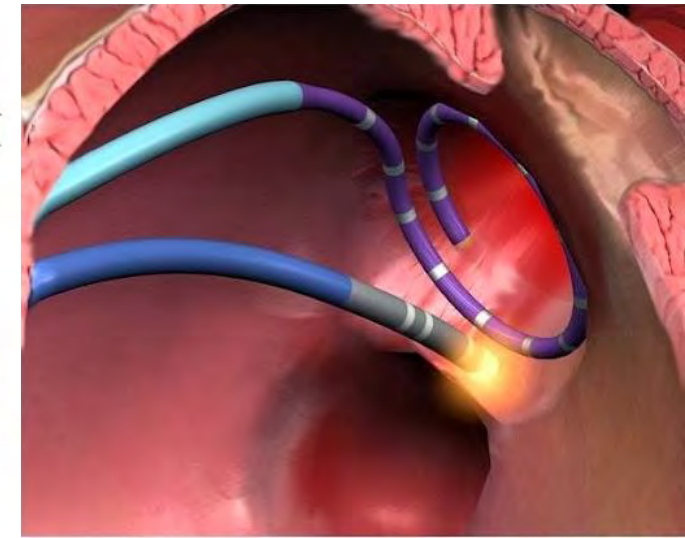
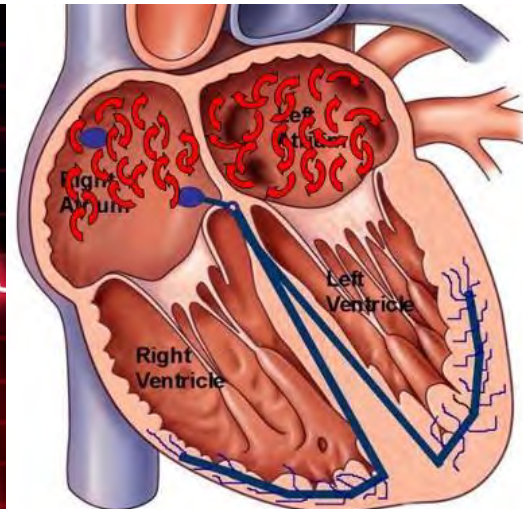
# Fibrillation auriculaire: quelle prise en charge en 2022?

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*10 mars 2022*



# Epidemiology

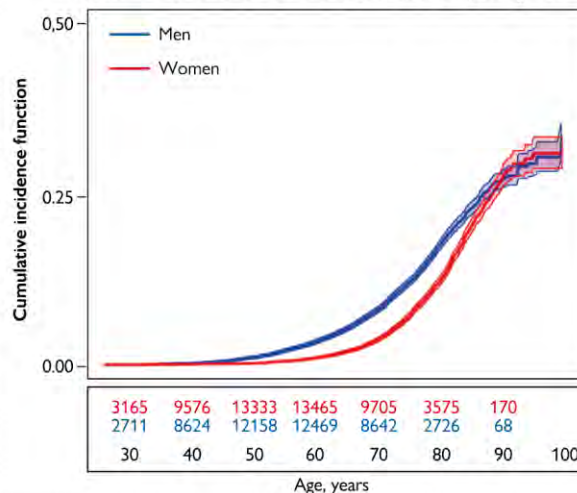
**LIFETIME RISK for AF**  
**1 in 3 individuals**



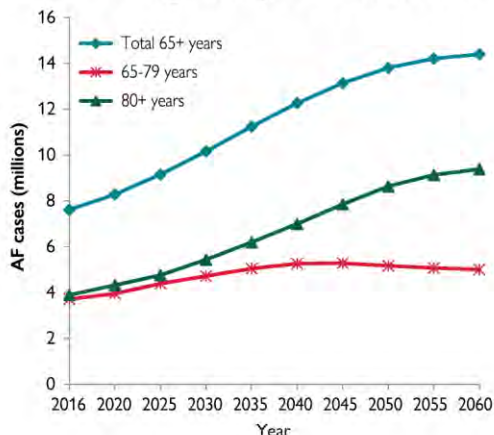
of European ancestry  
at index age of 55 years  
37.0% (34.3% to 39.6%)

**AF is more common in males**

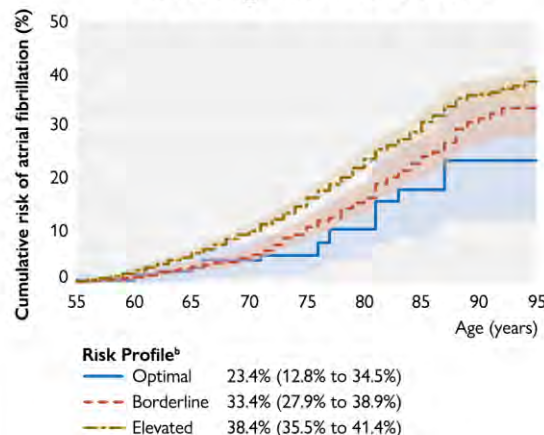
Cumulative incidence curves and 95% CIs  
for AF in women and men with death as a competing risk



**Projected increase in AF prevalence among elderly in EU 2016-2060**



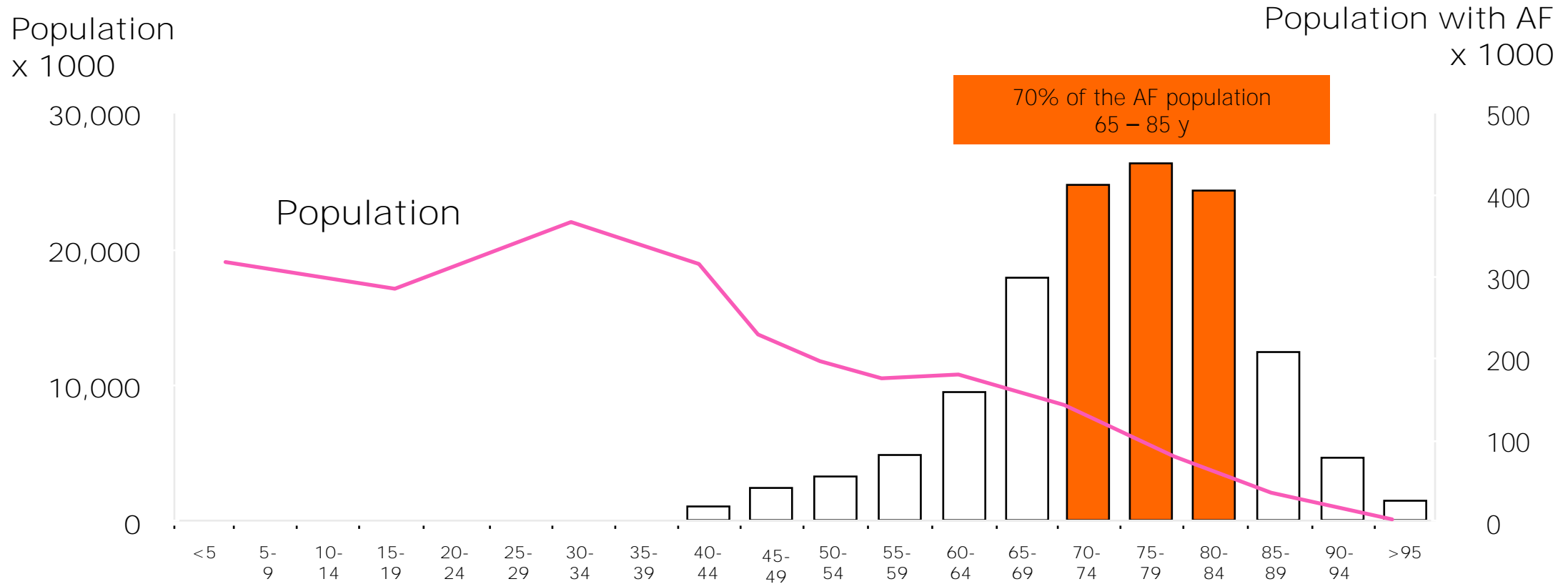
**Lifetime risk of AF increases with increasing risk factor burden<sup>a</sup>**



**Figure 2 (2)**  
**Epidemiology of AF:**  
**lifetime risk and**  
**projected rise in the**  
**incidence and**  
**prevalence**

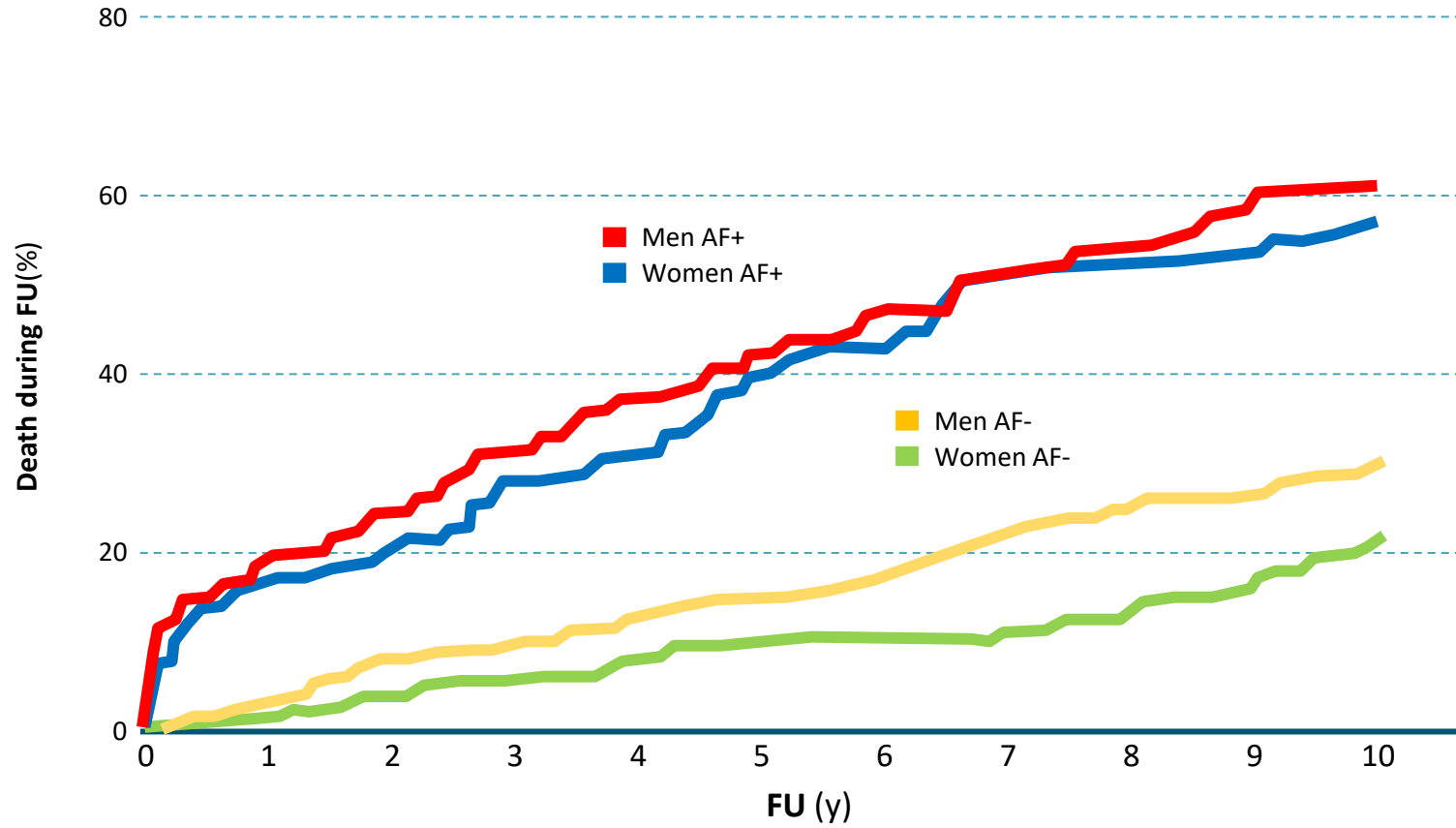
<sup>a</sup>Smoking, alcohol consumption, body mass index, BP, diabetes mellitus (type 1 or 2), and history of myocardial infarction or heart failure. <sup>b</sup>Risk profile: *optimal* – all risk factors are negative or within the normal range; *borderline* – no elevated risk factors but >1 borderline risk factor; *elevated* – >1 elevated risk factor.

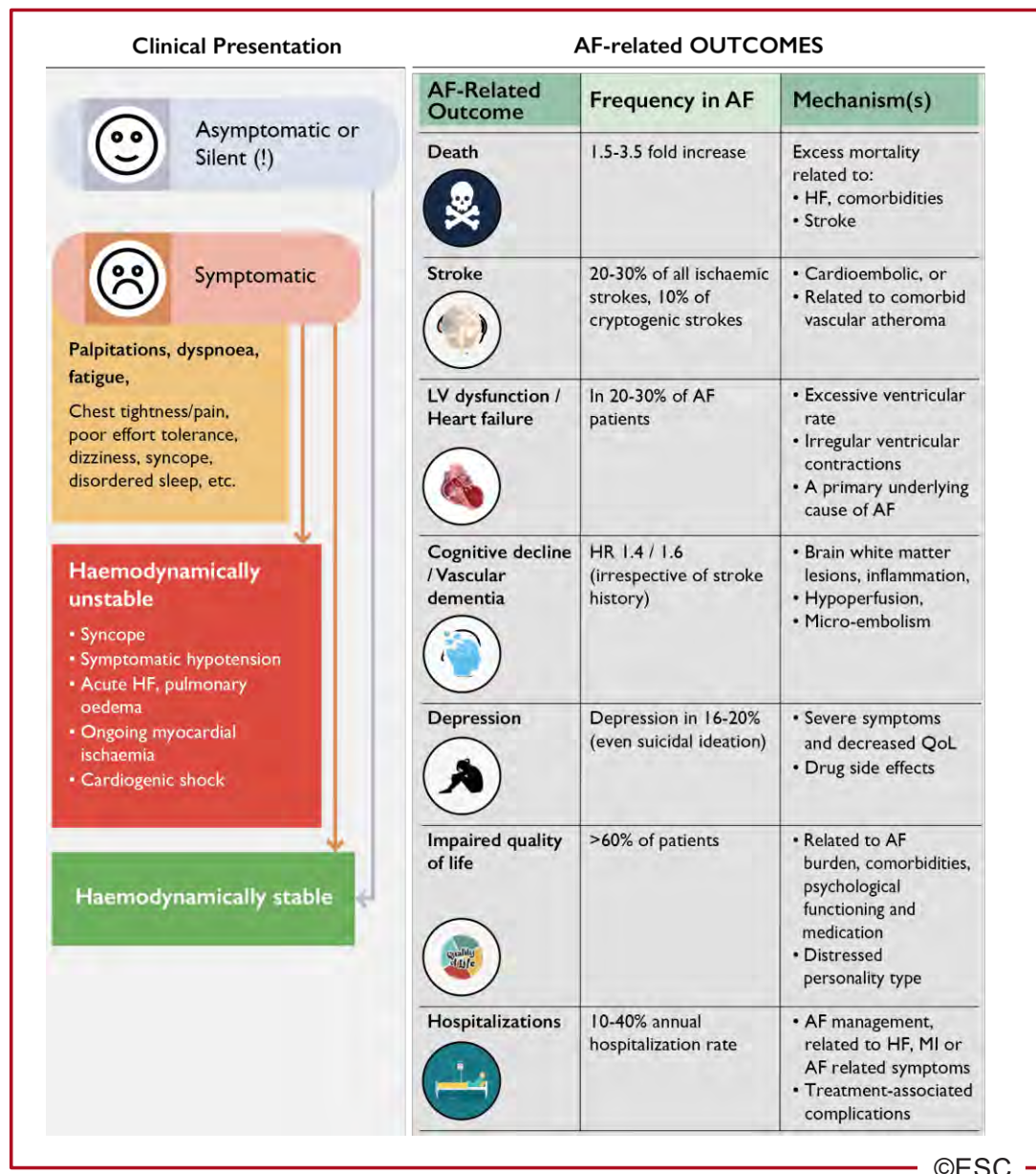
# Prevalence, Age distribution and Gender of patients with AF



- Based on data from 4 large population-based studies (PAF + sustained AF)
- Median age of pts with AF = 75 y old
- AF present in 2.3% of > 40 y, and 5.9% > 65 y old  $\Rightarrow$   $2.3 \times 10^6$  in the US
- 50% of AF population is >75 y and 32% > 80 y old

# AF and Mortality



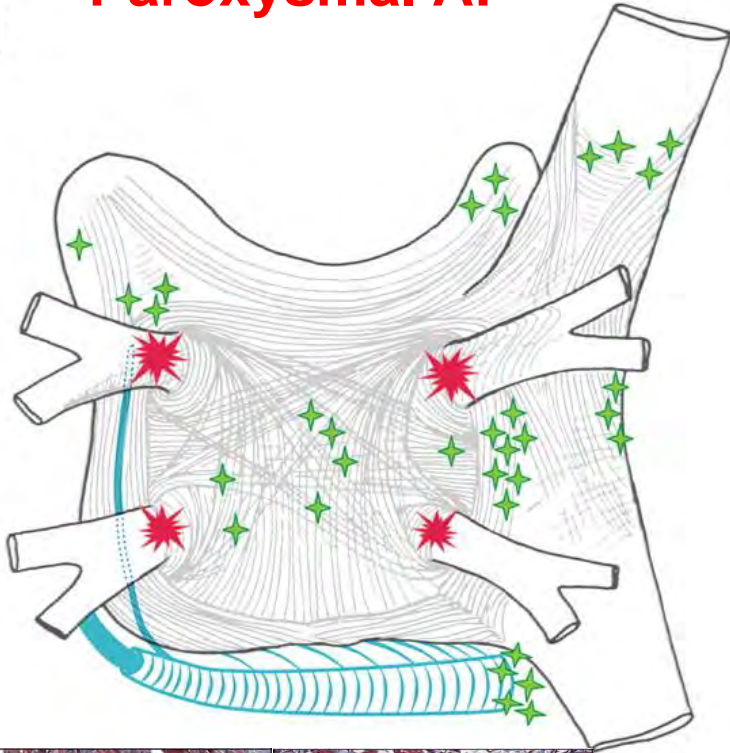


**Figure 4 Clinical presentation of AF and AF-related outcomes**

# Pathophysiology and mechanism of AF

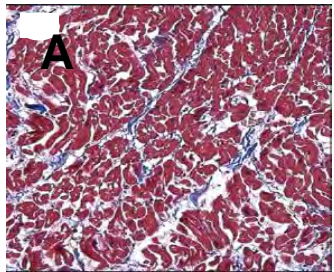
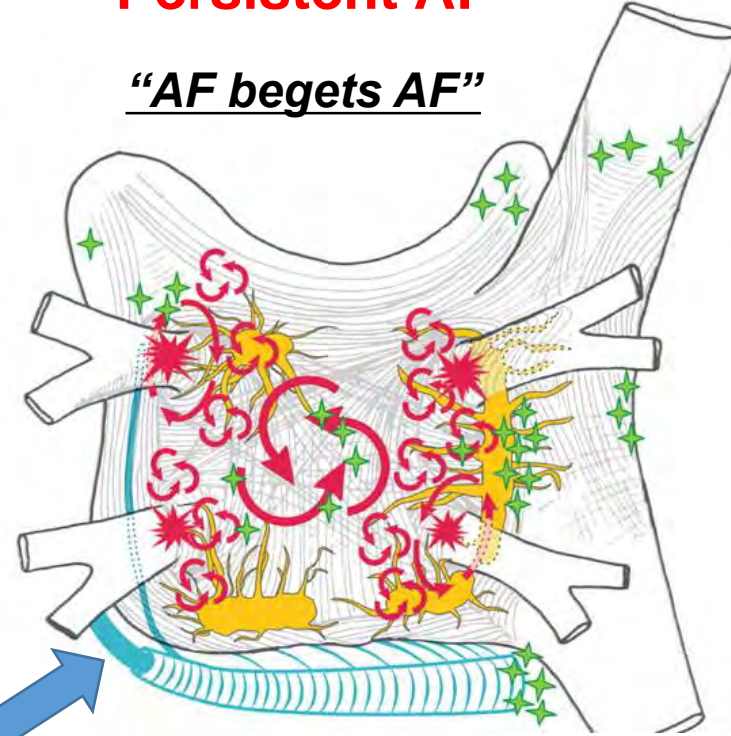
# Different Mechanism of Paroxysmal AF vs Persistent AF

