Tucet novinek,

kterých jste si třeba nevšimli a které mě pozitivně /negativně zaujaly na nových guidelines

Čihák R

INSTITUT KLINICKÉ A EXPERIMENTÁLNÍ MEDICÍNY
KLINIKA KARDIOLOGIE
IKEM

Guidelines fibrilace síní 2016

European Heart Journal Advance Access published August 27, 2016

European Heart Journal
doi:10.1093/eurheartj/ehw210

ESC GUIDELINES

2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS

The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC)

Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC

Endorsed by the European Stroke Org

Authors/Task Force Members: Paulus Kirchhof

ELSEVIER

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/crvasa

Cor et Vasa

Doporučení pro… | Guidelines

Doporučené postupy ESC 2016 pro léčbu fibrilace síní formulované ve spolupráci s EACTS.

Souhrn dokumentu připravený
Českou kardiologickou společností

ČESKÁ KARDIOLOGICKÁ SPOLEČNOST
THE CZECH SOCIETY OF CARDIOLOGY

(2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. Summary of the document prepared by the Czech Society of Cardiology)

Robert Čihák[a], Luděk Haman[b], Miloš Táborský[c]

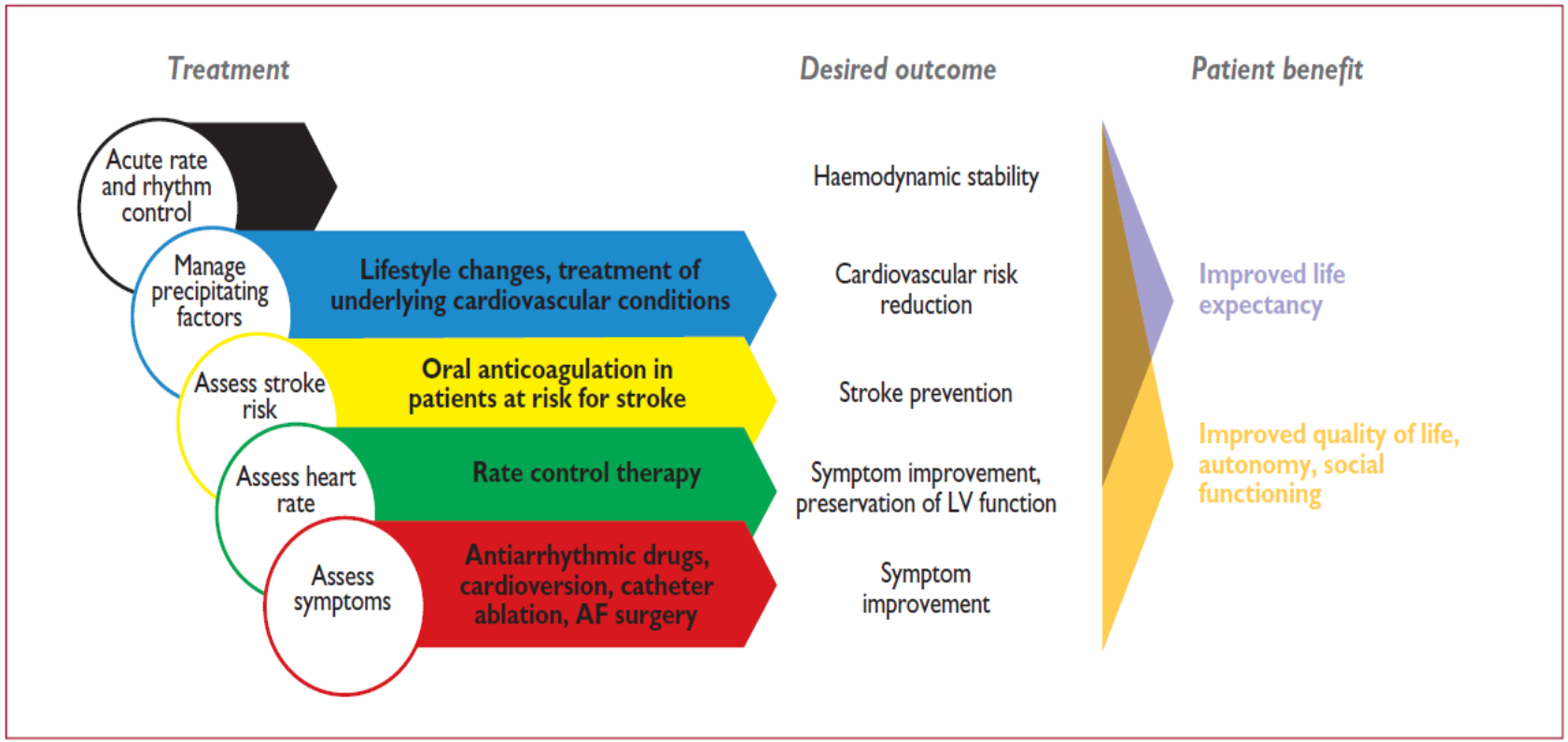
Morbidita a mortalita spojená s FS

Event	Association with AF
Death	Increased mortality, especially cardiovascular mortality due to sudden death, heart failure or stroke.
Stroke	20–30% of all strokes are due to AF. A growing number of patients with stroke are diagnosed with 'silent', paroxysmal AF.
Hospitalizations	10–40% of AF patients are hospitalized every year.
Quality of life	Quality of life is impaired in AF patients independent of other cardiovascular conditions.
Left ventricular dysfunction and heart failure	Left ventricular dysfunction is found in 20–30% of all AF patients. AF causes or aggravates LV dysfunction in many AF patients, while others have completely preserved LV function despite long-standing AF.
Cognitive decline and vascular dementia	Cognitive decline and vascular dementia can develop even in anticoagulated AF patients. Brain white matter lesions are more common in AF patients than in patients without AF.

