RUNG NHĨ – ATRIAL FIBRILLATION

ĐỊNH NGHĨA

Rối loạn nhịp nhanh trên thất với sự hoạt hóa hoạt động điện tâm nhĩ không đồng bộ và tâm nhĩ co bóp không hiệu quả.

Điện tâm đồ: đo ít nhất 30s để chẩn đoán.

(1) Khoảng R-R không đều

(2) Không thấy sóng P.

(3) Hoạt hóa nhĩ không đều.

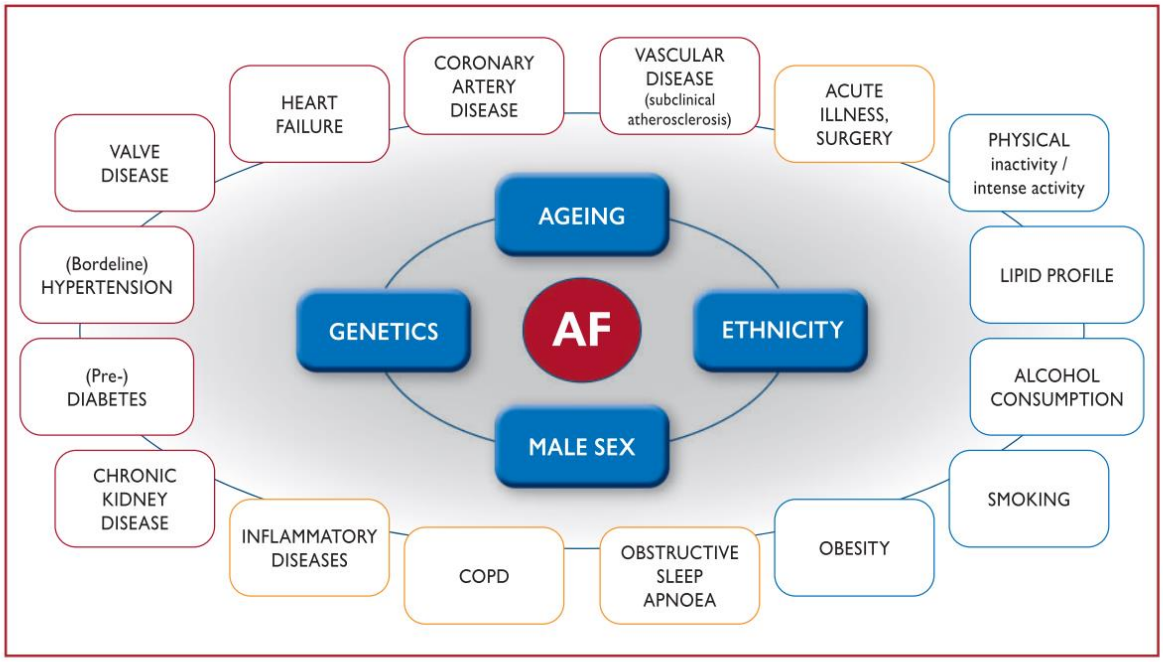

Figure 3 Summary of risk factors for incident AF[10,22,33,35–72] (*Supplementary Table 1* for full list). AF = atrial fibrillation; COPD = chronic obstructive pulmonary disease.

NGUYÊN NHÂN

Các nguyên nhân thường gặp là:

(1) Bệnh van tim

(2) Bệnh cơ tim thiếu máu cục bộ

(3) Suy tim

(4) Cường giáp

(5) Lớn tuổi

LÂM SÀNG

Triệu chứng của rối loạn nhịp: hồi hộp - đánh trống ngực, nặng ngực, mệt mỏi, chóng mặt, khó thở, lo lắng là 6 triệu chứng dùng để phân loại EHRA. Ngất.

Triệu chứng của nguyên nhân.

Triệu chứng biến chứng: tử vong, huyết khối tuần hoàn hệ thống, suy tim, phù phổi cấp…

PHÂN LOẠI

Hiện tại, AF do van và không do van đã không còn được định nghĩa. Rung nhĩ được phân loại: (1) lâm sàng có triệu chứng hoặc không có triệu chứng, (2) dưới lâm sàng (không biểu hiện trên ECG).

Rung nhĩ có thể chia theo thời gian chẩn đoán và khả năng chuyển lại nhịp xoang: rung nhĩ cơn, rung nhĩ kịch phát, rung nhĩ bền bỉ (persistent), rung nhĩ dai dẳng (long-standing), rung nhĩ vĩnh viễn (permanent).

Table 4 Classification of AF

AF pattern	Definition
First diagnosed	AF not diagnosed before, irrespective of its duration or the presence/severity of AF-related symptoms.
Paroxysmal	AF that terminates spontaneously or with intervention within 7 days of onset.
Persistent	AF that is continuously sustained beyond 7 days, including episodes terminated by cardioversion (drugs or electrical cardioversion) after ≥7 days
Long-standing persistent	Continuous AF of >12 months' duration when decided to adopt a rhythm control strategy.
Permanent	AF that is accepted by the patient and physician, and no further attempts to restore/maintain sinus rhythm will be undertaken. Permanent AF represents a therapeutic attitude of the patient and physician rather than an inherent pathophysiological attribute of AF, and the term should not be used in the context of a rhythm control strategy with antiarrhythmic drug therapy or AF ablation. Should a rhythm control strategy be adopted, the arrhythmia would be re-classified as 'long-standing persistent AF'.
Terminology that should be abandoned	
Lone AF	A historical descriptor. Increasing knowledge about the pathophysiology of AF shows that in every patient a cause is present. Hence, this term is potentially confusing and should be abandoned.[147]
Valvular/non-valvular AF	Differentiates patients with moderate/severe mitral stenosis and those with mechanical prosthetic heart valve(s) from other patients with AF, but may be confusing[148] and should not be used.
Chronic AF	Has variable definitions and should not be used to describe populations of AF patients.

AF = atrial fibrillation.