## Paroxysmal AF

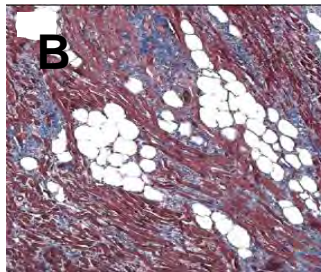


## Persistent AF

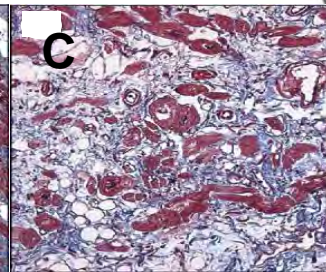
“AF begets AF”



**A**  
w/o AF  
5%



**B**  
Paroxysmal AF  
14%

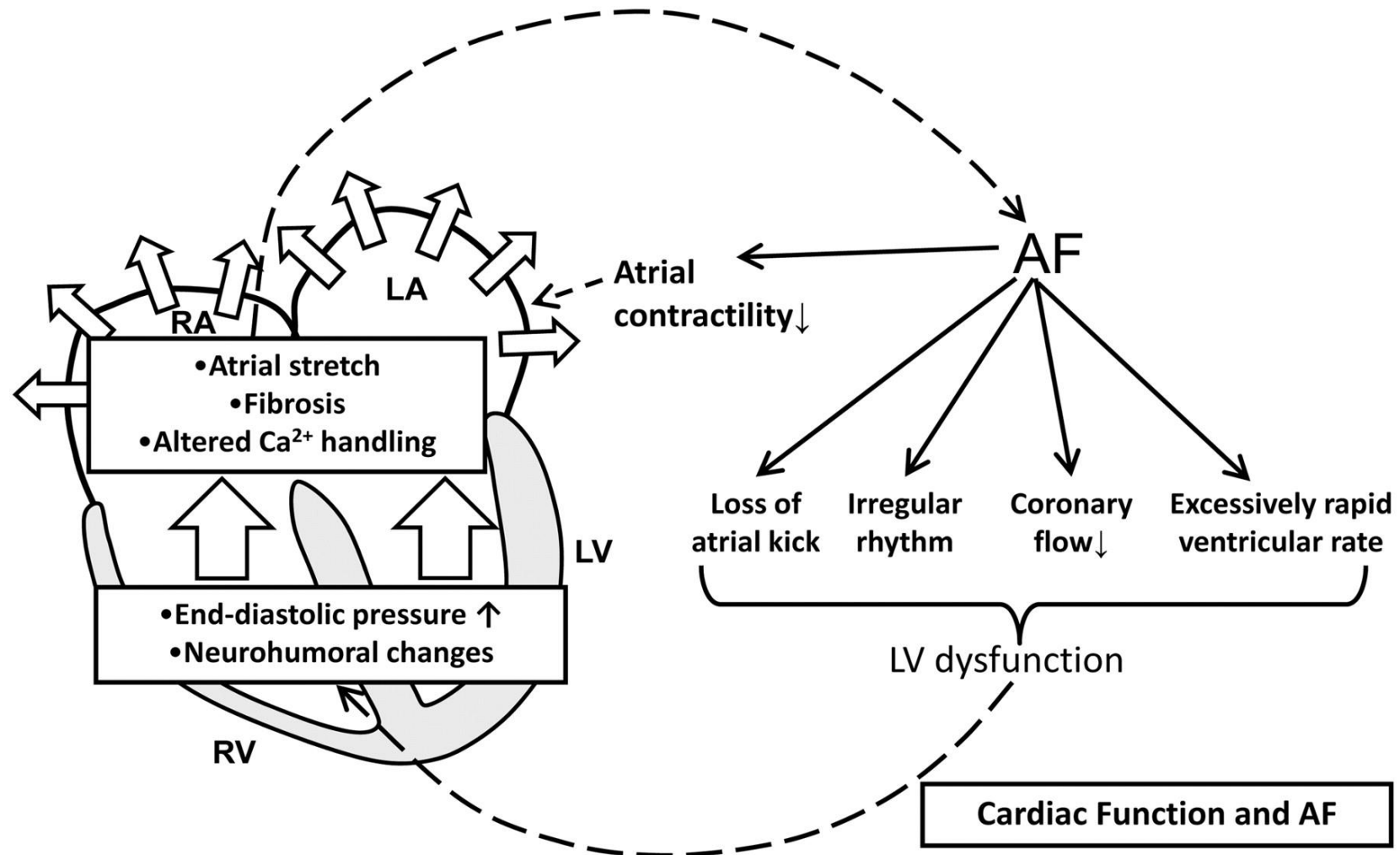


**C**  
Permanent AF  
51%

- Limited success of PV isolation (40-50%?)
- AF persists due to tissue and electrical remodeling:
  - AF itself (« AF begets AF »)
  - Secondary factors (HTN, valvular heart disease...)



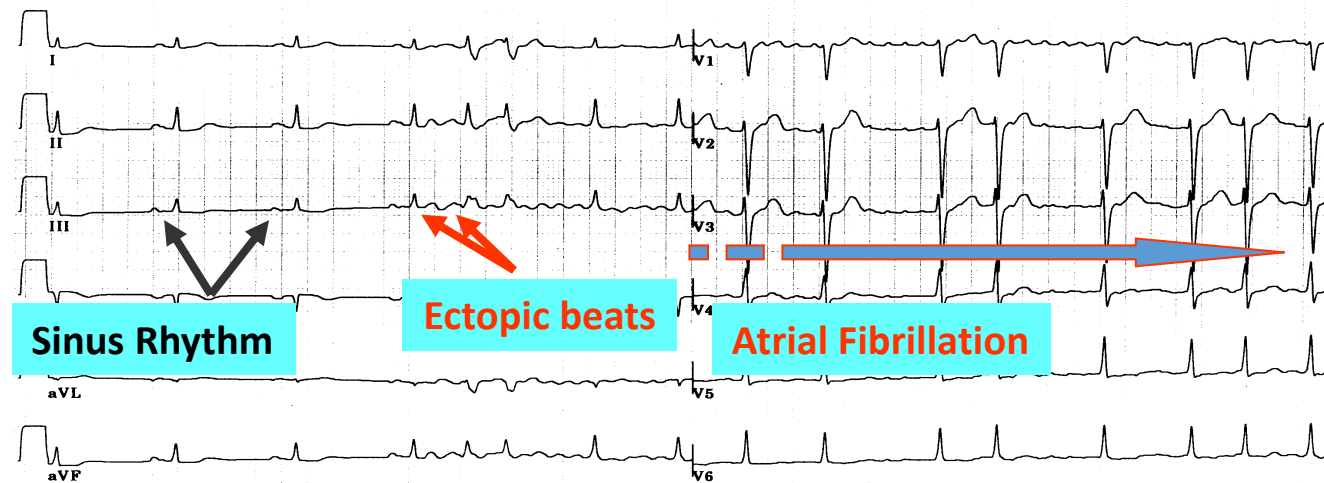
# “AF begets AF”



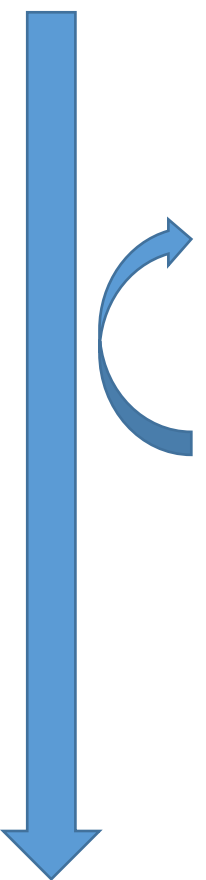
# Definition

# Definition

- **Supraventricular tachyarrhythmia with uncoordinated atrial electrical activation and consequently ineffective atrial contraction**
- **Irregularly irregular R-R intervals** (when atrioventricular conduction is not impaired)
- **Absence** of distinct repeating **P waves**
- Irregular atrial activations
- Minimum duration of an ECG tracing of AF required to establish the diagnosis of clinical AF is **at least 30 seconds**

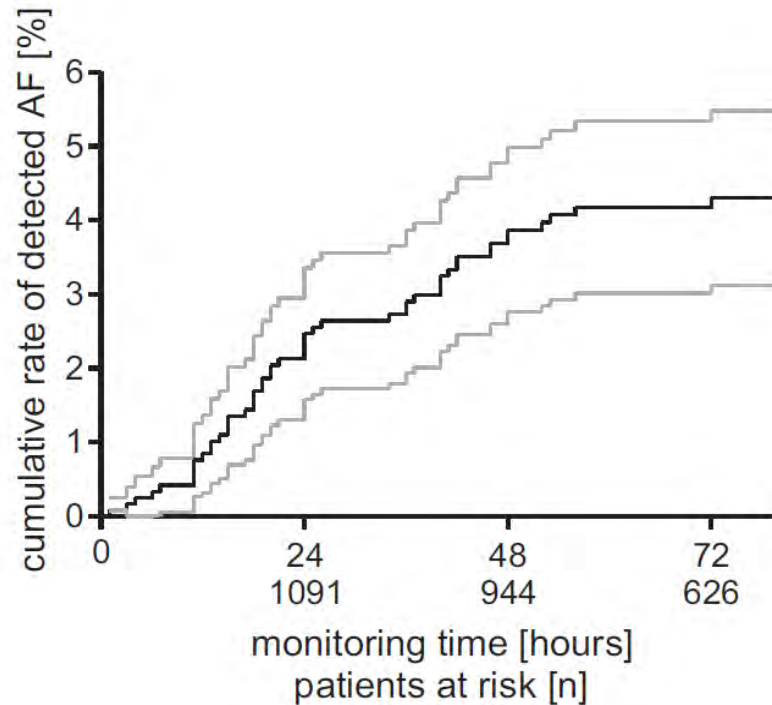


# Patterns of atrial fibrillation

- 
- **First diagnosed AF:**
    - AF that has **not been diagnosed before**, irrespective of the duration of the arrhythmia or the presence and severity of AF-related symptoms.
  - **Paroxysmal AF:**
    - AF that **terminates spontaneously or with intervention within 7 days of onset**
  - **Persistent AF:**
    - AF that lasts **longer than 7 days**, including episodes that are **terminated by cardioversion**, either with drugs or by direct current cardioversion, **after 7 days** or more.
  - **Long-standing persistent AF:**
    - Continuous AF lasting for **≥1 year** when it is decided to adopt a **rhythm control** strategy.
  - **Permanent AF:**
    - AF that is **accepted** by the patient (and physician). Hence, **rhythm control** interventions are, by definition, **not pursued** in patients with permanent AF. Permanent AF **represents a therapeutic attitude** of the patient and physician rather than an inherent pathophysiological attribute of AF. The term should not be used in the context of a rhythm control strategy with antiarrhythmic drug therapy or AF ablation

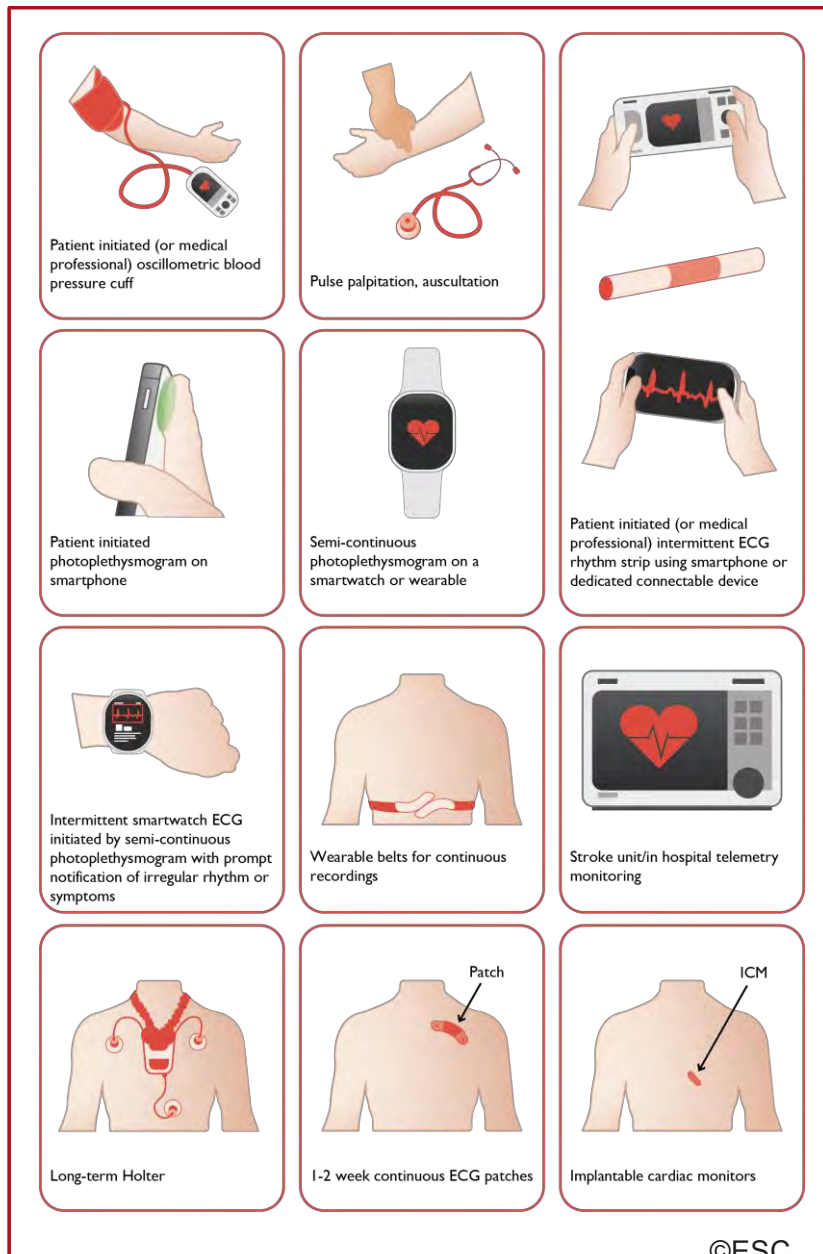
Screening

# Screening



AF detected in **4.3%** by 72h Holter monitor  
AF detected in **2.6%** by 24h Holter monitor

The **number needed to screen by 72-hour ECG** was **55 patients** for each additional AF diagnosis



**Figure 6** Systems used for AF screening

Pulse palpation, automated BP monitors, single-lead ECG devices, PPG devices, other sensors (using seismocardiography, accelerometers, and gyroscopes, etc.) used in applications for smartphones, wrist bands, and watches. Intermittent smartwatch detection through PPG or ECG recordings. Smartwatches and other ‘wearables’ can passively measure pulse rate from the wrist using an optical sensor for PPG and alerting the consumer of a pulse irregularity (based on a specific algorithm for AF detection analysing pulse irregularity and variability

# Recommendations for screening to detect AF (1)

Recommendations	Class	Level
Opportunistic screening for AF by pulse taking or ECG rhythm strip is recommended in patients $\geq 65$ years of age.	I	B
It is recommended to interrogate pacemakers and implantable cardioverter defibrillators on a regular basis for AHRE. <sup>a</sup>	I	B

<sup>a</sup>See *sections* for diagnostic criteria for AF and AHRE, and for the management of patients with AHRE.

