ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
ΙΔΡΥΘΕΝ ΤΟ 1837

ΠΡΟΓΡΑΜΜΑ ΜΕΤΑΠΤΥΧΙΑΚΩΝ ΣΠΟΥΔΩΝ
«ΚΑΡΔΙΟΜΕΤΑΒΟΛΙΚΗ ΙΑΤΡΙΚΗ»

ΑΕΕ και πρόληψη. Πρωτογενής Πρόληψη ΑΕΕ και η διαχείριση της αντιθρομβωτικής αγωγής στη Δευτερογενή Πρόληψη

Κακαλέτσης Νικόλαος

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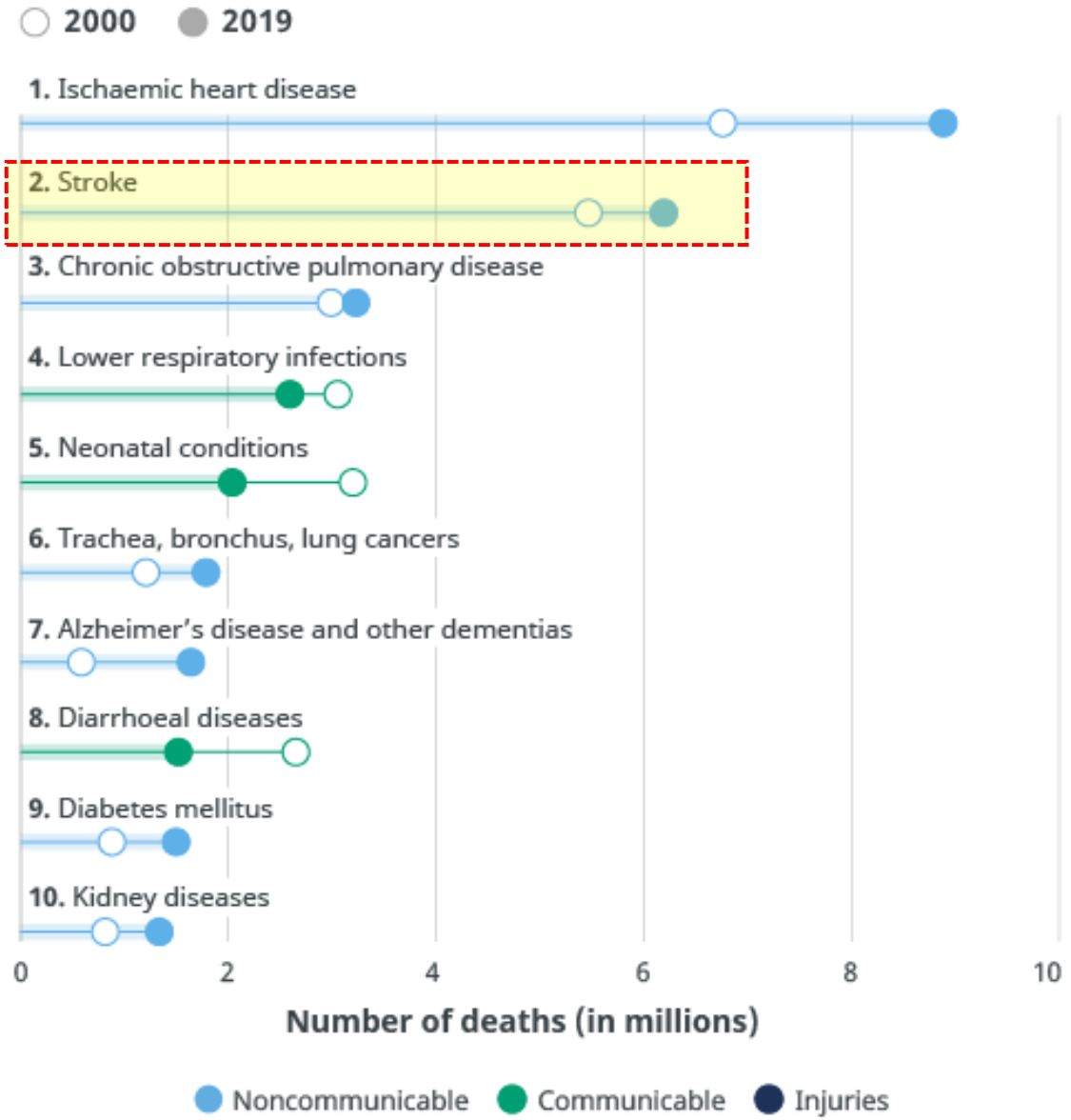
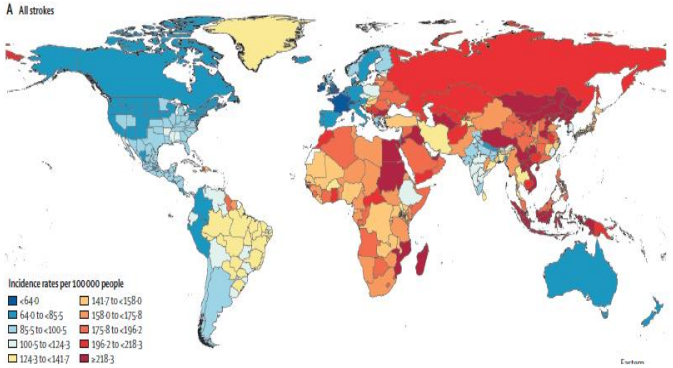
Μεταδιδακτορικός Ερευνητής Ιατρικής Σχολής Α.Π.Θ.
Stroke Research Fellow, HUS Helsinki University Hospital and University of Helsinki, Finland

5 Μαρτίου 2026

Leading causes of death globally



Source: WHO Global Health Estimates.



World Health Organization

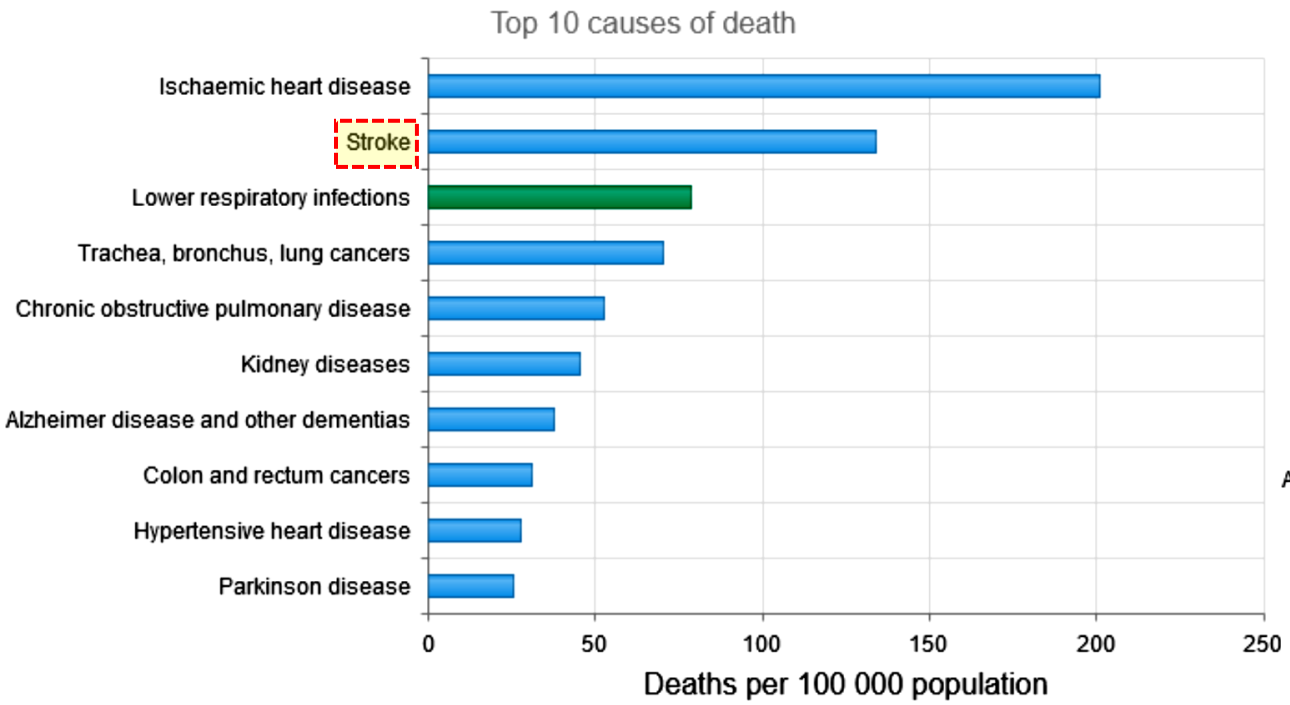
Top 10 global causes of disability-adjusted life years (DALYs) in 2019

1. Neonatal conditions
2. Ischaemic heart disease
3. Stroke
4. Lower respiratory infections
5. Diarrhoeal diseases
6. Road injury
7. Chronic obstructive pulmonary disease
8. Diabetes mellitus
9. Tuberculosis
10. Congenital anomalies

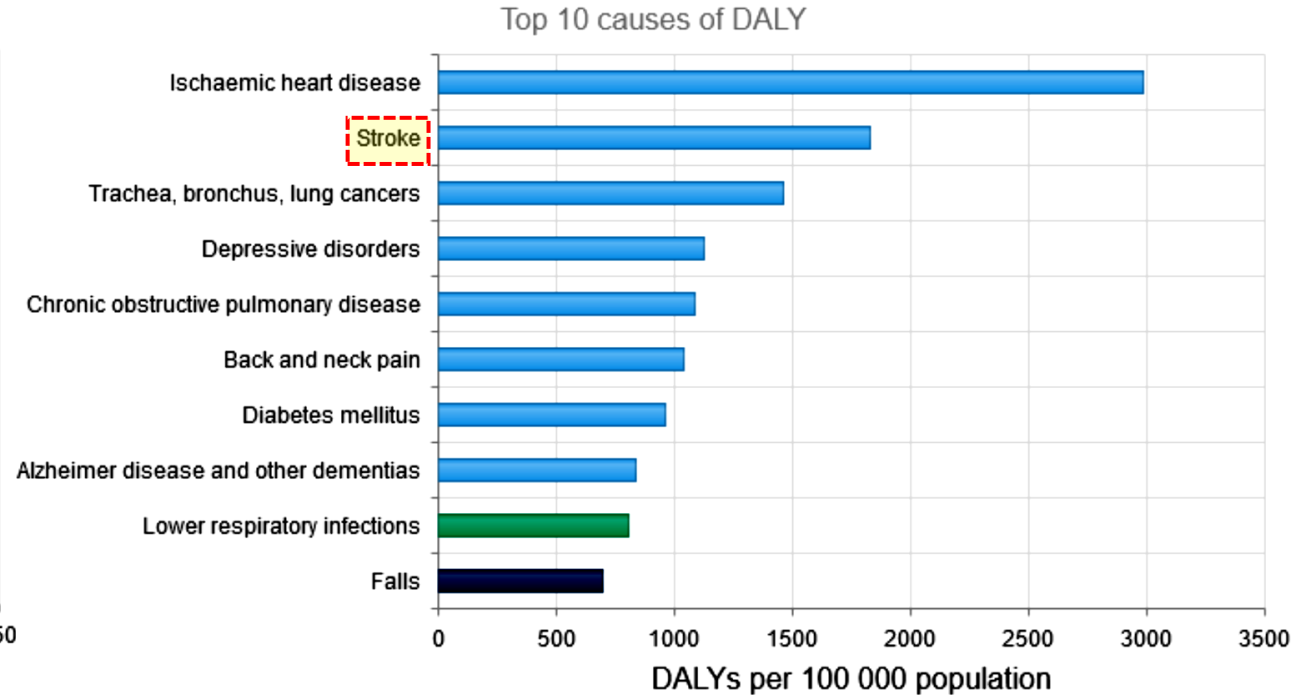
https://www.who.int/data/gho/data/themes/mortality-and-global-health-estimates

World Health Organization

Top 10 causes of death in Greece for both sexes aged all ages (2019)



Top 10 causes of DALY in Greece for both sexes aged all ages (2019)



https://www.who.int/data/gho/data/themes/mortality-and-global-health-estimates

Ischemic stroke is an etiologically heterogeneous syndrome

Stroke due to Small Vessel Disease (SVD)

ISCHEMIC STROKE

Non-SVD, non-embolic Stroke

Σηπτικό έμβολο - ενδοκαρδίτιδα

Non-thrombosis-mediated embolism

Non-thrombotic cardiogenic embolism

Embolic Stroke

Κολπική Μαρμαρυγή

Μεταλλική Βαλβίδα

Valvular Disease

Chamber embolism

Cardiogenic embolism

Ραγείσα αθηροσκληρωτική πλάκα στη καρωτίδα

Διαχωρισμός στη καρωτίδα

Atherosclerotic

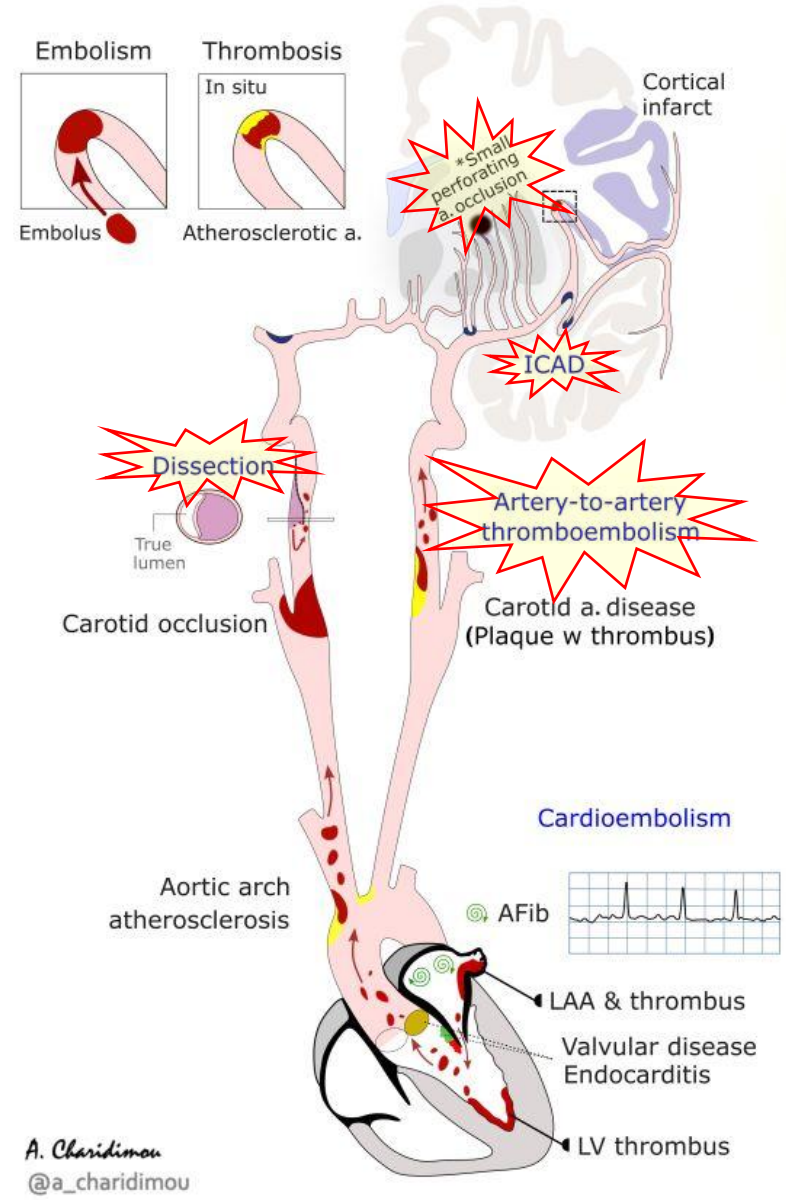
Non-Atherosclerotic

Arteriogenic embolism

Paradoxical embolism

ΕΒΦΘ κάτω άκρου και PFO

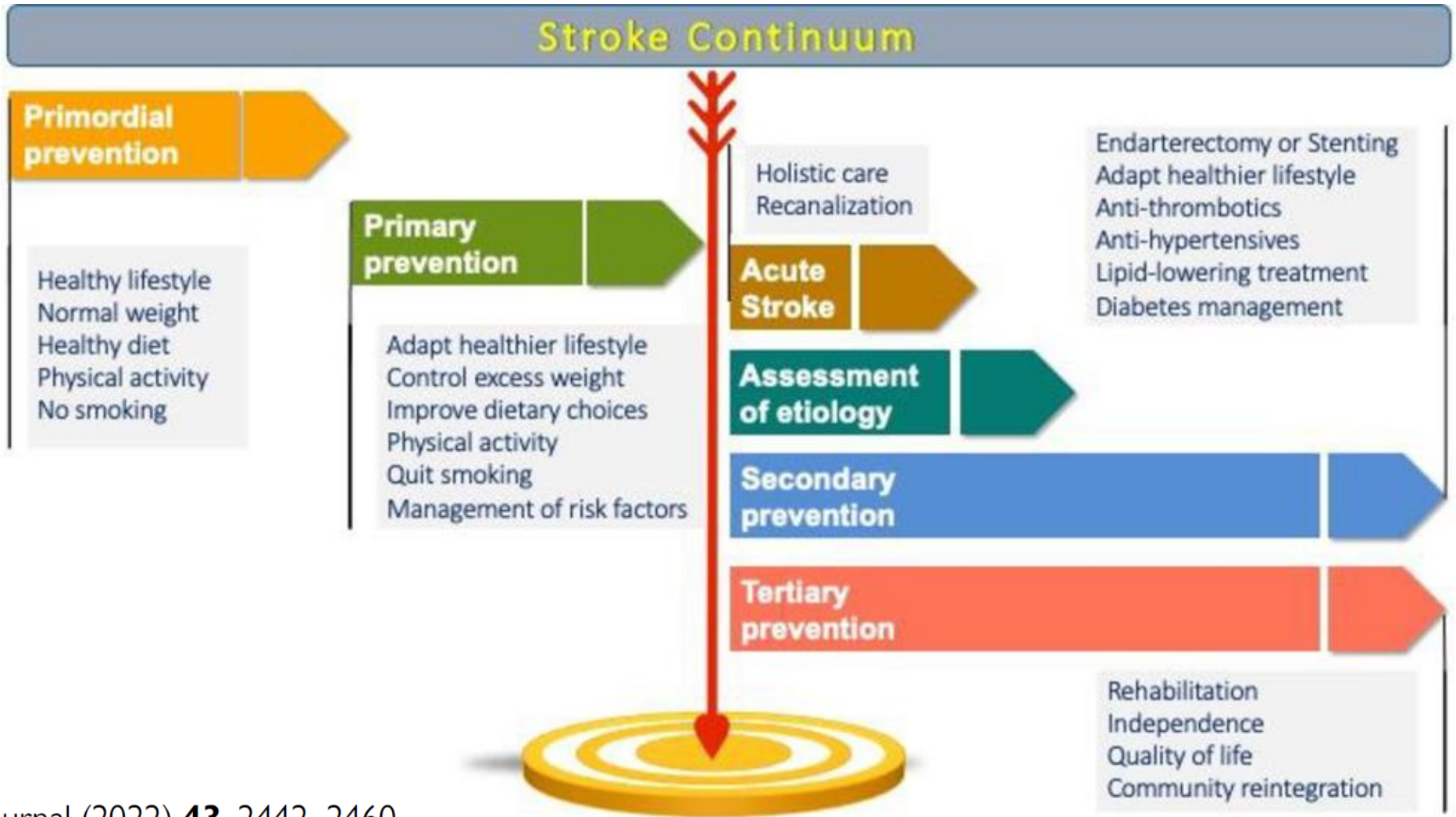
Thrombosis-mediated embolism



Ntaios G, Hart RG. Embolic stroke. Circulation. 2017;136:2403e2405.

Integrated care for optimizing the management of stroke and associated heart disease: a position paper of the European Society of Cardiology Council on Stroke

Gregory Y. H. Lip [1,2,3,4]*†, Deirdre A. Lane[1,2], Radosław Lenarczyk[3], Giuseppe Boriani [5], Wolfram Doehner [6], Laura A. Benjamin[7], Marc Fisher[8], Deborah Lowe[9], Ralph L. Sacco[10], Renate Schnabel[11], Caroline Watkins[12], George Ntaios [13], and Tatjana Potpara [4,14]†



European Heart Journal (2022) **43**, 2442–2460

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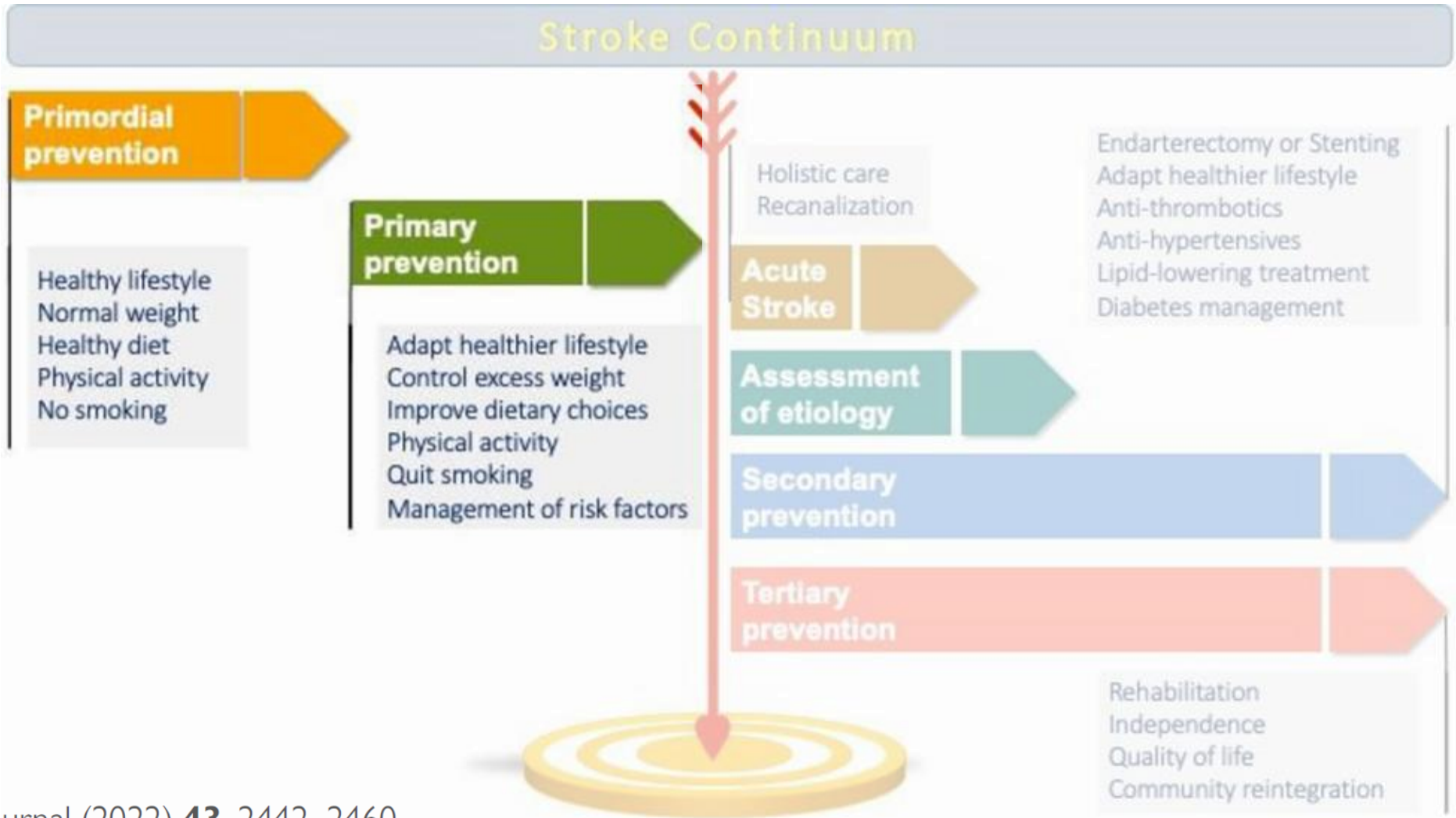

European Heart Journal (2022) **43**, 2442–2460

Global and regional effects of potentially modifiable risk factors associated with acute stroke in 32 countries (INTERSTROKE): a case-control study

INTERSTROKE

- 26,919 participants were recruited (1/2007 – 8/2015)
- from 32 countries (Asia, America, Europe, Australia, Middle East, Africa)
- 13,447 cases (10,388 AIS & 3,059 ICH) and 13,472 controls

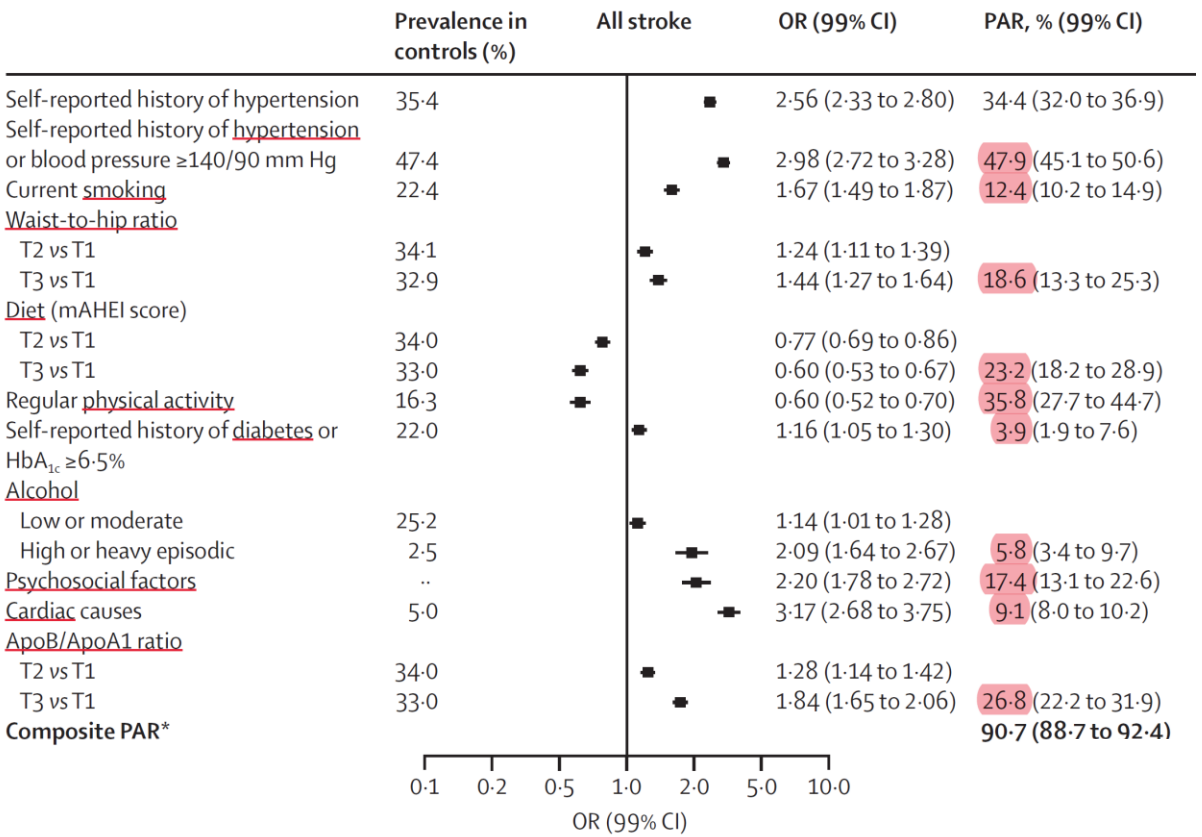

TABLE 1. TEN MODIFIABLE RISK FACTORS CONTRIBUTING TO 90% OF STROKES WORLDWIDE

Risk factor	Percent*
1. Hypertension	47.9%
2. Physical activity	35.8%
3. Apo/ApoA1 ratio	26.8%
4. Diet	23.2%
5. Waist-to-hip ratio	18.6%
6. Psychosocial factors	17.4%
7. Current smoker	12.4%
8. Cardiac causes	9.1%
9. Alcohol consumption	5.8%
10. Diabetes mellitus	3.9%

* Population attributable risk percent is the percent of the incidence of a disease in the population that is due to exposure. For example, 47.9% of all strokes in the world can be attributed to hypertension.

Lancet 2016 20;388:761-75

Stroke Risk Factors, Genetics, and Prevention

	Nonmodifiable Risk Factors	Modifiable Risk Factors
Ischemic stroke	Age	Hypertension
	Sex	Current smoking
	Race/ethnicity	Waist-to-hip ratio
		Diet
		Physical inactivity
		Hyperlipidemia
		Diabetes mellitus
		Alcohol consumption
		Cardiac causes
		Apolipoprotein B to A1
	Genetics*	

Circ Res. 2017;120:472-495

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Circ Res. 2017;120:472-495

Primary and Secondary Prevention of Ischemic Stroke and Cerebral Hemorrhage

JACC Focus Seminar

Lifestyle modifications including:

- healthy diet
- weight loss
- termination of smoking
- regular physical activity

are recommended.

JACC VOL. 75, NO. 15, 2020
APRIL 21, 2020:1804–18

ONTARGET

TRANSCEND

Relationship Between Healthy Diet and Risk of Cardiovascular Disease Among Patients on Drug Therapies for Secondary Prevention

A Prospective Cohort Study of 31 546 High-Risk Individuals From 40 Countries

mAHEI, modified Alternative Healthy Eating Index

Table 3. HRs and 95% CIs of the Composite Outcome for Individuals With Risk Factors or History of Diseases and According to Quintiles of the Modified Alternative Healthy Eating Index (Quintile 5 Versus 1, Healthiest Versus Unhealthiest)

	mAHEI				*P* for Trend
	Q2 vs Q1	Q3 vs Q1	Q4 vs Q1	Q5 vs Q1	
Hypertensive (n=26 307)	0.99 (0.91–1.08)	0.91 (0.83–1.01)	0.85 (0.77–0.95)	0.83 (0.74–0.92)	<0.0001
Normotensive (n=5239)	0.74 (0.58–0.95)	0.69 (0.53–0.88)	0.61 (0.47–0.78)	0.56 (0.42–0.74)	<0.0001
Diabetes mellitus, FPG ≥7 mg/dL (n=12 869)	0.96 (0.85–1.09)	0.91 (0.80–1.04)	0.86 (0.75–0.99)	0.75 (0.65–0.87)	<0.0001
No diabetes mellitus, FPG <7 mg/dL (n=18 676)	0.95 (0.84–1.07)	0.85 (0.74–0.96)	0.78 (0.69–0.90)	0.81 (0.71–0.92)	<0.0001
LDL median ≥2.80 mg/dL (n=15 254)	0.97 (0.87–1.09)	0.89 (0.79–1.00)	0.83 (0.73–0.95)	0.82 (0.72–0.94)	<0.001
LDL median <2.80 mg/dL (n=15 218)	0.94 (0.82–1.07)	0.87 (0.76–1.01)	0.82 (0.71–0.95)	0.76 (0.66–0.87)	<0.0001
With stroke/transient ischemic attack (n=6644)	0.94 (0.80–1.12)	0.82 (0.69–0.97)	0.79 (0.65–0.95)	0.78 (0.66–0.93)	<0.0001
Without stroke/transient ischemic attack (n=24 892)	0.96 (0.86–1.05)	0.90 (0.81–1.00)	0.83 (0.74–0.94)	0.78 (0.69–0.89)	<0.0001
With CAD (n=23 520)	0.97 (0.88–1.07)	0.85 (0.76–0.95)	0.83 (0.73–0.93)	0.78 (0.69–0.88)	<0.001
Without CAD (n=8026)	0.93 (0.77–1.12)	0.98 (0.83–1.16)	0.83 (0.69–0.99)	0.82 (0.69–0.98)	0.01
With PAD (n=4140)	0.92 (0.76–1.11)	1.02 (0.83–1.23)	0.77 (0.62–0.94)	0.92 (0.73–1.14)	0.1
Without PAD (n=27 406)	0.96 (0.88–1.06)	0.85 (0.77–0.95)	0.83 (0.74–0.93)	0.77 (0.68–0.86)	<0.0001

Patients in the healthier quintiles of mAHEI scores had a significantly *lower risk of CVD* (HR: **0.78**, 95%CI: 0.71-0.87).
The reductions in risk for CV death, myocardial infarction, and **stroke** were 35%, 14%, and **19%**, respectively.
The protective association was *consistent regardless of whether patients were receiving proven drugs*.

Conclusions

A **higher-quality diet** was associated with a **lower risk of recurrent CVD** events among **people ≥55** years of age with CVD or diabetes mellitus.
Highlighting the importance of healthy eating by health professionals would substantially reduce CVD recurrence and save lives globally.

Circulation. 2012;126:2705-2712

Primary Prevention of Cardiovascular Disease with a Mediterranean Diet Supplemented with Extra-Virgin Olive Oil or Nuts

PREDIMED

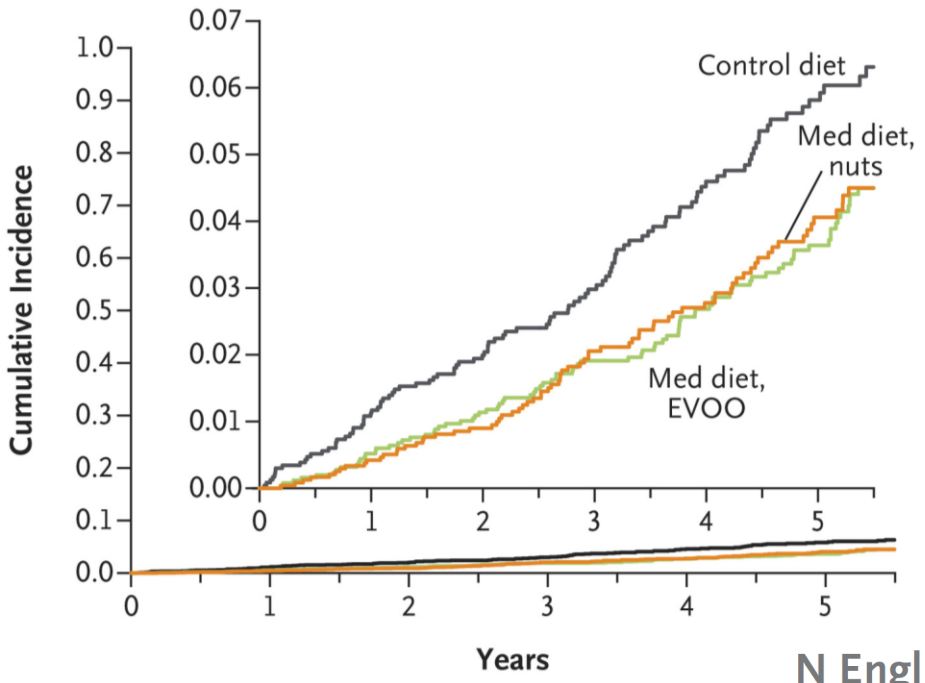
MeDiet + EVOO
N = 2543

MeDiet + Nuts
N = 2454

Control Diet
N = 2450

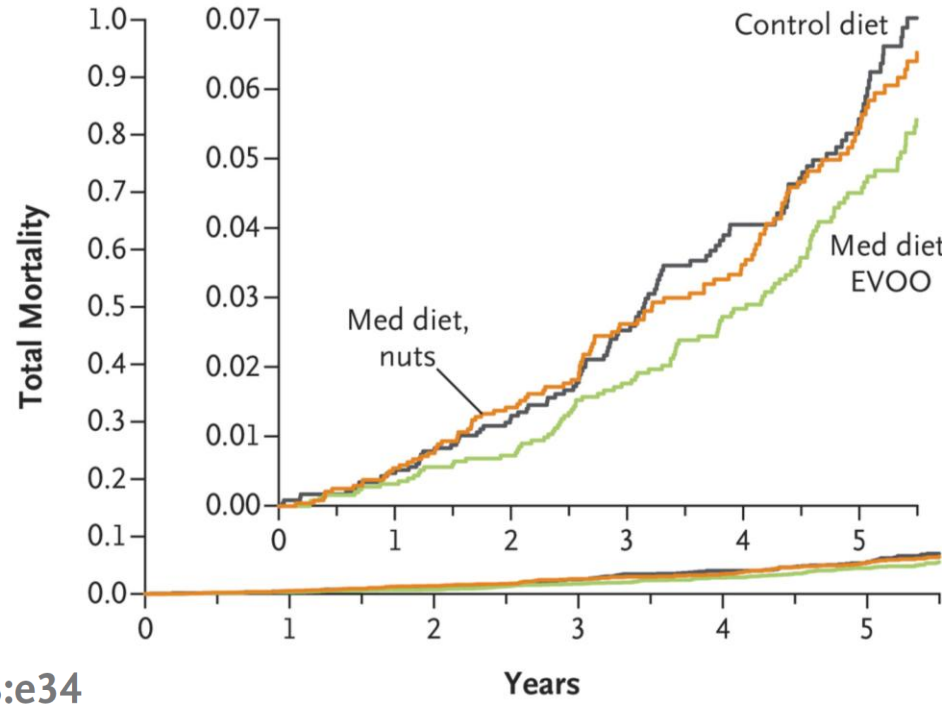
A **Primary End Point (acute myocardial infarction, stroke, or death from cardiovascular causes)**

Med diet, EVOO: hazard ratio, 0.69 (95% CI, 0.53–0.91)
Med diet, nuts: hazard ratio, 0.72 (95% CI, 0.54–0.95)



B **Total Mortality**

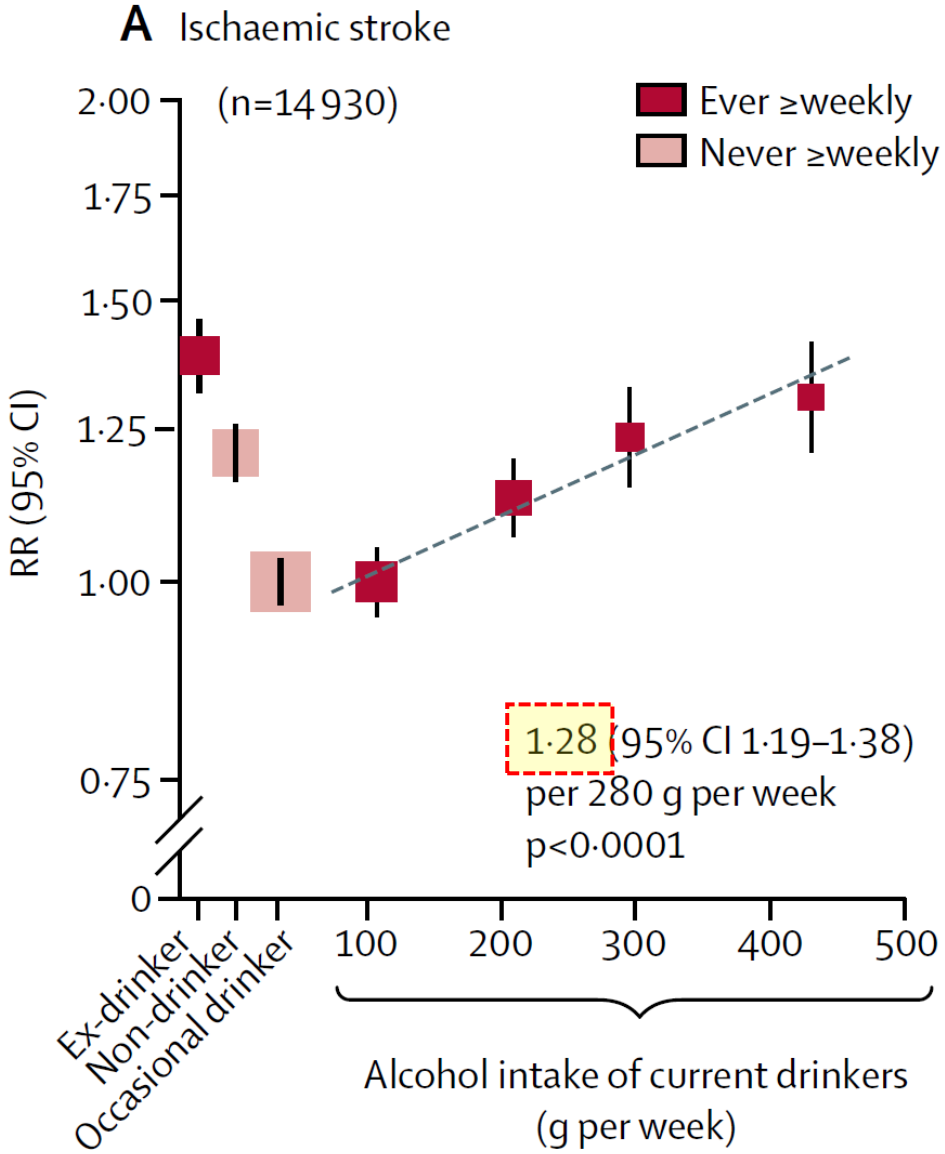
Med diet, EVOO: hazard ratio, 0.90 (95% CI, 0.69–1.18)
Med diet, nuts: hazard ratio, 1.12 (95% CI, 0.86–1.47)



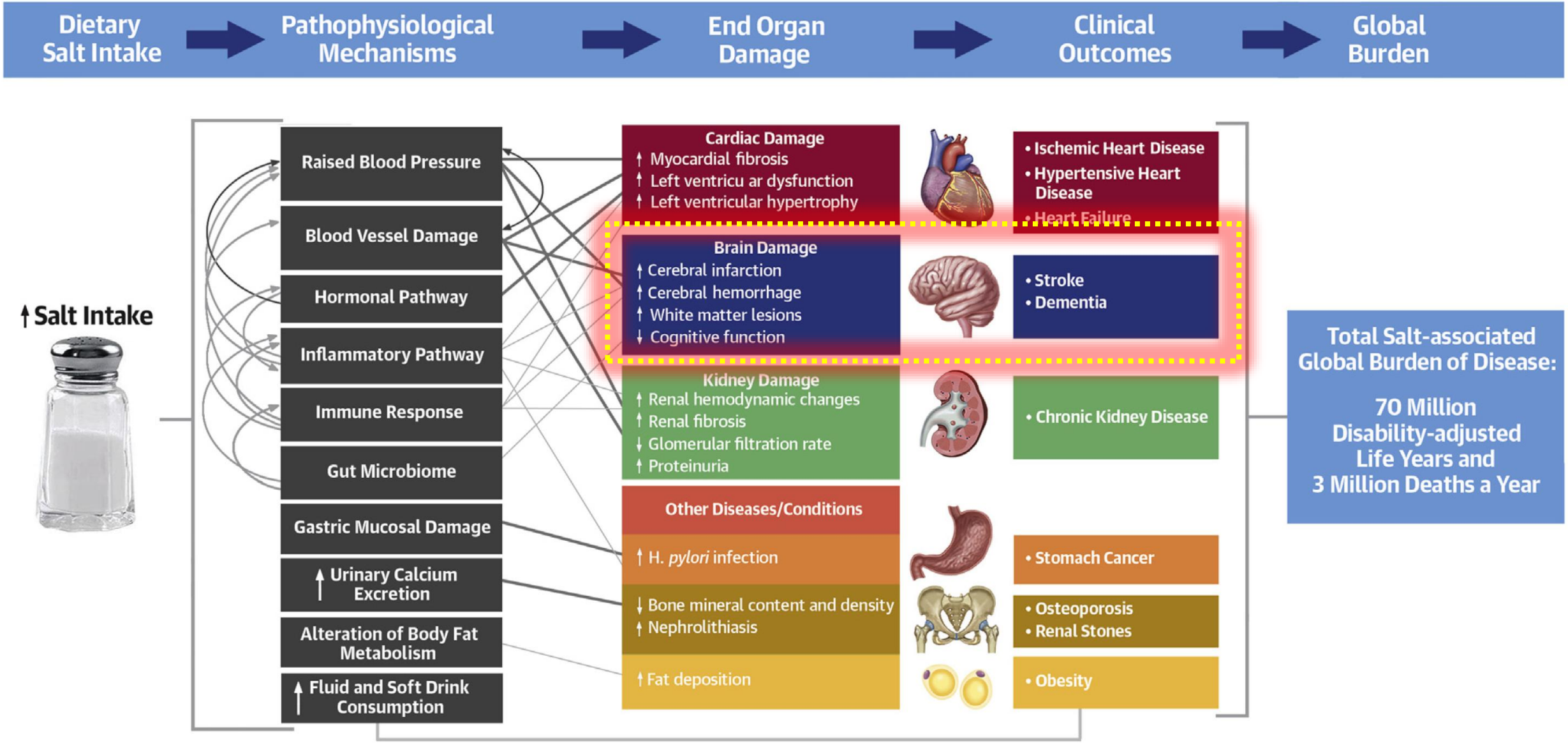
N Engl J Med 2018;378:e34

Conventional and genetic evidence on alcohol and vascular disease aetiology: a prospective study of 500 000 men and women in China

prospective China Kadoorie Biobank
enrolled 512,715 adults



Lancet 2019 4;393:1831-1842



He, F.J. et al. J Am Coll Cardiol. 2020;75(6):632-47.

How Far Should Salt Intake Be Reduced?

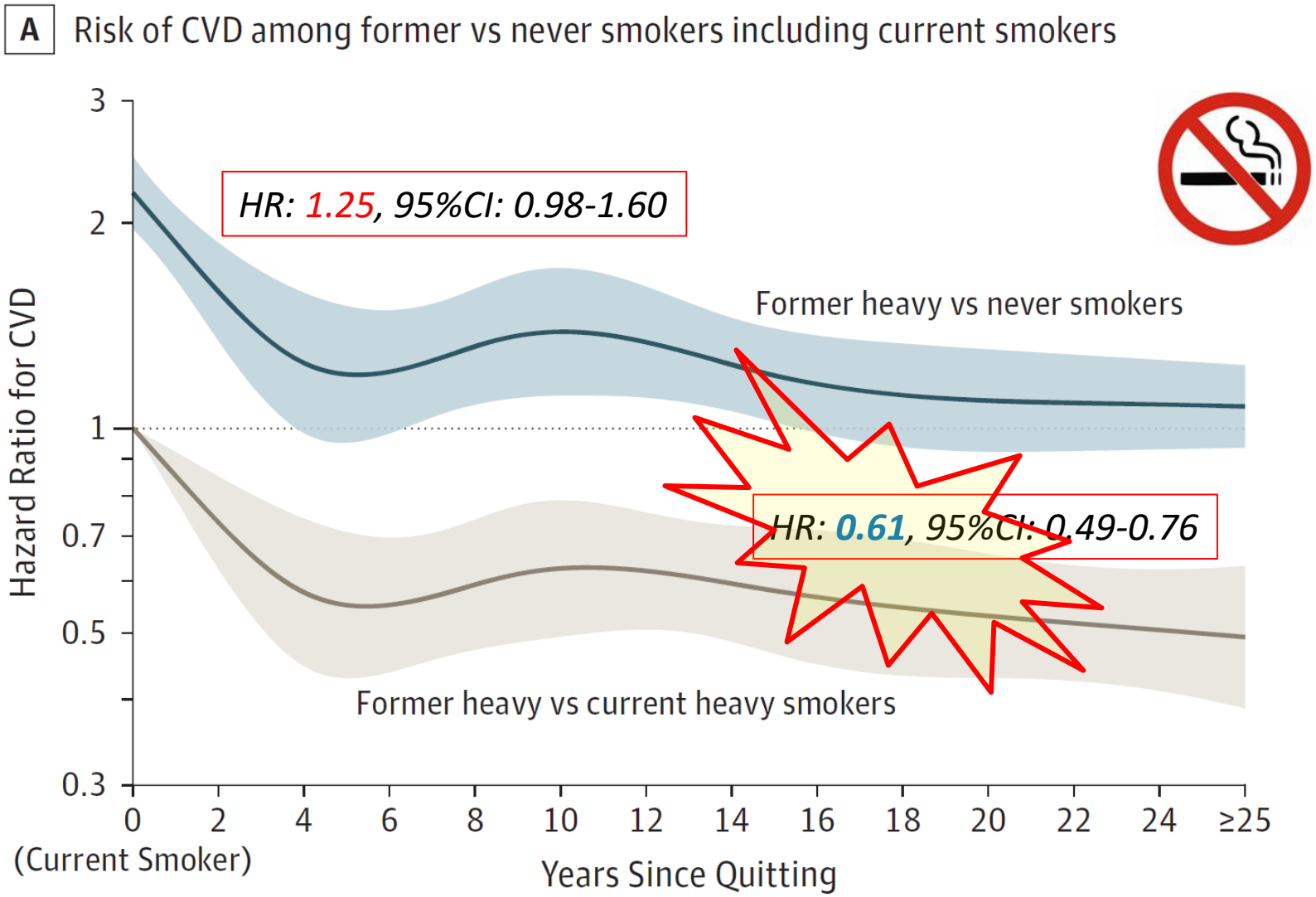
TABLE 2. Predicted Reductions in Stroke and IHD Deaths With Reductions in Salt Intake

Measure	Reduction in Salt Intake					
	3 g/d (50 mmol/d)		6 g/d (100 mmol/d)		9 g/d (150 mmol/d)	
	SBP	DBP	SBP	DBP	SBP	DBP
Fall in BP in all participants, mm Hg (from the meta-analysis)	2.5	1.4	5	2.8	7.5	4.2
Reduction in stroke death, %	12	14	23	25	32	36
Stroke deaths prevented in UK, n/y	7300	8300	13,700	15,500	19,300	21,600
Reduction in IHD death, %	9	10	16	19	23	27
IHD deaths prevented in UK, n/y	10,600	12,400	20,300	23,600	29,100	33,700

Hypertension. 2003;42:1093-1099

Association of Smoking Cessation With Subsequent Risk of Cardiovascular Disease

8,770 individuals from Framingham Heart Study participants without baseline CVD, mean age of 42.2 years and 45% male



CONCLUSIONS & RELEVANCE

Among heavy smokers, **smoking cessation** was associated with significantly **lower risk of CVD** within 5 years relative to current smokers. However, relative to never smokers, former smokers' CVD risk remained significantly elevated beyond 5 years after smoking cessation.

