Update in the Management of Atrial Fibrillation in the Emergency Department

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No Conflicts of Interest

Objectives

- To review the initial management of atrial fibrillation in acute setting
- Assessment for hemodynamic instability
- Indications for urgent cardioversion

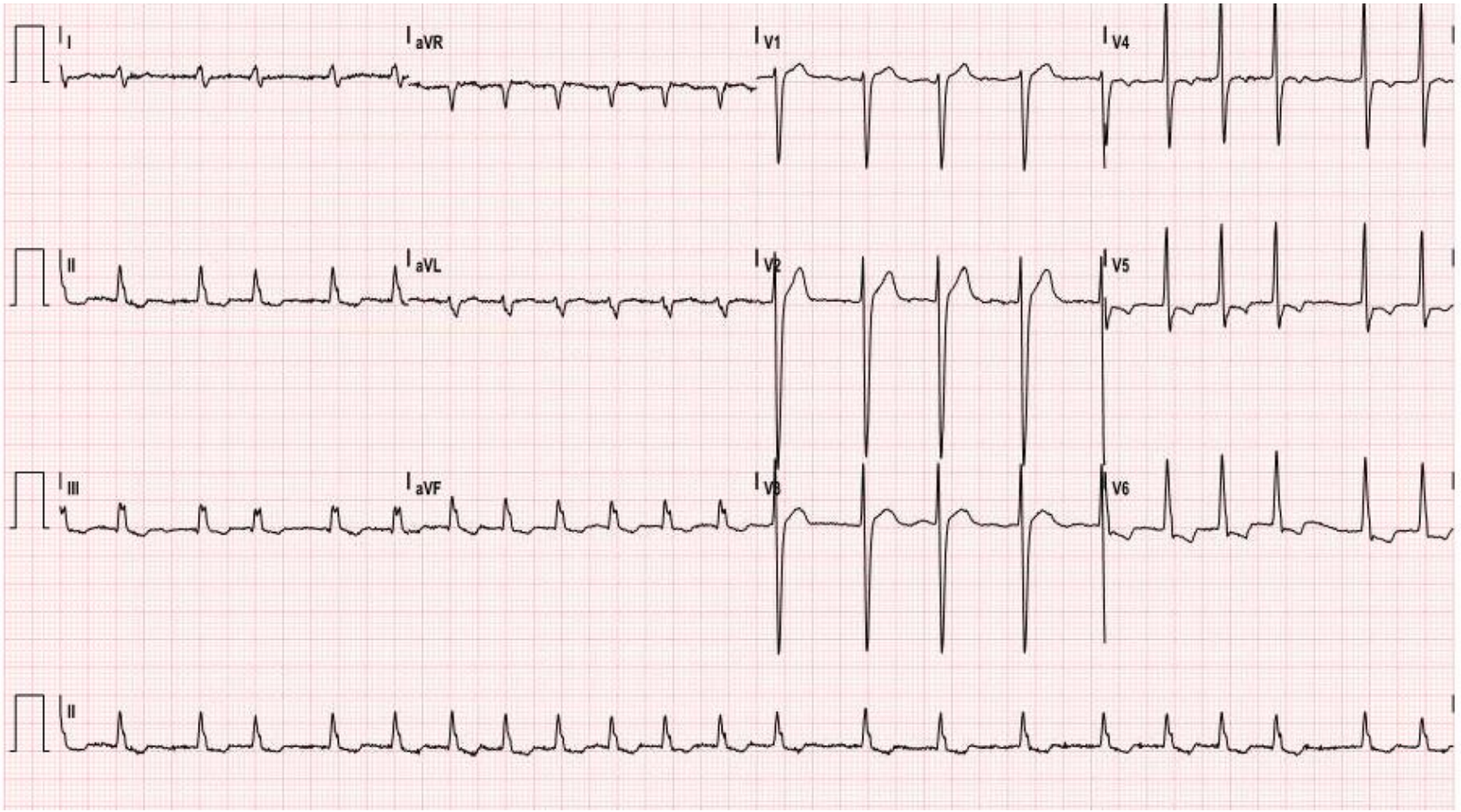
Clinical Case

- A 75 year old woman with PMHx of HTN, HLD and DM, CKD presents to ED for new onset dizziness, shortness of breath and palpitations that began 3 hours ago while patient was gardening in her lawn. She denies any associated chest pain and no actual loss of consciousness.

- Vital Signs: T: 37.5 C, BP 90s/60s (Baseline BP 115/80s), HR 140s-160s bpm and RR 24. A&O x3 with some facial grimmace. Cardiac exam is irregulary irregular without murmurs. Lungs CTAB. Remainder of exam unremarkable.

- She received a 2L bolus in the ED without increase in blood pressure

EKG



What is the next appropriate management for this patient?

- A) IV diltiazem
- B) Intubation
- C) Urgent Cardioversion
- D) IV pain control
- E) CT pulmonary angiogram

Definition

- **Atrial fibrillation (also called AFib or AF) is a quivering or irregular heartbeat (arrhythmia) that can lead to blood clots, stroke, heart failure and other heart-related complications.**

Here's how patients have described their experience:

- *"My heart flip-flops, skips beats, and feels like it's banging against my chest wall, especially if I'm carrying stuff up my stairs or bending down."*
- *"I was nauseated, light-headed, and weak. I had a really fast heartbeat and felt like I was gasping for air."*
- *"I had no symptoms at all. I discovered my AF at a regular check-up. I'm glad we found it early."*

2016 ESC Guidelines for the management of atrial fibrillation

European Heart Journal - doi:10.1093/eurheartj/ehw210

EUROPEAN SOCIETY OF CARDIOLOGY®

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 Organisation (ESO)

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General Considerations

- AF=most common sustained arrhytmia
- AF Prevalence=1.5 to 2% general population.
- AF+HF=increase mortality (Olson JACC 2006, Mamas EJHF 2009)

Epidemiology

- USA=2.7 to 6.1 M in 2010/2050=x by 2.5.
- Five fold increase of stroke.
- 3 fold increase of HF.
- 1.5 to 2 fold increase risk of death.
- Increase with age=+5.9% over 65 Y, +9% over 80 Y
- Framingham Study: **21% newly AF+HF** (Wang et al. Circulation 203)

Epidemiology

- In patients with preserved EF, AF is a predictor of HF (Sherazi Cardiol J 2011).
- ED visits for AF increased by 88% in 10 years=1% of all visits (McDonald et al. Ann Emerg Med 2008).
- 800000 to 1,000000 acute HF are managed by EPs (Fanarow C et al. Rev Cardiovasc Med 2003.)

Conditions predisposing to, or encouraging progression of AF

- Hypertension
- Symptomatic heart failure (NYHA II - IV) including tachycardiomyopathy
- Valvular heart disease
- Cardiomyopathies including primary electrical cardiac disease
- Atrial septal defect and other congenital heart defects
- Coronary artery disease
- Thyroid dysfunction and possibly subclinical thyroid dysfunction
- Obesity
- Diabetes mellitus
- Chronic obstructive pulmonary disease (COPD) and sleep apnoea
- Chronic renal disease

Consequences of AF

Thromboembolism
- **Stroke: 4.5× ↑risk**
- **Microemboli: ↓cognitive function**
- **Prothrombotic state**

Hospitalizations
- **Most common arrhythmia requiring hospitalization**
- **2-3× ↑risk for hospitalization**

- ↓*Quality of life*
- Palpitations, dyspnea, fatigue, ↓exercise tolerance

Impaired hemodynamics
- Loss of atrial kick
- Irregular ventricular contractions
- Heart failure
- Tachycardia-induced cardiomyopathy

- AF is an enormous contributor to the *growing cost* of medical care
- Estimated US cost burden: 15.7 billion

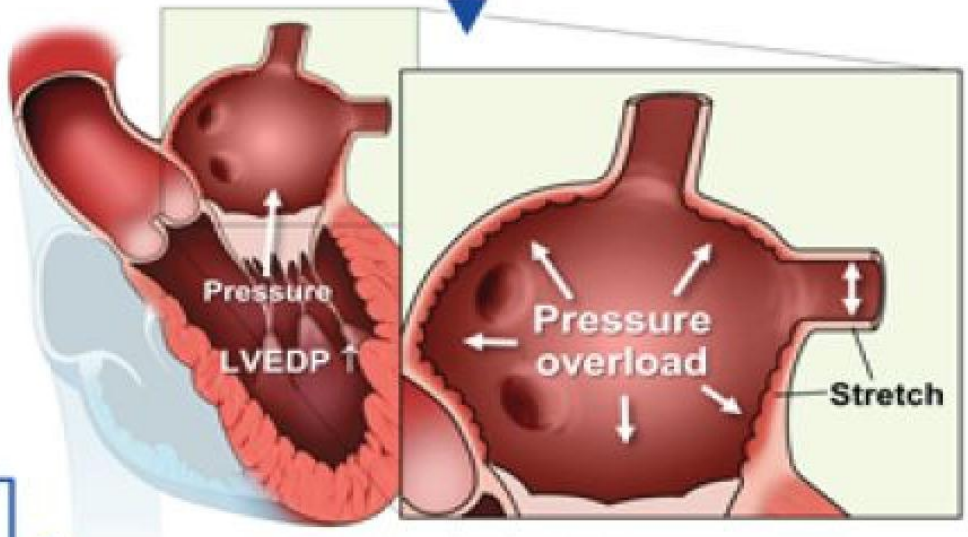
Pathogenesis of Atrial Fibrillation

Genetic predisposition

Predisposing factors
- Advanced age
- Hypertension
- Obesity
- Metabolic syndrome
- LV hypertrophy
- Left atrial enlargement
- Diastolic dysfunction

Predisposing factors
- Inflammation
- Oxidative stress
- Arterial stiffness
- ↑Vascular resistance
- ↓LV compliance