Onemocnění či stavy spojené s FS

Characteristic/comorbidity	Association with AF
Genetic predisposition (based on multiple common gene variants associated with AF)[64]	HR range 0.4–3.2
Older age[19] 50–59 years 60–69 years 70–79 years 80–89 years	HR: 1.00 (reference) 4.98 (95% CI 3.49–7.10) 7.35 (95% CI 5.28–10.2) 9.33 (95% CI 6.68–13.0)
Hypertension (treated) vs. none[19]	HR 1.32 (95% CI 1.08–1.60)
Heart failure vs. none[19]	HR 1.43 (95% CI 0.85–2.40)
Valvular heart disease vs. none[205]	RR 2.42 (95% CI 1.62–3.60)
Myocardial infarction vs. none[19]	HR 1.46 (95% CI 1.07–1.98)
Thyroid dysfunction[206, 207] Hypothyroidism Subclinical hyperthyroidism Overt hyperthyroidism	(reference: euthyroid) HR 1.23 (95% CI 0.77–1.97) RR 1.31 (95% CI 1.19–1.44) RR 1.42 (95% CI 1.22–1.63)
Obesity[19, 208] None (BMI <25 kg/m²) Overweight (BMI 25–30 kg/m²) Obese (BMI ≥31 kg/m²)	HR: 1.00 (reference) 1.13 (95% CI 0.87–1.46) 1.37 (95% CI 1.05–1.78)
Diabetes mellitus vs. none[19]	HR 1.25 (95% CI 0.98–1.60)
Chronic obstructive pulmonary disease[209] FEV1 ≥80% FEV1 60–80% FEV1 <60%	RR: 1.00 (reference) 1.28 (95% CI 0.79–2.06) 2.53 (95% CI 1.45–4.42)
Obstructive sleep apnoea vs. none[210]	HR 2.18 (95% CI 1.34–3.54)
Chronic kidney disease[211] None Stage 1 or 2 Stage 3 Stage 4 or 5	OR: 1.00 (reference) 2.67 (95% CI 2.04–3.48) 1.68 (95% CI 1.26–2.24) 3.52 (95% CI 1.73–7.15)
Smoking[212] Never Former Current	HR: 1.00 (reference) 1.32 (95% CI 1.10–1.57) 2.05 (95% CI 1.71–2.47)
Alcohol consumption[213] None 1– 6 drinks/week 7–14 drinks/week 15–21 drinks/week >21 drinks/week	RR: 1.00 (reference) 1.01 (95% CI 0.94–1.09) 1.07 (95% CI 0.98–1.17) 1.14 (95% CI 1.01–1.28) 1.39 (95% CI 1.22–1.58)
Habitual vigorous exercise[214] Non-exercisers <1 day/week 1–2 days/week 3–4 days/week 5–7 days/week	RR: 1.00 (reference) 0.90 (95% CI 0.68–1.20) 1.09 (95% CI 0.95–1.26) 1.04 (95% CI 0.91–1.19) 1.20 (95% CI 1.02–1.41)