JAMA. 2019;322(7):642-650

Physical activity and risk of ischemic stroke in the Northern Manhattan Study

Table 3 Risk of ischemic stroke associated with physical activity in the Northern Manhattan Study

Physical activity intensity	Unadjusted HR	95% CI	Partially adjusted HR*	95% CI	Fully adjusted HR†	95% CI
Any vs none	0.86	0.66–1.12	0.80	0.61–1.04	0.86	0.66–1.13
Light vs none	0.97	0.74–1.28	0.90	0.68–1.19	0.94	0.71–1.25
Moderate to heavy vs none	0.65	0.44–0.95	0.57	0.38–0.85	0.65	0.43–0.98
Moderate to heavy vs light to none combined	0.66	0.46–0.94	0.60	0.41–0.88	0.68	0.46–0.99

Neurology® 2009;73:1774–1779

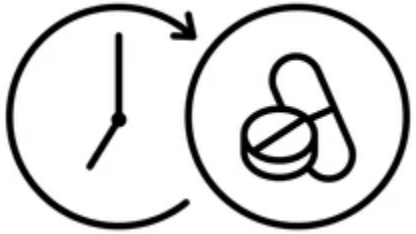
International Journal of Stroke
2021, Vol. 16(6) 738–750

The initial non-pharmacological approach is very important in patients at very high risk of future CV events, such as stroke or TIA patients:

- *increasing the potential of a **better physician-to-patient interaction**,*

&

- ***adherence to treatment**.*



Stroke Risk Factors, Genetics, and Prevention

	Nonmodifiable Risk Factors	Modifiable Risk Factors
Ischemic stroke	Age	Hypertension
	Sex	Current smoking
	Race/ethnicity	Waist-to-hip ratio
		Diet
		Physical inactivity
		Hyperlipidemia
		Diabetes mellitus
		Alcohol consumption
		Cardiac causes
		Apolipoprotein B to A1
	Genetics*	

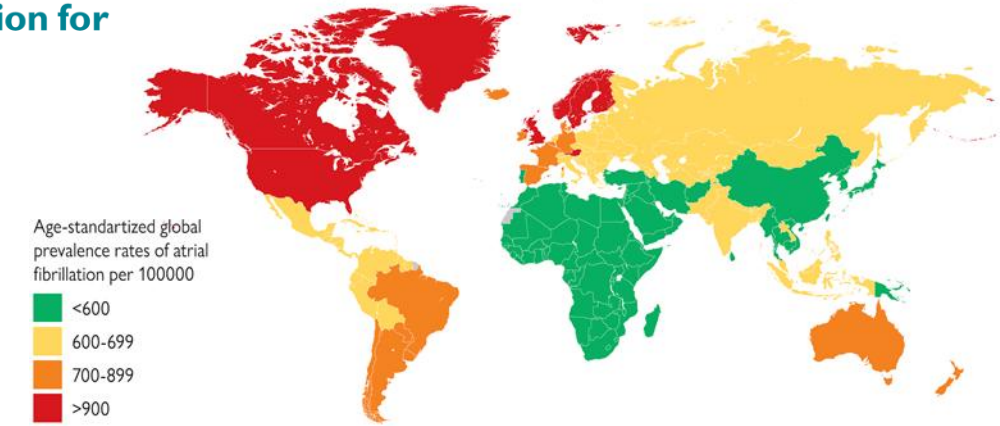
Circ Res. 2017;120:472-495

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)

ESC
European Society of Cardiology

GLOBAL PREVALENCE OF AF

(globally, 43.6 million individuals had prevalent AF/AFL in 2016)

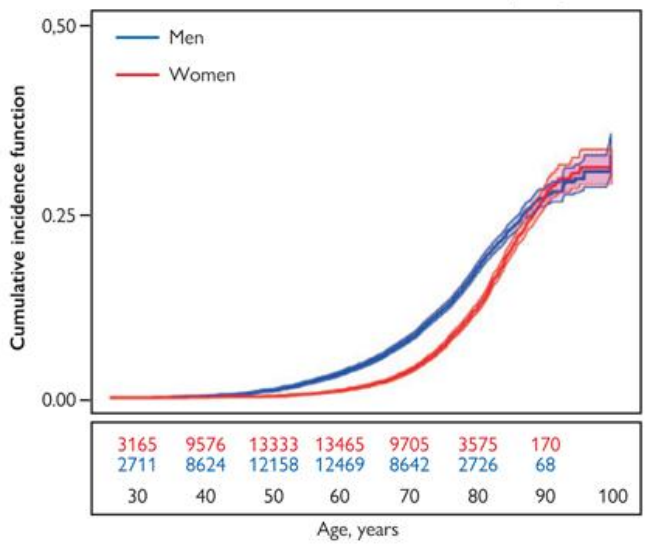


LIFETIME RISK for AF
1 in 3 individuals

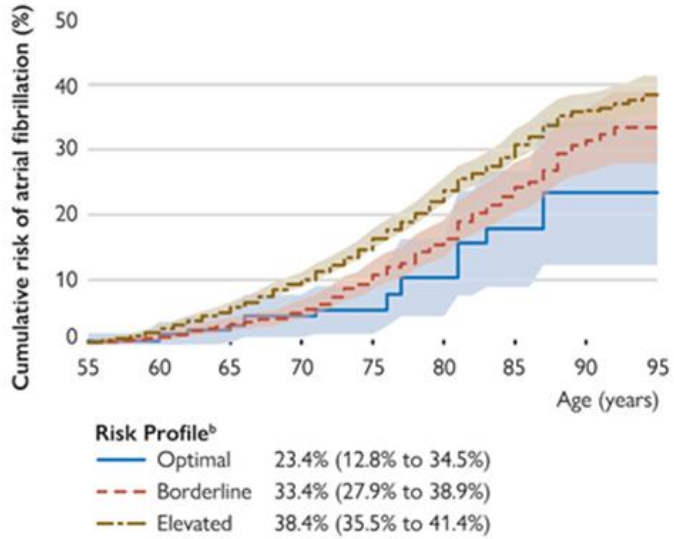
of European ancestry
at index age of 55 years
37.0% (34.3% to 39.6%)

AF is more common in males

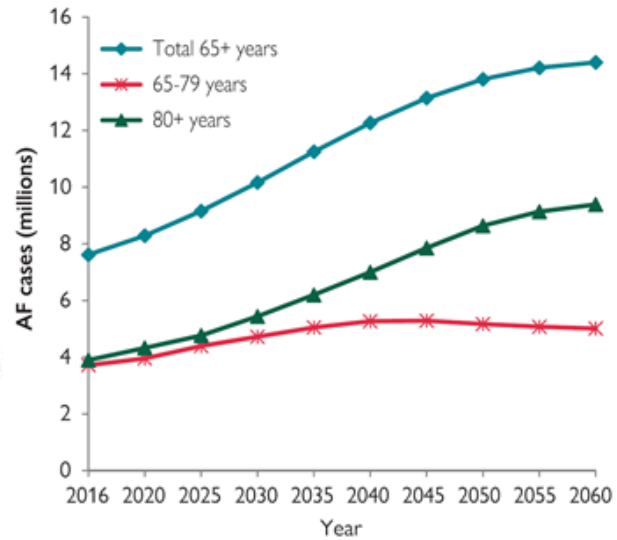
Cumulative incidence curves and 95% CIs
for AF in women and men with death as a competing risk



Lifetime risk of AF increases with increasing risk factor burden[a]

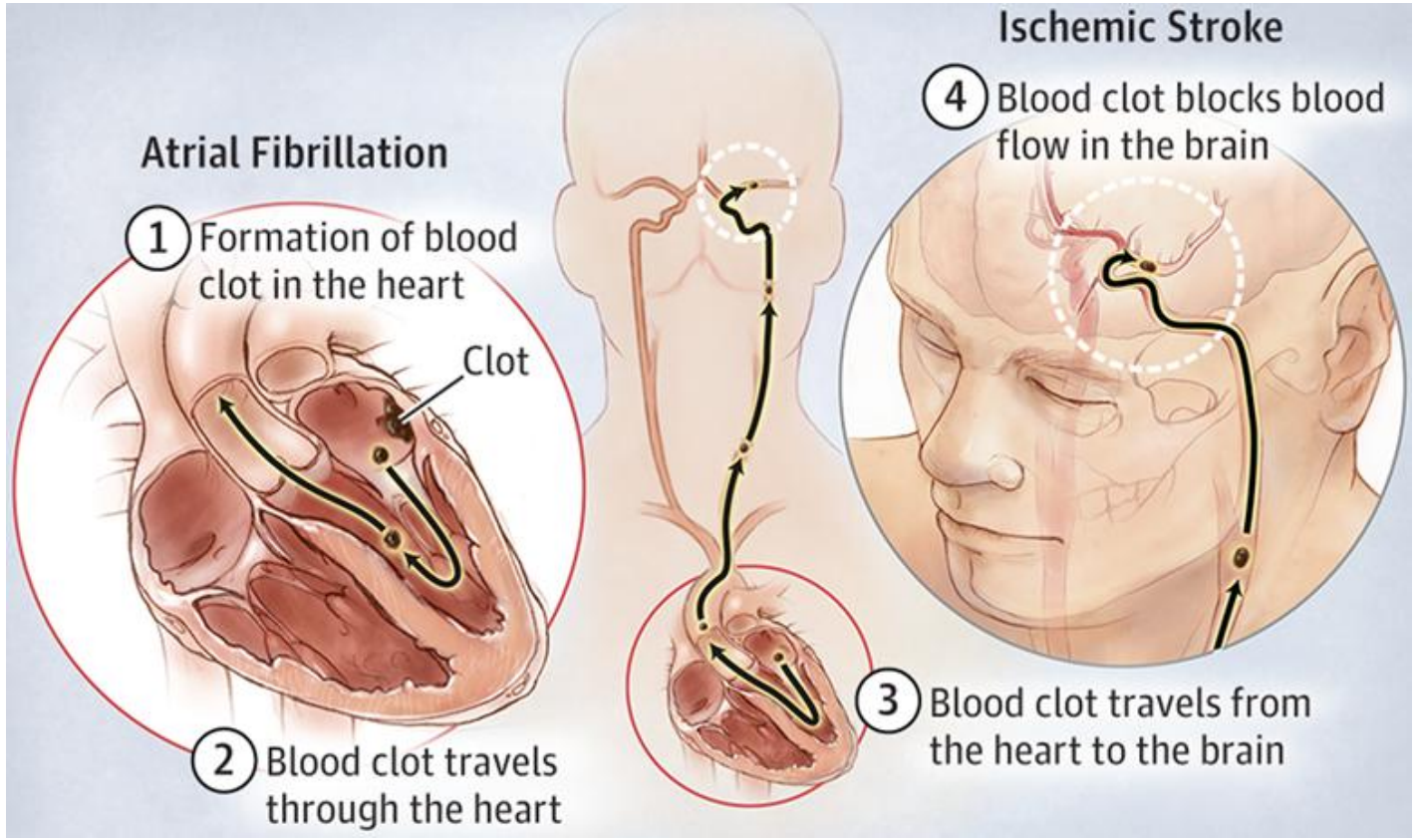


Projected increase in AF prevalence among elderly in EU 2016-2060



European Heart Journal (2020) **42**, 373–498

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)



CHA_2DS_2-VASc score Risk factors and definitions		Points awarded
C	**Congestive heart failure** Clinical HF, or objective evidence of moderate to severe LV dysfunction, or HCM	1
H	**Hypertension** or on antihypertensive therapy	1
A	**Age 75 years or older**	2
D	**Diabetes mellitus** Treatment with oral hypoglycaemic drugs and/or insulin or fasting blood glucose >125 mg/dL (7 mmol/L)	1
S	**Stroke**Previous stroke, TIA, or thromboembolism	2
V	**Vascular disease** Angiographically significant CAD, previous myocardial infarction, PAD, or aortic plaque	1
A	**Age 65 – 74 years**	1
Sc	**Sex category (female)**	1
Maximum score		**9**

European Heart Journal (2020) **42**, 373–498

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ESC European Society of Cardiology

European Heart Journal (2020) **42**, 373–498

Refining Clinical Risk Stratification for Predicting Stroke and Thromboembolism in Atrial Fibrillation Using a Novel Risk Factor-Based Approach

The Euro Heart Survey on Atrial Fibrillation

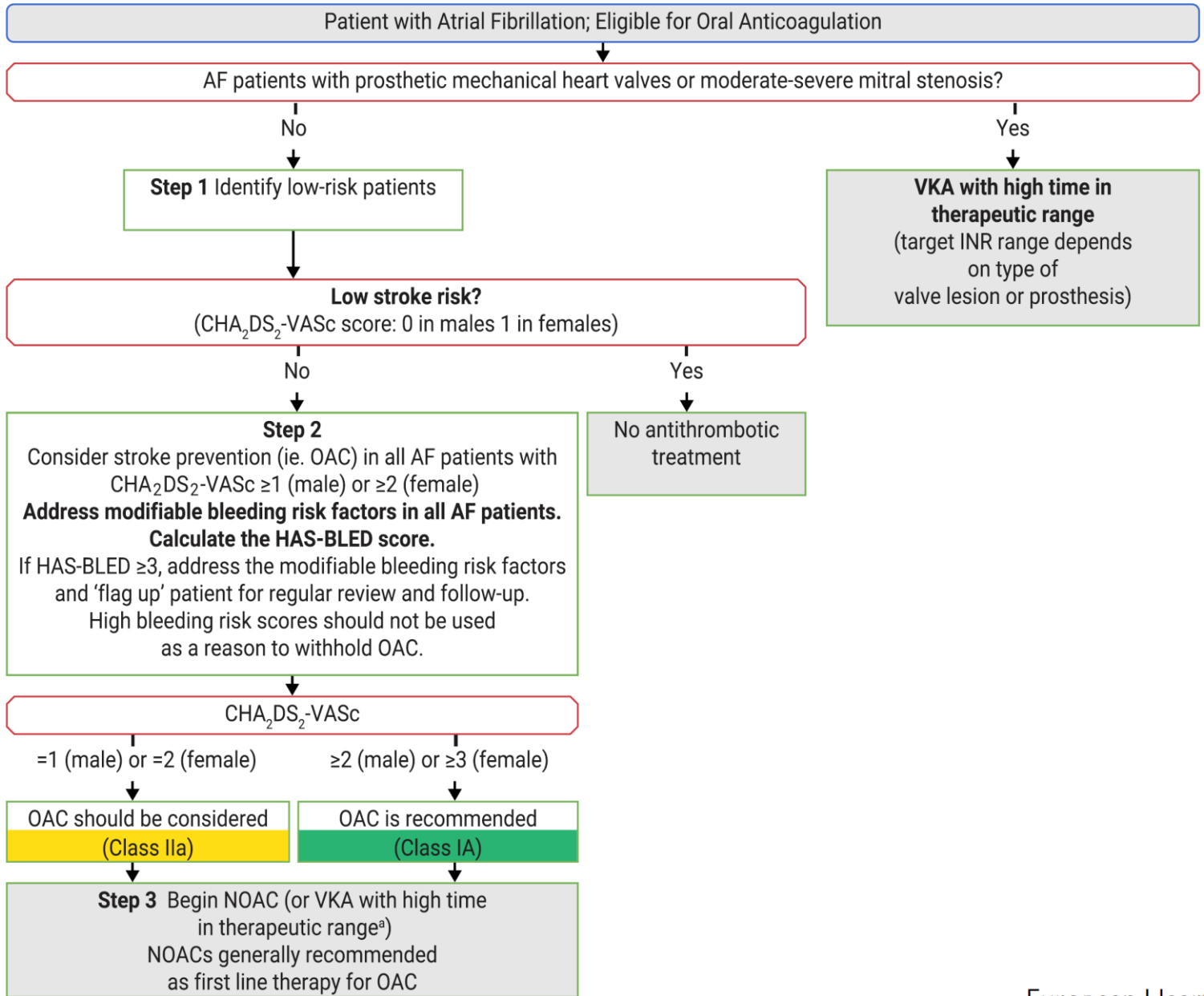
Table 6—*Stroke or Other TE at 1 Year Based on the 2009 Birmingham (CHA_2DS_2-VASc) Scoring System*

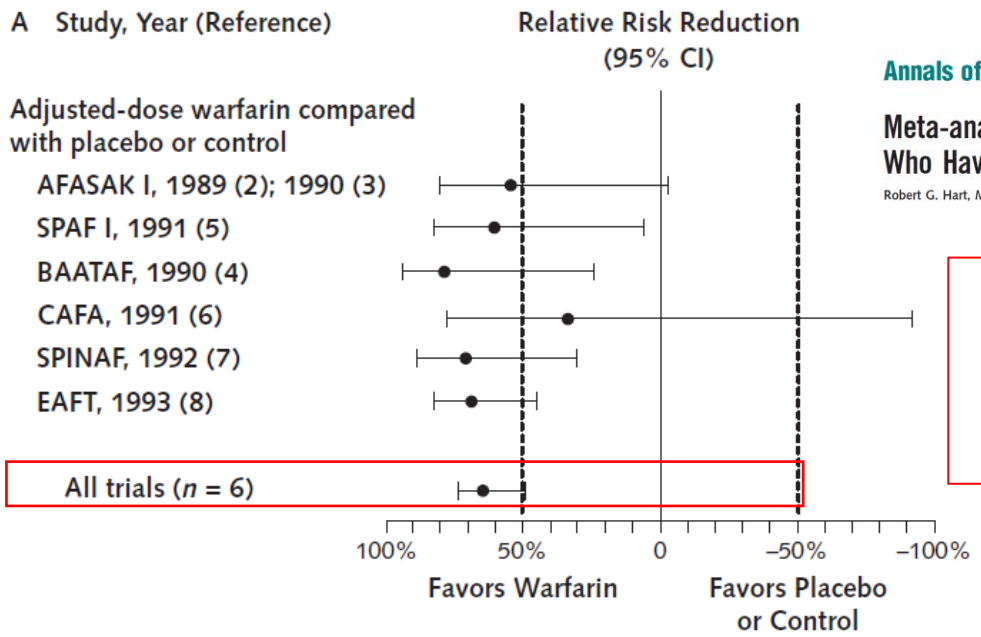
CHA_2DS_2-VASc Score	No.	Number of TE Events	TE Rate During 1 y (95% CI)	TE Rate During 1 y, Adjusted for Aspirin Prescription,[a] %
0	103	0	0% (0-0)	0
1	162	1	0.6% (0.0-3.4)	0.7
2	184	3	1.6% (0.3-4.7)	1.9
3	203	8	3.9% (1.7-7.6)	4.7
4	208	4	1.9% (0.5-4.9)	2.3
5	95	3	3.2% (0.7-9.0)	3.9
6	57	2	3.6% (0.4-12.3)	4.5
7	25	2	8.0% (1.0-26.0)	10.1
8	9	1	11.1% (0.3-48.3)	14.2
9	1	1	100% (2.5-100)	100
Total	1,084	25	*P* Value for trend 0.003	

CHEST 2010; 137(2):263–272

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)

ESC European Society of Cardiology



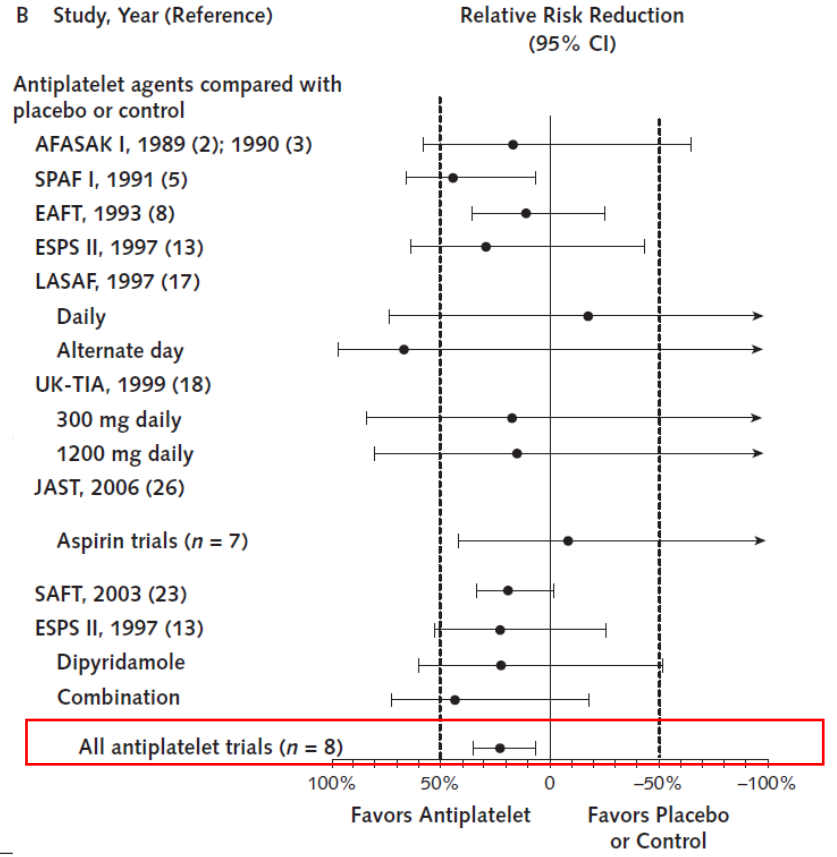


Annals of Internal Medicine | REVIEW

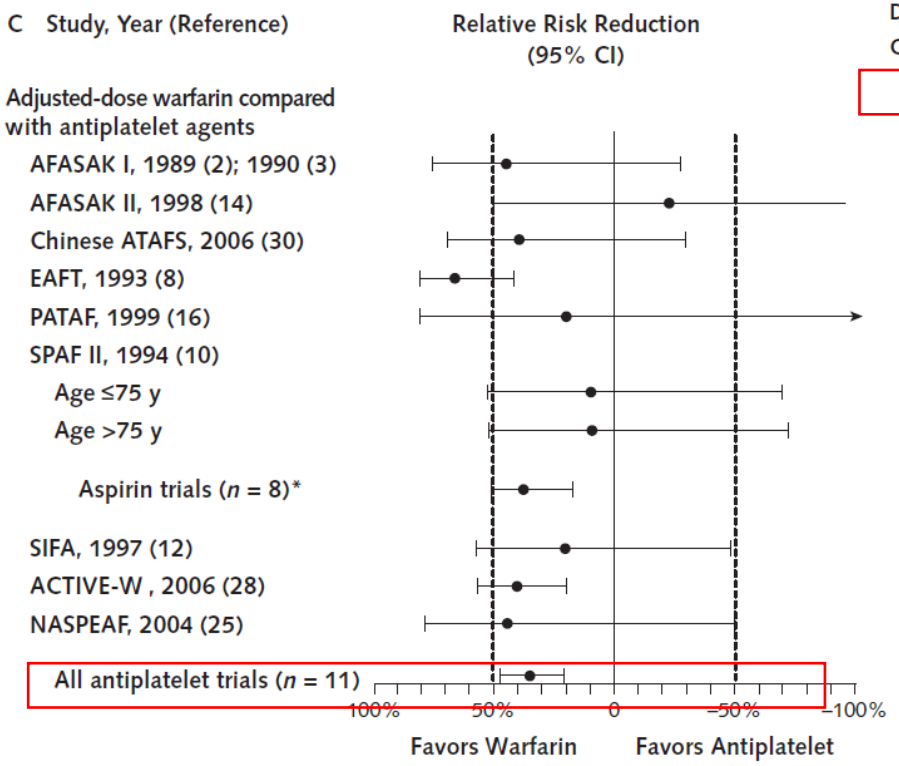
Meta-analysis: Antithrombotic Therapy to Prevent Stroke in Patients Who Have Nonvalvular Atrial Fibrillation

Robert G. Hart, MD; Lesly A. Pearce, MS; and Maria I. Aguilar, MD

Adjusted-dose warfarin and antiplatelet agents reduce stroke by approximately 60% and by approximately 20%, respectively, in patients who have AF.



Warfarin is substantially more efficacious (by approximately 40%) than antiplatelet therapy.



Ann Intern Med. 2007;146:857-867.

ROCKET AF

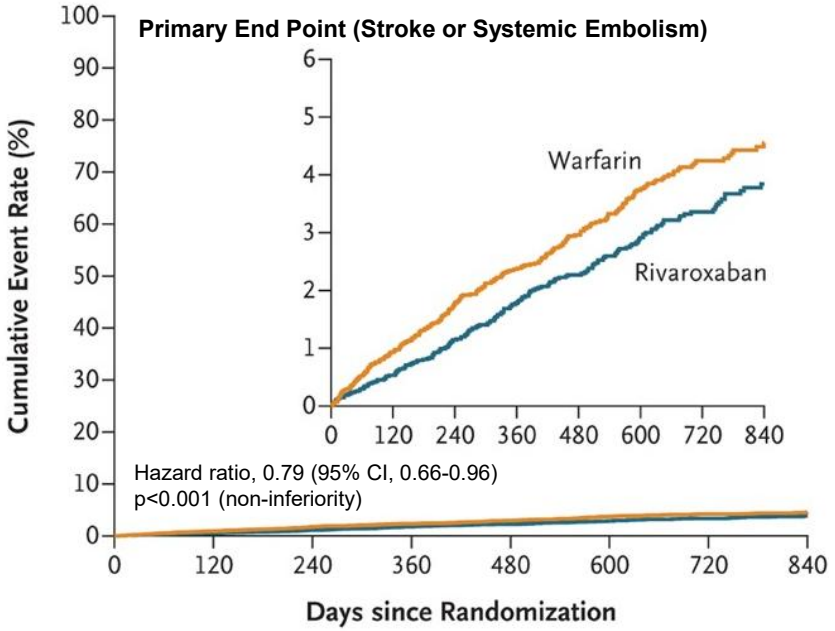
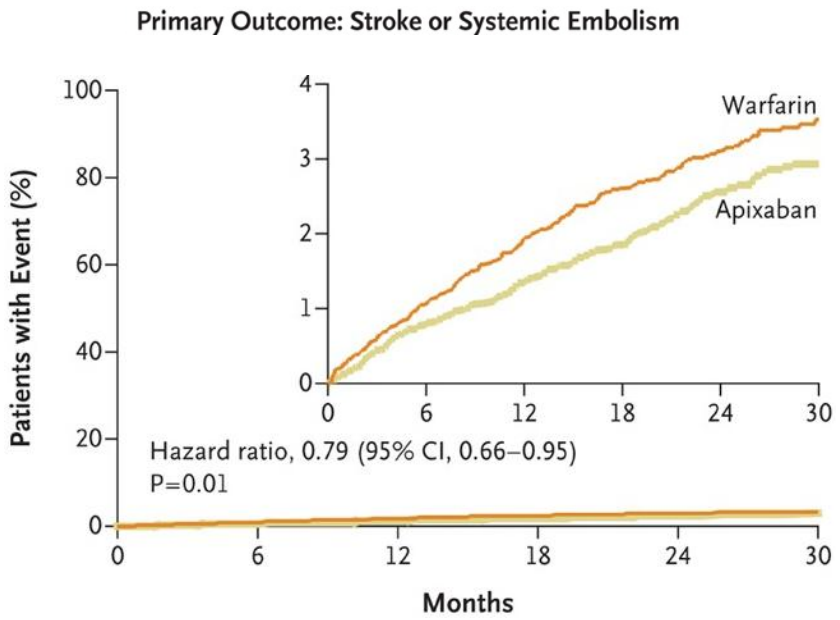


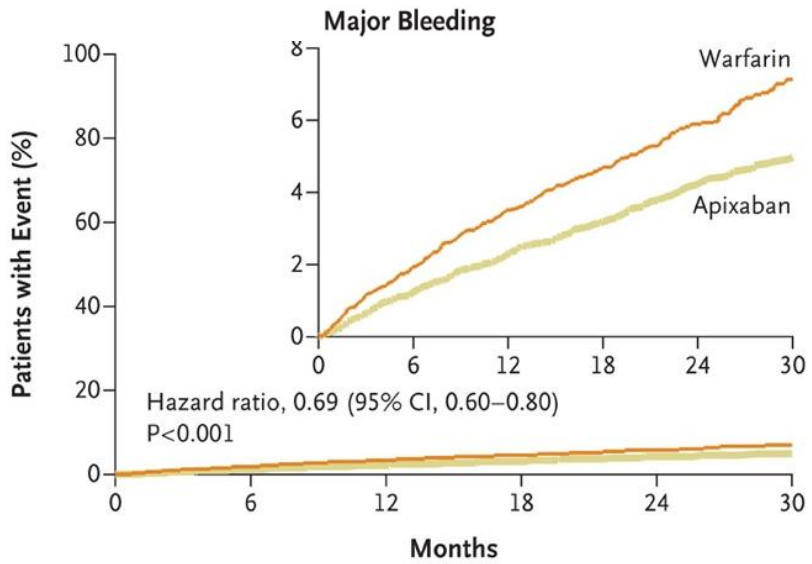
Table 3. Rates of Bleeding Events.*

Variable	Rivaroxaban (N=7111) Events no. (%)	Rivaroxaban (N=7111) Event Rate no./100 patient-yr	Warfarin (N=7125) Events no. (%)	Warfarin (N=7125) Event Rate no./100 patient-yr	Hazard Ratio (95% CI)†	P Value‡
Principal safety end point: major and nonmajor clinically relevant bleeding§	1475 (20.7)	14.9	1449 (20.3)	14.5	1.03 (0.96–1.11)	0.44
Major bleeding						
Any	395 (5.6)	3.6	386 (5.4)	3.4	1.04 (0.90–1.20)	0.58
Decrease in hemoglobin ≥2 g/dl	305 (4.3)	2.8	254 (3.6)	2.3	1.22 (1.03–1.44)	0.02
Transfusion	183 (2.6)	1.6	149 (2.1)	1.3	1.25 (1.01–1.55)	0.04
Critical bleeding¶	91 (1.3)	0.8	133 (1.9)	1.2	0.69 (0.53–0.91)	0.007
Fatal bleeding	27 (0.4)	0.2	55 (0.8)	0.5	0.50 (0.31–0.79)	0.003
Intracranial hemorrhage	55 (0.8)	0.5	84 (1.2)	0.7	0.67 (0.47–0.93)	0.02
Nonmajor clinically relevant bleeding	1185 (16.7)	11.8	1151 (16.2)	11.4	1.04 (0.96–1.13)	0.35

N Engl J Med 2011;365:883-91

ARISTOTLE





N Engl J Med 2011;365:981-92

RE-LY

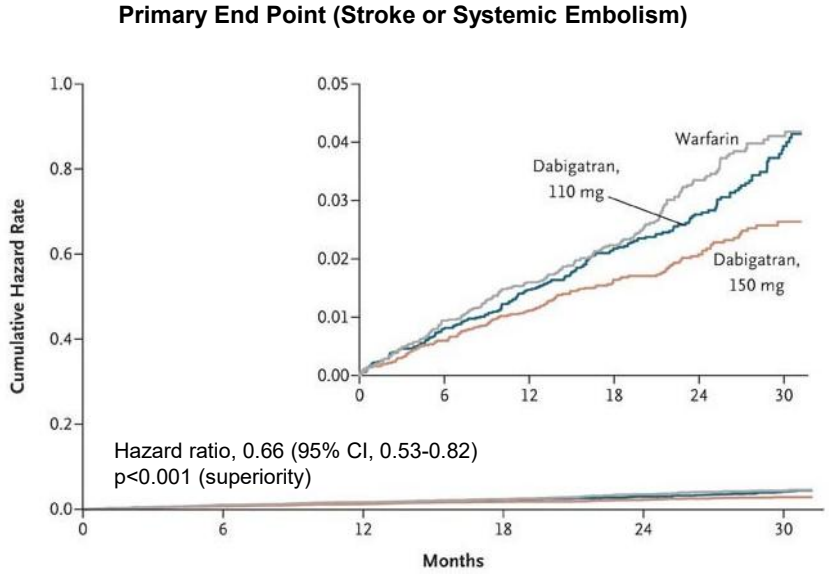
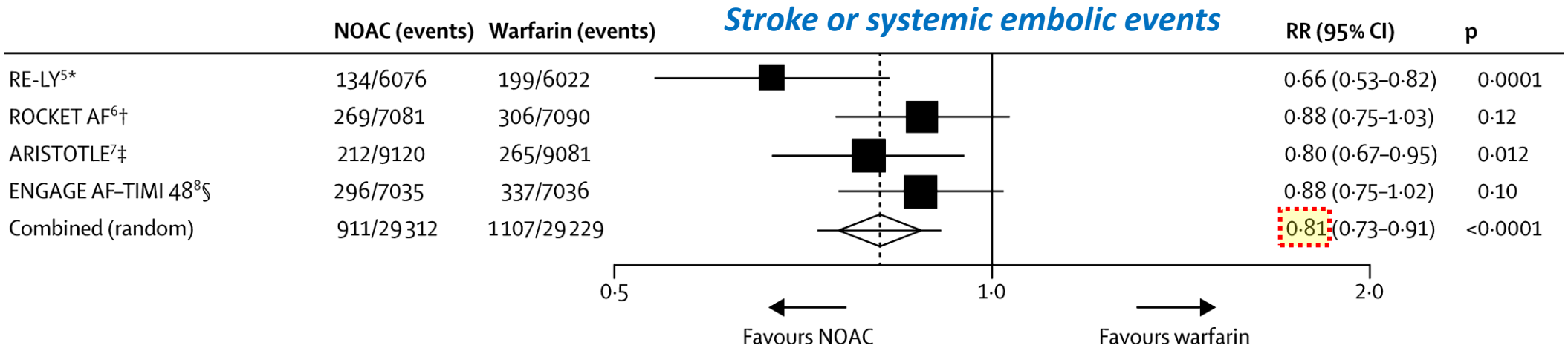


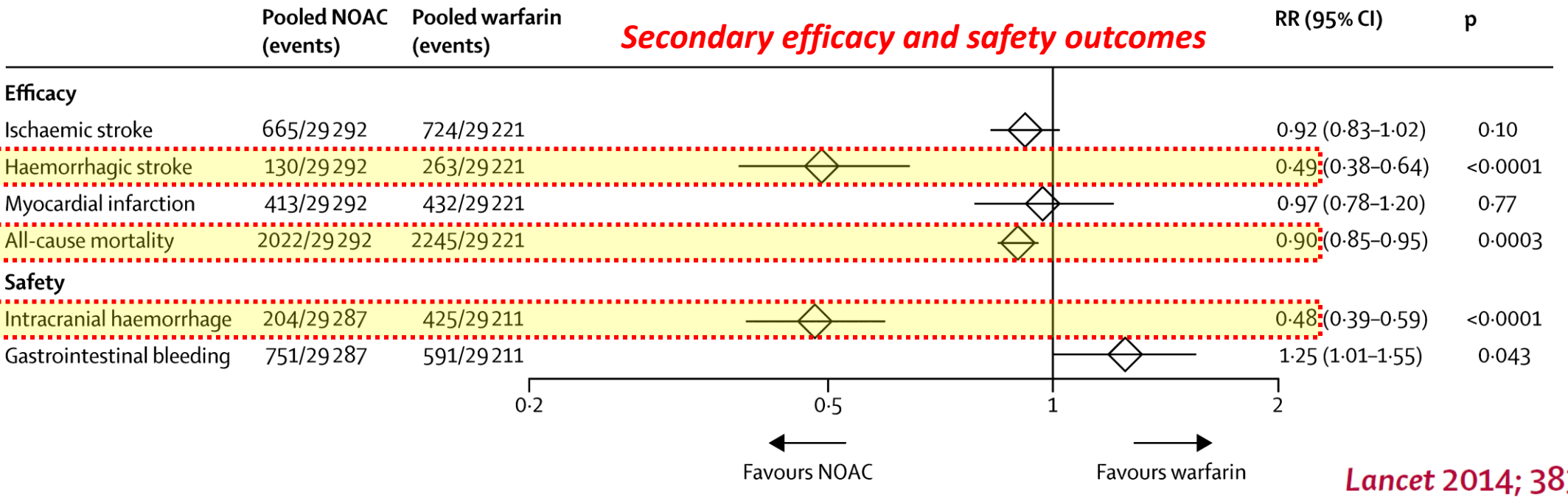
Table 3. Safety Outcomes, According to Treatment Group.*

Event	Dabigatran, 110 mg no. of patients	Dabigatran, 110 mg %/yr	Dabigatran, 150 mg no. of patients	Dabigatran, 150 mg %/yr	Warfarin no. of patients	Warfarin %/yr	Dabigatran, 110 mg, vs. Warfarin Relative Risk (95% CI)	Dabigatran, 110 mg, vs. Warfarin P Value	Dabigatran, 150 mg, vs. Warfarin Relative Risk (95% CI)	Dabigatran, 150 mg, vs. Warfarin P Value
Major bleeding	322	2.71	375	3.11	397	3.36	0.80 (0.69–0.93)	0.003	0.93 (0.81–1.07)	0.31
Life threatening	145	1.22	175	1.45	212	1.80	0.68 (0.55–0.83)	<0.001	0.81 (0.66–0.99)	0.04
Non–life threatening	198	1.66	226	1.88	208	1.76	0.94 (0.78–1.15)	0.56	1.07 (0.89–1.29)	0.47
Gastrointestinal†	133	1.12	182	1.51	120	1.02	1.10 (0.86–1.41)	0.43	1.50 (1.19–1.89)	<0.001
Minor bleeding	1566	13.16	1787	14.84	1931	16.37	0.79 (0.74–0.84)	<0.001	0.91 (0.85–0.97)	0.005
Major or minor bleeding	1740	14.62	1977	16.42	2142	18.15	0.78 (0.74–0.83)	<0.001	0.91 (0.86–0.97)	0.002
Intracranial bleeding	27	0.23	36	0.30	87	0.74	0.31 (0.20–0.47)	<0.001	0.40 (0.27–0.60)	<0.001
Extracranial bleeding	299	2.51	342	2.84	315	2.67	0.94 (0.80–1.10)	0.45	1.07 (0.92–1.25)	0.38
Net clinical benefit outcome‡	844	7.09	832	6.91	901	7.64	0.92 (0.84–1.02)	0.10	0.91 (0.82–1.00)	0.04

N Engl J Med 2009;361:1139-51

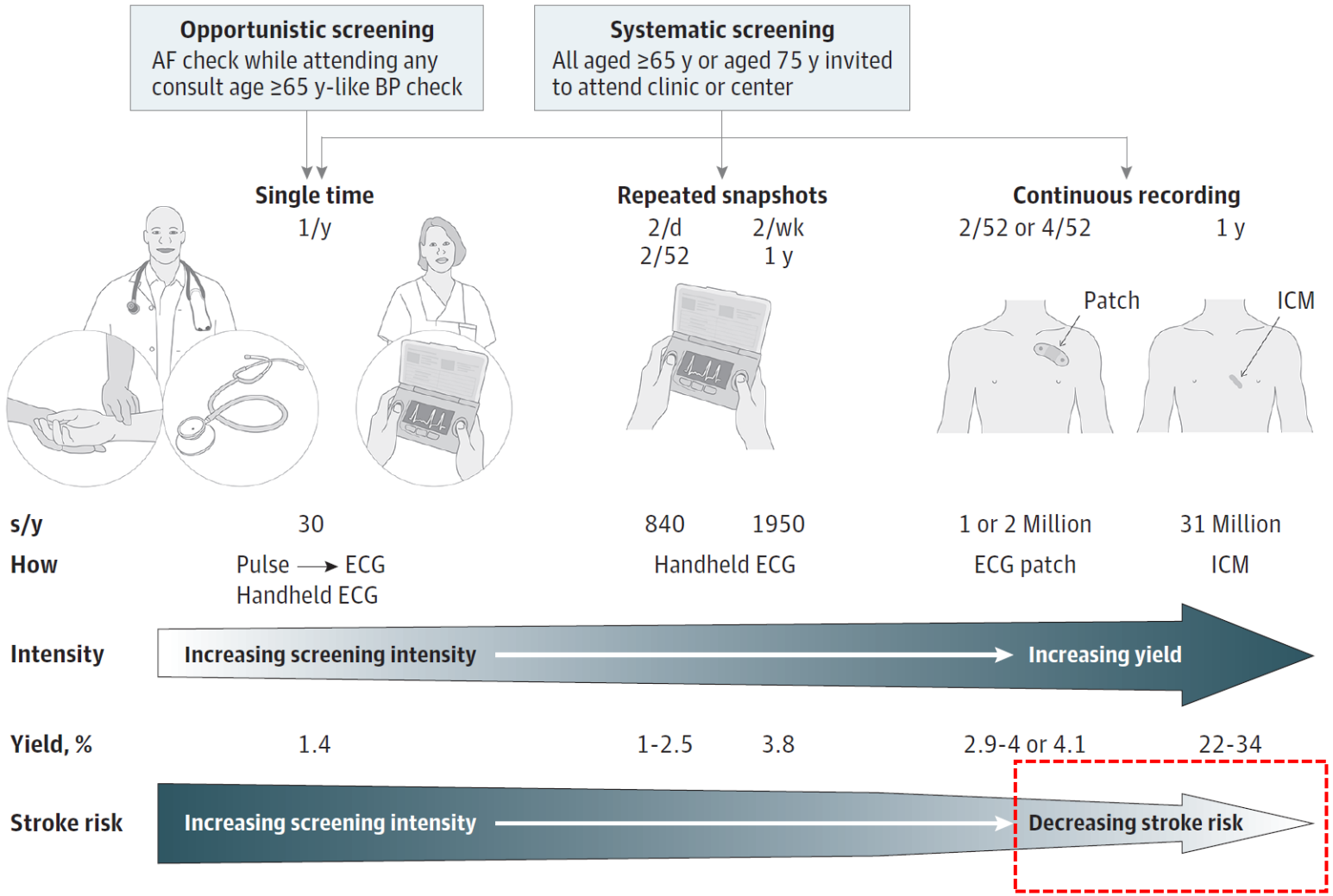
Comparison of the efficacy and safety of new oral anticoagulants with warfarin in patients with atrial fibrillation: a meta-analysis of randomised trials





Lancet 2014; 383: 955–62

Opportunistic Electrocardiogram Screening for Atrial Fibrillation to Prevent Stroke



JAMA Cardiol. 2019, 1;4(2):91-92

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)

ESC European Society of Cardiology

Recommendations	Class[a]	Level[b]
For stroke prevention in AF patients who are eligible for OAC, NOACs are recommended in preference to VKAs (excluding patients with mechanical heart valves or moderate-to-severe mitral stenosis).[423,424]	I	A
For stroke risk assessment, a risk-factor-based approach is recommended, using the CHA_2DS_2-VASc clinical stroke risk score to initially identify patients at 'low stroke risk' (CHA_2DS_2-VASc score = 0 in men, or 1 in women) who should not be offered antithrombotic therapy.[334,388]	I	A
OAC is recommended for stroke prevention in AF patients with CHA_2DS_2-VASc score ≥2 in men or ≥3 in women.[412]	I	A
OAC should be considered for stroke prevention in AF patients with a CHA_2DS_2-VASc score of 1 in men or 2 in women. Treatment should be individualized based on net clinical benefit and consideration of patient values and preferences.[338,378,380]	IIa	B
For bleeding risk assessment, a formal structured risk-score-based bleeding risk assessment is recommended to help identify non-modifiable and address modifiable bleeding risk factors in all AF patients, and to identify patients potentially at high risk of bleeding who should be scheduled for early and more frequent clinical review and follow-up.[388,395,404,406]	I	B

European Heart Journal (2020) **42**, 373–498

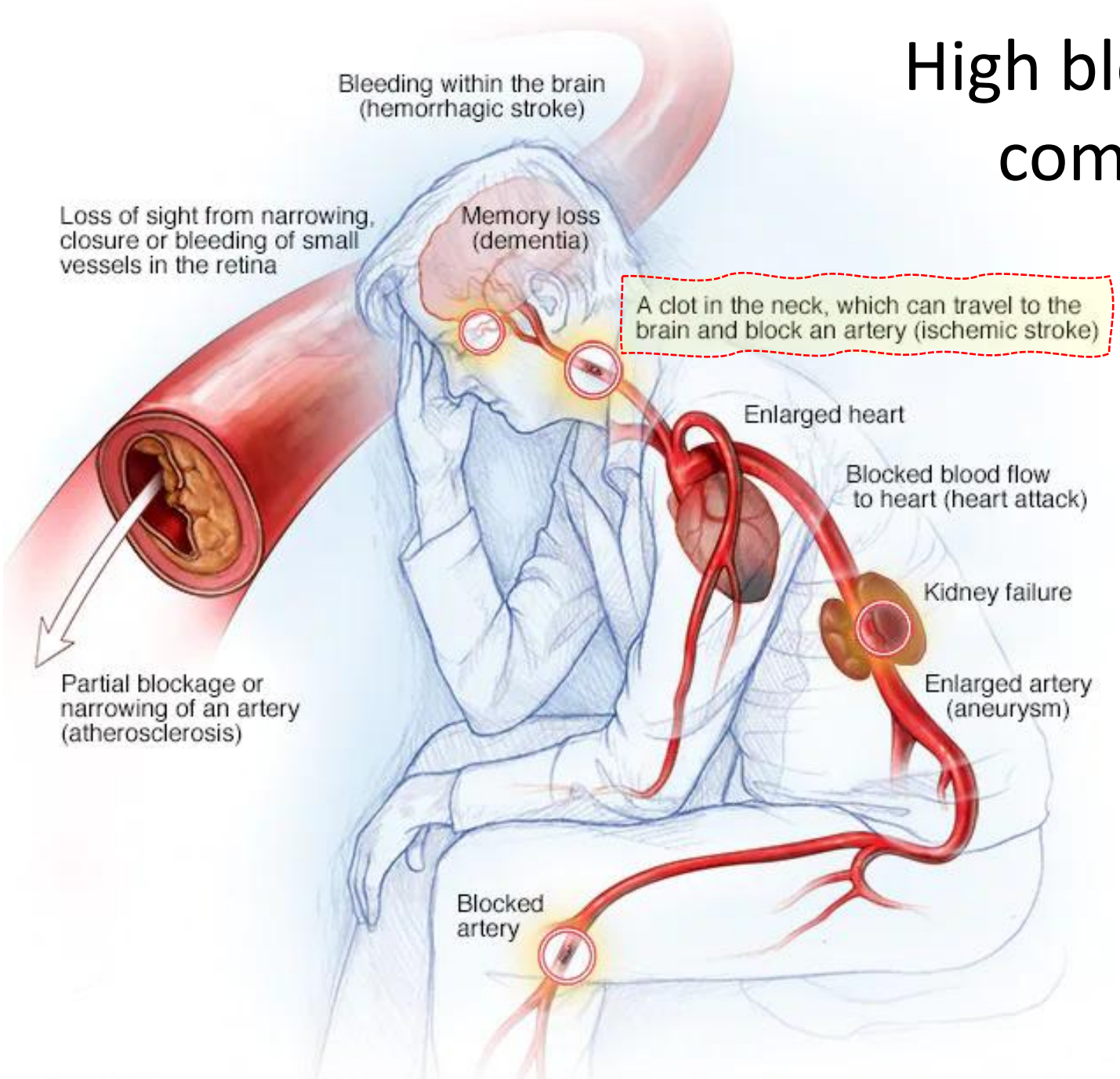
For a formal risk-score-based assessment of bleeding risk, the HAS-BLED score should be considered to help address modifiable bleeding risk factors, and to identify patients at high risk of bleeding (HAS-BLED score ≥3) for early and more frequent clinical review and follow-up.[388,395,404,406]	IIa	B
Stroke and bleeding risk reassessment at periodic intervals is recommended to inform treatment decisions (e.g. initiation of OAC in patients no longer at low risk of stroke) and address potentially modifiable bleeding risk factors.[c389,478,479]	I	B
In patients with AF initially at low risk of stroke, first reassessment of stroke risk should be made at 4 - 6 months after the index evaluation.[385–387]	IIa	B
If a VKA is used, a target INR of 2.0 - 3.0 is recommended, with individual TTR≥70%.[414]	I	B
In patients on VKAs with low time in INR therapeutic range (e.g. TTR<70%), recommended options are: • Switching to a NOAC but ensuring good adherence and persistence with therapy[415,416]; or	I	B
• Efforts to improve TTR (e.g. education/counselling and more frequent INR checks).[480]	IIa	B
Antiplatelet therapy alone (monotherapy or aspirin in combination with clopidogrel) is not recommended for stroke prevention in AF.[440,441,480,481]	III	A
Estimated bleeding risk, in the absence of absolute contraindications to OAC, should not in itself guide treatment decisions to use OAC for stroke prevention.	III	A
Clinical pattern of AF (i.e. first detected, paroxysmal, persistent, long-standing persistent, permanent) should not condition the indication to thromboprophylaxis.[160]	III	B
Recommendations for occlusion or exclusion of the LAA		
LAA occlusion may be considered for stroke prevention in patients with AF and contraindications for long-term anticoagulant treatment (e.g. intracranial bleeding without a reversible cause).[448,449,481,482]	IIb	B
Surgical occlusion or exclusion of the LAA may be considered for stroke prevention in patients with AF undergoing cardiac surgery.[459,483]	IIb	C

Stroke Risk Factors, Genetics, and Prevention

	Nonmodifiable Risk Factors	Modifiable Risk Factors
Ischemic stroke	Age	Hypertension
	Sex	Current smoking
	Race/ethnicity	Waist-to-hip ratio
		Diet
		Physical inactivity
		Hyperlipidemia
		Diabetes mellitus
		Alcohol consumption
		Cardiac causes
		Apolipoprotein B to A1
	Genetics*	

Circ Res. 2017;120:472-495

High blood pressure complications



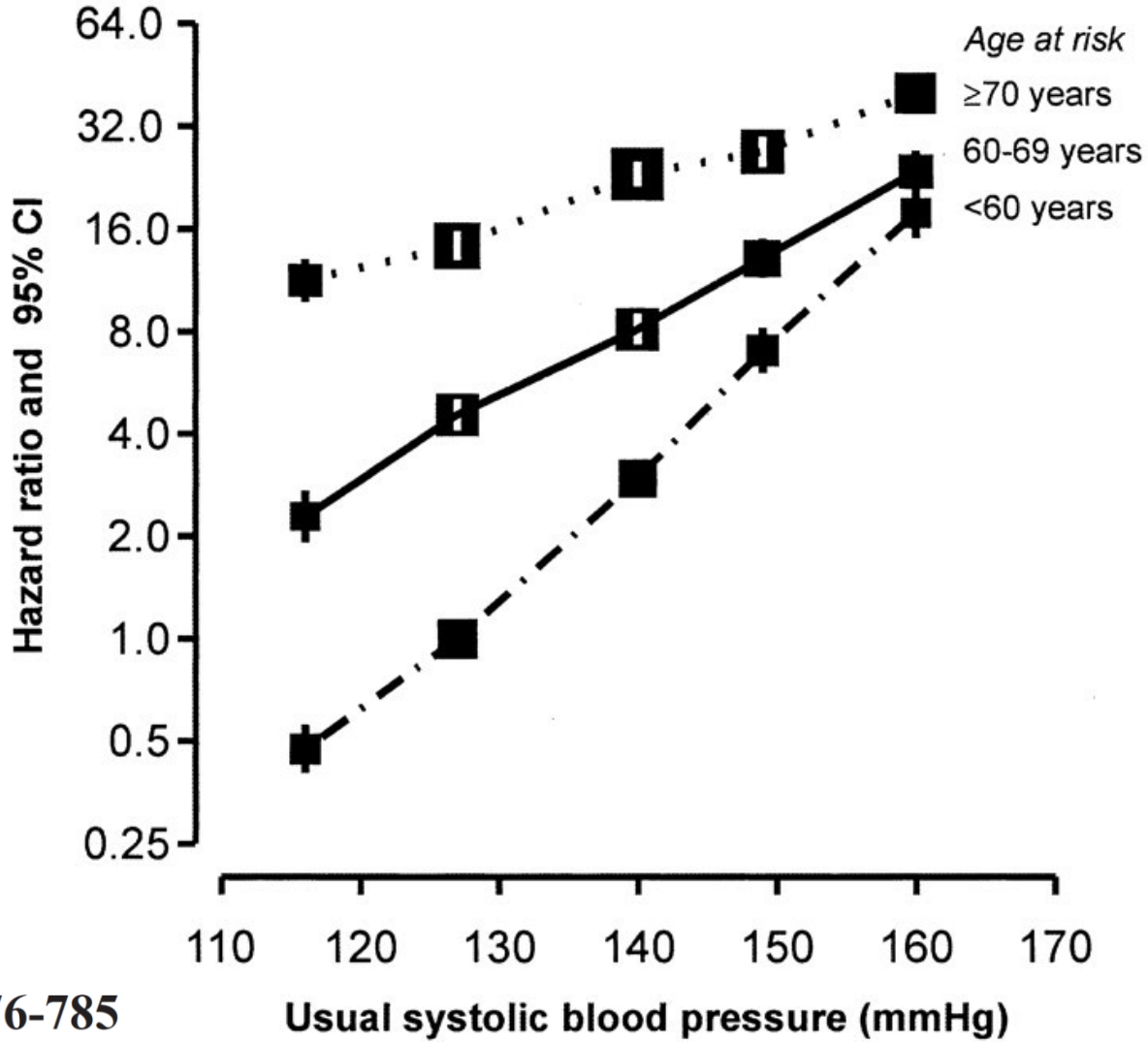
www.mayoclinic.org

Blood Pressure and Stroke

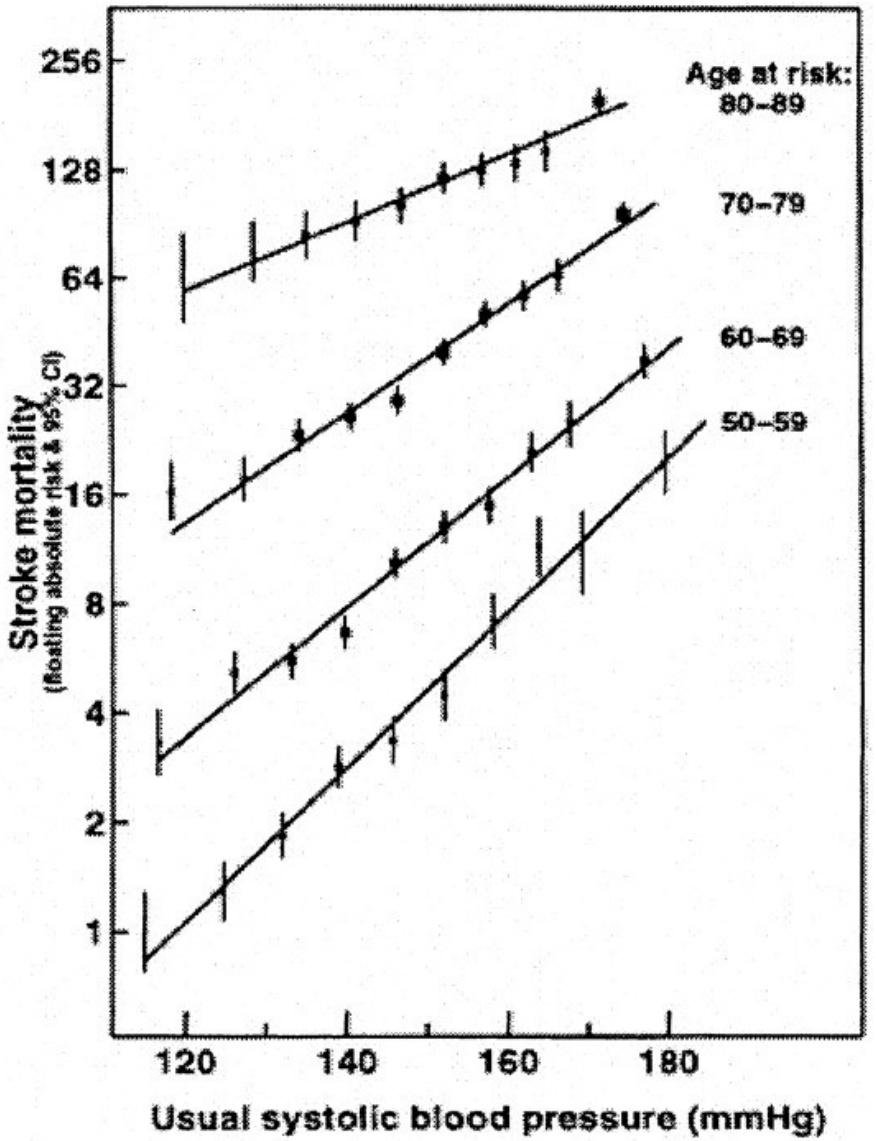
An Overview of Published Reviews

Usual SBP and **risk of stroke** by age, with data from **prospective** cohort study overviews.

