Τί πρέπει να γνωρίζει ο πνευμονολόγος για τα καρδιαγγειακά νοσήματα

Κώστας Τσιούφης

Πρόεδρος Ελληνικής Καρδιολογικής (2017-18)

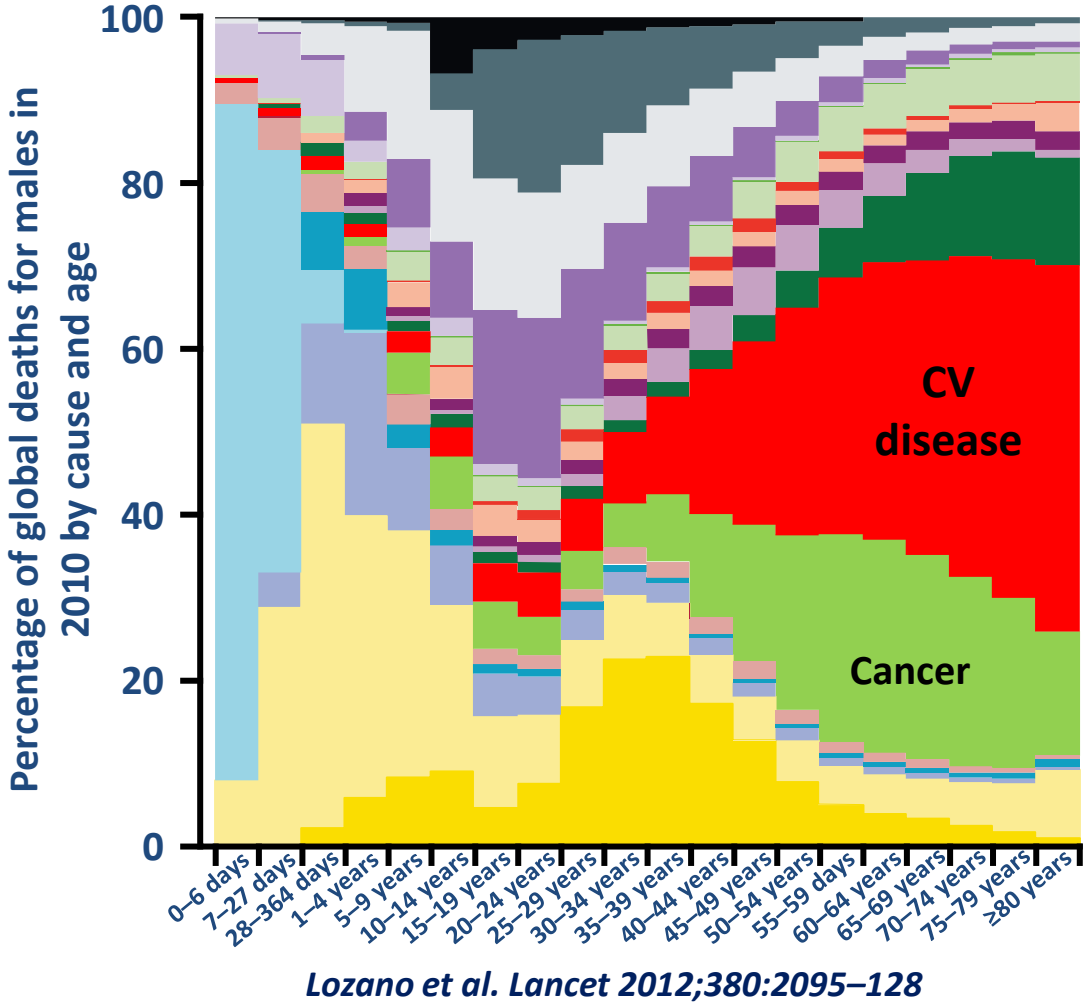
President of European Society of Hypertension (2017-19)

Δήλωση σύγκρουσης συμφερόντων

- **Καμία σύγκρουση συμφερόντων για τη συγκεκριμένη ομιλία**

✓ Έχω λάβει υποστήριξη συμμετοχής σε συνέδρια ή ερευνητική υποστήριξη ή τιμητική αμοιβή ομιλίας από Medtronic, St. Jude Medical, Bayer, Novartis, Astra-Zeneca, Boehringer In, Pfizer, Chiesi, Pharmanel, Sanofi, Vianex, Win-Medica, Elpen, Menarini

Globally, CVD is the most common non-communicable cause of death



Lozano et al. Lancet 2012;380:2095–128

ASCVD should be a public health priority

Globally, COPD is the fourth commonest cause of death

>By 2020 COPD will be ranked 3rd cause of death

> Pts with COPD die mainly from non-respiratory disease

25% die from CVD

20-33% from Cancer

> COPD is the comorbidity that most often delays the correct diagnosis of CHF

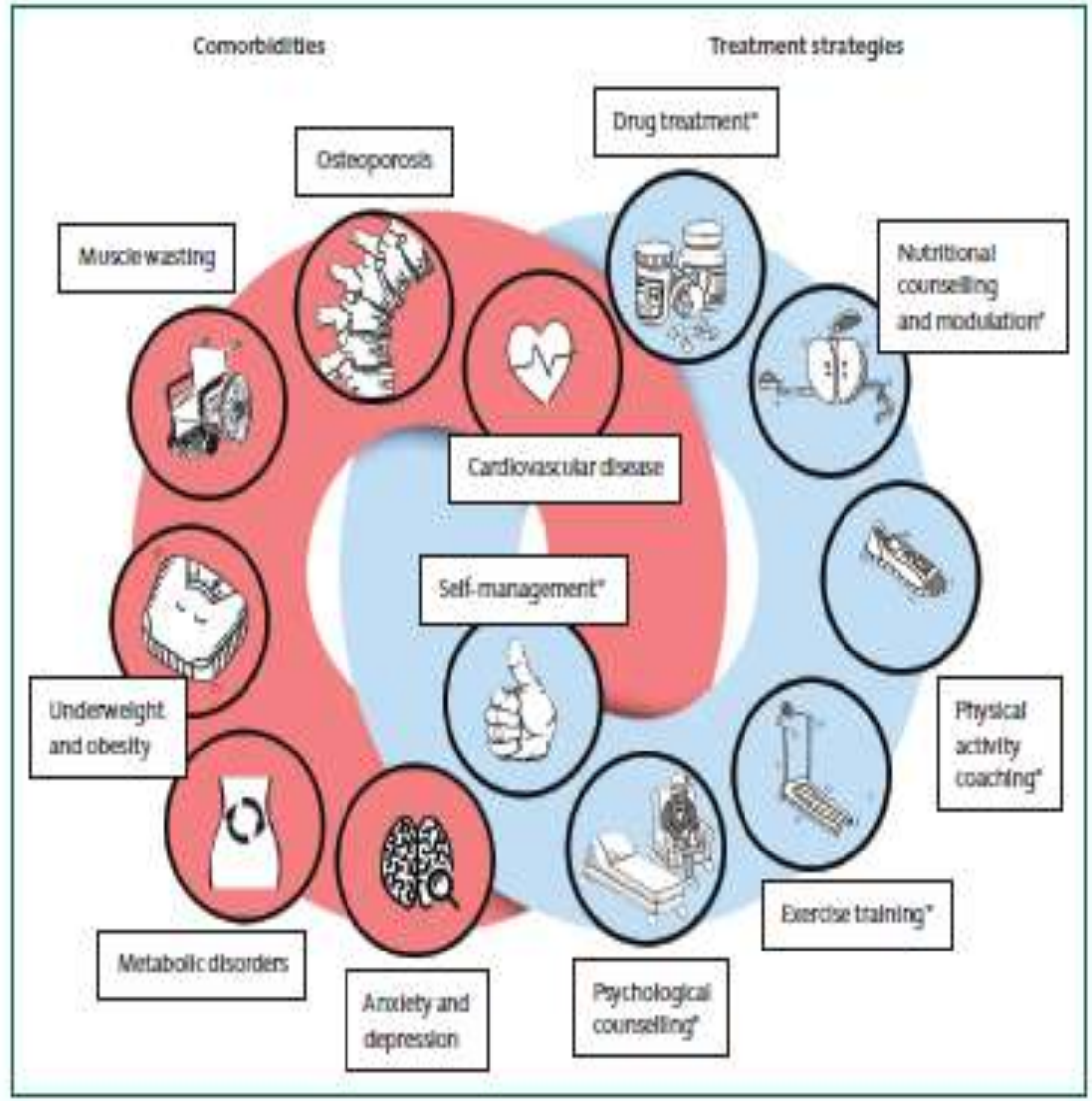
Cardiovascular Comorbidities

A Common Occurrence With COPD

- 2 to 3 times increased risk of CVD
- CVD comorbidities frequently undiagnosed and untreated
  - Most common CVDs: HTN, IHD, HF, AF
- Risk factors similar to those in general population: smoking, aging

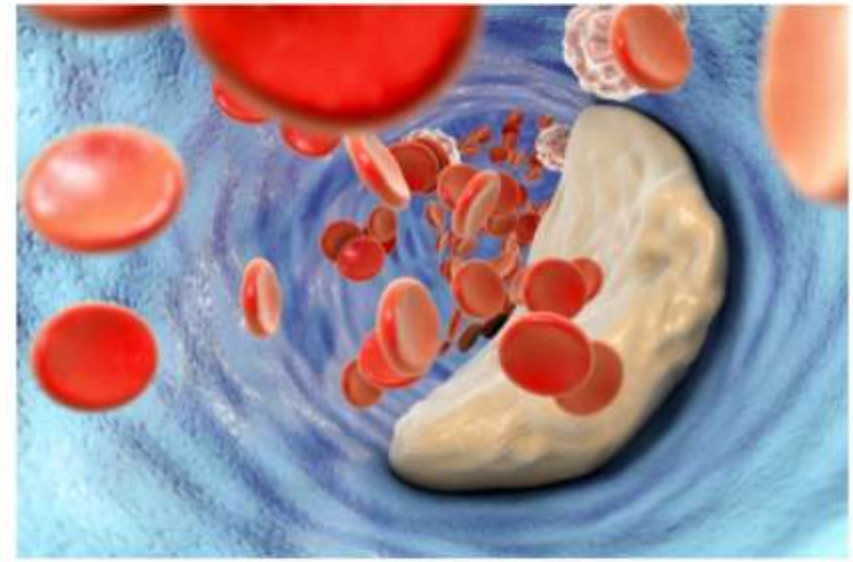
Onishi K. *J Cardiol.* 2017;70:128-134.

COPD phenotype



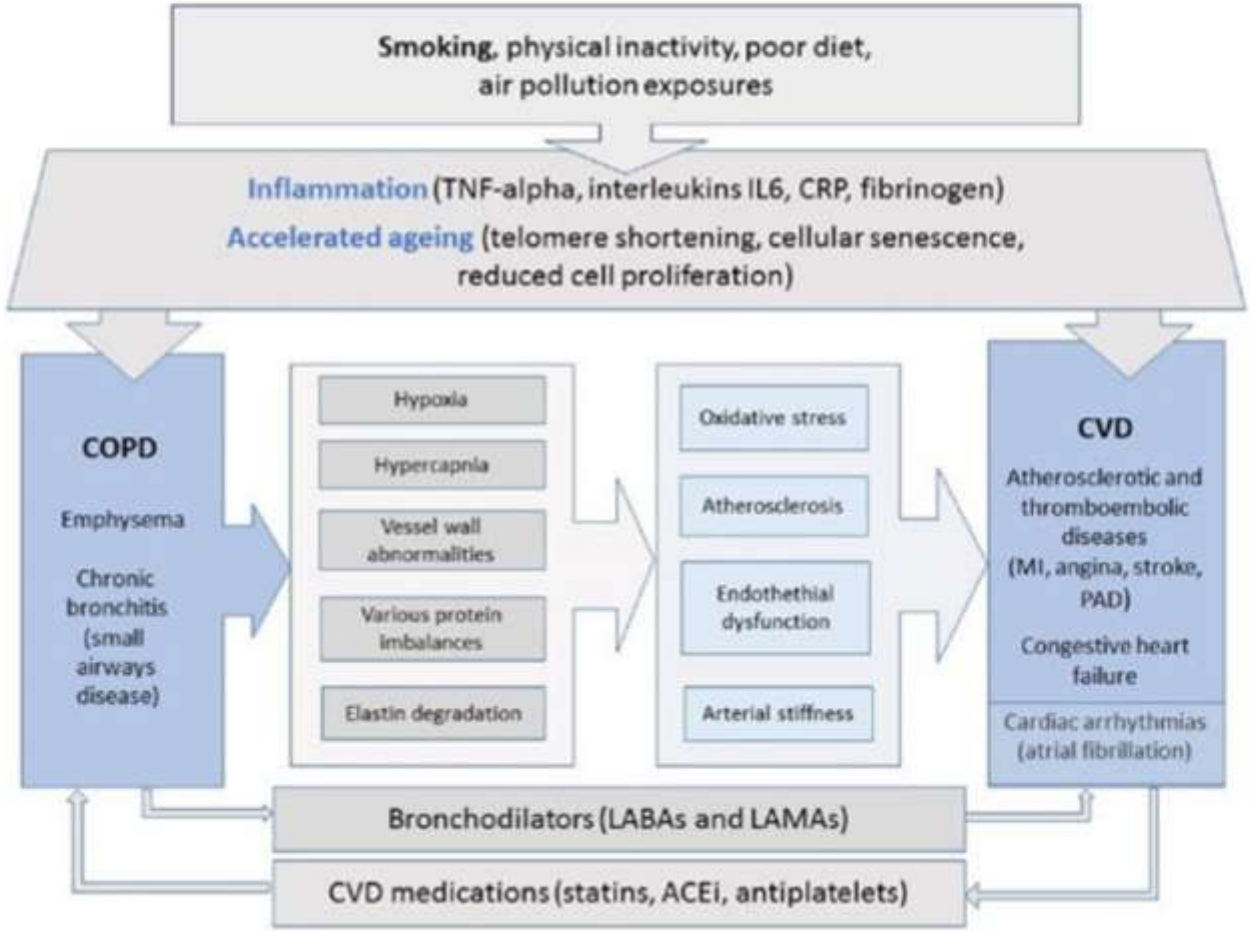
COPD and CVD
How Are They Linked?

- COPD: an independent risk factor for CVD
- Inflammation can lead to atherosclerotic plaque formation and progression of atherosclerosis[a]
  - Increased levels of circulating chemokines, cytokines, and acute phase reactants[b]
  - Platelet activation[c]



a. Libby P, et al. *Circulation.* 2002;105:1135-1143; b. Gan WQ, et al. *Thorax.* 2004;59:574-580; c. Maclay JD, et al. *Thorax.* 2011;66:769-774.