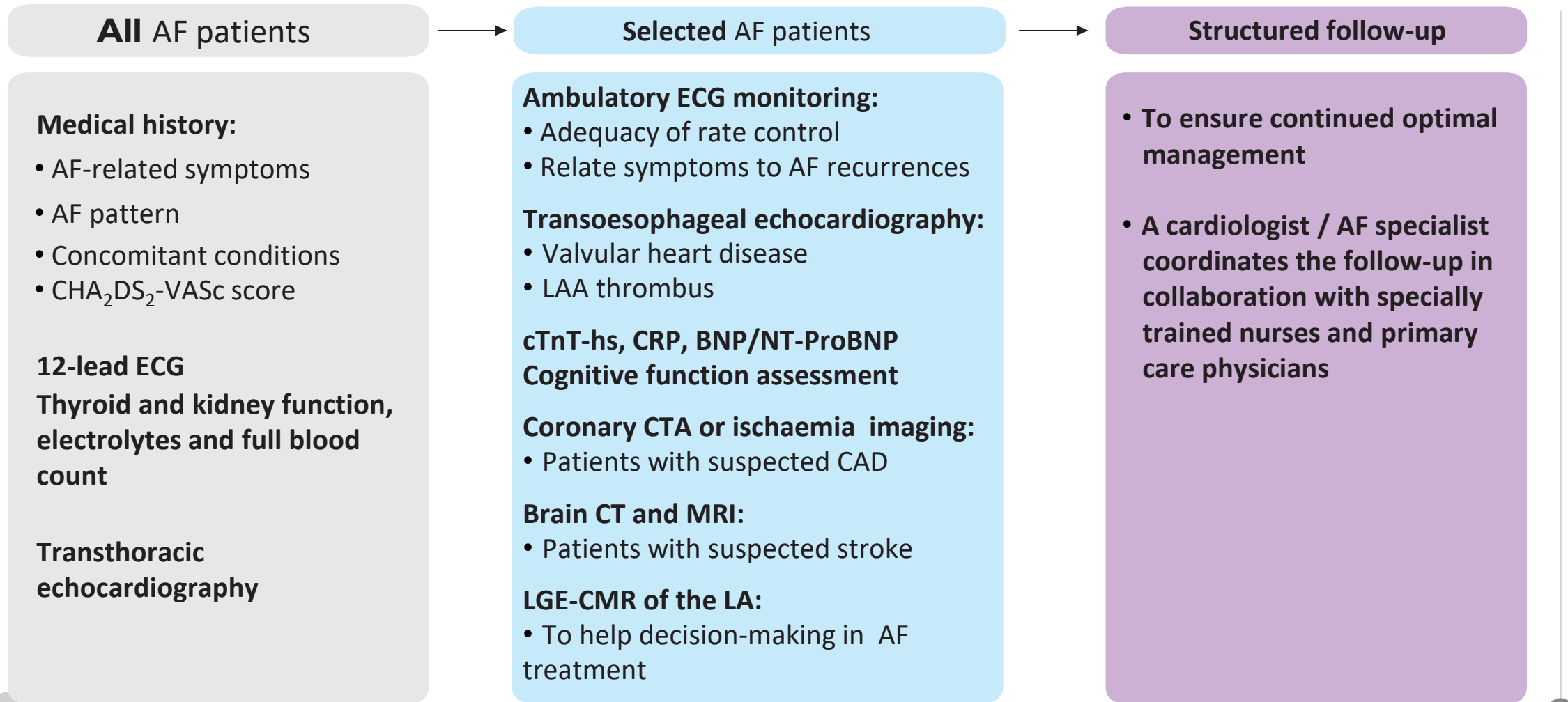


# Symptom burden of Atrial Fibrillation

- Poorer quality of life
- Lethargy
- Palpitations
- Dyspnoea
- Chest tightness
- Sleeping difficulties
- Psychosocial distress
- Cognitive impairment
- None (silent AF)

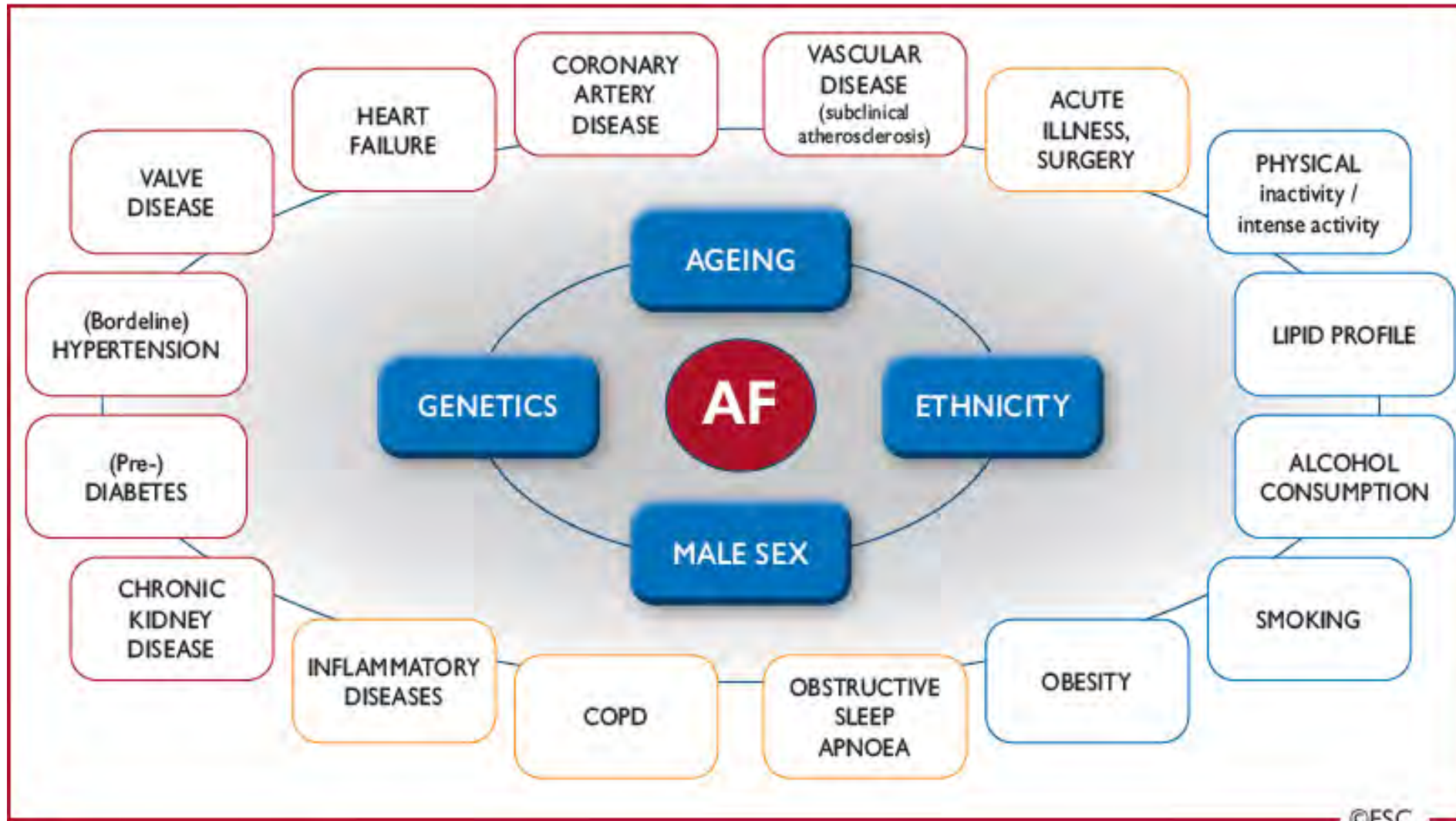
Modified EHRA score	Symptoms	Description
I	None	AF does not cause any symptoms
2a	Mild	Normal daily activity not affected by symptoms related to AF
2b	Moderate	Normal daily activity not affected by symptoms related to AF, but <b>patient troubled by symptoms</b>
3	Severe	Normal daily activity affected by symptoms related to AF
4	Disabling	Normal daily activity discontinued

# Figure 8 Diagnostic work-up and follow-up in AF patients



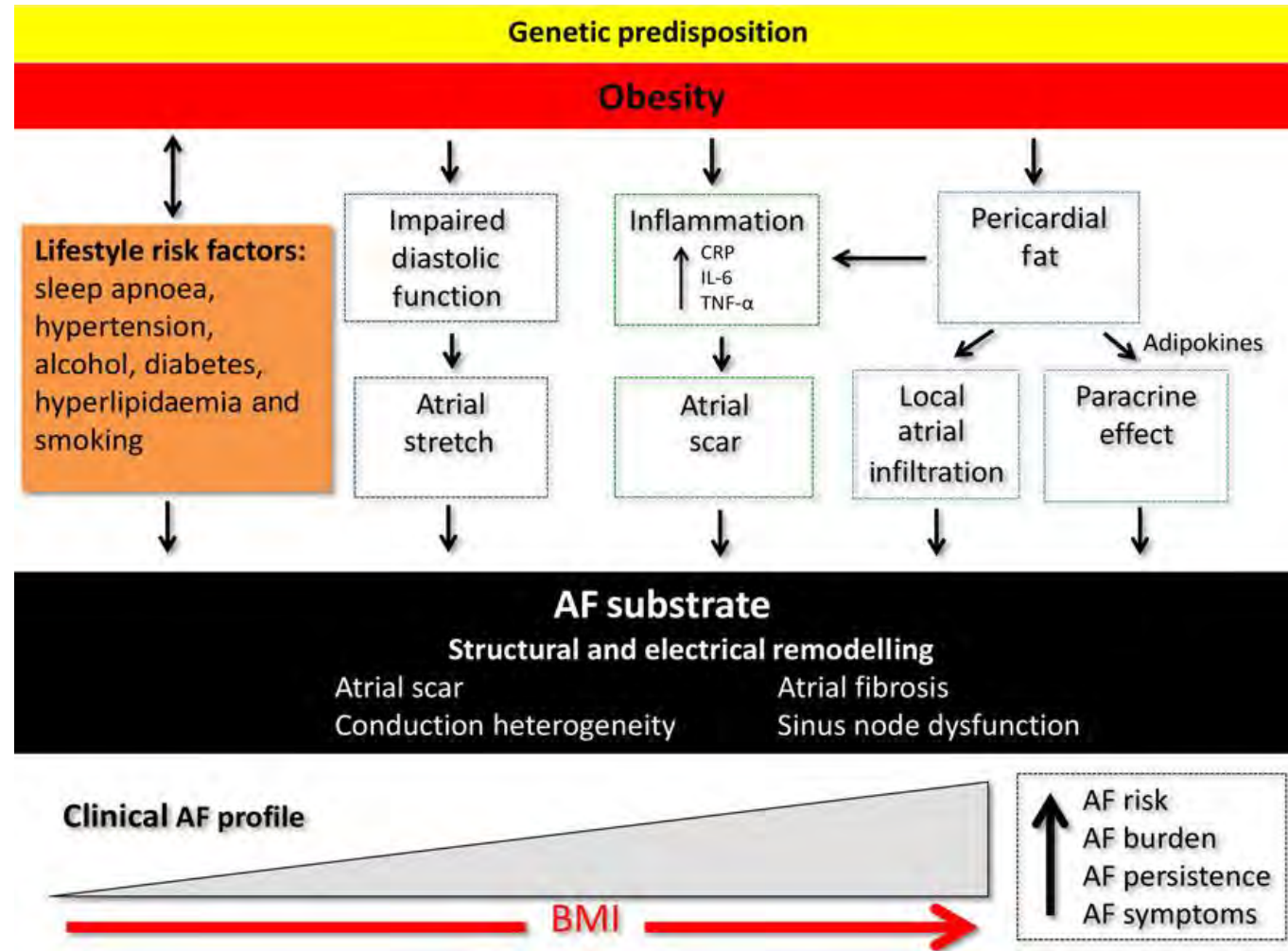
Risk Factors for developing AF

**Figure 3** Summary of risk factors for incident AF



# The Role of Obesity in AF

- Overweight: BMI 25-30kg/m<sup>2</sup>
- Obese: BMI >30kg/m<sup>2</sup>
- **Overweight populations have higher incidence, prevalence, severity and progression of AF** compared with their normal weight counterparts
- **Stable weight loss decreases AF burden and AF recurrence following treatment. Structural remodelling** in response to weight loss suggests that reverse remodelling of the AF substrate mediates improvement of arrhythmia profile
- **Obesity often co-exists with multiple AF risk factors that improve in response to weight loss, making a consolidated approach of weight loss and AF risk factor management preferable**



## The role of obesity in atrial fibrillation

Chrisan Joseph Nalliah<sup>1,2,3</sup>, Prashanthan Sanders<sup>3</sup>, Hans Kottkamp<sup>4</sup>, and Jonathan M. Kalman<sup>1,2\*</sup>

<sup>1</sup>Department of Cardiology, Royal Melbourne Hospital, Melbourne 3050, Australia; <sup>2</sup>Department of Medicine, University of Melbourne, Melbourne, Australia; <sup>3</sup>Centre for Heart Rhythm Disorders (CHRD), South Australian Health and Medical Research Institute (SAHMRI), University of Adelaide and Royal Adelaide Hospital, Adelaide, Australia; and <sup>4</sup>Department of Electrophysiology, Hirslanden Hospital, Zurich, Switzerland

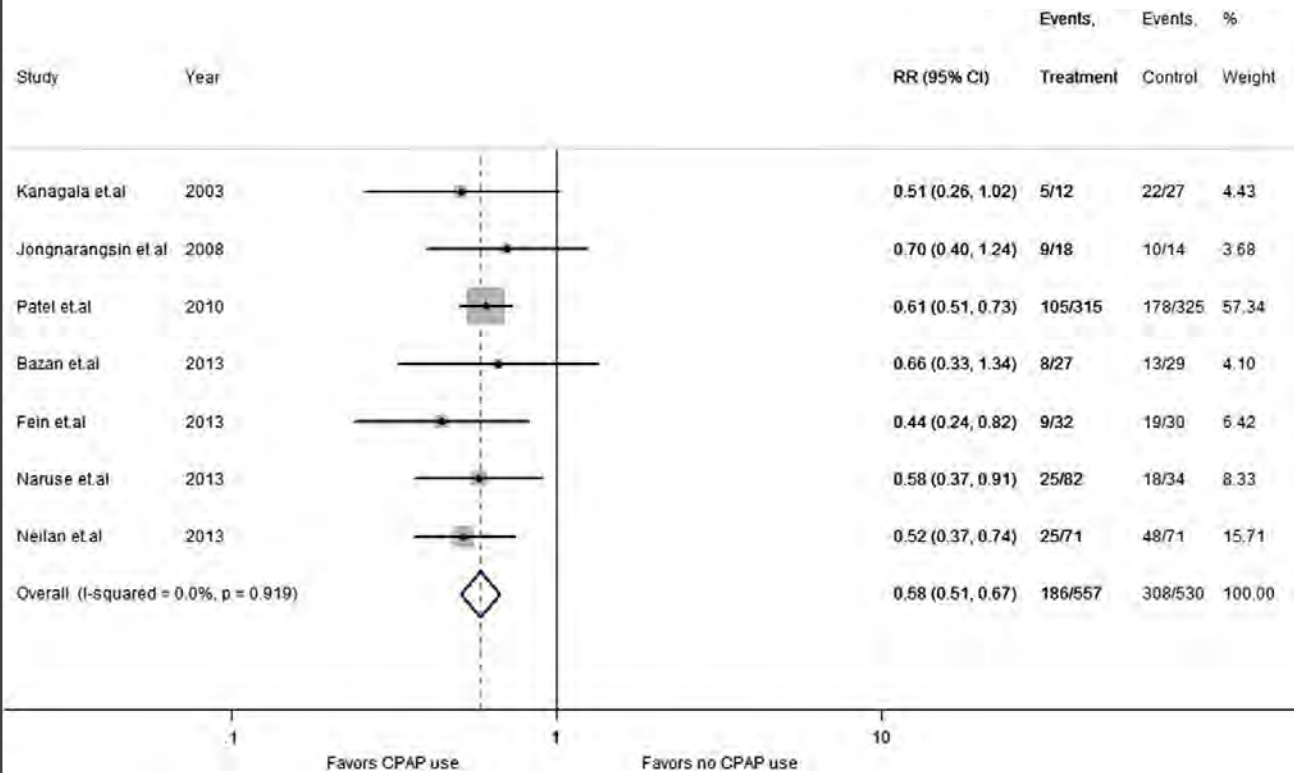
Received 19 June 2015; revised 10 August 2015; accepted 25 August 2015; online publish-ahead-of-print 14 September 2015



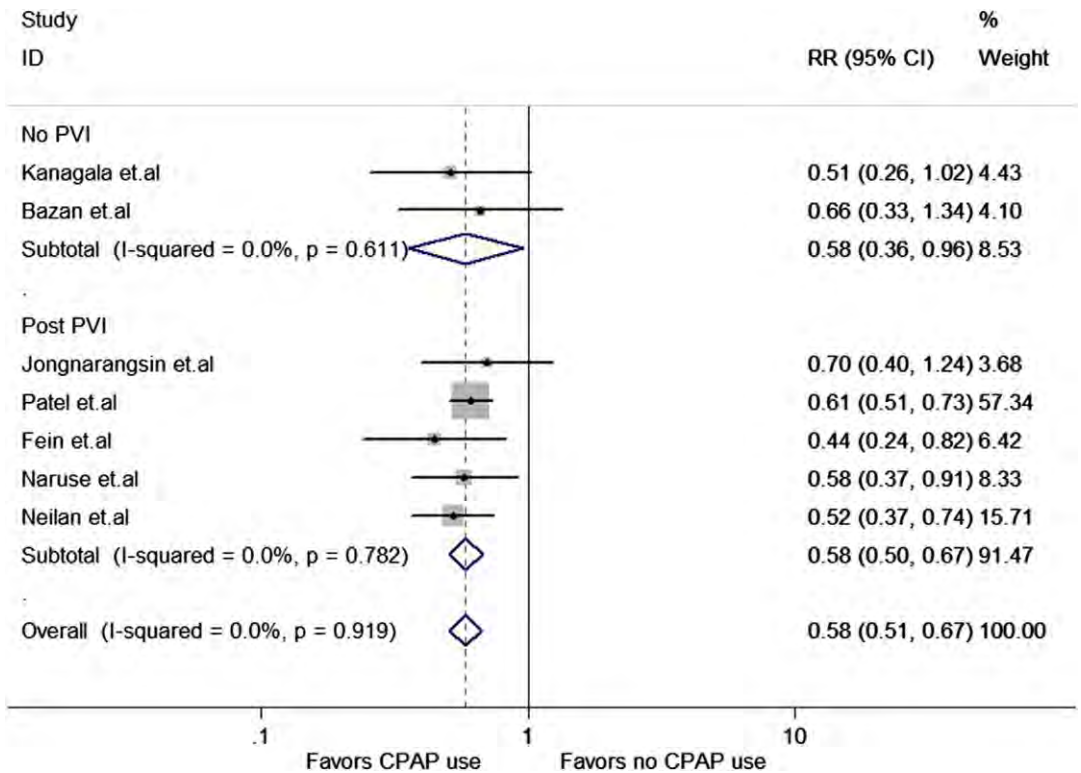
European Heart Journal (2016) 37, 1565–1572  
doi:10.1093/eurheartj/ehv486