Asia Pacific Cohort Studies Collaboration



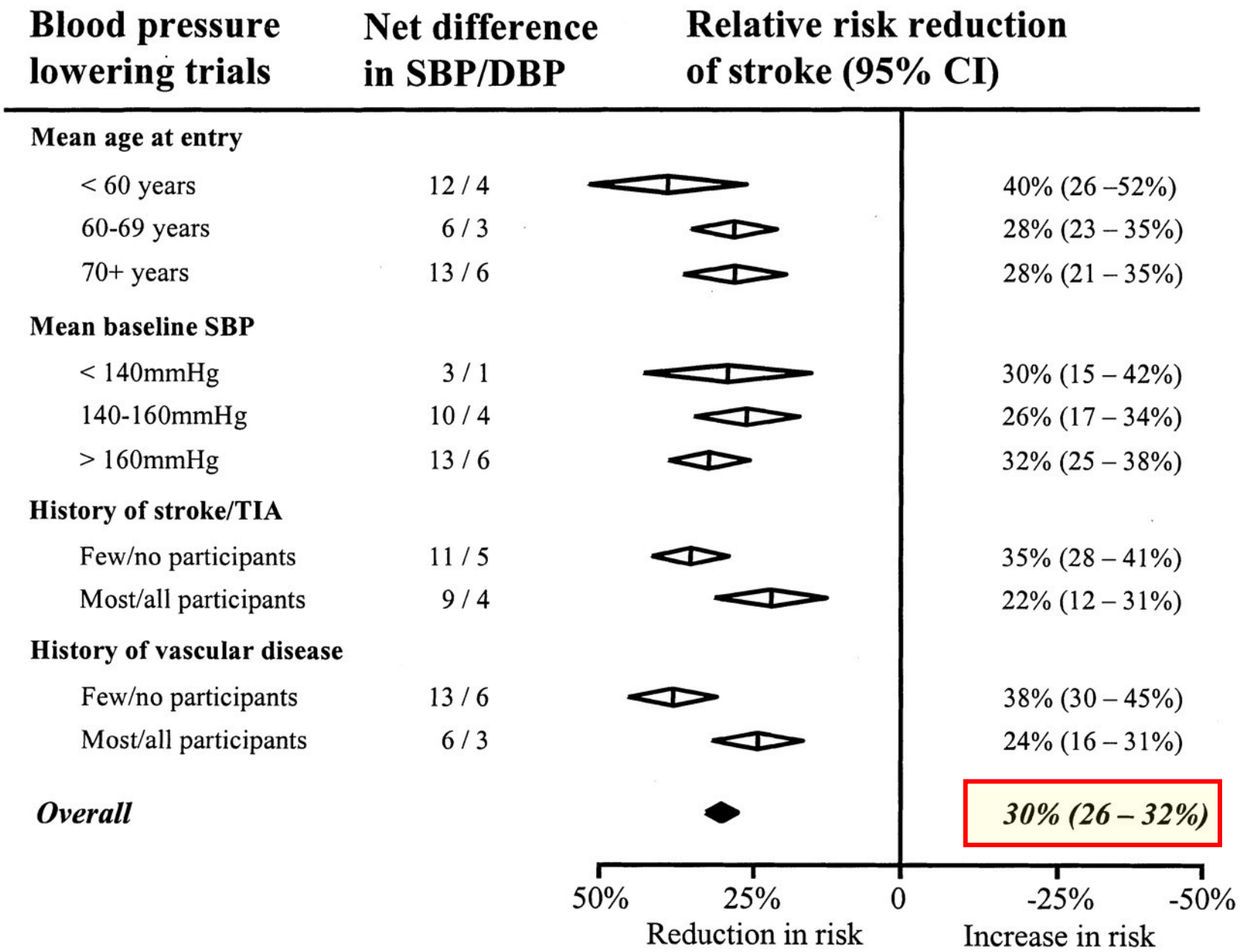
Prospective Studies Collaboration



Stroke. 2004;35:776-785

Blood Pressure and Stroke

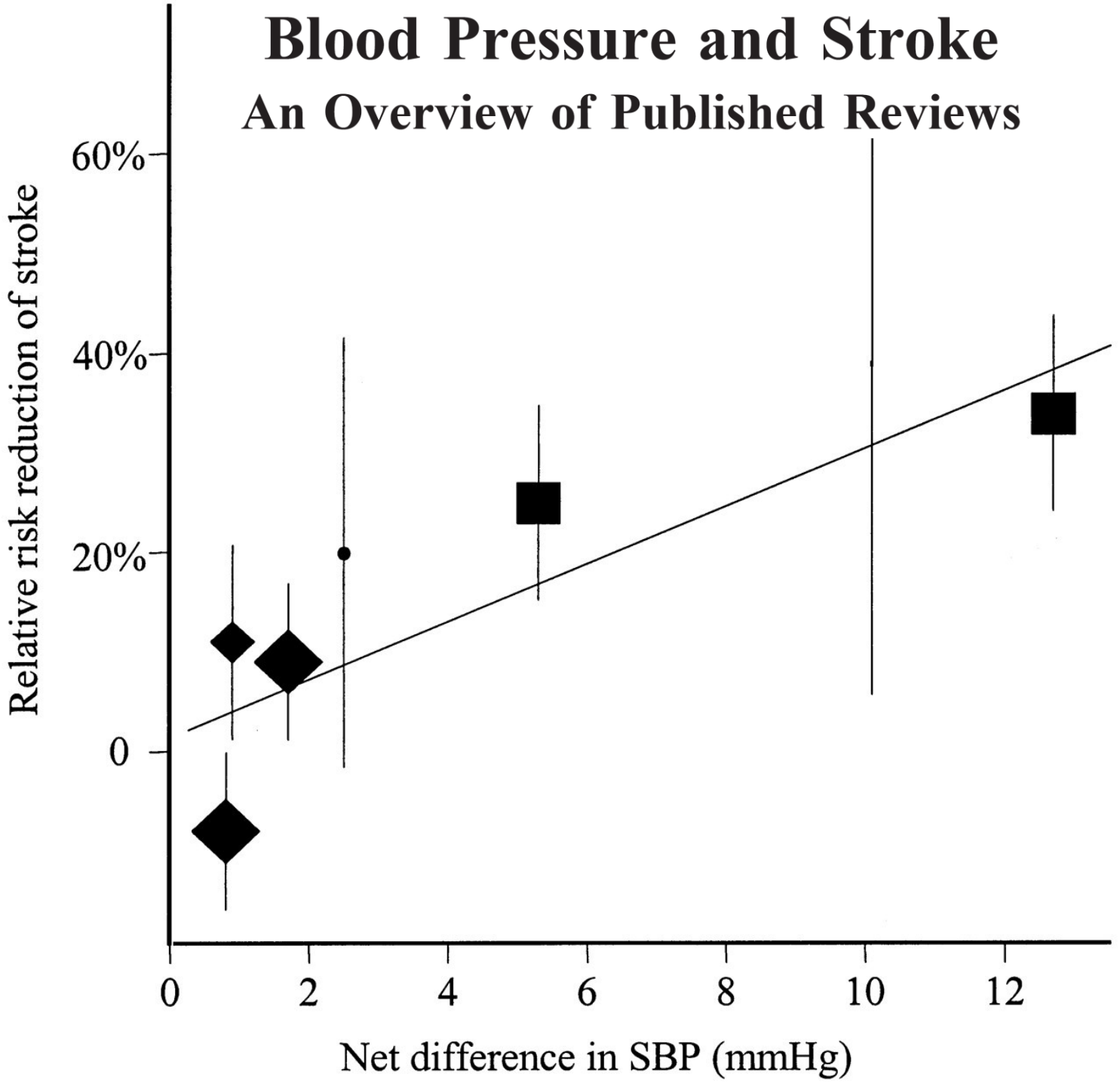
An Overview of Published Reviews



RCTs comparing **antihypertensive drugs** with a placebo (or no treatment) by subgroup

Stroke. 2004;35:776-785

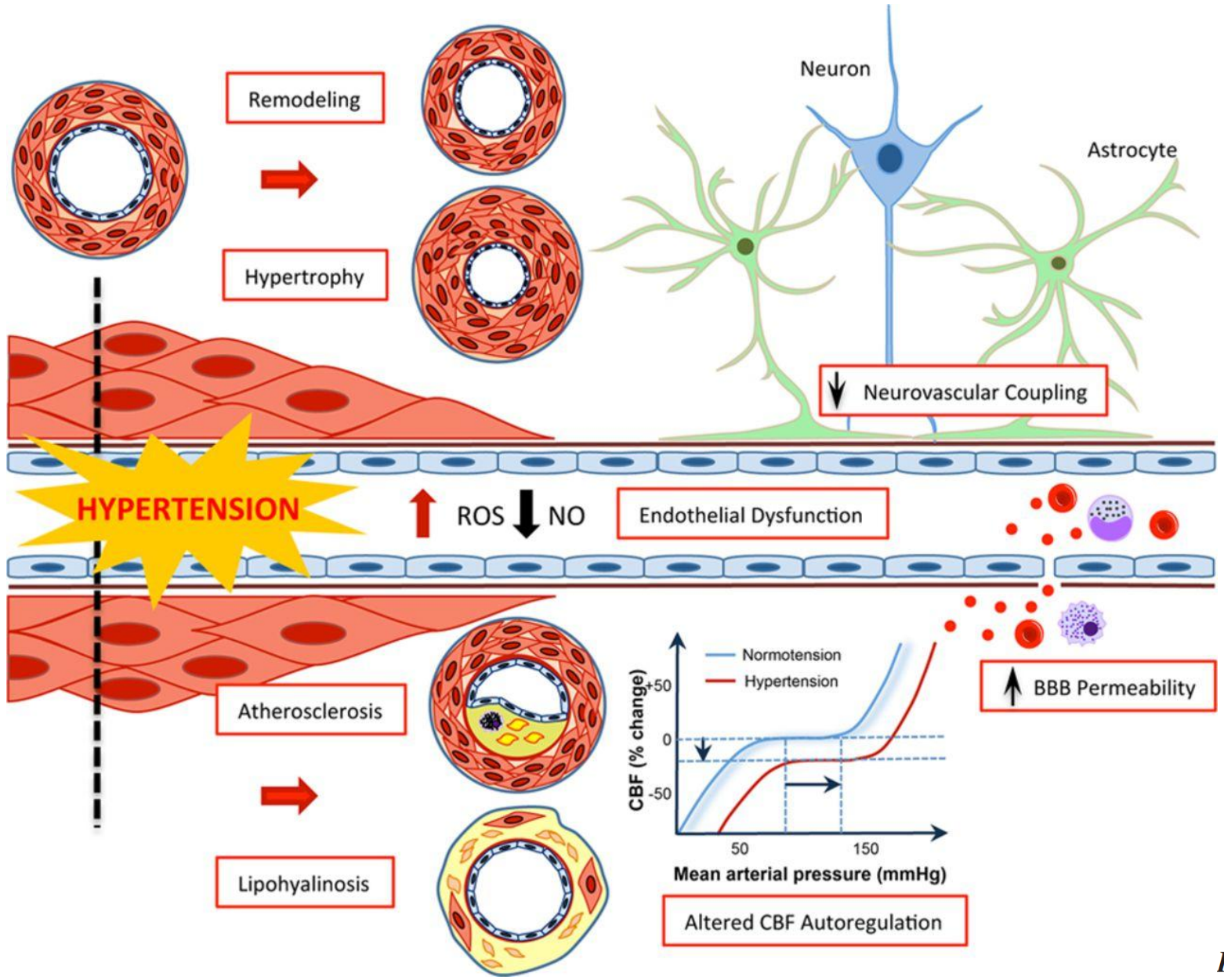
Net **reduction in SBP** and relative risk **reduction in stroke** in RCTs of BP lowering



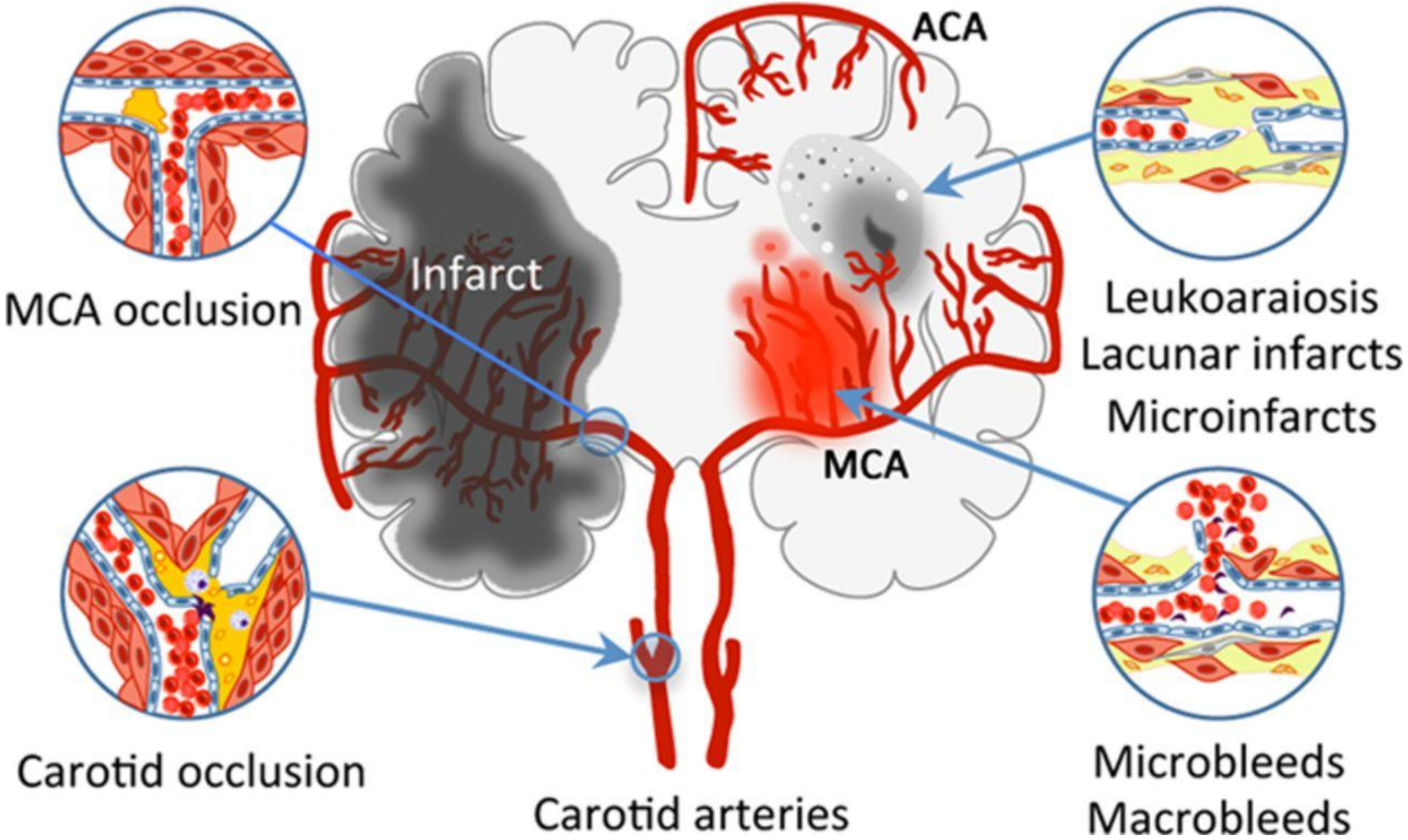
Stroke. 2004;35:776-785

Hypertension

A Harbinger of Stroke and Dementia

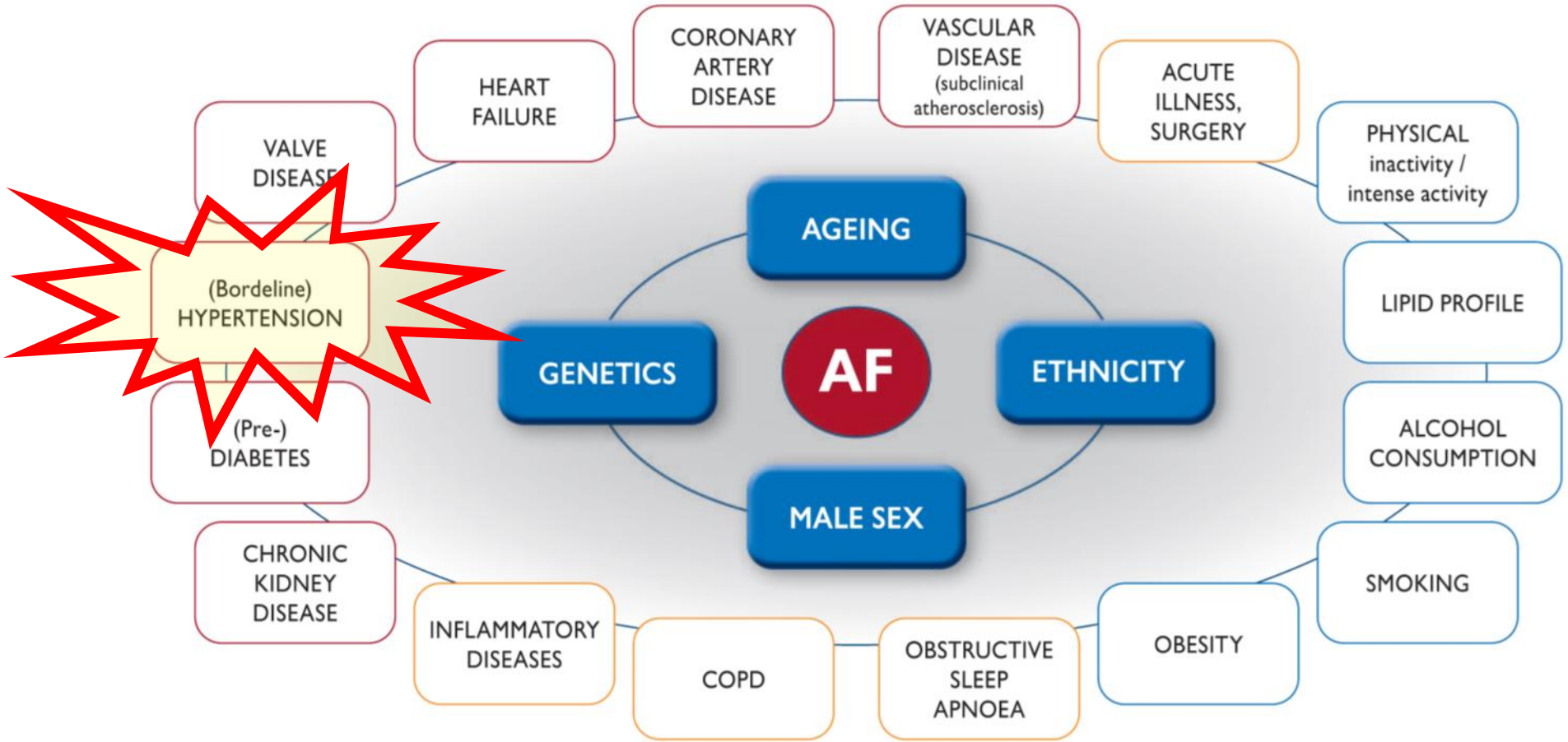


Hypertension. 2013;62:810-817



Hypertension. 2013;62:810-817

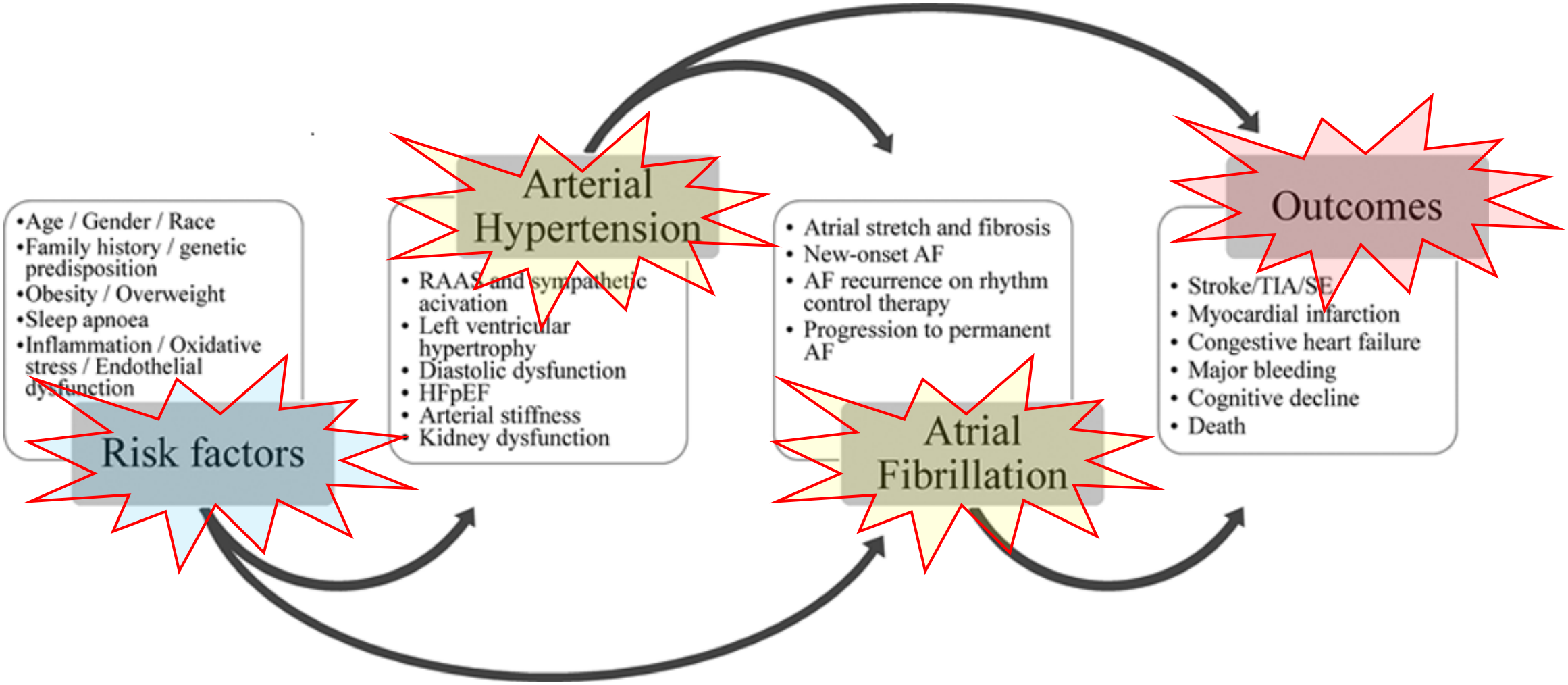
Summary of risk factors for incident AF



*Hypertension is the most common aetiological factor associated with the development of AF, and patients with hypertension have a **1.7-fold higher risk** of developing AF compared with normotensives*

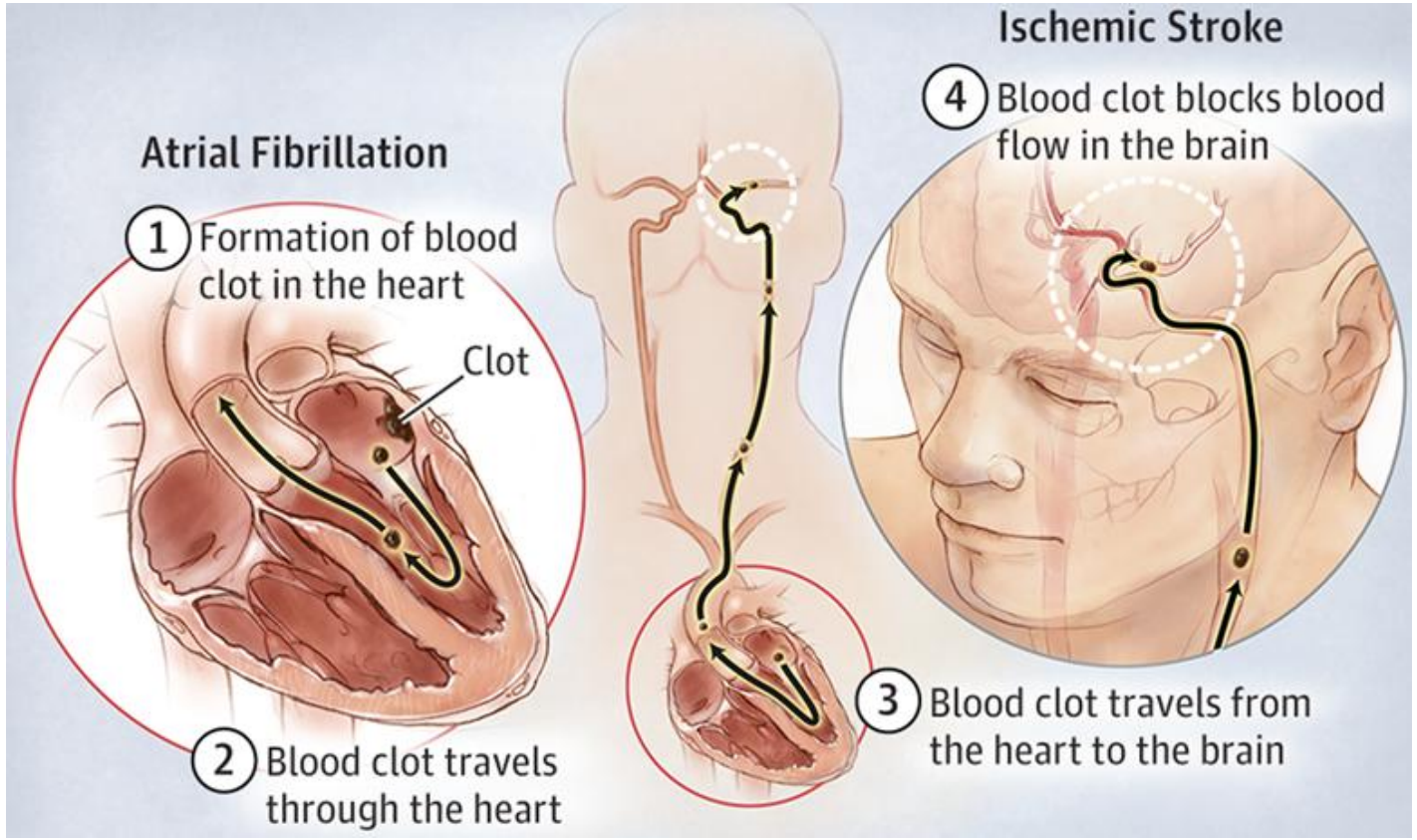
Atrial Fibrillation and Hypertension

Hypertension and atrial fibrillation axis in the cardiovascular disease continuum



Hypertension. 2017;70:854-861

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)



CHA_2DS_2-VASc score Risk factors and definitions		Points awarded
C	**Congestive heart failure** Clinical HF, or objective evidence of moderate to severe LV dysfunction, or HCM	1
H	**Hypertension** or on antihypertensive therapy	1
A	**Age 75 years or older**	2
D	**Diabetes mellitus** Treatment with oral hypoglycaemic drugs and/or insulin or fasting blood glucose >125 mg/dL (7 mmol/L)	1
S	**Stroke**Previous stroke, TIA, or thromboembolism	2
V	**Vascular disease** Angiographically significant CAD, previous myocardial infarction, PAD, or aortic plaque	1
A	**Age 65 – 74 years**	1
Sc	**Sex category (female)**	1
Maximum score		**9**

European Heart Journal (2020) **42**, 373–498

Ischemic stroke is an etiologically heterogeneous syndrome

Stroke due to Small Vessel Disease (SVD)

ISCHEMIC STROKE

Non-SVD, non-embolic Stroke

Embolic Stroke

Non-thrombosis-mediated embolism

Non-thrombotic cardiogenic embolism

Valvular Disease

Chamber embolism

Cardiogenic embolism

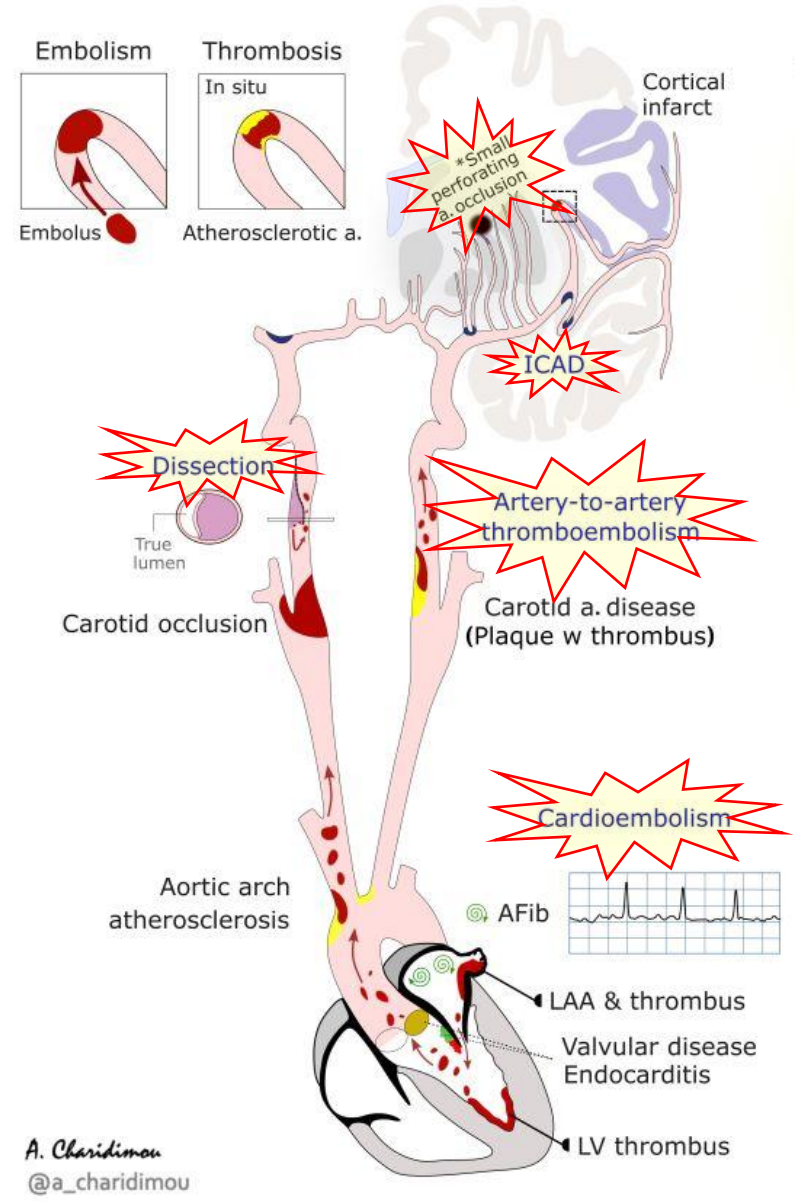
Atherosclerotic

Non-Atherosclerotic

Arteriogenic embolism

Paradoxical embolism

Thrombosis-mediated embolism



Ntaios G, Hart RG. Embolic stroke. Circulation. 2017;136:2403e2405.

ESC European Society of Cardiology

Summary of office blood pressure thresholds for treatment

Age group	Office SBP treatment threshold (mmHg)					Office DBP treatment threshold (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke/TIA	
18 - 65 years	≥140	≥140	≥140	≥140[a]	≥140[a]	≥90
65 - 79 years	≥140	≥140	≥140	≥140[a]	≥140[a]	≥90
≥80 years	≥160	≥160	≥160	≥160	≥160	≥90
Office DBP treatment threshold (mmHg)	≥90	≥90	≥90	≥90	≥90	

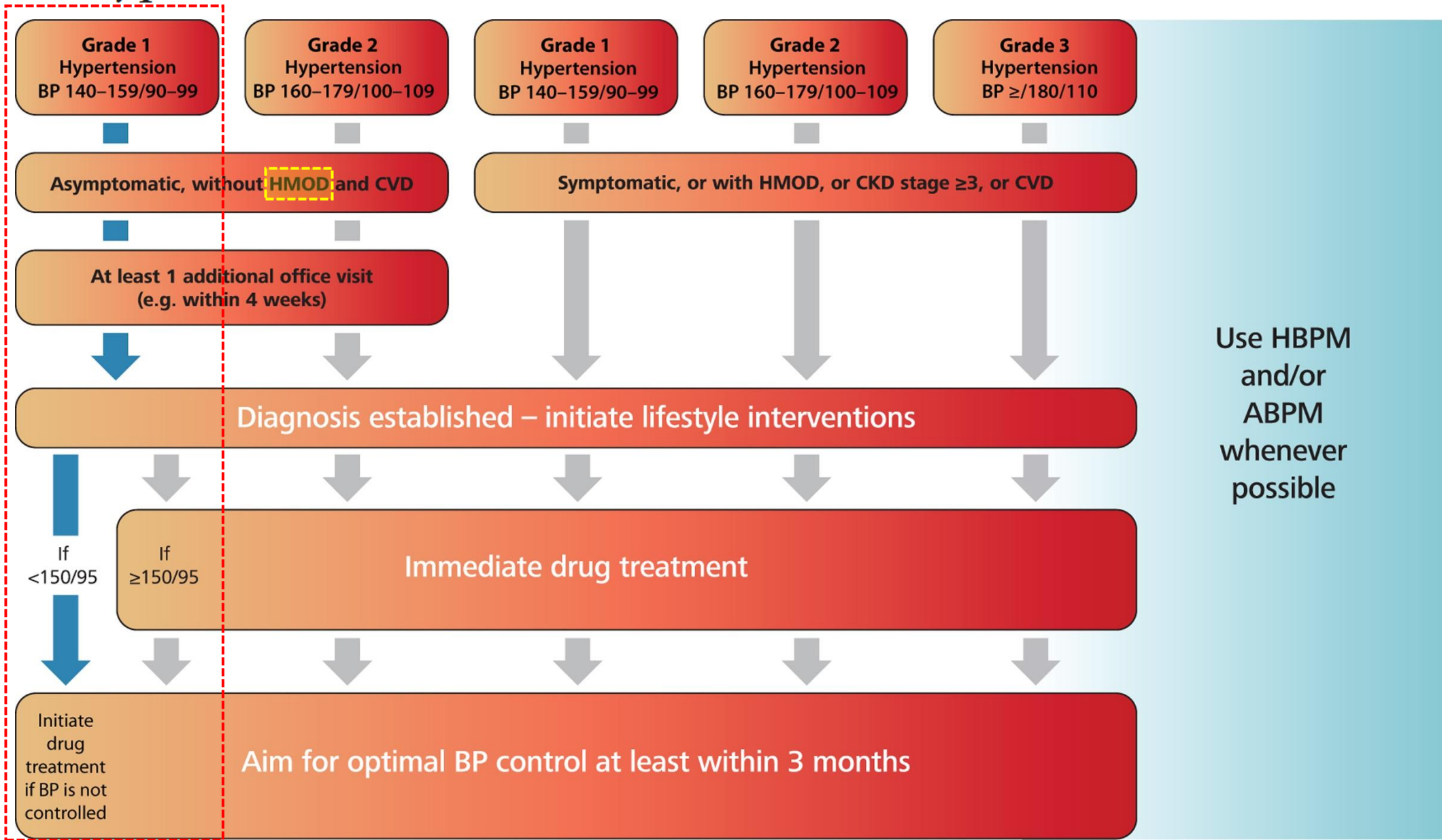
BP = blood pressure; CAD = coronary artery disease; CKD = chronic kidney disease; DBP = diastolic blood pressure; SBP = systolic blood pressure; TIA = transient ischaemic attack.

[a]Treatment may be considered in these very high-risk patients with high–normal SBP (i.e. SBP 130–140 mmHg).

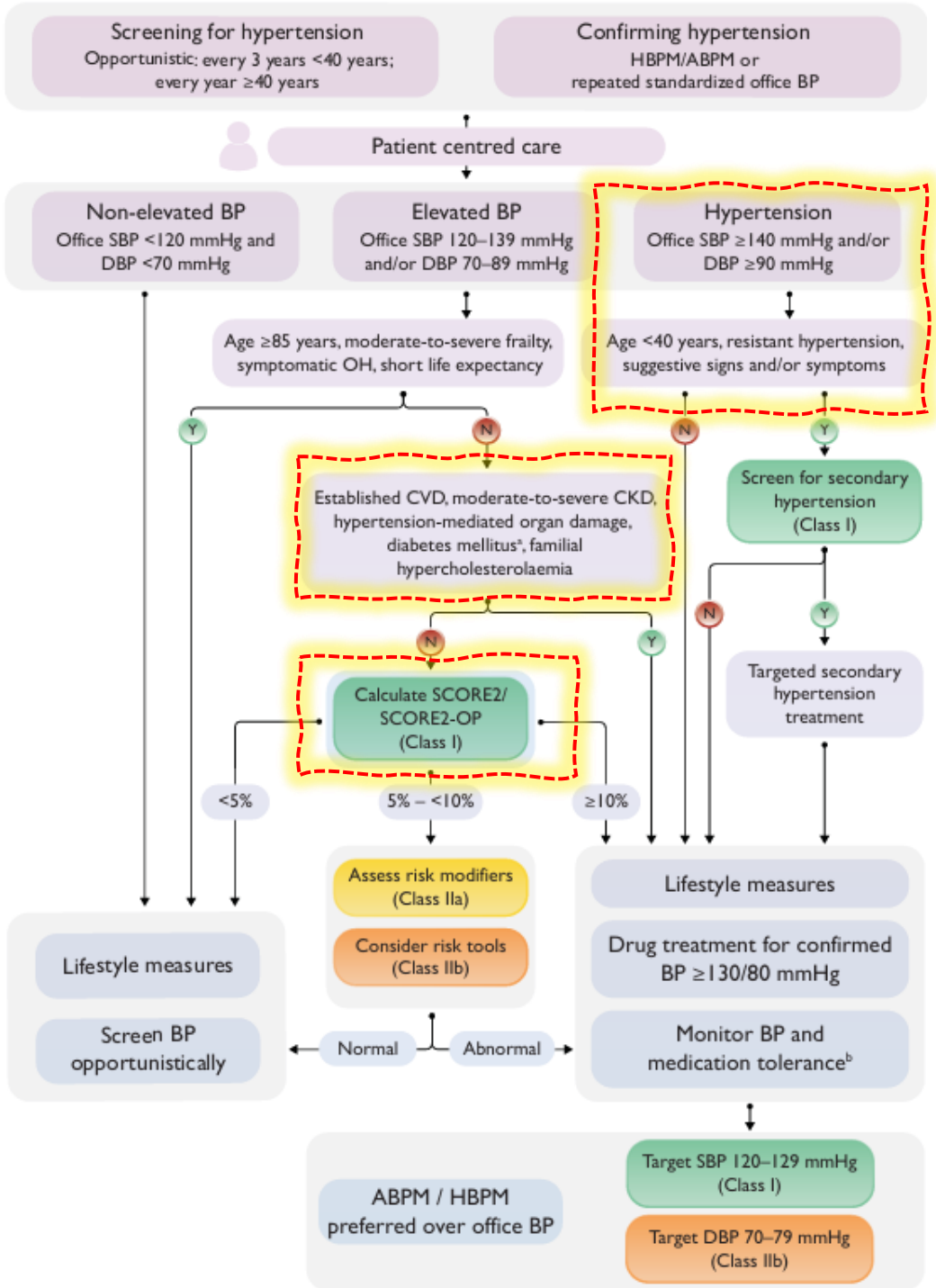
European Heart Journal (2018) **39**, 3021–3104

2023 ESH Guidelines for the management of arterial hypertension

Journal of Hypertension 2023, 41:1874–2071



2024 ESC Guidelines for the management of elevated blood pressure and hypertension



European Heart Journal (2024) **45**, 3912–4018

Stroke Risk Factors, Genetics, and Prevention

	Nonmodifiable Risk Factors	Modifiable Risk Factors
Ischemic stroke	Age	Hypertension
	Sex	Current smoking
	Race/ethnicity	Waist-to-hip ratio
		Diet
		Physical inactivity
		Hyperlipidemia
		Diabetes mellitus
		Alcohol consumption
		Cardiac causes
		Apolipoprotein B to A1
	Genetics*	

Circ Res. 2017;120:472-495

Prospective Associations of Fasting Insulin, Body Fat Distribution, and Diabetes With Risk of Ischemic Stroke

ATHEROSCLEROSIS RISK IN COMMUNITIES (ARIC) STUDY

Table 2—*Relative risks of ischemic stroke in relation to diabetes estimated from multivariable proportional hazards models (ARIC)*

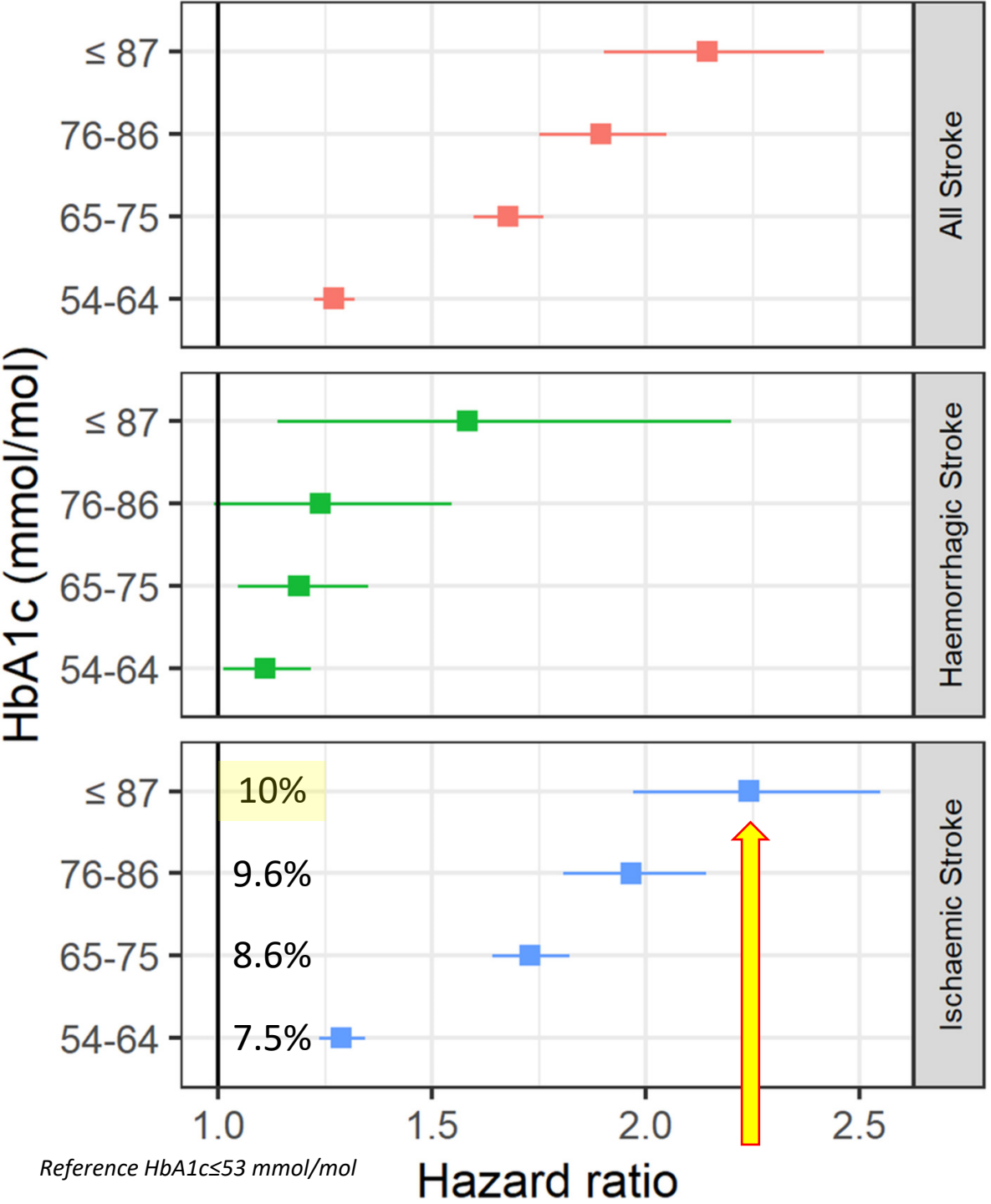
		Diabetes using fasting glucose ≥140 mg/dl			Diabetes using fasting glucose ≥126 mg/dl		
Model and adjustment variables	Events (*n*)	RR†	95% CI	*P* value	RR†	95% CI	*P* value
1. Age, sex, race, ARIC community, smoking, and education	187	3.70	2.7–5.1	<0.0001	3.23	2.4–4.4	<0.0001
2. Model 1 plus systolic blood pressure and antihypertensives	183	2.96	2.1–4.1	<0.0001	2.56	1.8–3.5	<0.0001
3. Model 2 plus HDL and LDL cholesterol	176	2.58	1.8–3.7	<0.0001	2.21	1.6–3.1	<0.0001
4. Model 3 plus von Willebrand factor	175	2.26	1.6–3.2	<0.0001	1.94	1.4–2.8	0.0002
5. Model 4 plus waist-to-hip ratio	175	2.22	1.5–3.2	<0.0001	1.90	1.3–2.7	0.0004

Also included as diabetes: nonfasting glucose ≥200 mg/dl, physician diagnosis of diabetes, or use of hypoglycemic medication. †The reference group is subjects without diabetes. RR, relative risk.

*The association of diabetes with ischemic stroke was strong, with relative risks of **2.0–4.0***

Diabetes Care 22:1077–1083, 1999

Risk of first stroke in people with type 2 diabetes and its relation to glycaemic control: A nationwide observational study



The risk of a first stroke with every 10mmol/mol (1%) increase in HbA1c category to a **more-than-double risk** (adjusted HR 2.14, 95% CI 1.90-2.42) in people with the highest HbA1c levels (10%) compared with the reference group (7%)

Outcome of a first stroke divided into ischaemic and haemorrhagic strokes in 406,271 people with type 2 diabetes in Sweden, from 1998-2015, according to glycaemic control

Diabetes Obes Metab. 2020;22:182–190

LONG-TERM COMPLICATIONS OF DIABETES MELLITUS

THE NEW ENGLAND JOURNAL OF MEDICINE June 10, 1993

ABC of arterial and venous disease

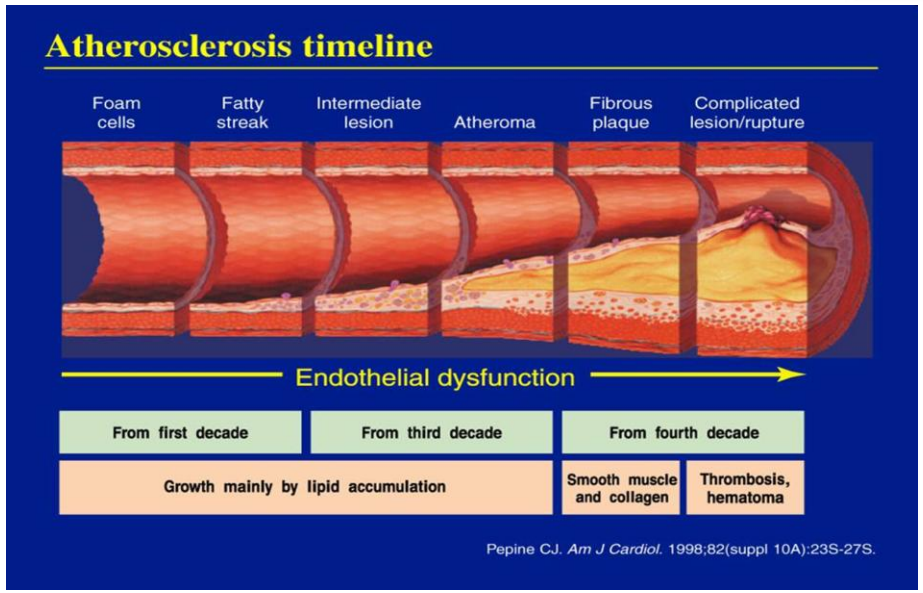
Vascular complications of diabetes

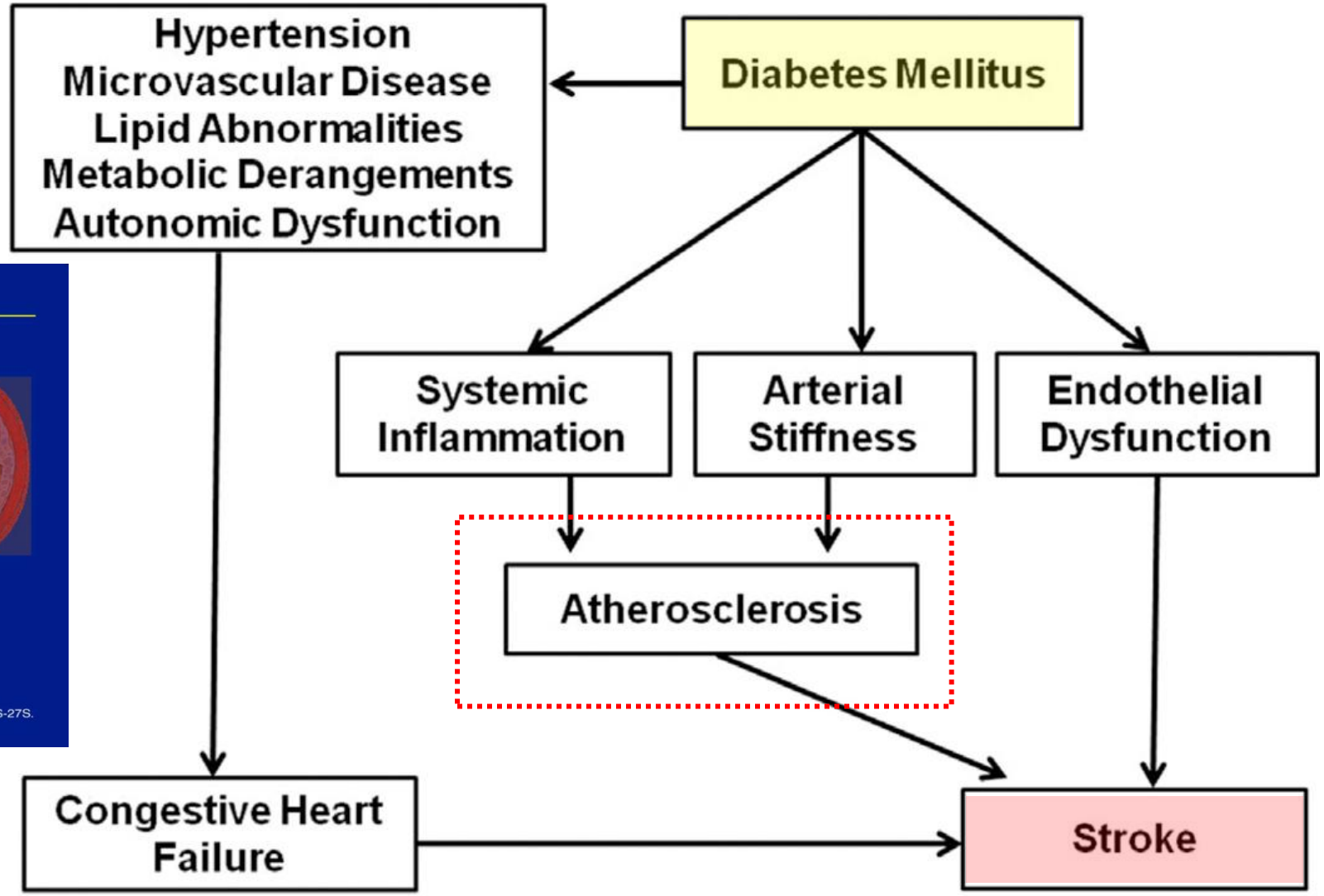
BMJ VOLUME 320 15 APRIL 2000

Vascular complications of diabetes

Microvascular	**Macrovascular**
Retinopathy	Ischaemic heart disease
Nephropathy	Stroke
Neuropathy	Peripheral vascular disease

Diabetes and Stroke: Epidemiology, Pathophysiology, Pharmaceuticals and Outcomes

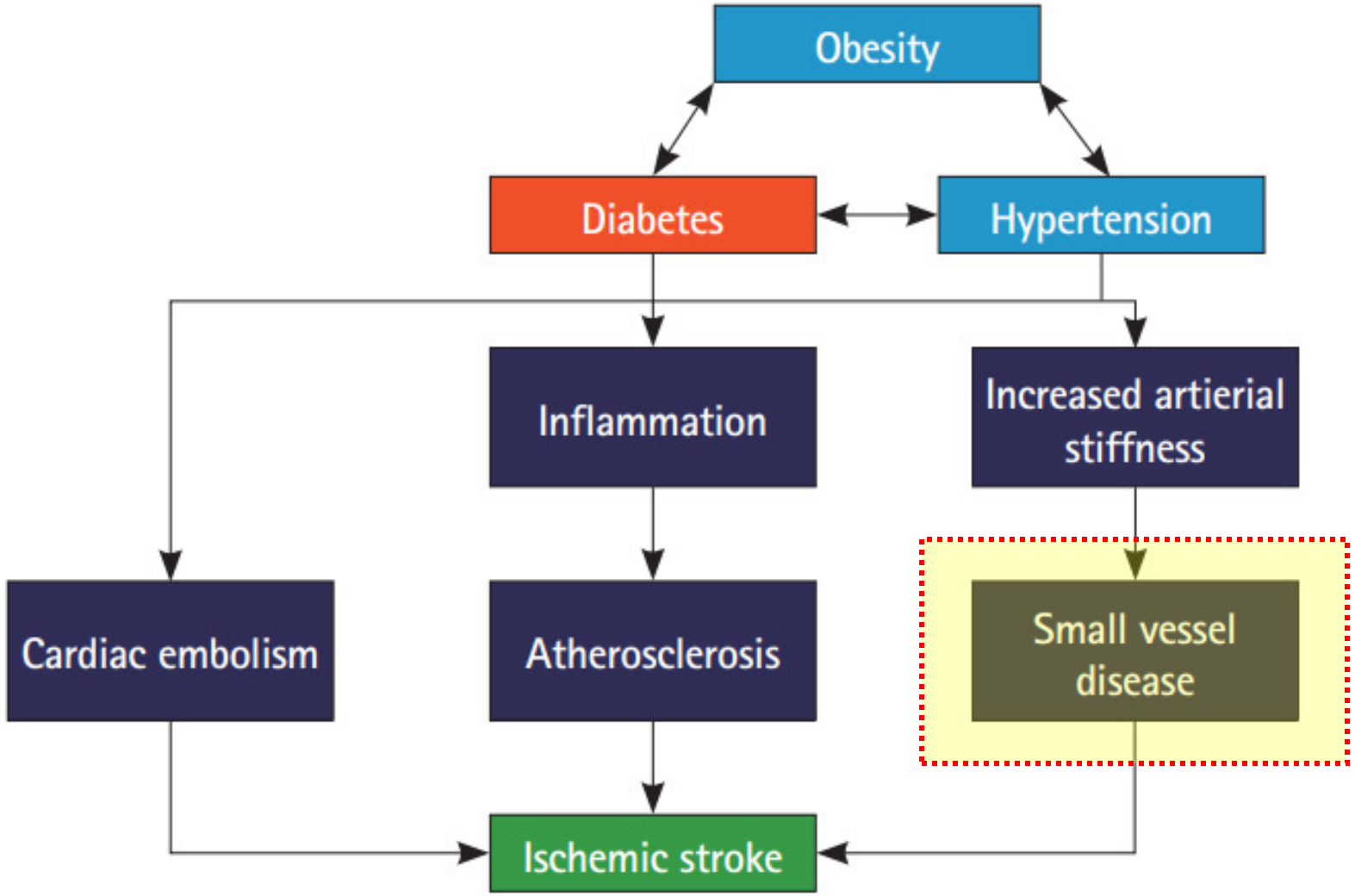




Possible mechanisms of stroke in individuals with diabetes

Am J Med Sci. 2016 April ; 351(4): 380–386

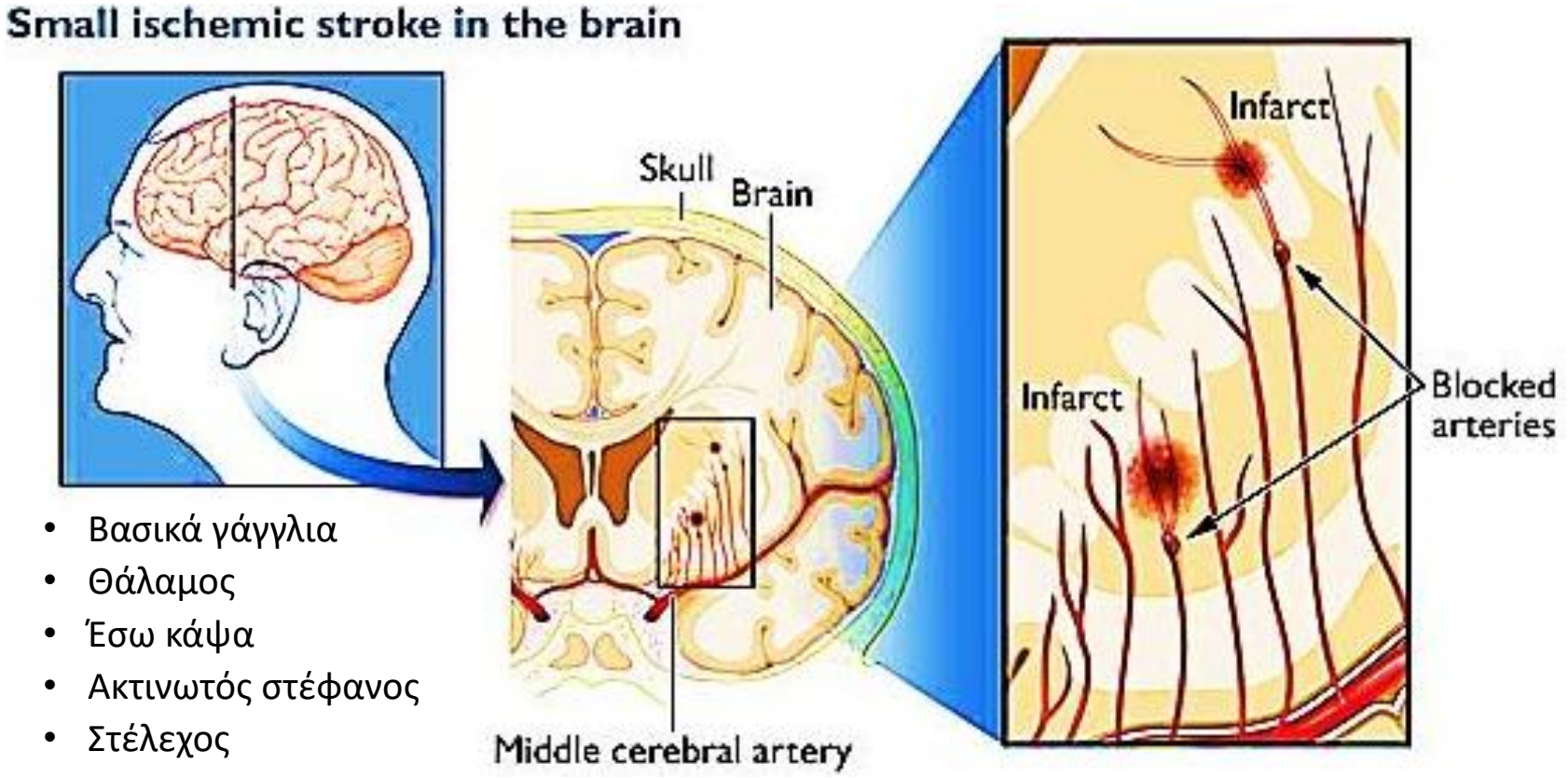
Diabetes and Stroke: What Are the Connections?