Apoptosis, ↑ fibrosis



- Δ connexins
- zig-zag conduction
- Electrical heterogeneity

Structural substrate

Electrophysiological substrate
Stretch-activated channels
Multiple reentry wavelet
Pulmonary vein focal/discharges
Dispersion of atrial refractoriness

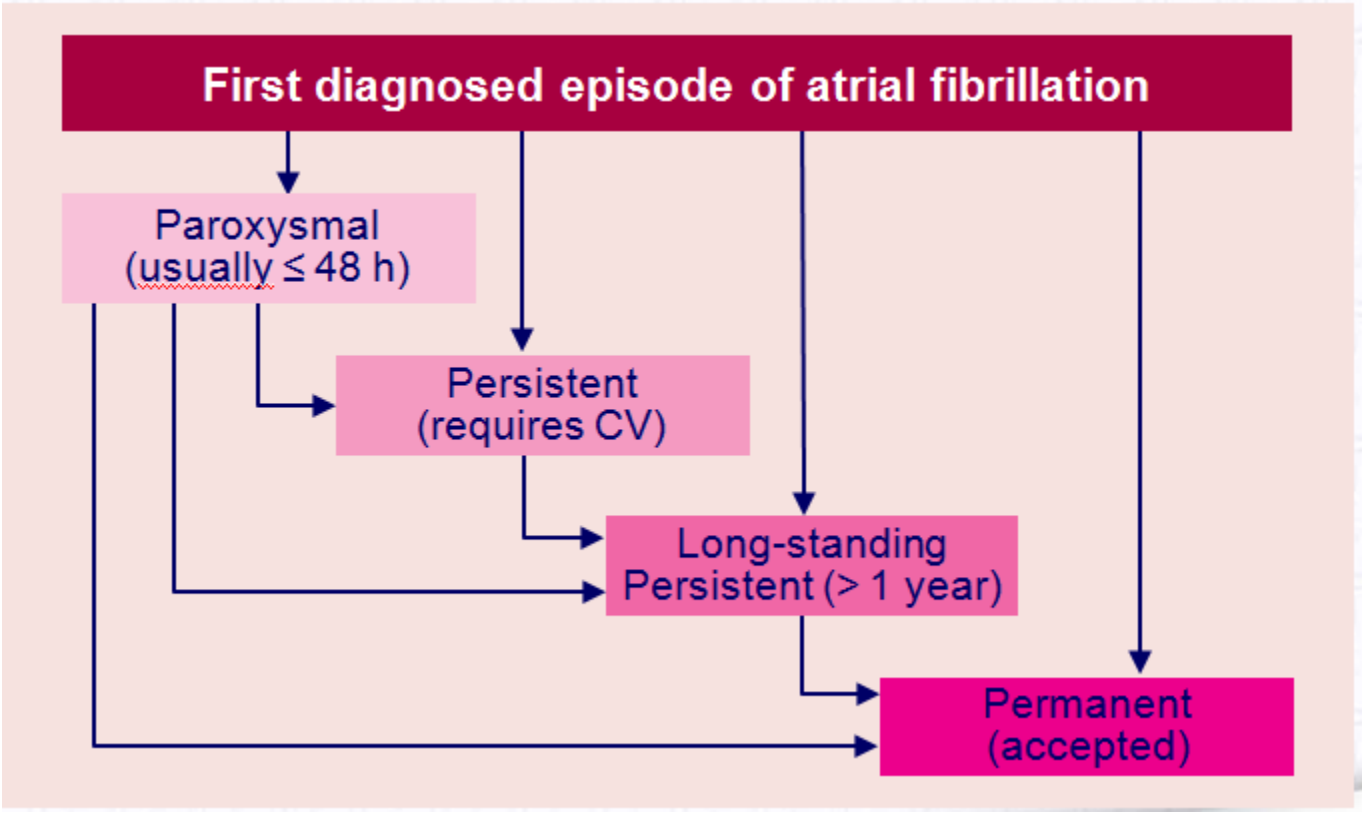
Triggers
- Atrial premature contractions
- Imbalance of autonomic nervous system
- Electrolyte imbalance
- Acute volume changes

Increased vulnerability

Triggers
- Atrial injury
- Atrial ischemia
- Volume overload
- Increased afterload
- Hemodynamic instability
- Sympathetic stimulation

AF

Types of Atrial Fibrillation



Initial Work-Up for patients AF in the ED

- A=Ensure a patent and protected airway
- B=Evaluate for adequate ventilation and listen for signs of pulmonary edema or precipitating pneumonia
- C=Determine hemodynamic stability and decide rate vs rythm control treatment
- D=Defibrillator pads anterior post chest for unstable patients
- E=ECG and evaluate thromboembolic risk and need of anticoagulation (CHA2DS2-VASc)

DIAGNOSTIC WORKUP

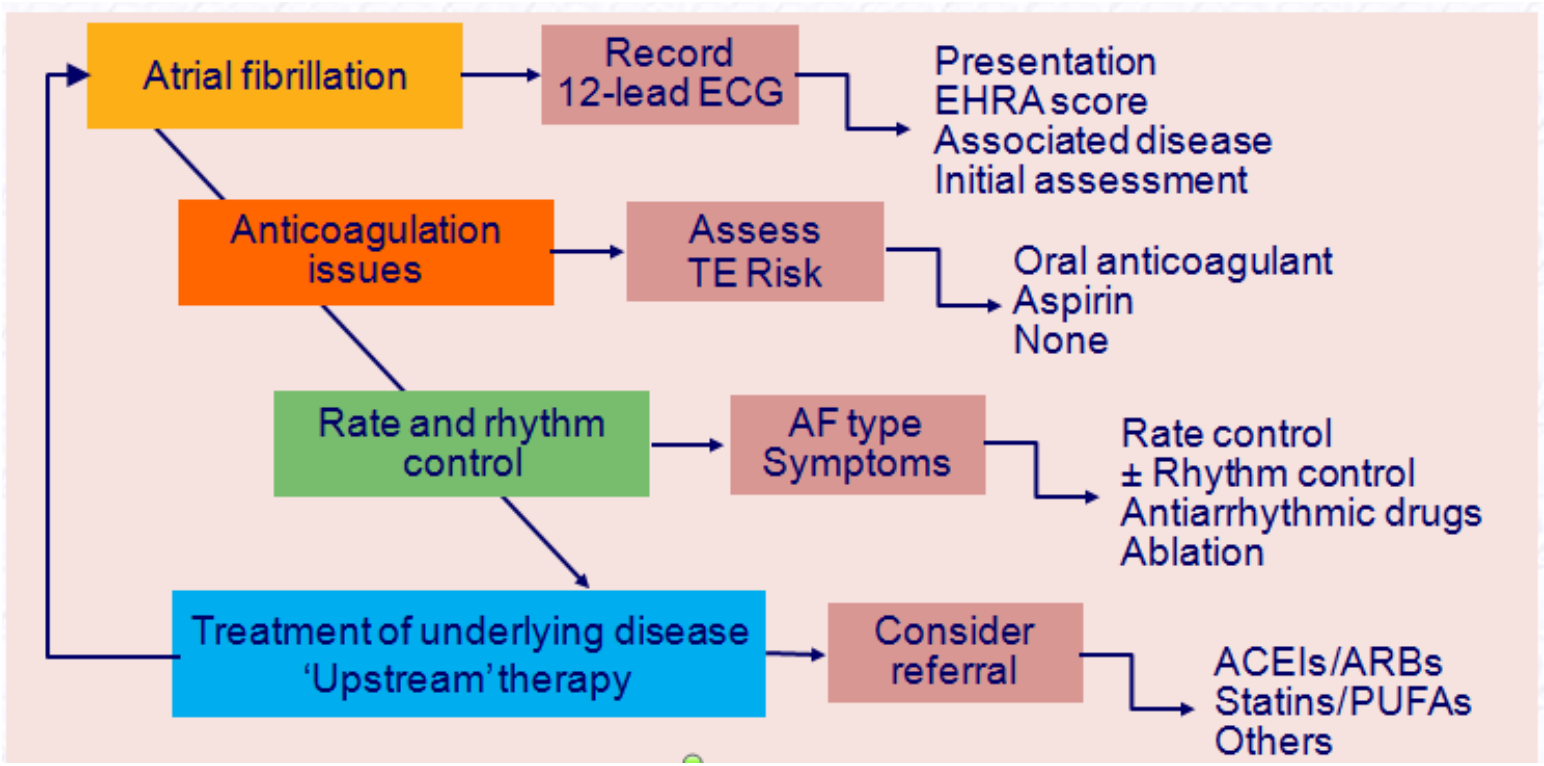
Identify Causes and Risk Factors

- **Minimum Evaluation**
- History and physical – BP, CV dz, Sleep Apnea
- Electrocardiogram – WPW, LVH, MI
- Echocardiogram – LVH, LAE, EF, Valve Dz
- Labs – TSH, Renal fxn, K+, Na+, Troponin…

Definition of Unstability in AF Patients

- Unstable angina
- Altered mental status
- Shortness of breath
- Focal neurologic deficit
- Signs of hypoperfusion
- **Heart failure**

The management cascade for patients with AF



ACEI = angiotensin-converting enzyme inhibitor; AF = atrial fibrillation; ARB = angiotensin receptor blocker; PUFA = polyunsaturated fatty acid; TE = thrombo-embolism.

Modified European Heart Rhythm Association (EHRA) symptom scale

Recommendations	Class	Level
Use of the modified EHRA symptom scale is recommended in clinical practice and research studies to quantify AF-related symptoms.	I	C

Modified EHRA score	Symptoms	Description
1	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 patient troubled by symptoms.
3	Severe	Normal daily activity affected by symptoms related to AF.
4	Disabling	Normal daily activity discontinued.