# Effect of Obstructive Sleep Apnea Treatment on Atrial Fibrillation Recurrence

## AF Recurrence in Users versus Nonusers of CPAP in patients with OSA



## AF Recurrence in Users versus Nonusers of CPAP in 2 groups of Patients with OSA: PVI and non-PVI groups



## Effect of Obstructive Sleep Apnea Treatment on Atrial Fibrillation Recurrence

A Meta-Analysis

Ashish Shukla, MD, MPH, Anthony Aizer, MD, MSc, Douglas Holmes, MD, Steven Fowler, MD, David S. Park, MD, PhD, Scott Bernstein, MD, Neil Bernstein, MD, Larry Chinitz, MD

# Alcohol and AF

## Potential Mechanisms for Acute Alcohol Consumption as a Trigger for AF

### CELLULAR EFFECTS

- Damage to gap junction intercellular channels
- Direct myocyte injury and/or inflammation
  - Acute oxidative stress

### AUTONOMIC EFFECTS

- Sympathetic activation (increased  $\beta$  receptor density)
  - Vagal inhibition
- Reduced heart rate variability

### ELECTROPHYSIOLOGICAL EFFECTS

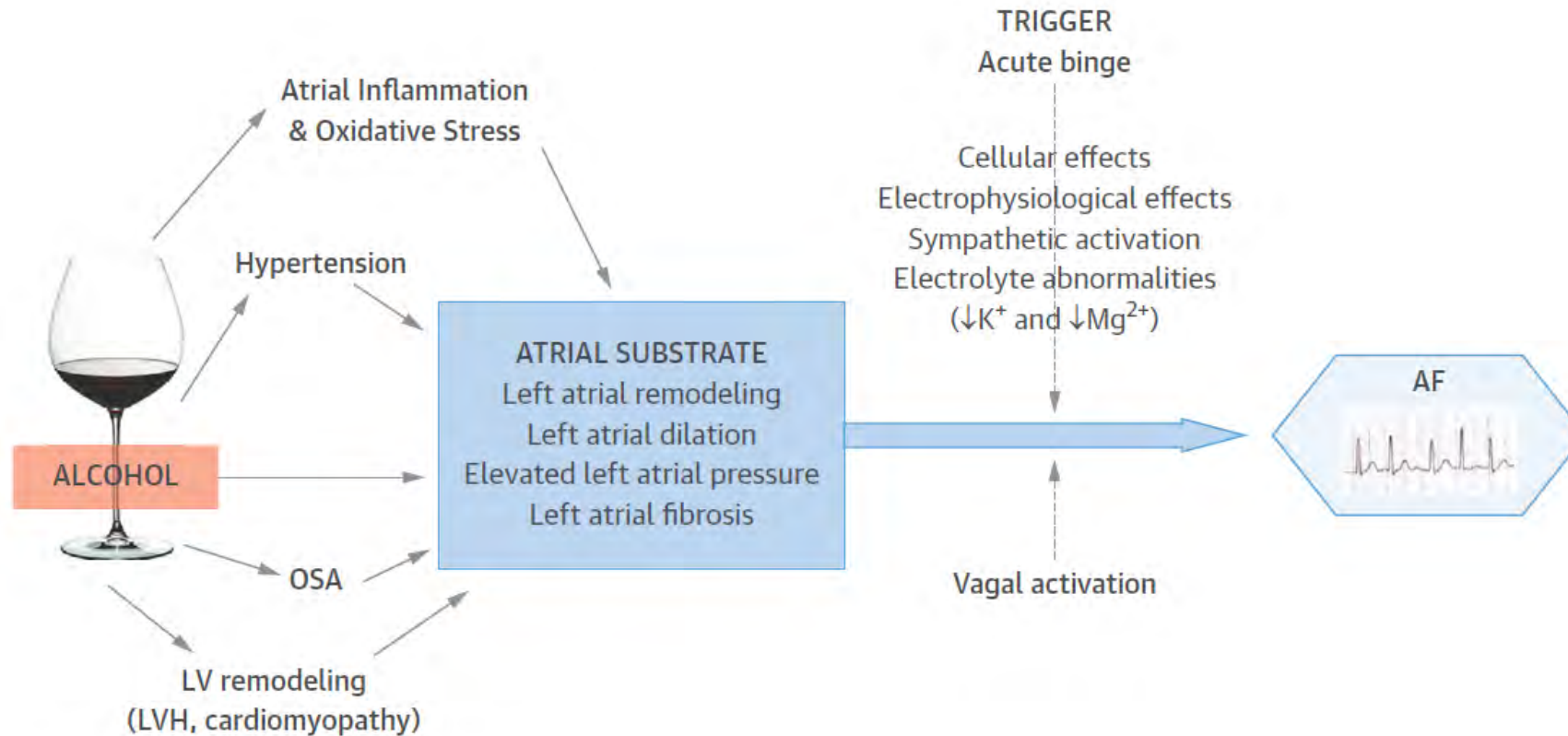
- Shorter atrial and pulmonary vein action potential
    - Shorter atrial effective refractory period
  - Slowing of intra- and inter-atrial conduction
    - Enhanced AV-nodal conduction
- RE-ENTRY

## Alcohol and Atrial Fibrillation

### A Sobering Review

# Alcohol and AF

## Habitual Alcohol Consumption and AF: Pathophysiology



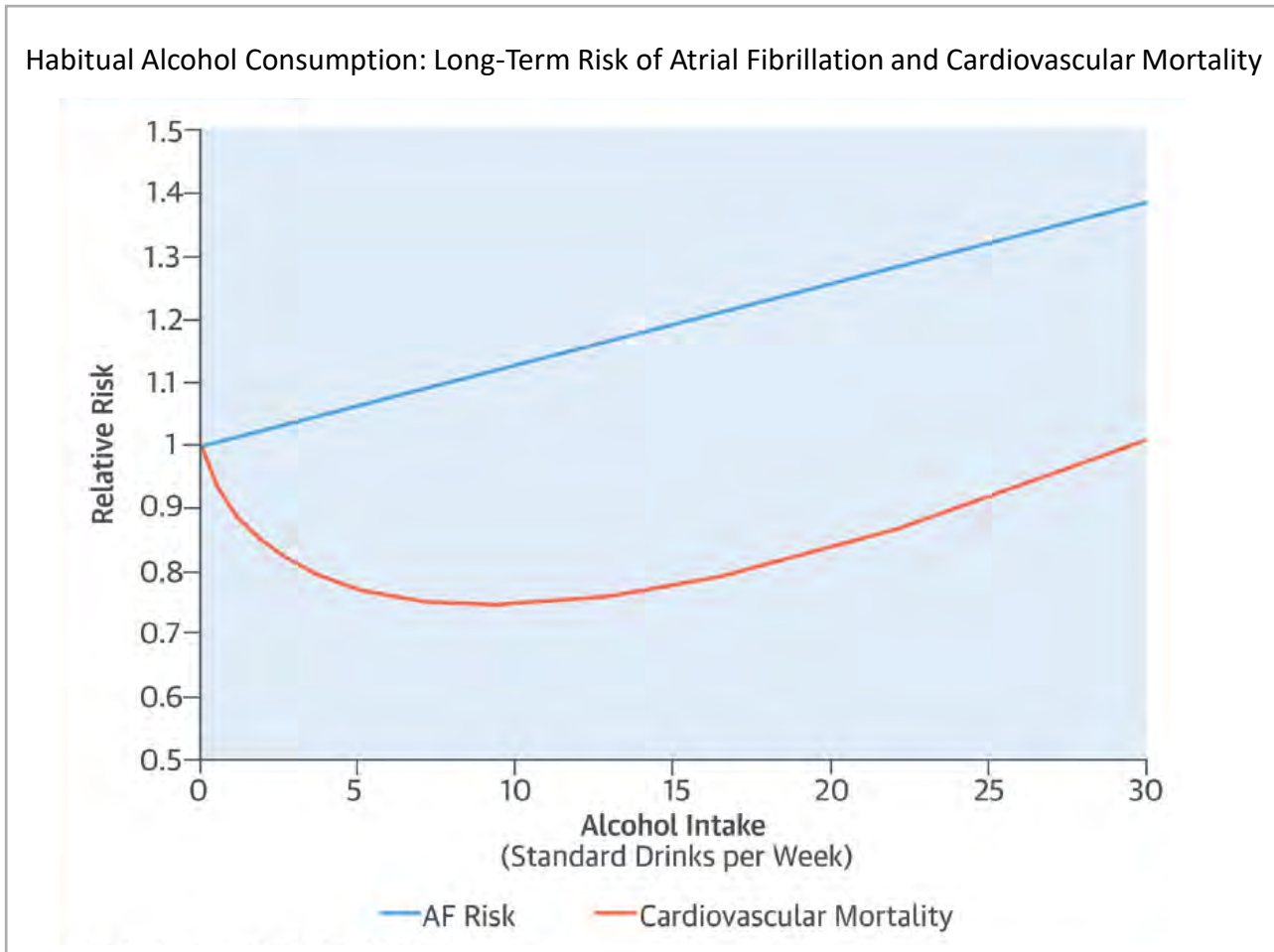
## Alcohol and Atrial Fibrillation

### A Sobering Review

Aleksandr Voskoboinik, MBBS,<sup>a,b,c</sup> Sandeep Prabhu, MBBS,<sup>a,b,c</sup> Liang-han Ling, MBBS, PhD,<sup>a,b,c</sup>  
Jonathan M. Kalman, MBBS, PhD,<sup>c,d</sup> Peter M. Kistler, MBBS, PhD<sup>a,b,c</sup>



# Alcohol and AF



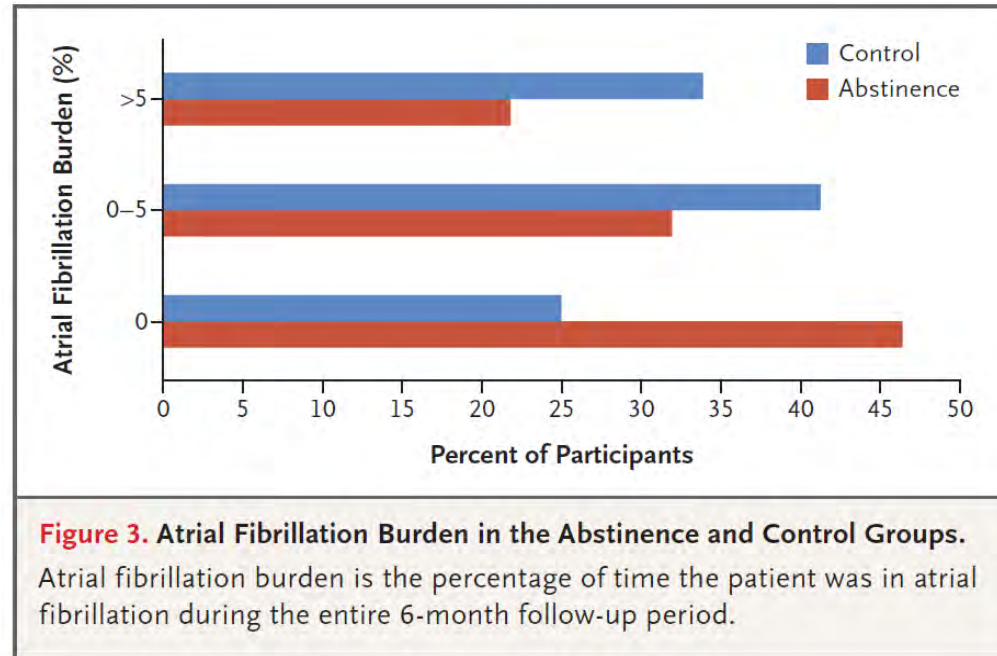
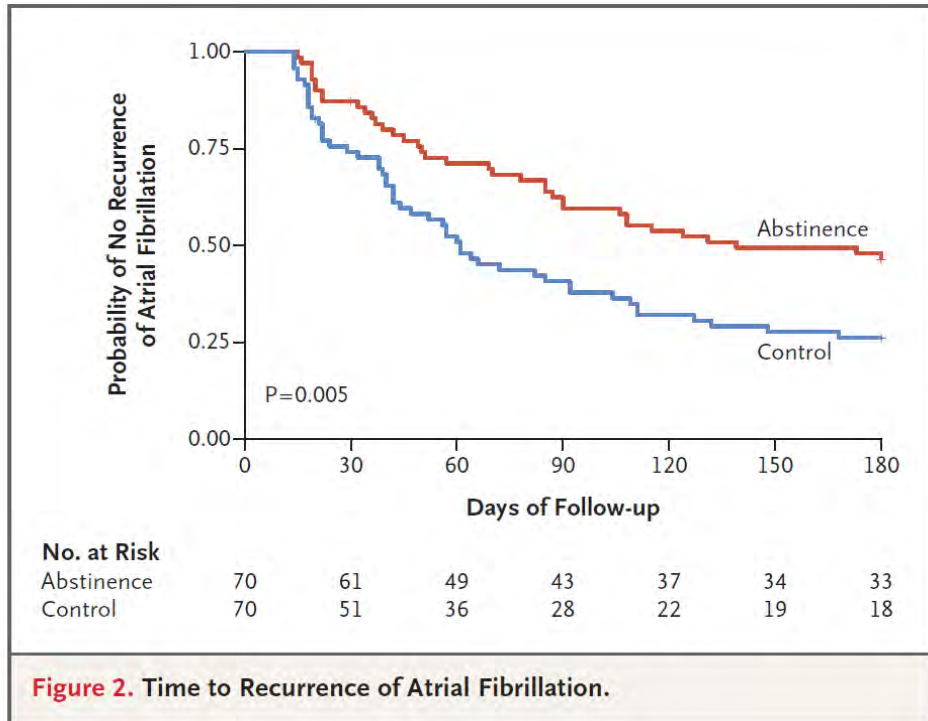
- Positive association between AF and alcohol (**binge drinking >5 standard drinks; moderate 7-21 drinks/weeks, heavy drinking >21 drinks/week**)
- 1 standard drink = 12g of alcohol
- **For each extra alcoholic drink per day, AF incidence increased 8%**
- Although a **small amount of alcohol is considered cardioprotective, these benefits do not extend to AF!**

## Alcohol and Atrial Fibrillation

### A Sobering Review

Aleksandr Voskoboinik, MBBS,<sup>a,b,c</sup> Sandeep Prabhu, MBBS,<sup>a,b,c</sup> Liang-han Ling, MBBS, PhD,<sup>a,b,c</sup>  
Jonathan M. Kalman, MBBS, PhD,<sup>c,d</sup> Peter M. Kistler, MBBS, PhD<sup>a,b,c</sup>

# Alcohol Abstinence in Drinkers with Atrial Fibrillation



- 85% Men
- Symptomatic Paroxysmal AF or symptomatic persistent AF with rhythm control strategy
- **Abstinance group:** reduced alcohol intake from 16.8±7.7 to 2.1±3.7 standard drinks per week (**reduction of 87%**)
- **Control group:** reduced alcohol intake from 16.4±6.9 to 13.2±6.5 drinks per week (**reduction of 19.5%**)

- AF recurred in 53% in abstinance group
- AF recurred in 73% in control group
- Abstinance group had a longer period before recurrence of AF than control group

ORIGINAL ARTICLE

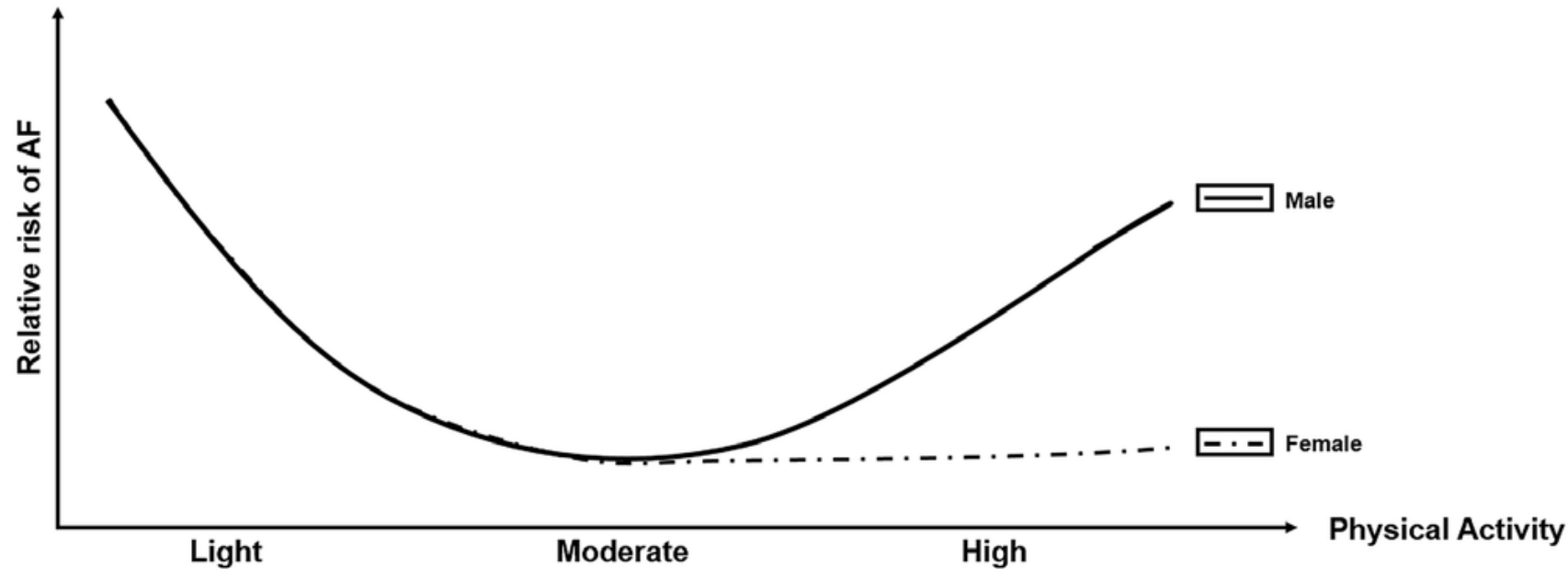
Alcohol Abstinence in Drinkers with Atrial Fibrillation

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The NEW ENGLAND JOURNAL of MEDICINE

N Engl J Med 2020;382:20-8.