Supplementary Table 3 Clinical types of AF.[111]

AF type	Clinical presentation	Possible pathophysiology
AF secondary to structural heart disease	AF in patients with LV systolic or diastolic dysfunction, long-standing hypertension with LVH, and/or other structural heart disease. The onset of AF in these patients is a common cause of hospitalization and a predictor of poor outcome.	Increased atrial pressure and atrial structural remodelling, together with activation of the sympathetic and renin-angiotensin system.
Focal AF	Patients with repetitive atrial runs and frequent, short episodes of paroxysmal atrial fibrillation. Often highly symptomatic, younger patients with distinguishable atrial waves (coarse AF), atrial ectopy, and/or atrial tachycardia deteriorating in AF.	Localized triggers, in most cases originating from the pulmonary veins, initiate AF. AF due to one or a few re-entrant drivers is also considered to be part of this type of AF.
Polygenic AF	AF in carriers of common gene variants that have been associated with early onset AF.	Currently under study. The presence of selected gene variants may also influence treatment outcomes.
Post-operative AF	New onset of AF (usually self-terminating) after major (typically cardiac) surgery in patients who were in sinus rhythm before surgery and had no prior history of AF.	Acute factors: inflammation, atrial oxidative stress, high sympathetic tone, electrolyte changes, and volume overload, possibly interacting with a pre-existing substrate.
AF in patients with mitral stenosis or prosthetic heart valves	AF in patients with mitral stenosis, after mitral valve surgery and in some cases other valvular disease.	Left atrial pressure (stenosis) and volume (regurgitation) load are the main drivers of atrial enlargement and structural atrial remodelling in these patients.
AF in athletes	Usually paroxysmal, related to duration and intensity of training.	Increased vagal tone and atrial volume.
Monogenic AF	AF in patients with inherited cardiomyopathies, including channelopathies.	The arrhythmogenic mechanisms responsible for sudden death are likely to contribute to the occurrence of AF in these patients.

TIẾP CẬN CHẨN ĐOÁN VÀ ĐIỀU TRỊ

A – Anticoagulation/Avoid stoke, B – Better symptom control, C-cardiovascular optimatize

Đánh giá ban đầu: (1) Rung nhĩ đáp ứng thất …, (2) Nguyên nhân là gì? (3) Thời gian? (4) Đã có huyết khối/nguy cơ huyết khối của bệnh nhân? (5) Nguy cơ xuất huyết.

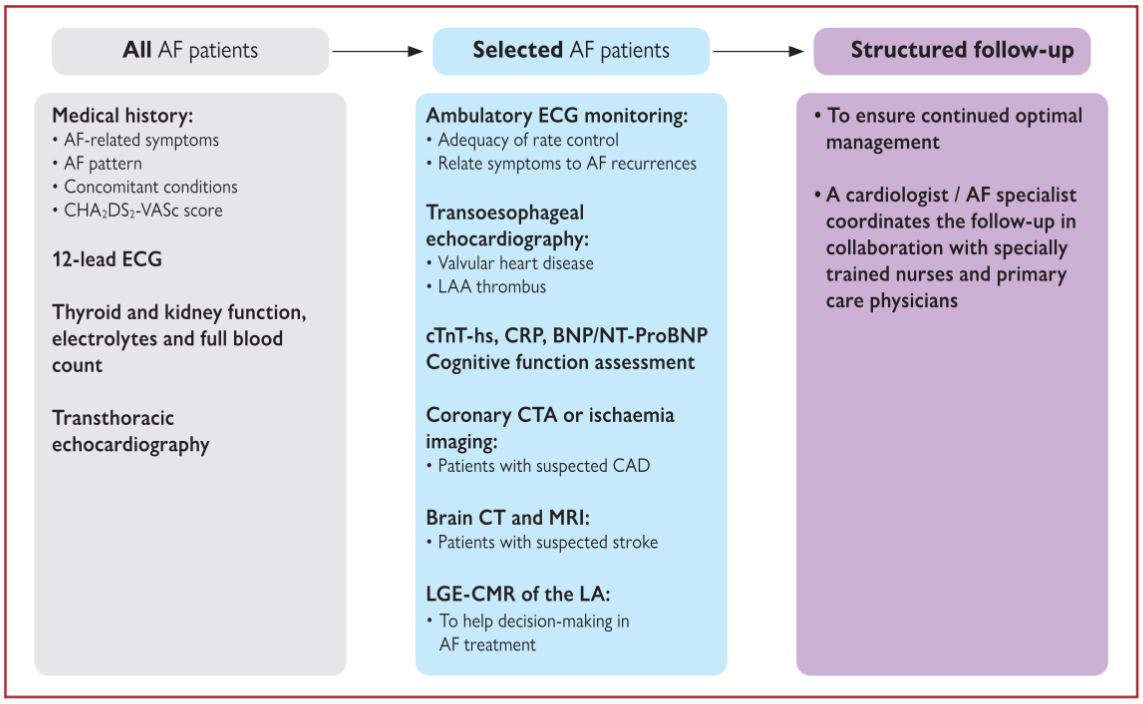

Figure 8 Diagnostic work-up and follow-up in AF patients. AF = atrial fibrillation; BNP = B-type natriuretic peptide; CHA_2DS_2-VASc = Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Stroke, Vascular disease, Age 65 - 74 years, Sex category (female); CAD = coronary artery disease; CRP = C-reactive protein; CT = computed tomography; CTA = computed tomography angiography; cTnT-hs = high-sensitivity cardiac troponin T; ECG = electrocardiogram; LAA = left atrial appendage; LGE-CMR = late gadolinium contrast-enhanced cardiac magnetic resonance; MRI = magnetic resonance imaging; NT-ProBNP = N-terminal (NT)-prohormone B-type natriuretic peptide.

Nguy cơ đột quỵ cao: CHA2DS2-VASc đánh ở bệnh nhân rung nhĩ không có van nhân tạo (cơ học, sinh học) hay hẹp van 2 lá trung bình đến nặng.

