Stadtspital Zürich

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USZ Universitäts Spital Zürich

Institut für Hausarztmedizin
Fortbildungsprogramm 2026:
Kardiologie in der Praxis

Rhythmusstörungen

Stephan Andreas Müller-Burri
Cardiology Triemli, Stadtspital Zürich
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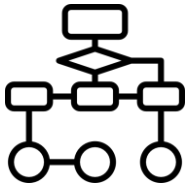
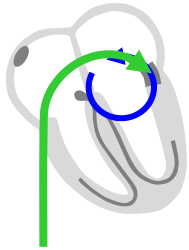
Learning objectives

With my case-based presentation, we will repeat:

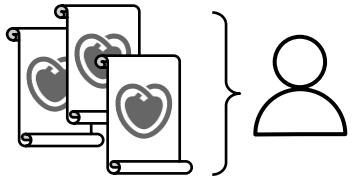


(1) The diagnostic pathway from symptoms to the diagnosis of patients with arrhythmias.



(2) Treatment strategies for common tachycardias.

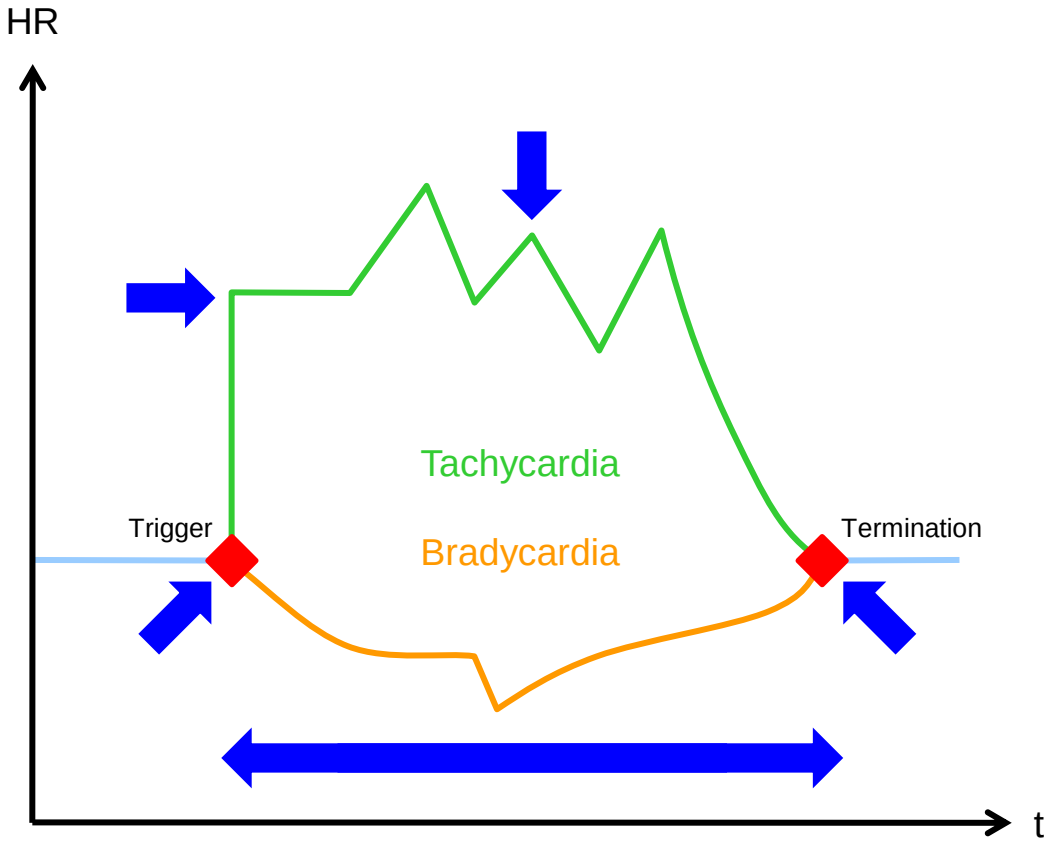
(3) Indications for the implantation of PM and ICD.



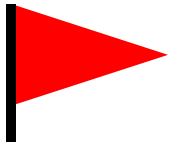
(4) How to develop guidelines based individualized treatment plans in complex patients.

Palpitation: History

Clinical characteristics of palpitation



Palpitation: Red flags



History

- Family history of heart disease, syncope, sudden cardiac arrest
- Established diagnosis of heart disease

Associated symptoms

- Occurrence during physical stress
- Dyspnea
- Chest pain (angina pectoris)
- Dizziness, syncope

Cardiological findings

- ECG abnormalities
- Ischemic or structural heart disease
- Documented arrhythmia during symptoms (correlation)

→ Consider further cardiological work-up / management

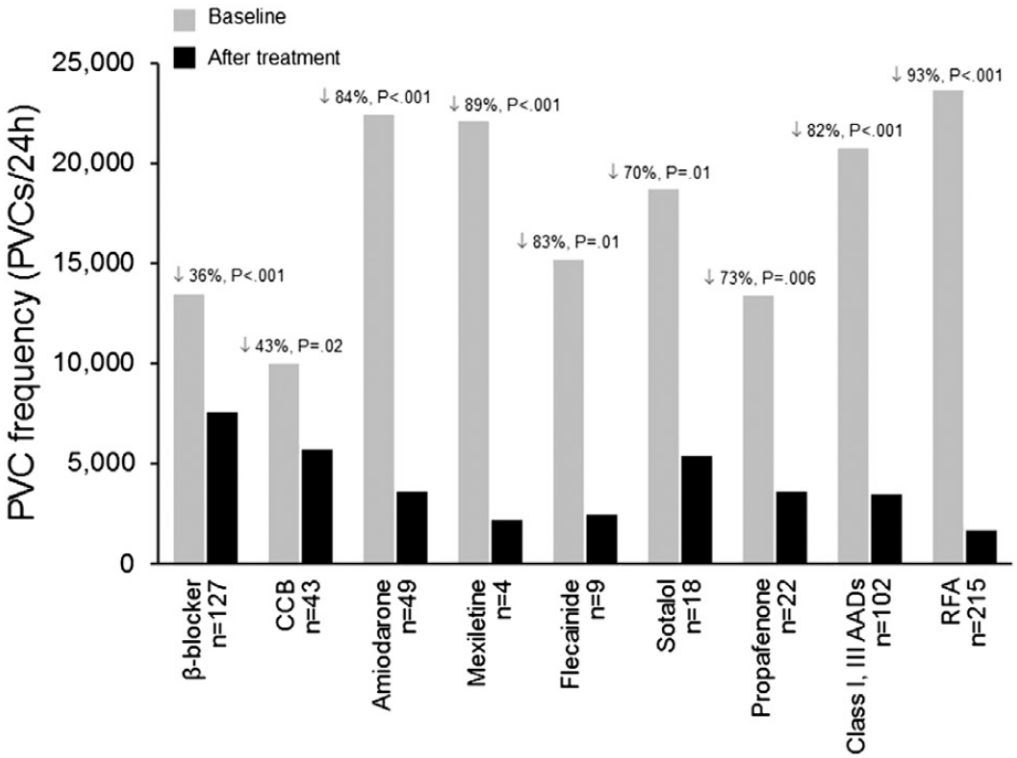
Outflow tract ventricular tachycardia

Efficacy of antiarrhythmic drugs

Mayo Clinic, retrospective analysis: 510 patients with frequent PVCs (>1000/24 h). 295 treated with AAD, 215 with radiofrequency ablation.

Results

AAD	N	PVC↓
β-blocker	127	-36%
CCB	43	-43%
Class I, III	102	-82%
RFA	215	-93%



Reduction in the frequency of PVCs after treatment with AAD or RFA.

1. Zhong L, et al. Heart Rhythm 2014;11:187-93.

Idiopathic PVC/VT

Therapeutic options according to the site of origin and presentation

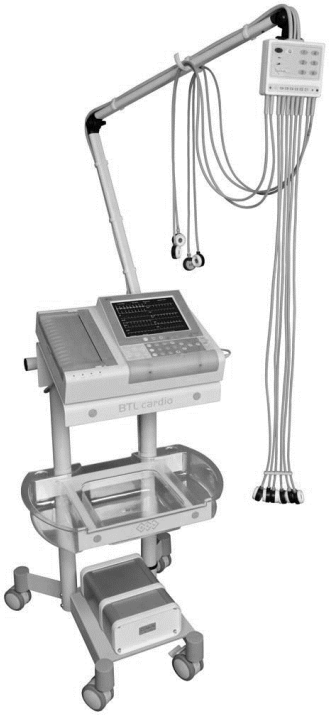
- RVOT and fascicular — Ablation > AAD
- Other site of origin — AAD (βB, CCB) > Ablation = flecainide
- LV dysfunction — Ablation > AAD (βB, flecainide, amiodarone)

		Ablation	Beta-blocker	CCB	Flecainide	Amiodarone
RVOT/fascicular PVC/VT: Symptomatic, normal LV function	symptomatic	Class I	Class IIa	Class IIa	Class IIa	Class III
PVC/VT other than RVOT/fascicular: Symptomatic, normal LV function	symptomatic	Class IIa	Class I	Class I	Class IIa	Class III
RVOT/fascicular PVC/VT: LV dysfunction	LVEF↓	Class I	Class IIa	Class III[a]	Class IIa[b]	Class IIa
PVC/VT other than RVOT/fascicular: LV dysfunction	LVEF↓	Class I	Class IIa	Class III[a]	Class IIa[b]	Class IIa
PVC: Burden >20%, asymptomatic, normal LV function		Class IIb				Class III

CCB, calcium channel blocker; LV, left ventricular; PVC, premature ventricular complex; RVOT, right ventricular outflow tract; VT, ventricular tachycardia.
[a]Intravenous calcium channel blockers.
[b]In selected patients (only moderate LV dysfunction).