Léčba FS – ovlivnění vyvolávajících příčin (upstream)

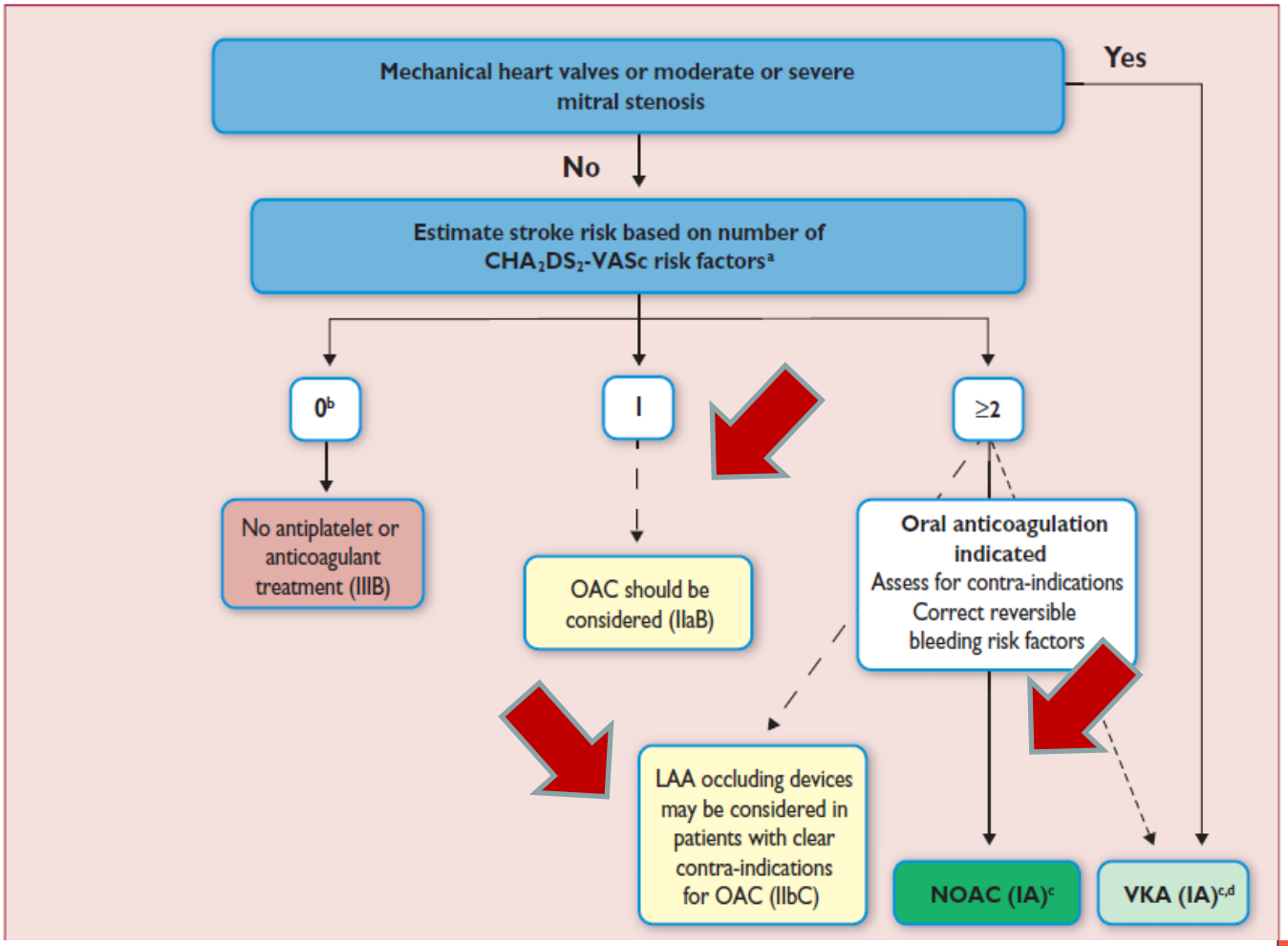


AF = atrial fibrillation; LV = left ventricular.

2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. European Heart Journal Advance Access published August 27, 2016

Guidelines fibrilace síní 2016

Doporučení k antikoagulační léčbě a k implantaci okluderu



After surgical occlusion or exclusion of the left atrial appendage, it is recommended to continue anticoagulation in at-risk patients with AF for stroke prevention.	I	B

Doporučení k antikoagulační léčbě (2)

Recommendations	Class[a]	Level[b]
Combinations of oral anticoagulants and platelet inhibitors increase bleeding risk and should be avoided in AF patients without another indication for platelet inhibition.	III (harm)	B
In male or female AF patients without additional stroke risk factors, anticoagulant or antiplatelet therapy is not recommended for stroke prevention.	III (harm)	B
Antiplatelet monotherapy is not recommended for stroke prevention in AF patients, regardless of stroke risk.	III (harm)	A
NOACs (apixaban, dabigatran, edoxaban, and rivaroxaban) are not recommended in patients with mechanical heart valves (Level of evidence B) or moderate-to-severe mitral stenosis (Level of evidence C).	III (harm)	B C