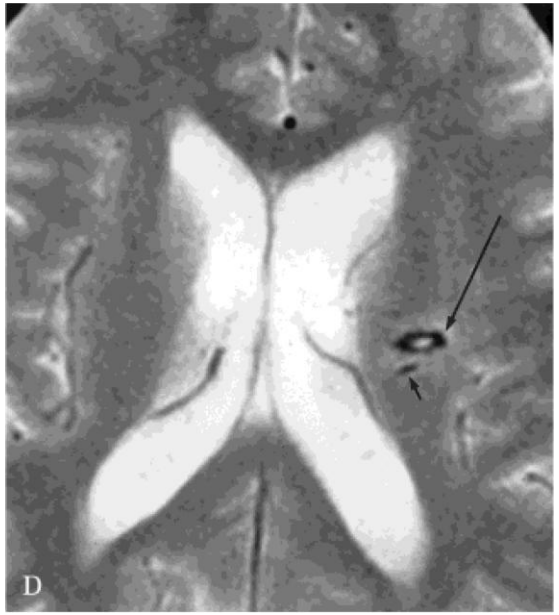
Journal of Stroke 2023;25(1):26-38

Νόσος Μικρών Αγγείων
Κενοχωριώδη → Lacunar

«Μικρά έμφρακτα (<15mm) της υποφλοιώδους περιοχής λόγω απόφραξης μεμονωμένων μικρών διατιτραίνοντων κλάδων»

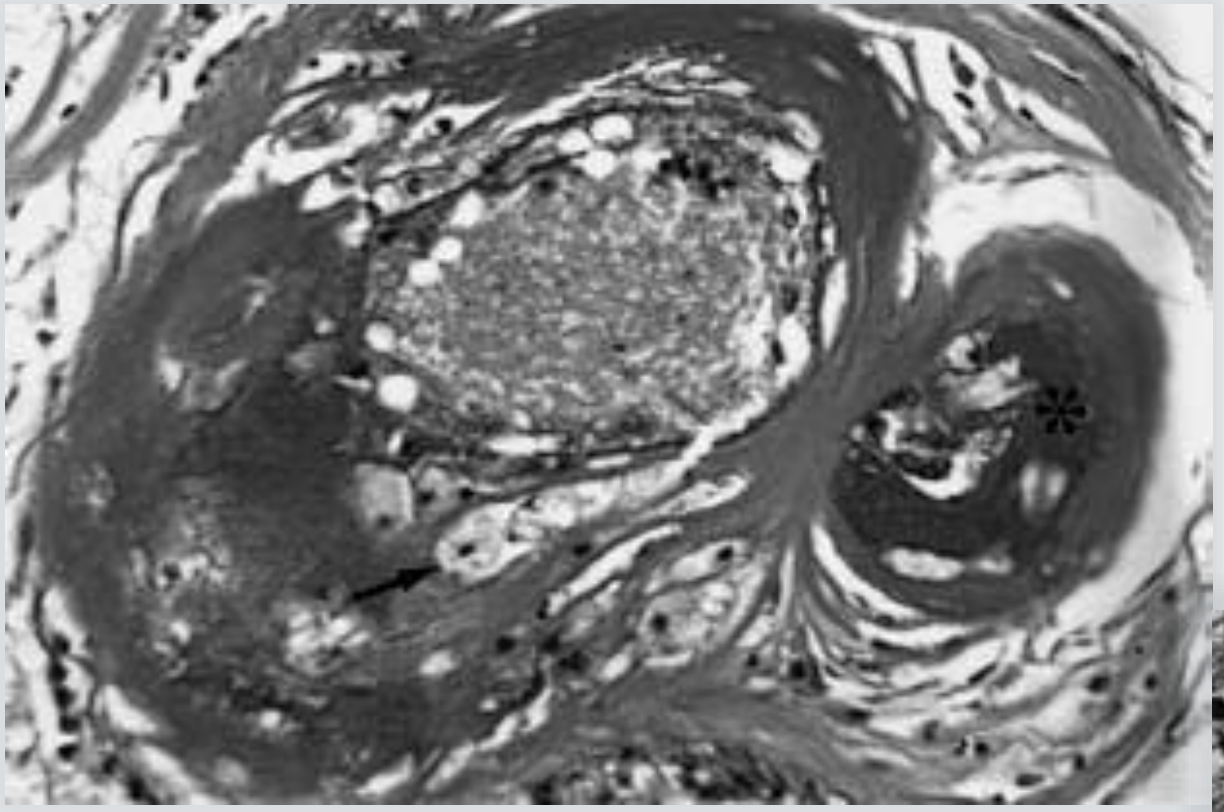


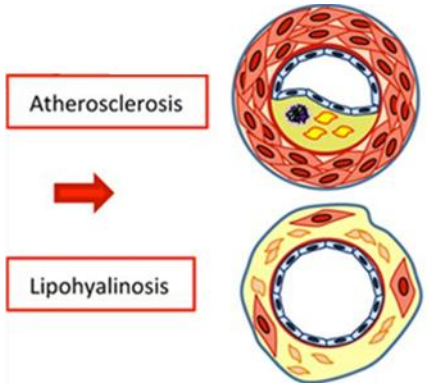
- Βασικά γάγγλια
- Θάλαμος
- Έσω κάψα
- Ακτινωτός στέφανος
- Στέλεχος

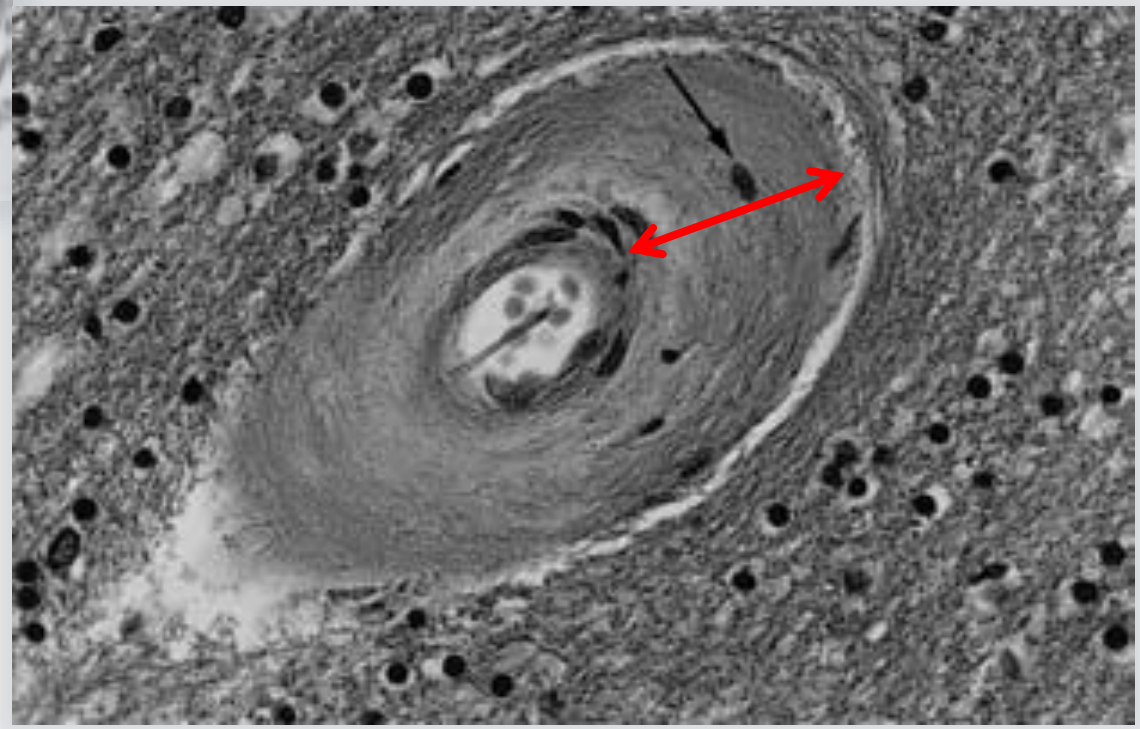


Ann Neurol 2001;50:208–215

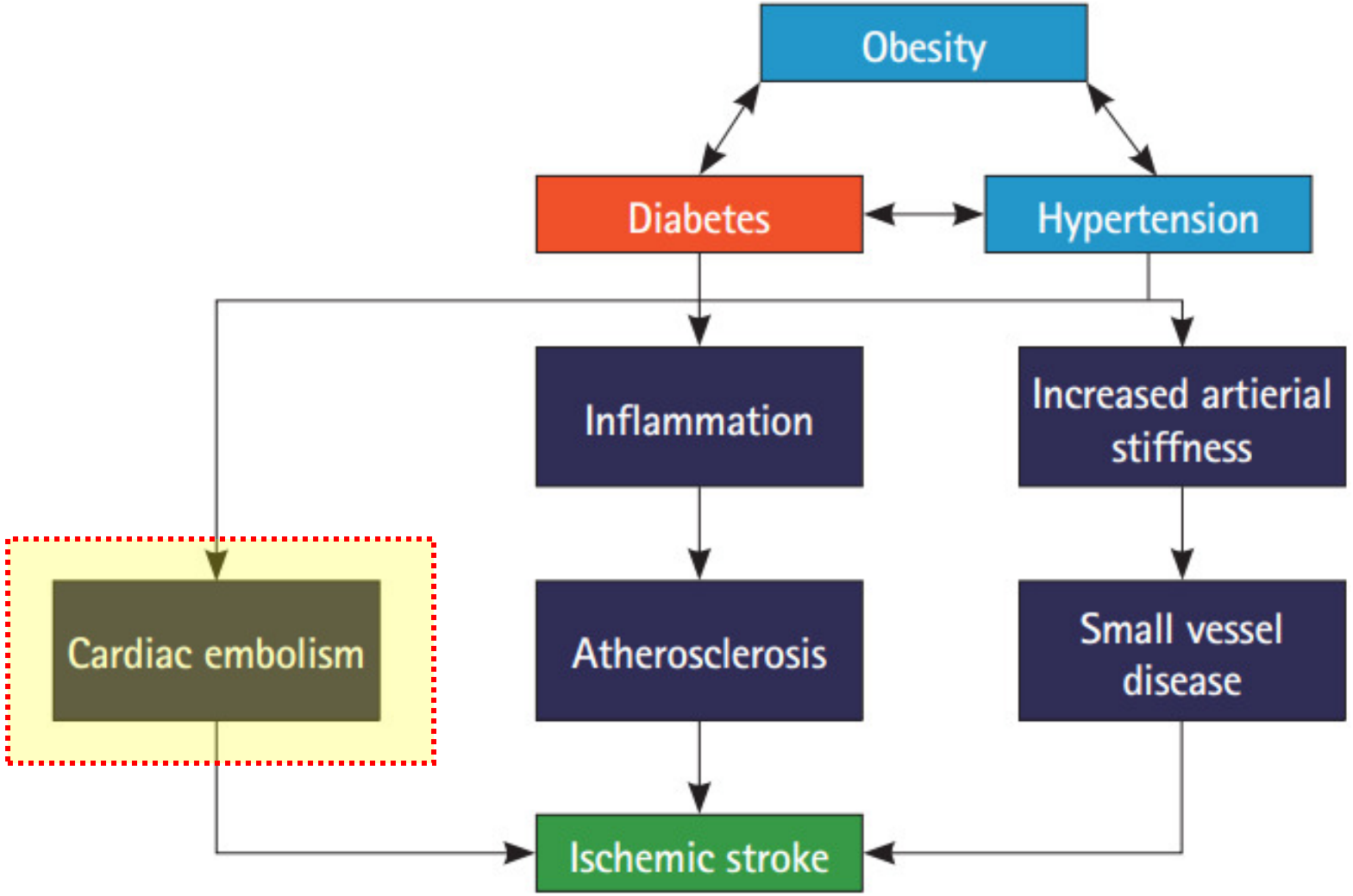
Brainin & Heiss, Textbook of Stroke Medicine, 2010




Atherosclerosis
Lipohyalinosis

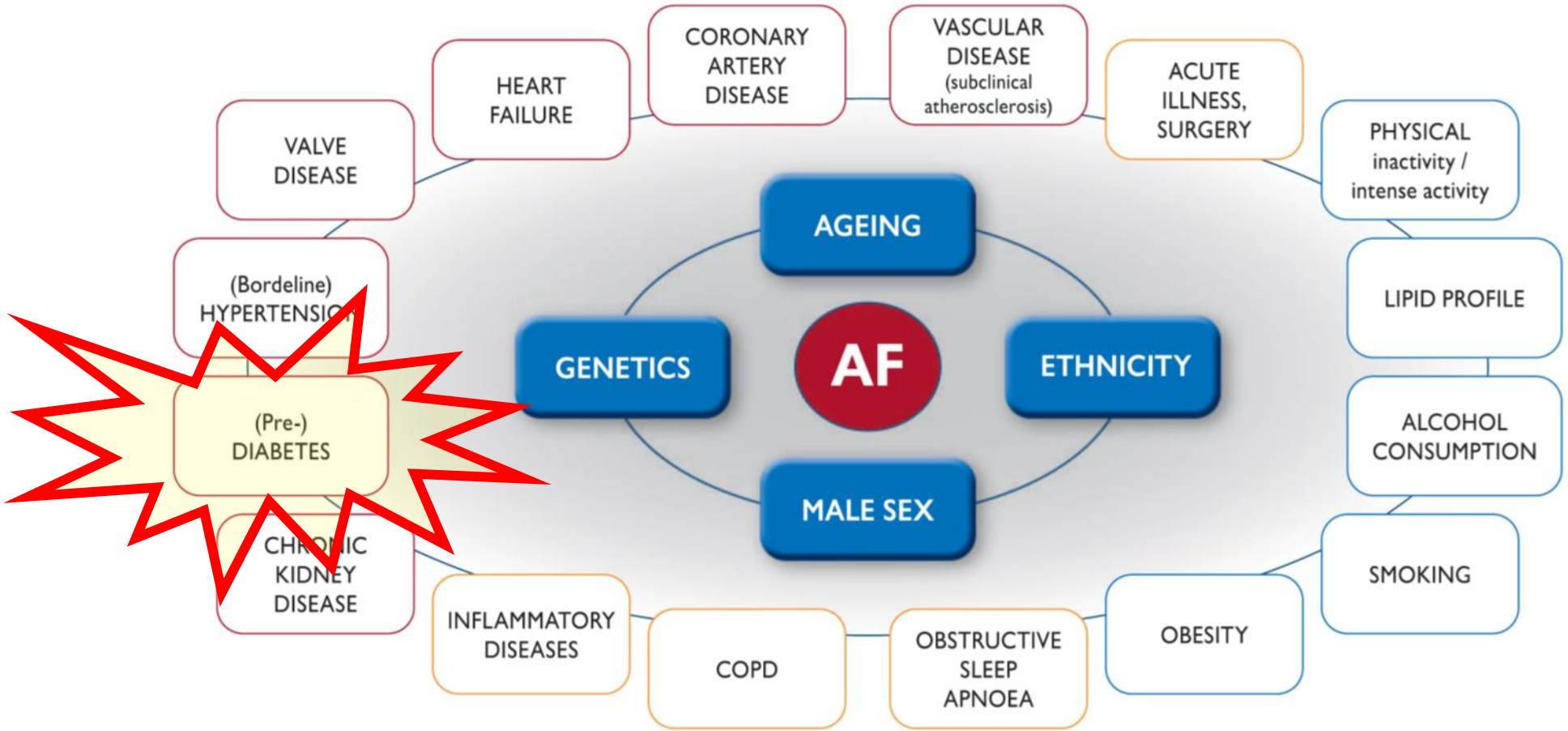


Diabetes and Stroke: What Are the Connections?

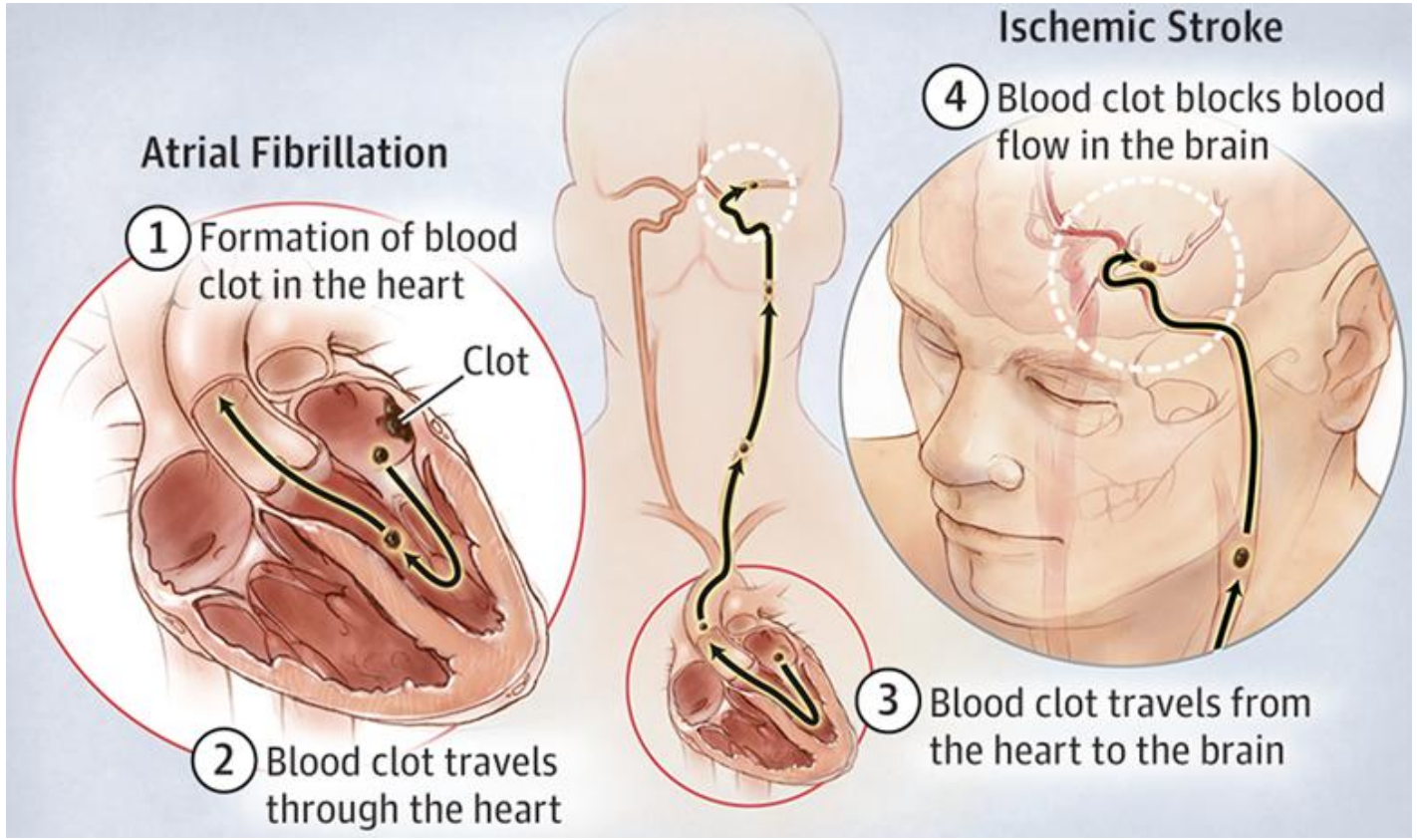


Journal of Stroke 2023;25(1):26-38

Summary of risk factors for incident AF



2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)



CHA_2DS_2-VASc score Risk factors and definitions		Points awarded
C	**Congestive heart failure** Clinical HF, or objective evidence of moderate to severe LV dysfunction, or HCM	1
H	**Hypertension** or on antihypertensive therapy	1
A	**Age 75 years or older**	2
D	**Diabetes mellitus** Treatment with oral hypoglycaemic drugs and/or insulin or fasting blood glucose >125 mg/dL (7 mmol/L)	1
S	**Stroke**Previous stroke, TIA, or thromboembolism	2
V	**Vascular disease** Angiographically significant CAD, previous myocardial infarction, PAD, or aortic plaque	1
A	**Age 65 – 74 years**	1
Sc	**Sex category (female)**	1
Maximum score		**9**

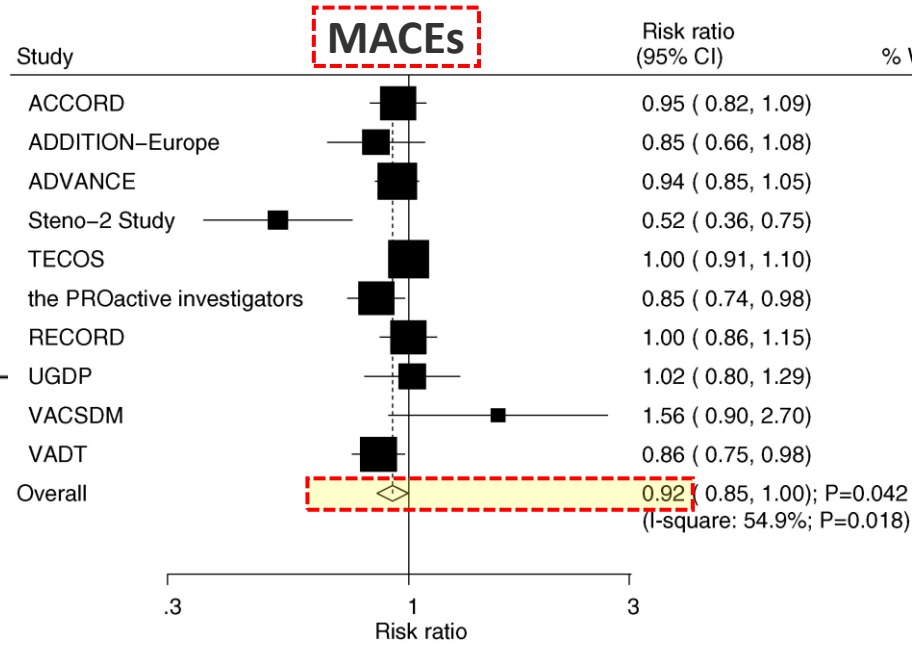
European Heart Journal (2020) **42**, 373–498

2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD

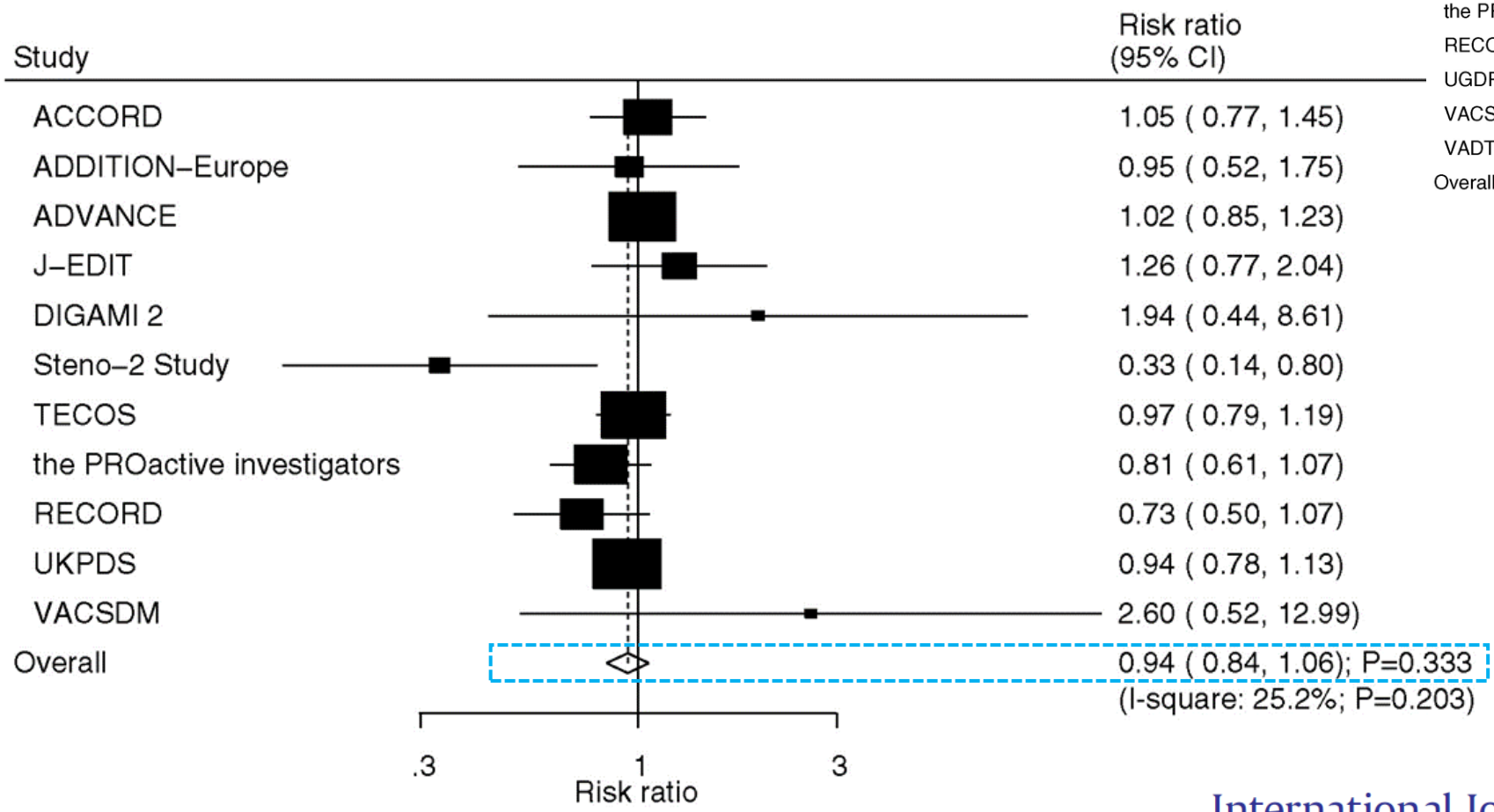
Recommendations	Class[a]	Level[b]
It is recommended to apply tight glucose control, targeting a near-normal HbA1c (<7.0% or <53 mmol/mol), to decrease microvascular complications in individuals with DM.[145–149]	I	A
It is recommended that HbA1c targets are individualized according to the duration of DM, comorbidities, and age.[122,150]	I	C
Avoidance of hypoglycaemia is recommended.[136,139,140,151]	I	C
The use of structured self-monitoring of blood glucose and/or continuous glucose monitoring should be considered to facilitate optimal glycaemic control.[141–144]	IIa	A
An HbA1c target of <7.0% (or <53 mmol/mol) should be considered for the prevention of macrovascular complications in individuals with DM.	IIa	C

European Heart Journal (2020) **41**, 255–323

Effects of intensive glucose lowering in treatment of type 2 diabetes mellitus on cardiovascular outcomes: A meta-analysis of data from 58,160 patients in 13 randomized controlled trials



B. stroke



Conclusion

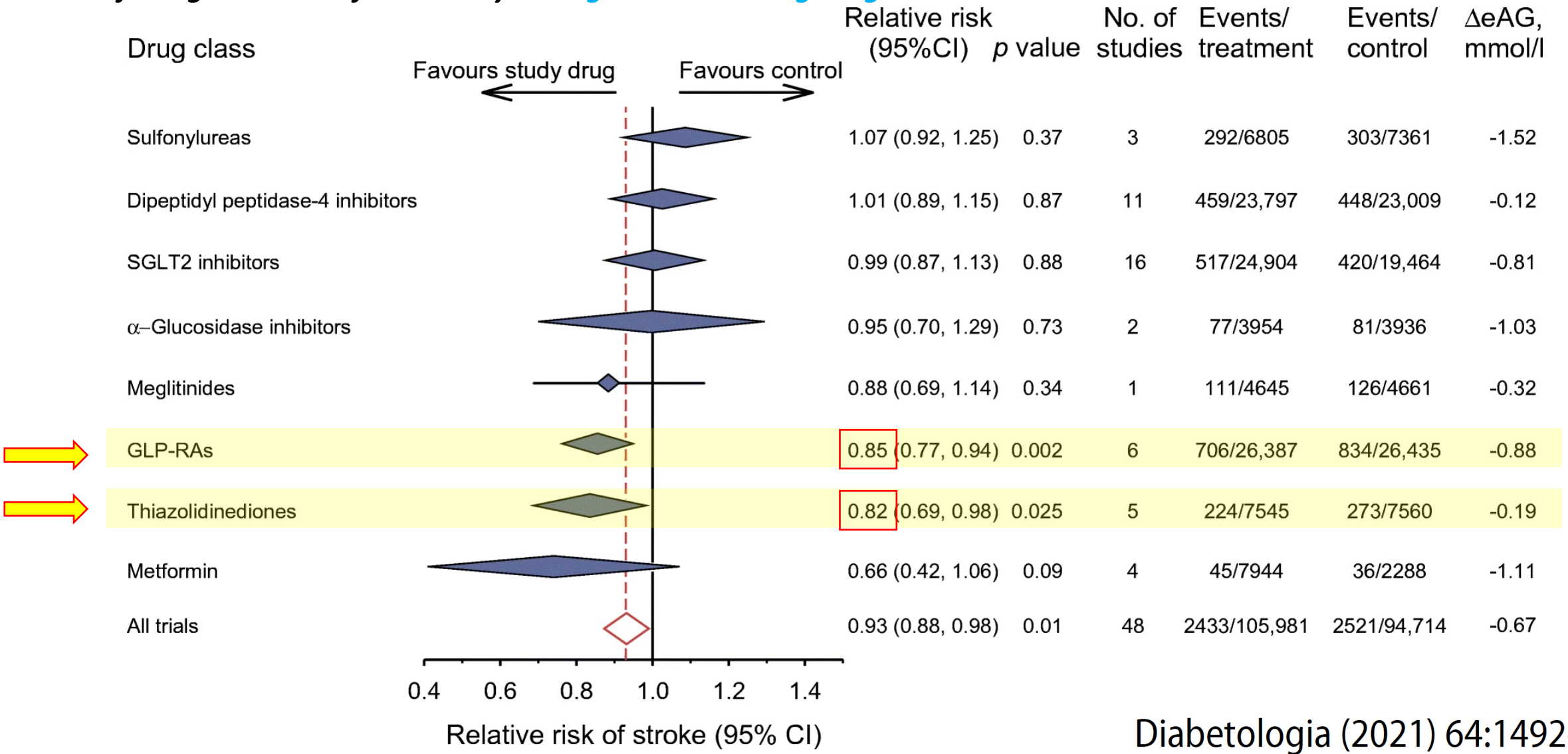
T2DM patients who received intensive glucose lowering therapy are associated with a reduced risk of MACEs and MI, whereas it has **no significant effect** on the risk of total mortality, cardiac death, stroke, and congestive heart failure.

These effects might differ when stratified by baseline characteristics in T2DM patients.

International Journal of Cardiology 218 (2016) 50–58

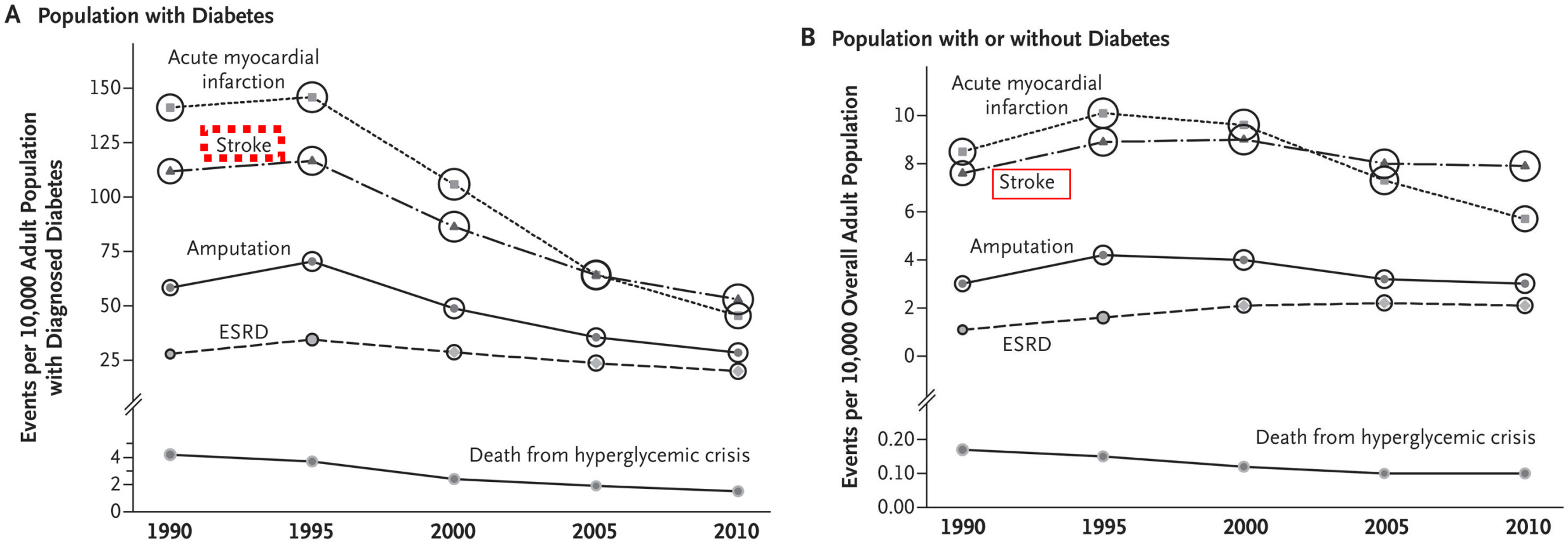
Impact of high glucose levels and glucose lowering on risk of ischaemic stroke: a Mendelian randomisation study and meta-analysis

Meta-analyses of risk of stroke (fatal and non-fatal) for randomised clinical intervention trials of more than 12 months' duration for eight classes of commonly used glucose-lowering drugs



Diabetologia (2021) 64:1492–1503

Changes in Diabetes-Related Complications in the United States, 1990–2010

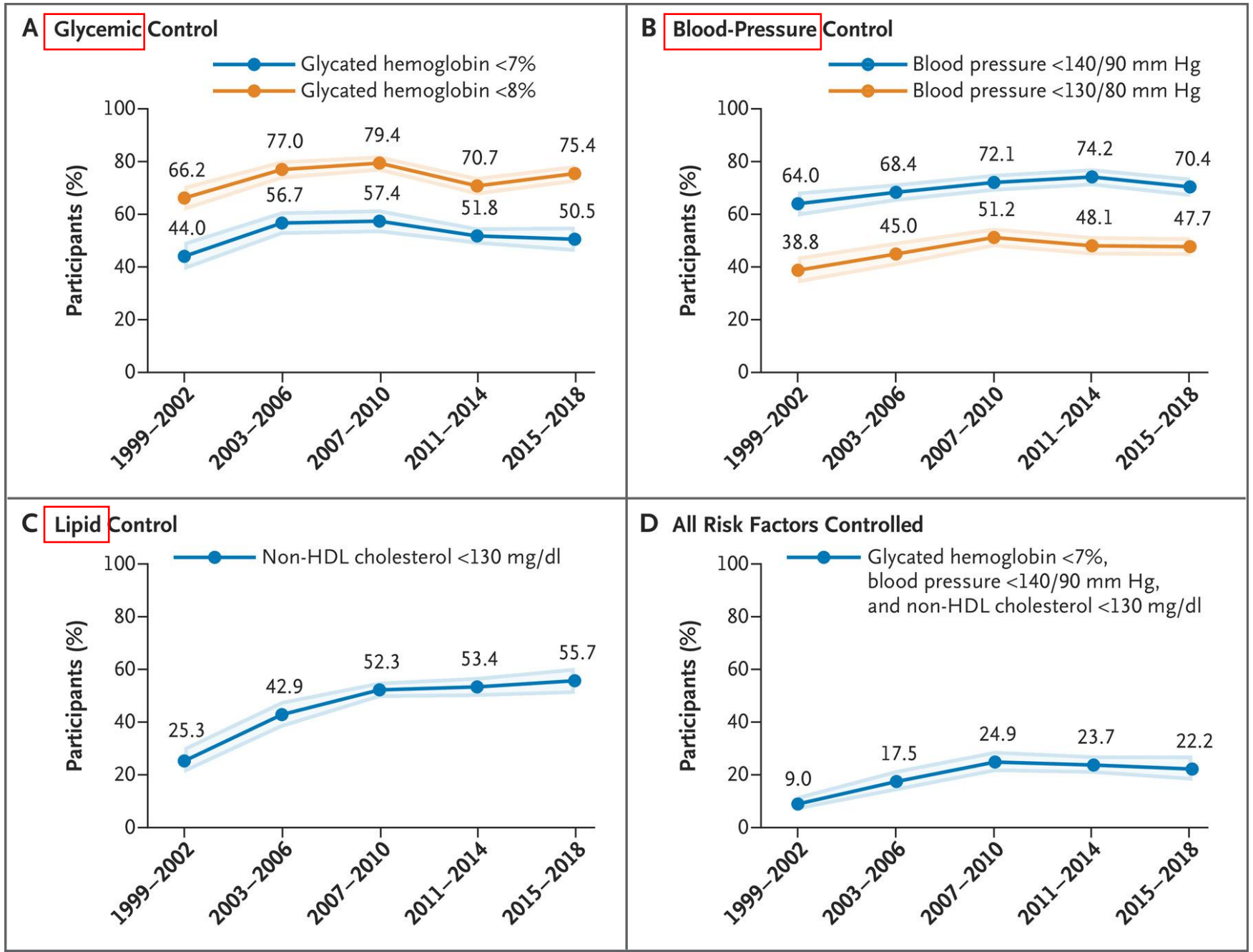


Rates of diabetes-related complications have declined substantially in the past two decades, but a large burden of disease persists because of the continued increase in the prevalence of diabetes

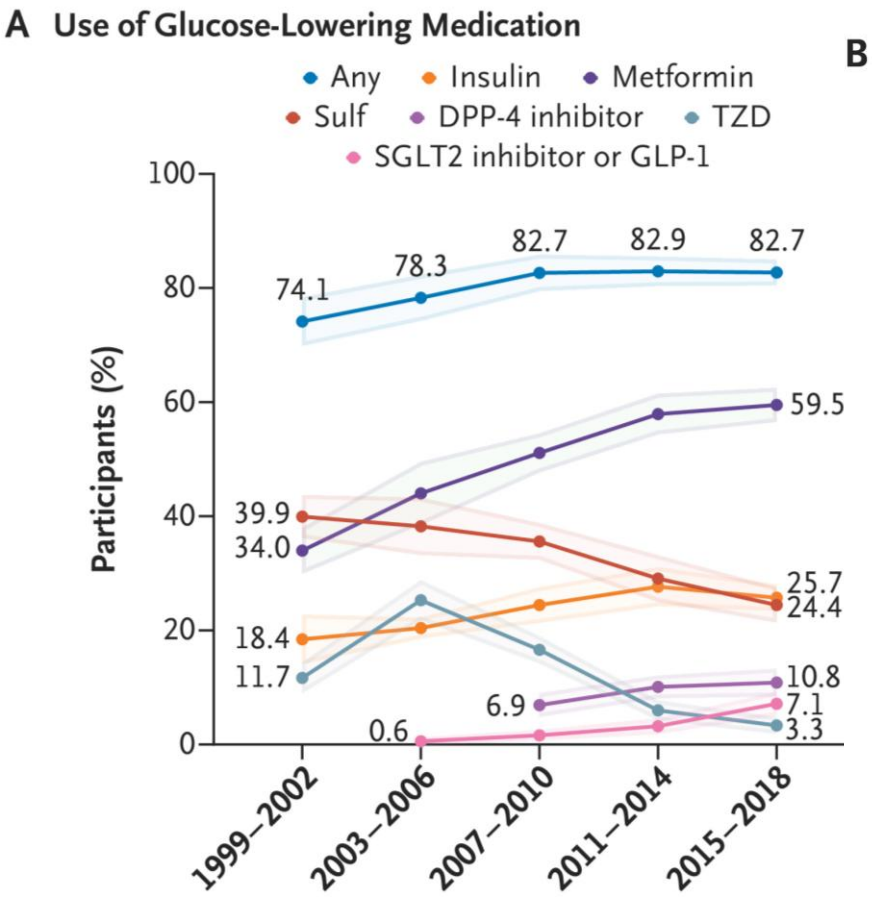
N Engl J Med 2014;370:1514-23

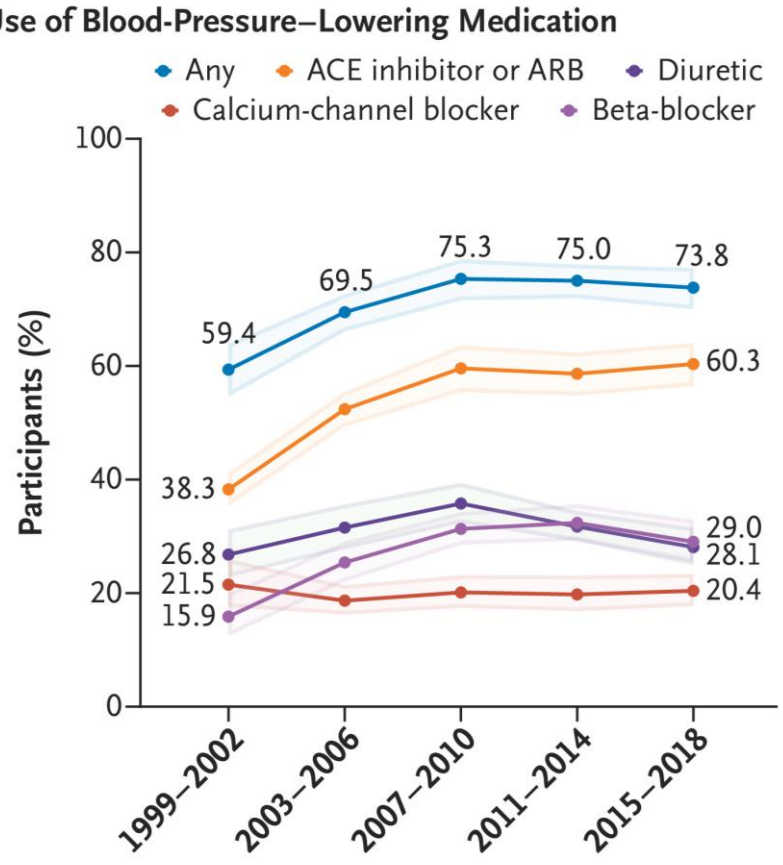
Trends in Diabetes Treatment and Control in U.S. Adults, 1999–2018

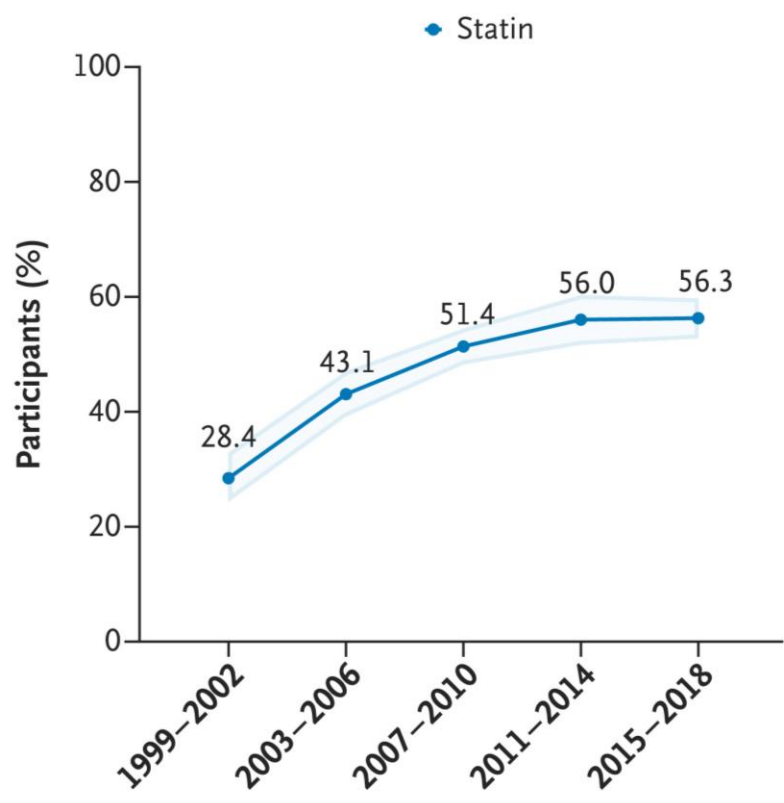
N Engl J Med 2021;384:2219-28



Trends in Diabetes Treatment and Control in U.S. Adults, 1999–2018







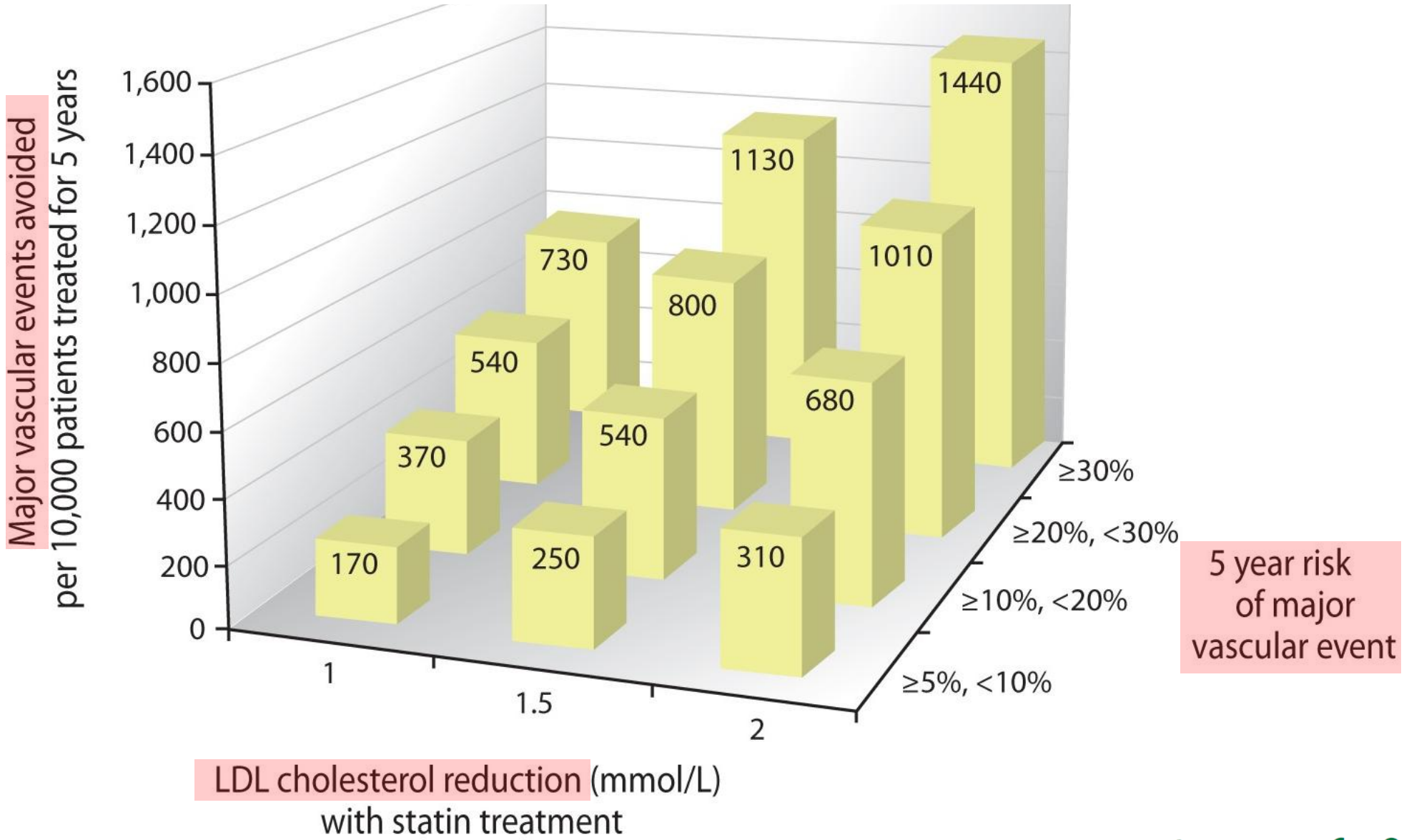
N Engl J Med 2021;384:2219-28

Stroke Risk Factors, Genetics, and Prevention

	Nonmodifiable Risk Factors	Modifiable Risk Factors
Ischemic stroke	Age	Hypertension
	Sex	Current smoking
	Race/ethnicity	Waist-to-hip ratio
		Diet
		Physical inactivity
		Hyperlipidemia
		Diabetes mellitus
		Alcohol consumption
		Cardiac causes
		Apolipoprotein B to A1
	Genetics*	