1. Zeppenfeld K, et al. 2022 ESC GL VT and SCD. EHJ 2022;00:1-130.

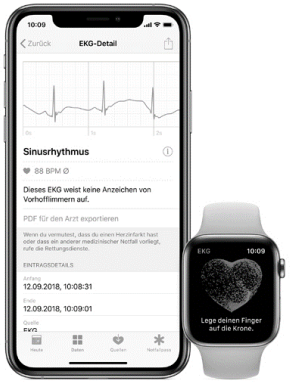
Diagnosis of cardiac arrhythmias



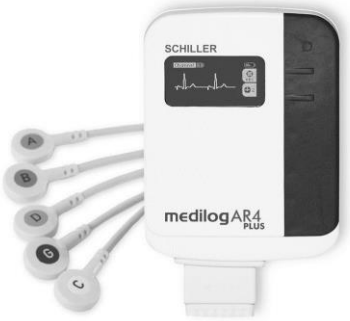
Standard 12-lead ECG

ECG patches

External loop recorder

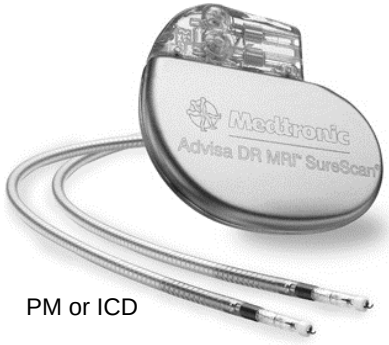


Smart Watch



Holter-ECG

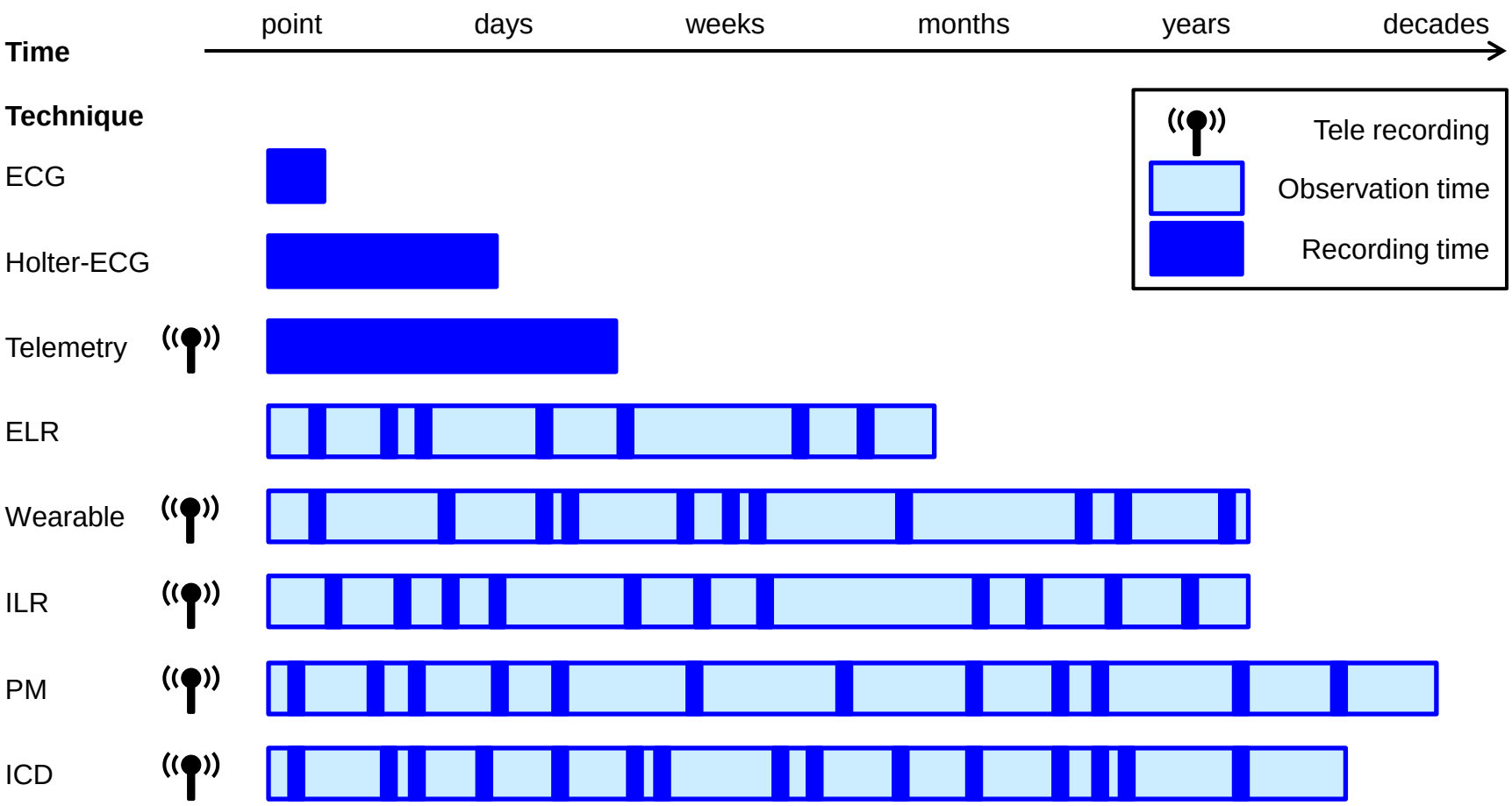
Implantable loop recorder



PM or ICD

1. Van Gelder IC, et al. 2024 ESC GL on AF. EHJ 2024;45:3314-414. 2. Bouzid Z, et al. Heart Rhythm 2022;19:1192-1201.

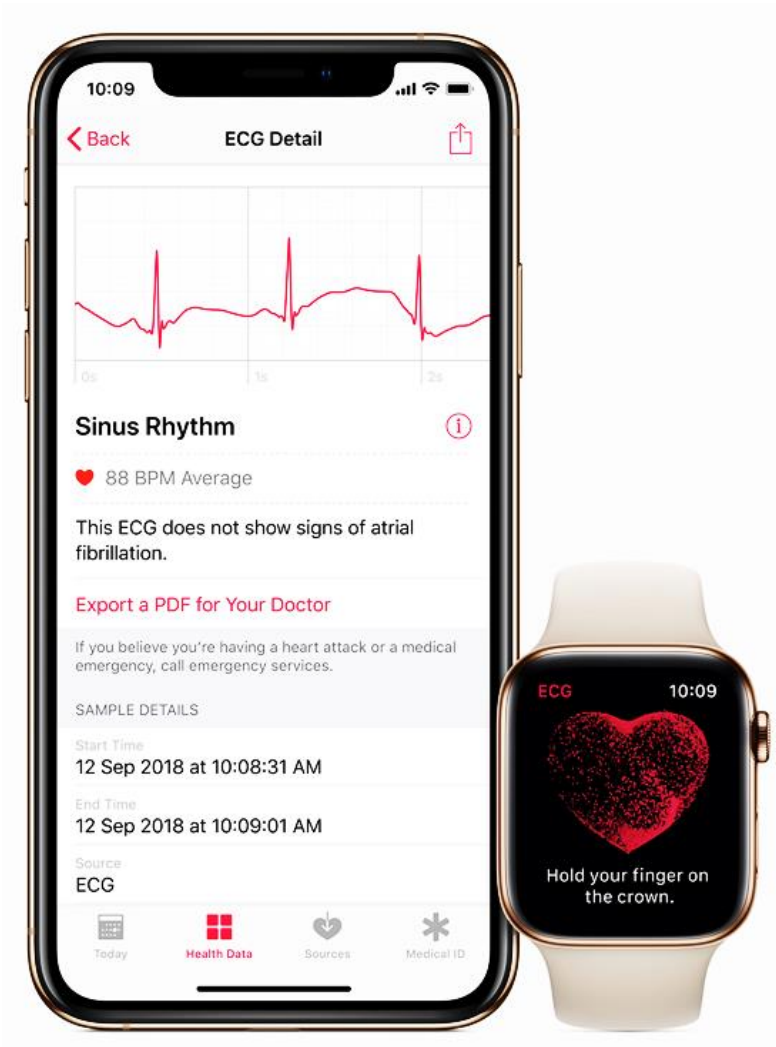
ECG recording tools

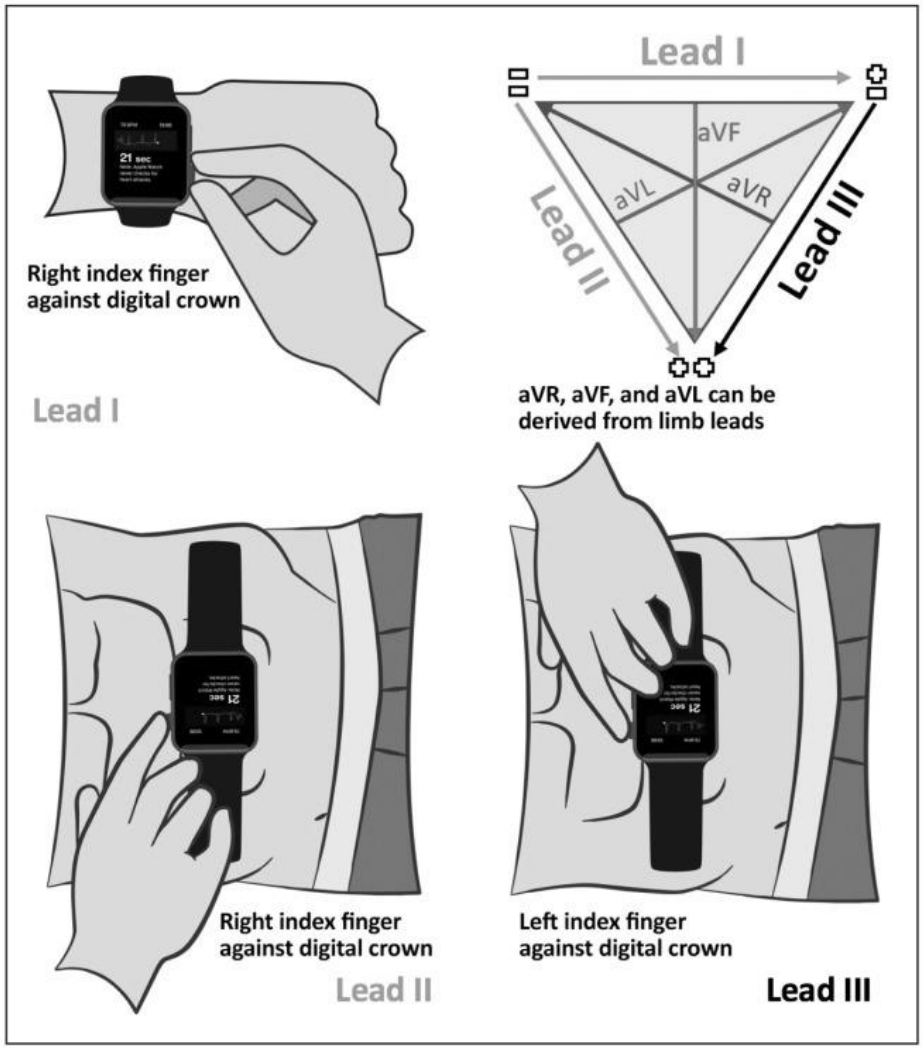


ECG denotes electrocardiogram, ELR external loop recorder, ILR implantable loop recorder, PM pacemaker, ICD internal cardioverter defibrillator.

1. Zimetbaum P, Goldmann A. Circulation 2010;122:1629-36. 2. Van Gelder IC, et al. 2024 ESC GL on AF. EHJ 2024;45:3314-414.

Smart Watch recordings





1. Apple.

Arrhythmia detection: wearable devices

Accuracy of wearable devices in measuring HR during SVT

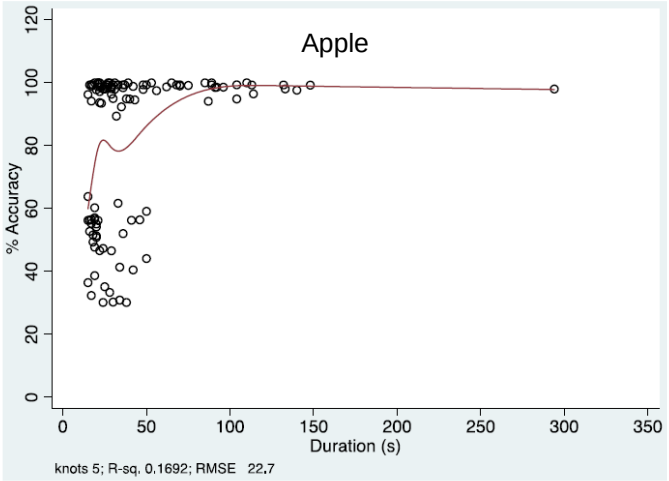
52 patients, wearable device during EPS for SVT.

Results: Accuracy of the detected HR

Episodes	Apple	Fitbit	Garmin	Polar
Duration <15 s	18.7%	19.5%	1.5%	37.7%
Duration 15-60 s	67.6%	42.4%	24.3%	65.5%
Duration >60 s	100%	35%	36%	90%

Conclusion

All wearable devices are inaccurate for short-duration SVT. Some devices are accurate for longer duration SVT.



1. Sequeira N, et al. Heart Rhythm 2020;17:854-9.

Management of wide QRS tachycardia

Wide QRS complex, regular tachycardia should routinely be treated as VT, unless the diagnosis of SVT with aberrancy or of SVT with pre-excitation is certain. (1)