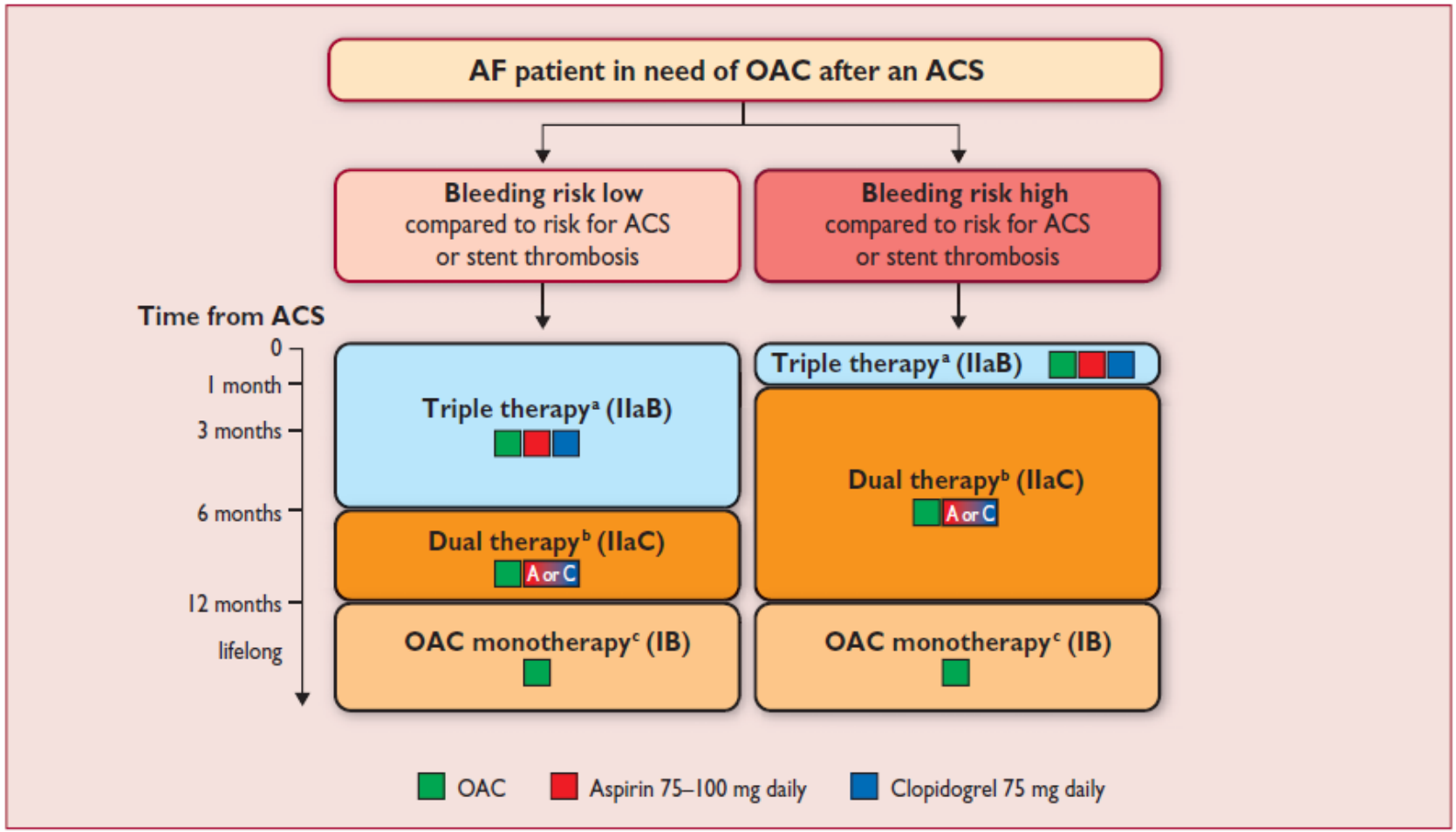
without another indication for platelet inhibition. (harm)
In male or female AF patients without additional stroke risk factors, anticoagulant or antiplatelet therapy is not recommended for stroke prevention. III (harm) B
Antiplatelet monotherapy is not recommended for stroke prevention in AF patients, regardless of stroke risk. III (harm) A
NOACs (apixaban, dabigatran, edoxaban, and rivaroxaban) are not recommended in patients with mechanical heart valves (Level of evidence B) or moderate-to-severe mitral stenosis (Level of evidence C). III (harm) B C

NOAC – chybí přehledná tabulka indikací u onemocnění chlopní

Tabulka 1 Indikace a kontraindikace podle typu a postižení chlopně pro užívání NOAC pacienty s FS

	Vhodné	Kontraindikované
Mechanická chlopenní náhrada		√
Středně těžká až těžká mitrální stenóza (obvykle revmatické etiologie)		√
Mírné a středně těžké jiné postižení nativní srdeční chlopně	√	
Těžká aortální stenóza	√	
	Omezené množství údajů	
	Většinou se provede intervence	
Biologická chlopeň[a]	√	
	(s výjimkou prvních 3 měsíců po výkonu)	
Plastika mitrální chlopně[a]	√	
	(s výjimkou prvních 3–6 měsíců po výkonu)	
PTAV a TAVI	√	
	(chybí však prospektivní údaje; může existovat potřeba kombinace s jedním nebo dvěma antiagregancii: zvážit riziko krvácení)	
Hypertrofická kardiomyopatie	√	
	(chybí však prospektivní údaje)	

NOAC – triple th



ACS = acute coronary syndrome; AF = atrial fibrillation; OAC = oral anticoagulation (using vitamin K antagonists or non-vitamin K antagonist oral anticoagulants); PCI = percutaneous coronary intervention.

[a]Dual therapy with OAC and aspirin or clopidogrel may be considered in selected patients, especially those not receiving a stent or patients at a longer time from the index event.

[b]OAC plus single antiplatelet.