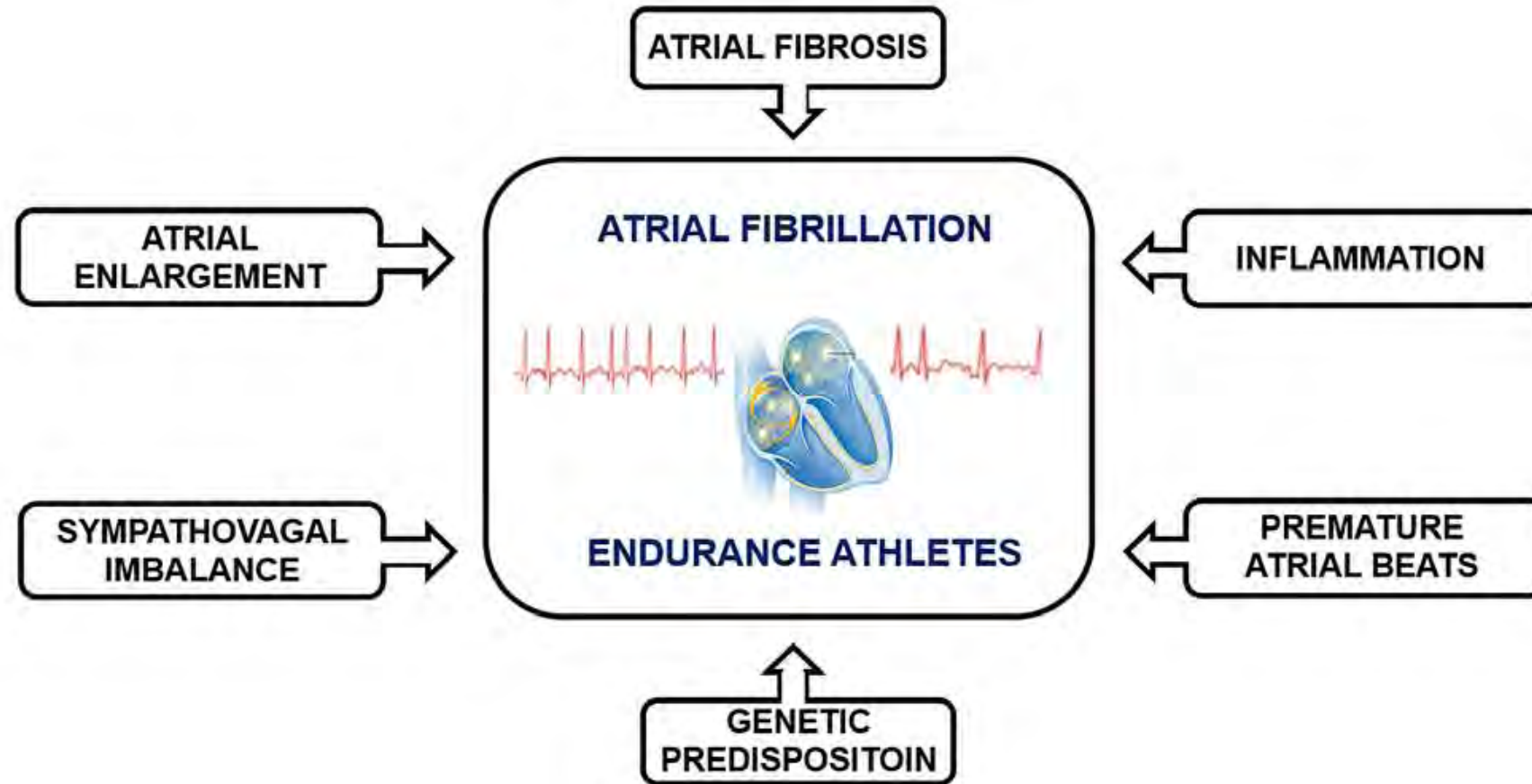
# Physical Activity and AF



- Proposed Cutoffs for the ascending flank of the U-Curve:
  - **5h/week vigorous intensity 30y**
  - **Accumulated sport practice >1500h**
- Gender difference might be explained by (in women):
  - Fewer comorbidities, shorter duration of exposure to vigorous exercise, lower sympathetic resting tone, lower BP, sex hormones...

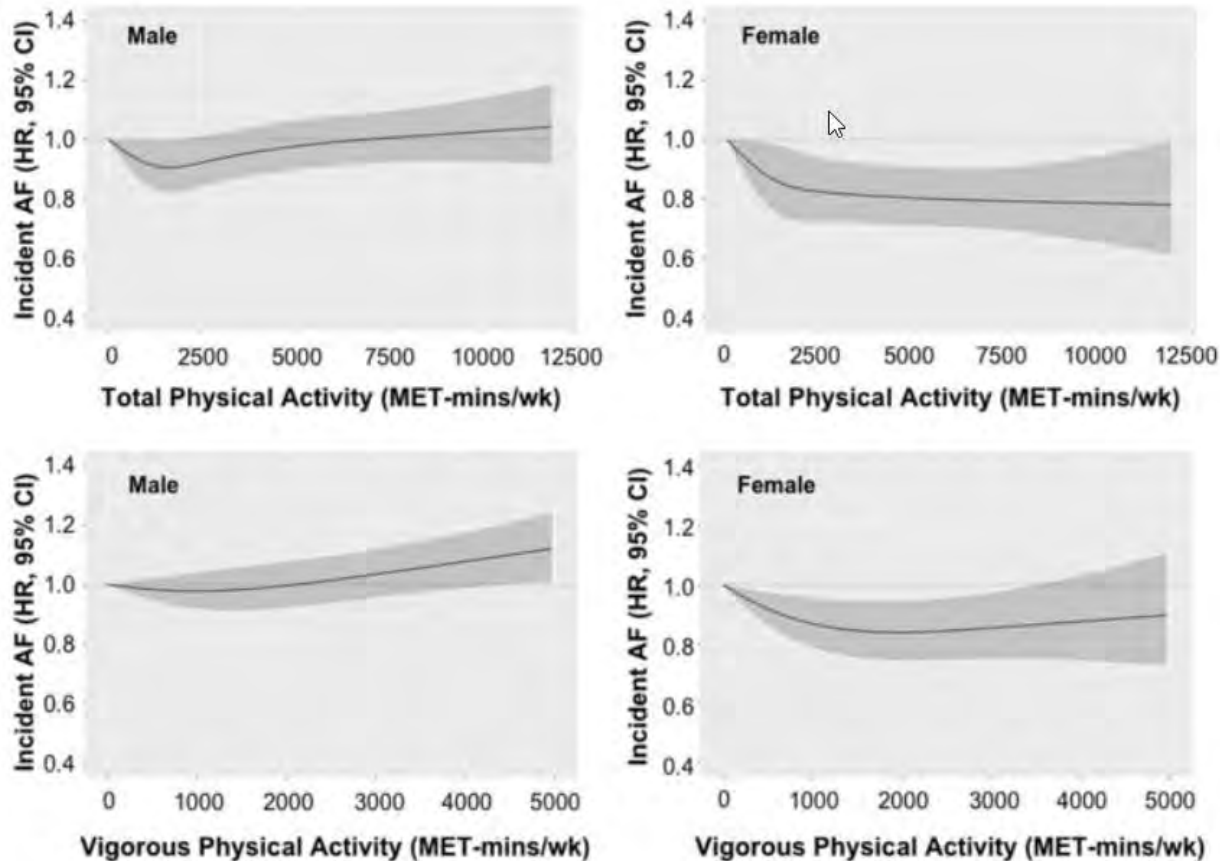
## Endurance Exercise and Atrial Fibrillation – A State of the Art Review

# Physical Activity and AF



## Endurance Exercise and Atrial Fibrillation – A State of the Art Review

# Association between physical activity and risk of incident AF



- 3 types of activity (walking, moderate, vigorous-intensity activities)
- Moderate 4-6METs
- Vigorous >6METs
- *Running at 10km/h ≈ 10METs*
- *Jogging at 8km/h ≈ 8METs*
- *Brisk walking at 6.4Km/h ≈ 5METs*
  
- Example: Brisk walk (6.4km/h) 3 days a week @ 5METs for 60minutes = 3x5x60 = 900METs/min/wk
- **Lower cut-off of 500 MET-min/wk to reflect the lower range of guideline-recommended physical activity**

Association between physical activity and risk of incident arrhythmias in 402 406 individuals: evidence from the UK Biobank cohort

Adrian D. Elliott<sup>1\*</sup>, Dominik Linz<sup>1</sup>, Ricardo Mishima<sup>1</sup>, Kadhim Kadhim<sup>1</sup>, Celine Gallagher<sup>1</sup>, Melissa E. Middeldorp<sup>1</sup>, Christian V. Verdicchio<sup>1</sup>, Jeroen M.L. Hendriks<sup>1</sup>, Dennis H. Lau<sup>1</sup>, Andre La Gerche<sup>2</sup>, and Prashanthan Sanders<sup>1</sup>

<sup>1</sup>Centre for Heart Rhythm Disorders, University of Adelaide and Royal Adelaide Hospital, Adelaide SA 5000, Australia; and <sup>2</sup>Sports Cardiology Laboratory, Baker Heart & Diabetes Institute, Melbourne, Victoria 3004, Australia

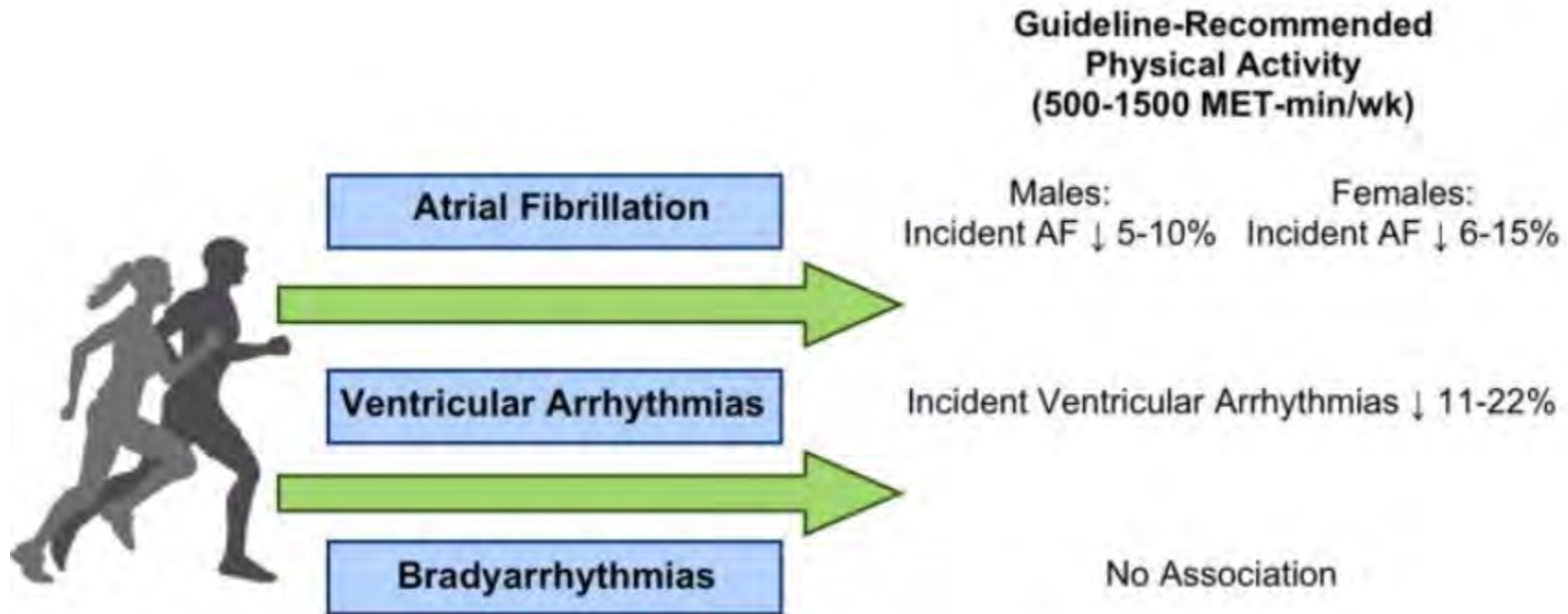


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European Heart Journal (2020) 41, 1479–1486  
doi:10.1093/eurheartj/ehz897

CLINICAL RESEARCH  
Arrhythmia/electrophysiology

# Association between physical activity and risk of incident AF



Association between physical activity and risk of incident arrhythmias in 402 406 individuals: evidence from the UK Biobank cohort

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CLINICAL RESEARCH  
Arrhythmia/electrophysiology

# Recommendations for lifestyle interventions and management of risk factors and concomitant diseases in patients with AF (1)

Recommendations	Class	Level
Identification and management of risk factors and concomitant diseases is recommended as an integral part of treatment in AF patients.	I	B
Modification of unhealthy lifestyle and targeted therapy of intercurrent conditions is recommended to reduce AF burden and symptom severity.	I	B
Opportunistic screening for AF is recommended in hypertensive patients.	I	B
Attention to good BP control is recommended in AF patients with hypertension to reduce AF recurrences and risk of stroke and bleeding.	I	B
In obese patients with AF, weight loss together with management of other risk factors should be considered to reduce AF incidence, AF progression, AF recurrences, and symptoms.	IIa	B

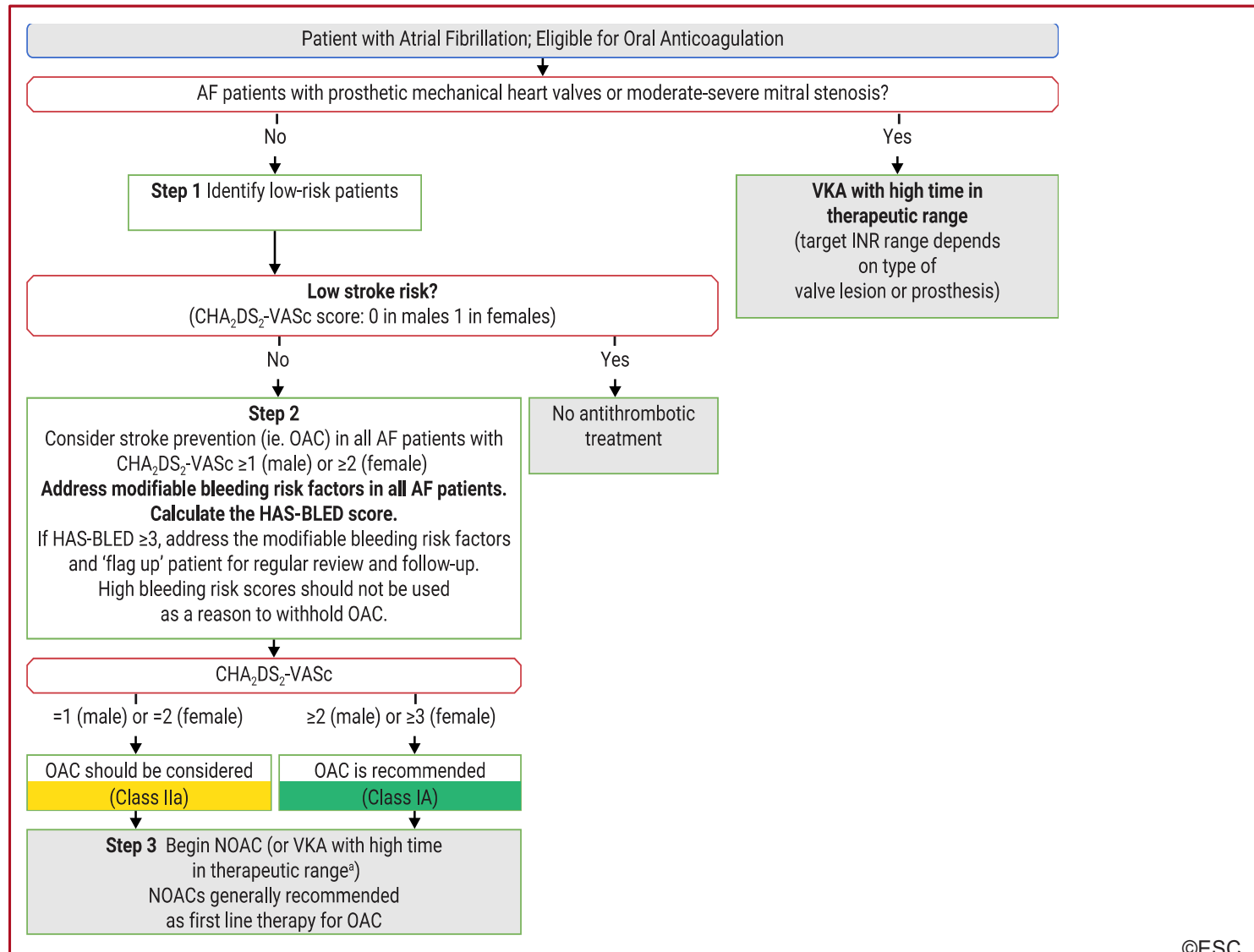
# Recommendations for lifestyle interventions and management of risk factors and concomitant diseases in patients with AF (2)

Recommendations	Class	Level
Advice and management to avoid alcohol excess should be considered for AF prevention and in AF patients considered for OAC therapy	<b>Ila</b>	<b>B</b>
Physical activity should be considered to help prevent AF incidence or recurrence, with the exception of excessive endurance exercise, which may promote AF.	<b>Ila</b>	<b>C</b>
Opportunistic screening for AF should be considered in patients with OSA.	<b>Ila</b>	<b>C</b>
Optimal management of OSA may be considered, to reduce AF incidence, AF progression, AF recurrences, and symptoms.	<b>Ilb</b>	<b>C</b>



# Stroke prevention in AF

# Stroke Prevention in AF



**Table 11** Clinical risk factors for stroke, transient ischaemic attack, and systemic embolism in the CHA<sub>2</sub>DS<sub>2</sub>-VASc score

CHA <sub>2</sub> DS <sub>2</sub> -VASc risk factor	Points
<b>Congestive heart failure</b> Signs/symptoms of heart failure or objective evidence of reduced left ventricular ejection fraction	+1
<b>Hypertension</b> Resting blood pressure >140/90 mmHg on at least two occasions or current antihypertensive treatment	+1
<b>Age 75 years or older</b>	+2
<b>Diabetes mellitus</b> Fasting glucose >125 mg/dL (7 mmol/L) or treatment with oral hypoglycaemic agent and/or insulin	+1
<b>Previous stroke, transient ischaemic attack, or thromboembolism</b>	+2
<b>Vascular disease</b> Previous myocardial infarction, peripheral artery disease, or aortic plaque	+1
<b>Age 65–74 years</b>	+1
<b>Sex category (female)</b>	+1

CHA<sub>2</sub>DS<sub>2</sub>-VASc = Congestive Heart failure, hypertension, Age ≥75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74, and Sex (female).