In case the mechanism of the arrhythmia is not fully understood, the arrhythmia should be treated as VT. (2)

If there is any doubt treat the arrhythmia as ventricular tachycardia!

1. Delacrétaz E. NEJM 2006;354:1039-51. 2. Brugada J, et al. 2019 ESC GL for the management of patients with SVT. EHJ 2020;41:655-720.

SVT: Chronic therapy

Dx	IC	II	Sotal	Amio	IV	Iva	Other	Ablation
SR tc	-	IIaC	-	-	IIbC	IIaB	IC*	IIaC
Efficacy	-	-	-	-	-	-	-	-
FAT	IIaC	IIaC	-	IIbC	IIaC	IIbC	-	IB
Efficacy	-	-	-	-	-	-	-	85%
AVNRT	-	IIaB	-	-	IIaB	-	-	IB
Efficacy	-	25%	-	-	25%	-	-	97%
AVRT	IIbB§	IIaB°	-	-	IIaB°	-	-	IB
Efficacy	-	-	-	-	-	-	-	92%
AFL	-	IIaC	-	IIbC	IIaC	-	-	IA/B
Efficacy	-	-	-	-	-	-	-	95% / 80%
AF	IA†	-	IA†	IA†	-	-	IA#†	IIaB / IA
Efficacy	33%	20%	33%	65%	-	-	33%	65-85%

Dx denotes diagnosis, Sotal sotalol, Amio amiodarone, Iva ivabradine, AVNRT AV node reentry tachycardia, AVRT AV reentrant tachycardia, FAT focal atrial tachycardia, AF atrial fibrillation, rhythm control. * Treatment of underlying disorder. ° only for orthodromic AVRT, § for anti-/orthodromic AVRT.† selected indication depending on the underlying heart disease. # Dronedarone.

1. Brugada J, et al. 2019 ESC GL for the management of patients with SVT. EHJ 2020;41:655-720. (adapted) 2. Kirchhof P, Benussi S, et al. 2016 ESC guidelines for the management of atrial fibrillation. Eur Heart J 2016;37:2893-962. 3. Katritsis DG, et al. Europace 2017;19:602-6.

Diagnosis of AF: ECG

Definition

Cardiac arrhythmia with the following characteristics:

1. The ECG shows absolutely irregular RR intervals (arrhythmia absoluta)
2. No distinct P waves on the ECG
3. Variable short atrial cycle length (<200 ms)

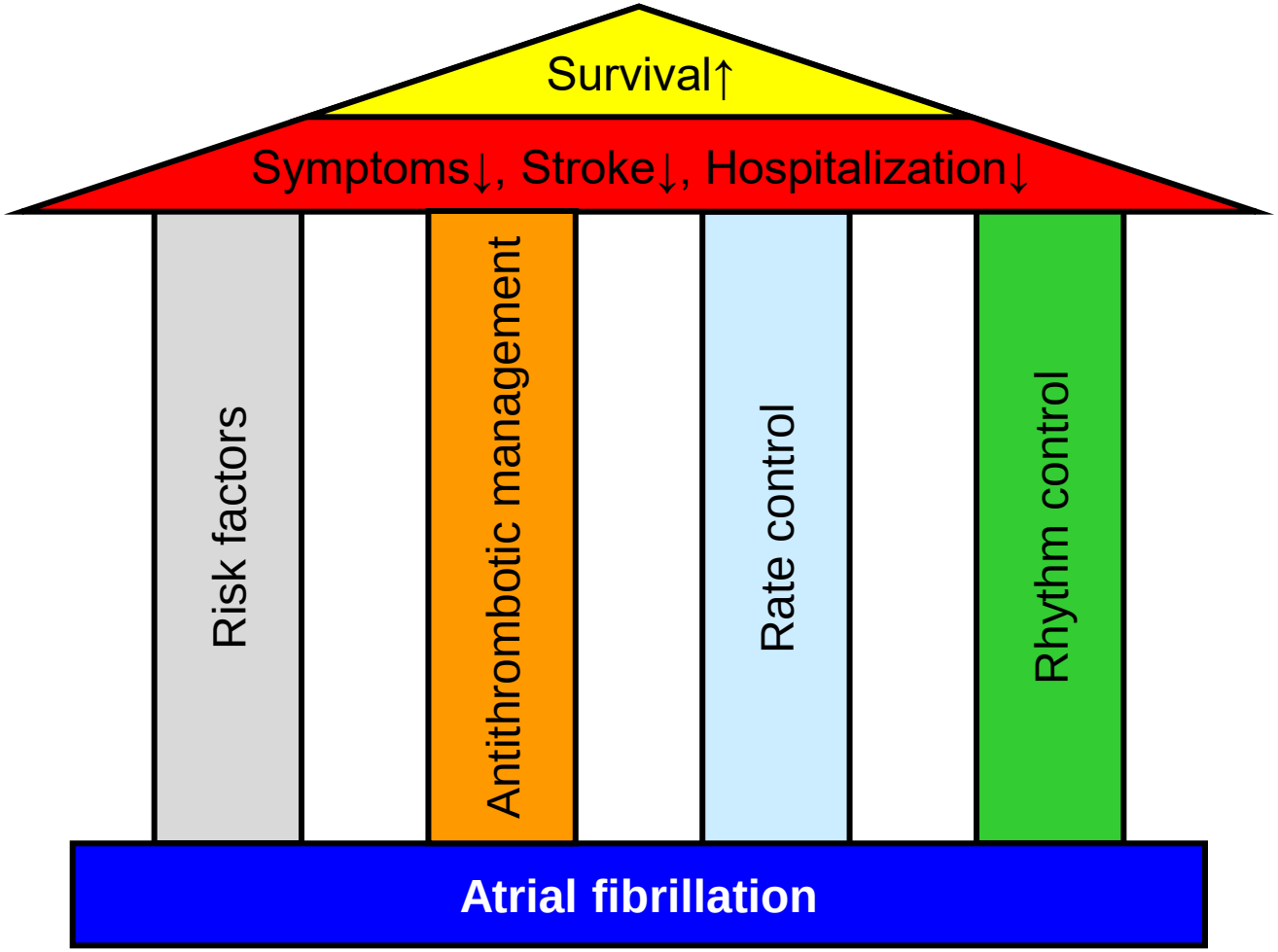
Minimal duration of an episode: >30 s (by accepted convention)