[c]Dual therapy with OAC and an antiplatelet agent (aspirin or clopidogrel) may be considered in patients at high risk of coronary events.

Rate control
stále platí doporučení založena na jediné menší studii

Recommendations for rate control

Recommendations	Class[a]	Level[b]	
Beta-blockers, digoxin, diltiazem, or verapamil are recommended to control heart rate in AF patients with LVEF ≥40%.	I	B	
Beta-blockers and/or digoxin are recommended to control heart rate in AF patients with LVEF <40%.	I	B	
Combination therapy comprising different rate controlling agents should be considered if a single agent does not achieve the necessary heart rate target.	IIa	C	
In patients with haemodynamic instability or severely depressed LVEF, amiodarone may be considered for acute control of heart rate.	IIb	B	
In patients with permanent AF (i.e. where no attempt to restore sinus rhythm is planned), antiarrhythmic drugs should not routinely be used	III (harm)	A	
A resting heart rate of <110 bpm (i.e. lenient rate control) should be considered as the initial heart rate target for rate control therapy.	IIa	B	560
Rhythm rather than rate control strategies should be considered as the preferred management in pre-excited AF and AF during pregnancy.	IIa	C	
Atrioventricular node ablation should be considered to control heart rate in patients unresponsive or intolerant to intensive rate and rhythm control therapy, accepting that these patients will become pacemaker dependent.	IIa	B	184, 564, 569

Kontrola rytmu – ablace a chirurgie

Recommendations	Class[a]	Level[b]
Catheter ablation of symptomatic paroxysmal AF is recommended to improve AF symptoms in patients who have symptomatic recurrences of AF on antiarrhythmic drug therapy (amiodarone, dronedarone, flecainide, propafenone, sotalol) and who prefer further rhythm control therapy, when performed by an electrophysiologist who has received appropriate training and is performing the procedure in an experienced centre.	I	A
Ablation of common atrial flutter should be considered to prevent recurrent flutter as part of an AF ablation procedure if documented or occurring during the AF ablation.	IIa	B
Catheter ablation of AF should be considered as first-line therapy to prevent recurrent AF and to improve symptoms in selected patients with symptomatic paroxysmal AF as an alternative to antiarrhythmic drug therapy, considering patient choice, benefit, and risk.	IIa	B
All patients should receive oral anticoagulation for at least 8 weeks after catheter (IIaB) or surgical (IIaC) ablation.	IIa	B C
Anticoagulation for stroke prevention should be continued indefinitely after apparently successful catheter or surgical ablation of AF in patients at high-risk of stroke.	IIa	C
When catheter ablation of AF is planned, continuation of oral anticoagulation with a VKA (IIaB) or NOAC (IIaC) should be considered during the procedure, maintaining effective anticoagulation.	IIb	B C
Catheter ablation should target isolation of the pulmonary veins using radiofrequency ablation or cryothermy balloon catheters.	IIa	B
AF ablation should be considered in symptomatic patients with AF and heart failure with reduced ejection fraction to improve symptoms and cardiac function when tachycardiomyopathy is suspected.	IIa	C
AF ablation should be considered as a strategy to avoid pacemaker implantation in patients with AF-related bradycardia.	IIa	C
Catheter or surgical ablation should be considered in patients with symptomatic persistent or long-standing persistent AF refractory to AAD therapy to improve symptoms, considering patient choice, benefit and risk, supported by an AF Heart Team.	IIa	C
Minimally invasive surgery with epicardial pulmonary vein isolation should be considered in patients with symptomatic AF when catheter ablation has failed. Decisions on such patients should be supported by an AF Heart Team.	IIa	B
Maze surgery, possibly via a minimally invasive approach, performed by an adequately trained operator in an experienced centre, should be considered by an AF Heart Team as a treatment option for patients with symptomatic refractory persistent AF or post-ablation AF to improve symptoms.	IIa	C
Maze surgery, preferably biatrial, should be considered in patients undergoing cardiac surgery to improve symptoms attributable to AF, balancing the added risk of the procedure and the benefit of rhythm control therapy.	IIa	A
Concomitant biatrial maze or pulmonary vein isolation may be considered in asymptomatic AF patients undergoing cardiac surgery.	IIb	C

IK+E M

Výskyt FS u různých KMP, kanálopatií …..