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## Table 10 Clinical risk factors in the HAS-BLED score (1)

Risk factors and definitions		Points awarded
<b>H</b>	<b>Uncontrolled hypertension</b> Systolic BP >160 mmHg	1
<b>A</b>	<b>Abnormal renal and/or hepatic function</b> Dialysis, transplant, serum creatinine >200 µmol/L, cirrhosis, bilirubin > × 2 upper limit of normal, AST/ALT/ALP >3 × upper limit of normal	1 point for each
<b>S</b>	<b>Stroke</b> Previous ischaemic or haemorrhagic <sup>a</sup> stroke	1
<b>B</b>	<b>Bleeding history or predisposition</b> Previous major haemorrhage or anaemia or severe thrombocytopenia	1

<sup>a</sup>Haemorrhagic stroke would also score 1 point under the 'B' criterion.

## Table 10 Clinical risk factors in the HAS-BLED score (2)

Risk factors and definitions		Points awarded
<b>L</b>	<b>Labile INR<sup>b</sup></b> TTR <60% in patient receiving VKA	1
<b>E</b>	<b>Elderly</b> Aged >65 years or extreme frailty	1
<b>D</b>	<b>Drugs or excessive alcohol drinking</b> Concomitant use of antiplatelet or non-steroidal anti-inflammatory drugs; and/or excessive <sup>c</sup> alcohol per week	1 point for each
<b>Maximum score</b>		<b>9</b>

<sup>b</sup>Only relevant if patient receiving a VKA.

<sup>c</sup>Alcohol excess or abuse refers to a high intake (e.g. >14 units per week), where the clinician assesses there would be an impact on health or bleeding risk.

# Non-Vitamin K antagonist oral anticoagulants

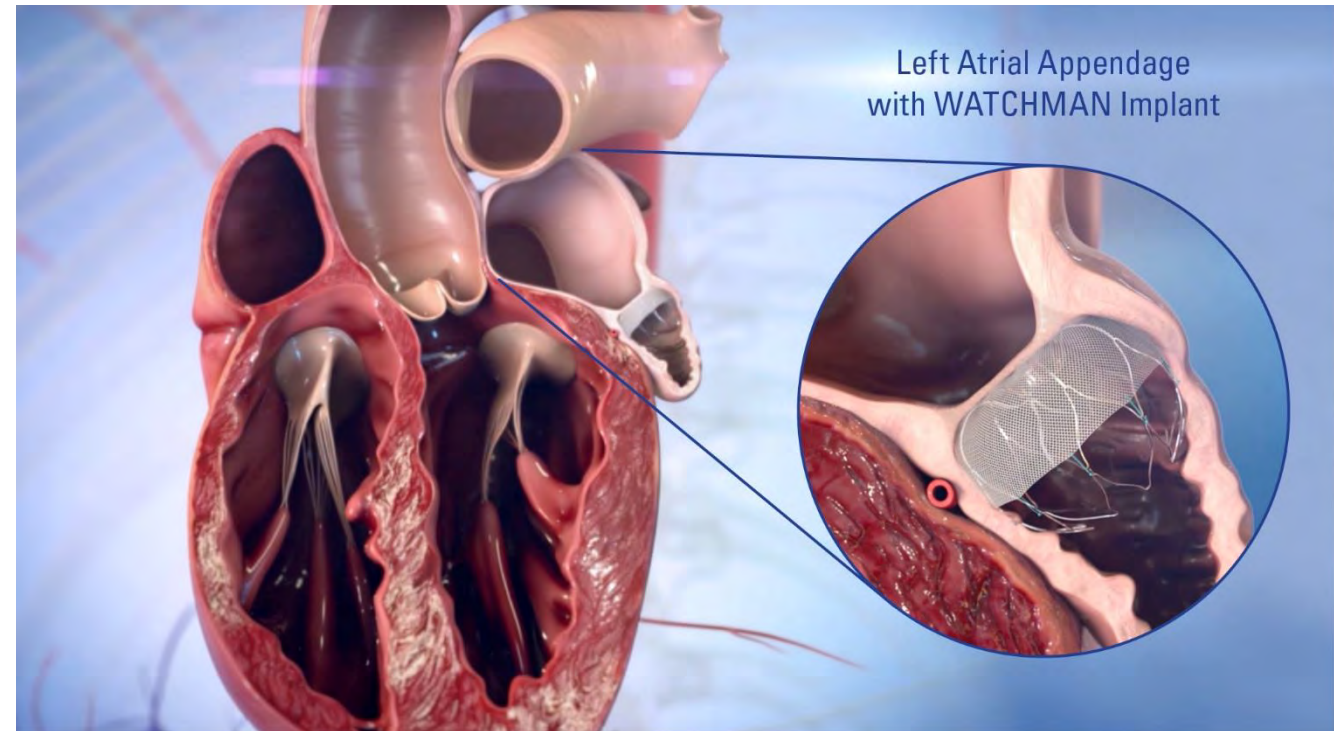
- **Direct thrombin inhibitor** : Dabigatran (Pradaxa<sup>®</sup>)
- **Factor Xa inhibitors**: Apixaban (Eliquis<sup>®</sup>), Edoxaban (Lixiana<sup>®</sup>), Rivaroxaban (Xarelto<sup>®</sup>)
- A meta-analysis based on the **high-dose treatment groups** of the pivotal studies of **warfarin vs. NOACs** included 42 411 patients receiving a NOAC and 29 272 receiving warfarin. NOACs in these dosages significantly **reduced stroke or systemic embolic events by 19% compared with warfarin** (RR 0.81; 95% CI 0.73– 0.91; P<0.0001), **mainly driven by a reduction in haemorrhagic stroke** (RR 0.49; 95% CI 0.38–0.64; P< 0.0001)<sup>1</sup>
- **Mortality is 10% lower** in patients randomized to NOAC therapy (RR 0.90; 95% CI 0.85 – 0.95; P=0.0003)<sup>1</sup>
- **Intracranial haemorrhage is halved** (RR 0.48; 95% CI 0.39 – 0.59; P<0.0001)<sup>1</sup>
- **Gastrointestinal bleeding events are more frequent** (RR 1.25; 95% CI 1.01 – 1.55; P=0.04)<sup>1</sup>

## Table 11 Dose selection criteria for NOACs

	Dabigatran	Rivaroxaban	Apixaban	Edoxaban
<b>Standard dose</b>	150 mg b.i.d.	20 mg o.d.	5 mg b.i.d.	60 mg o.d.
<b>Lower dose</b>	110 mg b.i.d.			
<b>Reduced dose</b>		15 mg o.d.	2.5 mg b.i.d.	30 mg o.d.
<b>Dose-reduction criteria</b>	Dabigatran 110 mg b.i.d. in patients with: <ul style="list-style-type: none"> <li>• Age ≥80 years</li> <li>• Concomitant use of verapamil, or</li> <li>• Increased bleeding risk</li> </ul>	CrCl 15–49 mL/min	At least 2 of 3 criteria: <ul style="list-style-type: none"> <li>• Age ≥80 years,</li> <li>• Body weight ≤60 kg, or</li> <li>• Serum creatinine ≥1.5 mg/dL (133 μmol/L)</li> </ul>	If any of the following: <ul style="list-style-type: none"> <li>• CrCl 15–50 mL/min,</li> <li>• Body weight ≤60 kg,</li> <li>• Concomitant use of dronedarone, ciclosporin, erythromycin, or ketoconazole</li> </ul>

# Left atrial appendage occlusion and exclusion

- Only one device (Watchman®) has been compared with VKA therapy in randomized trials<sup>1</sup>
- **LAA occlusion is non-inferior to VKA treatment for the prevention of stroke in AF patients with moderate stroke risk, with a possibility of lower bleeding rates in the patients who continued follow-up<sup>1</sup>**
- LAA occlusion may also **reduce stroke risk in patients with contraindications to OAC<sup>2</sup>**
- A large recent European registry reported a **high rate of implantation success (98%)**, with an acceptable procedure-related complication rate of **4% at 30 days<sup>3</sup>** (device embolization, pericardial effusion with or without tamponade, device thrombus with stroke, femoral hematoma)



1.Reddy VV, Doshi SK, Sievert H, Buchbinder M, Neuzil P, Huber K, Halperin JL, Holmes D. Percutaneous left atrial appendage closure for stroke prophylaxis in patients with atrial fibrillation: 2.3-Year Follow-up of the PROTECT AF (Watchman Left Atrial Appendage System for Embolic Protection in Patients with Atrial Fibrillation) Trial. Circulation 2013;127:720–729.  
2.Reddy VV, Mobius-Winkler S, Miller MA, Neuzil P, Schuler G, Wiebe J, Sick P, Sievert H. Left atrial appendage closure with the Watchman device in patients with a contraindication for oral anticoagulation: the ASAP study (ASA Plavix Feasibility Study With Watchman Left Atrial Appendage Closure Technology). J Am Coll Cardiol 2013;61:2551–2556.  
3. Boersma LV, Schmidt B, Betts TR, Sievert H, Tamburino C, Teiger E, Pokushalov E, Kische S, Schmitz T, Stein KM, Bergmann MW, EWOLUTION investigators. Implant success and safety of left atrial appendage closure with the WATCHMAN device: peri-procedural outcomes from the EWOLUTION registry. Eur Heart J 2016;37:2465–2474.

# Recommendations for the prevention of thromboembolic events in AF (5)

Recommendations for occlusion or exclusion of the LAA	Class	Level
LAA occlusion may be considered for stroke prevention in patients with AF and contraindications for long-term anticoagulant treatment (e.g. intracranial bleeding without a reversible cause).	<b>IIb</b>	<b>B</b>
Surgical occlusion or exclusion of the LAA may be considered for stroke prevention in patients with AF undergoing cardiac surgery.	<b>IIb</b>	<b>C</b>



# Cardioversion

# Cardioversion Recommendations

Recommendations	Class	Level
Electrical cardioversion of AF is recommended in patients with <b>acute haemodynamic instability</b> to restore cardiac output.	I	B
Cardioversion of AF (either electrical or pharmacological) is recommended in symptomatic patients with persistent or long-standing persistent AF as part of rhythm control therapy.	I	B
Pre-treatment with <b>amiodarone</b> , flecainide, ibutilide, or propafenone should be considered to enhance success of electrical cardioversion and prevent recurrent AF	IIa	B
In patients with <b>no history of ischaemic or structural heart disease, flecainide, propafenone, or vernakalant</b> are recommended for pharmacological cardioversion of new-onset AF	I	A
In selected patients with <b>infrequent recent-onset AF</b> and no significant structural or ischaemic heart disease, a single oral dose of flecainide or propafenone (the ' <b>pill in the pocket</b> ' approach) should be considered for patient-led cardioversion, following safety assessment.	IIa	B
In patients with <b>ischaemic and/or structural heart disease, amiodarone</b> is recommended for cardioversion of AF.	I	A
Vernakalant may be considered as an alternative to amiodarone for pharmacological conversion of AF in patients without hypotension, severe heart failure or severe structural heart disease (especially aortic stenosis).	IIb	B

# Stroke Risk Management Pericardioversion

Recommendations	Class	Level
Anticoagulation with heparin or a NOAC should be initiated <b>as soon as possible before every cardioversion</b> of AF or atrial flutter.	Ila	B
For cardioversion of AF/atrial flutter, effective anticoagulation is recommended for a <b>minimum of 3 weeks before</b> cardioversion.	I	B
Transoesophageal echocardiography (TOE) is recommended to exclude cardiac thrombus as an <b>alternative to preprocedural</b> anticoagulation when early cardioversion is planned.	I	B
Early cardioversion can be performed <b>without TOE</b> in patients with a definite duration of <b>AF &lt;48 hours</b> .	Ila	B
In patients at risk for stroke, anticoagulant therapy should be continued <b>long-term</b> after cardioversion according to the long-term anticoagulation recommendations, irrespective of the method of cardioversion or the apparent maintenance of sinus rhythm. In patients <b>without stroke risk factors, anticoagulation is recommended for 4 weeks after cardioversion</b> .	I	B
In patients where thrombus is identified on TOE, effective anticoagulation is recommended for at least 3 weeks.	I	C
In patients with a <b>definite duration of AF &lt;24 hours</b> and a very low stroke risk (CHA <sub>2</sub> DS <sub>2</sub> -VASc of 0 in men or 1 in women) post-cardioversion, <b>anticoagulation for 4 weeks may be omitted</b>	Ilb	C

Rate control

# Long-term heart rate control of AF

Perform echocardiogram (IC)  
Choose initial rate control therapy (IB) and combination therapy if required (IIaC)  
Target initial resting heart rate **<110 bpm** (IIaB), avoiding bradycardia

**LVEF <40%**

Beta-blocker

Digoxin

Atrioventricular node ablation should be considered to control heart rate in patients unresponsive or intolerant to intensive rate and rhythm control therapy, and not eligible for rhythm control by LA ablation, accepting that these patients will become pacemaker dependent.

Add digoxin

beta-blocker

**LVEF ≥40%**

Diltiazem/verapamil

Beta-blocker

Digoxin

Add digoxin

Add digoxin

verapamil or  
beta-blocker

**IIa**

**B**

Rhythm control

# Recommendations for rhythm control

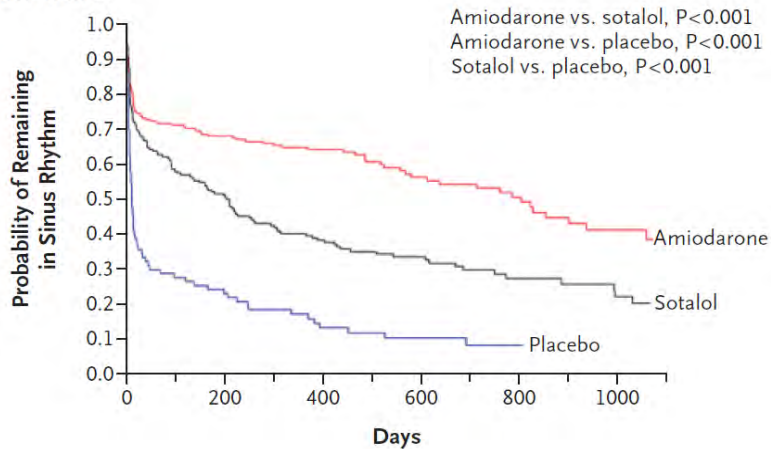
Recommendations	Class	Level
Rhythm control therapy is recommended for symptom and QoL improvement in symptomatic patients with AF.	I	A

Drug	Dose	Main Contra-indications and precautions	Warning signs warranting discontinuation	AV nodal slowing	Suggested ECG monitoring during initiation
<b>Amiodarone</b>	600mg in divided doses for 4 weeks, 400mg for 4 weeks, then 200mg once daily	Caution when using concomitant therapy with QT-prolonging drugs and in patients with SAN or AV node and conduction disease. The dose of VKAs and of digitalis should be reduced. Increased risk of myopathy with statins. Caution in patients with pre-existing liver disease.	<b>QT prolongation &gt;500 ms</b>	10–12 bpm in AF	<b>Baseline, 1 week, 4 weeks</b>
<b>Flecainide</b>	100-150mg twice daily	Contra-indicated if CrCl <50 mg/mL, liver disease, IHD or reduced LV ejection fraction Caution in the presence of SAN or AV node or conduction disease. CYP2D6 inhibitors (e.g. fluoxetine or tricyclic antidepressants) increase plasma concentration.	<b>QRS duration increases &gt;25% above baseline</b>	None	<b>Baseline, day 1, day 2–3</b>
<b>Propafenone</b>	150mg-300mg three times daily	Contra-indicated in IHD or reduced LV ejection fraction. Caution in the presence of SAN or AV node and conduction disease, renal or liver impairment, and asthma. Increases concentration of digitalis and warfarin.	<b>QRS duration increase &gt;25% above baseline</b>	Slight	<b>Baseline, day 1, day 2–3</b>
<b>Sotalol</b>	80-160mg twice daily	Contra-indicated in the presence of significant LV hypertrophy, systolic heart failure, asthma, pre-existing QT prolongation, hypokalaemia, CrCl<50 mg/mL. Moderate renal dysfunction requires careful adaptation of dose.	<b>QT interval &gt;500 ms, QT prolongation by &gt;60 ms upon therapy initiation</b>	Similar to high dose blockers	<b>Baseline, day 1, day 2–3</b>