Differential diagnosis

- **Atrial flutter:** regular organized activity (aCL >200 ms) with usually regular ventricular activation (2:1, 3:1, variable)
- **Multifocal atrial tachycardia:** P wave in front of every QRS complex. A least 3 different P wave morphologies on the ECG.
- **Regular supraventricular tachycardias:** e.g. AVNRT, AVRT, focal atrial tachycardia
- **Frequent atrial or ventricular ectopy** or **dual AV nodal conduction**

1. Camm AJ. et al. ESC 2010 guidelines for the management of atrial fibrillation. Eur Heart J 2010;31:2369-2429. 2. Practical Rate and Rhythm Management of Atrial Fibrillation, Pocket Guide, HRS, January 2010. 3. Kirchhof P, Benussi S, et al. 2016 ESC guidelines for the management of atrial fibrillation. Eur Heart J 2016;37:2893-962.

Management of AF



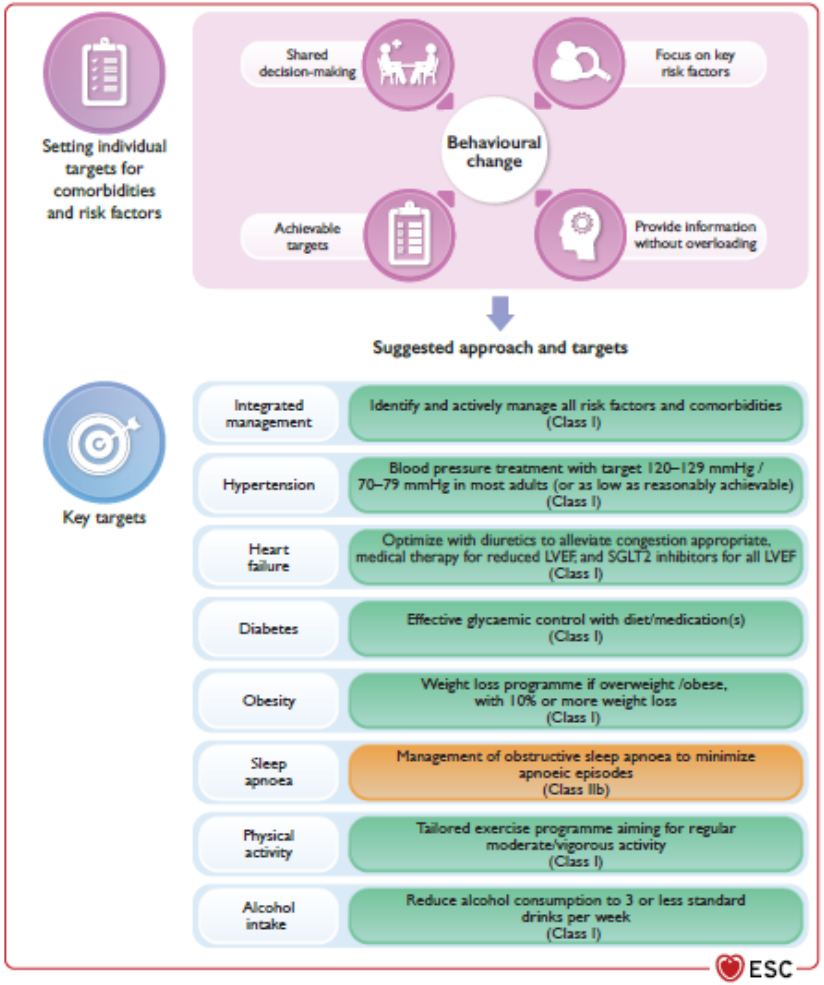
1. Kirchhof P, Benussi S, et al. EHJ 2016;37:2893-962. 2. Chung MK, et al. Circ 2020;141. DOI: 10.1161/CIR.0000000000000748. (adapted)

Integrated care model for AF management

Risk factor management

Comprehensive recommendations for I°/II° prevention

Risk factor	Target
• AHT	BD <130/80 mmHg
• Heart failure	OMT, SGLT2i
• Diabetes	Glycemic control
• Alcohol intake	≤3 drinks/week
• Obesity	Weight loss >10%
• Sleep apnea	CPAP (IIb)
• Physical activity	Regular activity



1. Van Gelder IC, et al. EHJ 2024;45:3314-414.

Antithrombotic management

CHA2DS2-VA, new definitions (1)

Parameters	Definition	Points
Chronic heart failure	Symptoms or signs of HF (irrespective of LVEF) or asymptomatic LVEF ≤40%	1
Hypertension	Resting BP >140/90 mmHg, or antihypertensive treatment	1
Age ≥75 years		2
Diabetes mellitus	According to currently accepted criteria, or glucose lowering therapy	1
Stroke/TIA/TE		2
Vascular disease	CAD: prior myocardial infarction, PCI, or significant CAD on imaging studies PVD: claudication, previous revascularization (incl. aorta), or complex aortic plaques (≥4 mm) on imaging	1
Age 65-74 years		1

Stroke Risk (2)	
Stroke rate	**%/y**
CHA_2DS_2-VASc	
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	15.20

1. Van Gelder IC, et al. 2024 ESC GL on AF. EHJ 2024;45:3314-414. 2. Camm AJ. et al. EHJ 2010;31:2369-2429.

Antithrombotic management

Anticoagulation

(1) Cardioversion (electrical/pharmacological) off anticoagulation only
- AF onset **<24 hours** (not <48 h)
- After exclusion of an intracardiac thrombus by TEE or CT

(2) New risk score: CHA_2DS_2-VA~~[Sc]~~: start OAC at ≥1 point

(3) Anticoagulation in all AF patients with HCM and cardiac amyloidosis

(4) NOAC as first line therapy, except

(5) VKA only for a) mechanical heart valves and
b) moderate to severe mitral valve stenosis

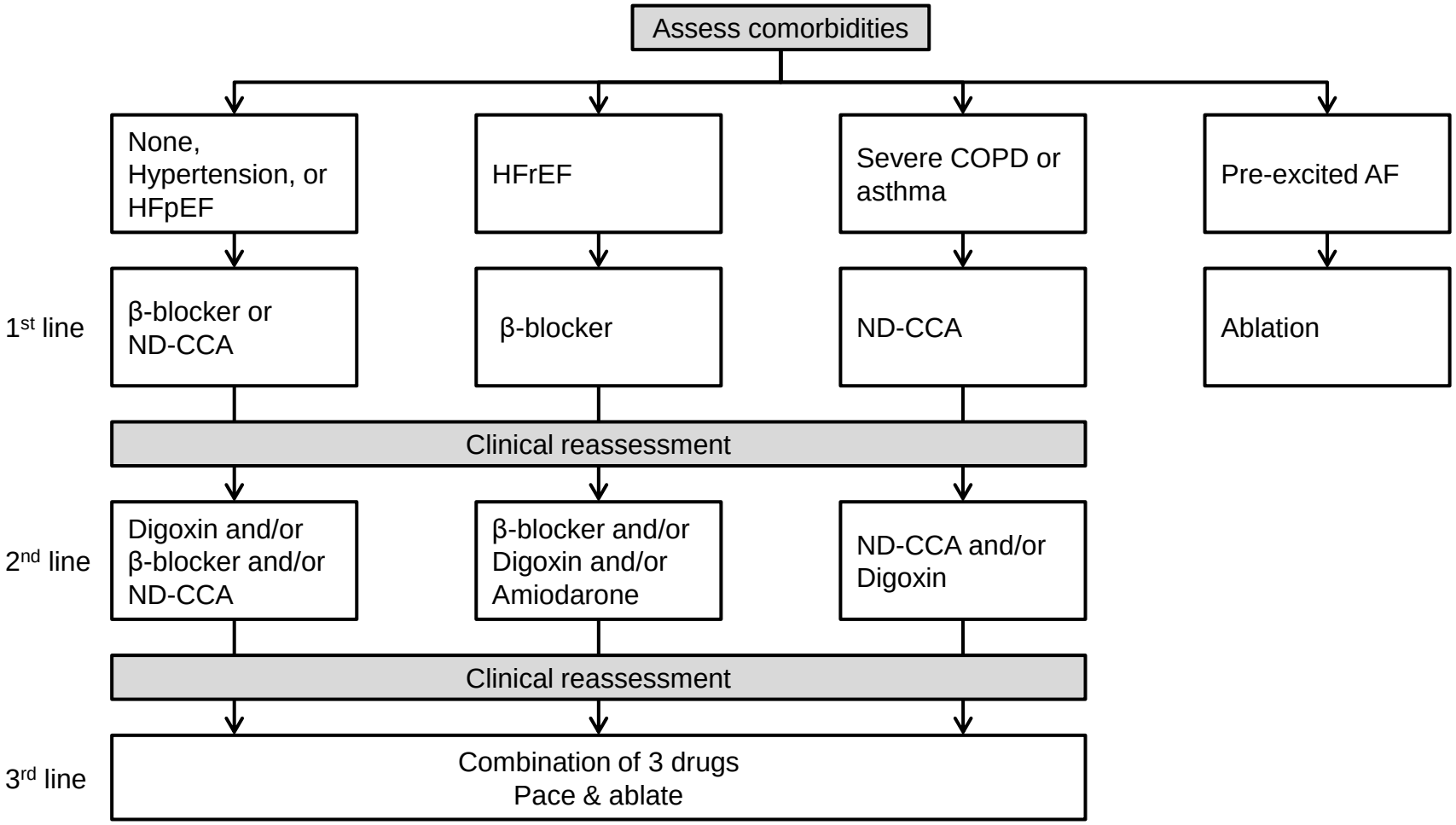
Bleeding risk

- Do not use bleeding risk scores (e.g. HAS-BLED) to decide starting or withdrawing OAC. (III)
- Manage all modifiable risk factors: AHT, antiplatelet drugs, unstable INR, LMWH bridging, NSAID, corticosteroids, alcohol, drug-drug interactions
- Offer PPI to avoid GI bleeding