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Risk factor-based point-based scoring system - CHA_2DS_2-VASc

Risk factor	Score
Congestive heart failure/LV dysfunction	1
Hypertension	1
Age ≥ 75 ans	2
Diabetes mellitus	1
Stroke/TIA/thrombo-embolism	2
Vascular disease*	1
Age 65-74	1
Sex category [i.e. femal sex]	1
Maximum score	**9**

*Prior myocardial infarction, peripheral artery disease, aortic plaque. Actual rates of stroke in contemporary cohorts may vary from these estimates.

Prediction of stroke and bleeding risk

Recommendations	Class	Level
The CHA_2DS_2-VASc score is recommended for stroke risk prediction in patients with AF.	I	A
Bleeding risk scores should be considered in AF patients on oral anticoagulation to identify modifiable risk factors for major bleeding.	IIa	B
Biomarkers such as high-sensitivity troponin and natriuretic peptide may be considered to further refine stroke and bleeding risk in AF patients.	IIb	B

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CHA2DS2-VASc	Stroke rate %/year
0	0%
1	1.3%
2	2.2%
3	3.2%
4	4.0%
5	6.7%
6	9.8%
7	9.6%
8	6.7%
9	15.2%

Anticoagulation, CHA_2DS_2VASc Score, and Thromboembolic Risk of Cardioversion of Acute Atrial Fibrillation (from the FinCV Study)

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The efficacy of the anticoagulation in preventing thromboembolic complications (TEC) and the usefulness of the CHA_2DS_2VASc score for assessing stroke risk during cardioversion of acute atrial fibrillation (AF) are unclear. Thus, our objectives were to assess the ability of the CHA_2DS_2VASc score to predict TEC and to evaluate the efficacy of anticoagulation in the prevention of TEC in Finnish CardioVersion (FinCV) study. The FinCV is a retrospective, multicenter study of 3,143 patients, who underwent 7,660 cardioversions for acute AF. The value of the CHA_2DS_2VASc score in predicting TEC was analyzed separately in cardioversions performed without and with anticoagulation. A total of 40 definite TEC (0.6%) occurred after 7,237 successful cardioversions and 1 stroke (0.2%) after 423 unsuccessful procedures. In 5,362 cardioversions performed without anticoagulation, the risk of definite TEC increased significantly from 0.4% in patients with a CHA_2DS_2VASc score of 0 to 1 to 2.3% in those with score of ≥5 ($p < 0.001$ for trend). The C-statistic of the CHA_2DS_2VASc score was 0.72 (0.61 to 0.83) in predicting definite TEC in non-anticoagulated patients with first cardioversion. The incidence of definite TEC was significantly lower in 2,298 cardioversions performed during anticoagulation (0.1% vs 0.7%, $p = 0.001$), and the preventive effect of anticoagulation was significant in patients with a score of ≥2 (0.2% vs 1.1%, $p = 0.001$). In conclusion, CHA_2DS_2VASc score is a strong predictor of TEC in cardioversion of acute AF performed without anticoagulation. Importantly, periprocedural anticoagulation reduced the risk of TEC by 82%. The overall risk of these complications was low after failed cardioversion. (Am J Cardiol 2016;117:1294–1298)

CLINICAL RESEARCH STUDY

THE AMERICAN JOURNAL of MEDICINE®

Non-Vitamin K Antagonist Oral Anticoagulants for Cardioversion in Atrial Fibrillation: An Updated Meta-analysis

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ABSTRACT

BACKGROUND: Non-vitamin K oral anticoagulants are now proven alternatives to vitamin K antagonists for stroke prevention in atrial fibrillation. However, there are few data on the efficacy and safety of their use for cardioversion, in which the risk of thromboembolic events is heightened.

METHODS: We performed a random-effects meta-analysis of patients undergoing both electrical and pharmacologic cardioversion for atrial fibrillation in the RE-LY, ROCKET-AF, ARISTOTLE, ENGAGE AF-TIMI 48, X-VeRT, and ENSURE-AF trials. We assessed Mantel-Haenszel pooled estimates of risk ratios (RRs) and 95% confidence intervals (CIs) for stroke/systemic embolism and major bleeding at ≤42 days of follow-up.

RESULTS: The analysis pooled 6148 patients in whom 6854 cardioversions for atrial fibrillation were performed. Compared with vitamin K antagonists, non-vitamin K antagonist oral anticoagulant therapy was associated with a similar risk of stroke/systemic embolism (RR, 0.82; 95% CI, 0.38-1.75) and major bleeding (RR, 0.98; 95% CI, 0.51-1.87). We found no significant statistical heterogeneity among studies (Cochrane Q $P = .75$, $I^2 = 0\%$ for stroke/systemic embolism; $P = .54$; $I^2 = 0\%$ for major bleeding).

CONCLUSIONS: The short-term incidence of thromboembolism and major bleeding after cardioversion on non-vitamin K antagonist oral anticoagulants was comparable to the incidence observed on dose-adjusted vitamin K antagonist therapy. Non-vitamin K antagonist oral anticoagulants are a reasonable alternative to vitamin K antagonists in patients undergoing cardioversion.

STROKE/SE

Study or Subgroup	NOACs Events	NOACs Total	WARFARIN Events	WARFARIN Total	Weight	Risk Ratio M-H, Random, 95% CI
ARISTOTLE	0	331	0	412		Not estimable
ENGAGE-AF	2	251	0	114	6.4%	2.28 [0.11, 47.15]
ENSURE-AF	3	1095	4	1104	26.1%	0.76 [0.17, 3.37]
RE-LY	7	1319	4	664	38.9%	0.88 [0.26, 3.00]
ROCKET-AF	2	138	1	132	10.2%	1.91 [0.18, 20.85]
X-VeRT	2	1002	3	502	18.3%	0.33 [0.06, 1.99]
Total (95% CI)		**4136**		**2928**	**100.0%**	**0.82 [0.38, 1.75]**
Total events	16		12			

Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 1.92$, df = 4 (P = 0.75); $I^2 = 0\%$
Test for overall effect: Z = 0.52 (P = 0.60)

Risk Ratio M-H, Random, 95% CI

0.02 0.1 1 10 50

Favors NOACs Favors VKAs

MAJOR BLEEDING

Study or Subgroup	NOACs Events	NOACs Total	VKAs Events	VKAs Total	Weight	Risk Ratio M-H, Fixed, 95% CI
ARISTOTLE	1	331	1	412	4.7%	1.24 [0.08, 19.82]
ENGAGE-AF	0	251	0	114		Not estimable
ENSURE-AF	3	1067	5	1082	26.1%	0.61 [0.15, 2.54]
RE-LY	15	1319	4	664	27.9%	1.89 [0.63, 5.67]
ROCKET-AF	0	138	2	132	13.4%	0.19 [0.01, 3.95]
Total (95% CI)		**4094**		**2903**	**100.0%**	**0.98 [0.51, 1.87]**
Total events	25		16			

Heterogeneity: $Chi^2 = 3.10$, df = 4 (P = 0.54); $I^2 = 0\%$
Test for overall effect: Z = 0.06 (P = 0.95)

Risk Ratio M-H, Fixed, 95% CI

0.01 0.1 1 10 100

Favors NOACs Favors VKAs

Stroke prevention in patients with atrial fibrillation (1)

Recommendations	Class	Level
Oral anticoagulation therapy to prevent thromboembolism is recommended for all male AF patients with a CHA_2DS_2-VASc score of 2 or more.	I	A
Oral anticoagulation therapy to prevent thromboembolism is recommended in all female AF patients with a CHA_2DS_2-VASc score of 3 or more.	I	A
Oral anticoagulation therapy to prevent thromboembolism should be considered in male AF patients with a CHA_2DS_2-VASc score of 1, considering individual characteristics and patient preferences.	IIa	B
Oral anticoagulation therapy to prevent thromboembolism should be considered in female AF patients with a CHA_2DS_2-VASc score of 2, considering individual characteristics and patient preferences.	IIa	B
Vitamin K antagonist therapy (INR 2.0–3.0 or higher) is recommended for stroke prevention in AF patients with moderate-to-severe mitral stenosis or mechanical heart valves.	I	B
When oral anticoagulation is initiated in a patient with AF who is eligible for a NOAC (apixaban, dabigatran, edoxaban, or rivaroxaban), a NOAC is recommended in preference to a Vitamin K antagonist.	I	A