Biological Pathways and Mechanisms Linking COPD and CVD



COPD exacerbations further increase the CV risk

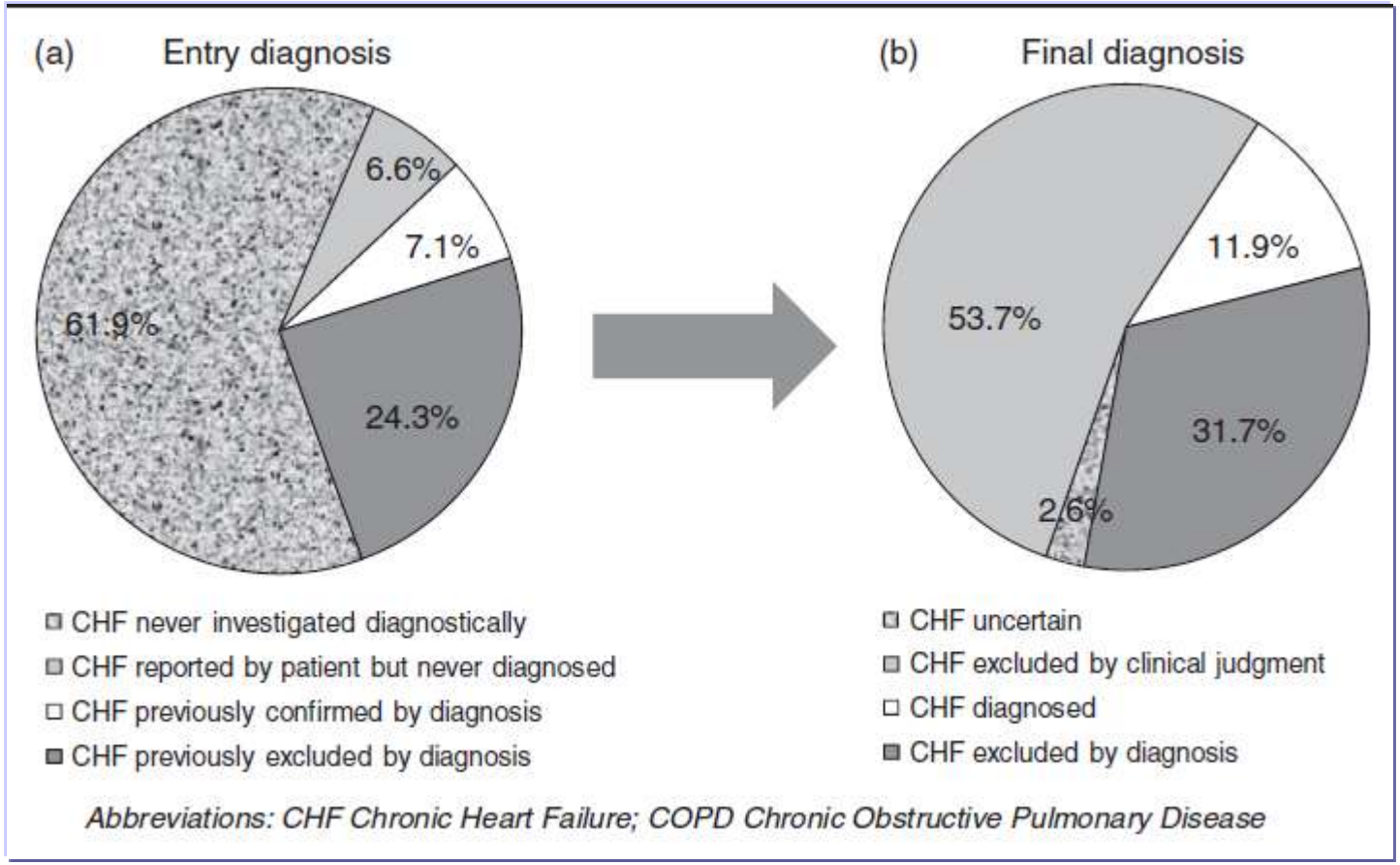
- Physical inactivity
- Hypoxia
- Tachycardia
- Increase in arterial stiffness
- Pulmonary hypertension
- Alterations in cardiac filling
- Increased platelet activation
- Use of high dose β2 agonists

COPD and CHF

Prevalence of coexisting COPD and CHF

SUSPIRIUM Registry

Presence of CHF as a comorbidity in COPD patients (a) before and (b) after diagnostic assessment

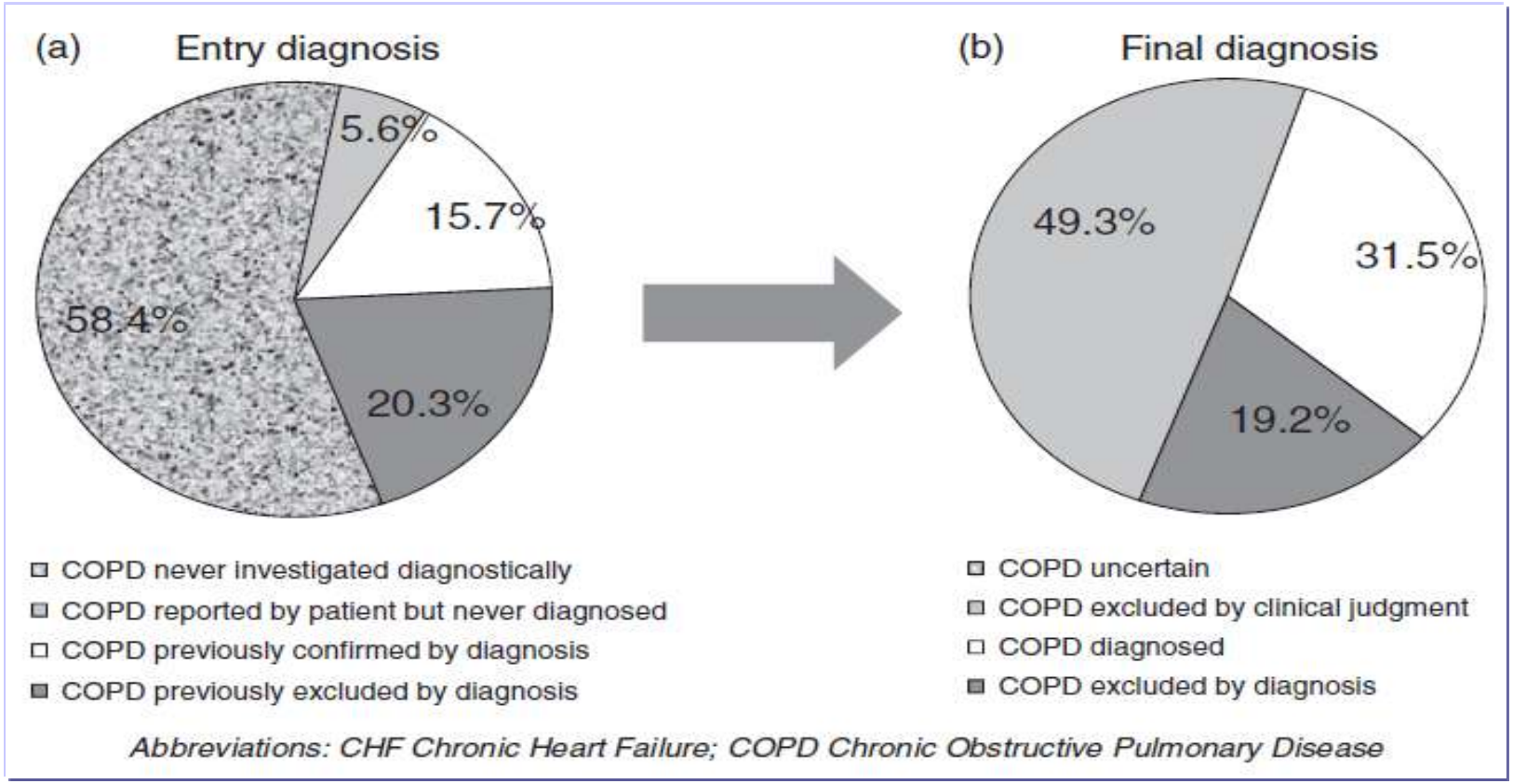


Griffo et al, European Journal of Preventive Cardiology 2017

Prevalence of coexisting COPD and CHF

SUSPIRIUM Registry

Presence of COPD as a comorbidity in CHF patients (a) before and (b) after diagnostic assessment



Griffo et al, European Journal of Preventive Cardiology 2017

Table 3.1 Definition of heart failure with preserved (HFpEF), mid-range (HFmrEF) and reduced ejection fraction (HFrEF)

Type of HF		HFrEF	HFmrEF	HFpEF
CRITERIA	1	Symptoms ± Signs[a]	Symptoms ± Signs[a]	Symptoms ± Signs[a]
	2	LVEF <40%	LVEF 40–49%	LVEF ≥50%
	3	–	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction (for details see Section 4.3.2).	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction (for details see Section 4.3.2).

BNP = B-type natriuretic peptide; HF = heart failure; HFmrEF = heart failure with mid-range ejection fraction; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; LAE = left atrial enlargement; LVEF = left ventricular ejection fraction; LVH = left ventricular hypertrophy; NT-proBNP = N-terminal pro-B type natriuretic peptide.

[a]Signs may not be present in the early stages of HF (especially in HFpEF) and in patients treated with diuretics.

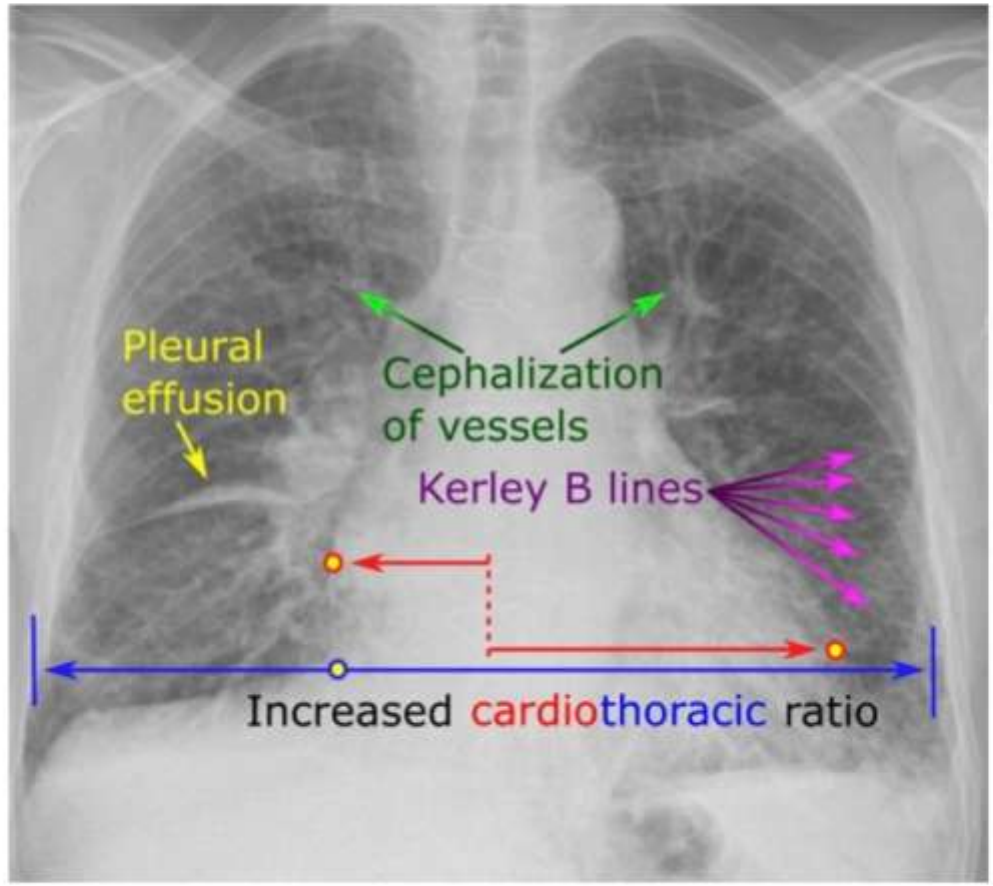
[b]BNP>35 pg/ml and/or NT-proBNP>125 pg/mL

Table 3.4 Aetiologies of heart failure

DISEASED MYOCARDIUM		
Ischaemic heart disease	Myocardial scar	
	Myocardial stunning/hibernation	
	Epicardial coronary artery disease	
	Abnormal coronary microcirculation	
	Endothelial dysfunction	
Toxic damage	Recreational substance abuse	Alcohol, cocaine, amphetamine, anabolic steroids.
	Heavy metals	Copper, iron, lead, cobalt.
	Medications	Cytostatic drugs (e.g. anthracyclines), immunomodulating drugs (e.g. interferons monoclonal antibodies such as trastuzumab, cetuximab), antidepressant drugs, antiarrhythmics, non-steroidal anti-Inflammatory drugs, anaesthetics.
	Radiation	
Immune-mediated and inflammatory damage	Related to infection	Bacteria, spirochaetes, fungi, protozoa, parasites (Chagas disease), rickettsiae, viruses (HIV/AIDS).
	Not related to infection	Lymphocytic/giant cell myocarditis, autoimmune diseases (e.g. Graves' disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hypersensitivity and eosinophilic myocarditis (Churg–Strauss).
Infiltration	Related to malignancy	Direct infiltrations and metastases.
	Not related to malignancy	Amyloidosis, sarcoidosis, haemochromatosis (iron), glycogen storage diseases (e.g. Pompe disease), lysosomal storage diseases (e.g. Fabry disease).
Metabolic derangements	Hormonal	Thyroid diseases, parathyroid diseases, acromegaly, GH deficiency, hypercortisolaemia, Conn's disease, Addison disease, diabetes, metabolic syndrome, phaeochromocytoma, pathologies related to pregnancy and peripartum.
	Nutritional	Deficiencies in thiamine, L-carnitine, selenium, iron, phosphates, calcium, complex malnutrition (e.g. malignancy, AIDS, anorexia nervosa), obesity.
Genetic abnormalities	Diverse forms	HCM, DCM, LV non-compaction, ARVC, restrictive cardiomyopathy (for details see respective expert documents), muscular dystrophies and laminopathies.
ABNORMAL LOADING CONDITIONS		
Hypertension		
Valve and myocardium structural defects	Acquired	Mitral, aortic, tricuspid and pulmonary valve diseases.
	Congenital	Atrial and ventricular septum defects and others (for details see a respective expert document).
Pericardial and endomyocardial pathologies	Pericardial	Constrictive pericarditis Pericardial effusion
	Endomyocardial	HES, EMF, endocardial fibroelastosis.
High output states		Severe anaemia, sepsis, thyrotoxicosis, Paget's disease, arteriovenous fistula, pregnancy.
Volume overload		Renal failure, iatrogenic fluid overload.
ARRHYTHMIAS		
Tachyarrhythmias		Atrial, ventricular arrhythmias.
Bradyarrhythmias		Sinus node dysfunctions, conduction disorders.

ARVC = arrhythmogenic right ventricular cardiomyopathy; DCM = dilated cardiomyopathy; EMF = endomyocardial fibrosis; GH = growth hormone; HCM = hypertrophic cardiomyopathy; HES = hypereosinophilic syndrome; HIV/AIDS = human immunodeficiency virus/acquired immune deficiency syndrome; LV = left ventricular.

Hidden Heart Failure



Assessing Patients With COPD and CVD
Echocardiogram

- ECHO (2D and 3D): noninvasive, readily available
- ECHO can identify:
  - LV contractile performance issues
  - Decreased ejection fraction
  - Problems with underlying diastolic dysfunction
  - Abnormalities in heart valves
  - Abnormalities in wall motion that may indicate a previous MI

Assessing Patients With COPD and CVD
Echocardiogram and PAH

- Mild elevation in PA pressures common with COPD
  - Mean PAP usually not > 35 mm Hg[a]
- ~5% of patients have arterial vasculopathy[a]
  - ECHO can differentiate patients with PAH from those without PAH
  - ECHO can determine which patients need right heart catheterization

a. Chaouat A, et al. *Eur Respir J*. 2008;32:1371-1385.

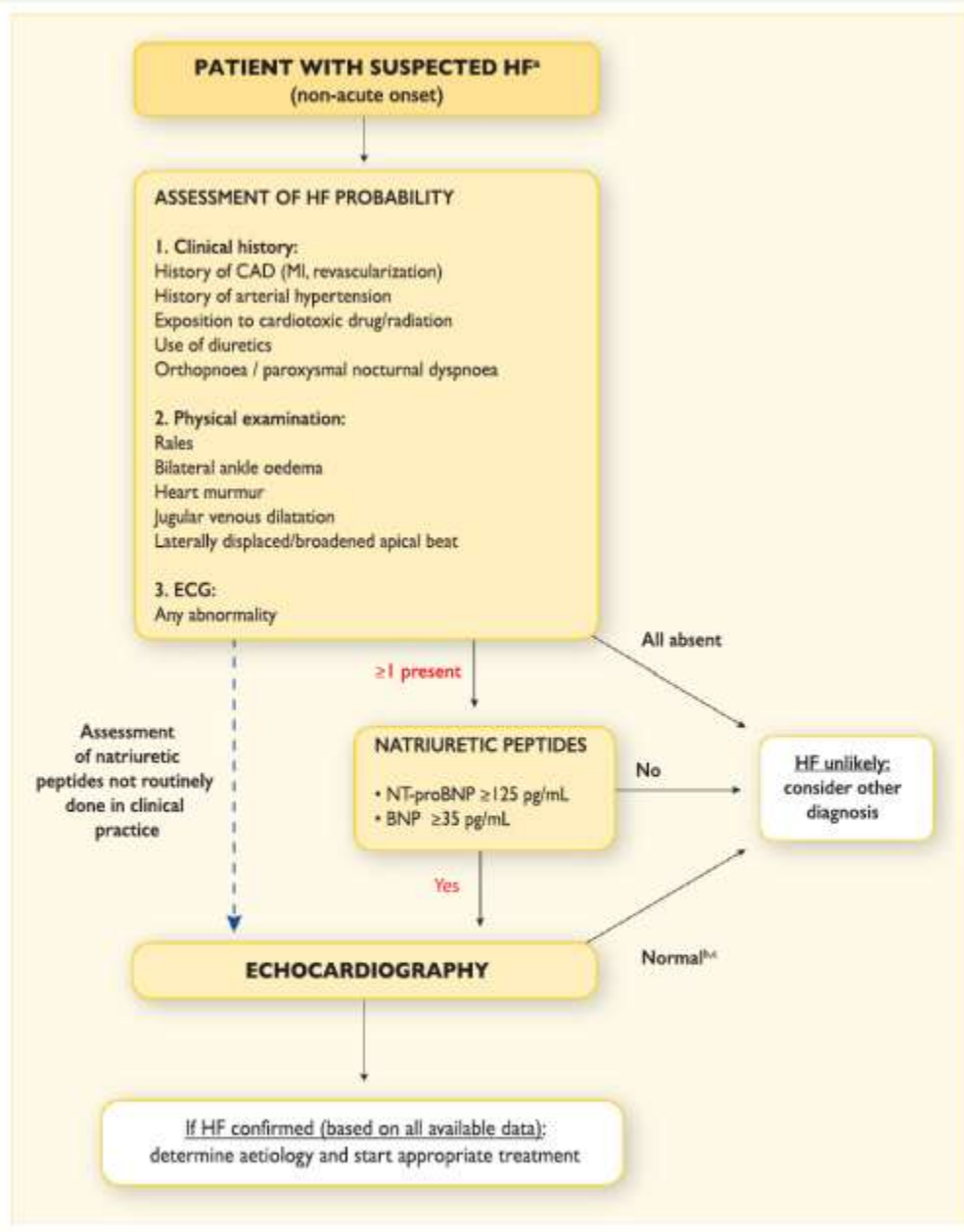


Figure 4.1 Diagnostic algorithm for a diagnosis of heart failure of non-acute onset
BNP = B-type natriuretic peptide; CAD = coronary artery disease; HF = heart failure; MI = myocardial infarction; NT-proBNP = N-terminal pro-B type natriuretic peptide.
[a]Patient reporting symptoms typical of HF (see *Table* 4.1).
[b]Normal ventricular and atrial volumes and function.
[c]Consider other causes of elevated natriuretic peptides (*Table* 12.3).