1. Van Gelder IC, et al. 2024 ESC GL on AF. EHJ 2024;45:3314-414.

Rate control

How can you achieve the optimal heart rate during AF?



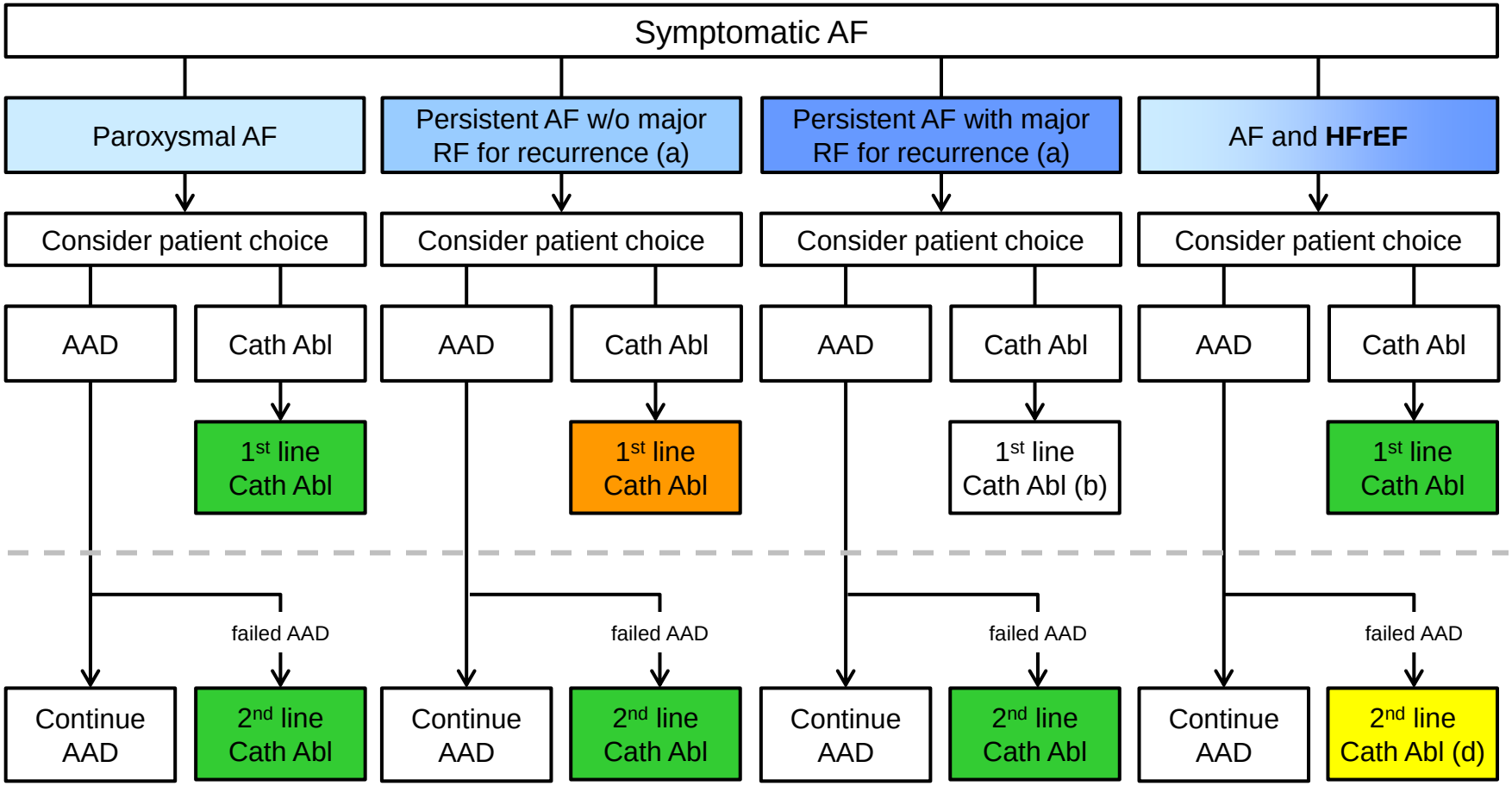
1. Van Gelder IC, et al. Lancet 2016;388:818-28 (adapted).

Antiarrhythmic drugs (AAD)

Drug	Dose	Contraindication	Side effects
Class I			
Flecainide	Oral: 50-150 mg, 2x/d	Structural heart disease CreaCl <50 ml/min	Bradyarrhythmias, fast AF, Torsades de pointes, Inotropy↓
Propafenone	Oral: 150-300 mg, 3x/d	Structural heart disease	c.f. Flecainide Gastrointestinal
Class III			
Sotalol	Oral: 80-160 mg, 2x/d	LV hypertrophy Systolic CHF QT prolongation CreaCl <50 ml/min	Bradyarrhythmias, Torsades de pointes, Inotropy↓
Dronedarone	Oral: 400 mg, 2x/d	Unstable CHF CHF NYHA III and IV Permanent AF CreaCl <30 ml/min	Gastrointestinal Crea↑ by 10-20 µmol/l Interaction with Digoxin, VKA and Dabigatran
Amiodarone	Oral loading: 600-800 mg/d Maintenance: 100-200 mg/d	Hyperthreoidism Pulmonary disease Lever disease	Bradyarrhythmias, QT interval↑, many extracardiac side effects, interaction with Digoxin, VKA, and others

1. Practical Rate and Rhythm Management of Atrial Fibrillation, Pocket Guide, HRS, January 2010. 2. ESC 2010 guidelines for the management of atrial fibrillation. Euro Heart J 2010. 3. Lip GYH, et al. Lancet 2012;379:648–61.

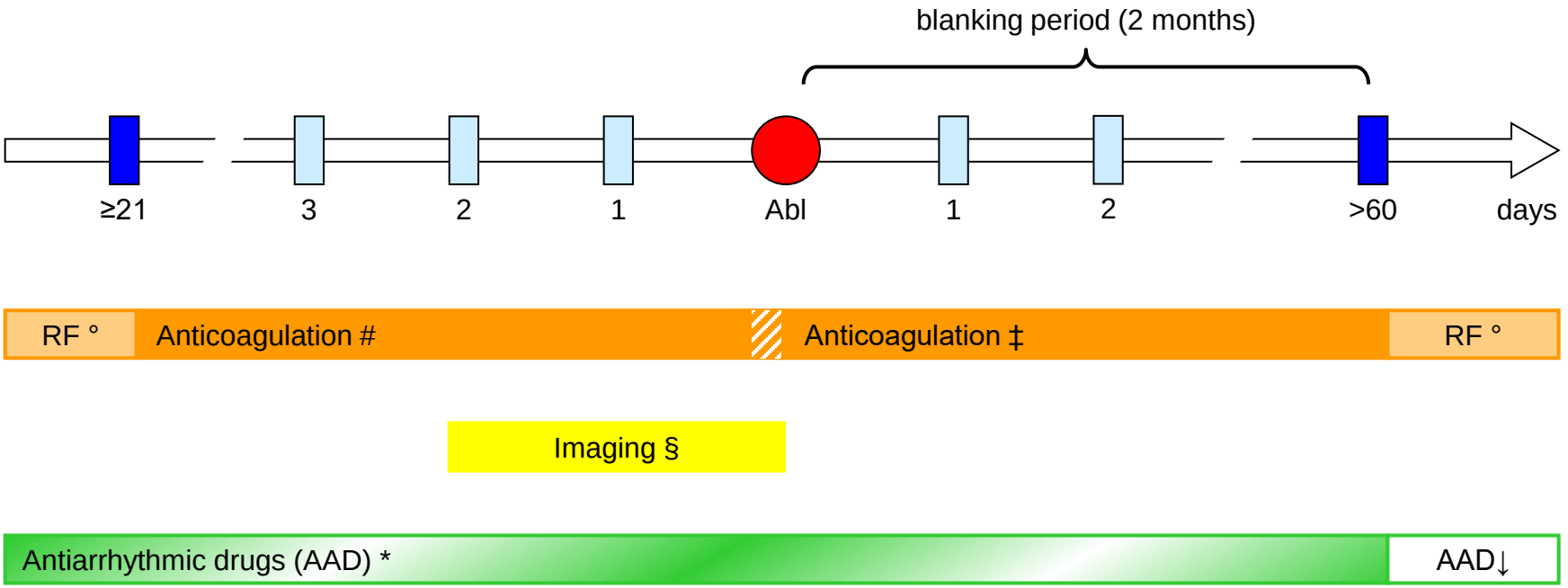
Catheter ablation (ESC GL 2020/24)



a) RF for AF recurrence: enlarged LA volume, advanced age, long AF duration, renal dysfunction, and other cardiovascular risk factors.
b) In rare individual circumstances, catheter ablation may be carefully considered as 1st line therapy.
c) Recommended to reverse LV dysfunction when tachycardiomyopathy is highly probably. d) To improve survival and reduce hospitalization.
Recommendations: ■ Class I, ■ Class IIa, ■ Class IIb.