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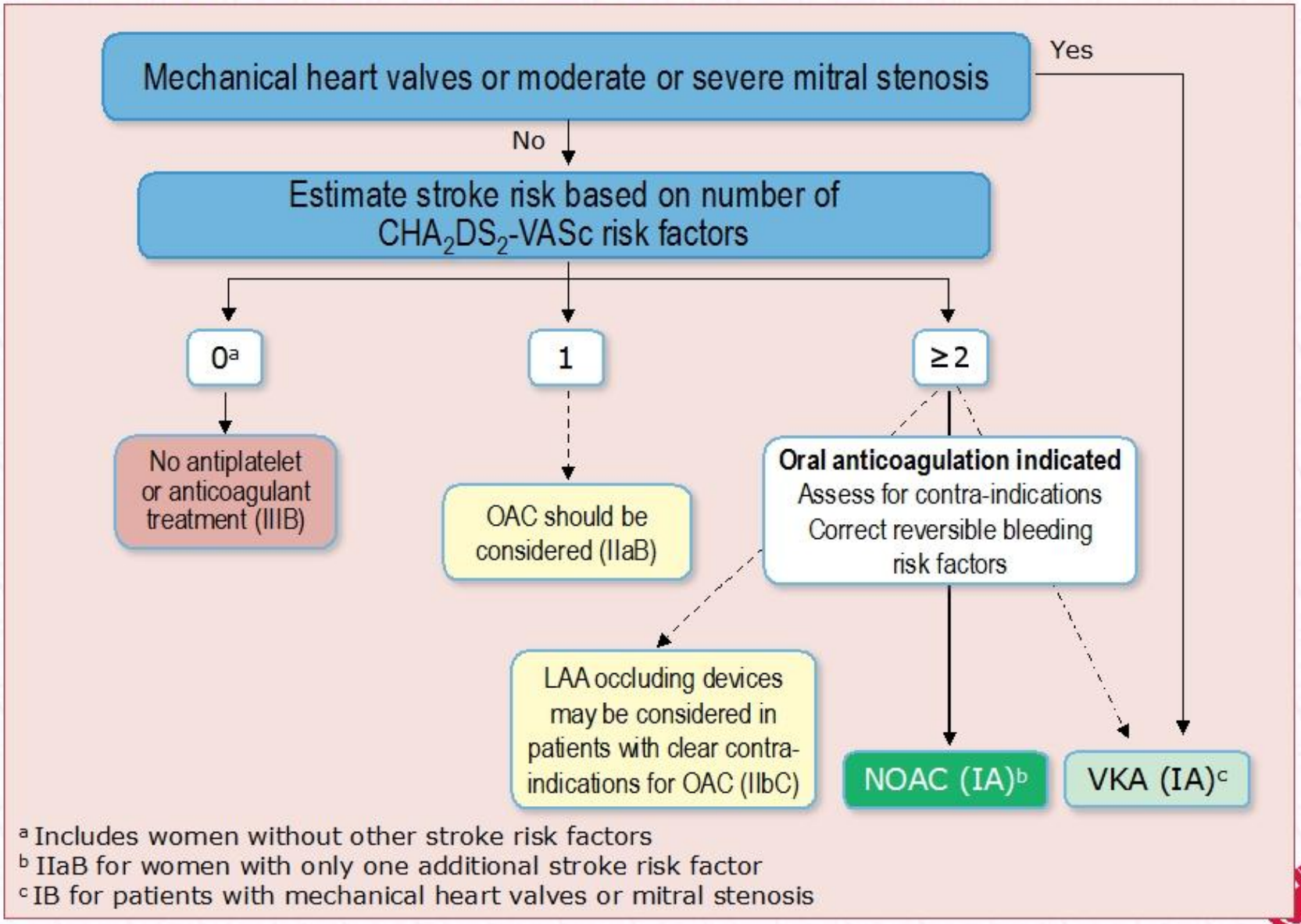
Stroke prevention in atrial fibrillation



[a] Includes women without other stroke risk factors
[b] IIaB for women with only one additional stroke risk factor
[c] IB for patients with mechanical heart valves or mitral stenosis

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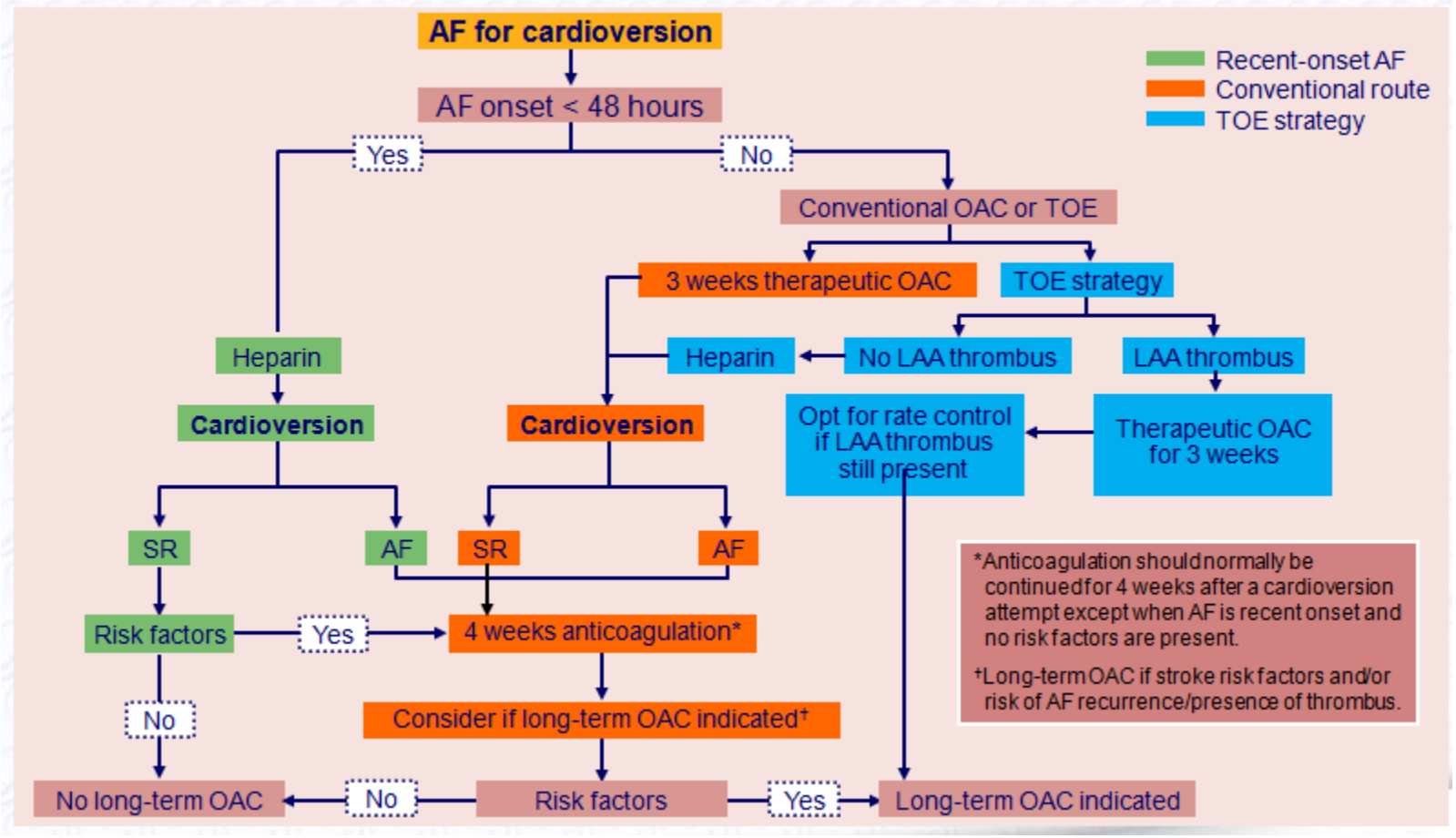
Cardioversion, TOE and anticoagulation



AF = atrial fibrillation; DCC = direct current cardioversion; LA = left atrium; LAA = left atrial appendage; OAC = oral anticoagulant; SR= sinus rhythm; TOE= transoesophageal echocardiography.

The HAS-BLED bleeding risk score

Letter	Clinical characteristic*	Points awarded
H	Hypertension	1
A	Abnormal renal and liver function (1 point each)	1 or 2
S	Stroke	1
B	Bleeding	1
L	Labile INRs	1
E	Elderly (e.g. age > 65 years)	1
D	Drugs or alcohol (1 point each)	1 or 2
		Maximum 9 points

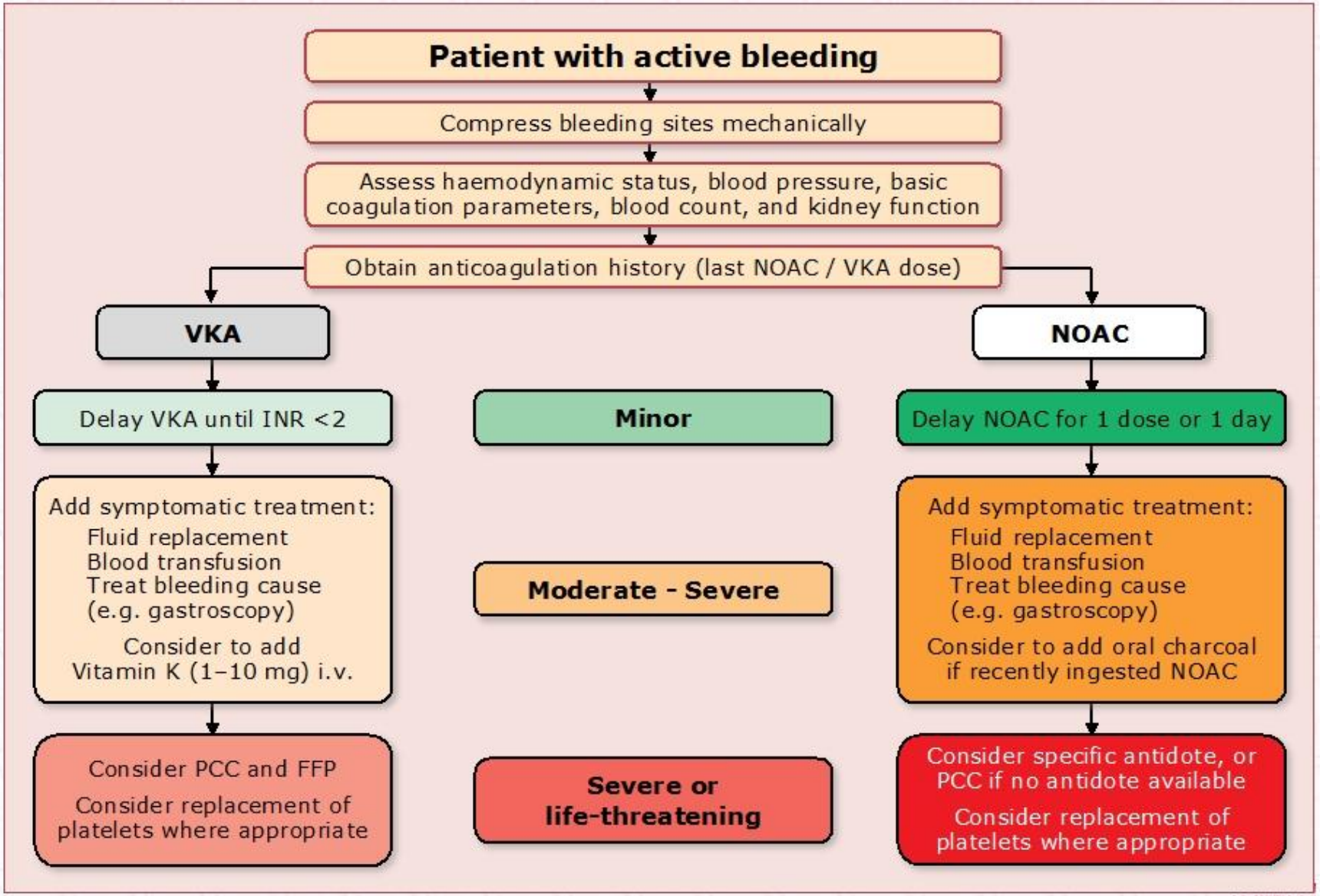
*Hypertension is defined as systolic blood pressure > 160 mmHg.