- CHA2DS2-VASc ≥ 2 ở nam, ≥ 3 ở nữ thì khuyến cáo dùng kháng đông đường uống: **NOACs hoặc VKA.**
- CHA2DS2-VASc ≥ 1 ở nam, ≥ 2 ở nữ thì cân nhắc sử dụng kháng đông.
- CHA2DS2-VASc < 1 ở nam, < 2 ở nữ thì không cần sử dụng.

HAS-BLED: Nguy cơ chảy máu lớn, nguy cơ cao khi ***HAS-BLED ≥ 3.*** Tuy nhiên, điểm số trên không phải là lý do tuyệt đối bắt buộc phải ngưng kháng đông nếu có chỉ định dùng.

Table 11 Dose selection criteria for NOACs

	Dabigatran	Rivaroxaban	Apixaban	Edoxaban
Standard dose	150 mg b.i.d.	20 mg o.d.	5 mg b.i.d.	60 mg o.d.
Lower dose	110 mg b.i.d.			
Reduced dose		15 mg o.d.	2.5 mg b.i.d.	30 mg o.d.
Dose-reduction criteria	Dabigatran 110 mg b.i.d. in patients with: • Age ≥80 years • Concomitant use of verapamil, or • Increased bleeding risk	CrCl 15 - 49 mL/min	At least 2 of 3 criteria: • Age ≥80 years, • Body weight ≤60 kg, or • Serum creatinine ≥1.5 mg/dL (133 µmol/L)	If any of the following: • CrCl 15 - 50 mL/min, • Body weight ≤60 kg, • Concomitant use of dronedarone, ciclosporine, erythromycin, or ketoconazole

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b.i.d. = bis in die (twice a day); CrCl = creatinine clearance; o.d. = *omni die* (once daily).

Apixaban ít gây chảy máu nhất, đặc biệt là ít gây tác dụng xuất huyết tiêu hóa.

Sử dụng bắt cầu kháng Vitamin K với Heparin khi có bằng chứng huyết khối, nếu không có huyết khối hiện hữu thì khởi động kháng Vitamin K đơn độc là đủ rồi. Vì nếu bệnh nhân đang có huyết khối, ức chế vitamin K sẽ gây hiện tượng tăng đông nghịch thường, do protein S và protein C giảm trước các yếu tố đông máu.

Table 8 **CHA_2DS_2-VASc score**[334]

CHA_2DS_2-VASc score Risk factors and definitions		Points awarded	Comment
C	**Congestive heart failure** Clinical HF, or objective evidence of moderate to severe LV dysfunction, or HCM	1	Recent decompensated HF irrespective of LVEF (thus incorporating HFrEF or HFpEF), or the presence (even if asymptomatic) of moderate-severe LV systolic impairment on cardiac imaging[335]; HCM confers a high stroke risk[336] and OAC is beneficial for stroke reduction.[337]
H	**Hypertension** or on antihypertensive therapy	1	History of hypertension may result in vascular changes that predispose to stroke, and a well-controlled BP today may not be well-controlled over time.[324] Uncontrolled BP - the optimal BP target associated with the lowest risk of ischaemic stroke, death, and other cardiovascular outcomes is 120 - 129/<80 mmHg.[338]
A	**Age 75 years or older**	2	Age is a powerful driver of stroke risk, and most population cohorts show that the risk rises from age 65 years upwards.[339] Age-related risk is a continuum, but for reasons of simplicity and practicality, 1 point is given for age 65 - 74 years and 2 points for age ≥75 years.
D	**Diabetes mellitus** Treatment with oral hypoglycaemic drugs and/or insulin or fasting blood glucose >125 mg/dL (7 mmol/L)	1	Diabetes mellitus is a well-established risk factor for stroke, and more recently stroke risk has been related to duration of diabetes mellitus (the longer the duration of diabetes mellitus, the higher the risk of thromboembolism[340]) and presence of diabetic target organ damage, e.g. retinopathy.[341] Both type 1 and type 2 diabetes mellitus confer broadly similar thromboembolic risk in AF, although the risk may be slightly higher in patients aged <65 years with type 2 diabetes mellitus compared to patients with type 1 diabetes mellitus.[342]
S	**Stroke**Previous stroke, TIA, or thromboembolism	2	Previous stroke, systemic embolism, or TIA confers a particularly high risk of ischaemic stroke, hence weighted 2 points. Although excluded from RCTs, AF patients with ICH (including haemorrhagic stroke) are at very high risk of subsequent ischaemic stroke, and recent observational studies suggest that such patients would benefit from oral anticoagulation.[343–345]
V	**Vascular disease** Angiographically significant CAD, previous myocardial infarction, PAD, or aortic plaque	1	Vascular disease (PAD or myocardial infarction) confers a 17 - 22% excess risk, particularly in Asian patients.[346–348] Angiographically significant CAD is also an independent risk factor for ischaemic stroke among AF patients (adjusted incidence rate ratio 1.29, 95% CI 1.08 - 1.53).[349] Complex aortic plaque on the descending aorta, as an indicator of significant vascular disease, is also a strong predictor of ischaemic stroke.[350]
A	**Age 65 – 74 years**	1	See above. Recent data from Asia suggest that the risk of stroke may rise from age 50 - 55 years upwards and that a modified CHA_2DS_2-VASc score may be used in Asian patients.[351,352]
Sc	**Sex category (female)**	1	A stroke risk modifier rather than a risk factor.[353]
Maximum score		**9**	

Mục tiêu INR

Khi sử dụng kháng vitamin K (wafarin, acenocoumarol) thì mục tiêu phụ thuộc vào loại van:

- Van cơ học: 2.5-3.5
- Rung nhĩ van tim bình thường hay van sinh học: INR = 2-3.
- Acenocumarol (Sintrom) 4 mg ½-1/4-1/8 viên (u).

Cần theo dõi INR hằng tháng, điều chỉnh theo sự thay đổi INR.

Cần tránh các loại thức ăn chứa nhiều vitamin K như rau củ quả có màu xanh đậm.