Table 20 Inherited cardiomyopathies, channelopathies, and pathways associated with atrial fibrillation

Syndrome	Gene	Functional alteration	AF prevalence	References
Long QT syndrome	KCNQ1 KCNH2 SCN5A ANK2 others	I_{Ks} ↓ I_{Kr} ↓ I_{Na} ↑ I_{NaK} ↓ Various effects	5–10%	846–850
Brugada syndrome	SCN5A GPDIL SCN1B CACNA1C CACNB2b others	I_{Na} ↓ I_{Na} ↓ I_{Na} ↓ I_{Ca} ↓ I_{Ca} ↓ others	10–20%	851–855
Short QT syndrome	KCNQ1 KCNH2 KCNJ2 CACNA1C CACNB2b	I_{Ks} ↑ I_{Kr} ↑ I_{K1} ↑ I_{Ca} ↓ I_{Ca} ↓	Up to 70%	853, 856–858
Catecholaminergic VT	RYR2 CASQ2	Abnormal Ca^{2+} release from sarcoplasmic reticulum	Variable but common	859–861
Hypertrophic cardiomyopathy	Sarcomeric genes		5–15%	862–864
Wolff-Parkinson-White syndrome	PRKAG		Variable	865
Holt-Oram syndrome	TBX5		Variable	866
Arrhythmogenic right ventricular cardiomyopathy	Several desmosomal genes, unknown gene loci	reduced mechanical cell-cell contacts	>40% in patients with VTs	867, 868

Gaps in evidence – přiznání toho, že mnohé (ještě) nevíme …

15. Gaps in evidence

There are some areas of AF management that are supported by excellent evidence from multiple, adequately powered randomized trials (e.g. oral anticoagulation). Other areas, such as rhythm control therapy, integrated AF management, and lifestyle modifications are clearly developing the required evidence, while areas such as rate control are in dire need of better studies to underpin future guidelines. Here, we identify areas in need of further research.

15.1 Major health modifiers causing atrial fibrillation

Atrial fibrillation has different causes in different patients. More research is needed into the major causes (and electrophysiological mechanisms) of AF in different patient groups.[176,1024] Such research should consider the major comorbidities associated with AF, and characterize the response to AF therapy in patients with different, pathophysiologically distinct types of AF.

15.2 How much atrial fibrillation constitutes a mandate for therapy?

Technological advances allow screening for an irregular pulse using patient-operated ECG devices, smartphones, and a variety of other technologies. These may be very useful to detect silent, undiagnosed AF.[157] Adequately powered studies evaluating the diagnostic accuracy of such technologies, the diagnostic yield in different populations, the shortest duration and pattern of atrial arrhythmias conveying a stroke risk, and the effect of ECG screening on outcomes are needed.

15.3 Atrial high-rate episodes (AHRE) and need for anticoagulation

All of the information on the benefit of OAC has been generated in patients with AF diagnosed by ECG. Technological advances allow ready detection of AHRE in patients with implanted devices and an atrial lead. Such patients are at increased stroke risk, but it is unclear whether they benefit from OAC. Controlled trials evaluating OAC in AHRE patients are ongoing and will provide evidence on the best antithrombotic therapy in these patients.

15.6 Left atrial appendage occlusion for stroke prevention

The most common justification for LAA occlusion devices in clinical practice is a perceived high bleeding risk and, less often, contraindications for OAC.[459] Unfortunately, LAA occluders have not been tested in such populations. Furthermore, LAA occluders have not been compared with NOAC therapy in patients at risk for bleeding, or with thoracoscopic LAA clipping. There is a clear need to conduct adequately designed and powered trials to define the clinical role of LAA occluders compared with NOAC therapy in patients with relative or absolute contraindications for anticoagulation, and/or in those suffering from an ischaemic stroke on anticoagulant therapy.