INR = international normalized ratio.

HAS-BLED

	Clinically Relevant Bleeding	Major Bleeding
0	7%	1%
1	8%	1%
2	11%	2%
3	16%	3%
4	15%	3%
≥5	38%	8%

Management of bleeding in anticoagulated AF patients



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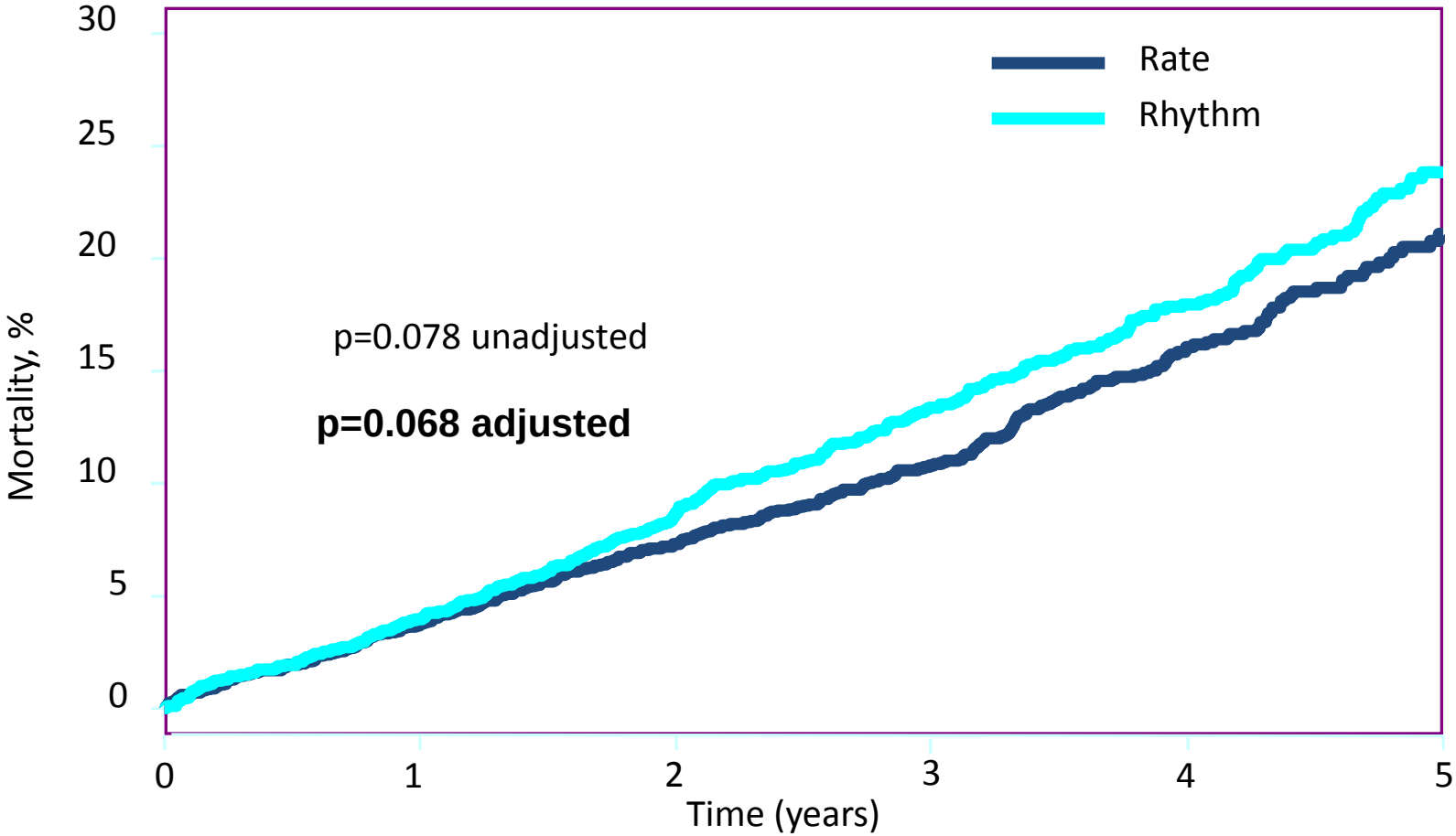
Indications for Urgent Direct Cardioversion

- Hemodynamic Instability:
  - Patient with decompensated heart failure
  - Active ischemia: if symptomatic with angina or evidence of ischemia/infarction on EKG
  - Evidence of organ hypoperfusion (altered mental status, cold clammy skin, acute kidney injury)

If Patient is Hemodynamically Stable

- Goal is ventricular rate control (<100 bpm) and anticoagulation
  - Resting HR goal should be 60-85 bpm in symptomatic patient
- Roughly 50% of patients with new onset AF will spontaneously convert to NSR spontaneously within 48 hours of onset
- Rate control or Rhythm control?
  - AFFIRM trial and RACE trial
    - No survival advantage in terms of stroke prevention rhythm control over rate control
- Rate control agents
  - Calcium Channel Blockers
  - Beta blockers (caution in patients with reactive airway disease)
  - Digoxin
  - Amiodarone (for patients intolerant or unresponsive to other agents)

AFFIRM: All-Cause Mortality



Rhythm N:	2033	1932	1807	1316	780	255
Rate N:	2027	1925	1825	1328	774	236

The AFFIRM Investigators. *N Engl J Med.* 2002;347:1825-1833.

TABLE 3. ADVERSE EVENTS.*

EVENT	OVERALL (N=4060)	RATE-CONTROL GROUP (N=2027)	RHYTHM-CONTROL GROUP (N=2033)	P VALUE
		no. of patients (%)		
Primary end point (death)	666 (26.3)	310 (25.9)	356 (26.7)	0.08†
Secondary end point (composite of death, disabling stroke, disabling anoxic encephalopathy, major bleeding, and cardiac arrest)	861 (32.3)	416 (32.7)	445 (32.0)	0.33
Torsade de pointes	14 (0.5)	2 (0.2)‡	12 (0.8)	0.007
Sustained ventricular tachycardia	15 (0.6)	9 (0.7)	6 (0.6)	0.44
Cardiac arrest followed by resuscitation				
Ventricular fibrillation or ventricular tachycardia	19 (0.6)	10 (0.7)	9 (0.5)	0.83
Pulseless electrical activity, bradycardia, or other rhythm	10 (0.3)	1 (<0.1)	9 (0.6)	0.01
Central nervous system event				
Total	211 (8.2)	105 (7.4)	106 (8.9)	0.93
Ischemic stroke§	157 (6.3)	77 (5.5)	80 (7.1)	0.79
After discontinuation of warfarin	69	25	44	
During warfarin but with INR <2.0	44	27	17	
Concurrent atrial fibrillation	67	42	25	
Primary intracerebral hemorrhage	34 (1.2)	18 (1.1)	16 (1.3)	0.73
Subdural or subarachnoid hemorrhage	24 (0.8)	11 (0.8)	13 (0.8)	0.68
Disabling anoxic encephalopathy	9 (0.3)	4 (0.2)	5 (0.4)	0.74
Myocardial infarction	140 (5.5)	67 (4.9)	73 (6.1)	0.60
Hemorrhage not involving the central nervous system	203 (7.3)	107 (7.7)	96 (6.9)	0.44
Systemic embolism	16 (0.5)	9 (0.5)	7 (0.4)	0.62
Pulmonary embolism	8 (0.3)	2 (0.1)	6 (0.5)	0.16
Hospitalization after base line	2594 (76.6)	1220 (73.0)	1374 (80.1)	<0.001

*Percentages were derived from a Kaplan–Meier analysis. P values were derived from the log-rank statistic.

†The P value in the case of death was based on the square root of the log-rank statistic, adjusted for 10 interim monitoring analyses.

‡One patient had crossed over to the rhythm-control group and was taking quinidine, and one patient had torsade de pointes 72 hours after mitral-valve replacement.

§Information on warfarin therapy was missing for two patients in the rate-control group and three patients in the rhythm-control group. Information on the presence of atrial fibrillation with the event was missing for 16 patients in the rate-control group and 13 patients in the rhythm-control group.

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Rhythm Control Versus Rate Control and Clinical Outcomes in Patients With Atrial Fibrillation

Results From the ORBIT-AF Registry

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ABSTRACT

OBJECTIVES The study sought to evaluate clinical outcomes in clinical practice with rhythm control versus rate control strategy for management of atrial fibrillation (AF).