Circ Res. 2017;120:472-495

Interpretation of the evidence for the efficacy and safety of statin therapy



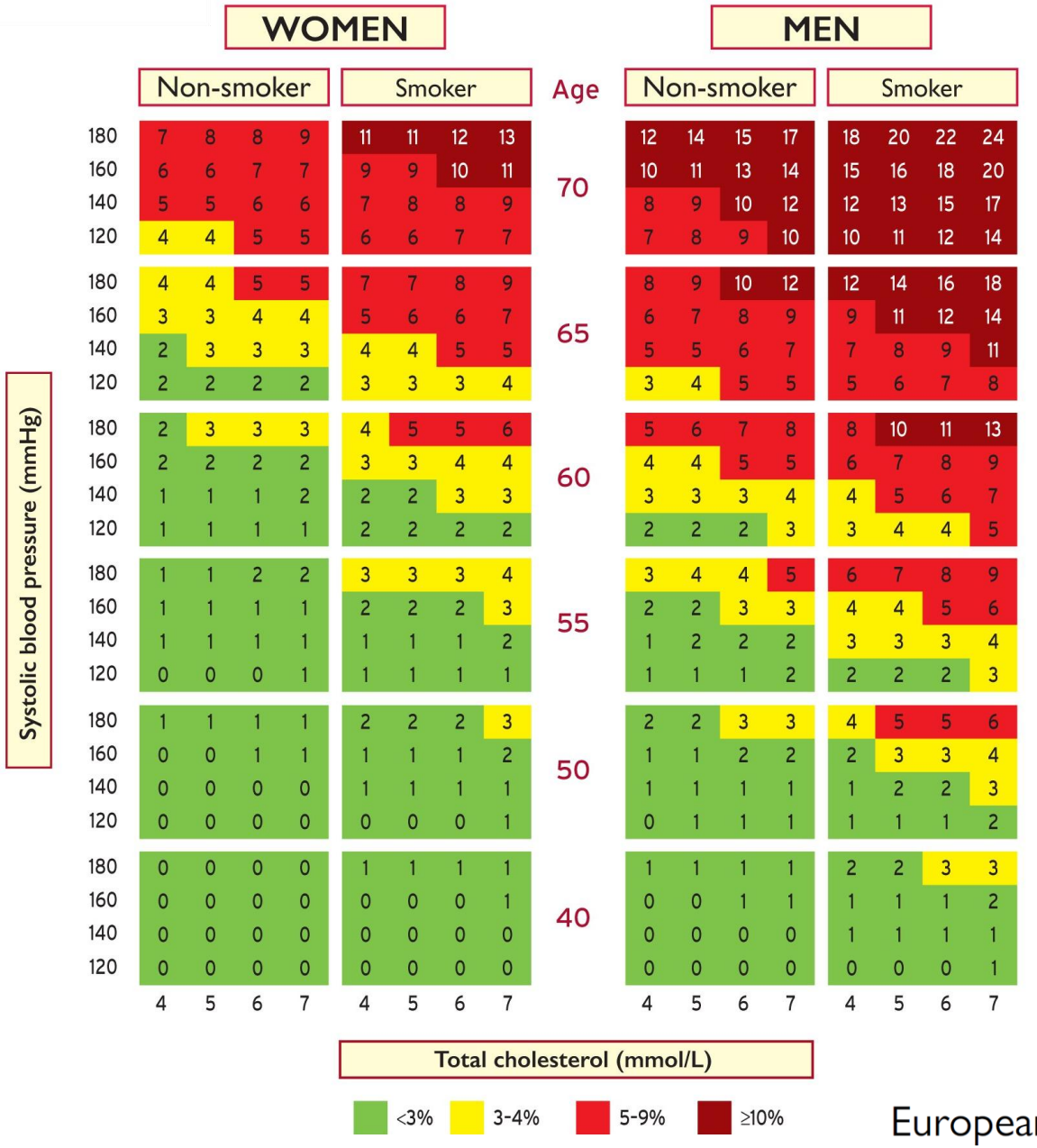
Lancet 2016; 388: 2532–61

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

SCORE Cardiovascular Risk Chart

10-year risk of fatal CVD

Low-risk regions of Europe

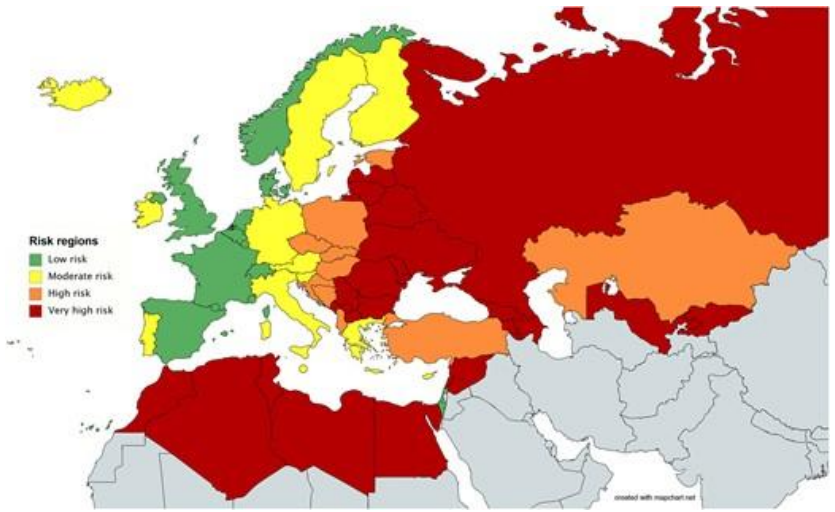


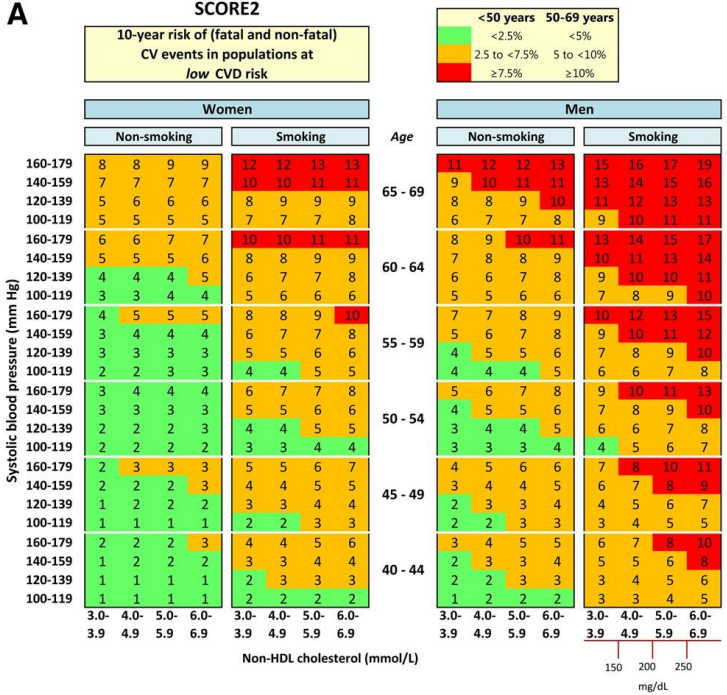
Systolic blood pressure (mmHg)	WOMEN Non-smoker				WOMEN Smoker				Age	MEN Non-smoker				MEN Smoker			
	4	5	6	7	4	5	6	7		4	5	6	7	4	5	6	7
180	7	8	8	9	11	11	12	13	70	12	14	15	17	18	20	22	24
160	6	6	7	7	9	9	10	11		10	11	13	14	15	16	18	20
140	5	5	6	6	7	8	8	9		8	9	10	12	12	13	15	17
120	4	4	5	5	6	6	7	7		7	8	9	10	10	11	12	14
180	4	4	5	5	7	7	8	9	65	8	9	10	12	12	14	16	18
160	3	3	4	4	5	6	6	7		6	7	8	9	9	11	12	14
140	2	3	3	3	4	4	5	5		5	5	6	7	7	8	9	11
120	2	2	2	2	3	3	3	4		3	4	5	5	5	6	7	8
180	2	3	3	3	4	5	5	6	60	5	6	7	8	8	10	11	13
160	2	2	2	2	3	3	4	4		4	4	5	5	6	7	8	9
140	1	1	1	2	2	2	3	3		3	3	3	4	4	5	6	7
120	1	1	1	1	2	2	2	2		2	2	2	3	3	4	4	5
180	1	1	2	2	3	3	3	4	55	3	4	4	5	6	7	8	9
160	1	1	1	1	2	2	2	3		2	2	3	3	4	4	5	6
140	1	1	1	1	1	1	1	2		1	2	2	2	3	3	3	4
120	0	0	0	1	1	1	1	1		1	1	1	2	2	2	2	3
180	1	1	1	1	2	2	2	3	50	2	2	3	3	4	5	5	6
160	0	0	1	1	1	1	1	2		1	1	2	2	2	3	3	4
140	0	0	0	0	1	1	1	1		1	1	1	1	1	2	2	3
120	0	0	0	0	0	0	0	1		0	1	1	1	1	1	1	2
180	0	0	0	0	1	1	1	1	40	1	1	1	1	2	2	3	3
160	0	0	0	0	0	0	0	1		0	0	1	1	1	1	1	2
140	0	0	0	0	0	0	0	0		0	0	0	0	1	1	1	1
120	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	1

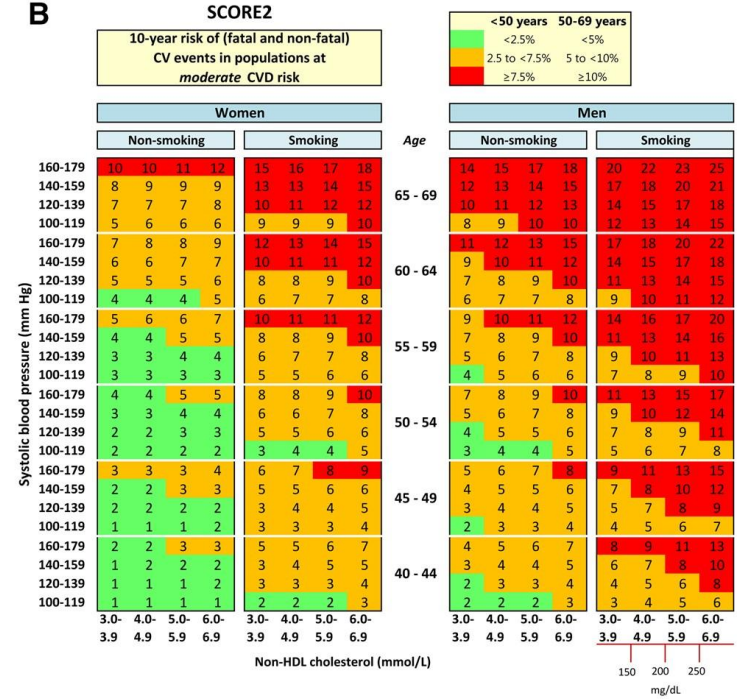
European Society of Cardiology

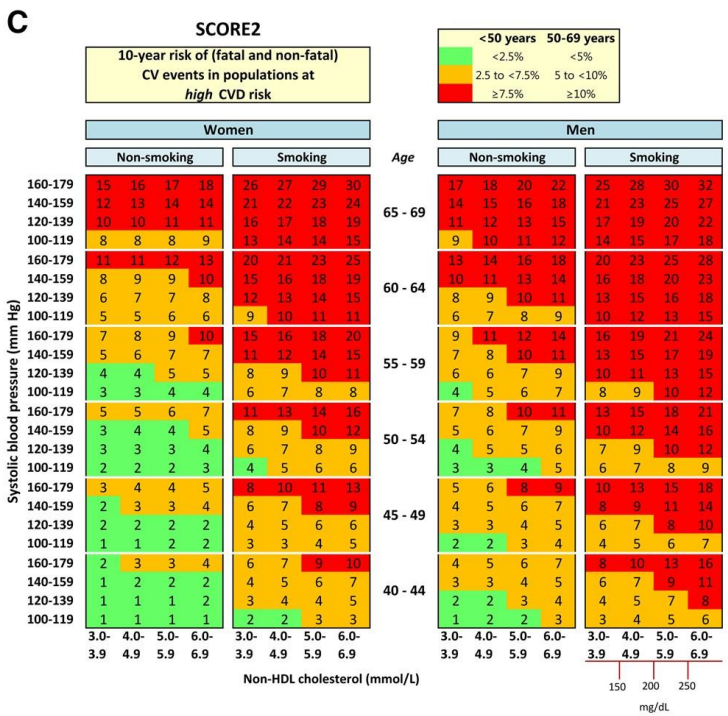
European Heart Journal (2020) **41**, 111–188

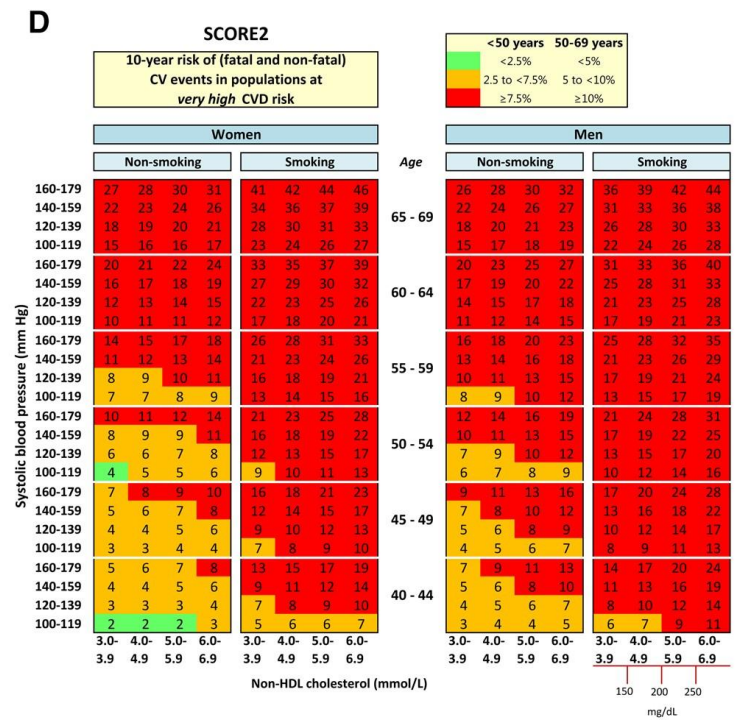
SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe











European Heart Journal (2021) **42**, 2439–2454

Systematic Coronary Risk Evaluation 2 (SCORE2)

Predicts 10-year CVD risk in patients without prior CVD or diabetes.

☆ Favorite Share

INSTRUCTIONS

Use in European patients aged 40-69 years without prior CVD or diabetes.

When to Use ⌄ | Why Use ⌄

Sex	**Male**	Female
Age	50	years
Smoking	Other	**Current**
SBP	150	mm Hg
Total cholesterol	160	mg/dL ⇆
HDL cholesterol	100	mg/dL ⇆

Very high double-check.

Risk region
See Evidence for definition of risk regions.

- Low
- **Moderate**
- High
- Very high

3.4 %

10-year risk of CVD

Copy Results | Next Steps »»

About the Creator

SCORE2 Working Group

Are you SCORE2 Working Group?

ESC Cardiovascular Risk Collaboration

Are you ESC Cardiovascular Risk Collaboration?

Also from MDCalc...

Related Calcs

- SCORE2-Diabetes
  Predicts 10-year CVD risk in patients with type 2 diabetes.
- SCORE2-OP
  Predicts 10-year CVD risk in older patients.
- PREVENT
  Predicts 10- and 30-year risk of CVD and CVD subtypes in patients aged 30-79 without known CVD.

Systematic Coronary Risk Evaluation 2-Older Persons (SCORE2-OP)

Predicts 10-year CVD risk in older patients.

INSTRUCTIONS

Use in patients ≥70 years.

When to Use ⌄ | Pearls/Pitfalls ⌄ | Why Use ⌄

Sex	**Male**	Female
Age	75	years
Diabetes	No	**Yes**
Smoking	**Other**	Current
SBP	150	mm Hg
Total cholesterol	150	mg/dL ⇆
HDL cholesterol	100	mg/dL ⇆

Very high double-check.

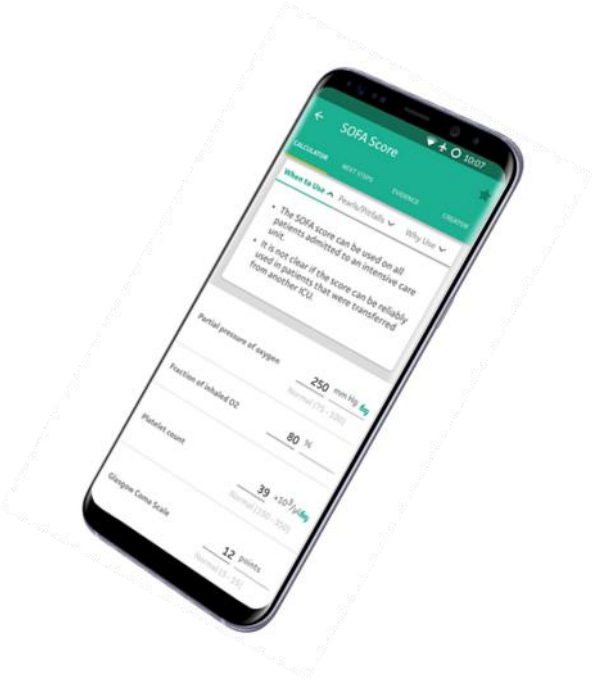
Risk region
See Evidence for definition of risk regions.

- Low
- **Moderate**
- High
- Very high

14.8 %

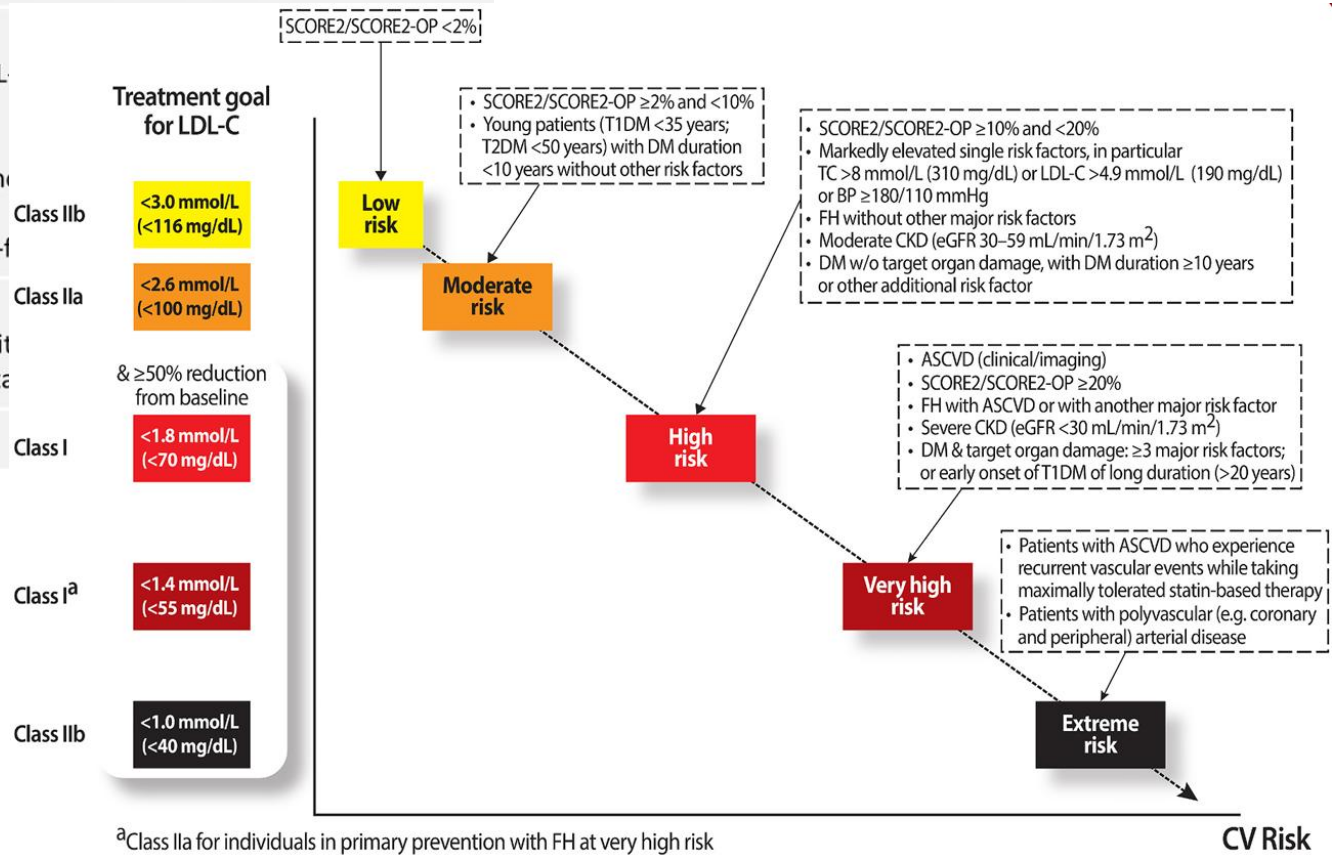
10-year risk of CVD

Copy Results | Next Steps »»



2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias

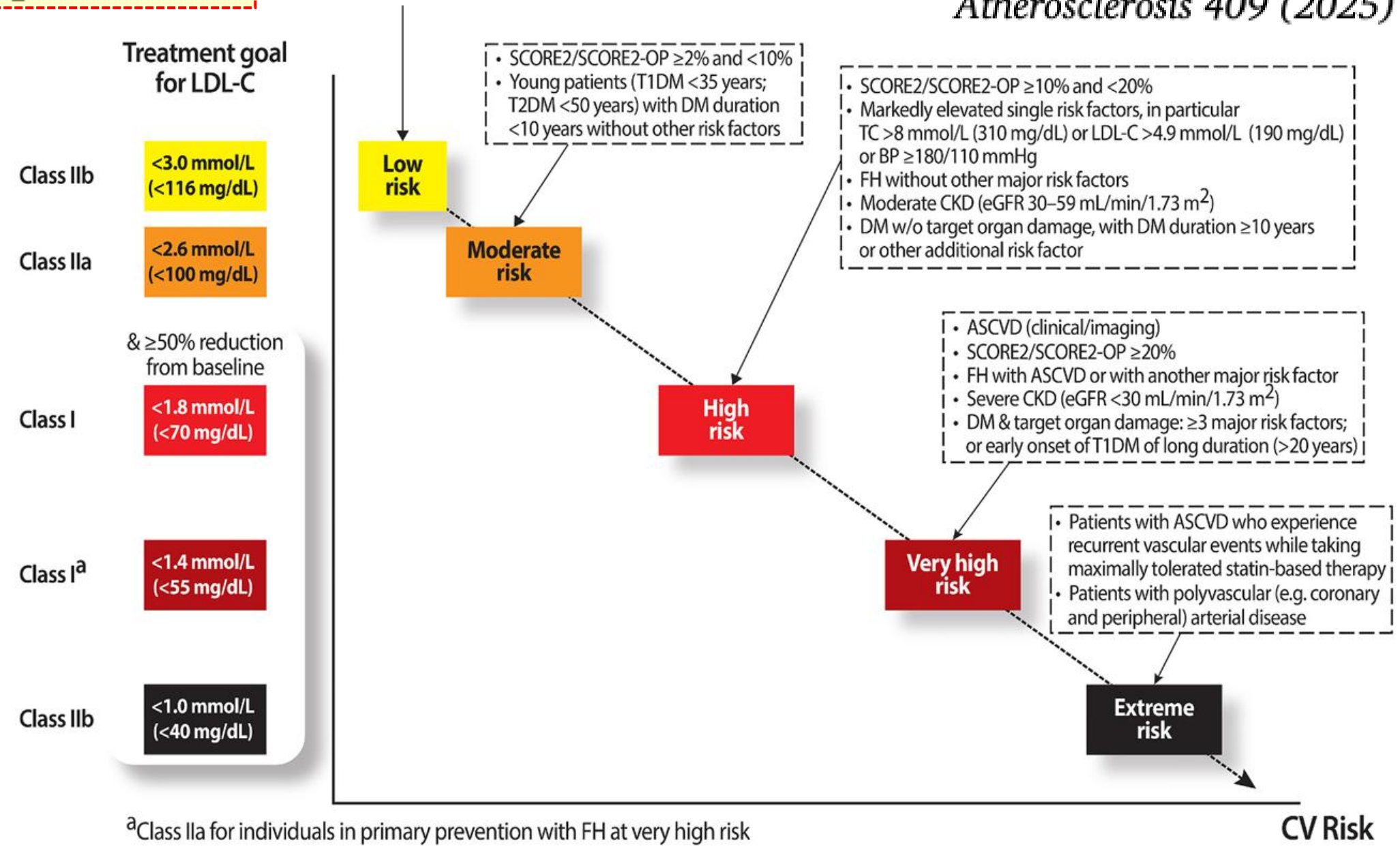
Very high risk	People with any of the following: • Documented ASCVD, either clinical or unequivocal on imaging. Documented ASCVD includes previous ACS (MI or unstable angina), chronic coronary syndromes, coronary revascularization (PCI, CABG, and other arterial revascularization procedures), stroke and TIA, and peripheral arterial disease. Unequivocally documented ASCVD on imaging includes those findings that are known to be predictive of clinical events, such as significant plaque[a] on coronary angiography or CT scan or on carotid or femoral ultrasound or markedly elevated CAC score by CT[b] • DM with target organ damage,[c] or at least three major risk factors, or early onset of T1DM of long duration (>20 years) • Severe CKD (eGFR <30 mL/min/1.73 m²) • A calculated SCORE2 or SCORE2-OP ≥20% for 10 year risk of fatal or non-fatal CVD • FH with ASCVD or with another major risk factor
High risk	People with any of the following: • Markedly elevated single risk factors, in particular TC >8 mmol/L (>310 mg/dL), LDL BP ≥180/110 mmHg • Patients with FH without other major risk factors • Patients with DM without target organ damage,[c] with DM duration ≥10 years or an • Moderate CKD (eGFR 30–59 mL/min/1.73 m²) • A calculated SCORE2 or SCORE2-OP ≥10% and <20% for 10 year risk of fatal or non-f
Moderate risk	People with any of the following: • Young patients (T1DM <35 years; T2DM <50 years) with DM duration <10 years, wit • Calculated SCORE2 or SCORE2-OP ≥2% and <10% for 10 year risk of fatal or non-fata
Low risk	• Calculated SCORE2 or SCORE2-OP <2% for 10 year risk of fatal or non-fatal CVD



Atherosclerosis 409 (2025) 120479

2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias

Atherosclerosis 409 (2025) 120479



2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias

Atherosclerosis 409 (2025) 120479

Total CV risk	Untreated LDL-C levels					
	<1.4 mmol/L (<55 mg/dL)	1.4 to <1.8 mmol/L (55 to <70 mg/dL)	1.8 to <2.6 mmol/L (70 to <100 mg/dL)	2.6 to <3.0 mmol/L (100 to <116 mg/ dL)	3.0 to <4.9 mmol/L (116 to <190 mg/ dL	≥4.9 mmol/L (≥190 mg/dL)[a]
Low	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle modification, consider adding drug if uncontrolled	N/A[a]
Moderate	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle modification, consider adding drug if uncontrolled	Lifestyle modification, consider adding drug if uncontrolled	N/A[a]
High	Lifestyle advice	Lifestyle advice	Lifestyle modification, consider adding drug if uncontrolled	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention
Very high primary prevention	Lifestyle modification, consider adding drug	Lifestyle modification, consider adding drug	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention
Very high secondary prevention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention	Lifestyle modification and concomittant drug intervention

2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias

Atherosclerosis 409 (2025) 120479

Box 1
Risk modifiers for consideration beyond the risk estimation based on the SCORE2 and SCORE2-OP algorithms

Demographic/clinical conditions.

- Family history of premature CVD (men: <55 years; women: <60 years)
- High-risk ethnicity (e.g. Southern Asian)
- Stress symptoms and psychosocial stressors
- Social deprivation
- Obesity
- Physical inactivity
- Chronic immune-mediated/inflammatory disorders
- Major psychiatric disorders
- History of premature menopause
- Pre-eclampsia or other hypertensive disorders of pregnancy
- Human immunodeficiency virus infection
- Obstructive sleep apnoea syndrome

Biomarkers.

- Persistently elevated hs-CRP (>2 mg/L)
- Elevated Lp(a) [>50 mg/dL (>105 nmol/L)].

CVD, cardiovascular disease; hs-CRP, high sensitivity C-reactive protein; Lp(a), lipoprotein(a).

Recommendations	Class[a]	Level[b]
Lp(a) levels above 50 mg/dL (105 nmol/L) should be considered in all adults as a CV risk-enhancing factor, with higher Lp(a) levels associated with a greater increase in risk.[37,101]	IIa	B

https://www.lpaclinicalguidance.com/

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

ESC
European Society of Cardiology

LDL-C	**Very-high risk in primary or secondary prevention:** A therapeutic regimen that achieves ≥50% LDL-C reduction from baseline[b] and an LDL-C goal of <1.4 mmol/L (<55 mg/dL). No current statin use: this is likely to require high-intensity LDL-lowering therapy. Current LDL-lowering treatment: an increased treatment intensity is required. **High risk:** A therapeutic regimen that achieves ≥50% LDL-C reduction from baseline[b] and an LDL-C goal of <1.8 mmol/L (<70 mg/dL). **Moderate risk:** A goal of <2.6 mmol/L (<100 mg/dL). **Low risk:** A goal of <3.0 mmol/L (<116 mg/dL).
Non-HDL-C	Non-HDL-C secondary goals are <2.2, 2.6, and 3.4 mmol/L (<85, 100, and 130 mg/dL) for very-high-, high-, and moderate-risk people, respectively.
ApoB	ApoB secondary goals are <65, 80, and 100 mg/dL for very-high-, high-, and moderate-risk people, respectively.
Triglycerides	No goal, but <1.7 mmol/L (<150 mg/dL) indicates lower risk and higher levels indicate a need to look for other risk factors.

European Heart Journal (2020) **41**, 111–188

Table 8 **Impact of specific lifestyle changes on lipid levels**

	Magnitude of the effect	Level
Lifestyle interventions to reduce TC and LDL-C levels		
Avoid dietary trans fats	++	A
Reduce dietary saturated fats	++	A
Increase dietary fibre	++	A
Use functional foods enriched with phytosterols	++	A
Use red yeast rice nutraceuticals	++	A
Reduce excessive body weight	++	A
Reduce dietary cholesterol	+	B
Increase habitual physical activity	+	B
Lifestyle interventions to reduce TG-rich lipoprotein levels		
Reduce excessive body weight	+	A
Reduce alcohol intake	+++	A
Increase habitual physical activity	++	A
Reduce total amount of dietary carbohydrates	++	A
Use supplements of n-3 polyunsaturated fats	++	A
Reduce intake of mono- and disaccharides	++	B
Replace saturated fats with mono- or polyunsaturated fats	+	B
Lifestyle interventions to increase HDL-C levels		
Avoid dietary trans fats	++	A
Increase habitual physical activity	+++	A
Reduce excessive body weight	++	A
Reduce dietary carbohydrates and replace them with unsaturated fats	++	A
Modest consumption in those who take alcohol may be continued	++	B
Quit smoking	+	B

EAS ESC European Society of Cardiology

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 - doi: 10.1093/eurheartj/ehz455)

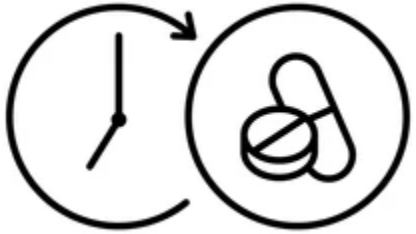
International Journal of Stroke
2021, Vol. 16(6) 738–750

The initial non-pharmacological approach is very important in patients at very high risk of future CV events, such as stroke or TIA patients:

- *increasing the potential of a **better physician-to-patient interaction**,*

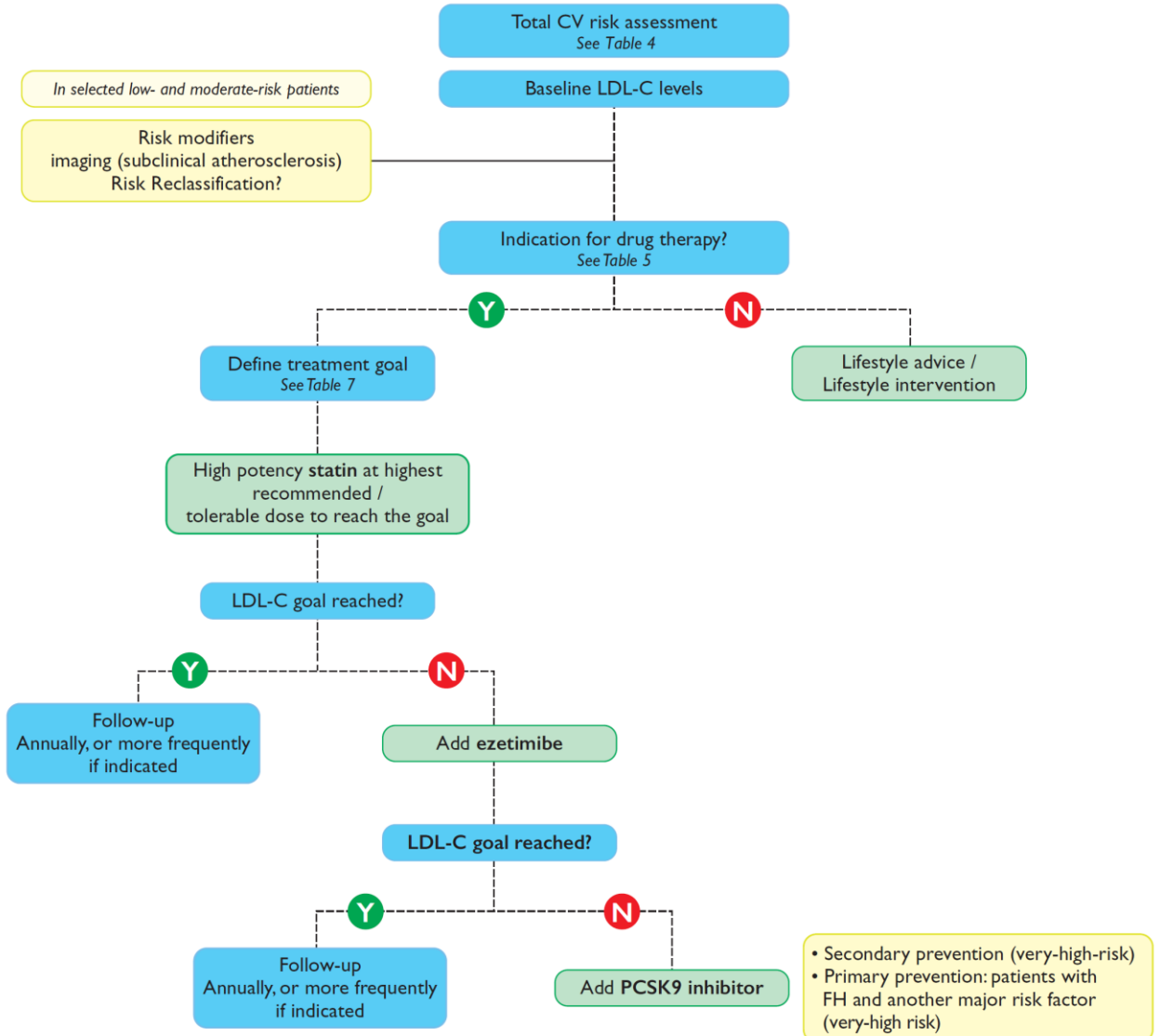
&

- ***adherence to treatment**.*



2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

ESC
European Society of Cardiology



European Heart Journal (2020) **41**, 111–188

Interpretation of the evidence for the efficacy and safety of statin therapy

	Daily dose of different statins				
	5 mg	10 mg	20 mg	40 mg	80 mg
Pravastatin	15%	20%	24%	29%	33%
Simvastatin	23%	27%	32%	37%	42%
Atorvastatin	31%	37%	43%	49%	55%
Rosuvastatin	38%	43%	48%	53%	58%

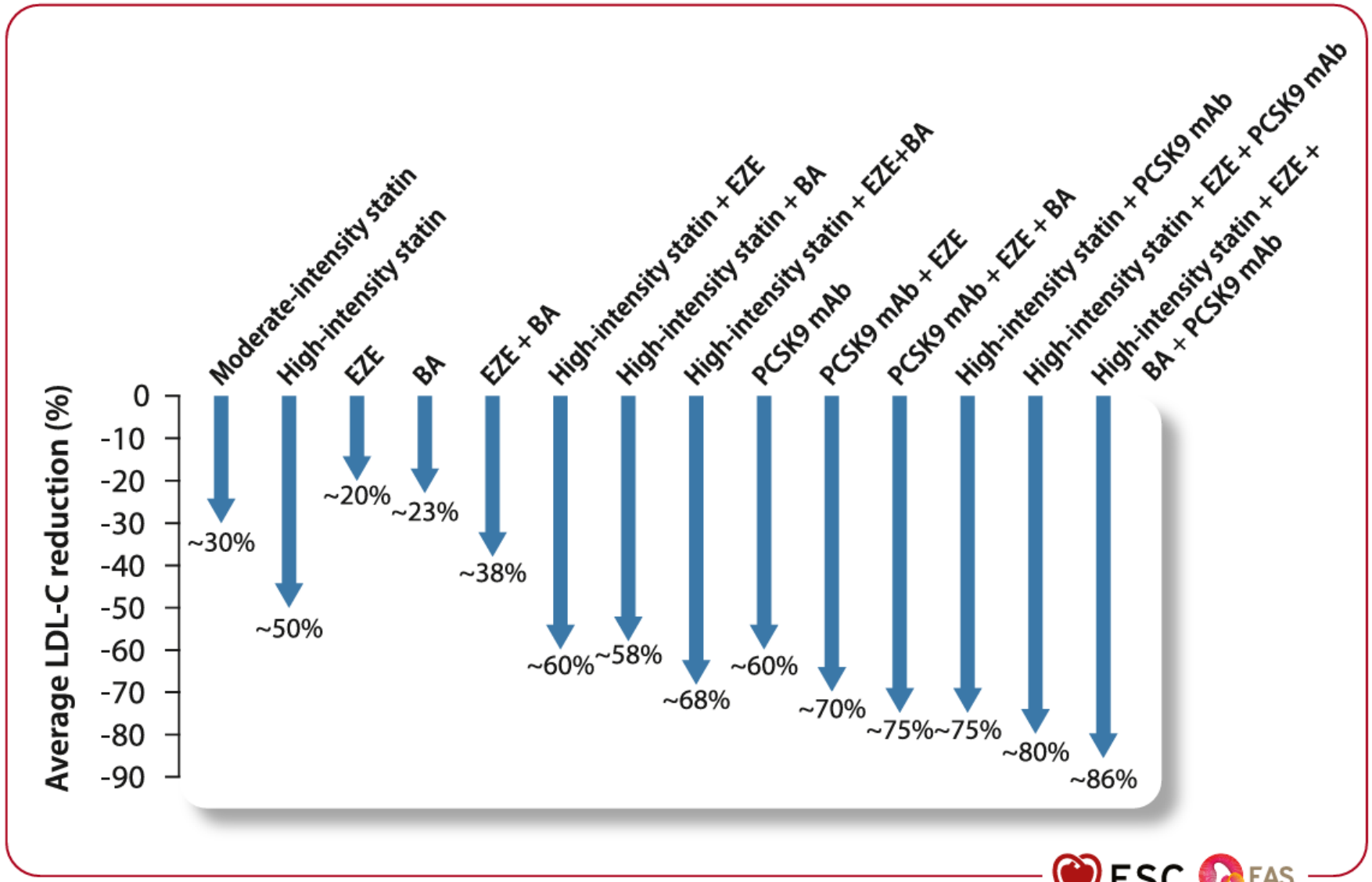
Shaded boxes indicate regimens that can produce about a halving or more in LDL cholesterol concentrations (largely irrespective of patient characteristics, including presenting concentrations of cholesterol). The 2016 cost for generic atorvastatin 40 mg daily in the UK is about £2 per 28 days of treatment;[184] rosuvastatin 20 mg daily currently costs about £25 per month,[185] but it became available as a generic in the USA during 2016.

Table 3: **Average relative reductions in LDL cholesterol concentrations with different doses of commonly used statins**[160,163]

Lancet 2016; 388: 2532–61

2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias

Atherosclerosis 409 (2025) 120479



ESC EAS

Monitor for statin-related adverse effects

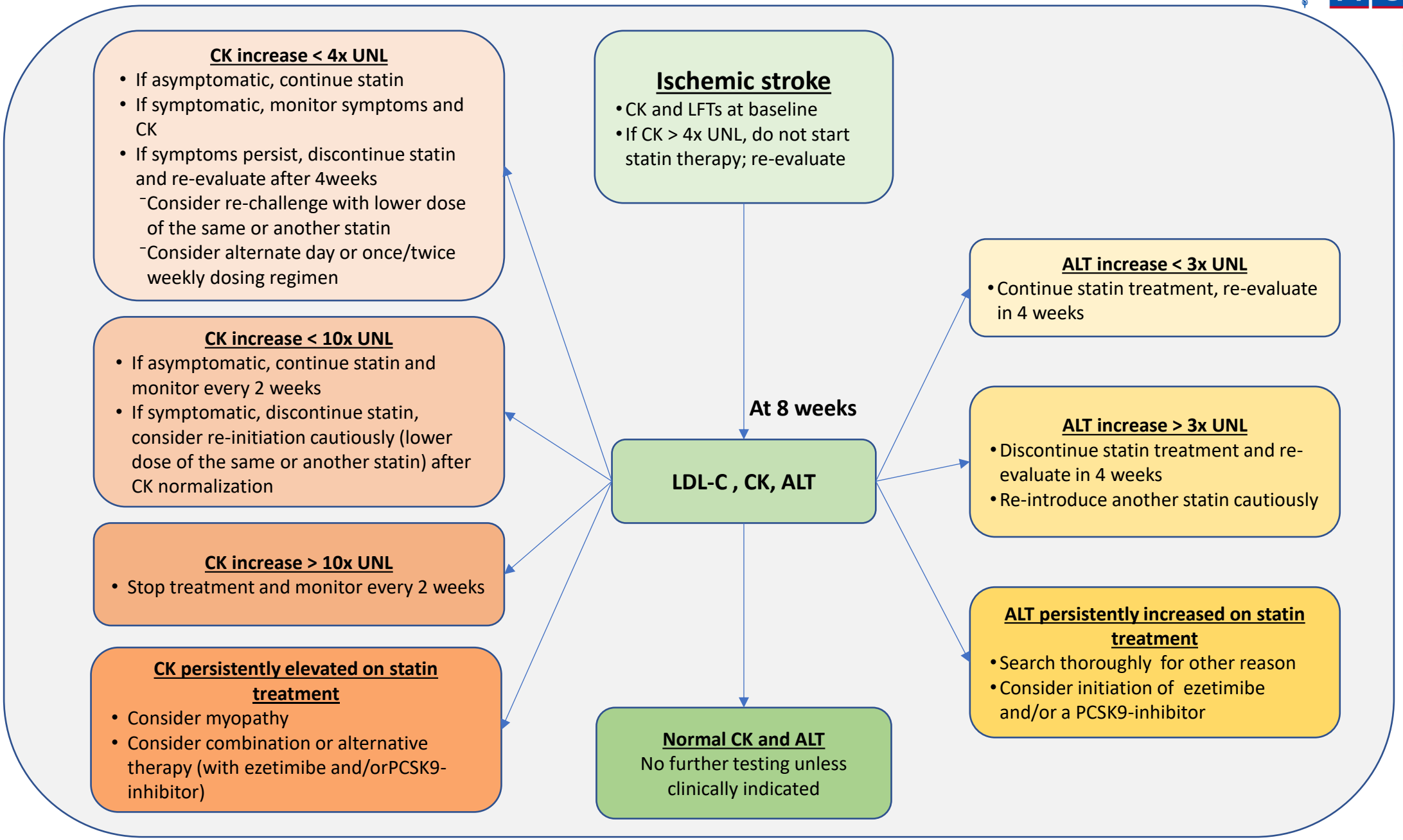
2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

ESC
European Society of Cardiology

Table 10 Drugs potentially interacting with statins metabolized by cytochrome P450 3A4 leading to increased risk of myopathy and rhabdomyolysis

Anti-infective agents	Calcium antagonists	Other
Itraconazole	Verapamil	Ciclosporin
Ketoconazole	Diltiazem	Danazol
Posaconazole	Amlodipine	Amiodarone
Erythromycin		Ranolazine
Clarithromycin		Grapefruit juice
Telithromycin		Nefazodone
HIV protease inhibitors		Gemfibrozil

European Heart Journal (2020) **41**, 111–188



International Journal of Stroke
2021, Vol. 16(6) 738-750

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

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Recommendations for the treatment of dyslipidaemias in older people (aged >65 years)

Recommendations	Class[a]	Level[b]
Treatment with statins is recommended for older people with ASCVD in the same way as for younger patients.[217]	I	A
Treatment with statins is recommended for primary prevention, according to the level of risk, in older people aged ≤75 years.[217]	I	A
Initiation of statin treatment for primary prevention in older people aged >75 years may be considered, if at high-risk or above.[217]	IIb	B
It is recommended that the statin is started at a low dose if there is significant renal impairment and/or the potential for drug interactions, and then titrated upwards to achieve LDL-C treatment goals.	I	C

European Heart Journal (2020) **41**, 111–188

2019 ESC/EAS Guidelines for the management of dyslipidaemias: *lipid modification to reduce cardiovascular risk*

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Testing lipids

How often should lipids be tested?

- Before starting lipid-lowering drug treatment, at least two measurements should be made, with an interval of 1–12 weeks, with the exception of conditions where prompt drug treatment is suggested, such as ACS and very high-risk patients.

How often should a patient's lipids be tested after starting lipid-lowering treatment?

- After starting treatment: 8 (±4) weeks.
- After adjustment of treatment: 8 (±4) weeks until the goal is achieved.

How often should lipids be tested once a patient has achieved the target or optimal lipid level?

- Annually (unless there are adherence problems or other specific reasons for more frequent reviews).

European Heart Journal (2020) **41**, 111–188

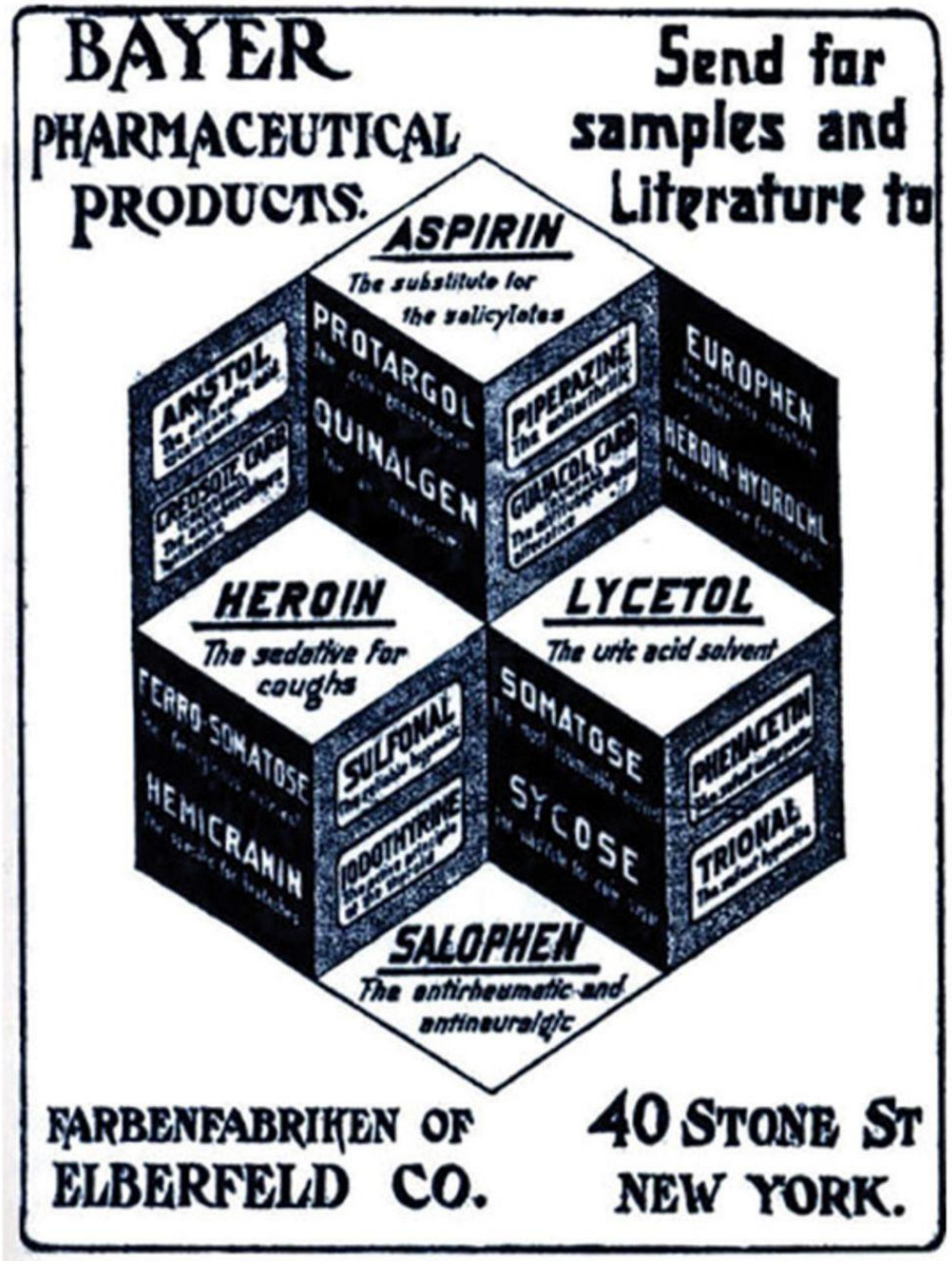
Primary and Secondary Prevention of Ischemic Stroke and Cerebral Hemorrhage

JACC Focus Seminar

ANTITHROMBOTIC THERAPY

JACC VOL. 75, NO. 15, 2020
APRIL 21, 2020:1804–18

Aspirin:
4,000 years and still learning



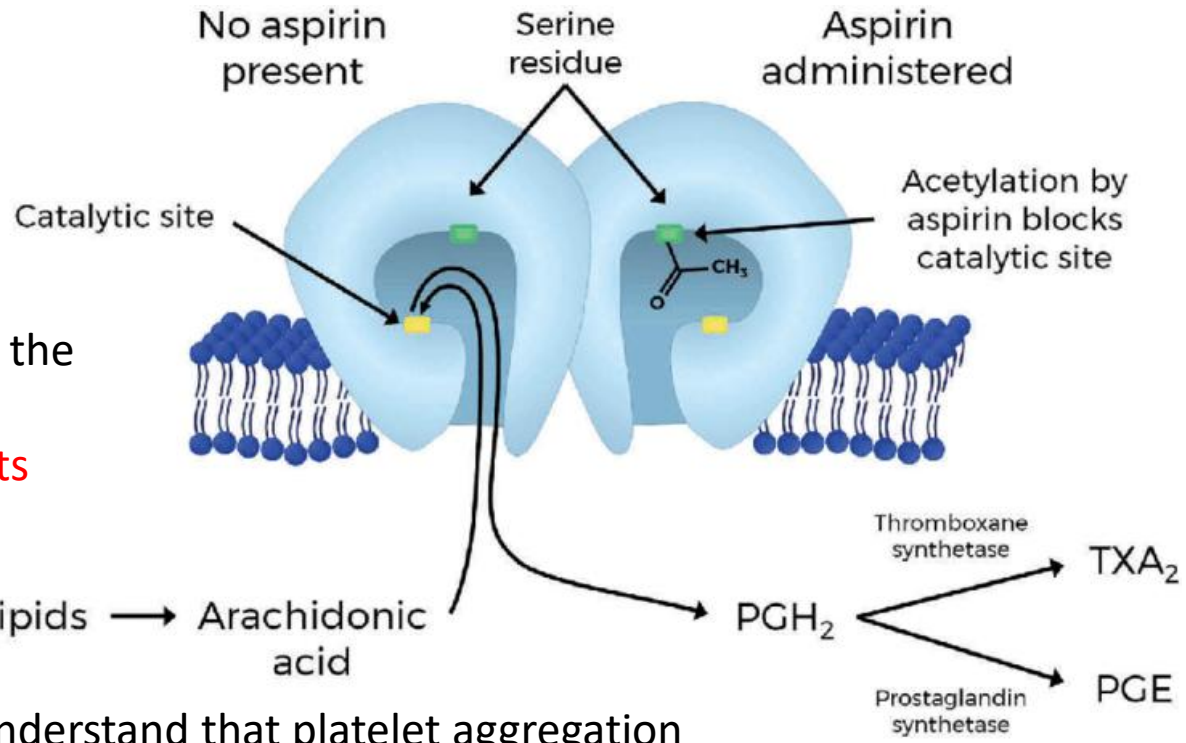
Aspirin: 4,000 years and still learning

Aspirin: The Story of a Wonder Drug

BMJ VOLUME 329 11 DECEMBER 2004

LEARNING HOW ASPIRIN WORKS (AND A FEW OTHER THINGS)

- In the late 1960s, **Weiss** et al reported that aspirin rapidly and irreversibly inhibits platelet aggregation.
- In parallel, using biological assays in work that eventually led to the Nobel Prize, **Vane** discovered that inflammation involves the de novo synthesis of prostaglandins and that aspirin directly inhibits this synthesis.



- Further work connecting these lines of investigation led us to understand that platelet aggregation is enhanced by the prostaglandin derivative thromboxane A2, produced by cy-clooxygenase-1, and that aspirin irreversibly inhibits this enzyme by acetylation.