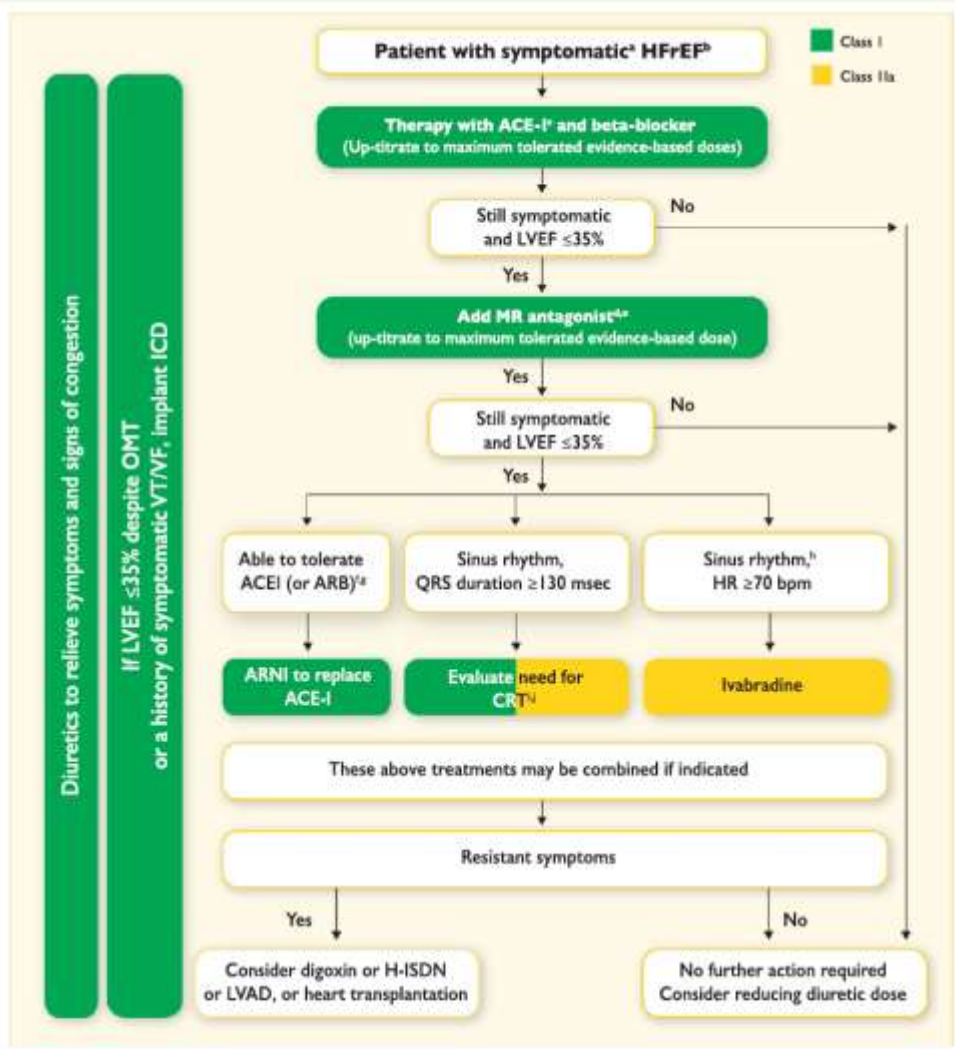
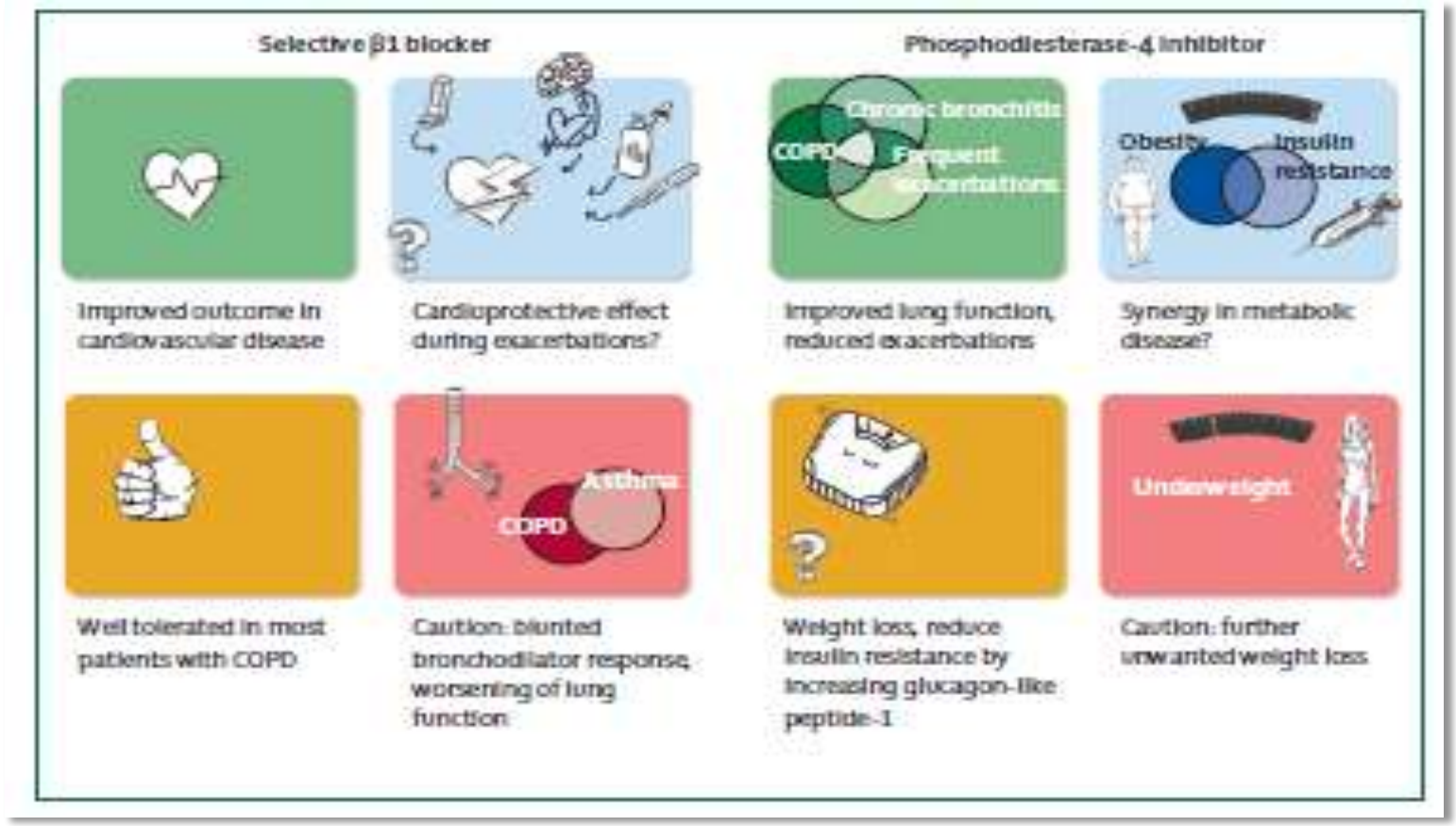


Figure 7.1 Therapeutic algorithm for a patient with symptomatic heart failure with reduced ejection fraction. Green indicates a class I recommendation; yellow indicates a class IIa recommendation. ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor neprilysin inhibitor; BNP = B-type natriuretic peptide; CRT = cardiac resynchronization therapy; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; H-ISDN = hydralazine and isosorbide dinitrate; HR = heart rate; ICD = implantable cardioverter defibrillator; LBBB = left bundle branch block; LVAD = left ventricular assist device; LVEF = left ventricular ejection fraction; MR = mineralocorticoid receptor; NT-proBNP = N-terminal pro-B type natriuretic peptide; NYHA = New York Heart Association; OMT = optimal medical therapy; VF = ventricular fibrillation; VT = ventricular tachycardia. [a]Symptomatic = NYHA Class II-IV. [b]HFrEF = LVEF <40%. [c]If ACE inhibitor not tolerated/contra-indicated, use ARB. [d]If MR antagonist not tolerated/contra-indicated, use ARB. [e]With a hospital admission for HF within the last 6 months or with elevated natriuretic peptides (BNP > 250 pg/ml or NTproBNP > 500 pg/ml in men and 750 pg/ml in women). [f]With an elevated plasma natriuretic peptide level (BNP ≥ 150 pg/mL or plasma NT-proBNP ≥ 600 pg/mL, or if HF hospitalization within recent 12 months plasma BNP ≥ 100 pg/mL or plasma NT-proBNP ≥ 400 pg/mL). [g]In doses equivalent to enalapril 10 mg b.i.d. [h]With a hospital admission for HF within the previous year. [i]CRT is recommended if QRS ≥ 130 msec and LBBB (in sinus rhythm). [j]CRT should/may be considered if QRS ≥ 130 msec with non-LBBB (in a sinus rhythm) or for patients in AF provided a strategy to ensure bi-ventricular capture in place (individualized decision). For further details, see Sections 7 and 8 and corresponding web pages.

Two examples of drug therapy with potential benefits and harms on outcomes of both COPD and other comorbidities



Intended drug effects are shown in green boxes, considerations in orange boxes, potential beneficial effects on comorbidity in blue boxes, and potential harms in red boxes.

COPD and HTN

Hypertension and COPD
Facts!

HTN is the most frequent comorbidity in patients with COPD

Coincidence of the two diseases may affect 2.5% of the adult population

Both conditions share similar environmental risks and, in addition, hypoxia may exacerbate risk.

Treatment of COPD with anticholinergic agents and long-acting beta-2 adrenoceptor agonists may adversely affect the CV system (increase heart rate and BP).

Are we all Hypertensive? And if so, why?

1. Genetics

2. Obesity

3. Immobility

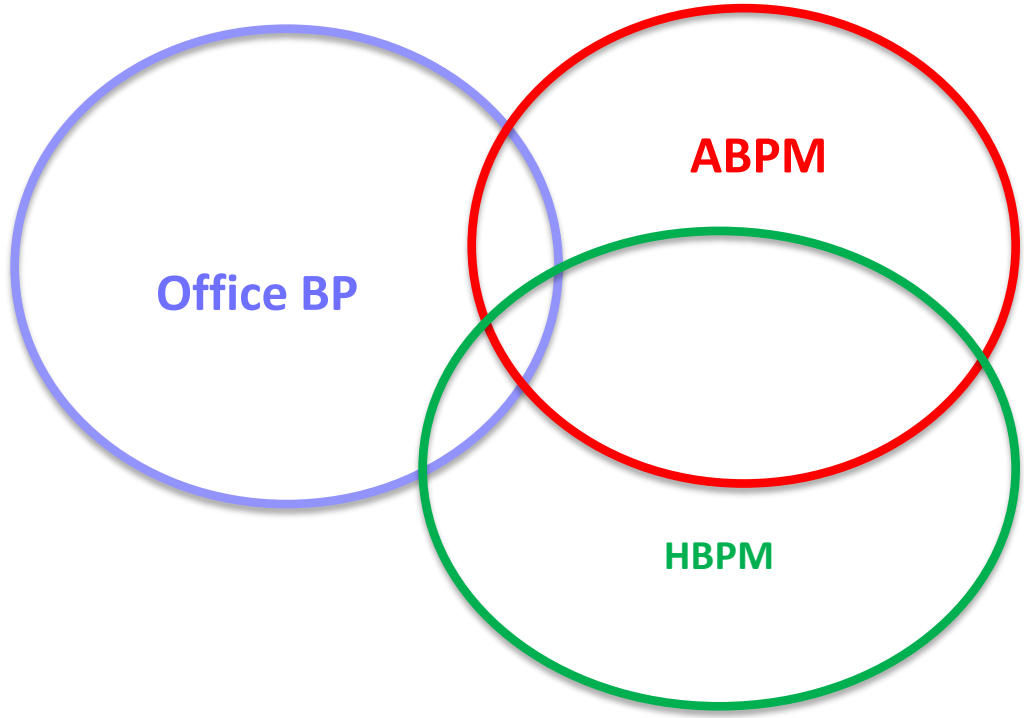
4. Alcohol

5. Nutrition

What is a Normal Blood Pressure? The Yanomani Indians

Diagnosis of HTN	=	Correct BP Measurement

Which BP should be used?



2018 ESC/ESH Guidelines for the management of arterial hypertension

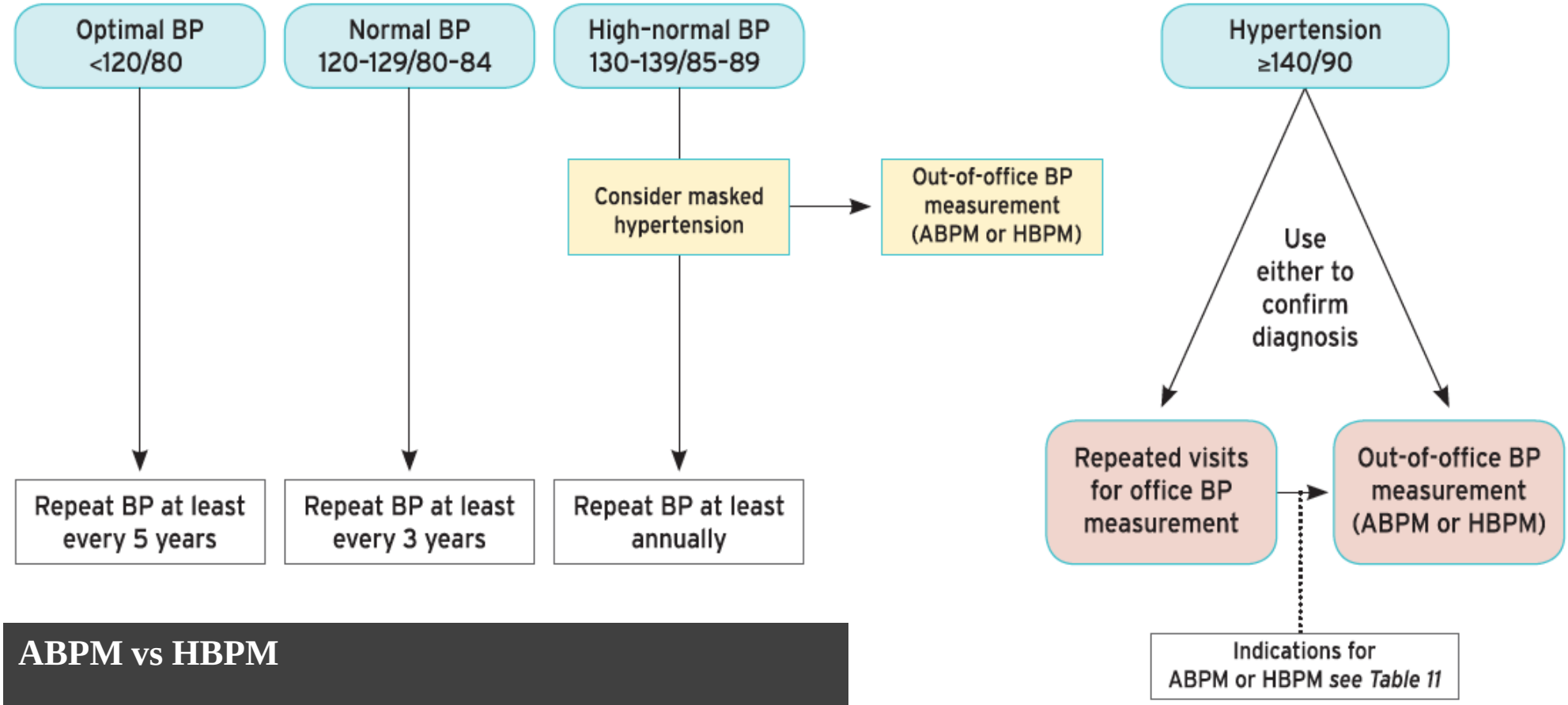
The Task Force for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension