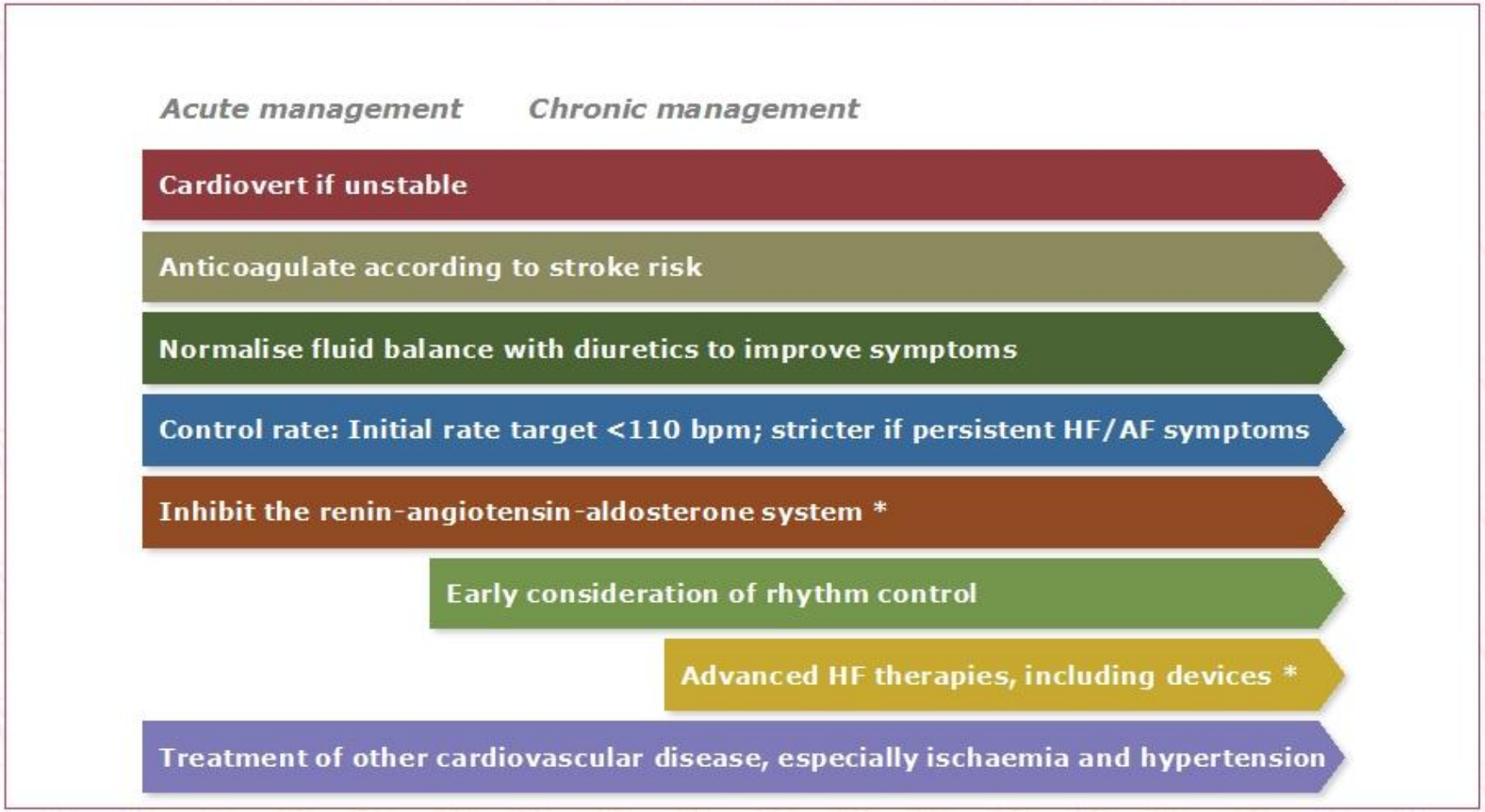
BACKGROUND Randomized trials have not demonstrated significant differences in stroke, heart failure, or mortality between rhythm and rate control strategies. The comparative outcomes in contemporary clinical practice are not well described.

METHODS Patients managed with a rhythm control strategy targeting maintenance of sinus rhythm were retrospectively compared with a strategy of rate control alone in a AF registry across various U.S. practice settings. Unadjusted and adjusted (inverse-propensity weighted) outcomes were estimated.

RESULTS The overall study population (N = 6,988) had a median of 74 (65 to 81) years of age, 56% were males, 77% had first detected or paroxysmal AF, and 68% had $CHADS_2$ score ≥2. In unadjusted analyses, rhythm control was associated with lower all-cause death, cardiovascular death, first stroke/non-central nervous system systemic embolization/transient ischemic attack, or first major bleeding event (all $p < 0.05$); no difference in new onset heart failure ($p = 0.28$); and more frequent cardiovascular hospitalizations ($p = 0.0006$). There was no difference in the incidence of pacemaker, defibrillator, or cardiac resynchronization device implantations ($p = 0.99$). In adjusted analyses, there were no statistical differences in clinical outcomes between rhythm control and rate control treated patients (all $p > 0.05$); however, rhythm control was associated with more cardiovascular hospitalizations (hazard ratio: 1.24; 95% confidence interval: 1.10 to 1.39; $p = 0.0003$).

CONCLUSIONS Among patients with AF, rhythm control was not superior to rate control strategy for outcomes of stroke, heart failure, or mortality, but was associated with more cardiovascular hospitalizations.
(J Am Coll Cardiol EP 2016;2:221–9) © 2016 by the American College of Cardiology Foundation.

Initial management of patients presenting acutely with atrial fibrillation and heart failure



* In patients with heart failure and reduced ejection fraction.

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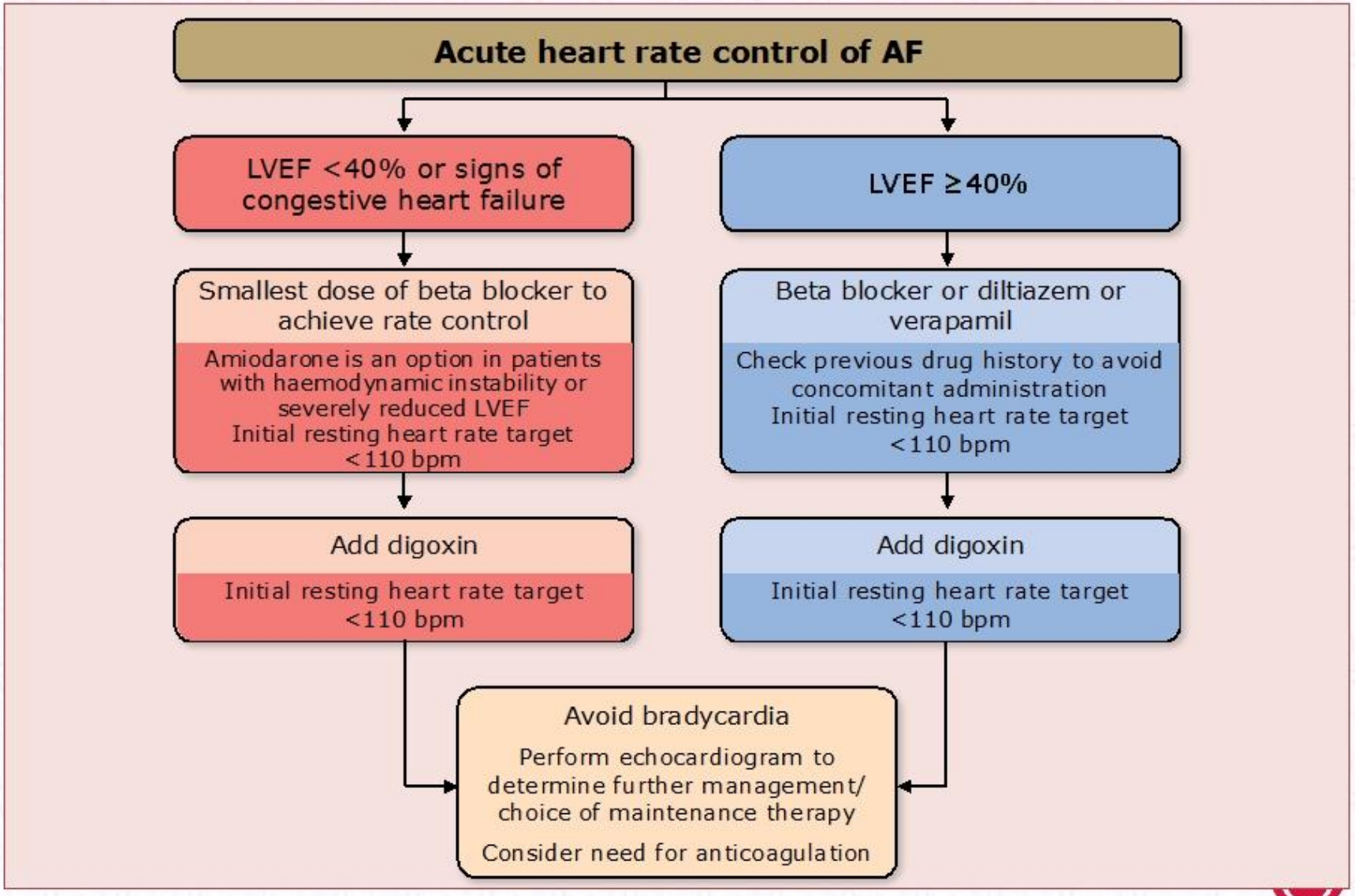
Drugs for rate control

	Intravenous administration	Usual oral maintenance dose
β-Blockers		
Metoprolol CR/XL	2.5–5 mg iv bolus over 2 min; up to 3 doses	100–200 mg o.d. (ER)
Bisoprolol	N/A	2.5–10 mg o.d.
Atenolol	N/A	25–100 mg o.d.
Esmolol	50–200 μg/kg/min iv	N/A
Propranolol	0.15 mg/kg iv over 1 min	10–40 mg t.i.d.
Carvedilol	N/A	3.125–25 mg b.i.d.
Non-dihydropyridine calcium channel antagonists		
Verapamil	0.0375–0.15 mg/kg iv over 2 min	40 mg b.i.d. to 360 mg (ER) o.d.
Diltiazem	N/A	60 mg t.i.d. to 360 mg (ER) o.d.
Digitalis glycosides		
Digoxin	0.5–1 mg	0.125 mg–0.5 mg o.d.
Digitoxin	0.4–0.6 mg	0.05 mg–0.1 mg o.d.
Others		
Amiodarone	5 mg/kg in 1 h, and 50 mg/h maintenance	100 mg–200 mg o.d.
Dronedarone[a]	N/A	400 mg b.i.d.