Expert Rev Cardiovasc Ther. 2019 Aug;17(8):561-573

2021 ESC Guidelines on cardiovascular disease prevention in clinical practice

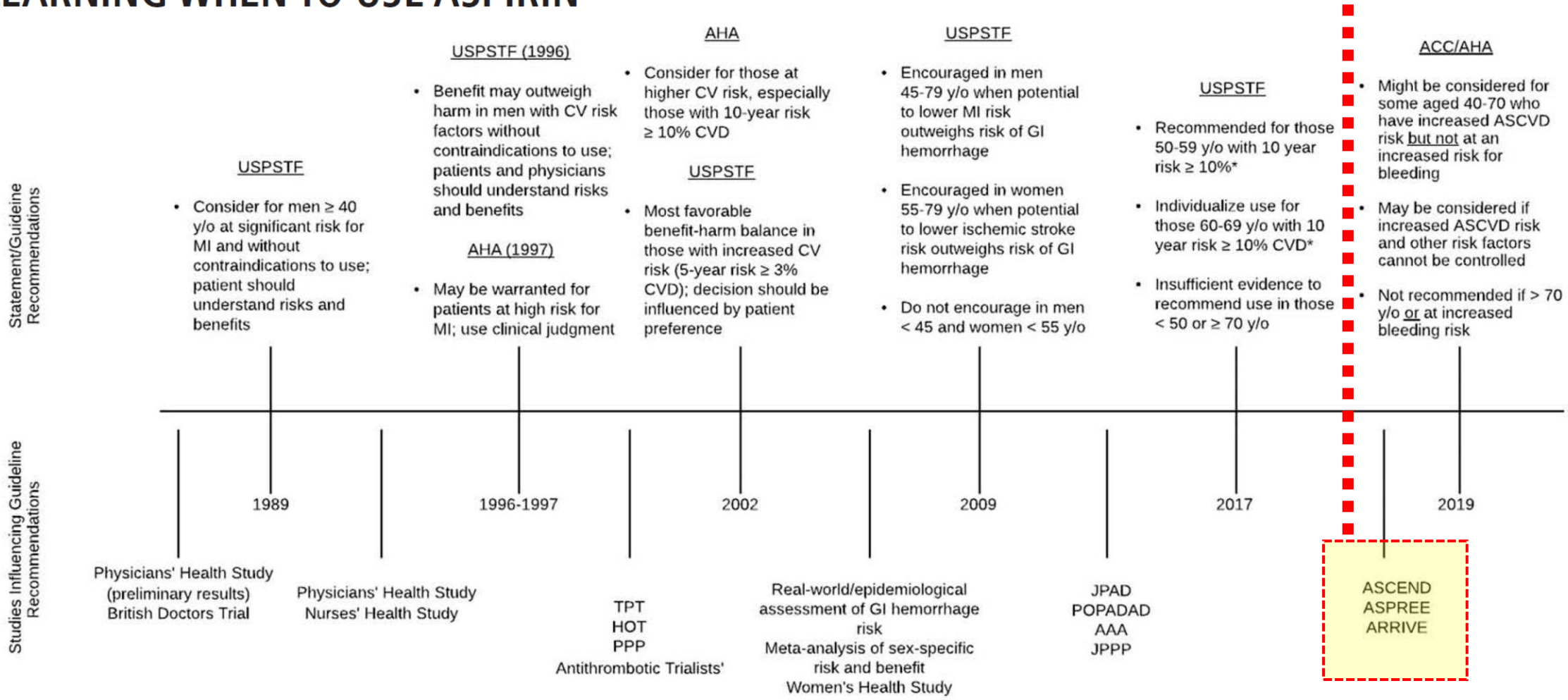
ESC
European Society of Cardiology

Recommendations	Class[a]	Level[b]
Aspirin 75 - 100 mg daily is recommended for secondary prevention of CVD.[619]	I	A

European Heart Journal (2021) **42**, 3227–3337

An aspirin a day? Clinical utility of aspirin therapy for the primary prevention of cardiovascular disease

LEARNING WHEN TO USE ASPIRIN



Expert Rev Cardiovasc Ther. 2019 Aug;17(8):561-573

Table 1. Trials of Aspirin for Primary Cardiovascular Prevention

Study	Year	Patients	Aspirin Dose	DM*	Mean or Median Follow-Up	Study Population	Primary Outcome Measure	Significant Efficacy
BDT[30]	1988	5139	300–500 mg/d	2%	5.6 y	Healthy men	CV death	No
PHS[31]	1989	22 071	325 mg every other day	4%	5 y	Healthy men	CV death	No
ETDRS[32]	1992	3711	650 mg/d	100%	5 y	DM†	All-cause mortality	No
ACBS[33]	1995	372	325 mg/d	19%	2.4 y	Carotid stenosis	Death, MI, stroke, TIA, stroke, MI, UA	No
HOT[34]	1998	18 790	75 mg/d	8%	3.8 y	Hypertension	CV death, MI, stroke	Yes
TPT[35]	1998	5085	75 mg/d	NR	6.7 y	CV risk factors	Coronary death and MI	Yes
PPP[36]	2001	4495	100 mg/d	17%	3.7 y	CV risk factors	CV death, nonfatal MI, stroke	No
ECLAP[37]	2004	518	100 mg/d	5%	3 y	Polycythemia vera	CV death, nonfatal MI, stroke, PE, VT	Yes
WHS[38]	2005	39 876	100 mg every other day	3%	10.1 y	Healthy women	CV death, nonfatal MI, stroke	No
CLIPS[39]	2007	366	100 mg/d	78%	2 y	PAD	CV death, MI, stroke	Yes
APLASA[40]	2007	98	81 mg/d	8%	2.3 y	AA syndrome	Acute thrombosis	No
POPADAD[41]	2008	1276	100 mg/d	100%	6.7 y	Diabetes, PAD	CV death, nonfatal MI, stroke, CLI	No
JPAD[42]	2008	2539	81–100 mg/d	100%	4.4 y	DM	Ischemic heart disease, stroke, PAD	No
AAA[43]	2010	3350	100 mg/d	3%	8.2 yr	PAD	CV death, MI, stroke, revascularization	No
JPPP[44]	2014	14 464	100 mg/d	34%	5.0 yr	CV risk factors	CV death, nonfatal MI, stroke	No

Circulation. 2016;134:1579–1594

Aspirin for Primary Prevention of Atherosclerotic Cardiovascular Disease

Advances in Diagnosis and Treatment

Table 1. Summary of the Major Aspirin Primary Prevention Trials

Trial (Acronym)	Aspirin Dose (Daily, Unless Noted)	Control Group	No. of Subjects (Aspirin/Control), Sex, Mean Age, y	Relative Risk (95% CI), Aspirin/Control Number of Events			
				Nonfatal MI	Nonfatal Stroke	Cardiovascular Mortality	All-Cause Mortality
British Doctors' Study (BDS)[26]	300-500 mg	No aspirin	3429/1710, M, 61	0.97 (0.67-1.41), 80/41	1.13 (0.72-1.77), 61/27	1.01 (0.74-1.37), 119/59	0.89 (0.74-1.08), 270/151
Physicians' Health Study (PHS)[27]	325 mg every other day	Placebo	11 037/11 034, M, 53	0.59 (0.47-0.74), 129/213	1.20 (0.91-1.59), 110/92	0.92 (0.66-1.28), 66/72	0.96 (0.80-1.14), 217/227
Early Treatment Diabetic Retinopathy Study (ETDRS)[28]	650 mg	Placebo	1856/1855, M/F, ~50	0.83 ($P \leq .05$), 524 (combined)	1.26 (0.89-1.80), 67/53	0.89 (0.76-1.04), 244/275	0.93 (0.79-1.09), 284/305
Thrombosis Prevention Trial (TPT)[29]	75 mg	Placebo	1268/1272, M, 57	0.65 (0.45-0.92), 47/73	0.64 (0.34-1.20), 16/25	1.05 (0.69-1.61), 42/40	1.03 (0.80-1.32), 113/110
Hypertension Optimal Treatment (HOT)[30]	75 mg	Placebo	9399/9391, M/F, 61	0.60 (0.45-0.81), 68/113	0.99 (0.78-1.24),[a] 146/148	0.95 (0.75-1.20), 133/140	0.93 (0.79-1.09), 284/305
Primary Prevention Project (PPP)[31]	100 mg	No aspirin	2226/2269, M/F, 64	0.69 (0.36-1.33), 15/22	0.84 (0.42-1.67), 15/18	0.56 (0.31-1.01), 17/31	0.81 (0.58-1.13), 62/78
Women's Health Study (WHS)[32]	100 mg every other day	Placebo	19 934/19 942, F, 54	1.01 (0.83-1.24), 184/181	0.81 (0.67-0.97), 198/244	0.95 (0.74-1.22), 120/126	0.95 (0.85-1.06), 609/642
Prevention of Progression of Arterial Disease and Diabetes (POPADAD)[33]	100 mg	Placebo	638/638, M/F, 60	0.98 (0.69-1.40), 55/56	0.71 (0.45-1.12), 29/41	1.23 (0.80-1.89), 43/35	0.93 (0.72-1.21), 94/101
Japanese Primary Prevention of Atherosclerosis with Aspirin for Diabetes (JPAD)[34]	81-100 mg	No aspirin	1262/1277, M/F, 65	1.35 (0.57-3.19), 12/9	1.01 (0.60-1.72), 27/27	0.10 (0.01-0.79), 1/10	0.91 (0.57-1.43), 33/38
Aspirin for Asymptomatic Atherosclerosis (AAA)[35]	100 mg	Placebo	1675/1675, M/F, 62	0.91 (0.65-1.28), 62/68	0.97 (0.62-1.52), 37/38	1.17 (0.72-1.89), 43/35	0.95 (0.85-1.06), 609/642
Japanese Primary Prevention Project (JPPP)[36]	100 mg	No aspirin	7220/7244, M/F, 70	0.53 (0.31-0.91), 20/38	1.00 (0.77-1.31), 109/109	1.02 (0.71-1.47), 58/57	0.98 (0.84-1.15), 297/303
Total[b]	All doses		118 445	0.78 (0.71-0.87)	0.95 (0.85-1.06)	0.94 (0.86-1.03)	0.94 (0.89-0.99)
Low-dose aspirin trials[b]	≤100 mg		87 524	0.83 (0.74-0.94)	0.86 (0.76-0.98)	0.97 (0.85-1.10)	0.95 (0.89-1.01)

Table 2. Summary of Recent Meta-Analyses of Aspirin for Primary Cardiovascular Prevention

Study Characteristic	ATT[45]	Bartolucci[46]	Raju[47]	Berger[48]	Seshasai[49]	Xie[50]	Raju[51]	Guirguis-Blake[52,53]
Publication date	2009	2011	2011	2011	2012	2014	2015	2016
Type	Patient level	Study level	Study level	Study level	Study level	Study level	Study level	Study level
Pooled patients	95 000	100 038	100 076	102 621	102 621	107 686	114 734	118 445
Summary measure	RaR (95% CI)	OR (95% CI)	RR (95% CI)	RR (95% CI)	OR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
Studies included	6	9	9	9	9	14	10	11
Follow-up	330,000 PY	NR	3.8–10.1 yr	710,053 PY	≈700,000 PY	734,170 PY	NR	3.6–10.1 y
Serious vascular events	0.88 (0.82–0.94)*	0.87 (0.80–0.93)*	0.88 (0.83–0.94)*	0.90 (0.85–0.96)*	0.90 (0.85–0.96)*	0.90 (0.85–0.95)*	0.89 (0.82–0.97)*	NR
Any MI	NR	NR	0.83 (0.69–1.00)*	0.86 (0.74–1.00)*	NR	0.86 (0.75–0.98)*	0.78 (0.65–0.94)*	NR
Fatal MI	NR	NR	NR	NR	1.06 (0.83–1.37)	NR	NR	NR
Nonfatal MI	0.77 (0.69–0.86)*	0.81 (0.67–0.99)*	NR	NR	0.80 (0.67–0.96)*	NR	0.80 (0.64–0.99)*	0.78 (0.71–0.87)*
All-cause death	NR	0.95 (0.88–1.01)	0.94 (0.88–1.00)*	0.94 (0.89–1.00)	0.94 (0.88–1.00)	0.94 (0.89–0.99)*	0.94 (0.89–1.00)	0.94 (0.89–0.99)*
Cardiovascular	0.97 (0.87–1.09)	0.96 (0.80–1.14)	0.96 (0.84–1.09)	0.99 (0.85–1.14)	0.99 (0.85–1.15)	1.04 (0.86–1.25)	0.95 (0.84–1.07)	0.94 (0.86–1.03)
Any stroke	0.95 (0.85–1.06)	0.92 (0.83–1.02)	NR	0.94 (0.84–1.06)	0.94 (0.84–1.06)	0.95 (0.87–1.05)	0.94 (0.84–1.06)	0.95 (0.85–1.06)
Hemorrhagic	1.32 (1.00–1.75)*	NR	1.36 (1.01–1.82)*	1.35 (1.01–1.81)*	NR	1.34 (1.01–1.79)*	1.43 (1.10–1.86)*	1.33 (1.03–1.71)*
Ischemic	0.86 (0.74–1.00)*	NR	0.86 (0.75–0.98)*	0.87 (0.73–1.02)	NR	0.86 (0.75–0.98)*	NR	NR
Major bleeding	1.54 (1.30–1.82)*	NR	1.66 (1.41–1.95)*	1.62 (1.31–2.00)*	NR	1.55 (1.35–1.78)*	1.69 (1.43–1.98)*	NR
Gastrointestinal	NR	NR	1.37 (1.15–1.62)*	1.29 (1.24–1.47)*	NR	NR	1.64 (1.30–2.07)*	1.59 (1.32–1.91)*

*Statistically significant.

Circulation. 2016;134:1579–1594

Guidelines for the Primary Prevention of Stroke

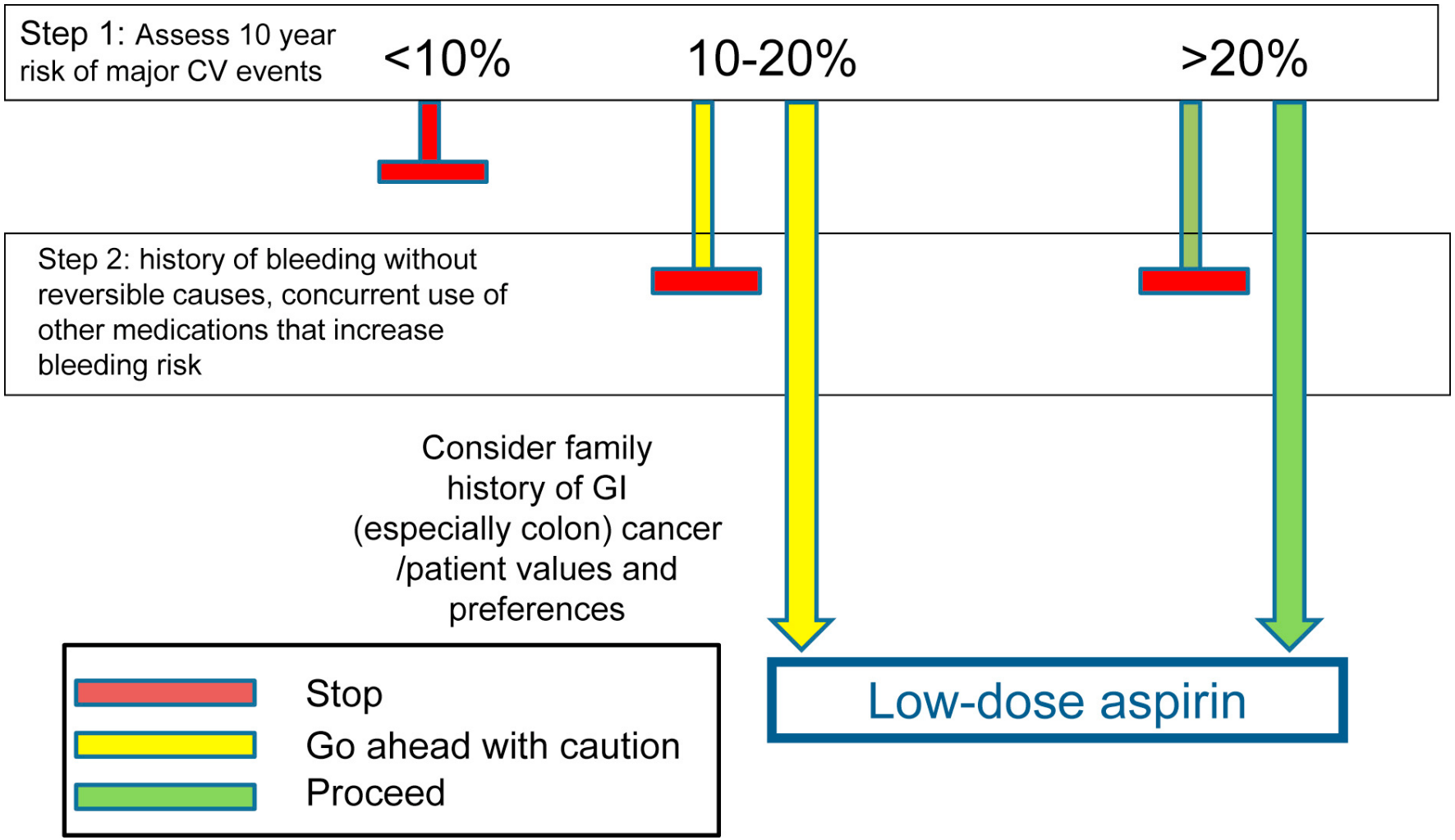
A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association

Section	2014 Recommendation	Description of Change from 2011
Diabetes mellitus	Control of blood pressure in accordance with an AHA/ACC/CDC advisory to a target of <140/90 mm Hg is recommended in patients with type 1 or type 2 diabetes mellitus (*Class I; Level of Evidence A*).	Reworded to reference AHA/ACC/CDC advisory
	The usefulness of aspirin for primary stroke prevention for patients with diabetes mellitus but low 10-y risk of cardiovascular disease is unclear (*Class IIb; Level of Evidence B*).	Deleted the phrase "however, administering aspirin may be reasonable"
Antiplatelet agents and aspirin	The use of aspirin for cardiovascular (including but not specific to stroke) prophylaxis is reasonable for people whose risk is sufficiently high (10-y risk >10%) for the benefits to outweigh the risks associated with treatment. A cardiovascular risk calculator to assist in estimating 10-y risk can be found online at http://my.americanheart.org/cvriskcalculator (*Class IIa; Level of Evidence A*).	Reworded to include cardiovascular risk calculator and link; changed from Class I to IIa
	Aspirin might be considered for the prevention of a first stroke in people with chronic kidney disease (ie, estimated glomerular filtration rate <45 mL·min^{-1}·1.73 m^{-2}) (*Class IIb; Level of Evidence C*). This recommendation does not apply to severe kidney disease (stage 4 or 5; estimated glomerular filtration rate <30 mL·min^{-1}·1.73 m^{-2}).	New recommendation
	Cilostazol may be reasonable for the prevention of a first stroke in people with peripheral arterial disease (*Class IIb; Level of Evidence B*).	New recommendation
	As a result of a lack of relevant clinical trials, antiplatelet regimens other than aspirin and cilostazol are not recommended for the prevention of a first stroke (*Class III; Level of Evidence C*).	New recommendation

Aspirin Therapy in Primary Cardiovascular Disease Prevention

A Position Paper of the European Society of Cardiology Working Group on Thrombosis

J Am Coll Cardiol 2014;64:319–27



Aspirin for Primary Cardiovascular Risk Prevention and Beyond in Diabetes Mellitus

Figure 3. Risk stratification approach for aspirin use in primary prevention of cardiovascular disease for a patient with diabetes mellitus, on the background assumption of optimal management of other cardiovascular disease risk factors.

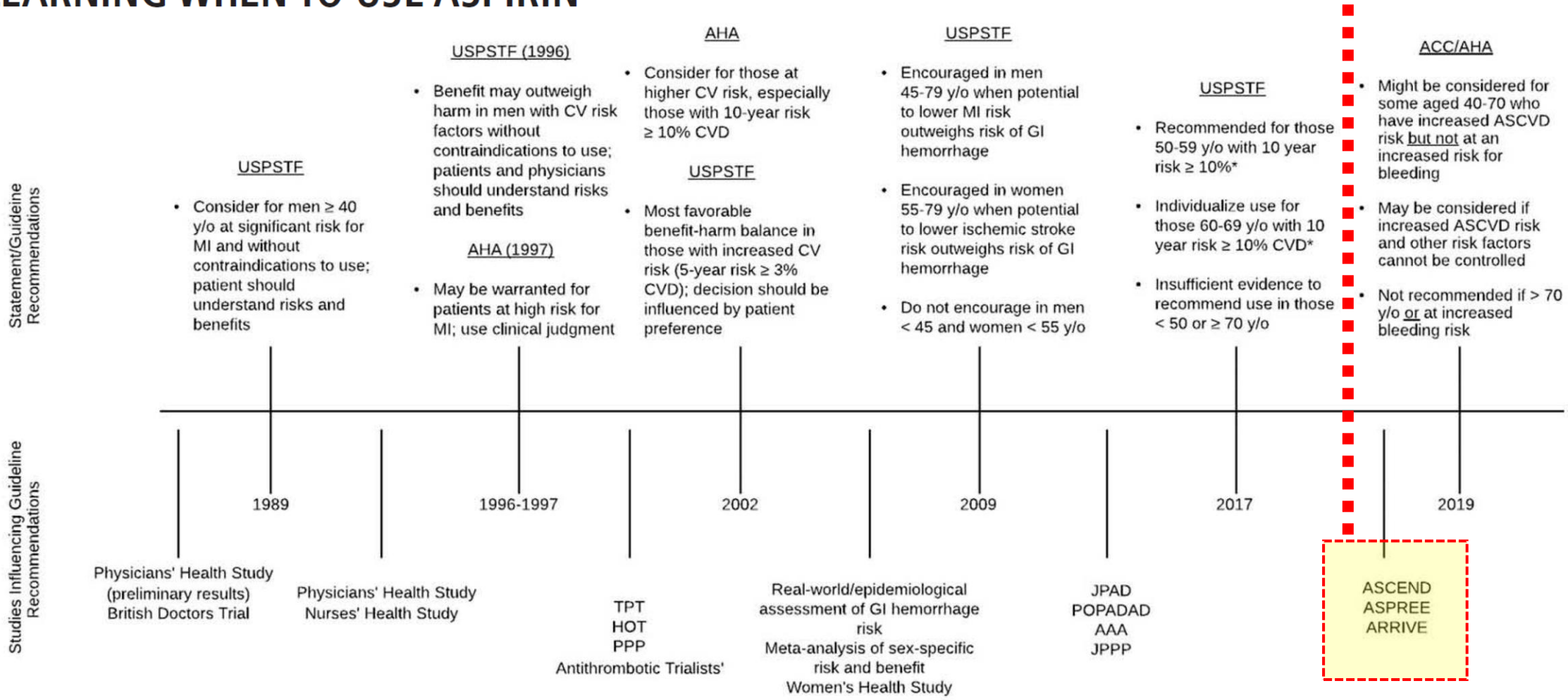
Age (years)	10-year CVD risk	Family history of CRC		No family history of CRC	
		HBR	no HBR	HBR	no HBR
<50	<5%	No ASA	No ASA	No ASA	No ASA
<50	5–10%	No ASA	Initiate ASA	No ASA	Clinical judgment
50–59	5–10%	No ASA	Initiate ASA	No ASA	Clinical judgment
50–59	10–20%	Clinical judgment	Initiate ASA	No ASA	Initiate ASA
60–69	10–20%	Clinical judgment	Initiate ASA	No ASA	Clinical judgment
≥70	≥20%	No ASA	Clinical judgment	No ASA	Clinical judgment

High bleeding risk (HBR) is defined as a history of bleeding without reversible causes and concurrent use of other medications that increase bleeding risk. **Clinical judgment** includes a balanced assessment of risk and benefits of aspirin therapy and factors patients' preference and willingness to comply with aspirin for the subsequent 10 years. **CRC** indicates colorectal cancer; and CVD, cardiovascular disease

Circulation. 2016;134:1579–1594

An aspirin a day? Clinical utility of aspirin therapy for the primary prevention of cardiovascular disease

LEARNING WHEN TO USE ASPIRIN

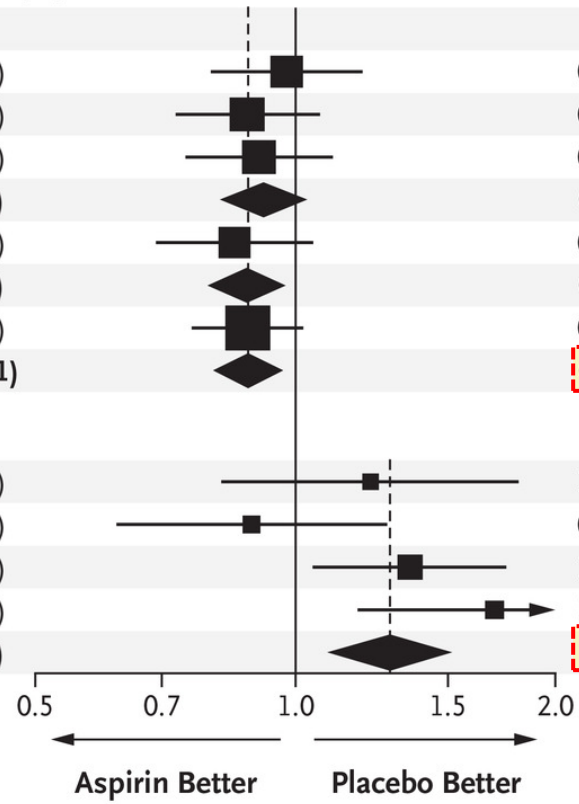


Expert Rev Cardiovasc Ther. 2019 Aug;17(8):561-573

Effects of Aspirin for Primary Prevention in Persons with Diabetes Mellitus

ASCEND

Type of Event	Aspirin (N=7740)	Placebo (N=7740)	Rate Ratio (95% CI)	P Value
	no. of participants with event (%)			
Vascular Outcomes				
Nonfatal myocardial infarction	191 (2.5)	195 (2.5)	0.98 (0.80–1.19)	
Nonfatal presumed ischemic stroke	202 (2.6)	229 (3.0)	0.88 (0.73–1.06)	
Vascular death excluding intracranial hemorrhage	197 (2.5)	217 (2.8)	0.91 (0.75–1.10)	
Any serious vascular event excluding TIA	**542 (7.0)**	**587 (7.6)**	**0.92 (0.82–1.03)**	
TIA	168 (2.2)	197 (2.5)	0.85 (0.69–1.04)	
Any serious vascular event including TIA	**658 (8.5)**	**743 (9.6)**	**0.88 (0.79–0.97)**	**0.01**
Any arterial revascularization	340 (4.4)	384 (5.0)	0.88 (0.76–1.02)	
Any serious vascular event or revascularization	**833 (10.8)**	**936 (12.1)**	**0.88 (0.80–0.97)**	
Major Bleeding				
Intracranial hemorrhage	55 (0.7)	45 (0.6)	1.22 (0.82–1.81)	
Sight-threatening bleeding in eye	57 (0.7)	64 (0.8)	0.89 (0.62–1.27)	
Serious gastrointestinal bleeding	137 (1.8)	101 (1.3)	1.36 (1.05–1.75)	
Other major bleeding	74 (1.0)	43 (0.6)	1.70 (1.18–2.44)	
Any major bleeding	**314 (4.1)**	**245 (3.2)**	**1.29 (1.09–1.52)**	**0.003**



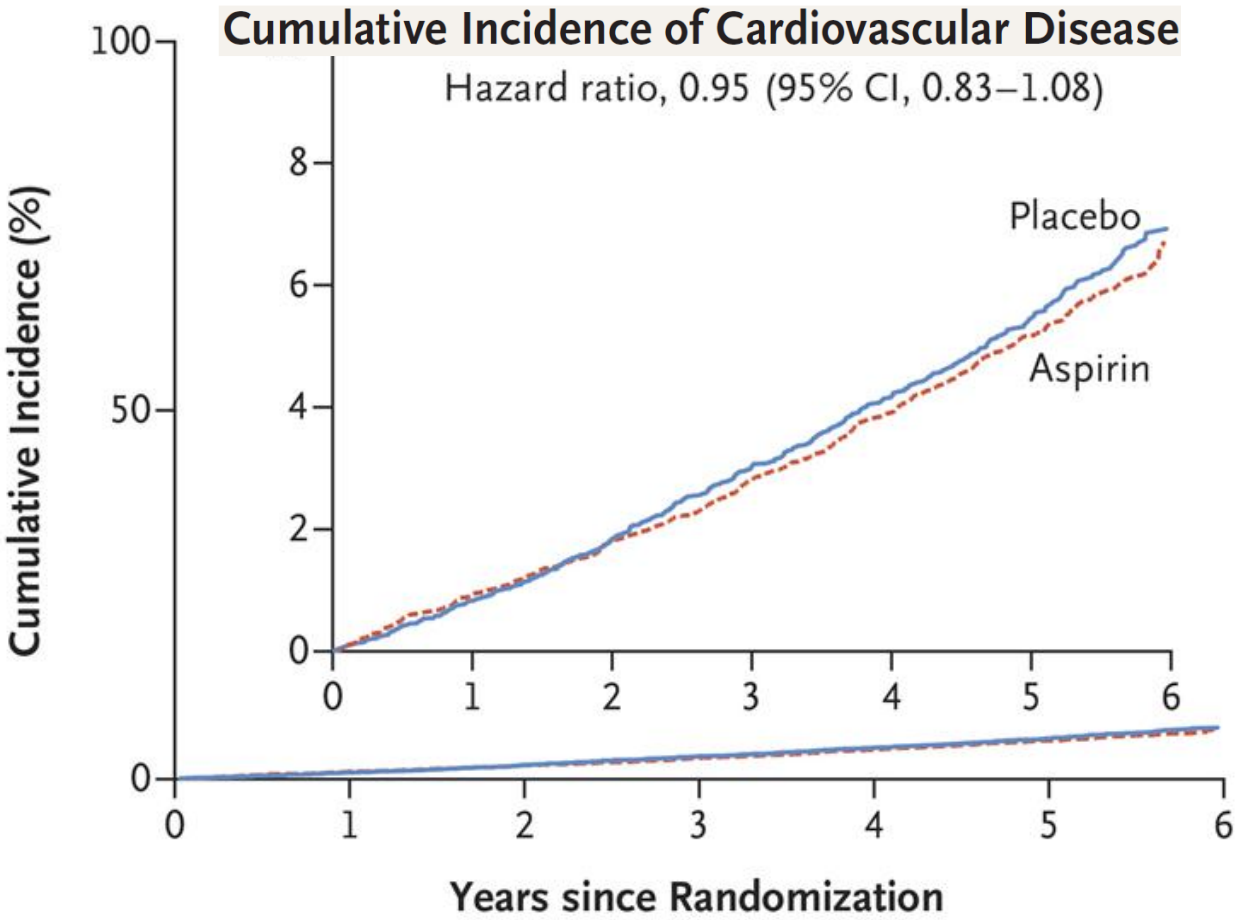
N Engl J Med 2018;379:1529-39

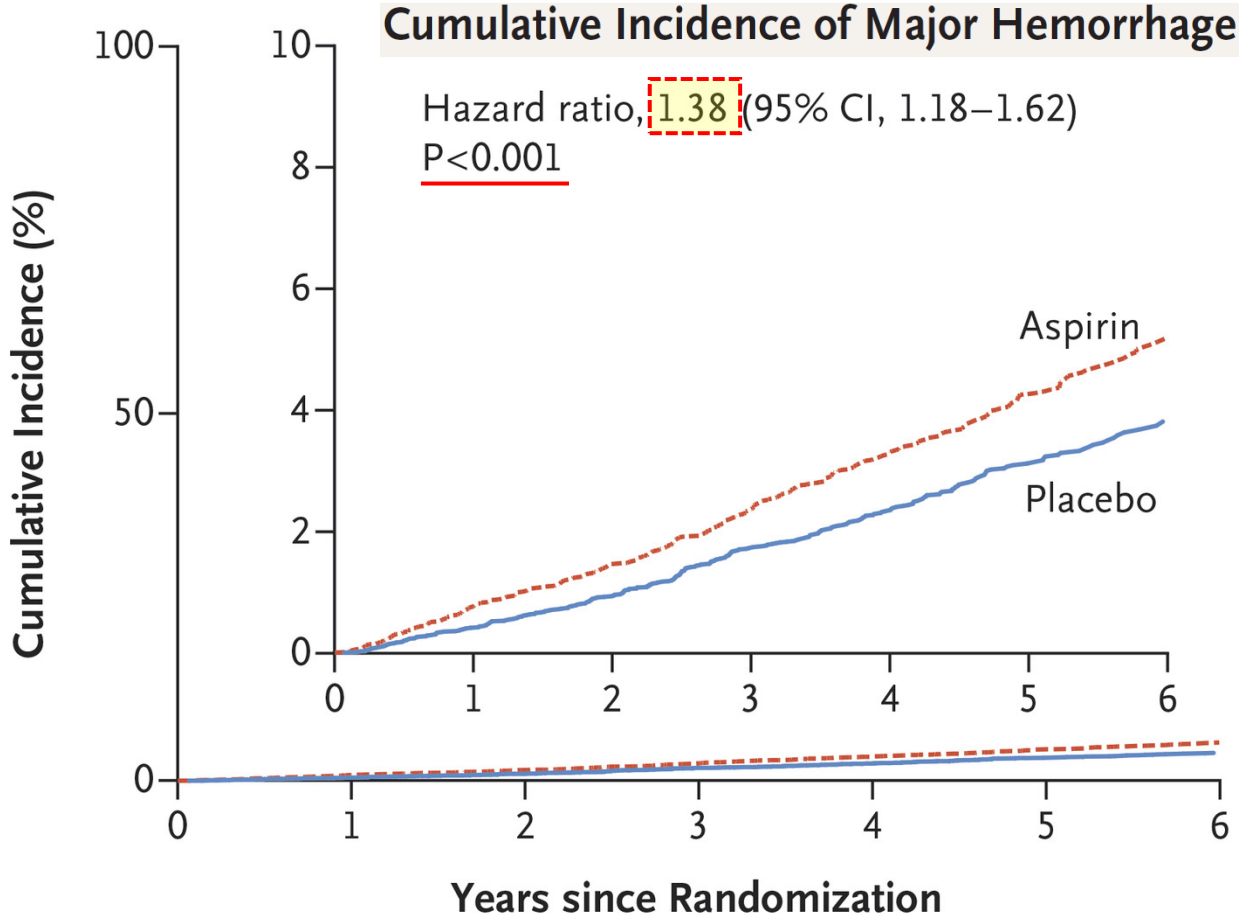
Effect of Aspirin on Cardiovascular Events and Bleeding in the Healthy Elderly

ASPREE

Conclusions

The use of low-dose aspirin as a primary prevention strategy in older adults resulted in a **significantly higher risk of major hemorrhage** and *did not result in a significantly lower risk of cardiovascular disease* than placebo.

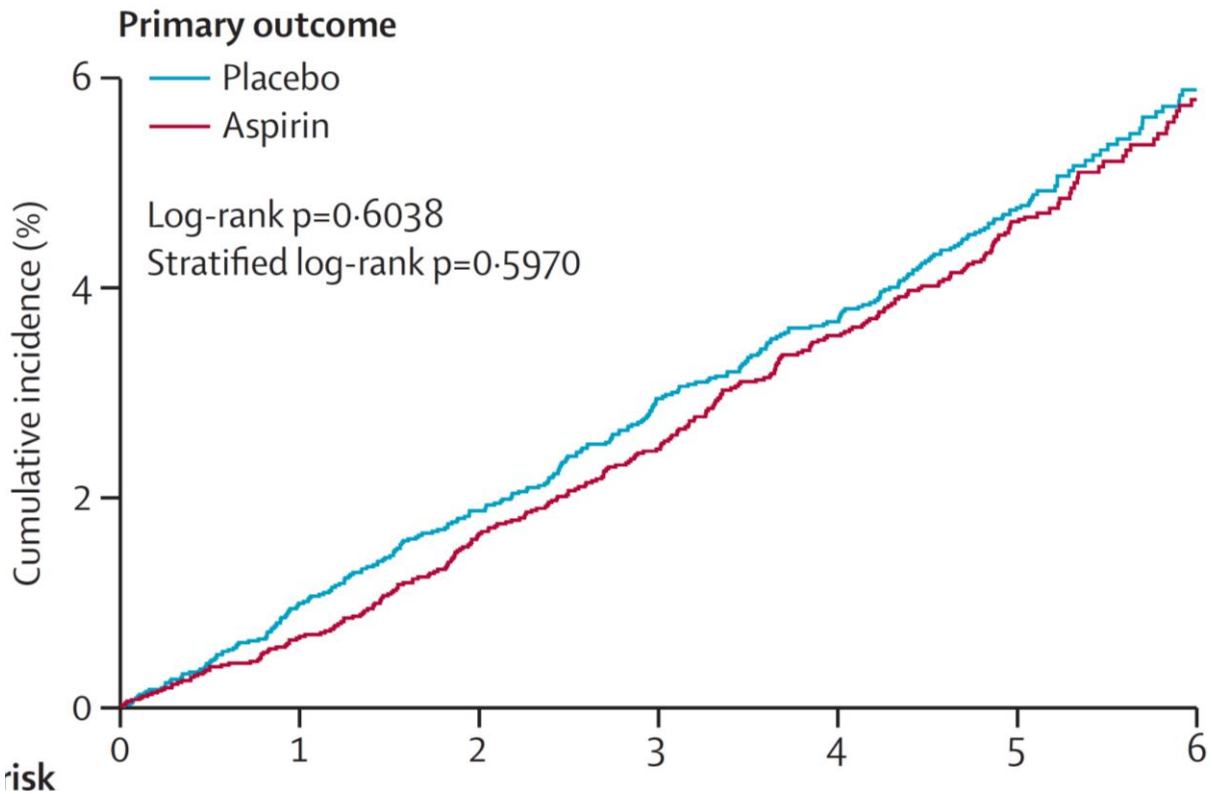


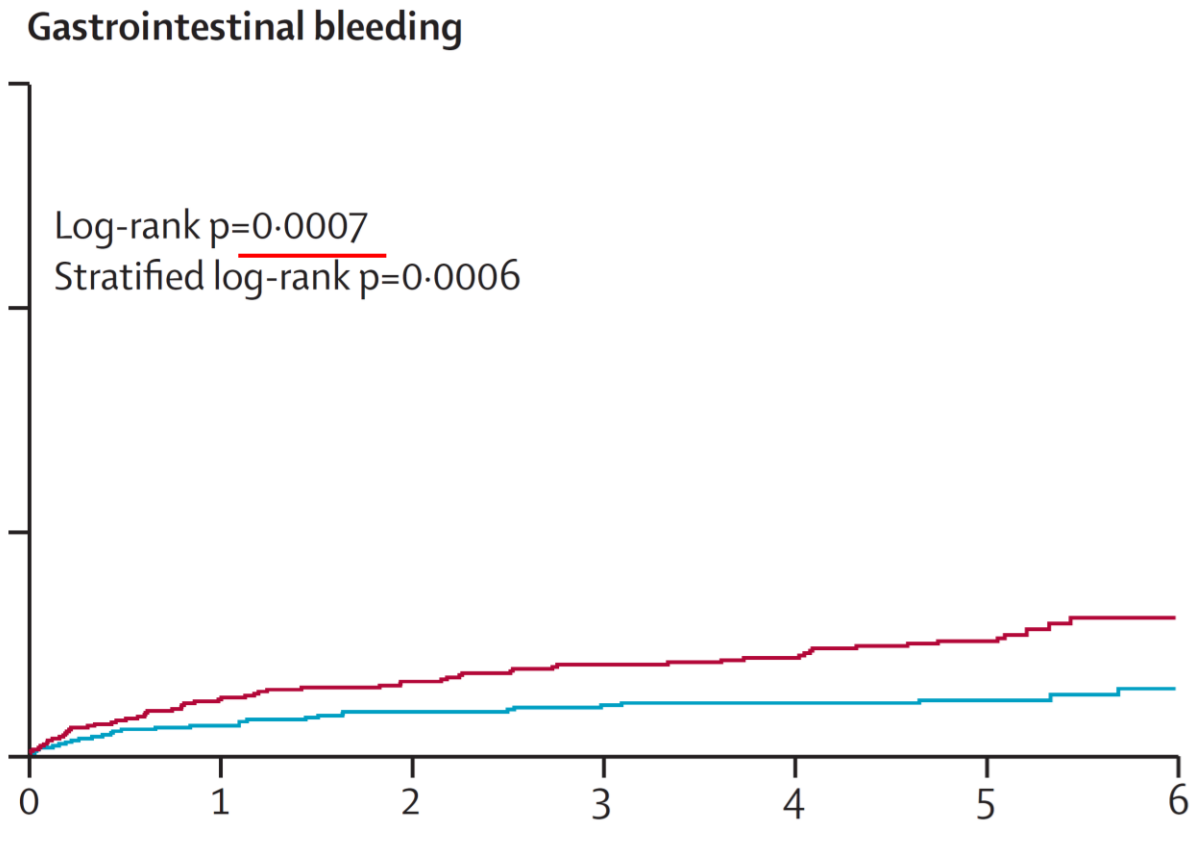


N Engl J Med 2018;379:1509-18

ARRIVE

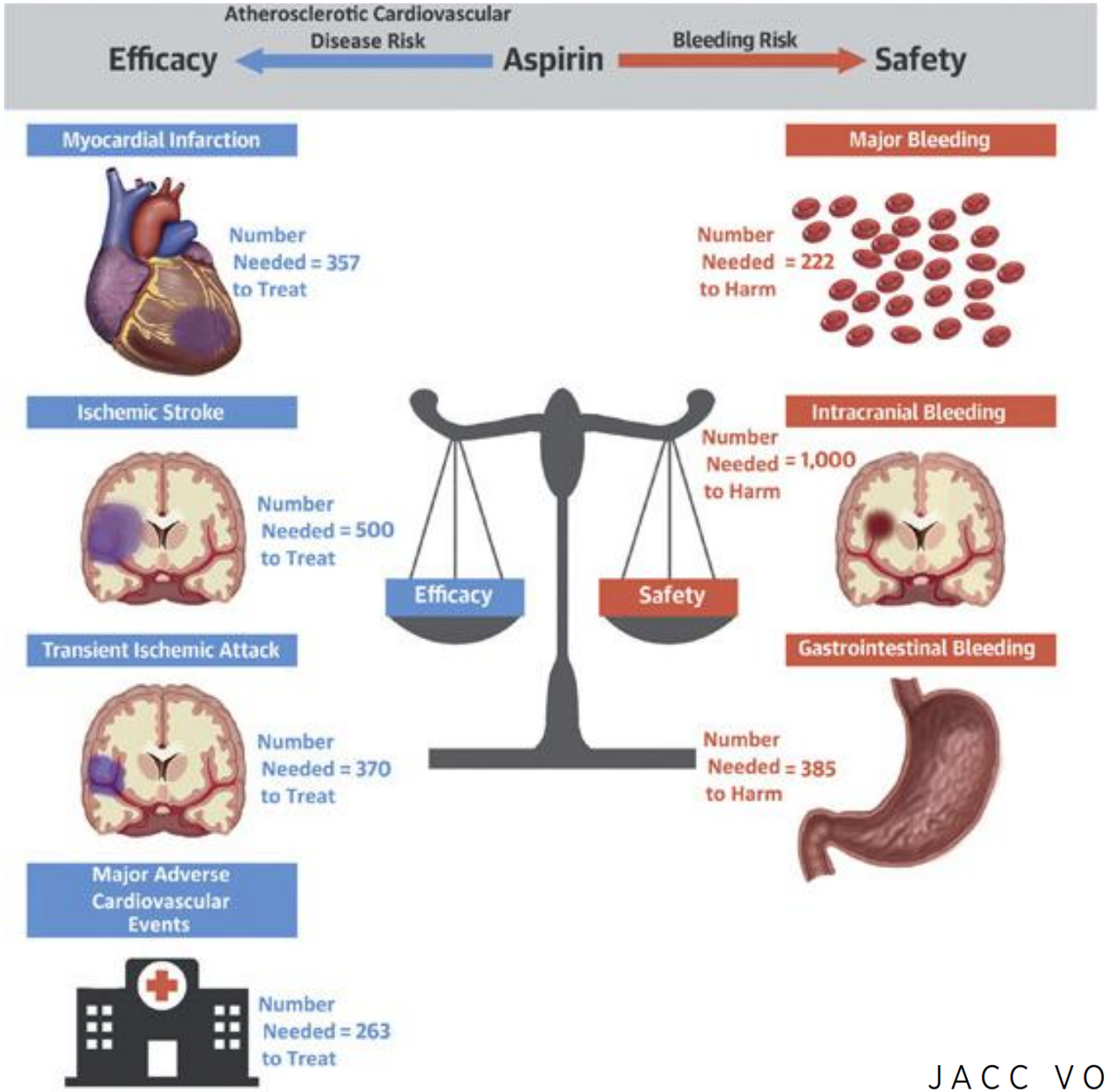
Use of aspirin to reduce risk of initial vascular events in patients at moderate risk of cardiovascular disease (ARRIVE): a randomised, double-blind, placebo-controlled trial





Lancet 2018; 392: 1036–46

Aspirin for Primary Prevention of Cardiovascular Events



2021 ESC Guidelines on cardiovascular disease prevention in clinical practice

Recommendations	Class[a]	Level[b]
Concomitant use of a proton pump inhibitor is recommended in patients receiving antiplatelet therapy who are at high risk of gastrointestinal bleeding.[622,623]	I	A

Recommendations		
In patients with DM at high or very high CVD risk, low-dose aspirin may be considered for primary prevention in the absence of clear contraindications.[5,624,625]	IIb	A
Antiplatelet therapy is not recommended in individuals with low/moderate CV risk due to the increased risk of major bleeding.[624,626–630]	III	A

European Heart Journal (2021) **42**, 3227–3337

2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease

Recommendations for Aspirin Use

Referenced studies that support recommendations are summarized in Online Data Supplements 17 and 18.

COR	LOE	Recommendations
IIb	A	1. Low-dose aspirin (75-100 mg orally daily) might be considered for the primary prevention of ASCVD among select adults 40 to 70 years of age who are at higher ASCVD risk but not at increased bleeding risk.[S4.6-1–S4.6-8]
III: Harm	B-R	2. Low-dose aspirin (75-100 mg orally daily) should not be administered on a routine basis for the primary prevention of ASCVD among adults >70 years of age.[S4.6-9]
III: Harm	C-LD	3. Low-dose aspirin (75-100 mg orally daily) should not be administered for the primary prevention of ASCVD among adults of any age who are at increased risk of bleeding.[S4.6-10]

Circulation. 2019;140:e596–e646

10. Cardiovascular Disease and Risk Management: Standards of Care in Diabetes—2025

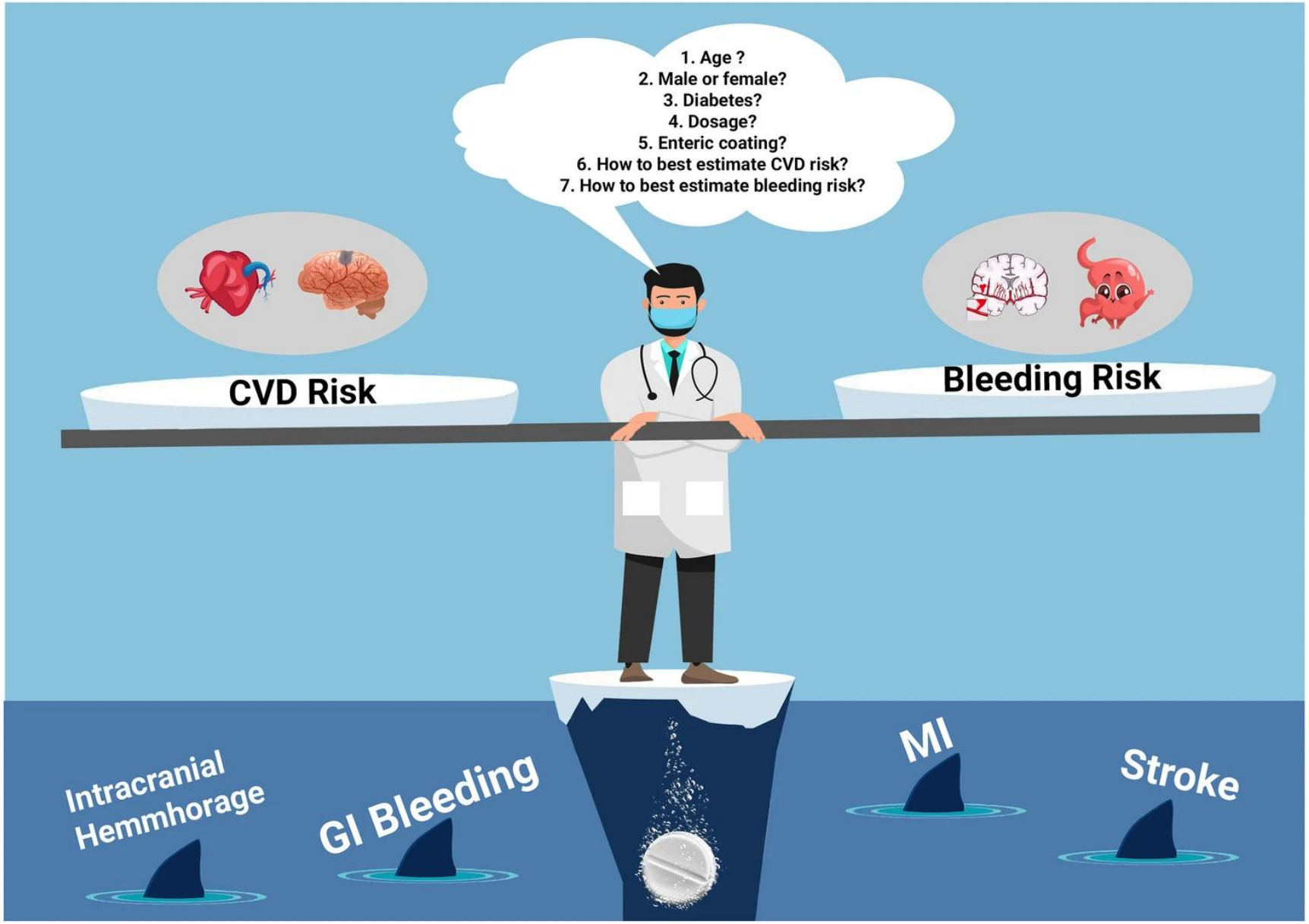
ANTIPLATELET AGENTS

10.38 Aspirin therapy (75–162 mg/day) may be considered as a primary prevention strategy in those with diabetes who are at increased cardiovascular risk after a comprehensive discussion with the individual on the benefits versus the comparable increased risk of bleeding. **A**

Diabetes Care 2025;48(Suppl. 1):S207–S238

Aspirin for Primary Prevention of Cardiovascular Diseases: "WALTZ" with the Evidence

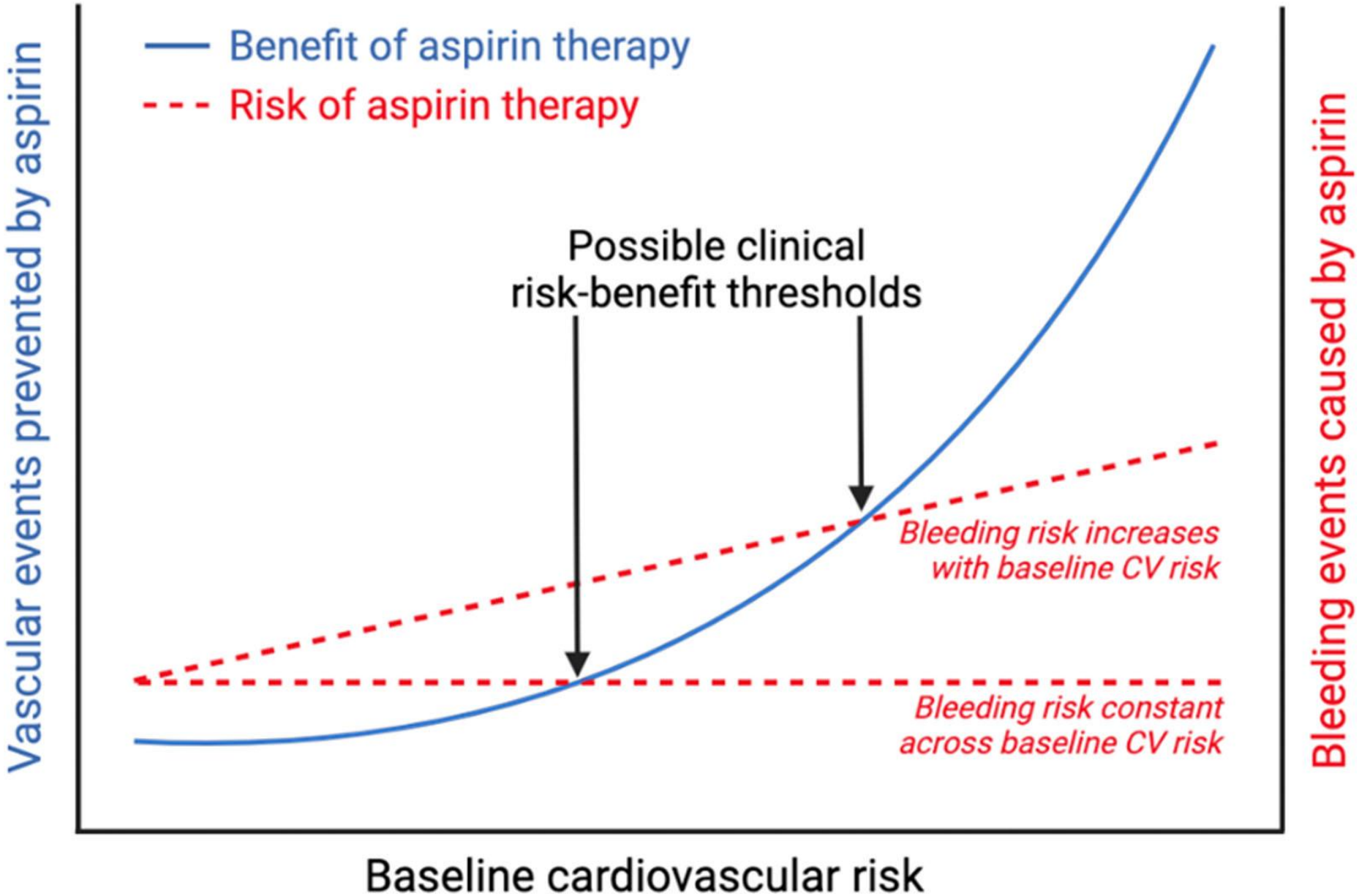
Kyriakos Dimitriadis[1] · Emilia Lazarou[1] · Panagiotis Tsioufis[1] · Stergios Soulaidopoulos[1] · Konstantinos Tsioufis[1]



Current Cardiology Reports (2022) 24:1139–1147

Aspirin for the Primary Prevention of Cardiovascular Disease: Time for a Platelet-Guided Approach

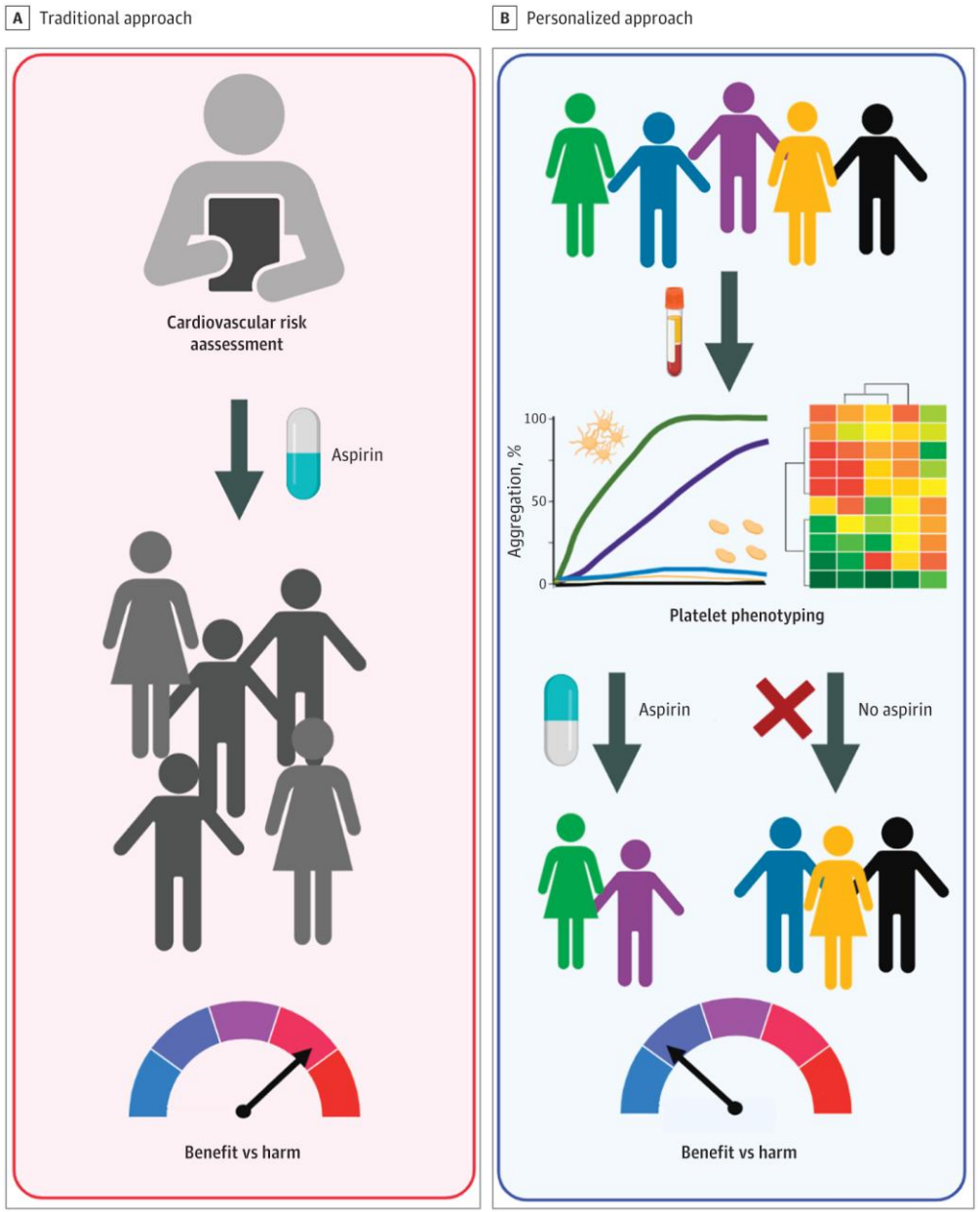
Arterioscler Thromb Vasc Biol. 2022;42:1207–1216.