1. Hindricks G, et al. 2020 ESC CL AF. EHJ 2020;42:373-498 (adapted). 1. Van Gelder IC, et al. 2024 ESC GL on AF. EHJ 2024;45:3314-414 (adapted).

Standard PVI protocol



* No specific recommendation for the periprocedural management of antiarrhythmic drug therapy
° Anticoagulation according to general recommendation (CHA_2DS_2-VA ≥1 or very risk for thromboembolism: hypertrophic cardiomyopathy, amyloidosis, rheumatic heart disease)
\# Anticoagulation for all patients (except patients with PAF and very low risk (i.e. CHA_2DS_2-VA = 0))
‡ Anticoagulation for all patients after left atrial ablation
§ Imaging (TEE, CT-LA, CMR, ICE) within 48 h before the procedure to exclude left atrial thrombus

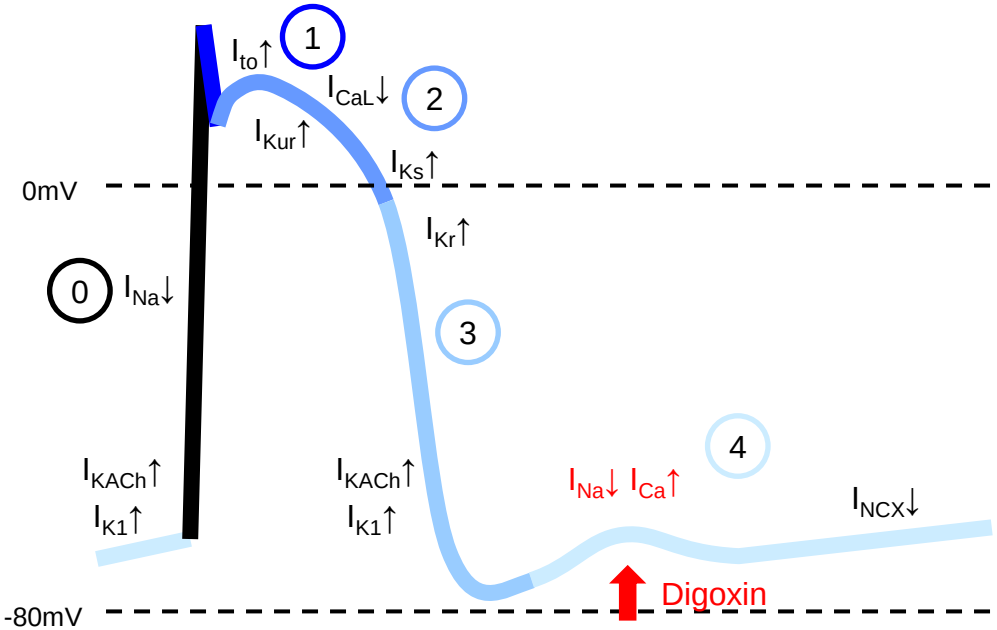
1. Van Gelder IC, et al. 2024 ESC GL on AF. EHJ 2024;45:3314-414. 2. Tzeis S, Gerstenfeld EP, et. al. Heart Rhythm 2024;21:e31-e149.

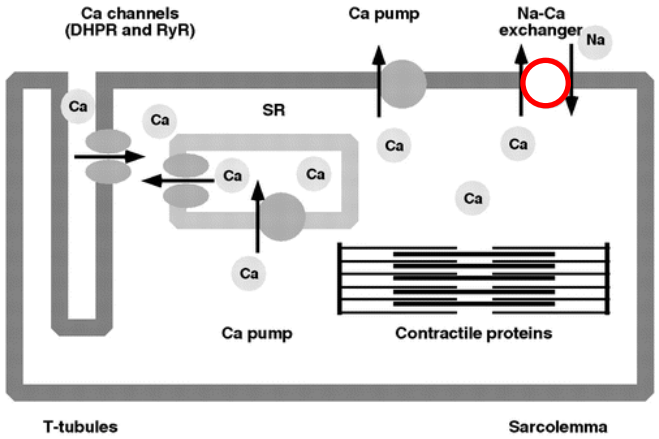
Digoxin toxicity

Actions of digoxin

- Inhibition of the Na^+/K^+-ATPase → intracellular $[Na^+]$↑ → intracellular $[Ca^{2+}]$↑ → Inotropy↑
- Increase in vagal tone → slowing of the vHR in AF

Mechanism of digitalis toxicity





Digoxin induced intracellular Ca^{2+} overload promotes the increased activity of the Na^+/Ca^{2+} exchanger in Phase 4 → delayed after depolarization (DAD).

1. Wellens HJJ, Conover MB. The ECG in Emergency Decision Making. 2nd Edition 2006 W.B. Saunders Company. p. 158-176. 2. Nattel S. in Mont L, Brugada J. A practical approach to clinical electrophysiology, 1st ed, 2010. p. 15-24. 3. Niggli E. Annu Rev Physiol 1999;61:311-35.

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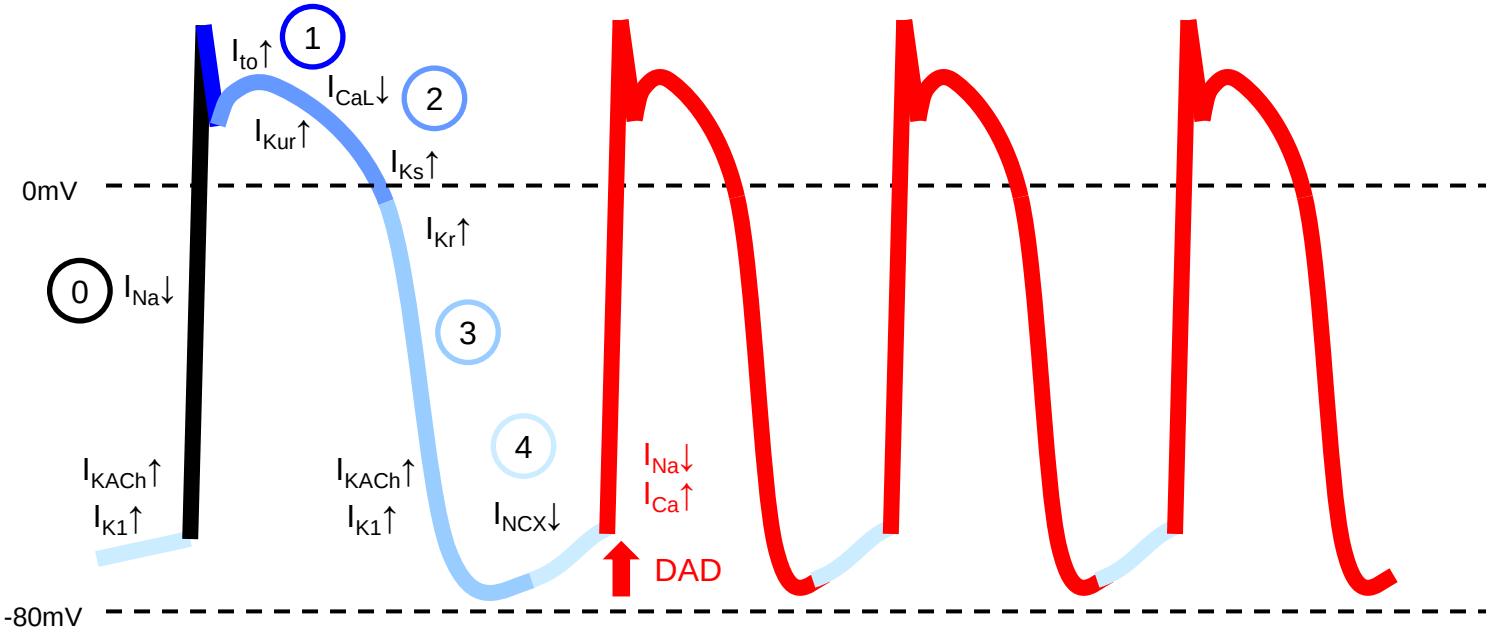

1. Wellens HJJ, Conover MB. The ECG in Emergency Decision Making. 2nd Edition 2006 W.B. Saunders Company. p. 158-176.

Digoxin toxicity

Plasma levels

Digoxin ≥ 1.7-2.0 nmol/l (no correlation with toxicity)

Risk factors for clinical manifestation

- Hypokalemia, especially as a trigger for cardiac arrhythmias
- Hypomagnesemia
- Hypercalcemia
- Myocardial ischemia

Symptoms

Gastrointestinal: anorexia, nausea, vomiting, and abdominal pain

Neurological: Changes in mental status as lethargy, fatigue, delirium, confusion, disorientation, and weakness.

Visual changes: chromatopsia, diplopia, photophobia, decreased visual acuity, photopsia, scotomas, or blindness

1. Wellens HJJ, Conover MB. The ECG in Emergency Decision Making. 2nd Edition 2006 W.B. Saunders Company. p. 158-176. 2. https://compendium.ch.