ER = extended release formulations; N/A = not applicable. [‡]Only in patients with non-permanent atrial fibrillation.

Acute heart rate control in atrial fibrillation



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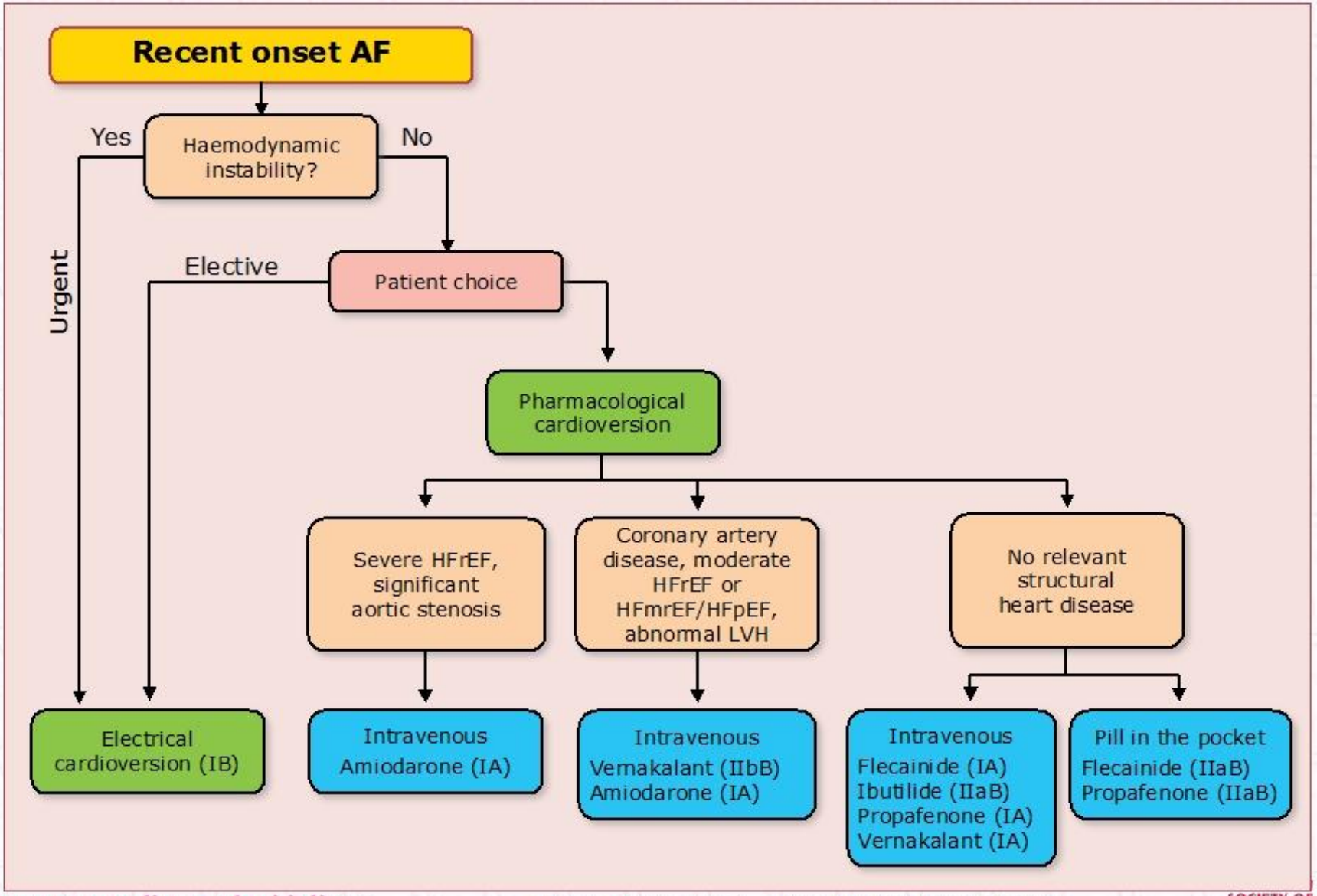
Urgent Cardioversion

- Electrical Cardioversion: sedate patient and place setting on direct synchronization then shock
  - Initial shock setting of 100J→ 200J→ 300J→ 360J until sinus rhythm returns
- Make sure you perform direct cardioversion with R wave synchronization to prevent an “R on T” phenomenon which can lead to V fib
- Restoration of normal sinus rhythm takes precedence over need for protection from thromboembolic risk
- Would recommend cardiology consult at this time

Cardioversion of recent onset of atrial fibrillation



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Drugs and doses for pharmacological conversion of (recent-onset) AF

Drug	Dose	Follow-up dose	Risks
Amiodarone	5 mg/kg i.v. over 1 h	50 mg/h	Phlebitis, hypotention. Will slow the ventricular rate. Delayed AF conversion to sinus rhythm.
Flecainide	2 mg/kg i.v. over 10 min, or 200-300 mg p.o.	N/A	Not suitable for patients with market structural heart disease; may prolong QRS duration, and hence the QT interval; and may inadvertently increase the ventricular rate due to conversion to atrial flutter and 1:1 conduction to the ventricles.
Ibutilide	1 mg i.v. over 10 min	1 mg i.v. over 10 min after waiting for 10 min	Can cause prolongation of the QT interval and torsades de pointes; watch for abnormal T-U waves or QT prolongation. Will slow the ventricular rate.
Propafenone	2 mg/kg i.v. over 10 min, or 450-600 mg p.o.		Not suitable for patients with market structural heart disease; may prolong QRS duration; will slightly slow the ventricular rate, but may inadvertently increase the ventricular rate due to conversion to atrial flutter and 1;1 conduction to the ventricules.
Vernakalant	3 mg/kg i.v. over 10 min	Second infusion of 2 mg/kg i.v. over 10 min after 15 min rest	So far only evaluated in clinical trials; recently approved.

ACS = acute coronary syndrome; AF = atrial fibrillation; DCC = direct current cardioversion; i.v. = intravenous; N/A = not applicable; NYHA, New York Heart Association; p.o. = per os; QRS = QRS duration; QT = QT interval; T-U = abnormal repolarization (T-U) waves.

Problems with drugs cardioversion

- **Proarrhythmia**:
  - VT with Flecainide, Propafenone in LVH, CAD, Decreased EF
  - Torsades in Dronedarone, Sotalol, Dofetilide
- **Organ Toxicity**:
  - Amiodarone, procainamide, quinidine
  - Organ Toxicity: Lupus, agranulocytosis, thrombocytopenia, optic neuritis, pulmonary fibrosis, hepatitis, etc.

Clinical Case Revisited

CHA_2DS_2-VASc=4=4% stroke risk

HAS-BLED=3=16% bleeding risk

Clinical Case Revisited

What is the next appropriate management for this patient?

A) IV diltiazem
B) Intubation
C) Urgent Cardioversion
D) IV pain control
E) CT pulmonary angiogram

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Atrial fibrillation patients with CHA_2DS_2-VASc > 1 benefit from oral anticoagulation prior to cardioversion☆

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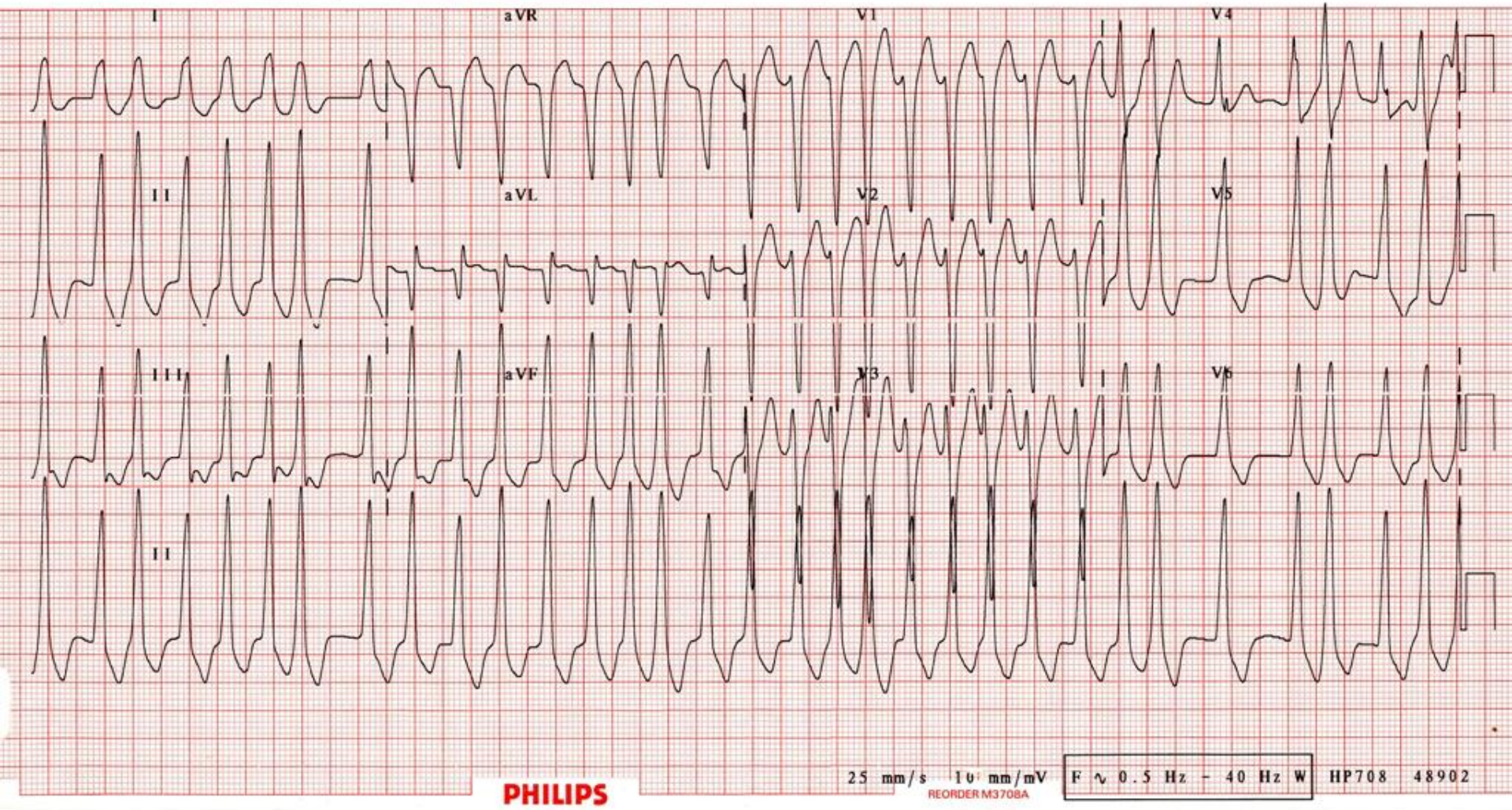
ABSTRACT

Background: Electrical cardioversion of atrial fibrillation is associated with an increased risk of embolic stroke, but is generally considered safe if performed within 48 h after onset. Our objective was to investigate if thromboembolism and bleeding in association with cardioversion of atrial fibrillation differed between patients with and without oral anticoagulation.