Editorial

Aspirin for Primary Prevention—Time to Rethink Our Approach

Jeffrey S. Berger, MD, MS



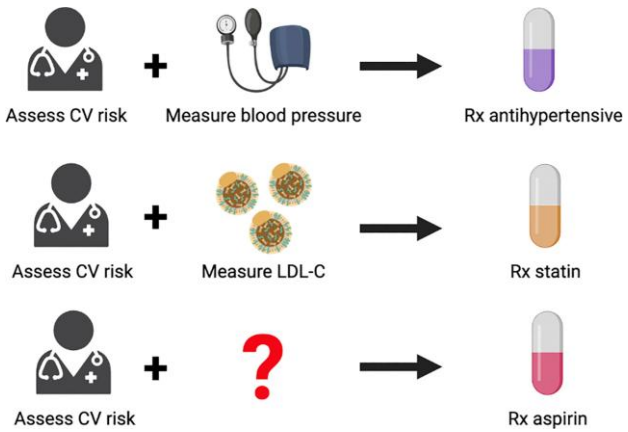
A ***precision medicine*** approach holds promise to improve the identification of individuals who may benefit from the use of aspirin for primary prevention of CVD.

Antihypertensive → elevated BP

Statin → elevated LDL

Antidiabetic → elevated GLU

Aspirin → ???



Arterioscler Thromb Vasc Biol. 2022;42:1207–1216.

What if clinicians were able to identify individuals based on their increased platelet activity (eg, the target of antiplatelet therapy) and an individual's likelihood of experiencing a platelet-mediated event?

JAMA Network Open. 2022;5(4):e2210144

Heart Disease and Stroke Statistics–2023 Update: A Report From the American Heart Association

Circulation. 2023;147:e93–e621

ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
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ΠΡΟΓΡΑΜΜΑ ΜΕΤΑΠΤΥΧΙΑΚΩΝ ΣΠΟΥΔΩΝ
«ΚΑΡΔΙΟΜΕΤΑΒΟΛΙΚΗ ΙΑΤΡΙΚΗ»

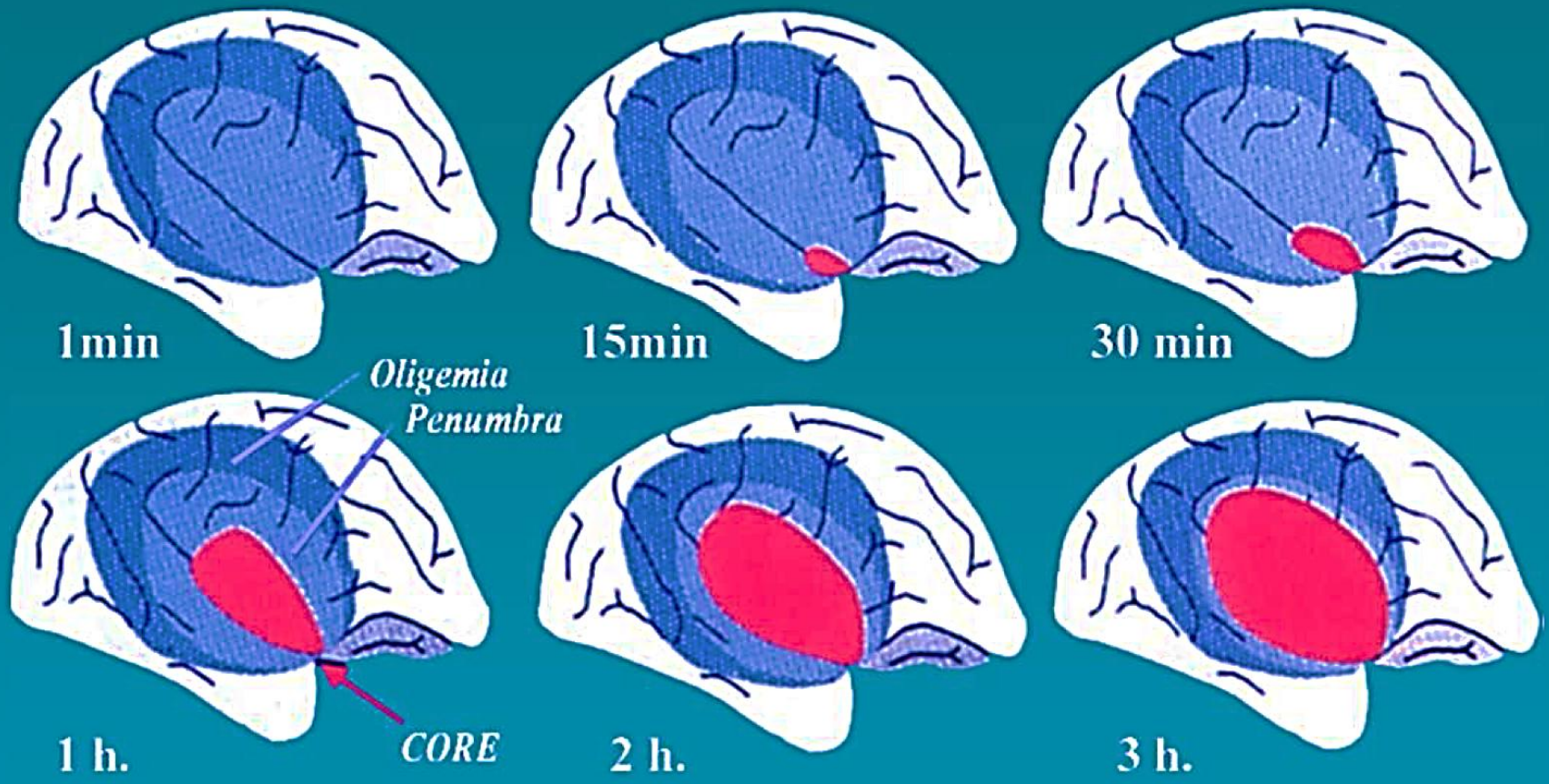
ΑΕΕ και πρόληψη. Πρωτογενής Πρόληψη ΑΕΕ και η διαχείριση της αντιθρομβωτικής αγωγής στη Δευτερογενή Πρόληψη

Κακαλέτσης Νικόλαος

Παθολόγος
Επιμελητής Β', Β' Παθολογική Κλινική, Γ.Ν.Θ. Ιπποκράτειο
Μεταδιδακτορικός Ερευνητής Ιατρικής Σχολής Α.Π.Θ.
Stroke Research Fellow, HUS Helsinki University Hospital and University of Helsinki, Finland

4 Μαρτίου 2026

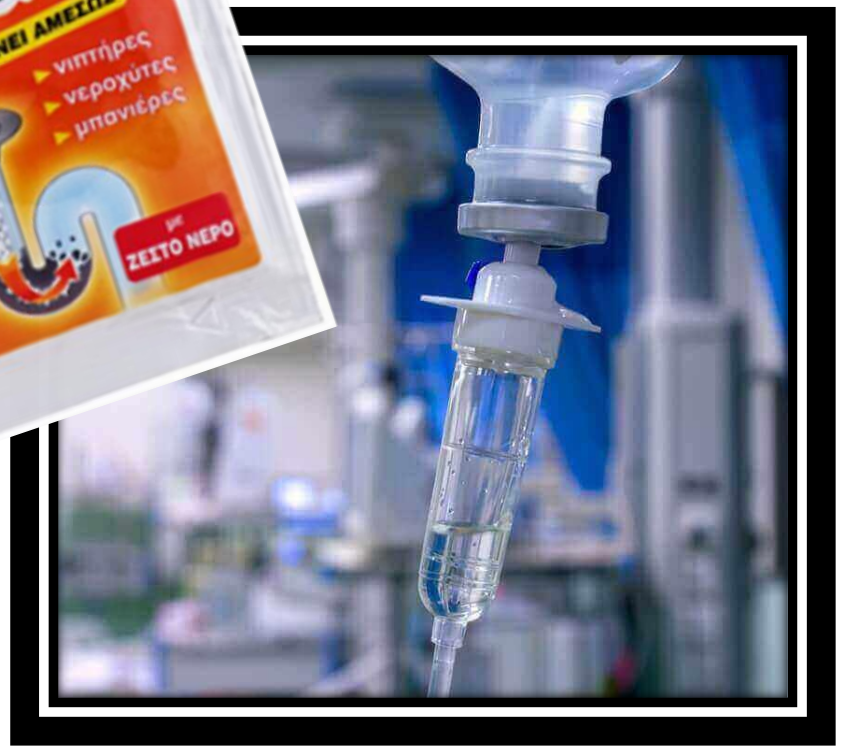
Growth of Infarction

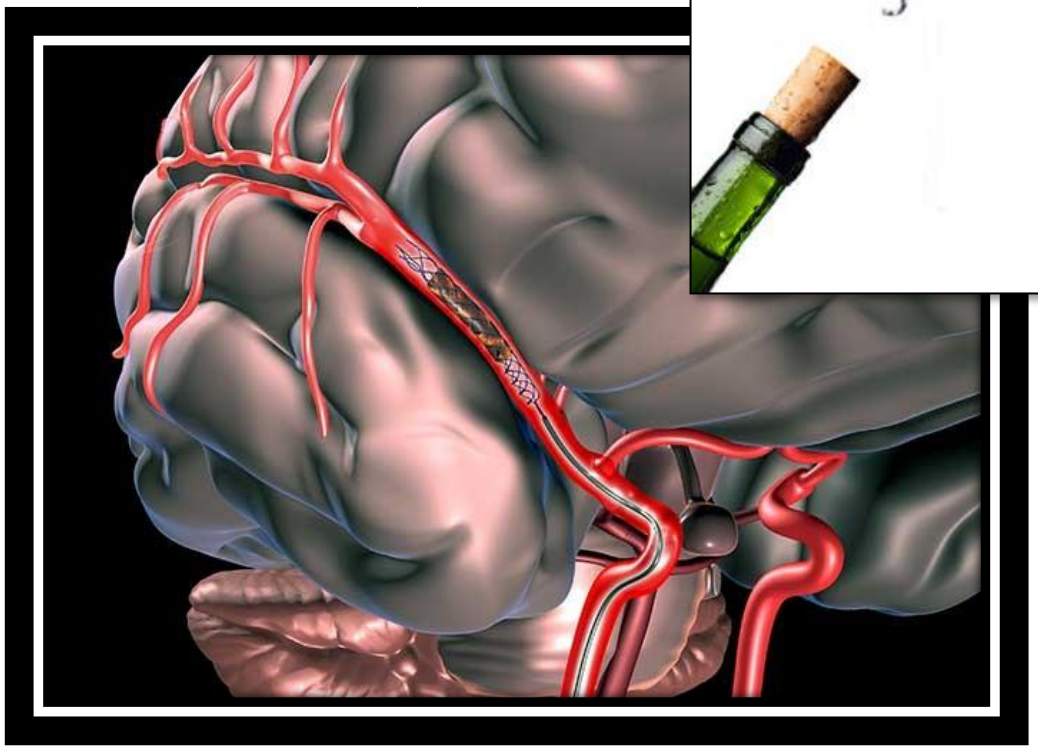


The Ischemic Penumbra: A Dynamic [time + space] Concept

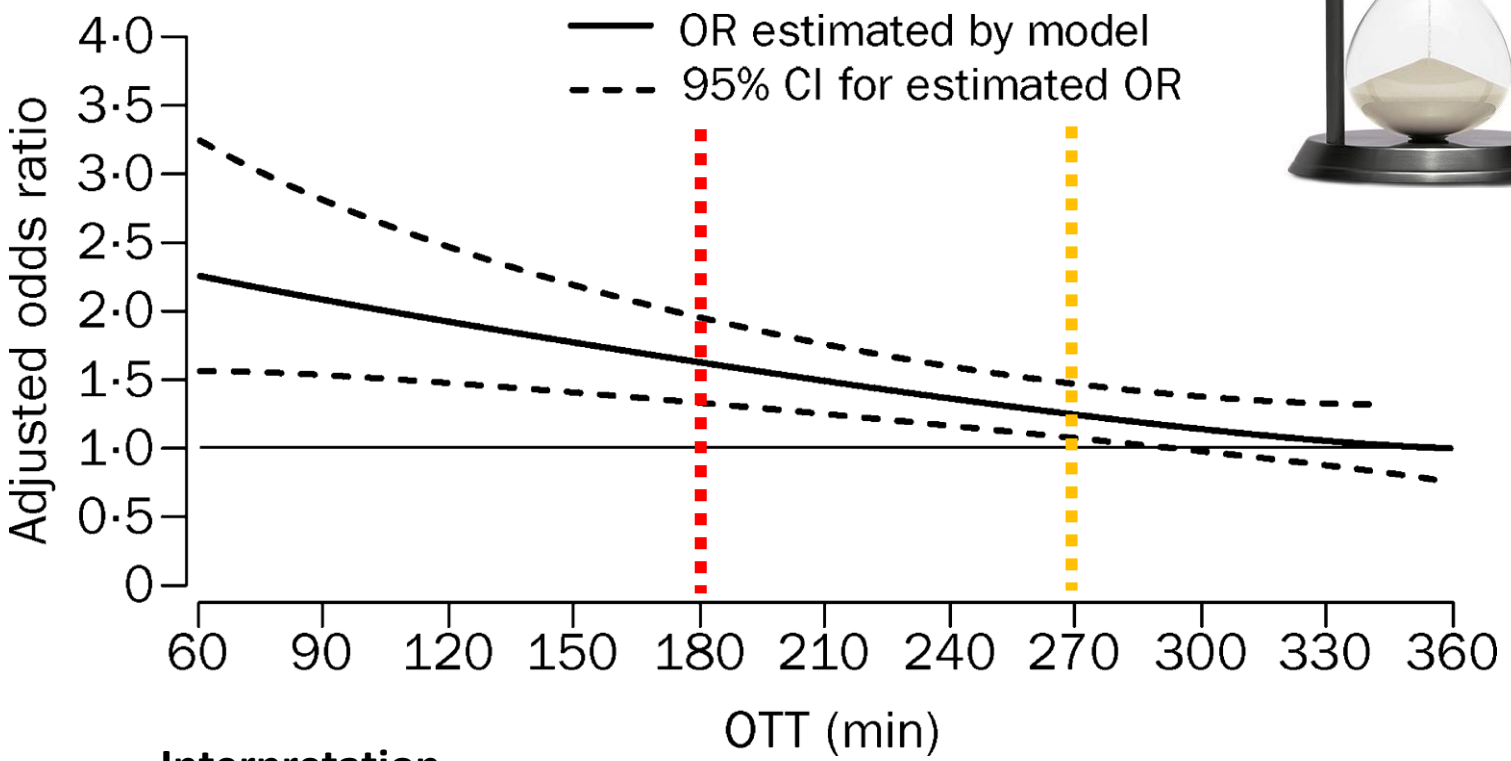
Baron JC, Cerebrovasc Dis 1999; 9: 193-201.

Intravenous thrombolysis & Mechanical thrombectomy





Association of outcome with early stroke treatment: pooled analysis of ATLANTIS, ECASS, and NINDS rt-PA stroke trials



Interpretation

The sooner that rt-PA is given to stroke patients, the greater the benefit, especially if started within 90 min. Our results suggest a potential benefit beyond 3 h, but this potential might come with some risks.

Lancet 2004; **363:** 768–74

ΣΥΣΤΑΣΕΙΣ ΓΙΑ ΤΗΝ ΕΝΔΟΦΛΕΒΙΑ ΘΡΟΜΒΟΛΥΣΗ ΣΕ ΑΣΘΕΝΕΙΣ ΜΕ ΟΞΥ ΙΣΧΑΙΜΙΚΟ ΑΓΓΕΙΑΚΟ ΕΓΚΕΦΑΛΙΚΟ ΕΠΕΙΣΟΔΙΟ.

Ένας κλινικός οδηγός από τον Ελληνικό Οργανισμό Εγκεφαλικών

1. Κάθε ασθενής με συμπτώματα οξέος ΑΕΕ θα πρέπει να αντιμετωπίζεται ως πιθανός υποψήφιος για θεραπεία με ενδοφλέβια θρομβόλυση εφόσον βρίσκεται εντός 4,5 ωρών από την έναρξη των συμπτωμάτων (Σύσταση 1Α). Στο πλαίσιο αυτό, θα πρέπει να του παραχωρείται άμεση πρόσβαση στον αξονικό ή μαγνητικό τομογράφο, ώστε να υποβληθεί σε επείγουσα απεικόνιση του εγκεφάλου όσο το δυνατόν ταχύτερα για να αποκλειστεί το ενδεχόμενο ενδοκράνιας αιμορραγίας (Σύσταση 1Γ).

HEART VESSELS & BRAIN | ΙΟΥΛΙΟΣ - ΣΕΠΤΕΜΒΡΙΟΣ 2017

Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual patient data from five randomised trials

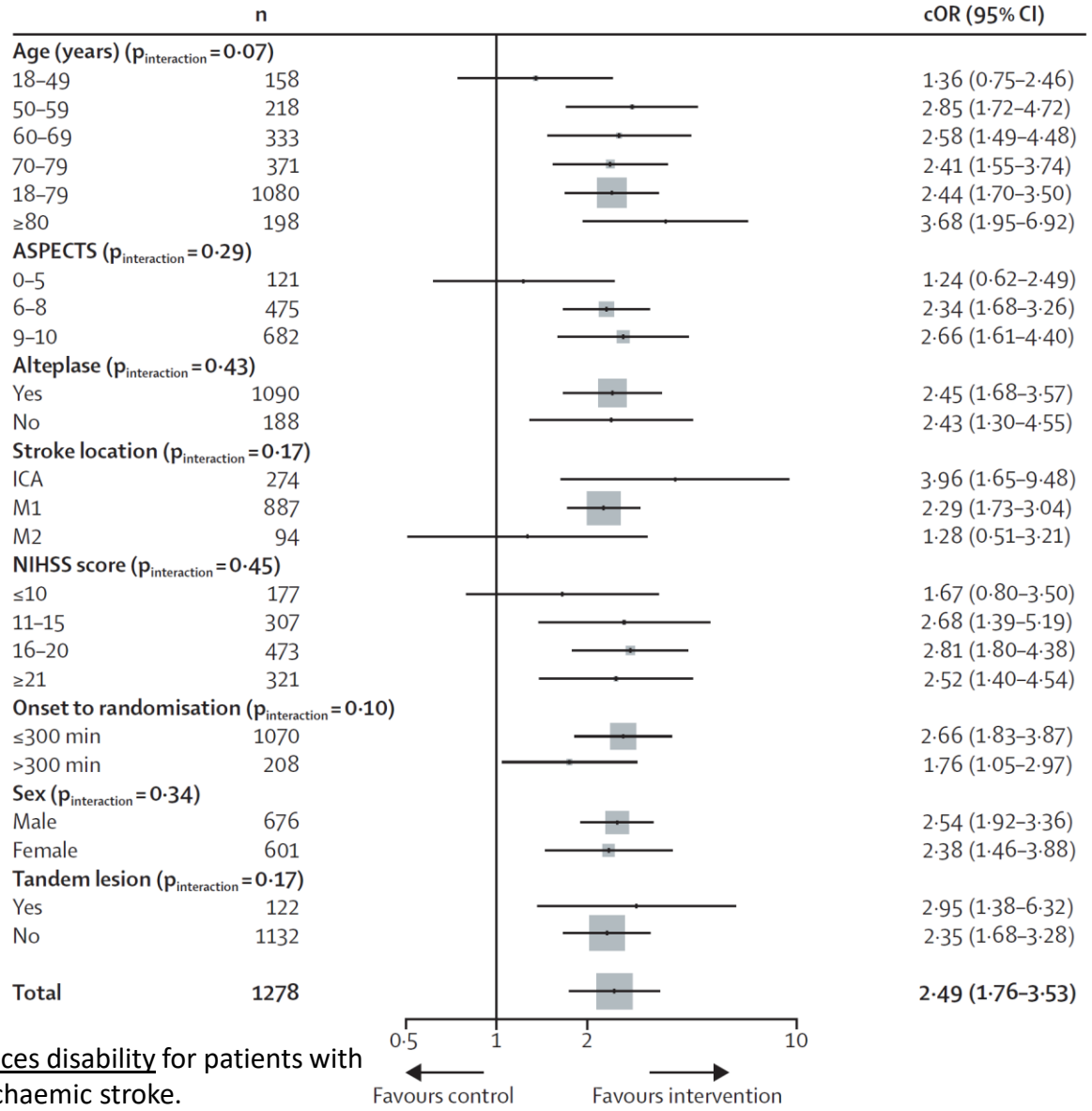
ΣΥΣΤΑΣΕΙΣ ΓΙΑ ΤΗΝ ΜΗΧΑΝΙΚΗ ΘΡΟΜΒΕΚΤΟΜΗ ΣΕ ΑΣΘΕΝΕΙΣ ΜΕ ΟΞΥ ΙΣΧΑΙΜΙΚΟ ΑΓΓΕΙΑΚΟ ΕΓΚΕΦΑΛΙΚΟ ΕΠΕΙΣΟΔΙΟ.

Ένας κλινικός οδηγός από τον Ελληνικό Οργανισμό Εγκεφαλικών.

1. Σε ασθενείς με σημαντικά νευρολογικά συμπτώματα λόγω ισχαιμικού αγγειακού εγκεφαλικού επεισοδίου (ΑΕΕ) με απόφραξη μεγάλου κλάδου της πρόσθιας ενδοκράνιας κυκλοφορίας, συνιστάται ενδαγγειακή θεραπεία με μηχανική θρομβεκτομή έως 6 ώρες μετά την έναρξη των συμπτωμάτων (1Α). Η παρουσία συνυπάρχουσας σύστοιχης εξωκράνιας καρωτιδικής νόσου δεν αποτελεί αντένδειξη (2Β). Μετά την πάροδο των 6 ωρών συνιστάται θρομβεκτομή σε επιλεγμένους ασθενείς (1Α). Επί απουσίας αντενδείξεων, οι ασθενείς θα πρέπει να λαμβάνουν προηγουμένως ενδοφλέβια θρομβόλυση με αλτεπλάση εφόσον αυτή δύναται να χορηγηθεί εντός 4.5 ωρών από την έναρξη της συμπτωματολογίας (1Α).

HEART VESSELS & BRAIN | ΑΠΡΙΛΙΟΣ - ΙΟΥΝΙΟΣ 2017

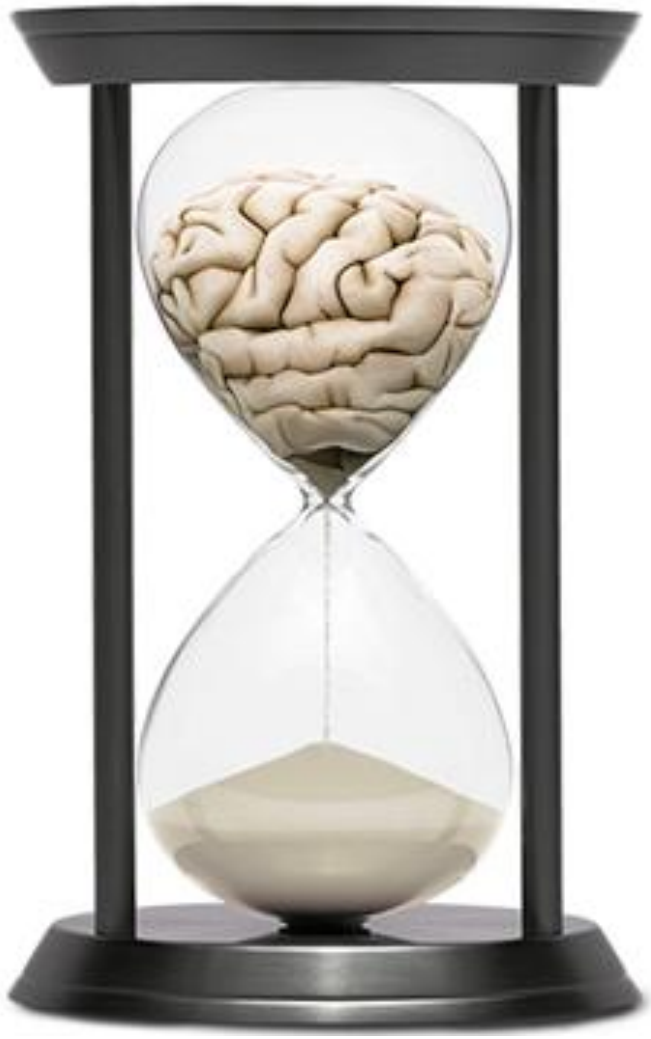
- MR CLEAN
- ESCAPE
- REVASCAT
- SWIFT PRIME
- EXTEND IA



In conclusion, **endovascular thrombectomy** reduces disability for patients with large vessel anterior circulation ischaemic stroke.
Benefits are seen across a wide range of age and initial stroke severity and apply to patients irrespective of eligibility for **intravenous alteplase**.

Lancet 2016; 387: 1723–31

« Time is brain »



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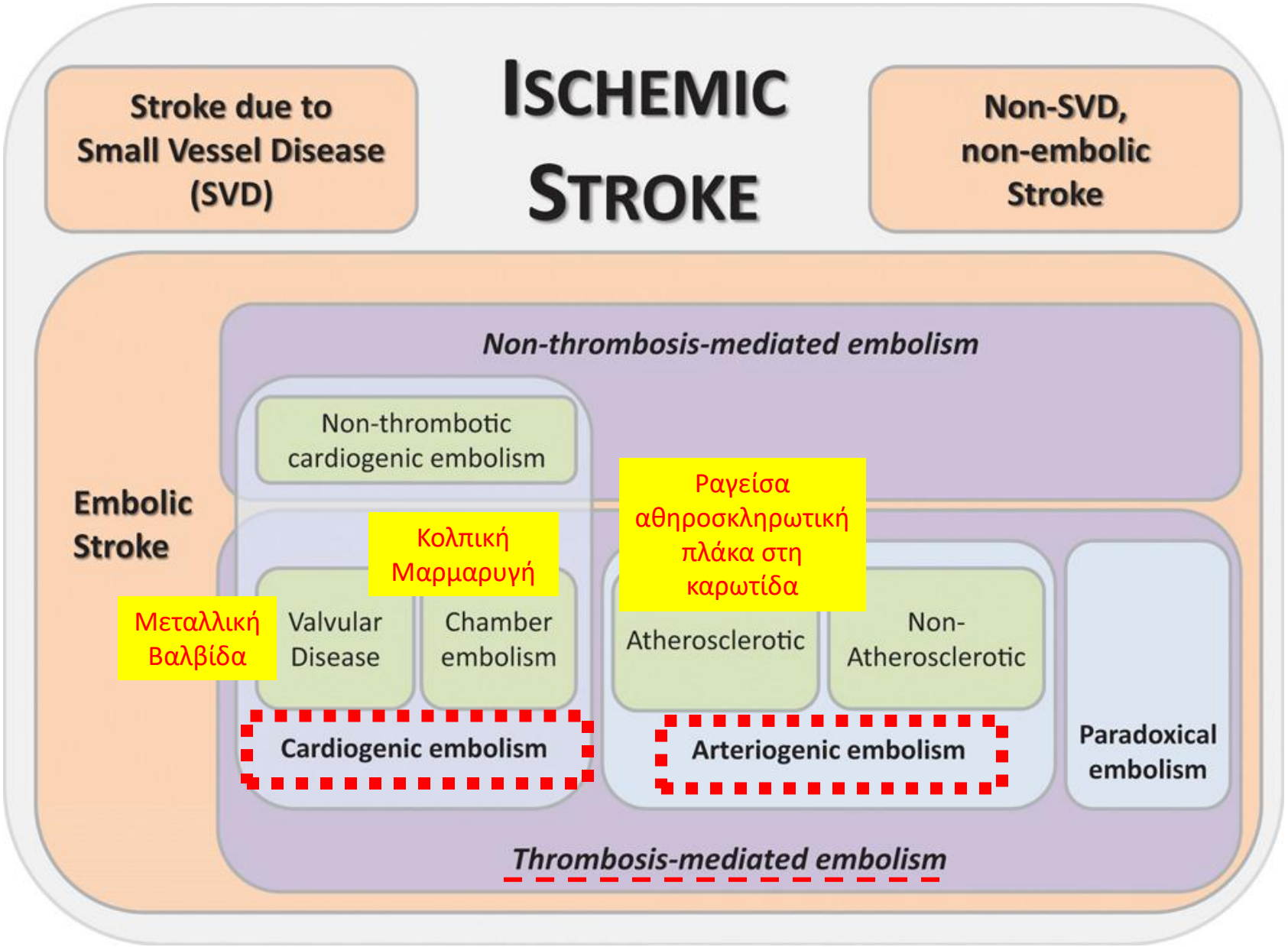
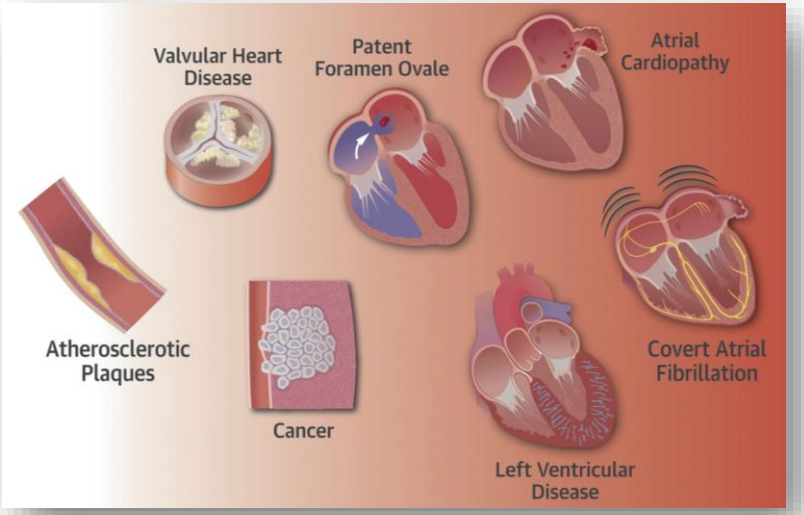
Μεταδιδακτορικός Ερευνητής Ιατρικής Σχολής Α.Π.Θ.
Stroke Research Fellow, HUS Helsinki University Hospital and University of Helsinki, Finland

5 Μαρτίου 2026

Ischemic stroke is an etiologically heterogeneous syndrome



Ntaios G, Hart RG. Embolic stroke. Circulation. 2017;136:2403e2405.



Ntaios G. Embolic Stroke of Undetermined Source: JACC Review Topic of the Week. J Am Coll Cardiol. 2020 Jan 28;75(3):333-340.

In order to optimize the ***secondary prevention strategy*** in a patient with ischemic stroke, it is rational to identify the underlying etiologic pathology.

VOLUME 8 | SUPPLEMENT 1 | 2017

ISSN 2459-380X

Hellenic Journal of Atherosclerosis

ΚΛΙΝΙΚΟΣ ΟΔΗΓΟΣ

Συστάσεις για την αντιθρομβωτική αγωγή σε ασθενείς με ισχαιμικό εγκεφαλικό επεισόδιο

Γ. Ντάιος, Γ. Ανδρικόπουλος, Ε. Αρναούτογλου, Ε. Βαβουρανάκης, Γ. Γεροτζιάφας, Ε. Κορομπόκη, Μ. Ματσάγκας, Χ. Μηλιώνης, Β. Παπαβασιλείου, Α. Πλωμαρίτογλου, Δ. Ρίχτερ, Σ. Σουρμελής, Κ. Σπέγγος, Κ. Τάκης, Κ. Τζιόμαλος, Α. Τσελέπης, Α. Ι. Χατζητόλιος, Κ. Βέμμος

Ένας κλινικός οδηγός από τον Ελληνικό Οργανισμό Εγκεφαλικών και το Ινστιτούτο Μελέτης και Εκπαίδευσης στη Θρόμβωση και την Αντιθρομβωτική Αγωγή

Ελληνικός Οργανισμός Εγκεφαλικών

ΑΘΗΝΑ, 2017

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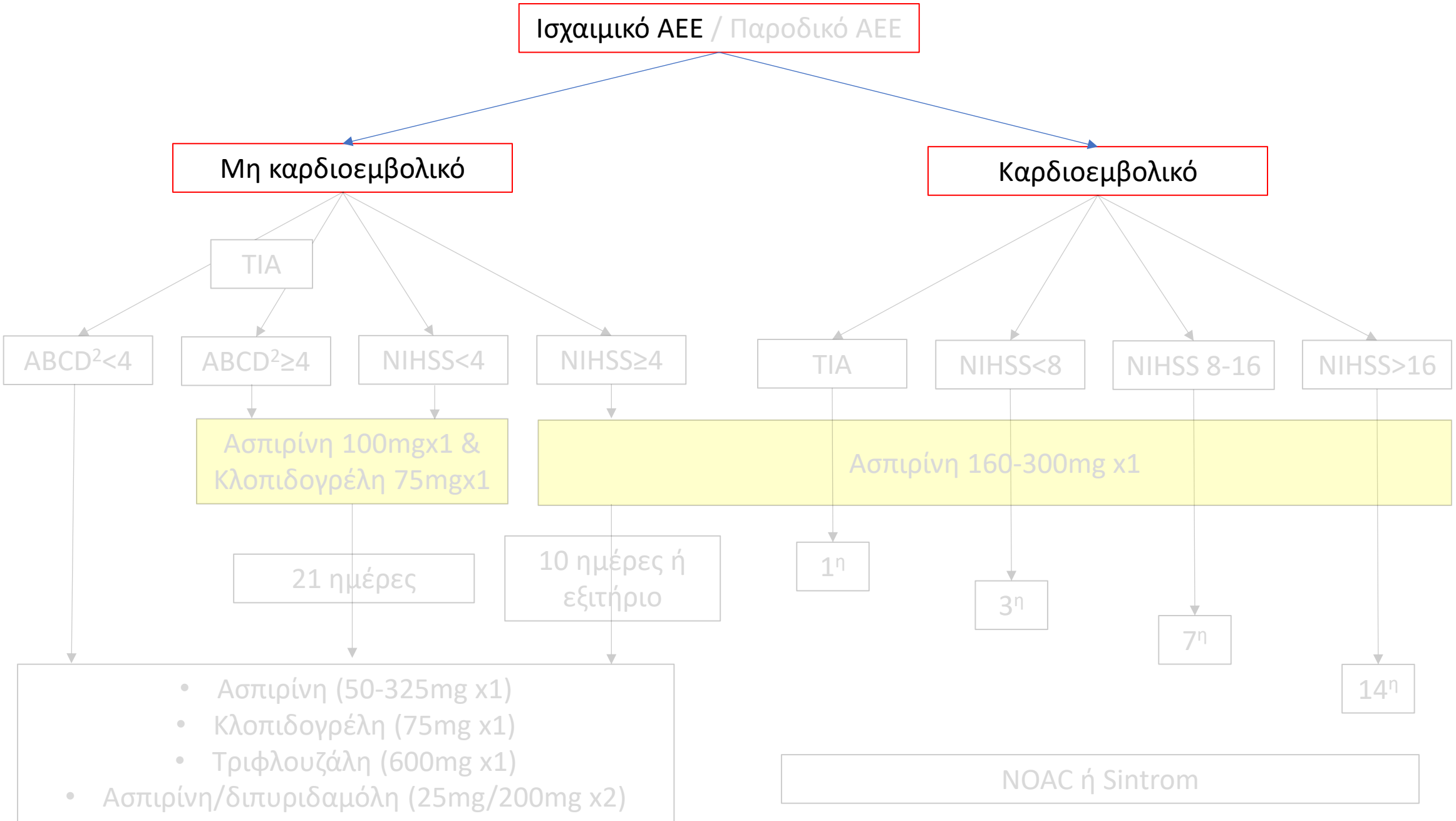
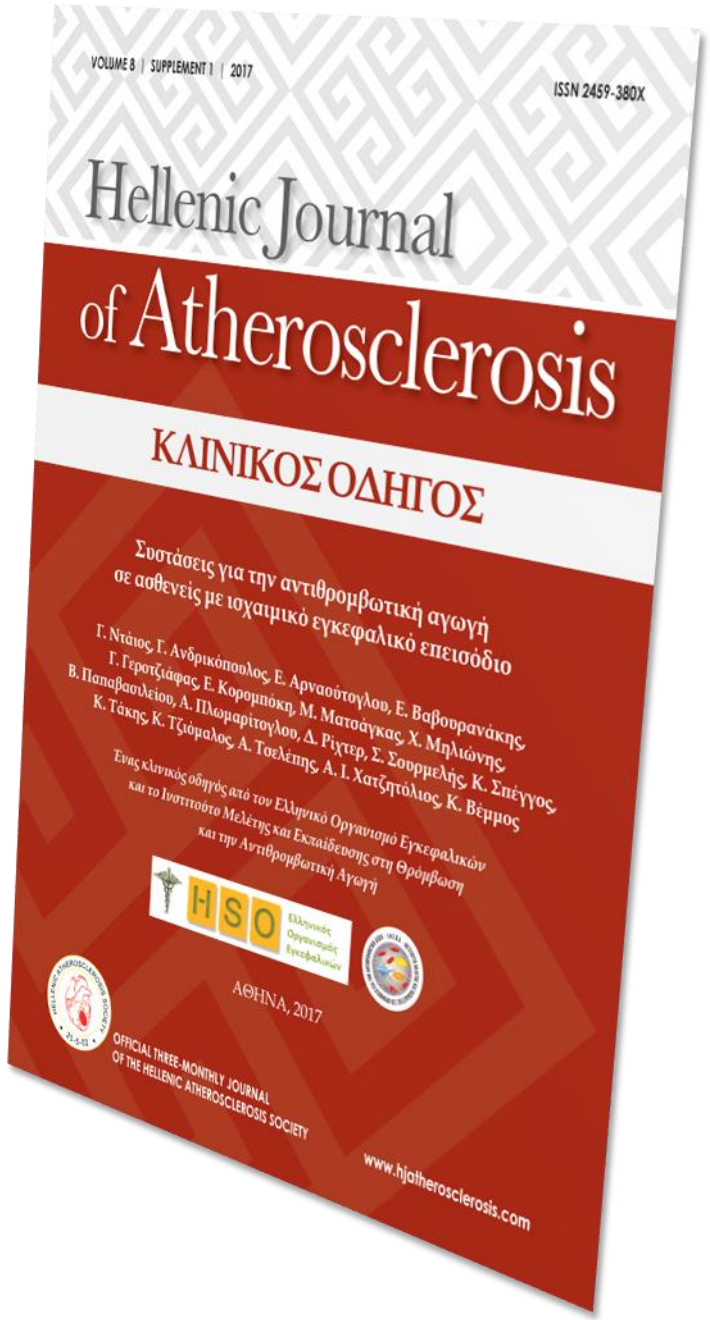

Ισχαιμικό ΑΕΕ / Παροδικό ΑΕΕ
Μη καρδιοεμβολικό
Καρδιοεμβολικό
TIA
ABCD²<4
ABCD²≥4
NIHSS<4
NIHSS≥4
TIA
NIHSS<8
NIHSS 8-16
NIHSS>16
Ασπιρίνη 100mgx1 & Κλοπιδογρέλη 75mgx1
Ασπιρίνη 160-300mg x1
21 ημέρες
10 ημέρες ή εξιτήριο
1η
3η
7η
14η
• Ασπιρίνη (50-325mg x1)
• Κλοπιδογρέλη (75mg x1)
• Τριφλουζάλη (600mg x1)
• Ασπιρίνη/διπυριδαμόλη (25mg/200mg x2)
NOAC ή Sintrom

1. Ασθενείς με ισχαιμικό (εγκατεστημένο ή παροδικό) εγκεφαλικό επεισόδιο χωρίς κολπική μαρμαρυγή ή μεταλλική βαλβίδα ή άλλη ένδειξη για αντιπηκτική αγωγή πρέπει να λαμβάνουν αντιαιμοπεταλιακή αγωγή (1A).



Cochrane Library

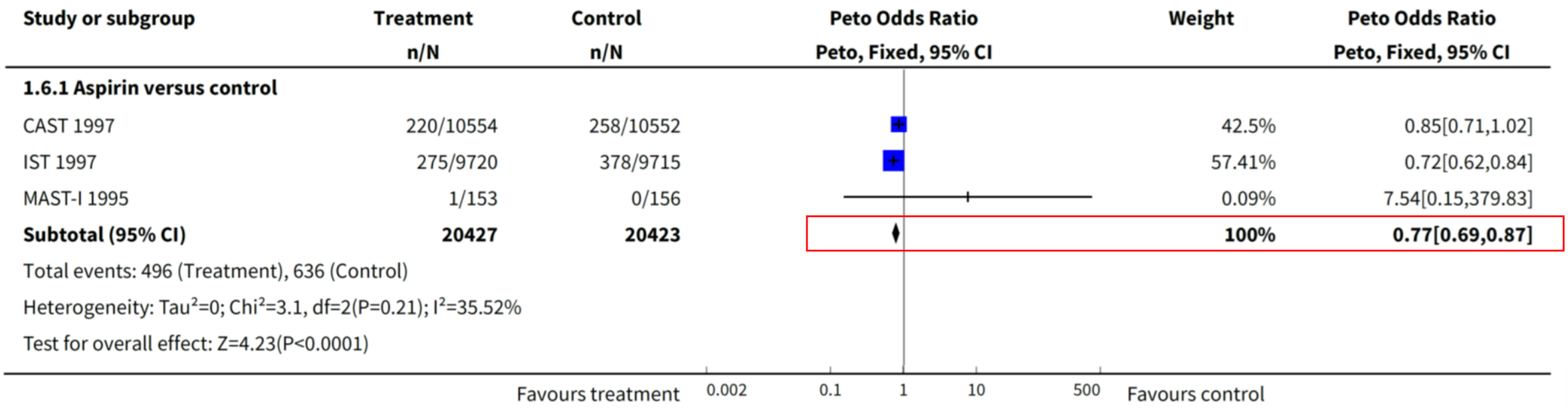
Cochrane Database of Systematic Reviews

Oral antiplatelet therapy for acute ischaemic stroke (Review)

Sandercock PAG, Counsell C, Tseng MC, Cecconi E

Study	Participants	Stroke	Intervention	Age	FU
CAST Chinese Acute Stroke Trial	21,106	<48h	aspirin 160mg vs placebo	28% >70 years	4 weeks or discharge
IST International Stroke Trial	19,435	<48h	sc heparin (5000 IU or 12 500 IU 12 hourly), aspirin 300 mg, both, or neither	61% >70 years	14 days or discharge
MAST-I Multicentre Acute Stroke Trial-Italy	309	<6h	aspirin 300 mg oral (or iv/rectal) 24 hourly vs no treatment	46% 61-75 years	10 days

Analysis 1.6. Comparison 1 Antiplatelet drug versus control in acute presumed ischaemic stroke, Outcome 6 Recurrent ischaemic/unknown stroke during treatment period.



Antiplatelet therapy with **aspirin 160 mg to 300 mg** daily, given orally (or by nasogastric tube or per rectum in people who cannot swallow) and started **within 48 hours** of onset of presumed ischaemic stroke, reduced the risk of early recurrent ischaemic stroke without a major risk of early haemorrhagic complications; long-term outcomes were improved.

Sandercock PA, Counsell C, Tseng MC, Cecconi E. Oral antiplatelet therapy for acute ischaemic stroke. Cochrane Database Syst Rev. 2014:CD000029

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association

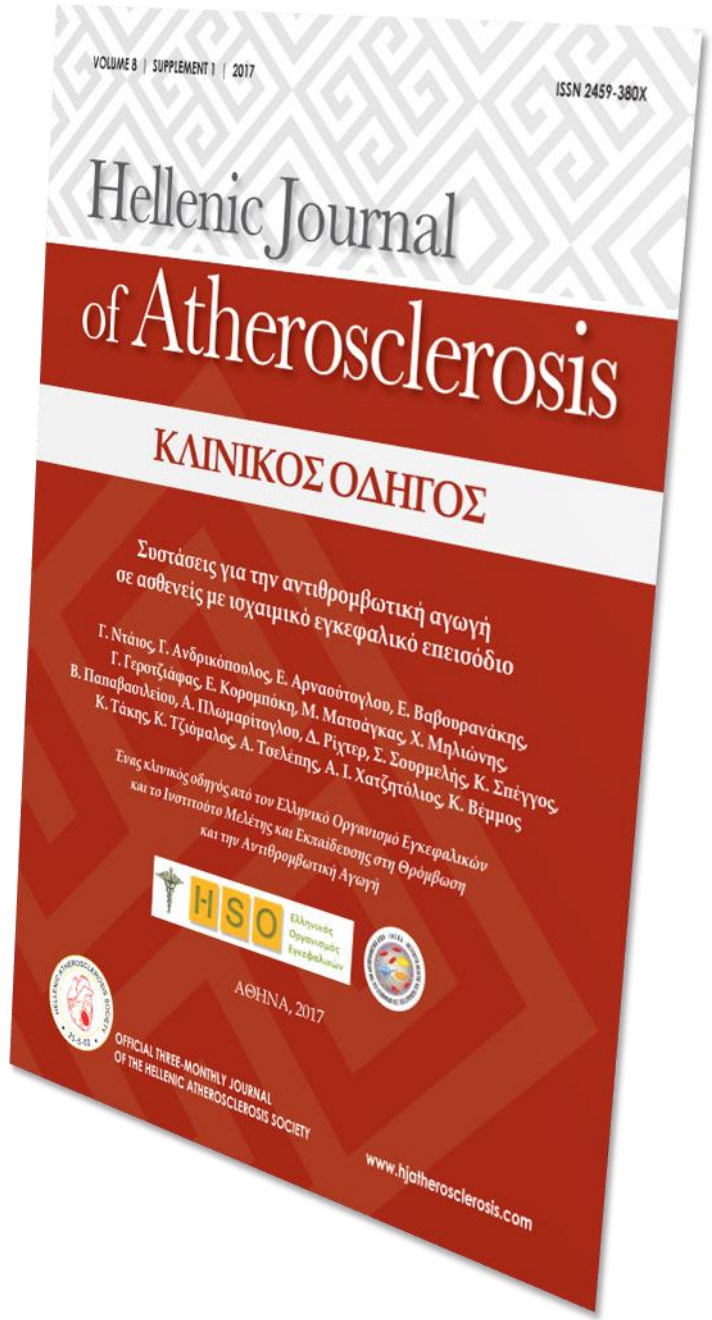
COR	LOE	Recommendations
General principles for early antiplatelet therapy		
1	A	1. In patients with AIS, administration of aspirin is recommended within 48 hours after stroke onset to reduce risk of death and dependency.[1–3]

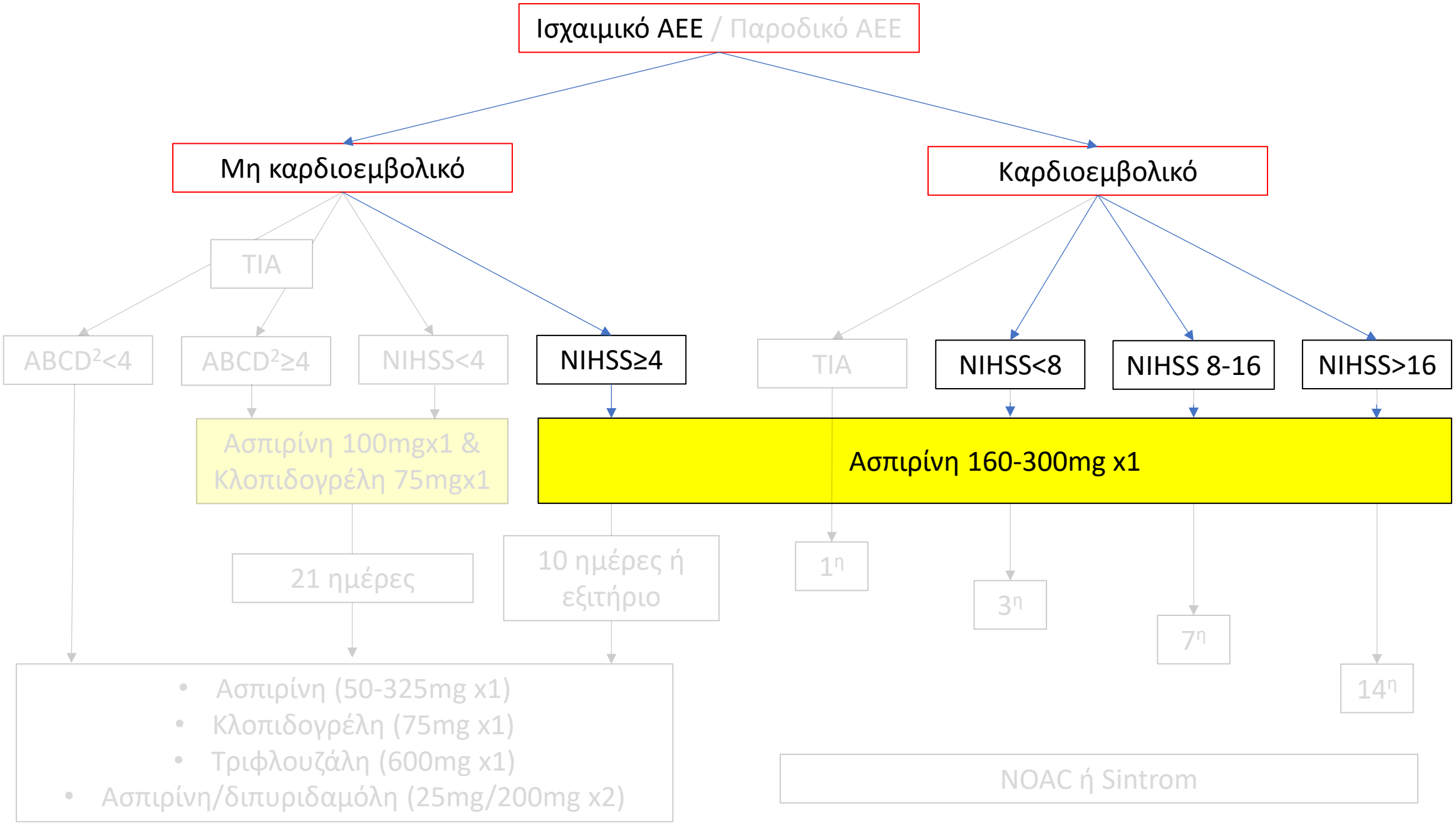
Stroke. 2026 Jan 26. Epub ahead of print.

4. Ασθενείς με οξύ ισχαιμικό εγκεφαλικό επεισόδιο πρέπει να λαμβάνουν ασπιρίνη (325 mg) αμέσως μόλις αποκλειστεί η αιμορραγία εγκεφάλου (1Α). Σε ασθενείς που έχουν υποβληθεί σε ενδοφλέβια θρομβόλυση, η αντιαιμοπεταλιακή αγωγή πρέπει να καθυστερεί 24 ώρες και να ξεκινά αφού αποκλειστεί η αιμορραγία εγκεφάλου (1Γ).