Digoxin toxicity: cardiac arrhythmias

Level	Bradyarrhythmias	Tachyarrhythmias
Sinus node	• Sinus bradycardia • SA block	• Sinus tachycardia
Atrium		• Ectopic atrial tachycardia (aHR usually < 250 bpm) with (2:1) AV block
AV node	• PR prolongation > 200 ms • 2nd degree AV block, type Wenckebach • 3rd degree AV block	• Junctional rhythm with narrow QRS at various HR (HR < 40-60-120 bpm)
Ventricle		• PVC, bigeminus • Ventricular tachycardias • Bidirectional ventricular tachycardia • Ventricular fibrillation

1. Wellens HJJ, Conover MB. The ECG in Emergency Decision Making. 2nd Edition 2006 W.B. Saunders Company. p. 158-176.

Digoxin toxicity: Treatment

Monitoring, supportive care

- Bed rest, avoid sympathetic stimulation
- Correct electrolyte abnormalities
  Note: Hyperkalemia reflects the degree of digoxin toxicity

Treatment of ventricular tachycardias

- Digitalis antibodies (Fab)
- Phenytoin (with a backup pacemaker)

Treatment of bradyarrhythmias

- Temporary ventricular pacing

Avoid

- Rapid pacing (→ acceleration of tachycardias)
- Sudden cessation of pacing (→ asystole without escape rhythm)
- Carotid sinus massage (→ worsening of arrhythmias → VF)

1. Wellens HJJ, Conover MB. The ECG in Emergency Decision Making. 2nd Edition 2006 W.B. Saunders Company. p. 158-176.

Digoxin in medical history

THE EFFECT OF DIGOXIN ON MORTALITY AND MORBIDITY IN PATIENTS WITH HEART FAILURE

THE DIGITALIS INVESTIGATION GROUP*

ABSTRACT

Background The role of cardiac glycosides in treating patients with chronic heart failure and normal sinus rhythm remains controversial. We studied the effect of digoxin on mortality and hospitalization in a randomized, double-blind clinical trial.

Methods In the main trial, patients with left ventricular ejection fractions of 0.45 or less were randomly assigned to digoxin (3397 patients) or placebo (3403 patients) in addition to diuretics and angiotensin-converting–enzyme inhibitors (median dose of digoxin, 0.25 mg per day; average follow-up, 37 months). In an ancillary trial of patients with ejection fractions greater than 0.45, 492 patients were randomly assigned to digoxin and 496 to placebo.

Results In the main trial, mortality was unaffected. There were 1181 deaths (34.8 percent) with digoxin and 1194 deaths (35.1 percent) with placebo (risk ratio when digoxin was compared with placebo, 0.99; 95 percent confidence interval, 0.91 to 1.07; P=0.80). In the digoxin group, there was a trend toward a decrease in the risk of death attributed to worsening heart failure (risk ratio, 0.88; 95 percent confidence interval, 0.77 to 1.01; P=0.06). There were 6 percent fewer hospitalizations overall in that group than in the placebo group, and fewer patients were hospitalized for worsening heart failure (26.8 percent vs. 34.7 percent; risk ratio, 0.72; 95 percent confidence interval, 0.66 to 0.79; P<0.001). In the ancillary trial, the findings regarding the primary combined outcome of death or hospitalization due to worsening heart failure were consistent with the results of the main trial.

Conclusions Digoxin did not reduce overall mortality, but it reduced the rate of hospitalization both overall and for worsening heart failure. These findings define more precisely the role of digoxin in the management of chronic heart failure. (N Engl J Med 1997;336:525-33.)

ALTHOUGH digoxin is one of the most commonly prescribed drugs for the treatment of heart failure, there is uncertainty about its long-term efficacy and safety.[1-3] Several recent short-term, randomized trials indicated that withdrawing digoxin worsens functional status, exercise capacity, and the left ventricular ejection fraction in patients with heart failure.[4,5] However, the long-term effect of digoxin on mortality and hospitalization for heart failure or other causes is unknown. We conducted a randomized, double-blind, placebo-controlled trial to evaluate the effects of digoxin (Lanoxin, Glaxo Wellcome) on mortality from any cause (the primary end point) and on hospitalization for heart failure (the secondary end point) over a three-to-five-year period in patients with heart failure and normal sinus rhythm.[6]

METHODS

Design

The rationale and design of the study and the base-line characteristics of the patients have been reported previously.[7] Patients were enrolled at 302 clinical centers in the United States and Canada. The study was organized and conducted by a Steering Committee representing the National Heart, Lung, and Blood Institute; the Department of Veterans Affairs Cooperative Studies Program; and cardiologists from the United States and Canada. An independent Data and Safety Monitoring Board monitored the progress of the study. The study was approved by the institutional review board at each participating center. All the patients gave written informed consent.

Eligibility

Patients were eligible for the main trial if they had heart failure and a left ventricular ejection fraction of 0.45 or less (6800 pa-

Rekha Garg, M.D., Richard Gorlin, M.D., Thomas Smith, M.D., and

Digitoxin in Patients with Heart Failure and Reduced Ejection Fraction

U. Bavendiek,[1] A. Großhennig,[2] J. Schwab,[3,4] D. Berliner,[1] A. Rieth,[5] L.S. Maier,[6] T. Gaspar,[7,8] N.H. Thomas,[2] X. Liu,[2] S. Schallhorn,[1] E. Angelini,[1] S. Soltani,[1] F. Rathje,[1] M.-A. Sandu,[1] W. Geller,[1] R. Hambrecht,[9] M. Zdravkovic,[10] S. Philipp,[11] D. Kosevic,[12] G. Nickenig,[13] D. Scheiber,[14] S. Winkler,[15] P.M. Becher,[16-18] P. Lurz,[19] M. Hülsmann,[20] S. Wiesner,[2] C. Schröder,[21] B. Neuhaus,[22] A. Seltmann,[22] H. von der Leyen,[23,24] C. Veltmann,[1,25] S. Störk,[26,27] M. Böhm,[28] A. Koch,[2] and J. Bauersachs,[1] for the DIGIT-HF Study Group*

ABSTRACT

BACKGROUND

The therapeutic efficacy of the cardiac glycoside digitoxin in patients with heart failure and reduced ejection fraction is not established.

METHODS

In this international, double-blind, placebo-controlled trial, we randomly assigned patients with chronic heart failure who had a left ventricular ejection fraction of 40% or less and a New York Heart Association (NYHA) functional class of III or IV or a left ventricular ejection fraction of 30% or less and an NYHA functional class of II in a 1:1 ratio to receive digitoxin (at a starting dose of 0.07 mg once daily) or matching placebo in addition to guideline-directed medical therapy. The primary outcome was a composite of death from any cause or hospital admission for worsening heart failure, whichever occurred first.

RESULTS

Among 1240 patients who underwent randomization, 1212 fulfilled the criteria for inclusion in the modified intention-to-treat population: 613 patients in the digitoxin group and 599 in the placebo group. Over a median follow-up of 36 months, a primary-outcome event occurred in 242 patients (39.5%) in the digitoxin group and 264 (44.1%) in the placebo group (hazard ratio for death or first hospital admission for worsening heart failure, 0.82; 95% confidence interval [CI], 0.69 to 0.98; P=0.03). Death from any cause occurred in 167 patients (27.2%) in the digitoxin group and 177 (29.5%) in the placebo group (hazard ratio, 0.86; 95% CI, 0.69 to 1.07). A first hospital admission for worsening heart failure occurred in 172 patients (28.1%) in the digitoxin group and 182 (30.4%) in the placebo group (hazard ratio, 0.85; 95% CI, 0.69 to 1.05). At least one serious adverse event occurred in 29 patients (4.7%) in the digitoxin group and 17 (2.8%) in the placebo group.

CONCLUSIONS

Treatment with digitoxin led to a lower combined risk of death from any cause or hospital admission for worsening heart failure than placebo among patients with heart failure and reduced ejection fraction who received guideline-directed medical therapy. (Funded by the German Federal Ministry of Research, Technology, and Space and others; DIGIT-HF EudraCT number, 2013-005326-38.)

The authors' full names, academic degrees, and affiliations are listed at the end of the article. Johann Bauersachs can be contacted at bauersachs.johann@mh-hannover.de or at the Department of Cardiology and Angiology, Hannover Medical School, Carl-Neuberg-Straße 1, 30625 Hannover, Germany.

*A complete list of members of the DIGIT-HF Study Group is provided in the Supplementary Appendix, available at NEJM.org.

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CME

1. The Digitalis Investigation Group, DIG trial. NEJM 1997;336:525-33. 2. Bavendiek U, et al. DIGIT-HF, NEJM 2025;393:1155-65.