Methods: Retrospective study of patients with atrial fibrillation undergoing electrical cardioversion from national Swedish health registries from January 1st 2006 until December 1st 2010. Main outcome measures were thromboembolism and bleeding.

Results: In total 22,874 atrial fibrillation patients underwent electrical cardioversion, 10,722 with and 12,152 without oral anticoagulation pre-treatment. Patients with low stroke risk (CHA_2DS_2-VASc 0–1) did not suffer from any thromboembolic complications within 30 days after cardioversion. After adjustment for factors included in CHA_2DS_2-VASc and after propensity score matching, patients without oral anticoagulation had higher risk for thromboembolic complications, odds ratio 2.54 (95% confidence interval 1.70–3.79) and odds ratio 2.51 (95% confidence interval 1.69–3.75). There were no significant differences regarding bleeding complications between patients with or without anticoagulation after adjustment for factors included in HAS-BLED, odds ratio 1.08 (95% confidence interval 0.51–2.25), nor after propensity score matching, odds ratio 1.00 (95% confidence interval

Conclusion: The results suggest that electrical cardioversion without prior anticoagulation may not be safe for patients with risk factors for thromboembolism (CHA_2DS_2-VASc score >1 point).


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s 10 mm/mV
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HP708 48902
PHILIPS
REORDER M3708A

Wolff-Parkinson-White Syndrome (WPW)

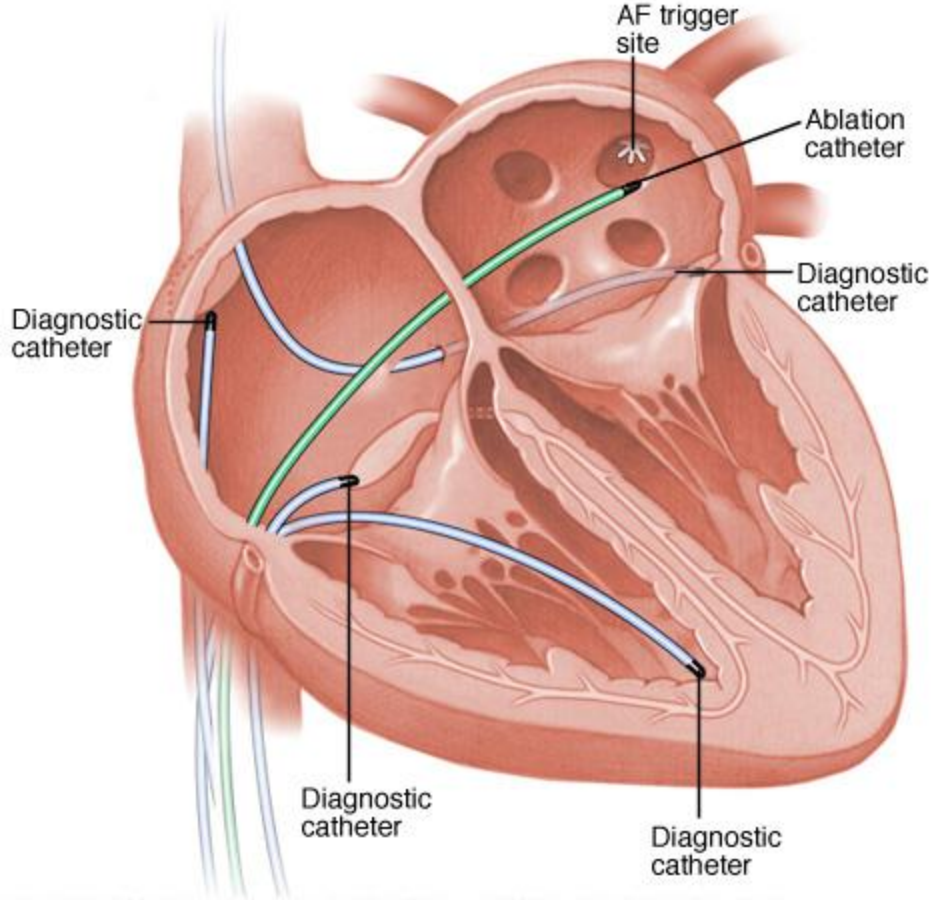
Differential of A.fib with wide QRS: **A.fib with aberrancy** (RBBB or LBBB – QRS usually has typical morphology), or **A.fib with pre-excitation** – eg, WPW: esp. when QRS morphology is bizarre, polymorphic and much faster than usual A.fib (sometimes approaching 300)

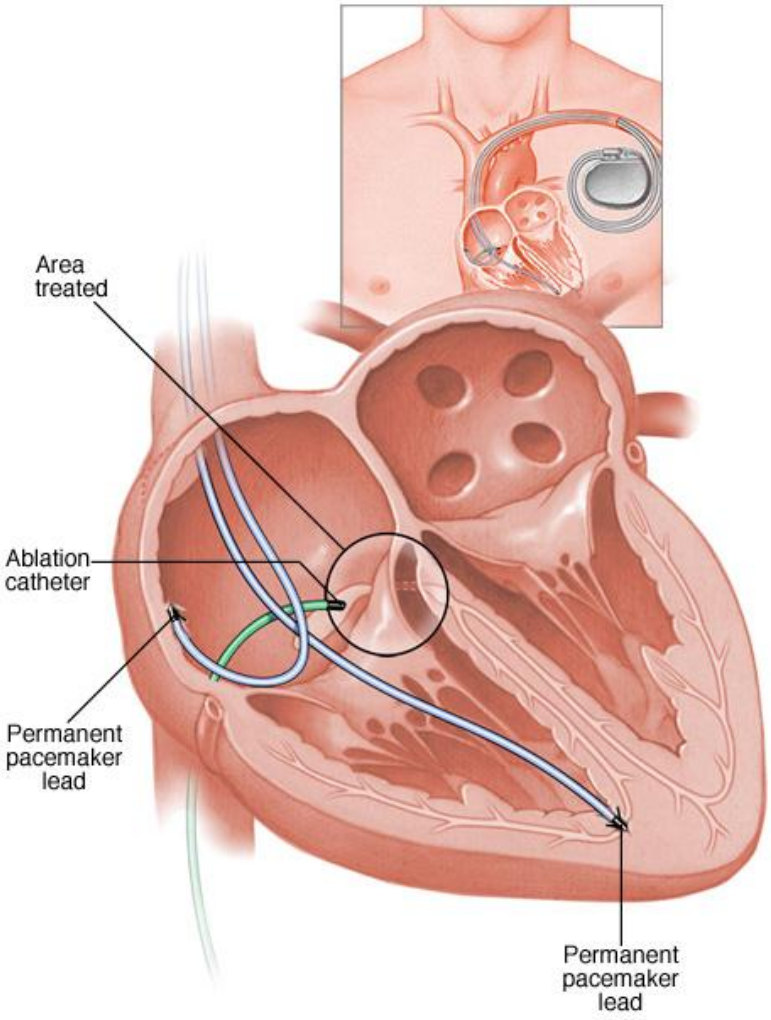
NEVER give AV nodal blocking agent (beta-blocker, calcium-channel blocker, adenosine, digoxin and even amiodarone) as the AV node will be blocked and impulses sent preferentially down the bypass tract – which doesn't have any slowing mechanism – and trigger VF

Treatment: electrical cardioversion, or procainamide is the safest medication

Key messages

- If patient is hemodynamically unstable in setting of atrial fibrillation (with hypotension, angina, decompensated heart failure…) then proceed with direct synchronized cardioversion
- Anticoagulation is protective
- Rate control is goal for Afib with RVR for symptomatic management
- Initial rate control agents are diltiazem or metoprolol





2016 AF guidelines in mobile apps

ESC pocket guidelines app

- **can be accessed free of charge**
- **over 58000 unique users**
- **25 titles, > 130 practical tools**
- **2016 ESC AF Guidelines integrated**
  - Tools supporting integrated AF care
  - Check the General AF Treatment Manager

ANDROID APP ON Google play

Available on the App Store

amazon

CATCH ME

To support integrated AF care, the ESC Guidelines task force and the CATCH ME consortium (www.catch-me.info) have developed state-of-the-art interactive tools underpinning integrated AF management. A first version including an overall treatment manager is integrated into the AF section of the ESC pocket guidelines app. Further CATCH ME tools for healthcare professionals and an associated app for AF patients will be released in late 2016 / early 2017.

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Everybody has to realize what he/she has to do

Traveling Fast or Traveling to Get Somewhere?

If you want to travel fast, you travel alone. If you want to go far, travel with others.

African Proverb