Authors/Task Force Members: Bryan Williams (ESC Chairperson) (UK)*, Giuseppe Mancia (ESH Chairperson) (Italy)*, Wilko Spiering (The Netherlands), Enrico Agabiti Rosei (Italy), Michel Azizi (France), Michel Burnier (Switzerland), Denis L. Clement (Belgium), Antonio Coca (Spain), Giovanni de Simone (Italy), Anna Dominiczak (UK), Thomas Kahan (Sweden), Felix Mahfoud (Germany), Josep Redon (Spain), Luis Ruilope (Spain), Alberto Zanchetti (Italy)†, Mary Kerins (Ireland), Sverre E. Kjeldsen (Norway), Reinhold Kreutz (Germany), Stephane Laurent (France), Gregory Y.H. Lip (UK), Richard McManus (UK), Krzysztof Narkiewicz (Poland), Frank Ruschitzka (Switzerland), Roland E. Schmieder (Germany), Evgeny Shlyakhto (Russia), Costas Tsioufis (Greece), Victor Aboyans (France), and Ileana Desormais (France)

Classification of office BP and definitions of hypertension grade

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	< 120	and	< 80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥ 180	and/or	≥ 110
Isolated systolic hypertension	≥ 140	and	< 90

Screening and diagnosis of hypertension



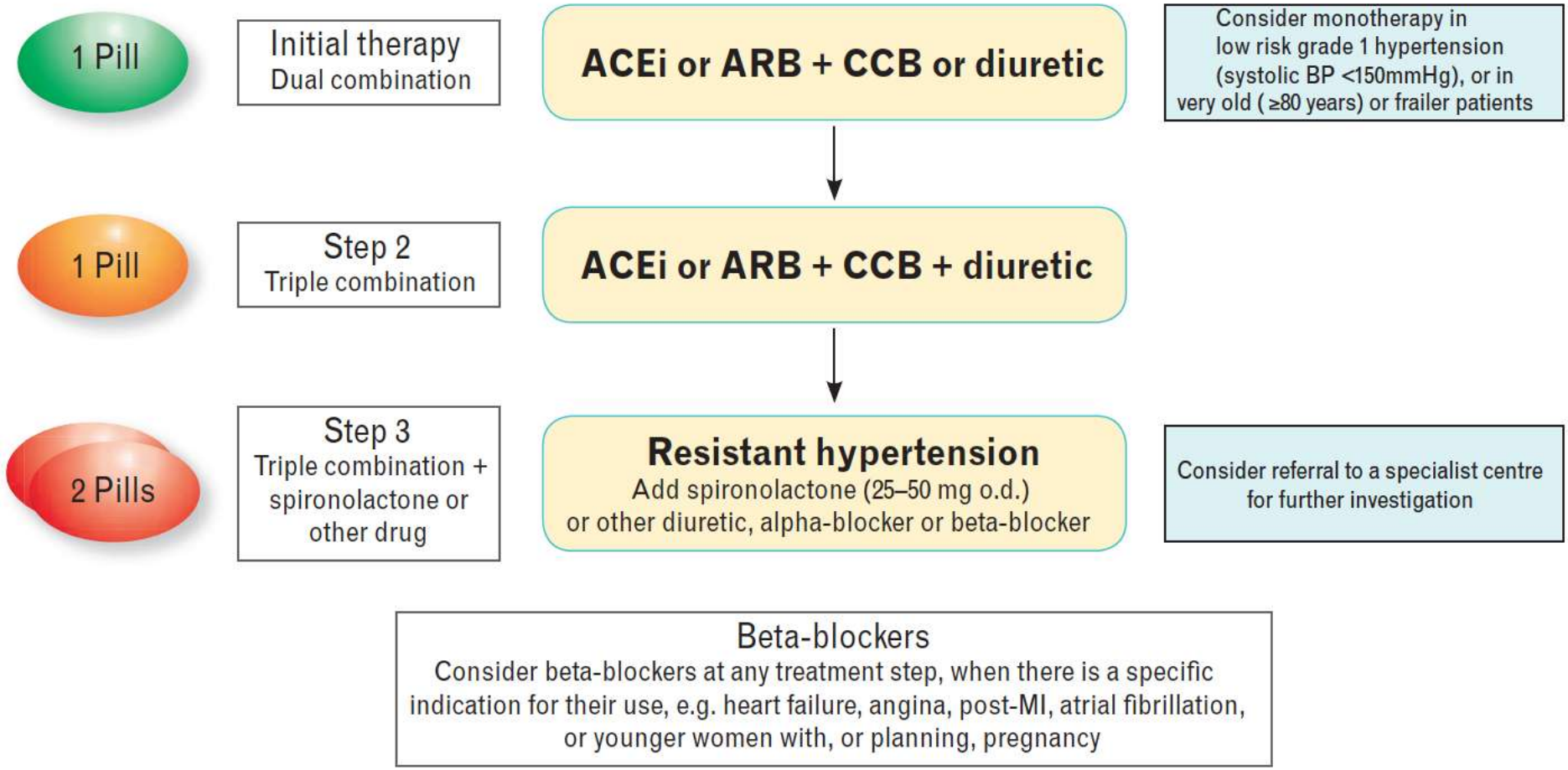
ABPM vs HBPM

"In general these methods should be regarded as complementary rather then absolute alternatives"

Office BP treatment target range

Age group	Office SBP treatment target ranges (mmHg)					Diastolic treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke/TIA	
18–65 years	**Target to 130** *or lower if tolerated* Not < 120	**Target to 130** *or lower if tolerated* Not < 120	**Target to < 140 to 130** *if tolerated*	**Target to 130** *or lower if tolerated* Not < 120	**Target to 130** *or lower if tolerated* Not < 120	< 80 to 70
65–79 years	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	< 80 to 70
≥ 80 years	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	**Target to < 140 to 130** *if tolerated*	< 80 to 70
Diastolic treatment target range(mmHg)	< 80 to 70	< 80 to 70	< 80 to 70	< 80 to 70	< 80 to 70	

Core drug-treatment strategy for uncomplicated hypertension



The core algorithm is also appropriate for most patients with HMOD, cerebrovascular disease, diabetes, or PAD

Hypertension and COPD
Selection of antihypertensive drugs vs their effects on pulmonary function

Management of hypertensive patients with COPD should include lifestyle changes, among which cessation of smoking is

essential.

- CCBs, ARBs or ACEIs, or the CCB/RAS blocker combination

are recommended as the initial drugs of choice.

If the BP response is poor, or depending on other comorbidities, thiazides or thiazide-like diuretics and beta1-selective beta-blockers can be considered.

Hypertension and COPD
Selection of antihypertensive drugs vs their effects on pulmonary function

beta-blockers

- May negatively affect the reduced basal lung function

May diminish the effectiveness of emergency beta-agonist administration,

May reduce the benefit of long acting beta-agonist treatment, and make the discrimination of asthma and COPD more difficult.

But

- there is evidence that in COPD these drugs maintain their CV-protective effects

Diuretic

- May decrease the plasma level of potassium

(in addition to the hypokalaemic effects of glucocorticoids andbeta2-adrenoceptor agonists),

May worsen carbon dioxide retention

(including metabolic alkalosis-related hypoxia in hypoventilated patients),

May increase haematocrit, and deteriorate mucus secretion in bronchi

We work together

ESH and the 35 National Hypertension EU societies and the 5 Associated Hypertension societies

We work together

ESH and the 191 ***Excellence Centres***

Blood Pressure Control in ESH Excellence Centers

BP-CON study

To determine

1. **Average BP values in different ESH Excellent Centers, countries and European regions (attended and unattended BP)**

2. **Prevalence of uncontrolled hypertensive patients** and analyze the most important **factors/predictors for treatment failure** in ESH Excellence Centers.

3. Preparation of **strategies aiming to increase hypertension control** globally but with specificities for particular countries/regions

Time frame: December 2018-December 2019

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Hypertension and Cardiovascular Prevention Clinic, 3rd Department of Internal Medicine, Evangelismos Hospital, Athens	Dr. Andreadis, Emmanuel
Hypertension Center of Sotiria Hospital, Athens	Prof. Stergiou George S.
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Hypertension Center, Department of Nephrology, University Hospital of Ioannina, Ioannina	Dr. Kalaitzidis, Rigas
Hypertension Centre, Department of Clinical Therapeutics, Medical School of Athens, Alexandra Hospital, Athens	Prof E.Manios
Hypertension Clinic, Cardiology Department, Asklepeion Hospital, Athens	Prof. Manolis, Athansios
Hypertension Clinic, Department of Cardiology, KAT General Hospital, Athens	Dr Grassos, Charalampos
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European Society of Hypertension

ESH Athens 2022

32ND EUROPEAN MEETING ON HYPERTENSION AND CARDIOVASCULAR PROTECTION

JUNE 17-22, 2022 - ATHENS, GREECE

MEGARON ATHENS INTERNATIONAL CONFERENCE CENTRE (MAICC)


European Society of Hypertension
6th Official pre-ESH Congress
Satellite Symposium
UPDATE ON
DIFFICULT TO CONTROL
HYPERTENSION
& RELATED
COMORBIDITIES
29-30 MARCH 2019
Benaki Museum Amphitheater
138 Piraeus Street, ATHENS
Chairman: C. Tsioufis
Co-chairman: R.E. Schmieder

ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
ΙΔΡΥΘΕΝ ΤΟ 1837

ΣΧΟΛΗ ΕΠΙΣΤΗΜΩΝ ΥΓΕΙΑΣ
ΤΜΗΜΑ ΙΑΤΡΙΚΗΣ

ΠΡΟΓΡΑΜΜΑ ΜΕΤΑΠΤΥΧΙΑΚΩΝ ΣΠΟΥΔΩΝ

ΑΡΤΗΡΙΑΚΗ ΥΠΕΡΤΑΣΗ ΚΑΙ ΣΥΝΟΔΑ ΚΑΡΔΙΑΓΓΕΙΑΚΑ - ΝΕΦΡΙΚΑ ΝΟΣΗΜΑΤΑ

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Βιβλιοθήκη Ιπποκράτειου Γ.Ν.Α., Έβρου 63-67, 115-27, Αθήνα

COPD and AF

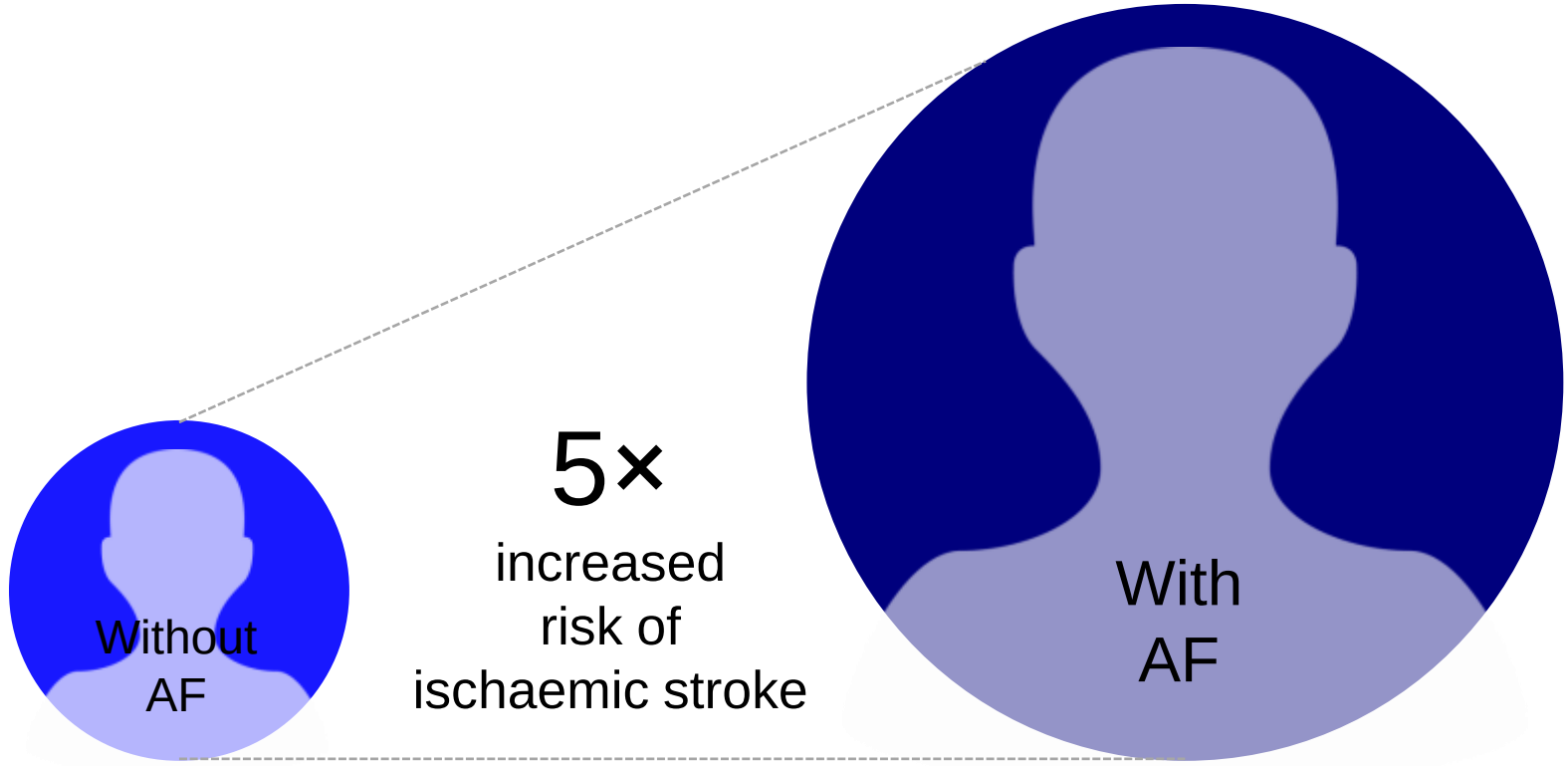
Atrial Fibrillation and Multi-focal Atrial Tachycardia in Patients With COPD

- Many patients with COPD have underlying structural changes predominantly to their atria
  - Atrial fibrillation is commonly observed in patients with COPD
  - β-1 blockers can help control heart rate
- Patients who present with COPD exacerbations are also at increased risk for multi-focal atrial tachycardia

Falk JA, et al. *Proc Am Thorac* Soc. 2008;5:543-548.