15.7 Anticoagulation in atrial fibrillation patients after a bleeding or stroke event

At least 2% of anticoagulated patients with AF will experience a serious bleeding event per year. Observational data suggest that OAC can be reinitiated even after an intracerebral bleeding event.[460,484] Controlled studies evaluating different anticoagulation and stroke prevention interventions are urgently needed to provide evidence on the best management of patients who have suffered a bleeding event that would usually lead to withholding OAC. Some studies (e.g. APACHE-AF [Apixaban versus Antiplatelet drugs or no antithrombotic drugs after anticoagulation-associated intraCerebral HaEmorrhage in patients with Atrial Fibrillation][1025]) are ongoing, but adequately powered trials are needed. Similarly, prospectively collected data are needed on the stroke prevention and bleeding risk following (re-)initiation of OAC after stroke or intracranial bleeding.

15.8 Anticoagulation and optimal timing of non-acute cardioversion

Based on retrospective data, previous recommendations on the safe time-window in which a cardioversion can be performed in new-onset AF used ≤48 h as the 'gold standard' for non-protected cardioversion. However, new evidence has emerged that initiating pre-cardioversion anticoagulation in patients with AF episodes of <24 h or even <12 h would provide even better

IK+E M

Shrnutí guidelines pro ty, pro které je 1 stránka doporučení maximum

17. A short summary of the management of atrial fibrillation patients

Here, we provide 17 simple rules to guide the diagnosis and management of AF patients according to the 2016 ESC Guidelines for the management of atrial fibrillation developed in cooperation with EACTS.

(1) Use ECG screening in at-risk populations for AF, especially stroke survivors and the elderly.
(2) Document AF by ECG before starting treatment.
(3) Evaluate all AF patients by clinical evaluation, ECG, and echocardiogram for underlying cardiovascular conditions such as hypertension, heart failure, valvular heart disease, and others.
(4) Provide tailored information and education to AF patients to empower them to support AF management.
(5) Propose lifestyle changes to all suitable AF patients to make their management more effective.
(6) Treat underlying cardiovascular conditions adequately, e.g. valve repair or replacement in AF patients with significant valvular heart disease, treatment of heart failure, or management of hypertension, among others.
(7) Use oral anticoagulation in all AF patients unless they are at low risk for stroke based on the CHA_2DS_2VASc score or have true contraindications for anticoagulant therapy.
(8) Anticoagulate patients with atrial flutter similar to AF. Offer isthmus ablation to symptomatic flutter patients.
(9) Reduce all modifiable bleeding risk factors in all AF patients on oral anticoagulation, e.g. by treating hypertension, minimizing the duration and intensity of concomitant antiplatelet and non-steroidal anti-inflammatory drug therapy, treating anaemia and eliminating causes for blood loss, maintaining stable INR values in patients on VKAs, and moderating alcohol intake.
(10) Check ventricular rate in all AF patients and use rate control medications to achieve lenient rate control.
(11) Evaluate AF-related symptoms in all AF patients using the modified EHRA symptoms scale. Whenever patients have AF-related symptoms, aim to improve symptoms by adjustment of rate control therapy and by offering antiarrhythmic drugs, cardioversion, or catheter or surgical ablation.
(12) Select antiarrhythmic drugs based on their safety profile and consider catheter or surgical ablation when antiarrhythmic drugs fail.
(13) Do not offer routine genetic testing in AF patients unless there is suspicion of an inherited cardiac condition.
(14) Do not use antiplatelet therapy for stroke prevention in AF.
(15) Do not permanently discontinue oral anticoagulation in AF patients at increased risk of stroke unless such a decision is taken by a multidisciplinary team.
(16) Do not use rhythm control therapy in asymptomatic AF patients, nor in patients with permanent AF.
(17) Do not perform cardioversion or catheter ablation without anticoagulation, unless an atrial thrombus has been ruled out transoesophageal echocardiogram.