Table 10 **Clinical risk factors in the HAS-BLED score**[395]

Risk factors and definitions		Points awarded
H	**Uncontrolled hypertension** SBP >160 mmHg	1
A	**Abnormal renal and/or hepatic function** 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
S	**Stroke** Previous ischaemic or haemorrhagic[a] stroke	1
B	**Bleeding history or predisposition** Previous major haemorrhage or anaemia or severe thrombocytopenia	1
L	**Labile INR**[b] TTR <60% in patient receiving VKA	1
E	**Elderly** Aged >65 years or extreme frailty	1
D	**Drugs or excessive alcohol drinking** Concomitant use of antiplatelet or NSAID; and/or excessive[c] alcohol per week	1 point for each
Maximum score		**9**

ALP = alkaline phosphatase; ALT = alanine aminotransferase; AST = aspartate aminotransferase; SBP = systolic blood pressure; INR = international normalized ratio; NSAID = Non-steroidal anti-inflammatory drug; TTR = time in therapeutic range; VKA = vitamin K antagonist.
[a]Haemorrhagic stroke would also score 1 point under the 'B' criterion.
[b]Only relevant if patient receiving a VKA.
[c]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.

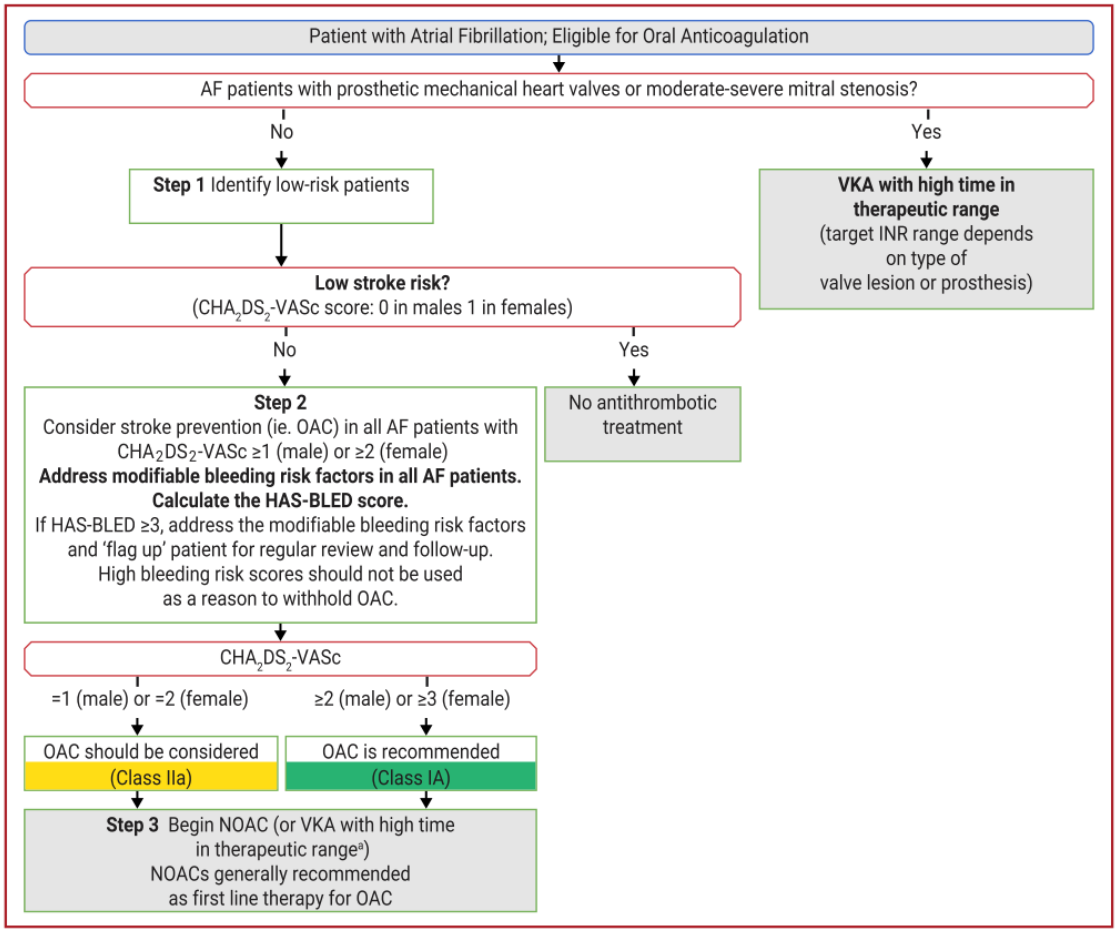


Figure 12 'A' - Anticoagulation/Avoid stroke: The 'AF 3-step' pathway. AF = atrial fibrillation; CHA_2DS_2-VASc = Congestive heart failure, Hypertension, Age >75 years, Diabetes mellitus, Stroke, Vascular disease, Age 65 - 74 years, Sex category (female); HAS-BLED = Hypertension, Abnormal

Better symptom controls

(1) Kiểm soát tần số tim

- Mục tiêu: < 80 lúc nghỉ (*tối ưu 60-65 lần/phút*), < 110 lúc vận động trung bình.

- Lựa chọn dựa vào: (1) bệnh nền (có suy tim?), (2) triệu chứng (có huyết khối chưa?), (3) tác dụng phụ của thuốc.
- **Các thuốc:**
  - ***First-line***: beta-blockers, tuy nhiên không dùng ở suy tim cấp và hen nặng.
    - Thuốc và liều tối đa cao hơn trong suy tim: Bisoprolol, carvedilol, Metoprolol, nebivolol.
  - ***NonDHP-CCB***: verapamil, diltiazem.
    - Chống chỉ định ở bệnh nhân suy tim phân suất tống máu giảm, vì tăng nguy cơ nhồi máu cơ tim, suy tim cấp, tử vong.
    - HF mà EF ≥ 40% thì vẫn sử dụng được.
  - ***Digoxin***: suy tim có rung nhĩ đáp ứng thất nhanh
  - ***Amiodarone:*** chọn lựa cuối cùng (***last resort***)
    - Chỉ được pha trong Glucose, không sử dụng Normal saline để pha.
- **Không dùng thuốc**: Đặt máy tạo nhịp tạm thời.
- Trong bệnh cảnh cấp tính, đặt biệt trong suy tim cấp, chức năng LV giảm nặng, amiodarone là sự lựa chọn.