Patients with AF have about fivefold increased risk of ischaemic stroke versus Patients with no AF

Framingham Heart Study (n=5070)



Risk ratio=4.8; $p<0.001$

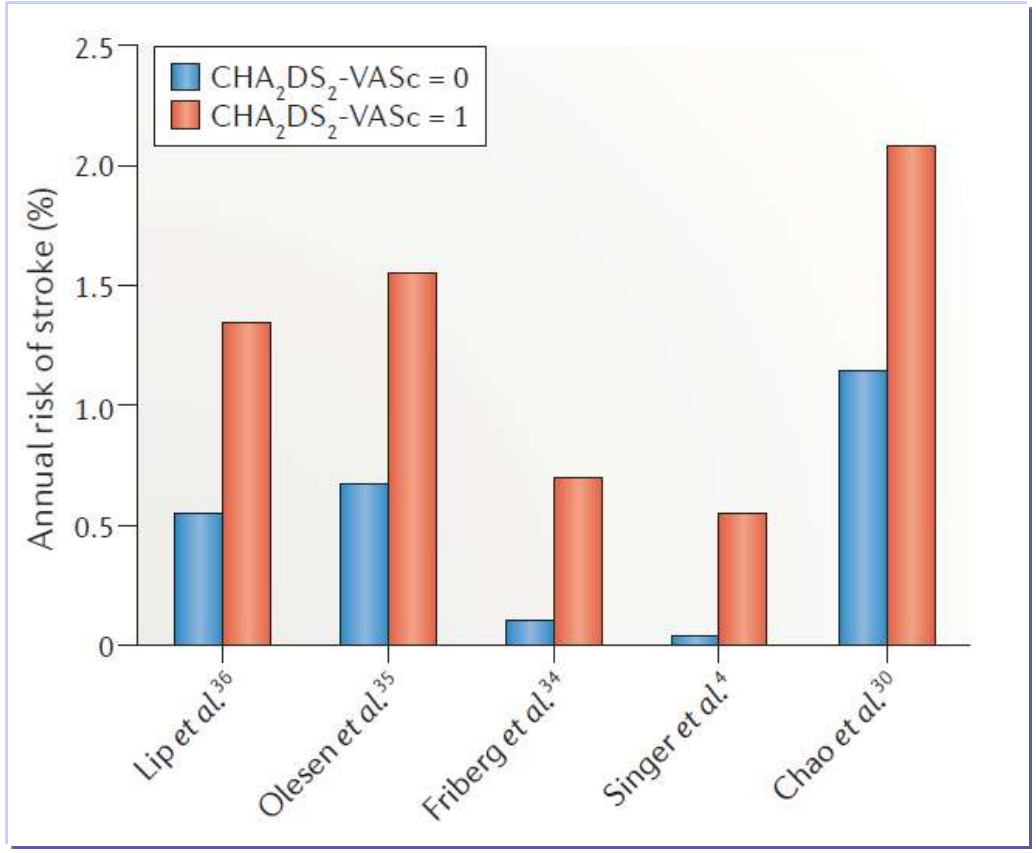
Wolf *et al*, 1991

Clinical risk factors for stroke, transient ischaemic attack, and systemic embolism

CHA_2DS_2-VASc risk factor	Points
Congestive heart failure Signs/symptoms of heart failure or objective evidence of reduced left-ventricular ejection fraction	1
Hypertension Resting blood pressure >140/90 mmHg on at least two occasions or current antihypertensive treatment	1
Age 75 years or older	2
Diabetes mellitus Fasting glucose >125 mg/dL (7 mmol/L) or treatment with oral hypoglycaemic agent and/or insulin	1
Previous stroke, transient ischaemic attack, or thromboembolism	2
Vascular disease Previous myocardial infarction, peripheral artery disease, or aortic plaque	1
Age 65–74 years	1
Sex category (female)	1

European Heart Journal - doi:10.1093/eurheartj/ehw210

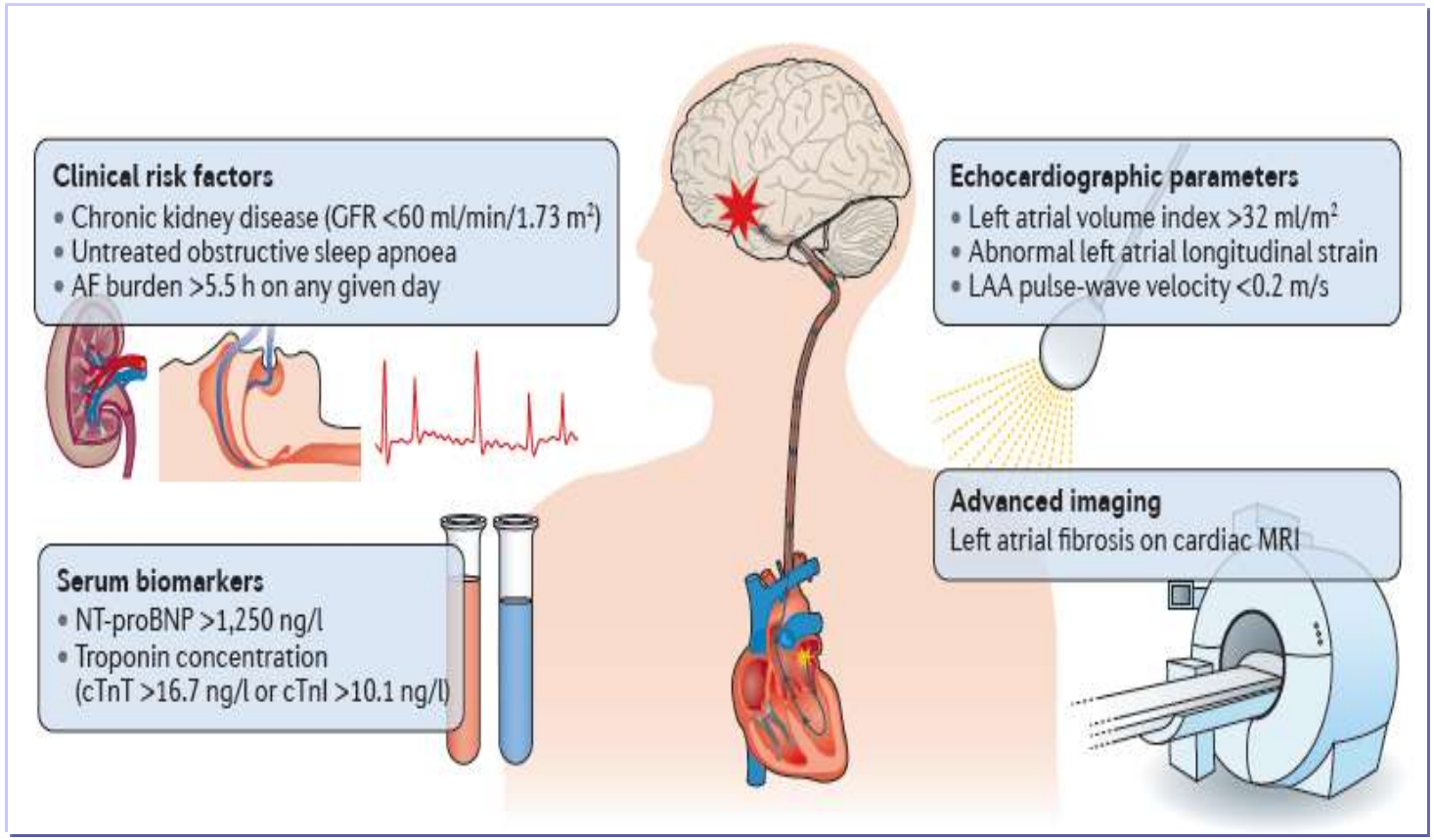
Annual risk of stroke in major studies according to CHA2DS2-VASc scores



Patients with a CHA2DS2 -VASc score = 0 are generally regarded to be at a very low risk of thromboembolism (consistently ≤1% annually). Stroke risk is more heterogeneous in patients with a score = 1 (a difference that is mainly owing to study methodology and end point definitions). However, annual stroke rates across studies are consistently ≤2% annually. For each study, the annual stroke rate over the longest available follow-up period is displayed.

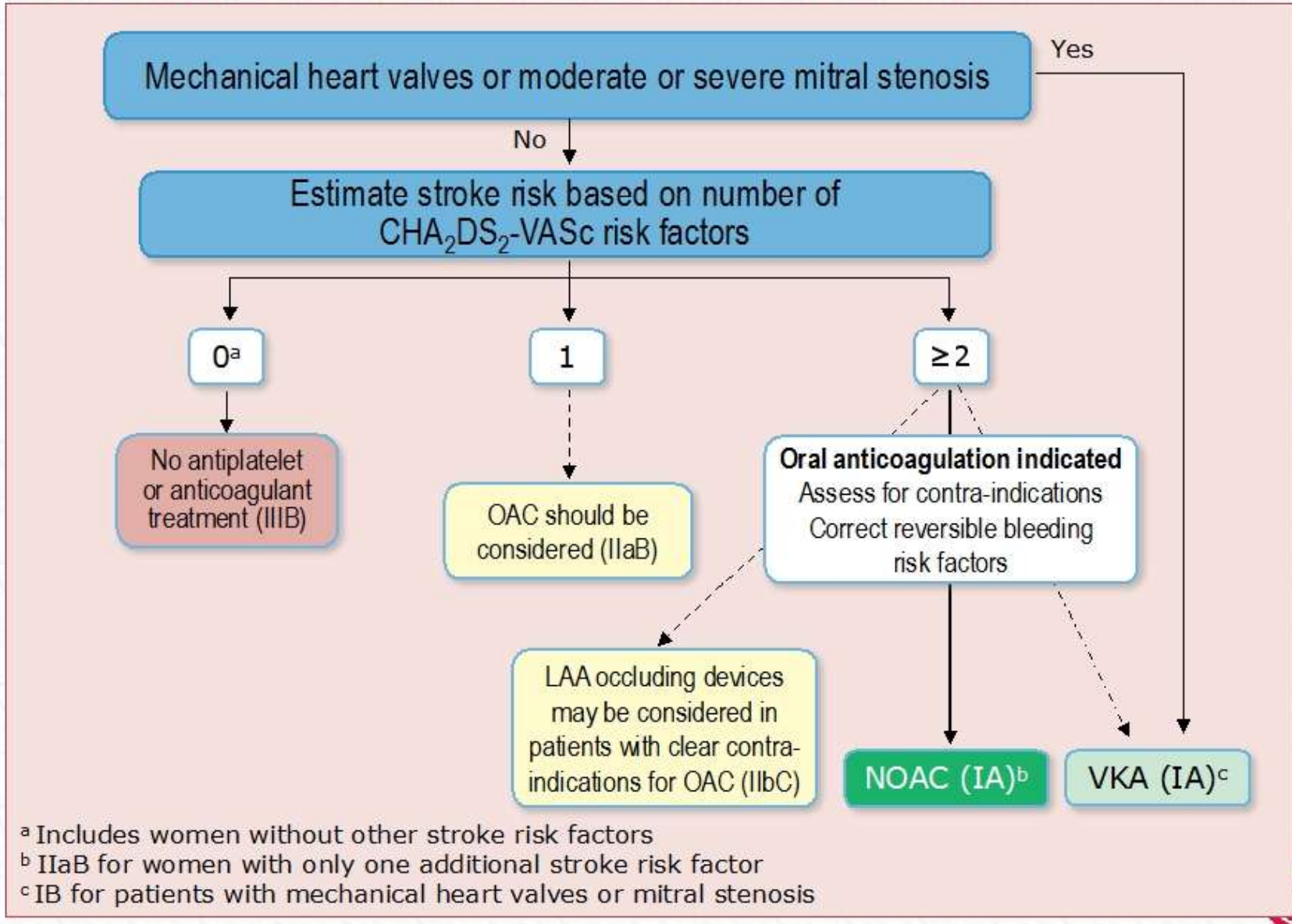
Nature Reviews Cardiology 2016;13:549-559

Novel markers of increased stroke risk in AF



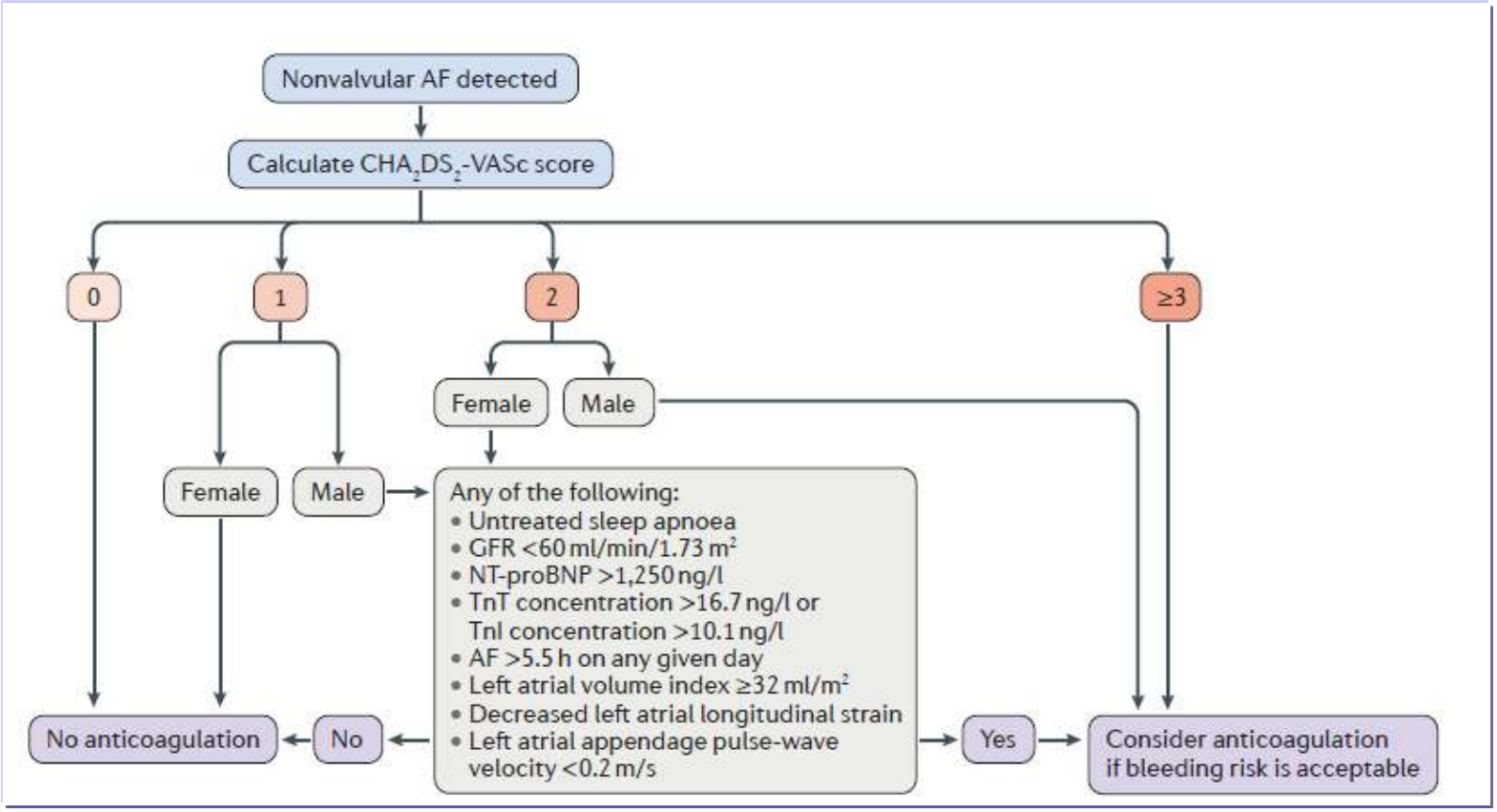
Nature Reviews Cardiology 2016;13:549-559

Stroke prevention in atrial fibrillation



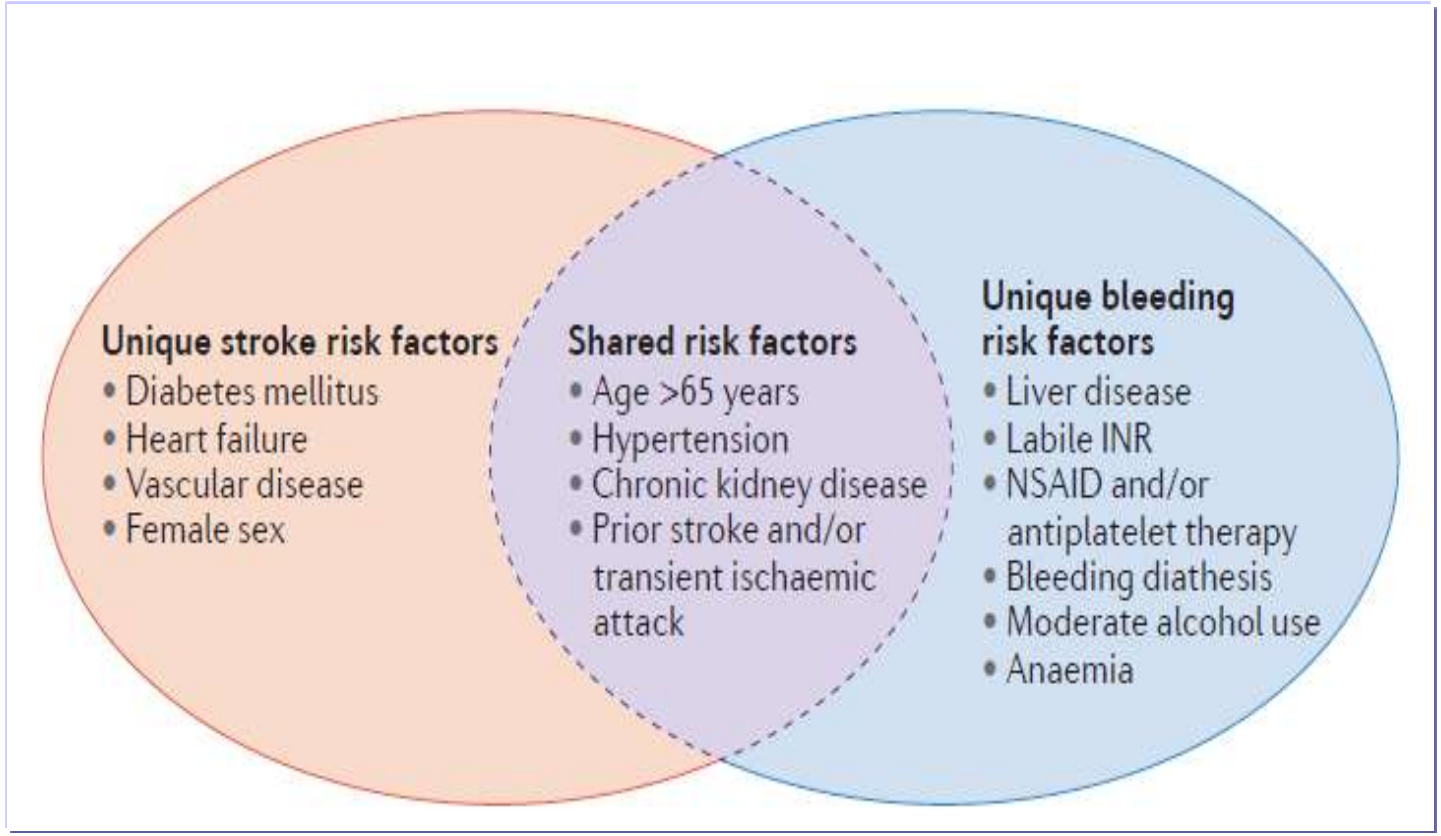
European Heart Journal - doi:10.1093/eurheartj/ehw210

Proposed algorithm for stroke risk assessment in atrial fibrillation (AF). Male patients with a CHA2DS2-VASc score = 1 or female patients with a CHA2DS2-VASc score = 2 are frequently borderline candidates for anticoagulation. The presence of novel stroke risk factors determines a higher stroke risk, and might favour anticoagulation.