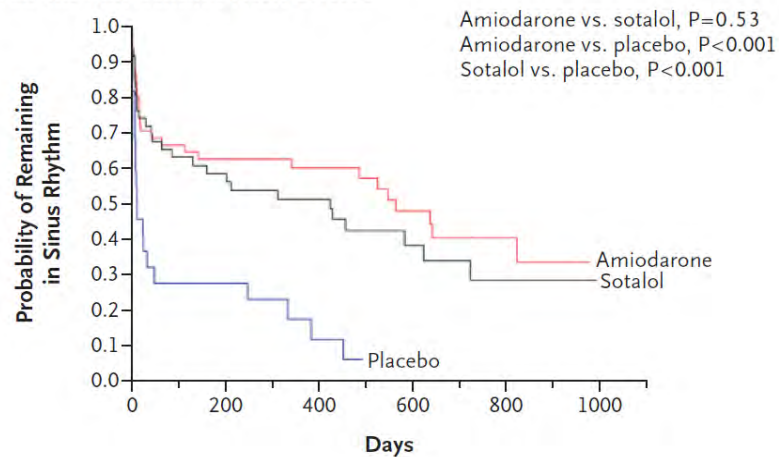


# Amiodarone vs Sotalol

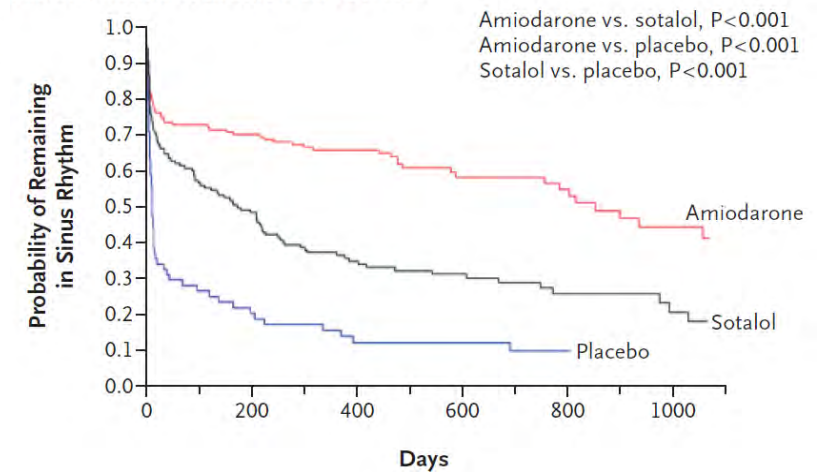
A All Patients



Patients with Ischemic Heart Disease

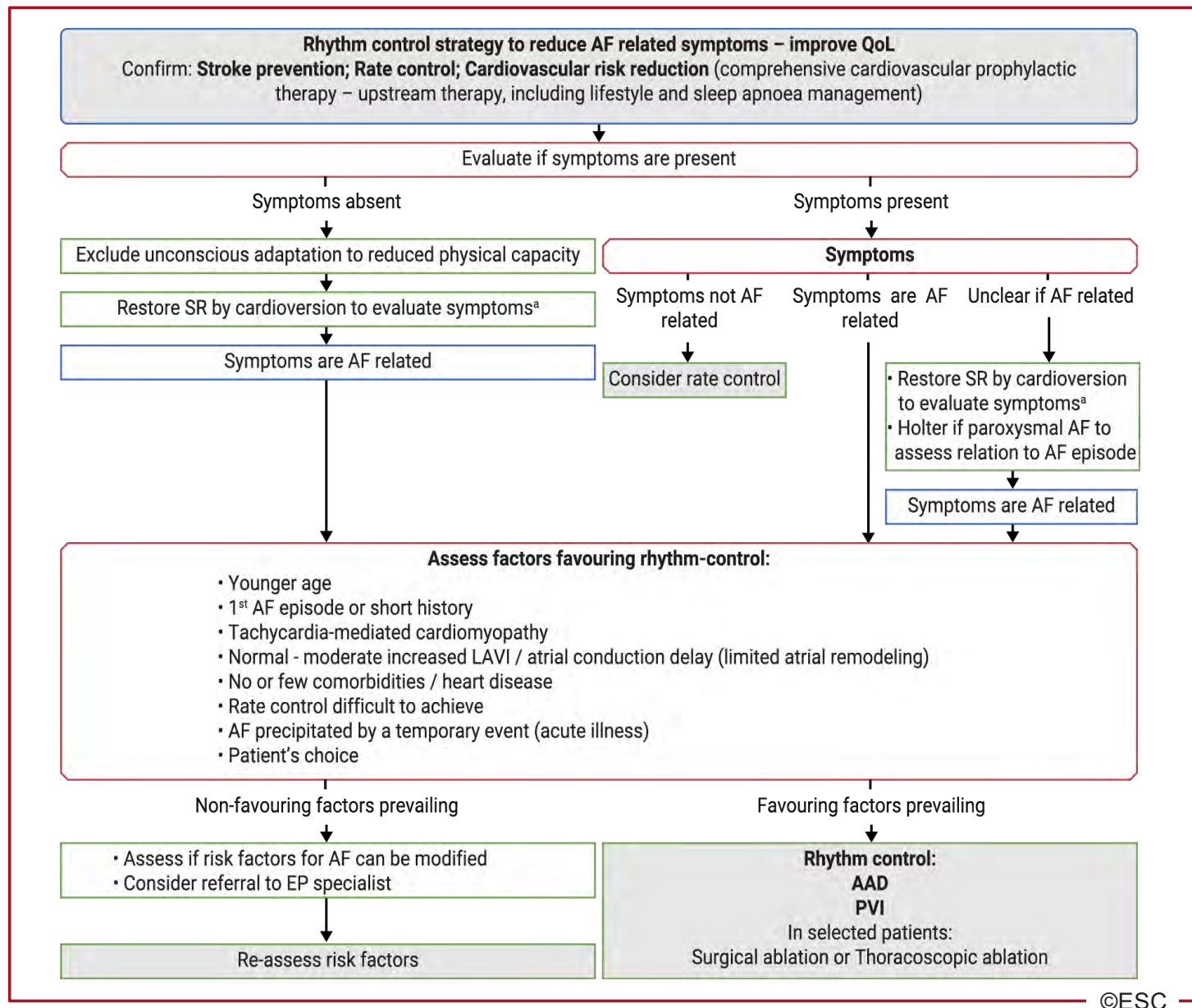


Patients without Ischemic Heart Disease



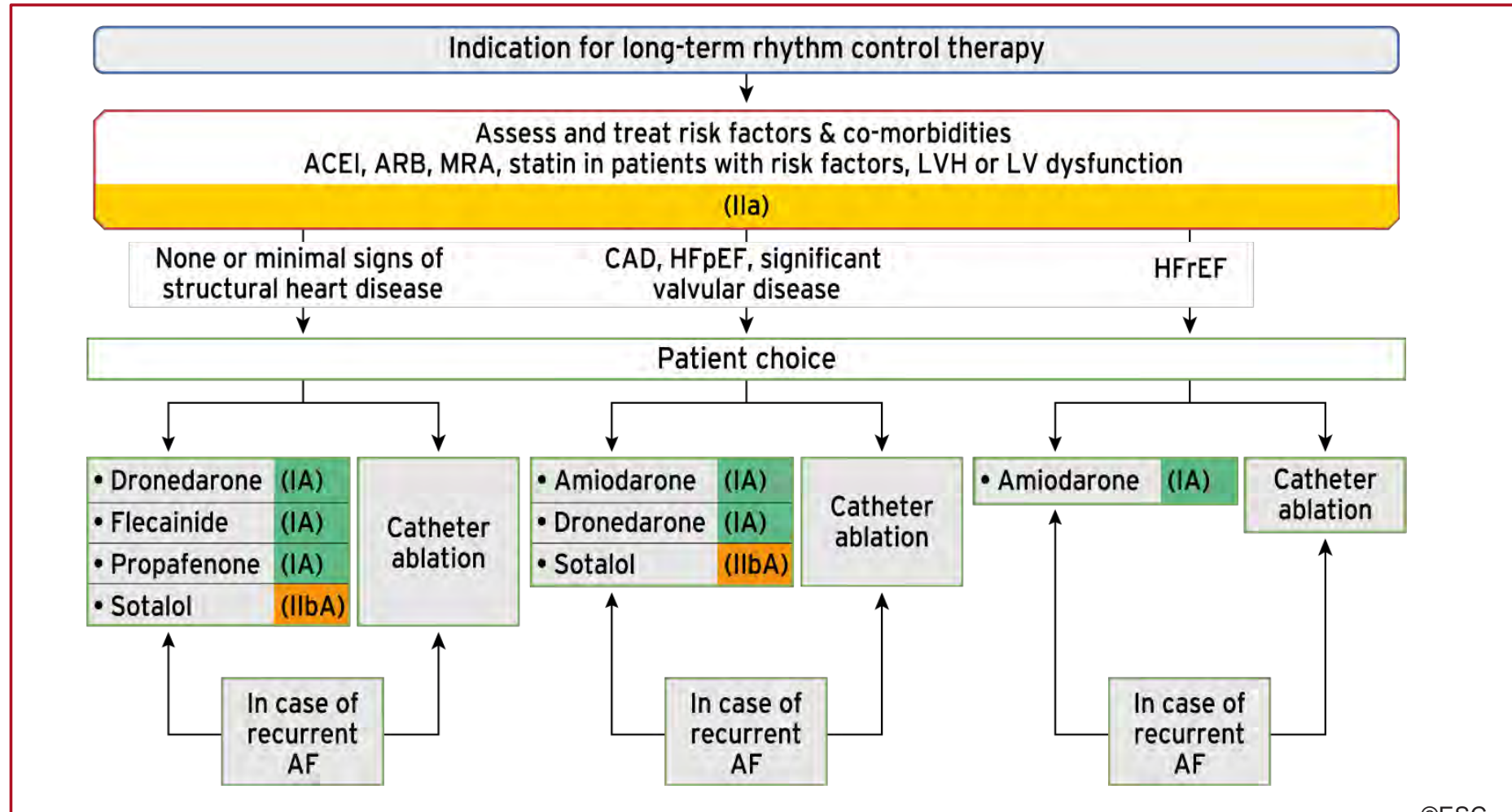
**Amiodarone and sotalol are equally efficacious in converting atrial fibrillation to sinus rhythm. Amiodarone is superior for maintaining sinus rhythm, but both drugs have similar efficacy in patients with ischemic heart disease.**

ORIGINAL ARTICLE



<sup>a</sup>Consider cardioversion to confirm that the absence of symptoms is not due to unconscious adaptation to reduced physical and/or mental capacity.

# Figure 19 Long-term rhythm control therapy



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Rate control vs rhythm  
control

# Rate vs rhythm control

<b>Rhythm control</b>	<b>Rate control</b>
<p><b>Advantages</b></p> <ul style="list-style-type: none"><li>• Fewer symptoms</li><li>• Better exercise tolerance</li><li>• Improved haemodynamic function</li><li>• Less need for anticoagulation</li></ul>	<p><b>Advantages</b></p> <ul style="list-style-type: none"><li>• Avoidance of antiarrhythmic drugs</li><li>• Good efficacy of rate control drugs</li><li>• Fewer admissions to hospital</li><li>• More cost effective</li><li>• Risk of stroke similar to rhythm control</li><li>• Mortality similar to rhythm control</li></ul>
<p><b>Disadvantages</b></p> <ul style="list-style-type: none"><li>• Side effects of antiarrhythmic drugs</li><li>• Poor efficacy of antiarrhythmic drugs</li><li>• Expensive</li><li>• High rates of recurrence</li><li>• Increased admissions to hospital</li></ul>	<p><b>Disadvantages</b></p> <ul style="list-style-type: none"><li>• Risks of anticoagulation</li><li>• Risk of tachycardiomyopathy</li><li>• Symptoms of persisting arrhythmia</li><li>• Atrial remodelling (permanent)</li></ul>

# Rate vs rhythm control

The strategy of restoring and maintaining sinus rhythm had no clear advantage over the strategy of controlling the ventricular rate and allowing atrial fibrillation to persist.

**Trend toward increased mortality** in association with the **rhythm-control** strategy (P=0.08)

The rates of the **composite end point** of *death, disabling stroke, disabling anoxic encephalopathy, major bleeding, and cardiac arrest* were also **similar in the two groups** (P=0.33).

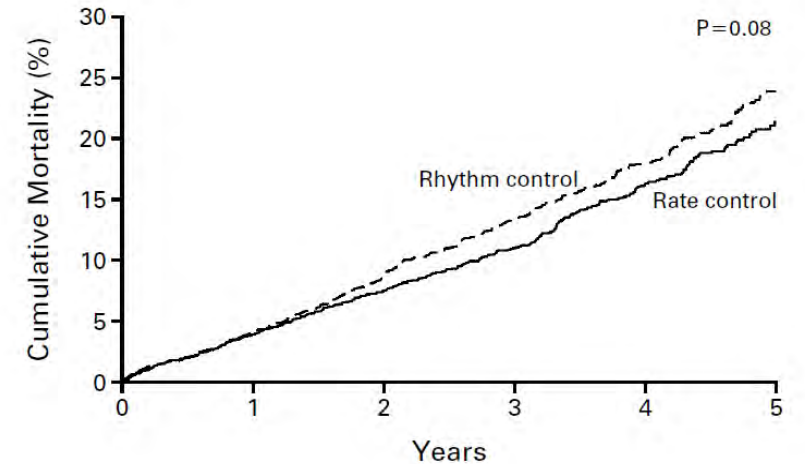
The majority of strokes in both groups occurred in patients who had **stopped taking warfarin or whose INR was subtherapeutic** at the time of the stroke, in general agreement with previously reported observations.

**Torsade de pointes or bradycardic arrest** occurred **more often in the rhythm-control group** than in the rate-control group.

The patients in the **rhythm-control group** were significantly **more likely to be hospitalized and have adverse drug effects** than those in the rate-control group

This study also suggest **that continuous anticoagulation is warranted in all patients with atrial fibrillation and risk factors for stroke, even when sinus rhythm appears to be restored and maintained.**

## Mortality (any cause)



No. OF DEATHS	number (percent)					
Rhythm control	0	80 (4)	175 (9)	257 (13)	314 (18)	352 (24)
Rate control	0	78 (4)	148 (7)	210 (11)	275 (16)	306 (21)

**Figure 1.** Cumulative Mortality from Any Cause in the Rhythm-Control Group and the Rate-Control Group.

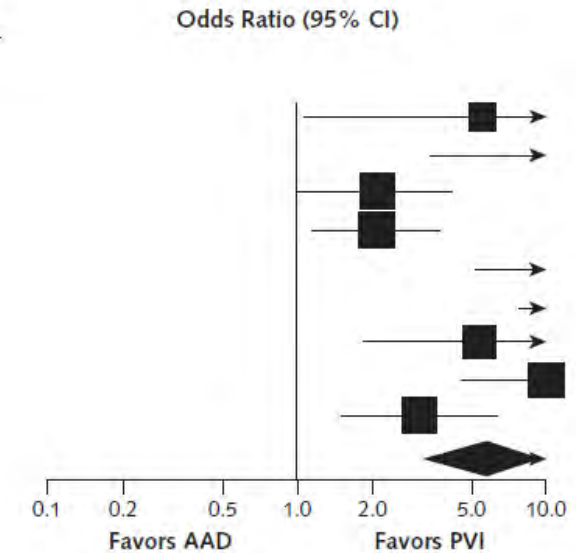


# Rate vs rhythm control

Maintenance of SR AAD vs PVI

E.

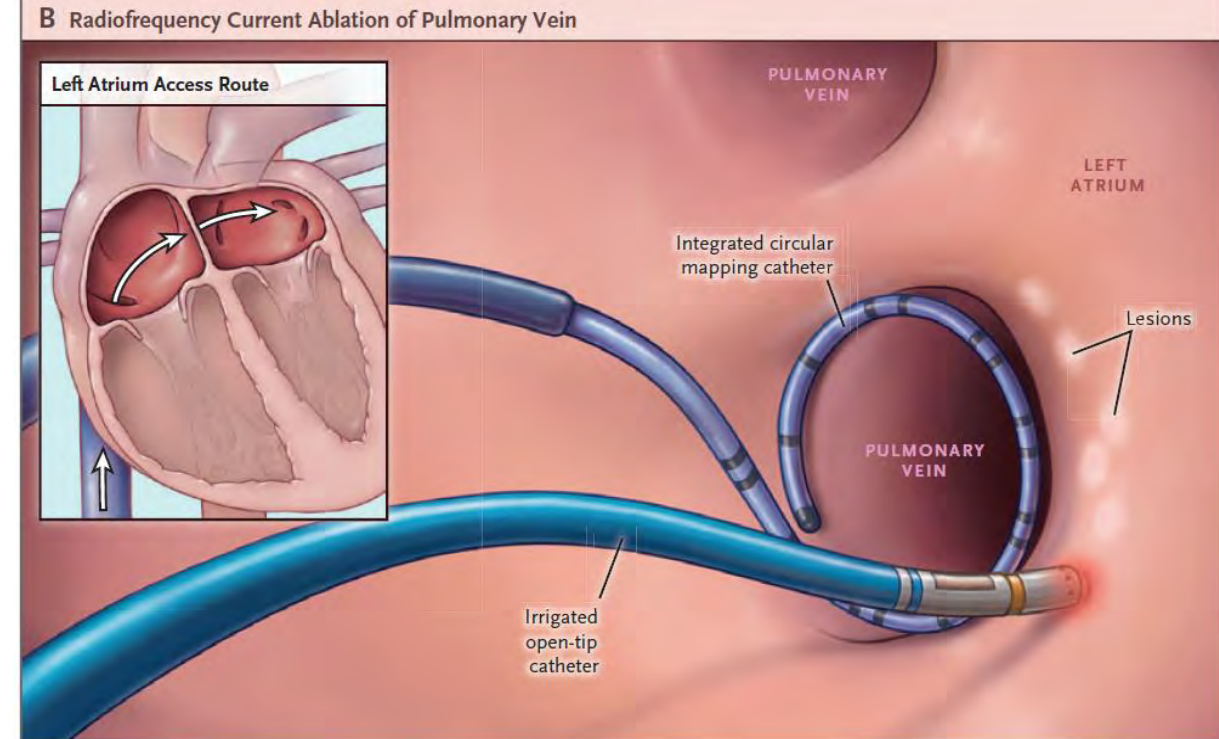
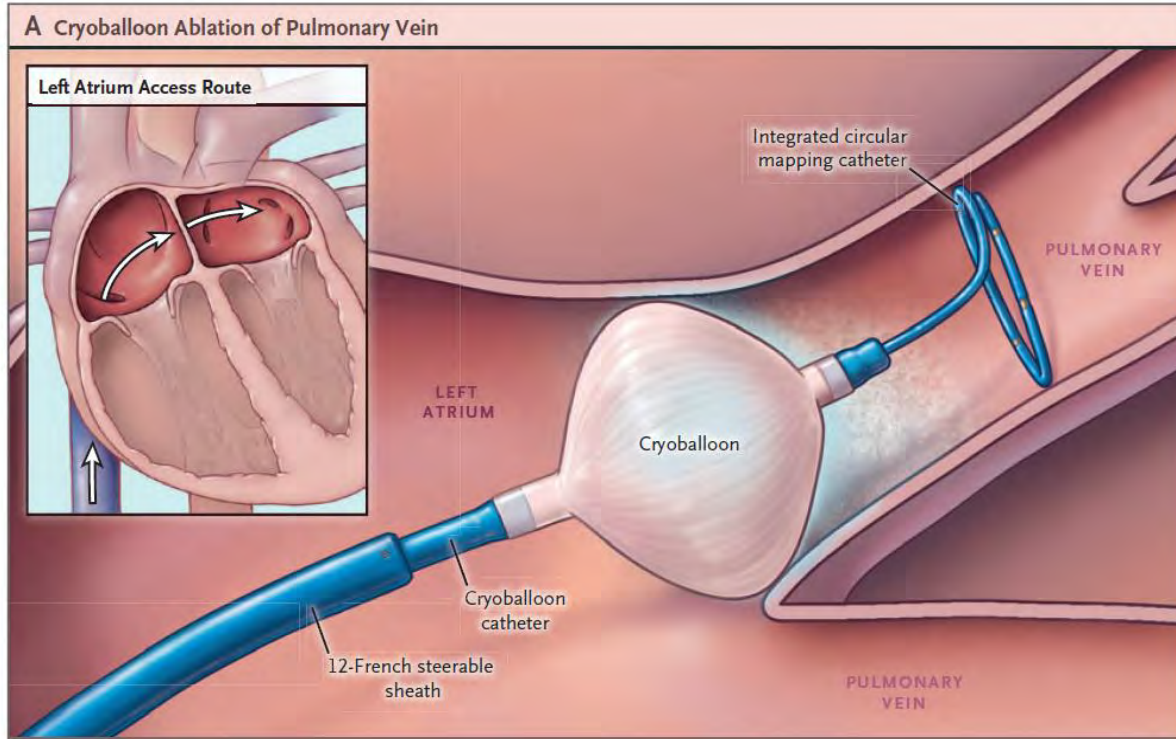
Study, Year (Reference)	Odds Ratio (95% CI)	Maintenance of Sinus Rhythm/Total, n/N	
		PVI	AAD
Krittayaphong et al, 2003 (147)	5.500 (1.065–28.416)	11/14	6/15
Wazni et al, 2005 (157)	11.846 (3.387–41.433)	28/32	13/35
Oral et al, 2006 (114)	2.066 (1.028–4.155)	57/77	40/69
Pappone et al, 2006 (115)	2.048 (1.130–3.711)	72/99	56/99
Stabile et al, 2006 (119)	13.300 (5.069–34.894)	38/68	6/69
Jaïs et al, 2008 (143)	24.769 (8.634–71.059)	46/52	13/55
Forleo et al, 2009 (112)	5.333 (1.839–15.471)	28/35	15/35
Wilber et al, 2010 (126)	9.917 (4.509–21.808)	70/106	10/61
Mont et al, 2014 (132)	3.059 (1.494–6.263)	69/98	21/48
Overall	5.874 (3.180–10.849)		