Table 13 **Drugs for rate control in AF[a]**

	Intravenous administration	Usual oral maintenance dose	Contraindicated
Beta-blockers[b]			
Metoprolol tartrate	2.5 - 5 mg i.v. bolus; up to 4 doses	25 - 100 mg *b.i.d.*	In case of asthma use beta-1-blockers Contraindicated in acute HF and history of severe bronchospasm
Metoprolol XL (succinate)	N/A	50 - 400 mg *o.d.*	
Bisoprolol	N/A	1.25 - 20 mg *o.d.*	
Atenolol[c]	N/A	25 - 100 mg *o.d.*	
Esmolol	500 µg/kg i.v. bolus over 1 min; followed by 50 - 300 µg/kg/min	N/A	
Landiolol	100 µg/kg i.v. bolus over 1 min, followed by 10 - 40 µg/kg/min; in patients with cardiac dysfunction: 1 - 10 µg/kg/min	N/A	
Nebivolol	N/A	2.5 - 10 mg *o.d.*	
Carvedilol	N/A	3.125 - 50 mg *b.i.d.*	
Non-dihydropyridine calcium channel antagonists			
Verapamil	2.5 - 10 mg i.v. bolus over 5 min	40 mg b.i.d. to 480 mg (extended release) o.d.	Contraindicated in HFrEF Adapt doses in hepatic and renal impairment
Diltiazem	0.25 mg/kg i.v. bolus over 5 min, then 5 - 15 mg/h	60 mg t.i.d. to 360 mg (extended release) o.d.	
Digitalis glycosides			
Digoxin	0.5 mg i.v. bolus (0.75 - 1.5 mg over 24 hours in divided doses)	0.0625 - 0.25 mg o.d.	High plasma levels associated with increased mortality Check renal function before starting and adapt dose in CKD patients
Digitoxin	0.4 - 0.6 mg	0.05 - 0.1 mg o.d.	High plasma levels associated with increased mortality
Other			
Amiodarone	300 mg i.v. diluted in 250 mL 5% dextrose over 30 - 60 min (preferably via central venous cannula), followed by 900 - 1200 mg i.v. over 24 hours diluted in 500 - 1000 mL via a central venous cannula	200 mg o.d. after loading 3 × 200 mg daily over 4 weeks, then 200 mg daily[536] [d] (reduce other rate controlling drugs according to heart rate)	In case of thyroid disease, only if no other options