Sick Sinus Syndrome: Treatment

Sinus node dysfunction (1)

Recommendations	Class[a]	Level[b]
In patients with SND and a DDD pacemaker, minimization of unnecessary ventricular pacing through programming is recommended.[144,151,159,164,166–169]	I	A
Pacing is indicated in SND when symptoms can clearly be attributed to bradyarrhythmias.[14,128–131]	I	B
Pacing is indicated in symptomatic patients with the bradycardia–tachycardia form of SND in order to correct bradyarrhythmias and enable pharmacological treatment, unless ablation of the tachyarrhythmia is preferred.[17,20,21,136–138,170,171]	I	B
In patients who present chronotropic incompetence and have clear symptoms during exercise, DDD with rate-responsive pacing should be considered.[172,173]	IIa	B
AF ablation should be considered as a strategy to avoid pacemaker implantation in patients with AF-related bradycardia or symptomatic pre-automaticity pauses, after AF conversion, taking into account the clinical situation.[136–139,174]	IIa	C
In patients with the bradycardia–tachycardia variant of SND, programming of atrial ATP may be considered.[164,165]	IIb	B
In patients with syncope, cardiac pacing may be considered to reduce recurrent syncope when asymptomatic pause(s) >6 s due to sinus arrest is documented.[133,134]	IIb	C
Pacing may be considered in SND when symptoms are likely to be due to bradyarrhythmias, when the evidence is not conclusive.	IIb	C
Pacing is not recommended in patients with bradyarrhythmias related to SND that are asymptomatic or due to transient causes that can be corrected and prevented.[33]	III	C

Indications for pacing:

(1) Correlation between symptoms and bradycardia (IB)

(2) Tachy-brady syndrome to enable AAD therapy (IB)

(3) Syncope and documented asymptomatic pause(s) >6 s due to sinus arrest (IIb,C)

No indication for pacing:

Asymptomatic or reversible sinus node disease (III,C)

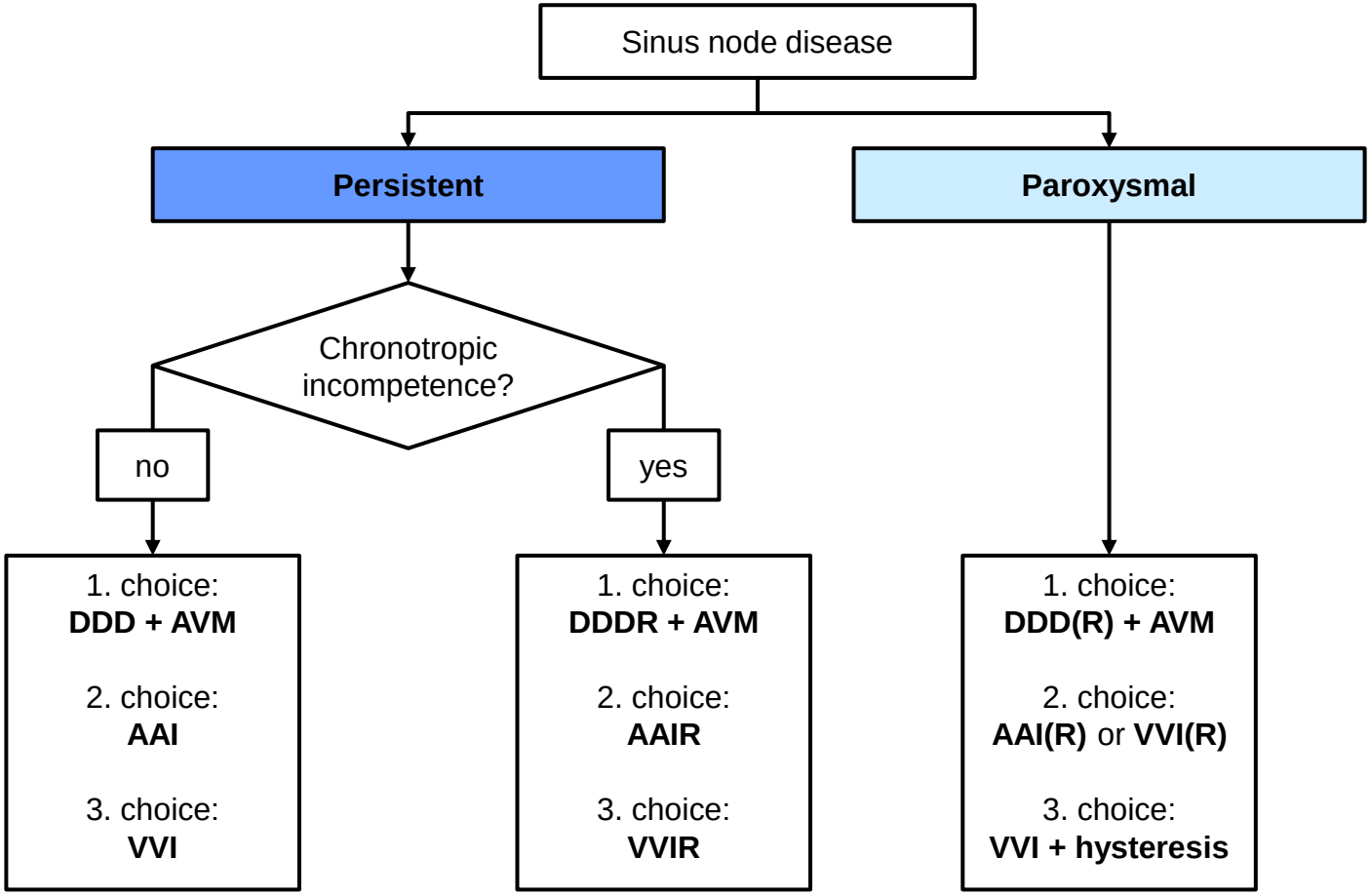
Other therapeutic option:

Consider catheter ablation for AF/AFL in patients with tachy-brady syndrome (IIa,C)

1. Glikson M, et al. ESC GL PM and CRT. EHJ 2021;42:3427-520.

Optimal pacing mode for bradycardia

ESC Guidelines: Sinus node disease (1,2)



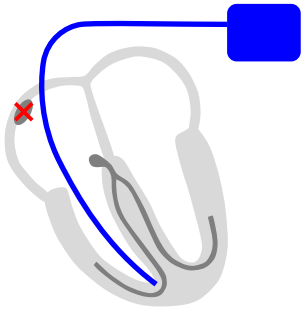
1. Brignole M, et al. ESC GL PM and CRT. EHJ 2013;34,2281-329. 2. Glikson M, et al. ESC GL PM and CRT. EHJ 2021;42:3427-520.

Available pacing systems for SSS

Transvenous pacemakers

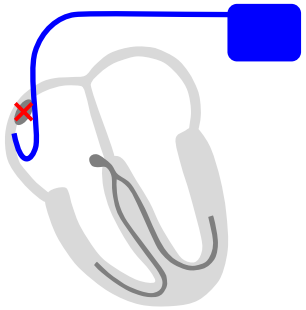
All systems +/- RV / CSP / LV lead

1-chamber pacing



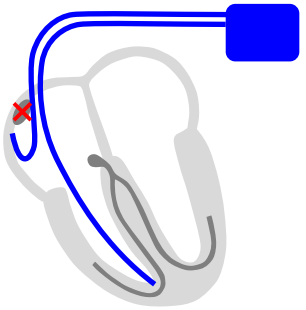
→ AV synchrony
→ ventricular dyssynchrony

AAI pacing



→ Minimal hardware
→ No RV pacing (backup)

2-chamber pacing



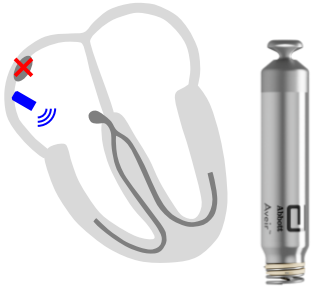
→ AV synchrony
→ ventricular dyssynchrony

Leadless pacemakers

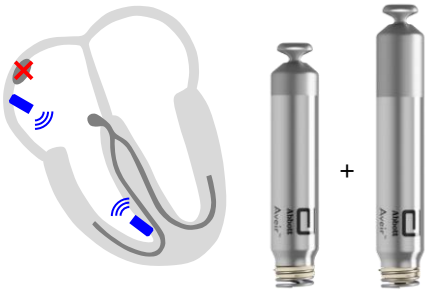
1-chamber pacing

or

AAI pacing



2-chamber pacing



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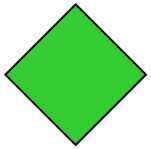
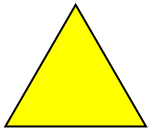
1. Brignole M, et al. ESC GL PM and CRT. EHJ 2013;34,2281-329. 2. Glikson M, et al. ESC GL PM and CRT. EHJ 2021;42:3427-520.

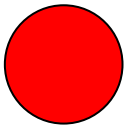
Summary



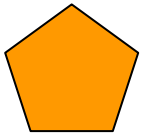
Case 1: **Palpitation due to ventricular extrasystole**
(1) History and workup / red flags / AAD and ablation



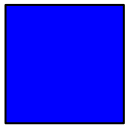
Case 2: **Supraventricular tachycardia (e.g. AVRT)**
(2) Tools for ECG monitoring / treatment strategy for SVT



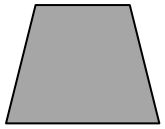
Case 3: **Paroxysmal atrial fibrillation**
(3) Diagnosis and management (4 pillars of the temple)



Case 4: **Digoxin intoxication**
(4) Tips and tricks for the safe use of digoxin



Case 5: **Sick Sinus Syndrome**
(5) PM only for symptomatic patients / PM systems



Case 6: **Rhythm management in heart failure patients**
(6) Guideline based, but individualized treatment plans

1. Brugada J, et al. 2019 ESC GL SVT. EHJ 2020;41:655-720. 2. Van Gelder IC, et al. 2024 ESC GL on AF. EHJ 2024;45:3314-414. 3. Zeppenfeld K, et al. 2022 ESC GL on VA & SCD. EHJ 2022;43:3997-4126. 4. Glikson M, et al. ESC GL PM and CRT. EHJ 2021;42:3427-520. 5. McDonagh TA, et al. ESC GL CHF. EHJ 2021;42:355-726. 6. McDonagh TA, et al. EHJ 2023;44:3627-39.