# Catheter Ablation vs Medical Therapy



# Catheter Ablation of AF



ORIGINAL ARTICLE

## Cryoballoon or Radiofrequency Ablation for Paroxysmal Atrial Fibrillation

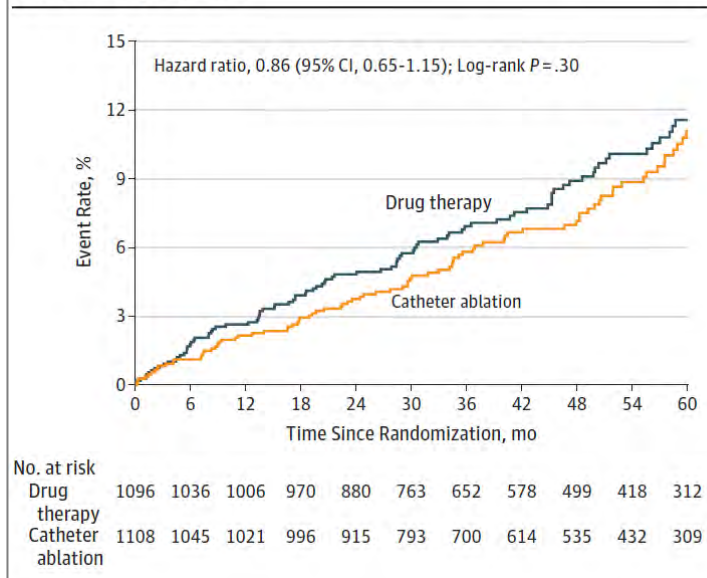
Karl-Heinz Kuck, M.D., Josep Brugada, M.D., Alexander Fürnkranz, M.D., Andreas Metzner, M.D., Feifan Ouyang, M.D., K.R. Julian Chun, M.D., Arif Elvan, M.D., Ph.D., Thomas Arentz, M.D., Kurt Bestehorn, M.D., Stuart J. Pocock, Ph.D., Jean-Paul Albenque, M.D., Ph.D., and Claudio Tondo, M.D., Ph.D., for the FIRE AND ICE Investigators®

N Engl J Med 2016;374:2235-45.

# Catheter Ablation vs Medical Therapy

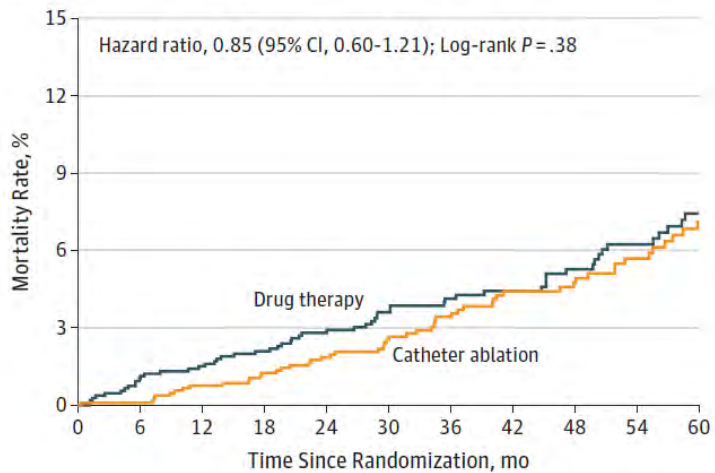
Cumulative risk of **death, disabling stroke, serious bleeding or cardiac arrest**

Figure 2. Kaplan-Meier Estimates of the Incidence of the Primary End Point



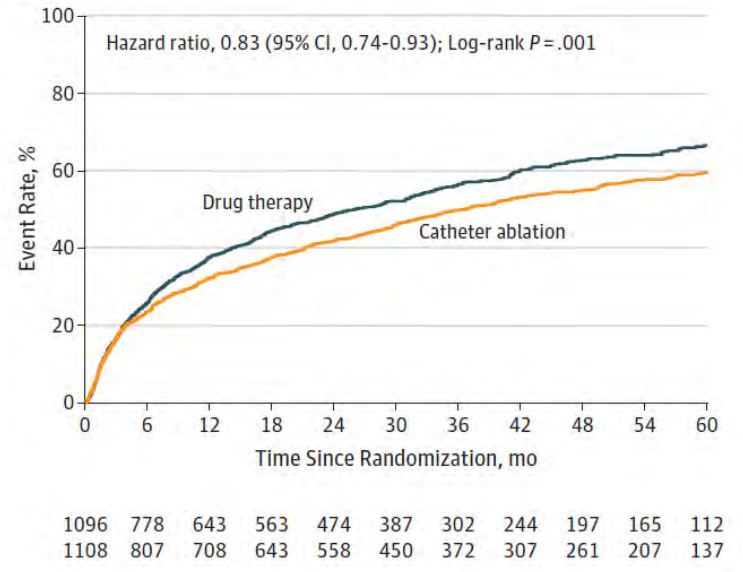
All cause mortality

A All-cause mortality



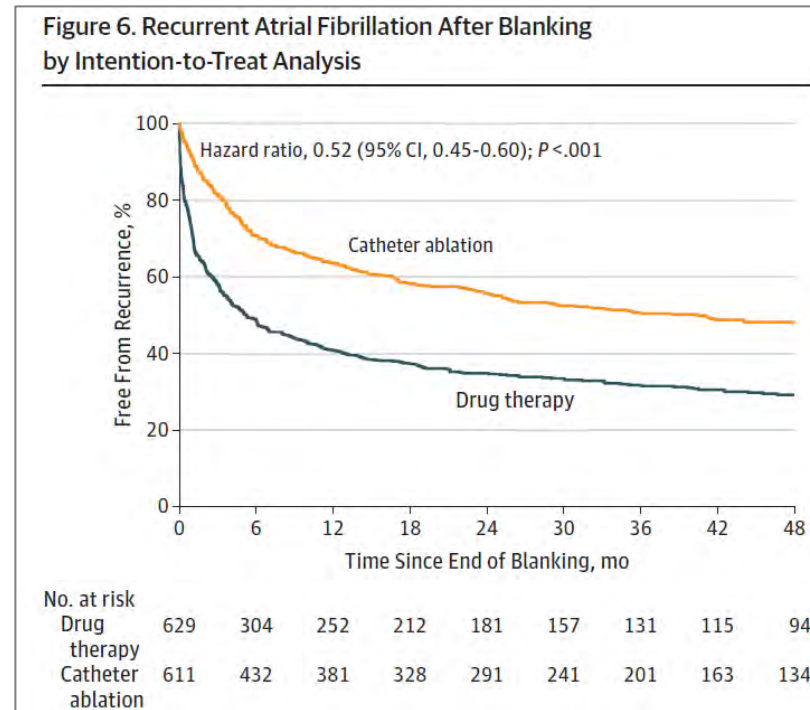
Mortality or Cardiovascular Hospitalization

B Mortality or cardiovascular hospitalization



Among patients with **AF**, the strategy of **catheter ablation**, compared with medical therapy, **does not significantly reduce the primary composite end point of death, disabling stroke, serious bleeding, or cardiac arrest.**

# Catheter Ablation vs Medical Therapy



**Catheter ablation is associated with a lower AF recurrence rate than drug therapy (50% vs 69% at 3years post blanking follow-up).**

# Catheter Ablation vs Medical Therapy

Figure 1. Patients Who Reported Being in Atrial Fibrillation Currently or Within the Past Month

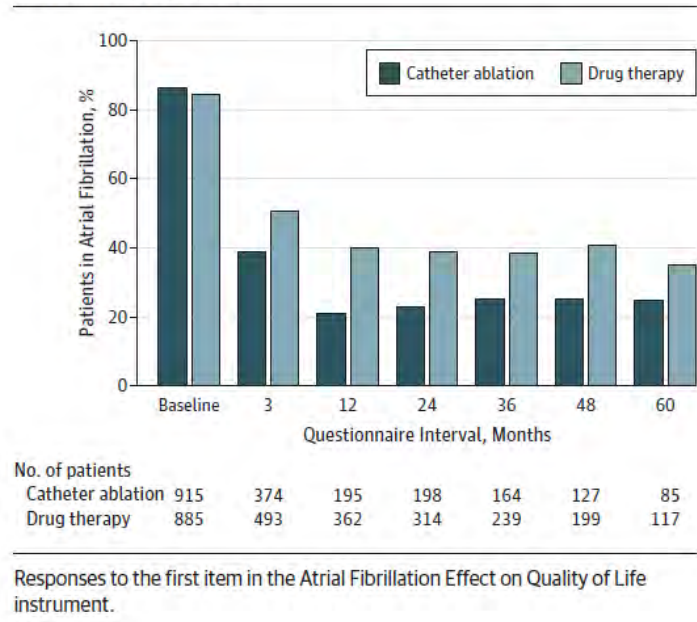
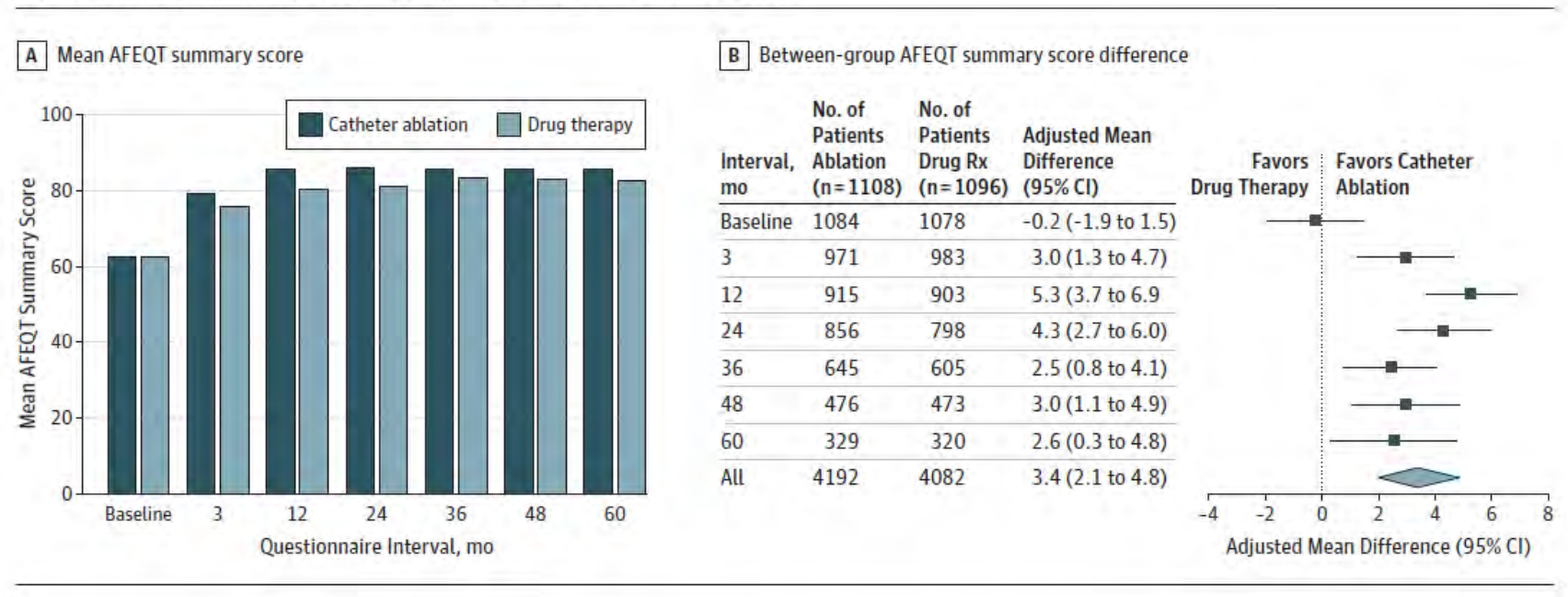
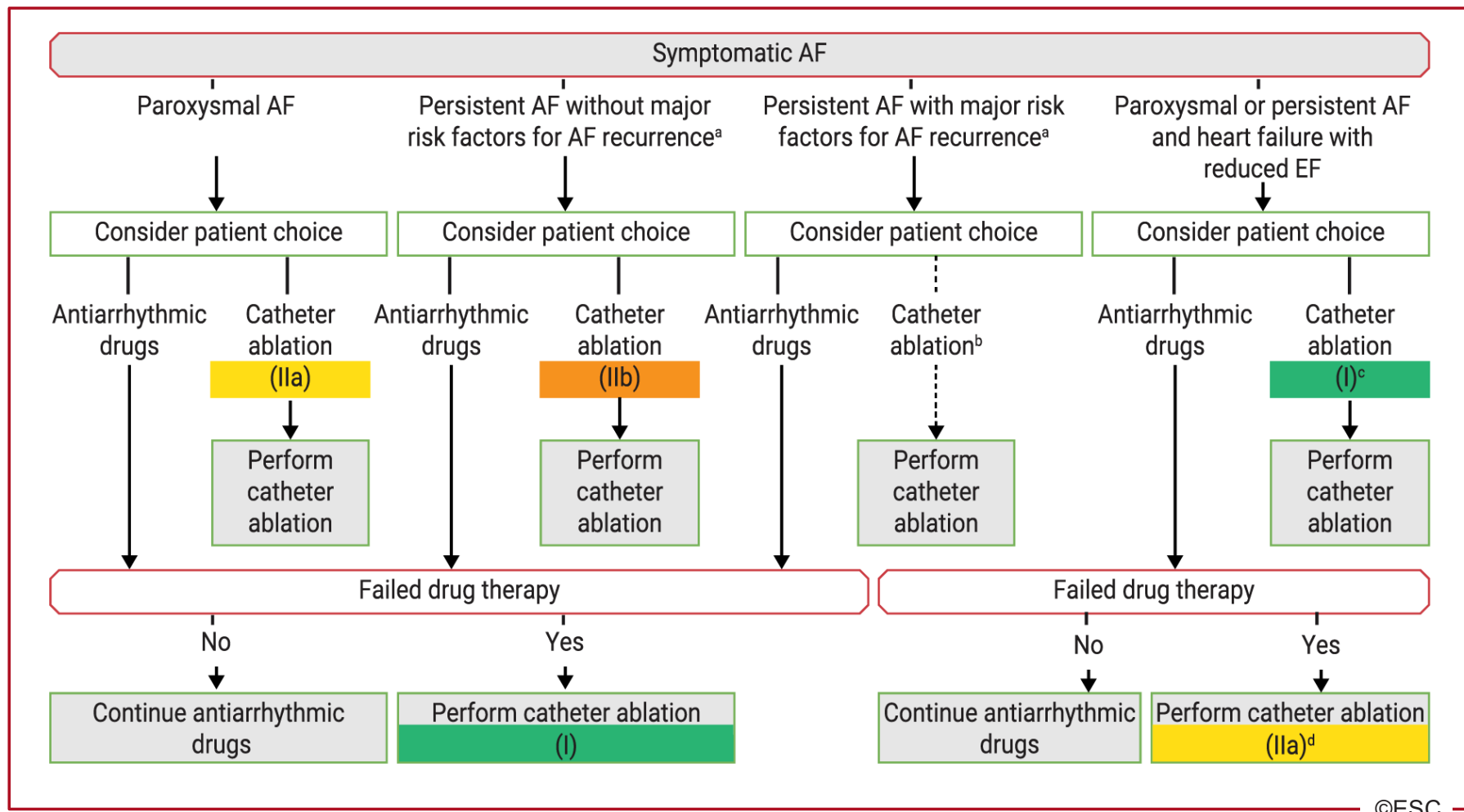


Figure 2. Atrial Fibrillation Effect on Quality of Life (AFEQT) Summary Scores



Catheter **ablation** provides **incremental symptomatic and QOL benefits over drug therapy** that is clinically important and statistically significant for patients with AF

# Figure 17 Indications for catheter ablation of symptomatic AF



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<sup>a</sup>Significantly enlarged LA volume, advanced age, long AF duration, renal dysfunction, and other cardiovascular risk factors. <sup>b</sup>In rare individual circumstances, catheter ablation may be carefully considered as first-line therapy. <sup>c</sup>Recommended to reverse LV dysfunction when tachycardiomyopathy is highly probable. <sup>d</sup>To improve survival and reduce hospitalization.

# Follow-up after Catheter Ablation of AF

- **Recurrences beyond the first month** post-ablation are generally predictive of late recurrences, but recurrent symptoms may be due to ectopic beats or other non-sustained arrhythmia; conversely the presence of asymptomatic AF after ablation is well described
- Monitoring may be performed with intermittent **ECG, Holter, external or implanted loop recorder**, or smartphone monitor. Patients should be **first reviewed at a minimum of 3 months and annually thereafter**
- Continuing **AAD treatment for 6 weeks to 3 months** may reduce early AF recurrences, rehospitalizations and cardioversions during this period
- AADs may be **weaned, ceased, or continued** according to **symptoms and rhythm status**
- **OAC therapy is continued for 2 months** following ablation in all patients. **Beyond this time**, a decision to continue OAC is determined **primarily by the presence of CHA<sub>2</sub>-DS<sub>2</sub>-VASc stroke risk factors rather than the rhythm status!**

# Conclusions

## Pillars of AF Management



# Fin

Merci pour votre attention!