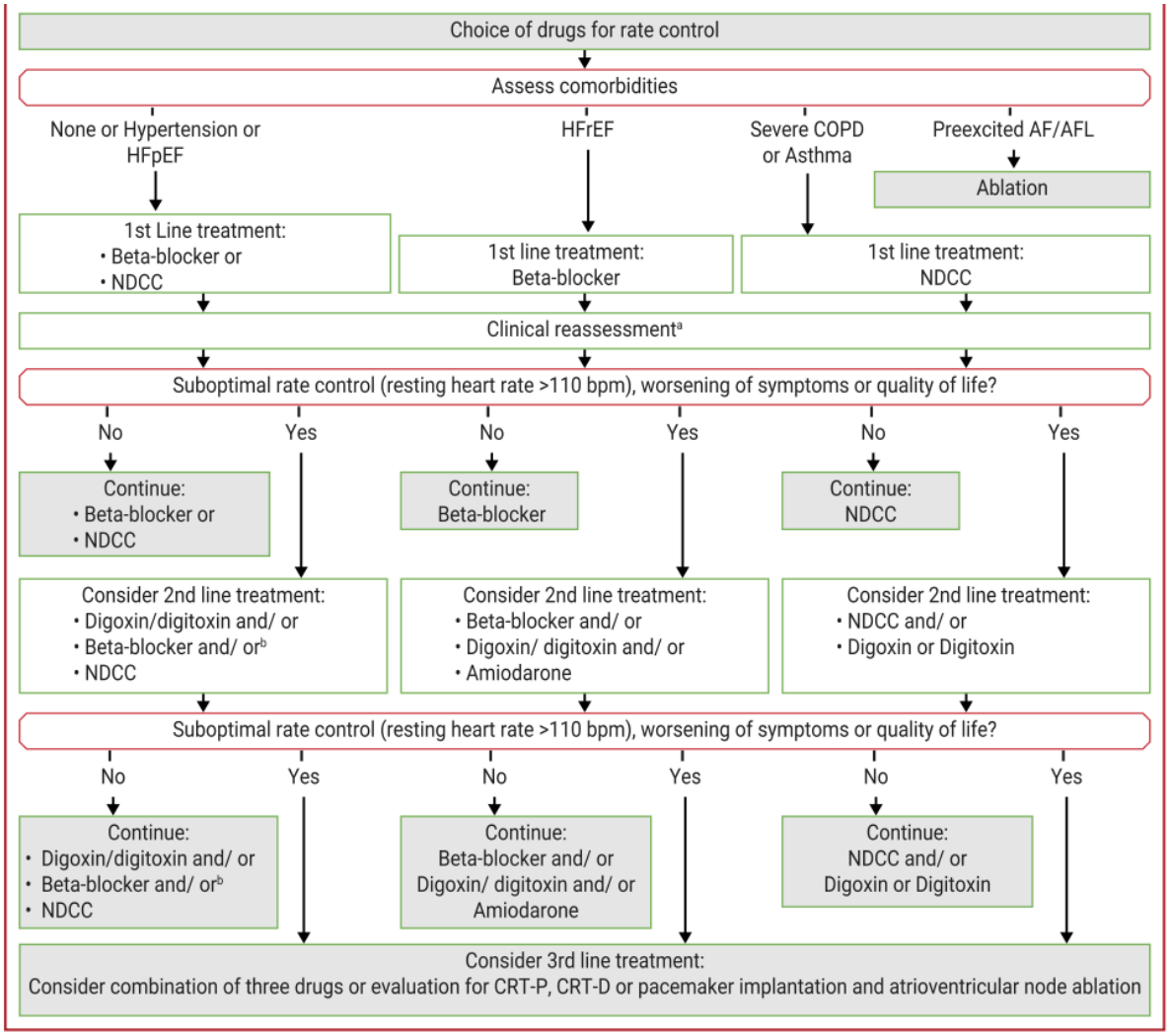


Figure 14 Choice of rate control drugs.[490] AF = atrial fibrillation; AFL = atrial flutter; COPD = chronic obstructive pulmonary disease; CRT-D = cardiac resynchronization therapy defibrillator; CRT-P = cardiac resynchronization therapy pacemaker; HFpEF = heart failure with preserved ejection fraction; HFrEF =

Recommendations for ventricular rate control in patients with AF[a]

Recommendations	Class[b]	Level[c]
Beta-blockers, diltiazem, or verapamil are recommended as first-choice drugs to control heart rate in AF patients with LVEF≥40%.[492,507,511,529]	I	B
Beta-blockers and/or digoxin are recommended to control heart rate in AF patients with LVEF<40%.[486,491,502,512,530–532]	I	B
Combination therapy comprising different rate controlling drugs[d] should be considered if a single drug does not achieve the target heart rate.[533,534]	IIa	B
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.[488]	IIa	B
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.[516,523,535,536]	IIa	B
In patients with haemodynamic instability or severely depressed LVEF, intravenous amiodarone may be considered for acute control of heart rate.[504,514,515]	IIb	B

AF = atrial fibrillation; bpm = beats per minute; ECG = electrocardiogram; LA = left atrial; LVEF = left ventricular ejection fraction.
[a]See *section 11* for ventricular rate control in various concomitant conditions and AF populations
[b]Class of recommendation.
[c]Level of evidence.
[d]Combining beta-blocker with verapamil or diltiazem should be performed with careful monitoring of heart rate by 24-h ECG to check for bradycardia.[488]

(2) Chuyển nhịp tim (Rhythm control)

Dựa vào: (1) thời gian xuất hiện rung nhĩ, (2) đã xuất hiện huyết khối chưa, (3) tính cấp bách của việc chuyển nhịp.

Lựa chọn: Chuyển nhịp bằng thuốc, hoặc không thuốc (sốc điện).