Nature Reviews Cardiology 2016;13:549-559

Unique and shared risk factors for bleeding and stroke.



Nature Reviews Cardiology 2016;13:549-559

Πότε να διακοπεί το NOAC πριν από προγραμματισμένη χειρουργική παρέμβαση

Τελευταία λήψη φαρμάκου προ εκλεκτικής χειρουργικής παρέμβασης

	Dabigatran		Apixaban		Edoxaban *		Rivaroxaban	
	Χωρίς σημαντικό αιμορραγικό κίνδυνο και με δυνατότητα για τοπική αιμόσταση: παρεμβείτε στα χαμηλά επίπεδα (δηλ ≥12h ή 24h μετά την τελευταία λήψη)							
	Χαμηλού κινδύνου	Υψηλού κινδύνου	Χαμηλού κινδύνου	Υψηλού κινδύνου	Χαμηλού κινδύνου	Υψηλού κινδύνου	Χαμηλού κινδύνου	Υψηλού κινδύνου
CrCl ≥80 ml/min	≥24h	≥48h	≥24h	≥48h	Χωρίς δεδομένα	Χωρίς δεδομένα	≥24h	≥48h
CrCl 50–80 ml/min	≥36h	≥72h	≥24h	≥48h	Χωρίς δεδομένα	Χωρίς δεδομένα	≥24h	≥48h
CrCl 30–50 ml/min §	≥48h	≥96h	≥24h	≥48h	Χωρίς δεδομένα	Χωρίς δεδομένα	≥24h	≥48h
CrCl 15–30 ml/min §	Χωρίς ένδειξη	Χωρίς ένδειξη	≥36h	≥48h	Χωρίς δεδομένα	Χωρίς δεδομένα	≥36h	≥48h
CrCl <15 ml/min	Χωρίς επίσημη ένδειξη							

*no EMA approval yet.;

Europace 2013;15 (5): 625-651

Επεμβάσεις που δεν απαιτούν υποχρεωτικά διακοπή της αγωγής

Κάντε την παρέμβαση στις χαμηλές συγκεντρώσεις του NOAC. Λάβετε υπόψη τη διενέργεια 18-24 h μετά την τελευταία λήψη και επανέναρξη 6 h αργότερα

- Οδοντιατρικές επεμβάσεις
  - Εξαγωγή 1 έως 3 δοντιών
  - Παραδοντικές επεμβάσεις
  - Διάνοιξη αποστήματος
  - Τοποθέτηση εμφυτεύματος
- Οφθαλμολογία
  - Επέμβαση καταρράκτη ή γλαυκώματος
- Ενδοσκόπηση χωρίς χειρουργείο
- Επιφανειακή επέμβαση (π.χ. διάνοιξη αποστήματος, μικρές δερματολογικές επεμβάσεις)

Europace 2013;15 (5): 625-651

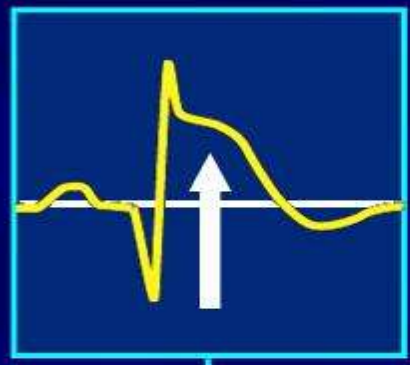
COPD and ACS

Ο ασθενής με οξύ στεφανιαίο σύνδρομο

Παθοφυσιολογικό υπόστρωμα

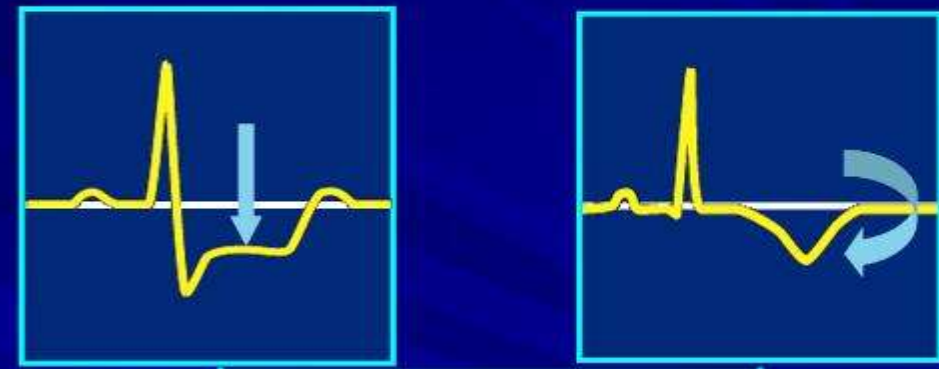
ACS:Spectrum of Clinical Presentation

ST-Elevation-MI



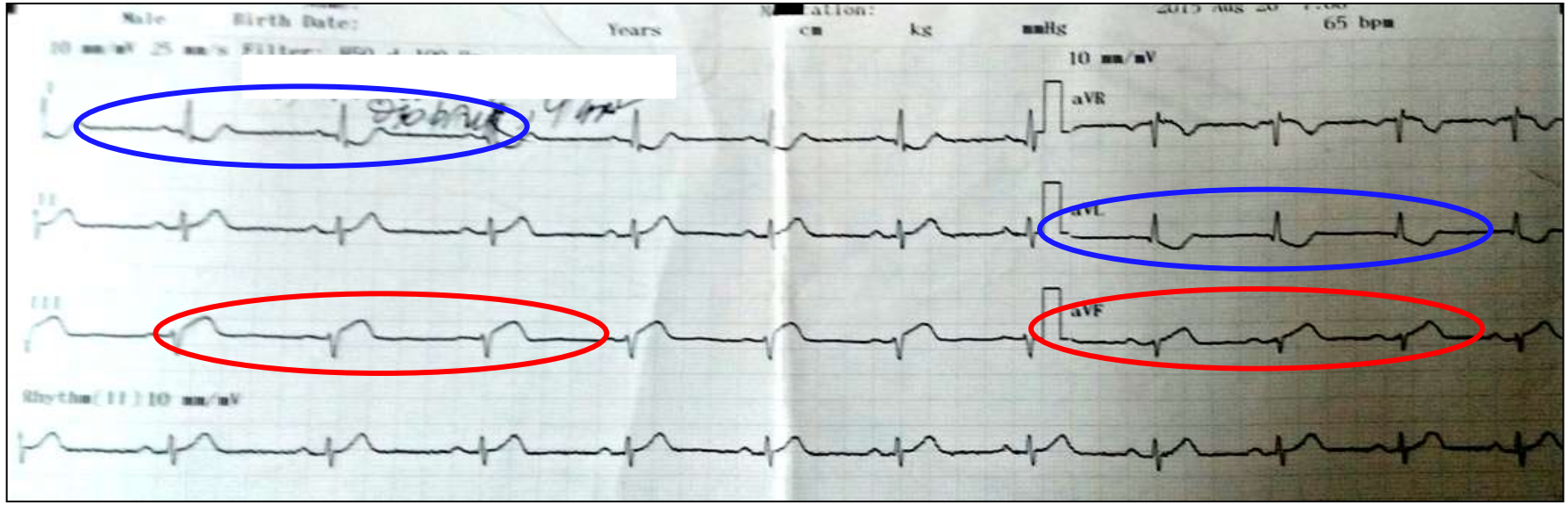
- Consistent Clinical Presentation
- Higher Short-Term Mortality
- More established Therapies
  - Direct PCI
  - Thrombolysis

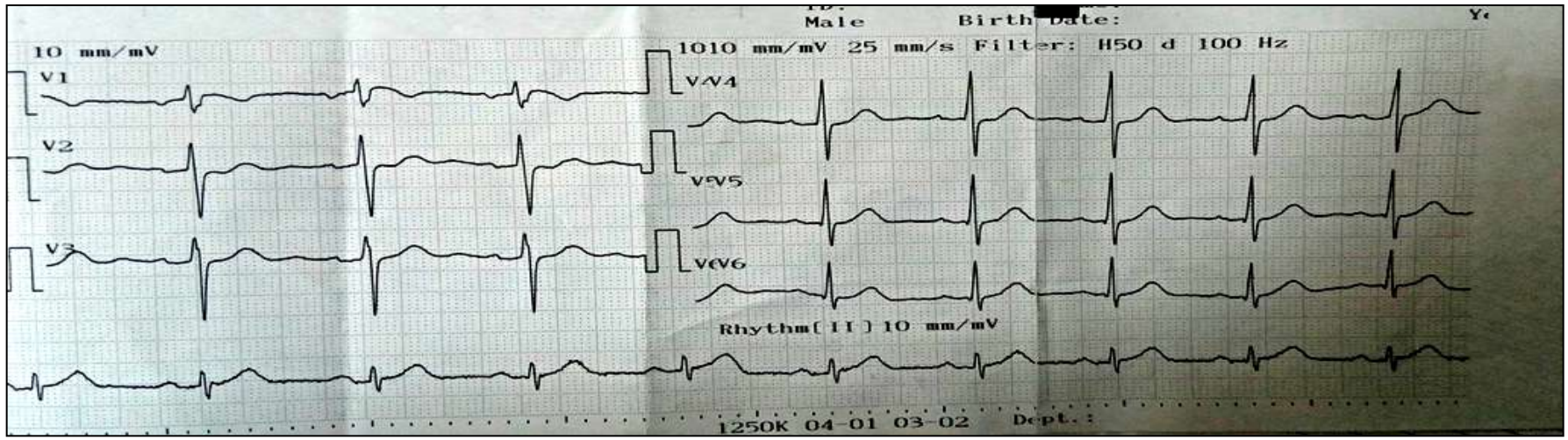
Non-ST-Elevation ACS



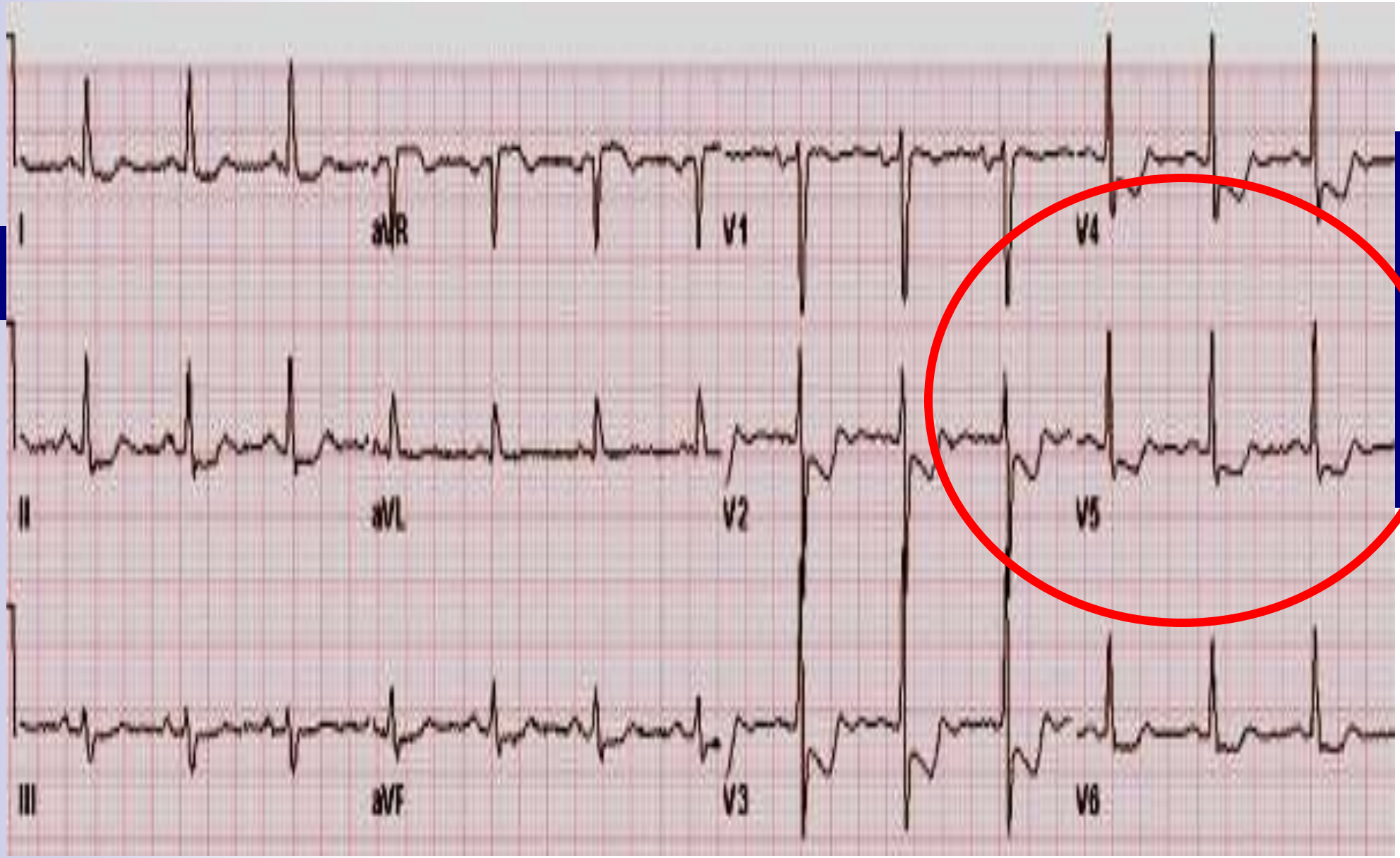
- Variable Clinical Presentation
- Acute Mortality Low, Long-term = High
- Therapy Varies Widely

Ανδρας ηλικίας 54 ετών καπνιστής, υπερτασικός και υπερλιπιδαιμικός παρουσίασε προκάρδιο συσφικτικό άλγος προ 30 λεπτών





Ανδρας ηλικίας 70 ετών με ισορικό ΣΔ, ΑΥ και υπερλιπιδαιμίας παρουσιάζει από 24ώρου βραχείας διάρκειας (3-5 λεπτά) επεισόδια στηθαγχικού πόνου στη ηρεμία



Elevations of Cardiac Troponin Values because of Myocardial Injury

Injury related to primary myocardial ischaemia

- Plaque rupture.
- Intraluminal coronary artery thrombus formation.

Injury related to supply/demand imbalance of myocardial ischaemia

- Tachy-/brady-arrhythmias.
- Aortic dissection or severe aortic valve disease.
- Hypertrophic cardiomyopathy.
- Cardiogenic, hypovolaemic, or septic shock.
- Severe respiratory failure.
- Severe anaemia.
- Hypertension with or without LVH.
- Coronary spasm.
- Coronary embolism or vasculitis.
- Coronary endothelial dysfunction without significant CAD.

Injury not related to myocardial ischaemia

- Cardiac contusion, surgery, ablation, pacing, or defibrillator shocks.
- Rhabdomyolysis with cardiac involvement.
- Myocarditis.
- Cardiotoxic agents, e.g. anthracyclines, herceptin.

Multifactorial or indeterminate myocardial injury

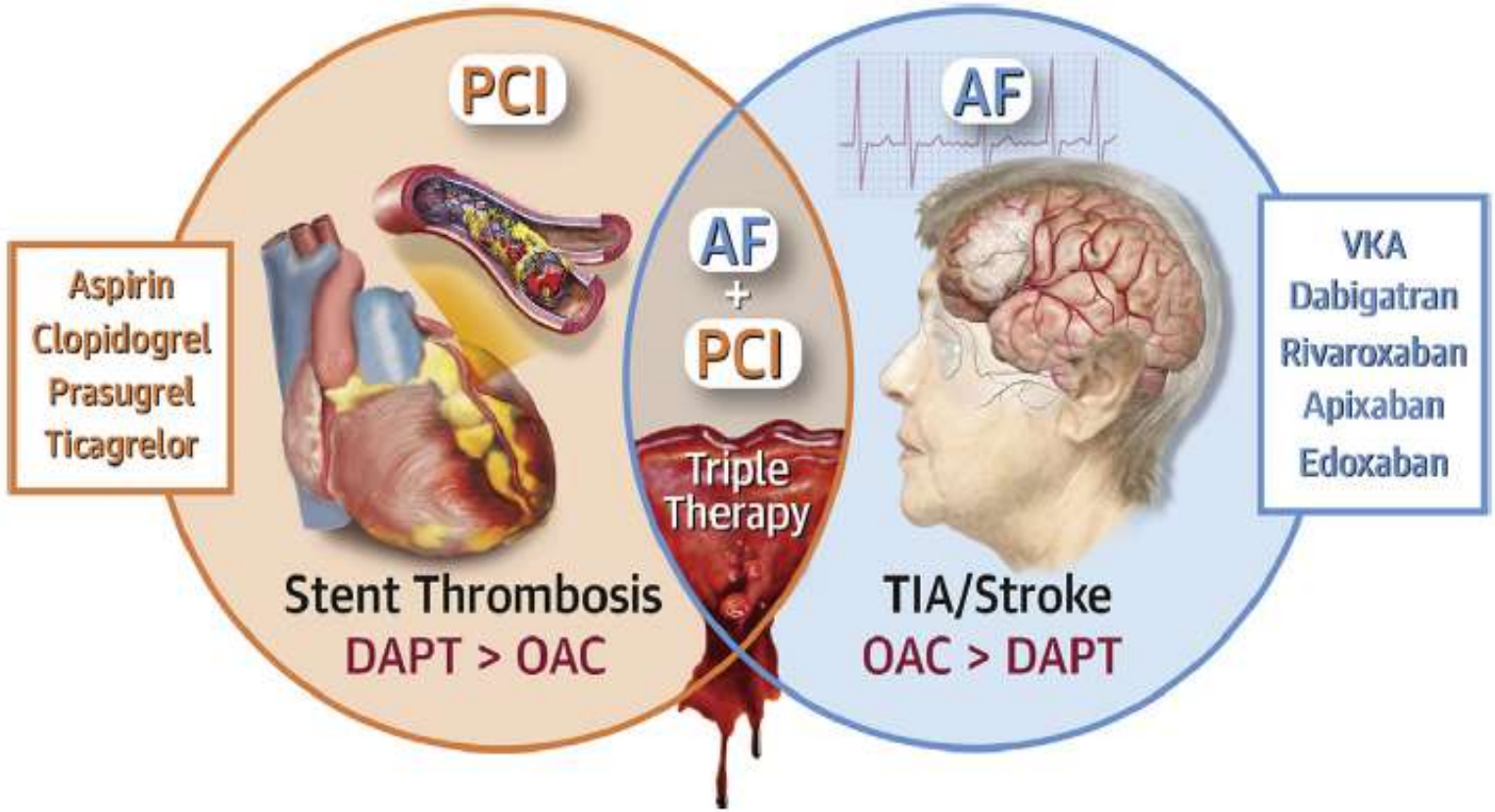
- Heart failure.
- Stress (Takotsubo) cardiomyopathy.
- Severe pulmonary embolism or pulmonary hypertension.
- Sepsis and critically ill patients.
- Renal failure.
- Severe acute neurological diseases, e.g. stroke, subarachnoid haemorrhage.
- Infiltrative diseases, e.g. amyloidosis, sarcoidosis.
- Strenuous exercise.

www.escardio.org/guidelines

European Heart Journal (2012) 33: 2551–2567
doi:10.1093/eurheartj/ehs184

EUROPEAN SOCIETY OF CARDIOLOGY®

FIGURE 1 Clinical Challenge in Patients With AF Undergoing PCI



Capodanno and Angiolillo JACC Intv 2017;10:1086

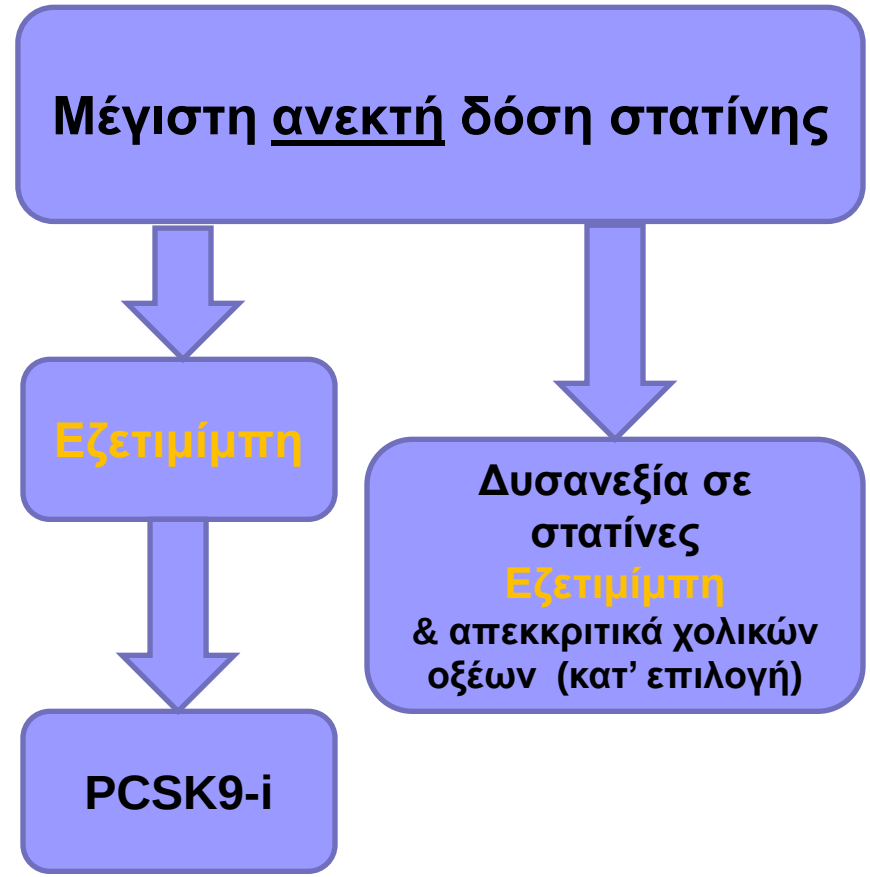
2016 ESC/EAS Guidelines for the Management of Dyslipidaemias

European Heart Journal (2016) **37**, 2999–3058
doi:10.1093/eurheartj/ehw272

Recommendations	Class [a]	Level [b]	Ref [c]
In patients at VERY HIGH CV risk[d], an LDL-C goal of <1.8 mmol/L (70 mg/dL) or a reduction of at least 50% if the baseline LDL-C[e] is between 1.8 and 3.5 mmol/L (70 and 135 mg/dL) is recommended.	I	B	61, 62, 65, 68, 69, 128
In patients at HIGH CV risk[d], an LDL-C goal of <2.6 mmol/L (100 mg/dL), or a reduction of at least 50% if the baseline LDL-C[e] is between 2.6 and 5.2 mmol/L (100 and 200 mg/dL) is recommended.	I	B	65, 129
In subjects at LOW or MODERATE risk[d] an LDL-C goal of <3.0 mmol/L (<115 mg/dL) should be considered.	IIa	C	-

Αλγόριθμος αντιμετώπισης

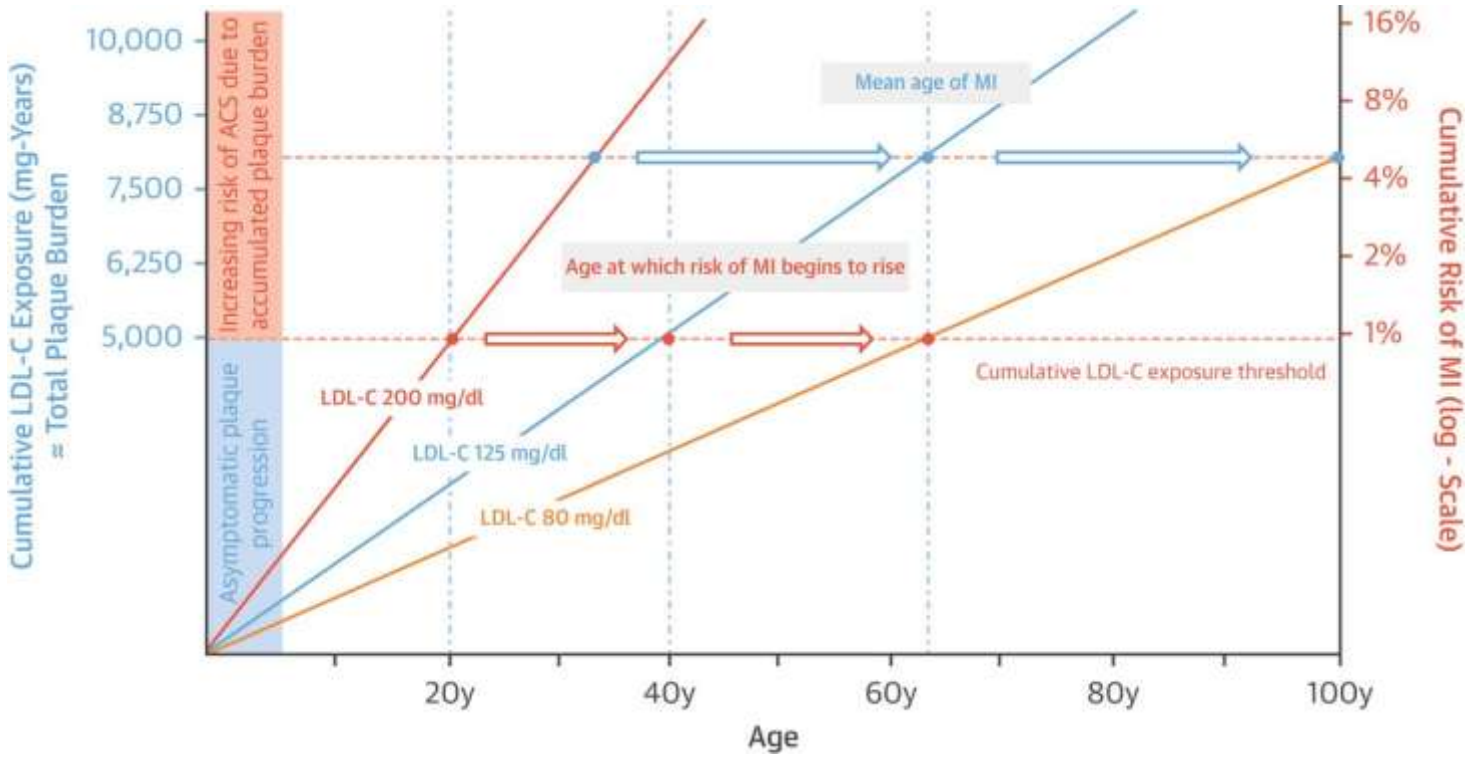
Recommendations	Class [a]	Level [b]
Prescribe statin up to the highest recommended dose or highest tolerable dose to reach the goal.	I	A
In the case of statin intolerance, ezetimibe or bile acid sequestrants, or these combined, should be considered.	IIa	C
If the goal is not reached, statin combination with a cholesterol absorption inhibitor should be considered.	IIa	B
If the goal is not reached, statin combination with a bile acid sequestrant may be considered.	IIb	C
In patients at very high-risk, with persistent high LDL-C despite treatment with maximal tolerated statin dose, in combination with ezetimibe or in patients with statin intolerance, a PCSK9 inhibitor may be considered.	IIb	C



European Heart Journal doi:10.1093/eurheartj/ehw272

Life time exposure to LDL matters? The younger the better?

Lower cumulative exposure LDL can slow plaque progression and delay the onset of myocardial infarction and other acute coronary syndromes

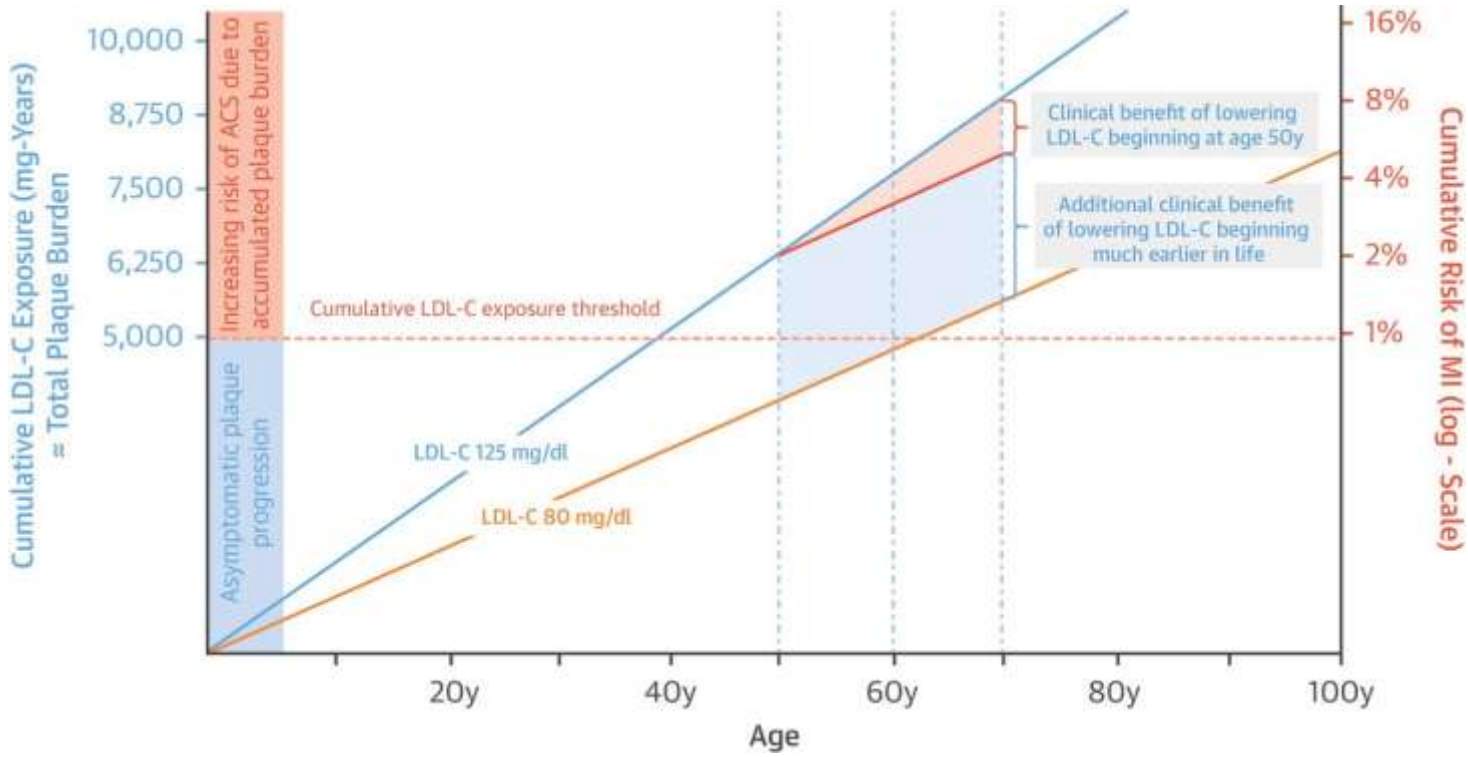


Brian A. Ference et al. JACC 2018;72:1141-1156

JACC

Life time exposure to LDL matters? The younger the better?