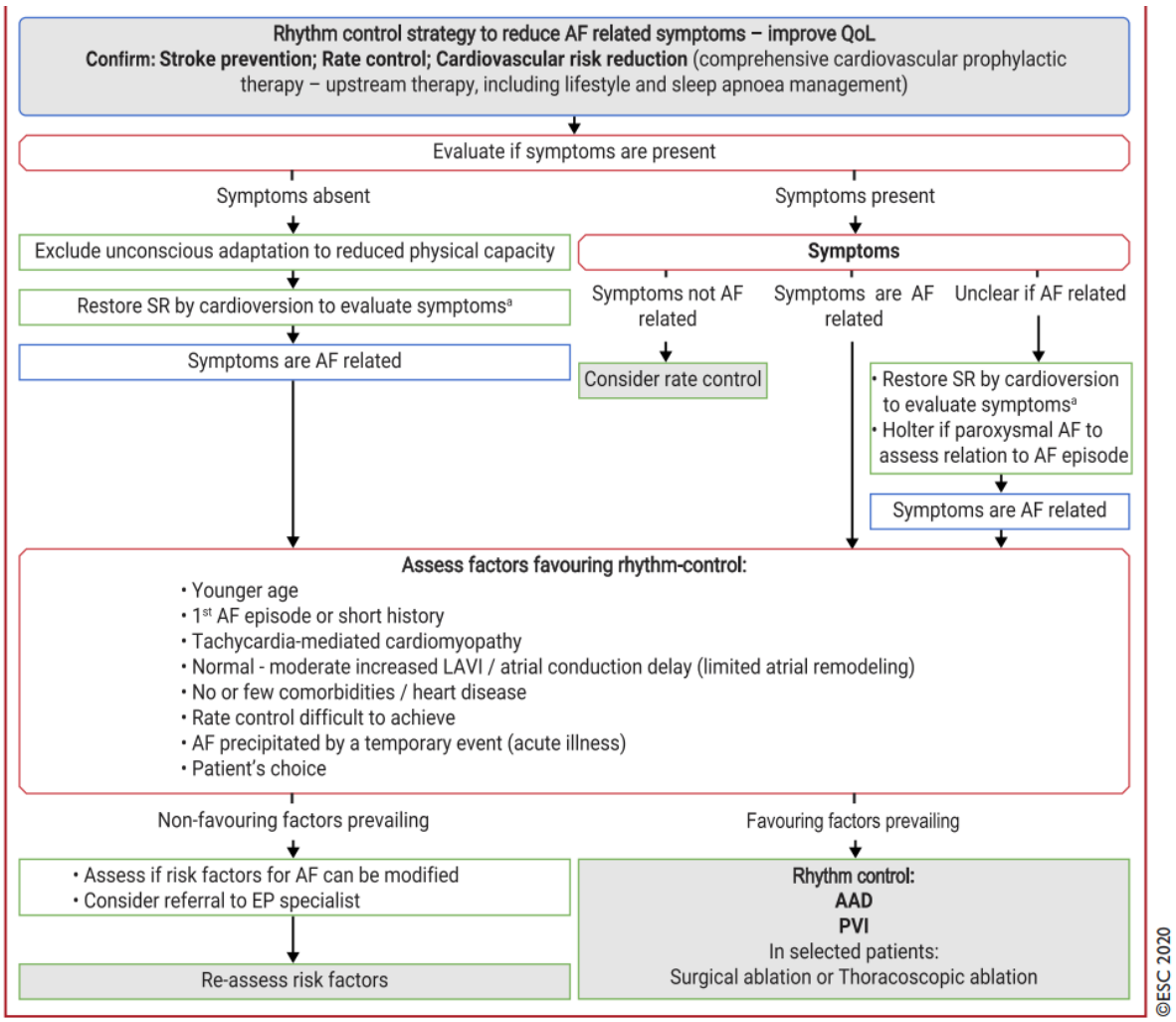


Figure 15 Rhythm control strategy. AAD = antiarrhythmic drug; AF = atrial fibrillation; CMP = cardiomyopathy; CV = cardioversion; LAVI = left atrial volume index; PAF = paroxysmal atrial fibrillation; PVI = pulmonary vein isolation; QoL = quality of life; SR = sinus rhythm. [a]Consider cardioversion to confirm that the absence of symptoms is not due to unconscious adaptation to reduced physical and/or mental capacity.

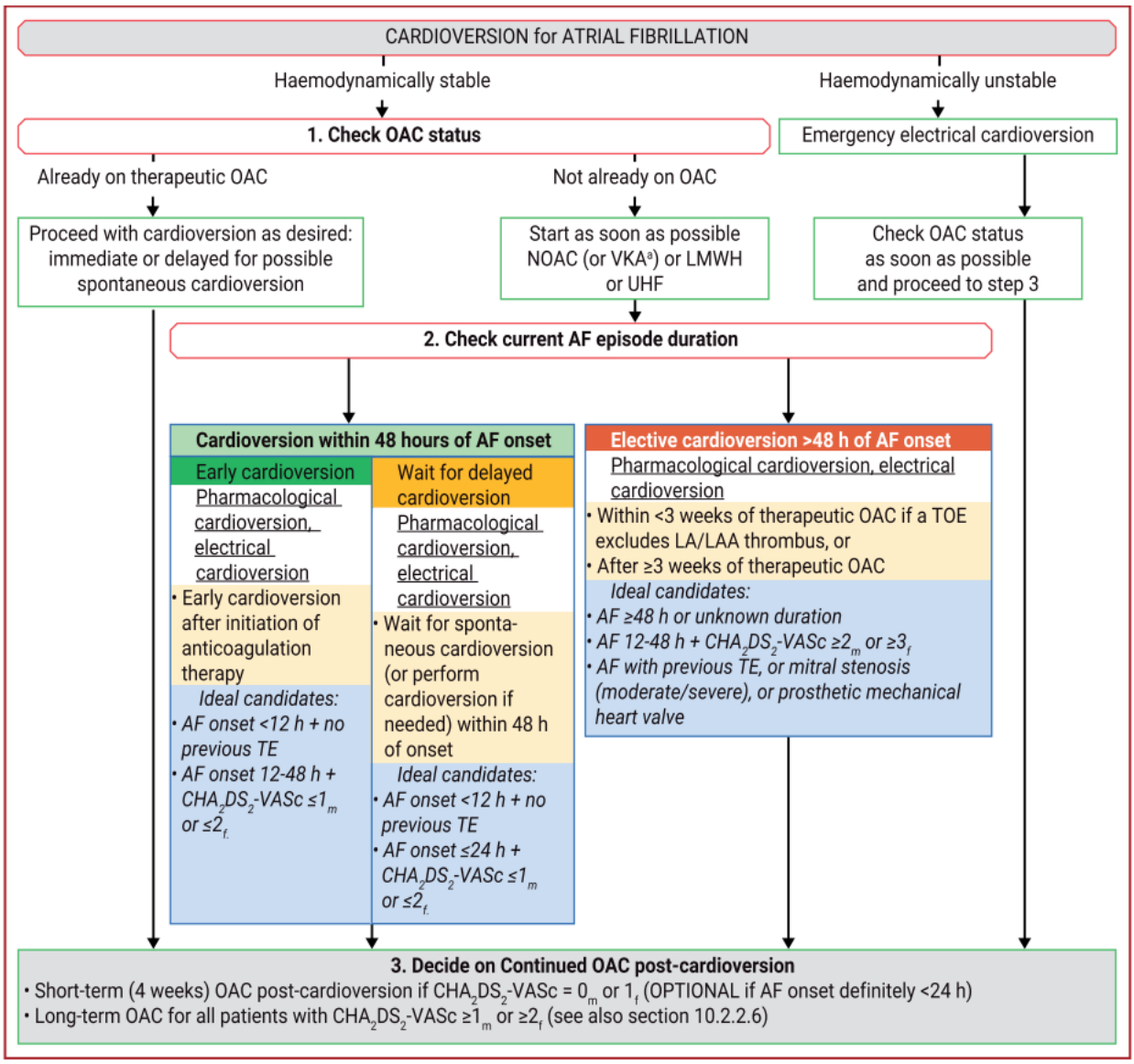


Figure 16 Flowchart for decision making on cardioversion of AF depending on clinical presentation, AF onset, oral anticoagulation intake, and risk factors

Thuốc chuyển nhịp có: Amiodarone, Ibutilide, Flecanide…

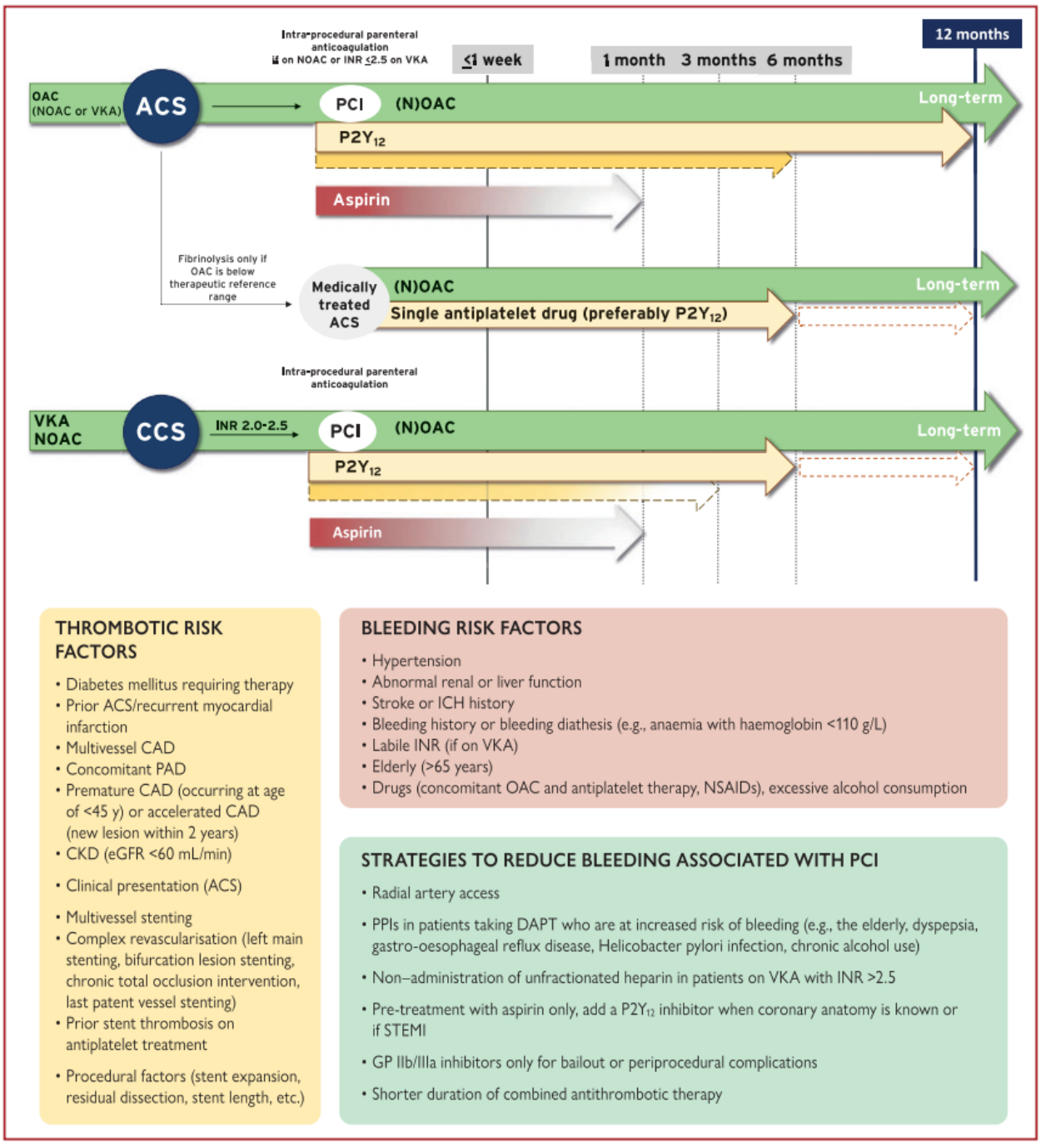
Tóm tắt:

(1) Chuyển nhịp tốt nhất là có bằng chứng khởi phát ***AF < 48h***, có thể không cần siêu âm tim và tiền căn không có thuyên tắc mạch (VN chắc khó).

(2) Nếu trên 48 giờ, cần sử dụng kháng đông ≥ 3 tuần, *hoặc* có thể chuyển nhịp trong vòng 3 tuần thì **phải có bằng chứng không có huyết khối buồng nhĩ/tiểu nhĩ trái bằng siêu âm tim qua ngả thực quản.**

(3) Sau khi chuyển nhịp, cần dùng kháng đông đường uống ≥ 4 tuần nếu CHA2DS2-VASc 0 ở nam, 1 ở nữ, còn nếu ≥ 1 ở nam và ≥ 2 ở nữ thì dùng OAC kéo dài.

Trường hợp ACS đã PCI: Ngưng Aspirin sau 1 tuần, dùng P2Y12 inhibitors phối hợp OAC kéo dài.



THROMBOTIC RISK FACTORS

- Diabetes mellitus requiring therapy
- Prior ACS/recurrent myocardial infarction
- Multivessel CAD
- Concomitant PAD
- Premature CAD (occurring at age of <45 y) or accelerated CAD (new lesion within 2 years)
- CKD (eGFR <60 mL/min)
- Clinical presentation (ACS)
- Multivessel stenting
- Complex revascularisation (left main stenting, bifurcation lesion stenting, chronic total occlusion intervention, last patent vessel stenting)
- Prior stent thrombosis on antiplatelet treatment
- Procedural factors (stent expansion, residual dissection, stent length, etc.)

BLEEDING RISK FACTORS

- Hypertension
- Abnormal renal or liver function
- Stroke or ICH history
- Bleeding history or bleeding diathesis (e.g., anaemia with haemoglobin <110 g/L)
- Labile INR (if on VKA)
- Elderly (>65 years)
- Drugs (concomitant OAC and antiplatelet therapy, NSAIDs), excessive alcohol consumption

STRATEGIES TO REDUCE BLEEDING ASSOCIATED WITH PCI

- Radial artery access
- PPIs in patients taking DAPT who are at increased risk of bleeding (e.g., the elderly, dyspepsia, gastro-oesophageal reflux disease, Helicobacter pylori infection, chronic alcohol use)
- Non–administration of unfractionated heparin in patients on VKA with INR >2.5
- Pre-treatment with aspirin only, add a $P2Y_{12}$ inhibitor when coronary anatomy is known or if STEMI
- GP IIb/IIIa inhibitors only for bailout or periprocedural complications
- Shorter duration of combined antithrombotic therapy

20 Post-procedural management of patients with AF and ACS/PCI (full-outlined arrows represent a default strategy; graded/dashe