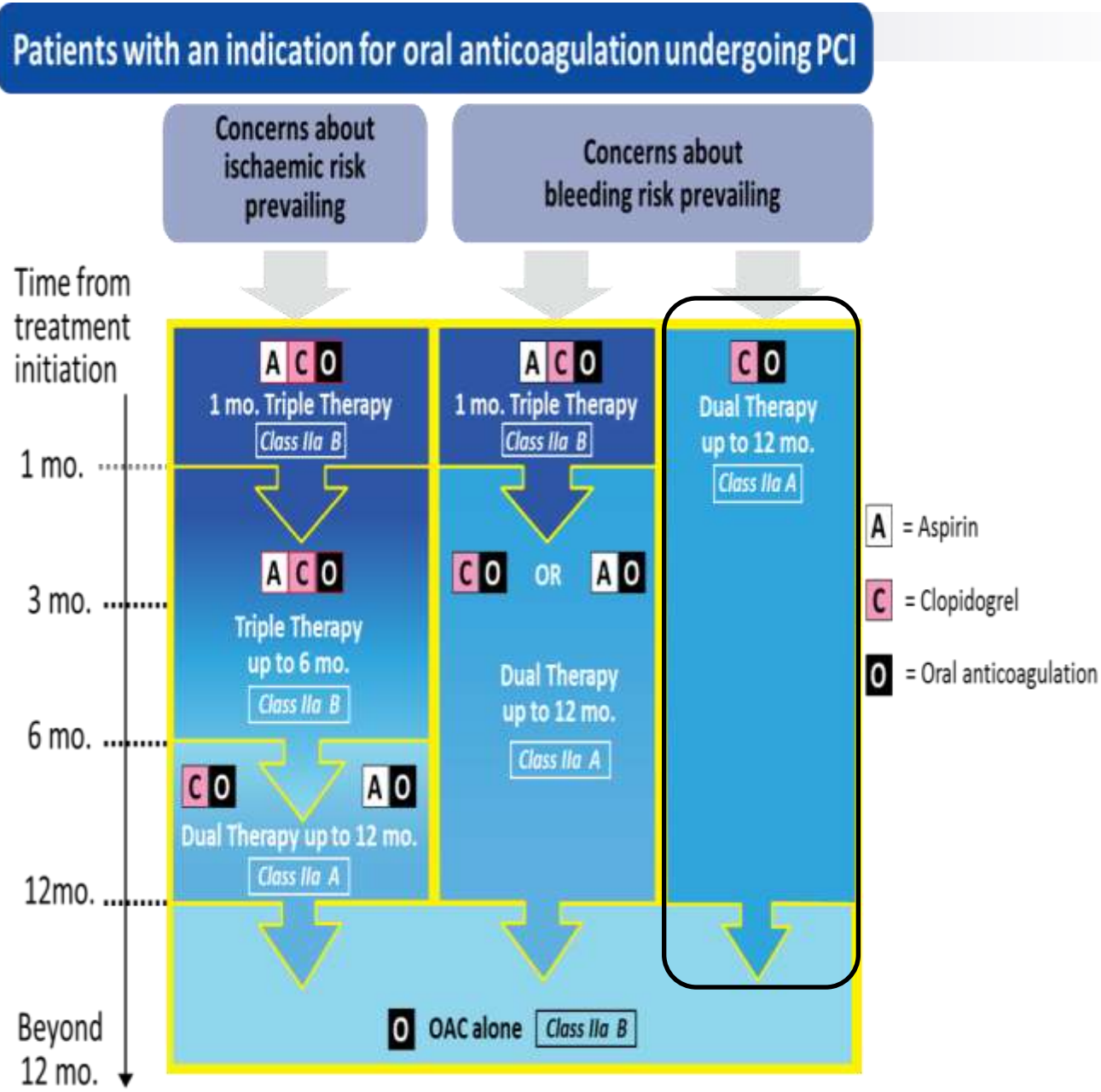
Effect of Primary and Primordial Prevention on Progression of Atherosclerosis and Risk of Acute Cardiovascular Events



Brian A. Ference et al. JACC 2018;72:1141-1156

JACC

Algorithm for dual antiplatelet therapy (DAPT) in patients with an indication for oral anticoagulation undergoing percutaneous coronary intervention (PCI)



www.escardio.org/guidelines

2017 ESC Focused Update on DAPT in Coronary Artery Disease, developed in collaboration with EACTS (European Heart Journal 2017 - doi:10.1093/eurheartj/ehx419)

Αντιθρομβωτική αγωγή σε ασθενείς που υποβάλλονται σε αγγειοπλαστική και χρήζουν από τους στόματος αντιπηκτικά

2017 ESC focused update on dual antiplatelet therapy in coronary artery disease developed in collaboration with EACTS

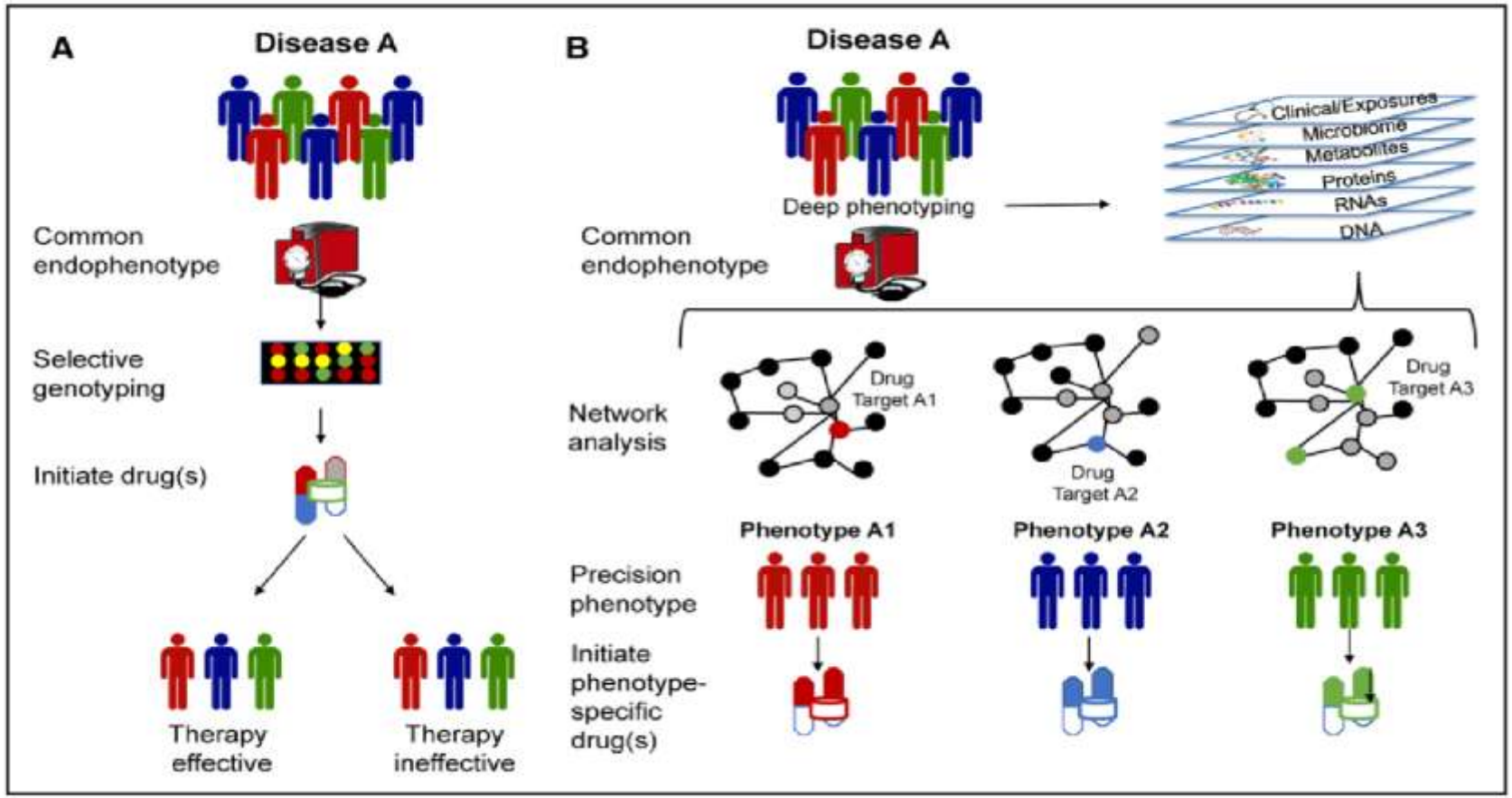
When a NOAC is used in combination with aspirin and/or clopidogrel, the lowest approved dose effective for stroke prevention tested in AF trials should be considered.[c]	IIa	C

- **Apixaban** 5 mg b.i.d or apixaban 2.5 mg b.i.d. *if at least two of* the following: age >_80 years, body weight <_60 kg or serum creatinine level >_1.5 mg/dL (133 lmol/ L);
- **Dabigatran** 110 mg b.i.d.;
- **Rivaroxaban** 20mg q.d. or rivaroxaban 15 mg q.d. if CrCl 30–49 mL/min.

European Heart Journal (2017) **0**, 1–48
doi:10.1093/eurheartj/ehx419

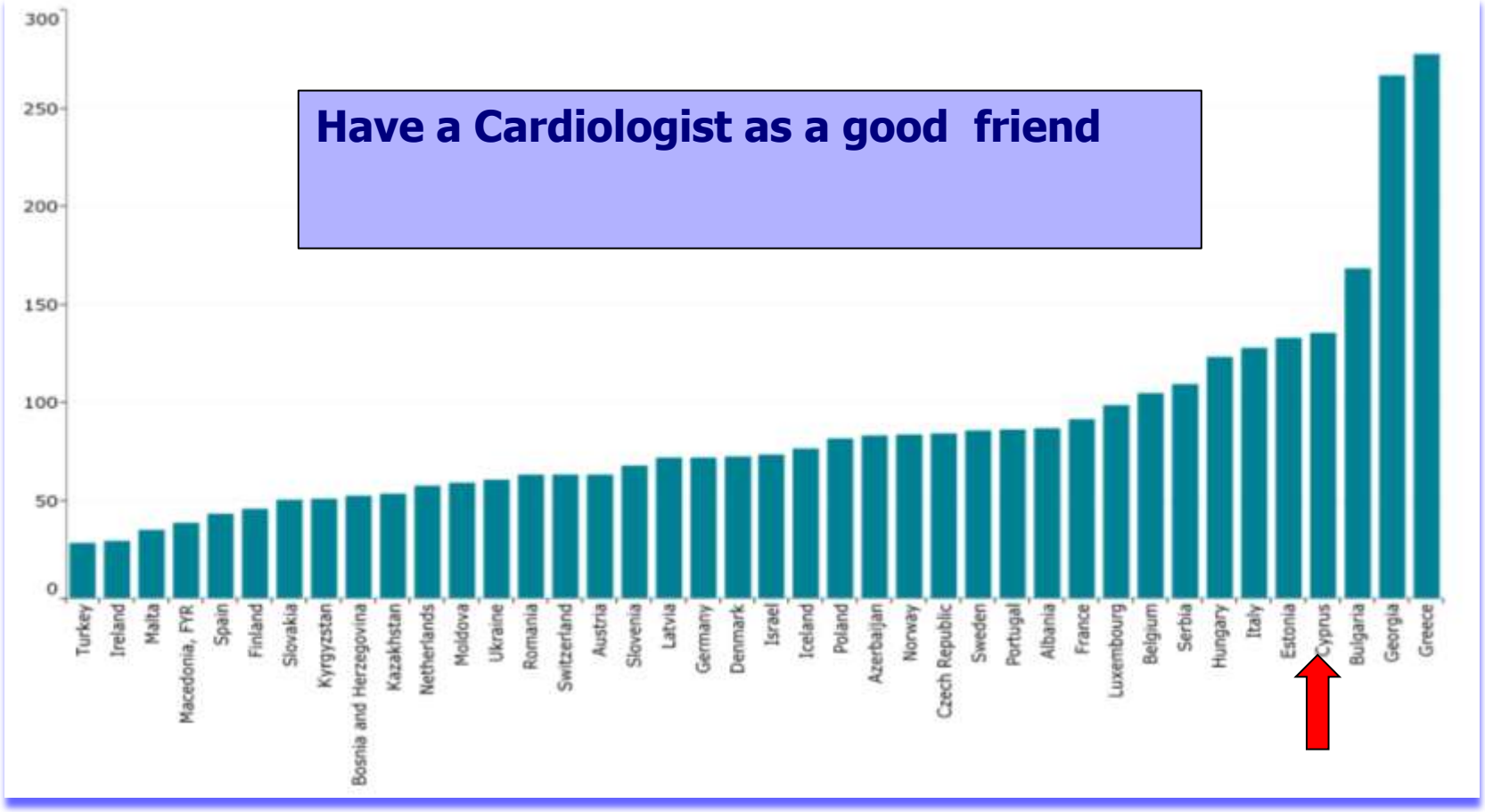
Precision Medicine is coming and should improve care

Biomarker Driven Therapeutics – Treat the Specific Phenotype



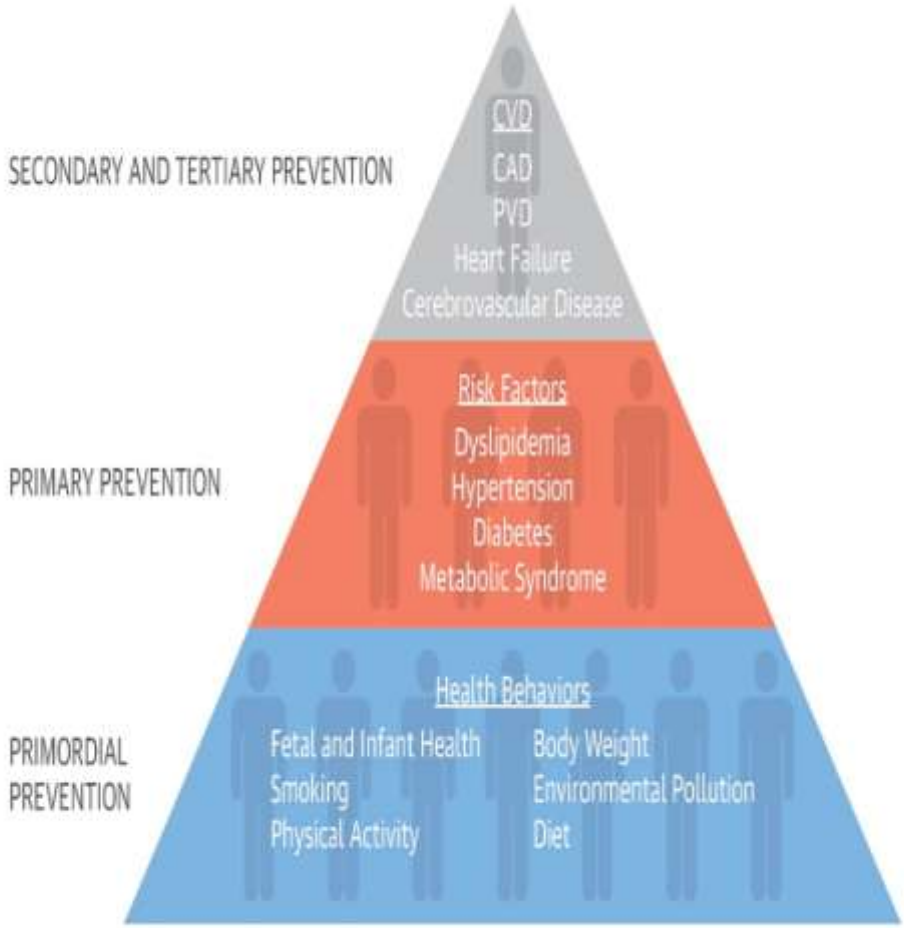
Leopold JA, Loscalzo J. **Circ Res** 2018;122(9):1302-1315

Cardiologists (per million people), 2015 or latest year



Source: ESC Atlas of Cardiology database 2017, data on file.

CENTRAL ILLUSTRATION: Cardiovascular Disease Prevention and Health Promotion



Hong, K.N. et al. J Am Coll Cardiol. 2017;70(17):2171-85.

"Prevention is better than cure"

Hippocrates

Everything in excess is opposed to nature

Hippocrates