9. Σε ασθενείς με οξύ ισχαιμικό εγκεφαλικό επεισόδιο και ένδειξη αντιπηκτικής αγωγής, η έναρξη της αντιπηκτικής αγωγής πρέπει να καθυστερεί για κάποιο χρονικό διάστημα ώστε να μειωθεί ο κίνδυνος αιμορραγικής μετατροπής του εγκεφαλικού εμφράκτου ...

Στο μεσοδιάστημα ο ασθενής θα πρέπει να λαμβάνει ασπιρίνη.




Ισχαιμικό ΑΕΕ / Παροδικό ΑΕΕ
Μη καρδιοεμβολικό
Καρδιοεμβολικό
TIA
ABCD²<4
ABCD²≥4
NIHSS<4
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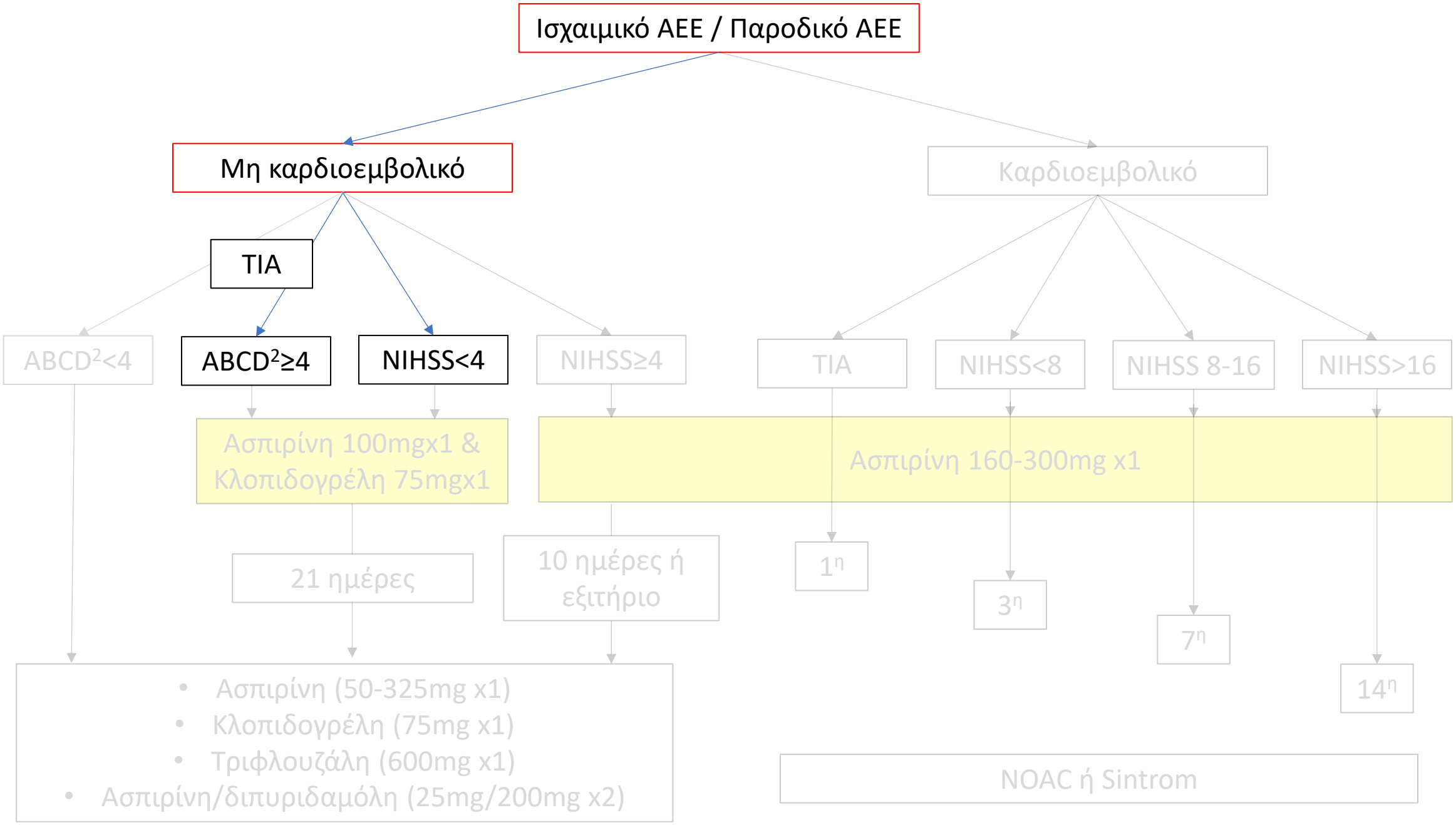
• Ασπιρίνη/διπυριδαμόλη (25mg/200mg x2)
NOAC ή Sintrom


Ισχαιμικό ΑΕΕ / Παροδικό ΑΕΕ
Μη καρδιοεμβολικό
Καρδιοεμβολικό
TIA
ABCD²<4
ABCD²≥4
NIHSS<4
NIHSS≥4
TIA
NIHSS<8
NIHSS 8-16
NIHSS>16
Ασπιρίνη 100mgx1 & Κλοπιδογρέλη 75mgx1
Ασπιρίνη 160-300mg x1
21 ημέρες
10 ημέρες ή εξιτήριο
1η
3η
7η
14η
Ασπιρίνη (50-325mg x1)
Κλοπιδογρέλη (75mg x1)
Τριφλουζάλη (600mg x1)
Ασπιρίνη/διπυριδαμόλη (25mg/200mg x2)
NOAC ή Sintrom

National Institutes of Health Stroke Scale (NIHSS)

	ΒΑΘΜΟΙ
1. Επίπεδο συνείδησης	
1Α) Γενικό επίπεδο συνείδησης	
• Εγρήγορση με έντονες ανταποκρίσεις.	0
• Απουσία εγρήγορσης – απαιτούνται μικρά ερεθίσματα για να επιτευχθεί ανταπόκριση, να δοθούν απαντήσεις ή να εκτελεσθούν εντολές.	1
• Βυθιότητα – Απαιτούνται επανειλημμένα ερεθίσματα για να μπορέσει ο ασθενής να παρακολουθήσει τον εξεταστή ή πολύ έντονα ακόμη και επώδυνα ερεθίσματα για να προκληθεί κινητική αντίδραση	2
• Κώμα – Μόνο αντανακλαστικές αντιδράσεις (κινητικές ή του αυτόνομου νευρικού) ή πλήρης απουσία οποιασδήποτε αντίδρασης	3
1Β) Ερώτηση ηλικίας και τρέχοντος μήνα	
• Σωστές απαντήσεις και στις δύο ερωτήσεις	0
• Μια μόνο σωστή απάντηση	1
• Καμία σωστή απάντηση	2
1Γ) Άνοιγμα και κλείσιμο βλεφάρων και σχηματισμός γροθιάς με το μη παρετικό άκρο	
• Σωστή εκτέλεση και των δύο δοκιμασιών	0
• Σωστή εκτέλεση μιας μόνο δοκιμασίας	1
• Καμία δοκιμασία δεν εκτελείται σωστά	2
2. Κινήσεις οφθαλμών (οριζόντιο επίπεδο)	
• Φυσιολογικές	0
• Μερική πάρεση – ανώμαλη κίνηση οφθαλμού τουλάχιστον σε έναν οφθαλμό	1
• Συζυγής απόκλιση οφθαλμών ή παράλυση που δεν διορθώνονται κατά την προσπάθεια ελέγχου του οφθαλμοκινητικού αντανακλαστικού (oculocephalic maneuver)	2
3. Οπτικά πεδία	
• Καμία απώλεια όρασης	0
• Μερική ημιανοψία	1
• Πλήρης ημιανοψία	2
• Αμφοτερόπλευρη ημιανοψία (τύφλωση που συμπεριλαμβάνει την φλοιϊκής αιτιολογίας τύφλωση)	3
4. Πάρεση μυών προσώπου	
• Φυσιολογικές συμμετρικές κινήσεις προσώπου	0
• Ήπια έκπτωση μυϊκής ισχύος (αποπλάτυνση της ρινοχειλικής αύλακας, ασυμμετρία στο χαμόγελο)	1
• Μερική πάρεση (πλήρης ή σχεδόν πλήρης παράλυση μόνο στο κατώτερο τμήμα του προσώπου)	2
• Πλήρης έτερο- ή αμφοτερό-πλευρη πάρεση (ανώτερο και κατώτερο τμήμα του προσώπου)	3

	Δ	Α
5. Μυϊκή ισχύς άνω άκρων (κάθε άνω άκρο εξετάζεται για 10 δευτερόλεπτα και βαθμολογείται χωριστά)		
• Το άκρο διατηρείται στην αρχική θέση εξέτασης	0	0
• Προοδευτική πτώση άνω άκρου, χωρίς να ακουμπήσει στο σώμα ή το κρεβάτι εντός των 10 δευτερολέπτων	1	1
• Προοδευτική πτώση άνω άκρου, που ακουμπά στο σώμα ή το κρεβάτι πριν την παρέλευση των 10 δευτερολέπτων	2	2
• Καμία ένδειξη προσπάθειας διατήρησης στη θέση εξέτασης (καμία προσπάθεια αντιβαρικής κίνησης)	3	3
• Δεν παρατηρείται καμία ένδειξη μυϊκής ισχύος στο άκρο (π.χ. κίνηση δακτύλων)	4	4
6. Μυϊκή ισχύς κάτω άκρων (κάθε κάτω άκρο εξετάζεται για 5 δευτερόλεπτα και βαθμολογείται χωριστά)	Δ	Α
• Το άκρο διατηρείται στην αρχική θέση εξέτασης	0	0
• Προοδευτική πτώση κάτω άκρου, χωρίς να ακουμπήσει στο σώμα ή το κρεβάτι εντός των 5 δευτερολέπτων	1	1
• Προοδευτική πτώση κάτω άκρου, που ακουμπά στο σώμα ή το κρεβάτι πριν την παρέλευση των 5 δευτερολέπτων	2	2
• Καμία ένδειξη προσπάθειας διατήρησης στη θέση εξέτασης (καμία προσπάθεια αντιβαρικής κίνησης)	3	3
• Δεν παρατηρείται καμία ένδειξη μυϊκής ισχύος στο άκρο (π.χ. κίνηση δακτύλων)	4	4

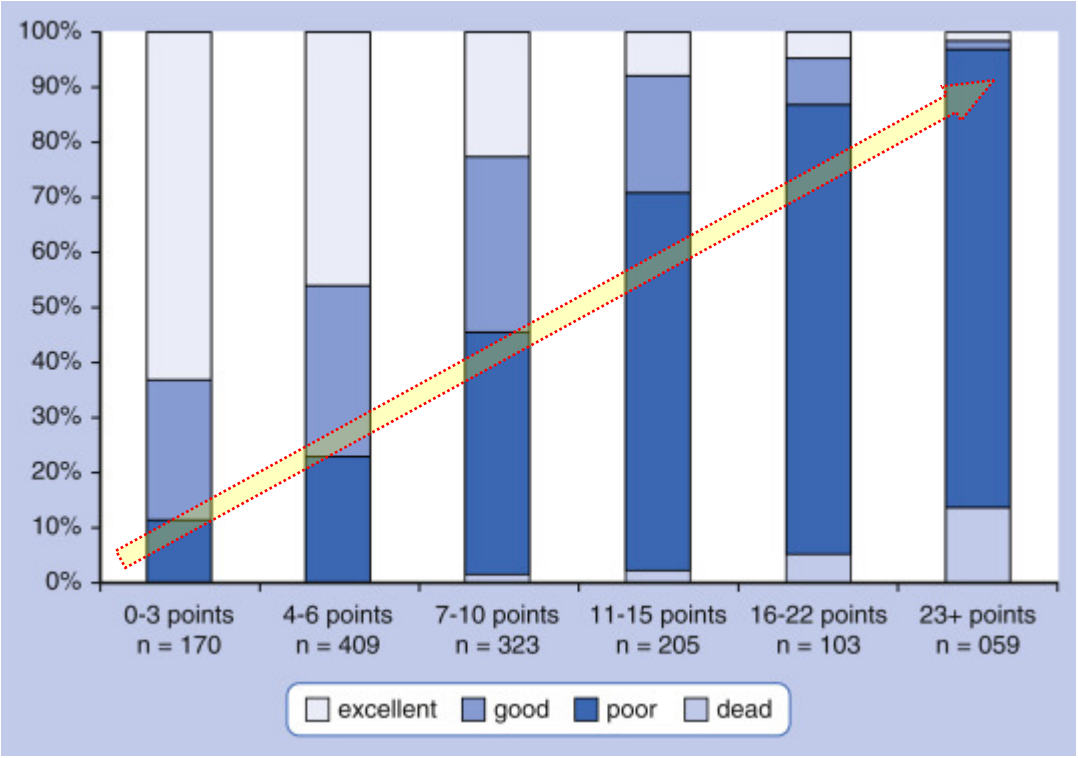
7. Αταξία άκρου/ων (δοκιμασίες δείκτη-ρινός) και πτέρνας-κνήμης)	
• Απουσία αταξίας	0
• Αταξία σε ένα μόνο άκρο	1
• Αταξία σε δύο άκρα	2
8. Αισθητικότητα	
• Φυσιολογική	0
• Ήπια ή ενδιάμεσης βαρύτητας απώλεια αισθητικότητας (ο ασθενής όμως μπορεί ακόμη να αισθανθεί ότι τον αγγίζουμε στη διάρκεια της δοκιμασίας)	1
• Σοβαρή ή πλήρης απώλεια της αισθητικότητας (ο ασθενής δεν έχει την αίσθηση του αγγίγματος, του νυγμού ή του επώδυνου ερεθίσματος που προκαλεί ο εξεταστής)	2
9. Λόγος/Αφασία (εκτίμηση με τη χρήση των εικόνων της αυθεντικής κλίμακας)	
• Φυσιολογικός λόγος, καμία ένδειξη αφασίας	0
• Ήπια ή ενδιάμεσης βαρύτητας αφασία	1
• Σοβαρή αφασία	2
• Πλήρης απουσία λόγου, σφαιρική αφασία	3
10. Δυσαρθρία	
• Απουσία δυσαρθρίας	0
• Ήπια ή ενδιάμεσης βαρύτητας δυσαρθρία	1
• Σοβαρή δυσαρθρία (ακατάληπτος λόγος που δεν μπορεί να αποδοθεί σε αφασία) ή αναρθρία	2
11. Αναγνώριση ταυτόχρονων αμφοτερόπλευρων ερεθισμάτων	
• Καμία διαταραχή	0
• Αδυναμία αναγνώρισης ενός εκ των οπτικών, απτικών και οπτικοχωρικών ακουστικών ερεθισμάτων	1
• Αδυναμία αναγνώρισης τουλάχιστον δύο διαφορετικών τύπων ερεθισμάτων. Ο ασθενής μπορεί να μην αναγνωρίζει κάποιο από τα άκρα του ή να προσανατολίζεται μόνο προς μια μεριά του χώρου.	2

ΣΥΣΤΑΣΕΙΣ ΓΙΑ ΤΗΝ ΕΝΔΟΦΛΕΒΙΑ ΘΡΟΜΒΟΛΥΣΗ ΣΕ ΑΣΘΕΝΕΙΣ ΜΕ ΟΞΥ ΙΣΧΑΙΜΙΚΟ ΑΓΓΕΙΑΚΟ ΕΓΚΕΦΑΛΙΚΟ ΕΠΕΙΣΟΔΙΟ.

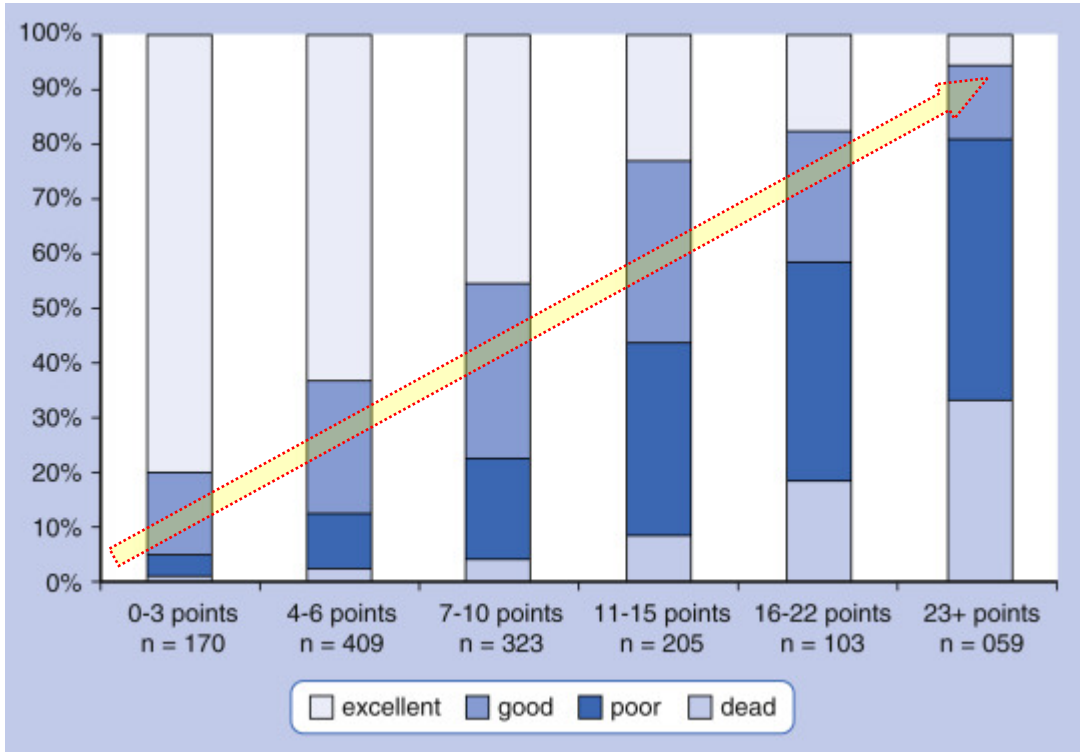
Ένας κλινικός οδηγός από τον Ελληνικό Οργανισμό Εγκεφαλικών

Συνιστάται η **εξάσκηση** αλλά και **διαδικτυακή εκπαίδευση** των επαγγελματιών υγείας που εμπλέκονται στην αντιμετώπιση ασθενών με αγγειακά εγκεφαλικά επεισόδια στην κλίμακα NIHSS πριν από τη χρήση του παραπάνω πίνακα.

National Institutes of Health Stroke Scale



Effect of baseline National Institutes of Health Stroke Scale score on outcome at **7 days**.



Effect of baseline National Institutes of Health Stroke Scale score on outcome at **3 months**.

Adams HP Jr., Davis PH, Leira EC, et al: Baseline NIH Stroke Scale score strongly predicts outcome after stroke: a report of the Trial of Org 10172 in Acute Stroke Treatment [TOAST], Neurology 53[1]:127, 1999

https://www.mdcalc.com/nih-stroke-scale-score-nihss

NIH Stroke Scale/Score (NIHSS)

Calculates the NIH Stroke Scale for quantifying stroke severity.

INSTRUCTIONS

The NIH Stroke Scale has many caveats buried within it. If your patient has prior known neurologic deficits e.g. prior weakness, hemi- or quadriplegia, blindness, etc. or is intubated, has a language barrier, etc., it becomes especially complicated. In those cases, consult the NIH Stroke Scale website. MDCalc's version is an attempt to clarify many of these confusing caveats, but cannot and should not be substituted for the official protocol.

Rules:

- Score what you see, not what you think.
- Score the first response, not the best response (except Item 9 - Best Language).
- Don't coach.

When to Use | Pearls/Pitfalls | Why Use

1A: Level of consciousness
May be assessed casually while taking history

Alert; keenly responsive	0
Arouses to minor stimulation	**+1**
Requires repeated stimulation to arouse	+2
Movements to pain	+2
Postures or unresponsive	+3

8 points

NIH Stroke Scale

Copy Results | Next Steps

About the Creator

Dr. Patrick D. Lyden

Are you Dr. Patrick D. Lyden?

Also from MDCalc...

Related Calcs

- tPA Contraindications
- Modified NIH Stroke Scale
- THRIVE Score

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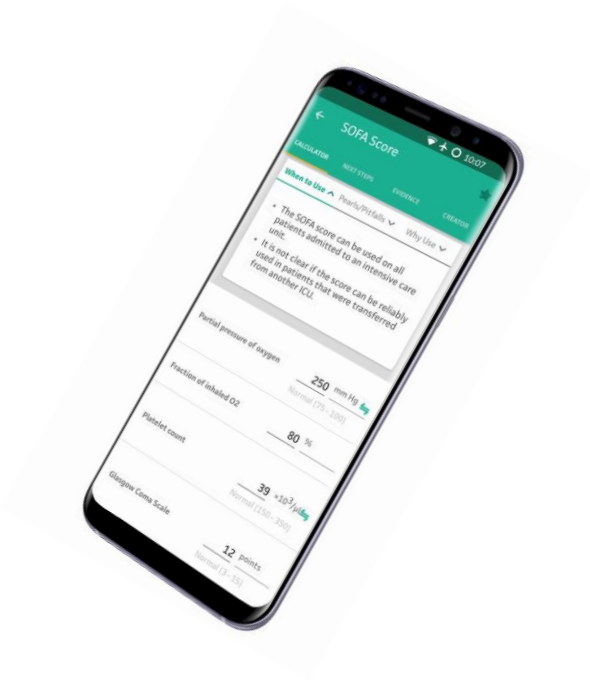
Partner Content

MDCalc Stroke CME
Get up to 10 hours of stroke CME

Calculated Decisions: NIHSS
Emergency Medicine Practice

Content Contributors

- Daniel Runde, MD



ABCD² Score for TIA

	ABCD2 items (score: 0–7)	Points
A	Age: ≥60 years	1
B	Blood pressure: ≥140/90mm Hg	1
C	Clinical features: -unilateral weakness or -speech impairment without weakness	 2 1
D	Duration of symptoms: ≥60 minutes or 10–59 minutes	 2 1
D	Diabetes: (on medication/insulin)	1

Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack

S Claiborne Johnston, Peter M Rothwell, Mai N Nguyen-Huynh, Matthew F Giles, Jacob S Elkins, Allan L Bernstein, Stephen Sidney

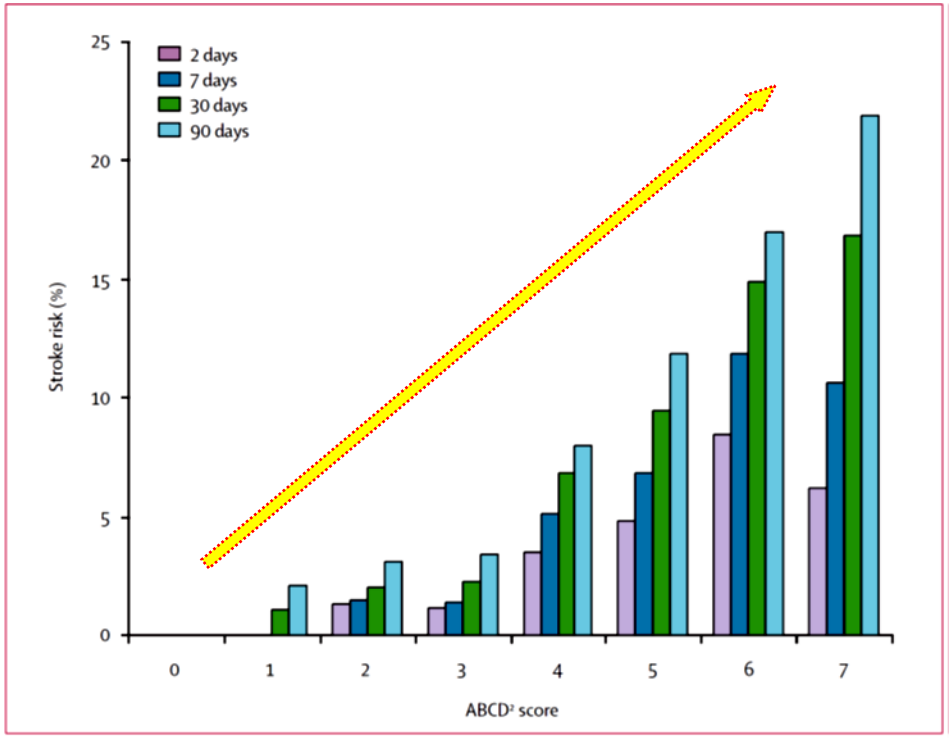


Figure: Short-term risk of stroke by ABCD² score in six groups combined (n=4799)

Lancet 2007; 369: 283–92

Higher ABCD² Score Predicts Patients Most Likely to Have True Transient Ischemic Attack

S. Andrew Josephson, MD; Stephen Sidney, MD, MPH; Trinh N. Pham, MA; Allan L. Bernstein, MD; S. Claiborne Johnston, MD, PhD

Conclusions

Among patients diagnosed by emergency department physicians with TIA, higher ABCD² score was associated with a greater likelihood that the diagnosis was confirmed on expert review. The predictive power of the ABCD² model is therefore partially explained by identification of those patients likely to have experienced a true TIA, an important aspect of the score when used **by non-neurologists**. However, higher ABCD² scores still remained predictive of 90-day stroke rate in the group of patients judged to have a true TIA by an expert neurologist.

Stroke 2008;49:3096-3098

https://www.mdcalc.com/abcd2-score-tia

ABCD² Score for TIA

Estimates the risk of stroke after a suspected transient ischemic attack (TIA).

When to Use | Pearls/Pitfalls | Why Use

Age ≥ 60 years	No 0	**Yes +1**
BP ≥ 140/90 mmHg Initial BP. Either SBP ≥ 140 or DBP ≥ 90.	No 0	**Yes +1**
Clinical features of the TIA	**Unilateral weakness**	**+2**
	Speech disturbance without weakness	+1
	Other symptoms	0
Duration of symptoms	<10 minutes	0
	10-59 minutes	**+1**
	≥ 60 minutes	+2
History of diabetes	No 0	**Yes +1**

6 points

Per the validation study, 6-7 points: High Risk
2-Day Stroke Risk: 8.1%
7-Day Stroke Risk: 11.7%
90-Day Stroke Risk: 17.8%

Copy Results | Next Steps

About the Creator

Dr. S. Claiborne Johnston

Are you Dr. S. Claiborne Johnston?

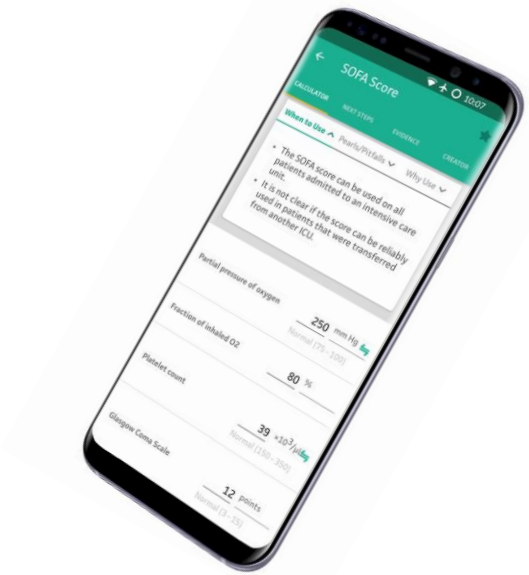
Also from MDCalc...

Related Calcs

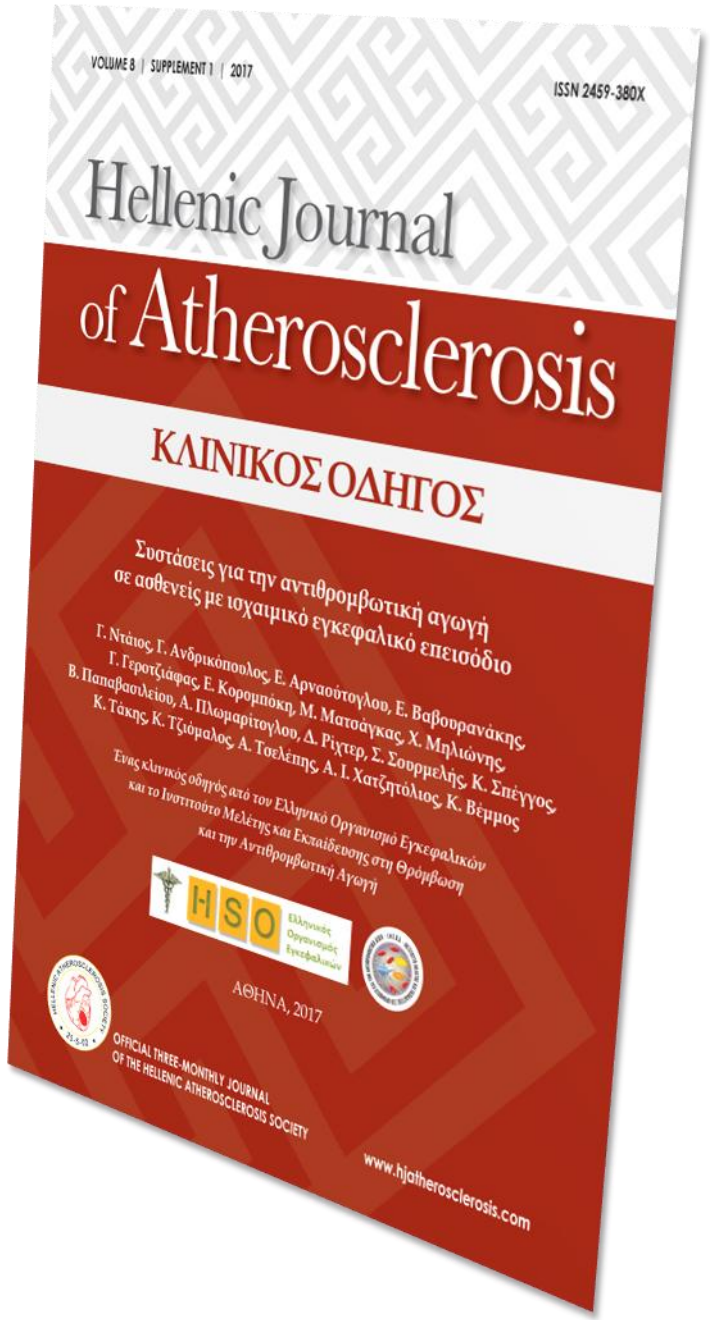
- CHADS₂ Score
- THRIVE Score
- Modified Rankin Score

Content Contributors

- Daniel Runde, MD



5. Ασθενείς με παροδικό εγκεφαλικό επεισόδιο υψηλού κινδύνου (ABCD2 ≥ 4) ή μικρής βαρύτητας εγκατεστημένο ισχαιμικό εγκεφαλικό επεισόδιο (NIHSS score κατά την εισαγωγή <4) χωρίς κολπική μαρμαρυγή ή μεταλλική βαλβίδα ή άλλη ένδειξη για αντιπηκτική αγωγή θα μπορούσαν να λάβουν διπλή αντιαιμοπεταλιακή αγωγή με ασπιρίνη και κλοπιδογρέλη για 21 ημέρες μετά το επεισόδιο (2Β). Στη συνέχεια πρέπει να συνεχίσουν να λαμβάνουν αντιαιμοπεταλιακή αγωγή όπως αυτή περιγράφεται στο εδάφιο 2.

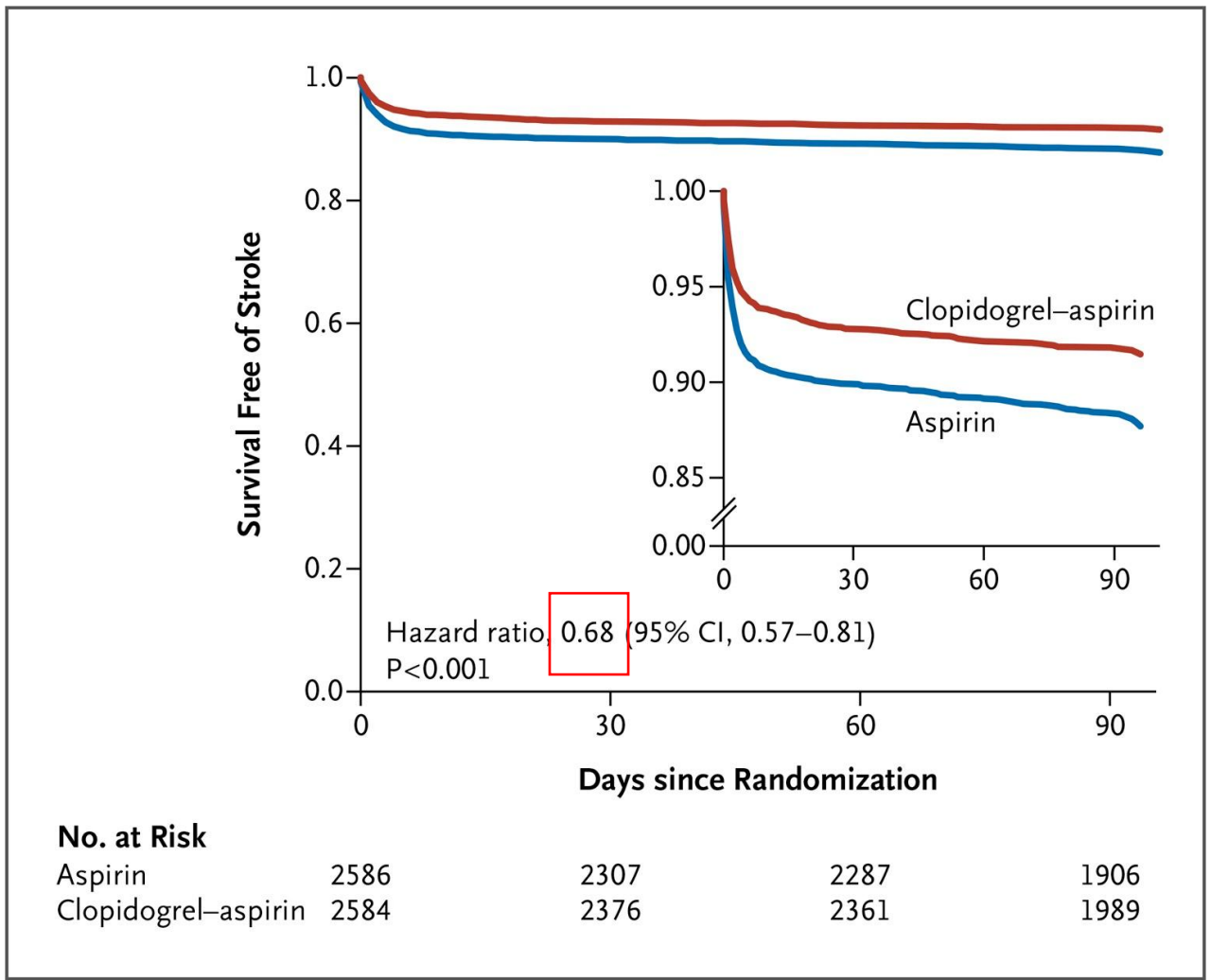


The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Clopidogrel with Aspirin in Acute Minor Stroke or Transient Ischemic Attack

- Randomized, double-blind, placebo-controlled trial conducted at 114 centers in China,
- **5,170** patients within 24 hours after the onset of minor ischemic stroke or high-risk TIA
- combination therapy with **clopidogrel and aspirin** (clopidogrel at an initial dose of 300 mg, followed by 75 mg per day for 90 days, plus aspirin at a dose of 75 mg per day for the first 21 days) or to **placebo plus aspirin** (75 mg per day for 90 days). All participants received open-label aspirin at a clinician-determined dose of 75 to 300 mg on day 1.
- The primary outcome was **stroke (ischemic or hemorrhagic)** during 90 days of follow-up in an intention-to-treat analysis.



CHANCE

Table 2. Efficacy and Safety Outcomes.

Outcome	Aspirin (N=2586) Patients with Event no.	Aspirin (N=2586) Event Rate %	Clopidogrel and Aspirin (N=2584) Patients with Event no.	Clopidogrel and Aspirin (N=2584) Event Rate %	Hazard Ratio (95% CI)	P Value
Primary outcome						
Stroke	303	11.7	212	8.2	0.68 (0.57–0.81)	<0.001
Secondary outcomes						
Stroke, myocardial infarction, or death from cardiovascular causes	307	11.9	216	8.4	0.69 (0.58–0.82)	<0.001
Ischemic stroke	295	11.4	204	7.9	0.67 (0.56–0.81)	<0.001
Hemorrhagic stroke	8	0.3	8	0.3	1.01 (0.38–2.70)	0.98
Myocardial infarction	2	0.1	3	0.1	1.44 (0.24–8.63)	0.69
Death from cardiovascular causes	5	0.2	6	0.2	1.16 (0.35–3.79)	0.81
Death from any cause	10	0.4	10	0.4	0.97 (0.40–2.33)	0.94
Transient ischemic attack	47	1.8	39	1.5	0.82 (0.53–1.26)	0.36
Safety outcomes						
Bleeding*						
Severe	4	0.2	4	0.2	0.94 (0.24–3.79)	0.94
Moderate	4	0.2	3	0.1	0.73 (0.16–3.26)	0.68
Mild	19	0.7	30	1.2	1.57 (0.88–2.79)	0.12
Any bleeding	41	1.6	60	2.3	1.41 (0.95–2.10)	0.09

Conclusions

Among patients with TIA or minor stroke who can be treated within 24 hours after the onset of symptoms, the **combination of clopidogrel and aspirin is superior** to aspirin alone for reducing the risk of stroke in the first 90 days and **does not increase the risk of hemorrhage**.

N ENGL J MED 369;1 NEJM.ORG JULY 4, 2013

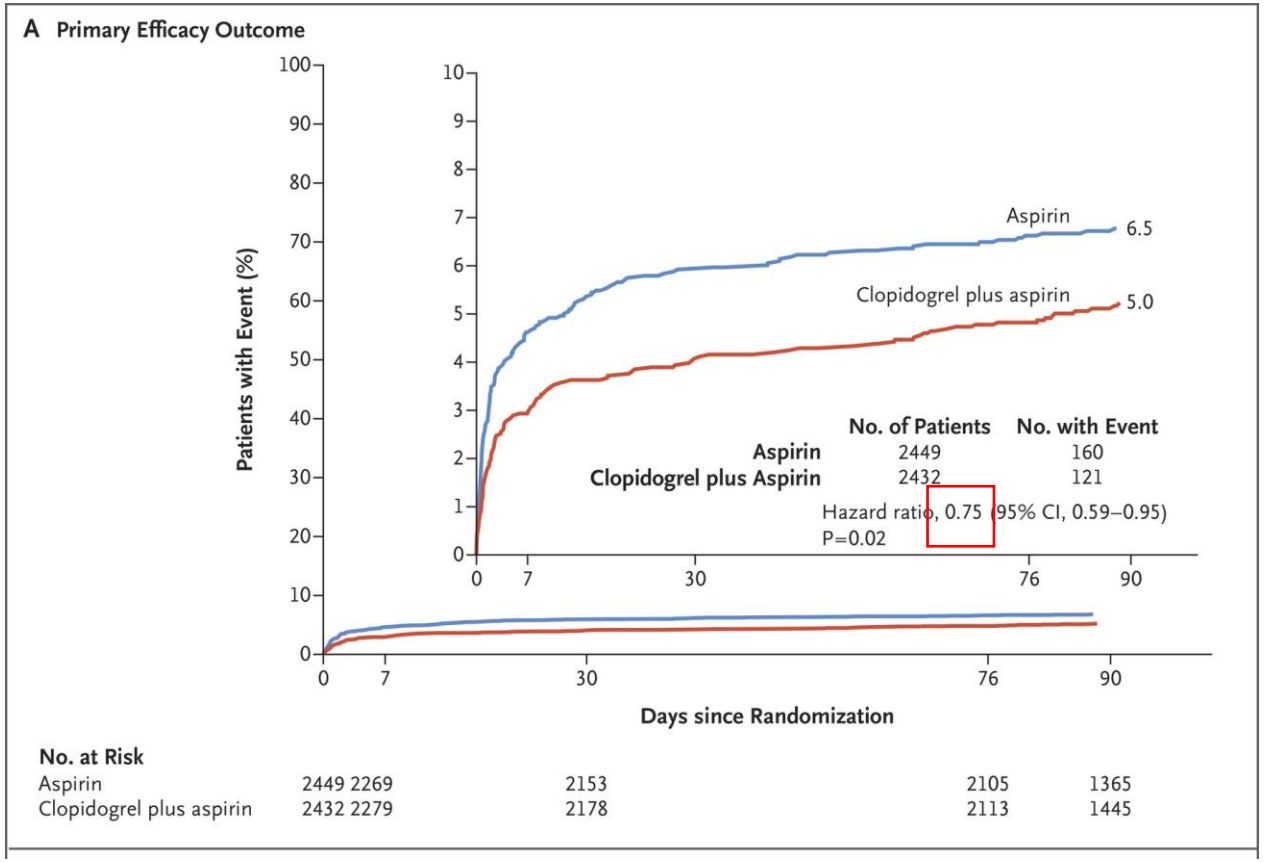
The NEW ENGLAND JOURNAL of MEDICINE

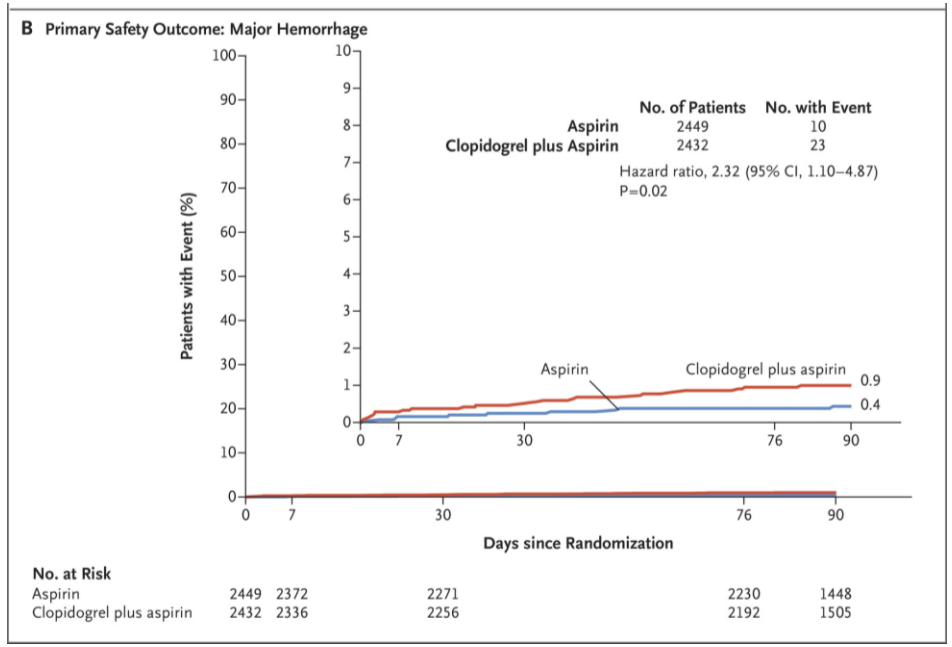
ESTABLISHED IN 1812 JULY 19, 2018 VOL. 379 NO. 3

Clopidogrel and Aspirin in Acute Ischemic Stroke and High-Risk TIA

POINT

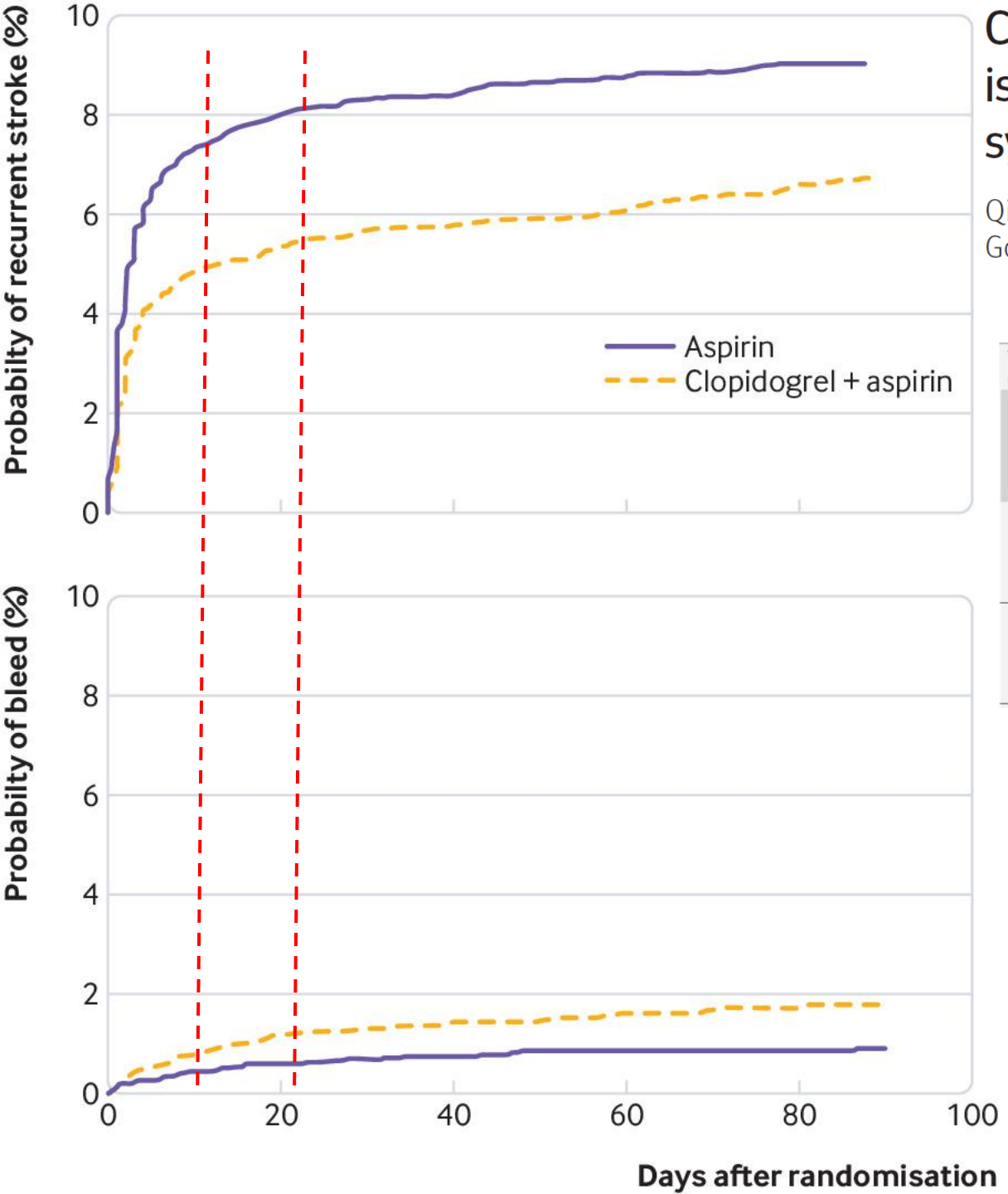
- Randomized, double-blind, placebo-controlled trial
- **4,881** patients (269 international sites) with minor ischemic stroke or high-risk TIA
- Clopidogrel at a loading dose of 600 mg on day 1, followed by **75 mg per day, plus aspirin** (at a dose of 50 to 325 mg per day) or the same range of doses of **aspirin alone**.
- Primary efficacy outcome in a time-to-event analysis was the risk of a composite of **major ischemic events**, which was defined as ischemic stroke, myocardial infarction, or death from an ischemic vascular event, at 90 days.





Conclusions

In patients with minor ischemic stroke or high-risk TIA, those who received a combination of clopidogrel and aspirin had a **lower risk of major ischemic events** but a **higher risk of major hemorrhage** at 90 days than those who received aspirin alone.



Clopidogrel plus aspirin versus aspirin alone for acute minor ischaemic stroke or high risk transient ischaemic attack: systematic review and meta-analysis

Qiukui Hao,[1,2] Malavika Tampi,[3] Martin O'Donnell,[4] Farid Foroutan,[2] Reed AC Siemieniuk,[2] Gordon Guyatt[2]

10,447 participants

BMJ 2018;364:k5108

Table 3 | GRADE evidence profile: Dual antiplatelet with clopidogrel and aspirin for 10-21 days versus 22-90 days after transient ischaemic attack (TIA) or minor stroke

Outcome; timeframe	Study results and measurements	Absolute effect estimates	
		Stop clopidogrel, continue aspirin	Continue clopidogrel and aspirin
Ischaemic stroke; 90 days	Odds ratio 1.47 (95% CI 0.84 to 2.56). Based on data from 4406 patients in one study. Follow-up 90 days	10/1000	14/1000
		Difference: 4 more per 1000 (95% CI 2 fewer to 11 more)	
Moderate or severe bleeding; 90 days	Odds ratio 2.20 (95% CI 0.83 to 5.78). Based on data from 4599 patients in one study. Follow-up 90 days	3/1000	6/1000
		Difference: 3 more per 1000 (95% CI 1 fewer to 7 more)	

Conclusion

Dual antiplatelet therapy with clopidogrel and aspirin given within 24 hours after high-risk TIA or minor ischaemic stroke reduces the risk of subsequent stroke by about 2%, with few serious adverse consequences.

Discontinuation of dual antiplatelet therapy as early as 10 days, and no later than 21 days, after initiation is likely to maximise its net benefit.

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association

Dual antiplatelet therapy for minor AIS and high-risk TIA		
1	A	12. In patients with minor (NIHSS score ≤3) noncardioembolic AIS or high-risk TIA (ABCD^2 score ≥4) who did not receive IVT, DAPT (aspirin and clopidogrel with loading dose of clopidogrel) should be initiated early (within 24 hours after symptom onset) and continued for 21 days, followed by single antiplatelet therapy (SAPT) to reduce the 90-day risk of recurrent ischemic stroke.[19–25]

Stroke. 2026 Jan 26. Epub ahead of print.

European Stroke Organisation expedited recommendation for the use of short-term dual antiplatelet therapy early after minor stroke and high-risk TIA

EUROPEAN STROKE JOURNAL

European Stroke Journal
0(0) 1–5

Recommendation 1

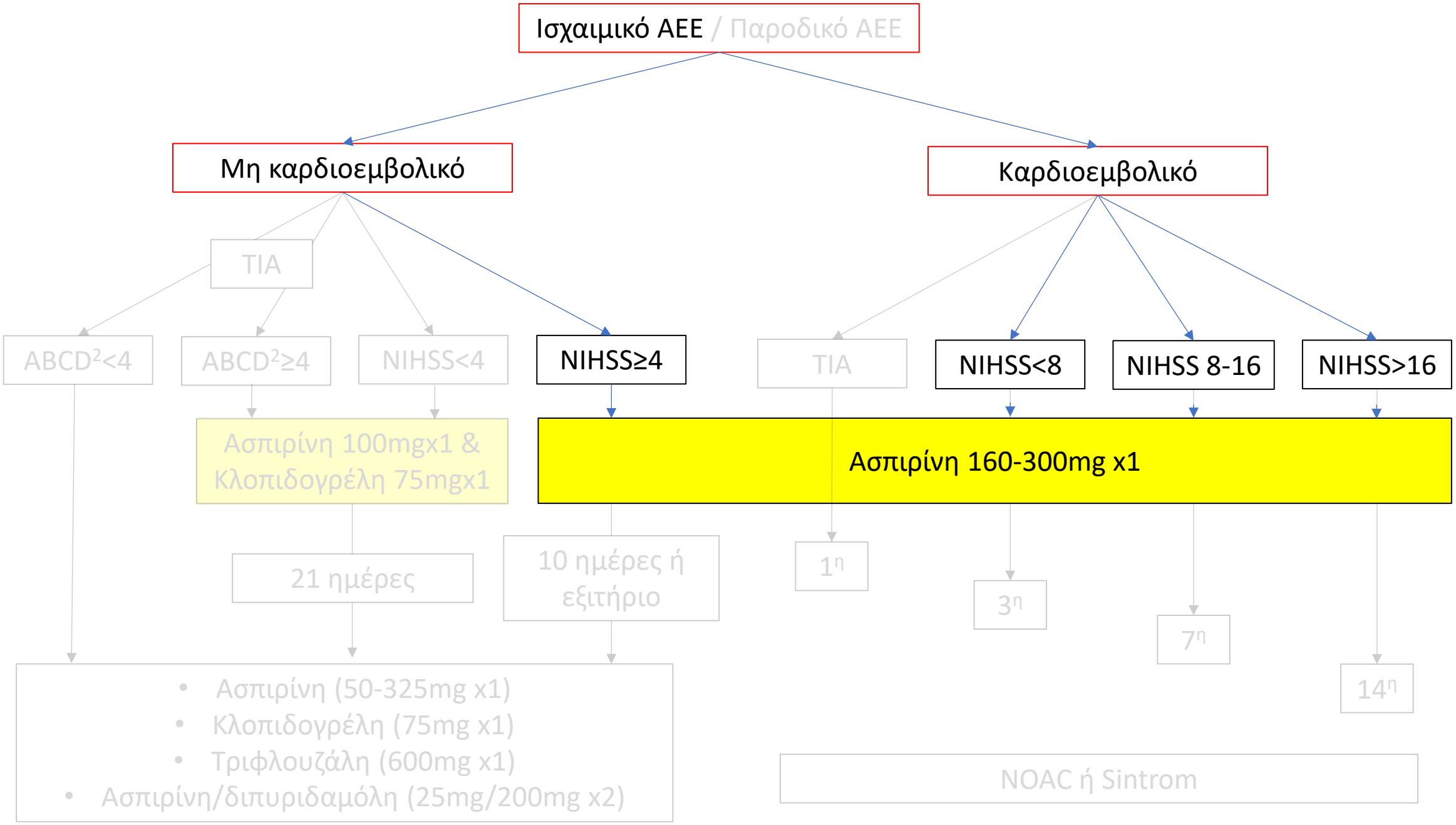
In people with a non-cardioembolic minor ischaemic stroke (NIHSS score of 3 or less) or high-risk TIA (ABCD2 score of 4 or more) in the past 24 hours, we recommend 21-days of dual antiplatelet therapy with aspirin and clopidogrel, followed by antiplatelet monotherapy thereafter.

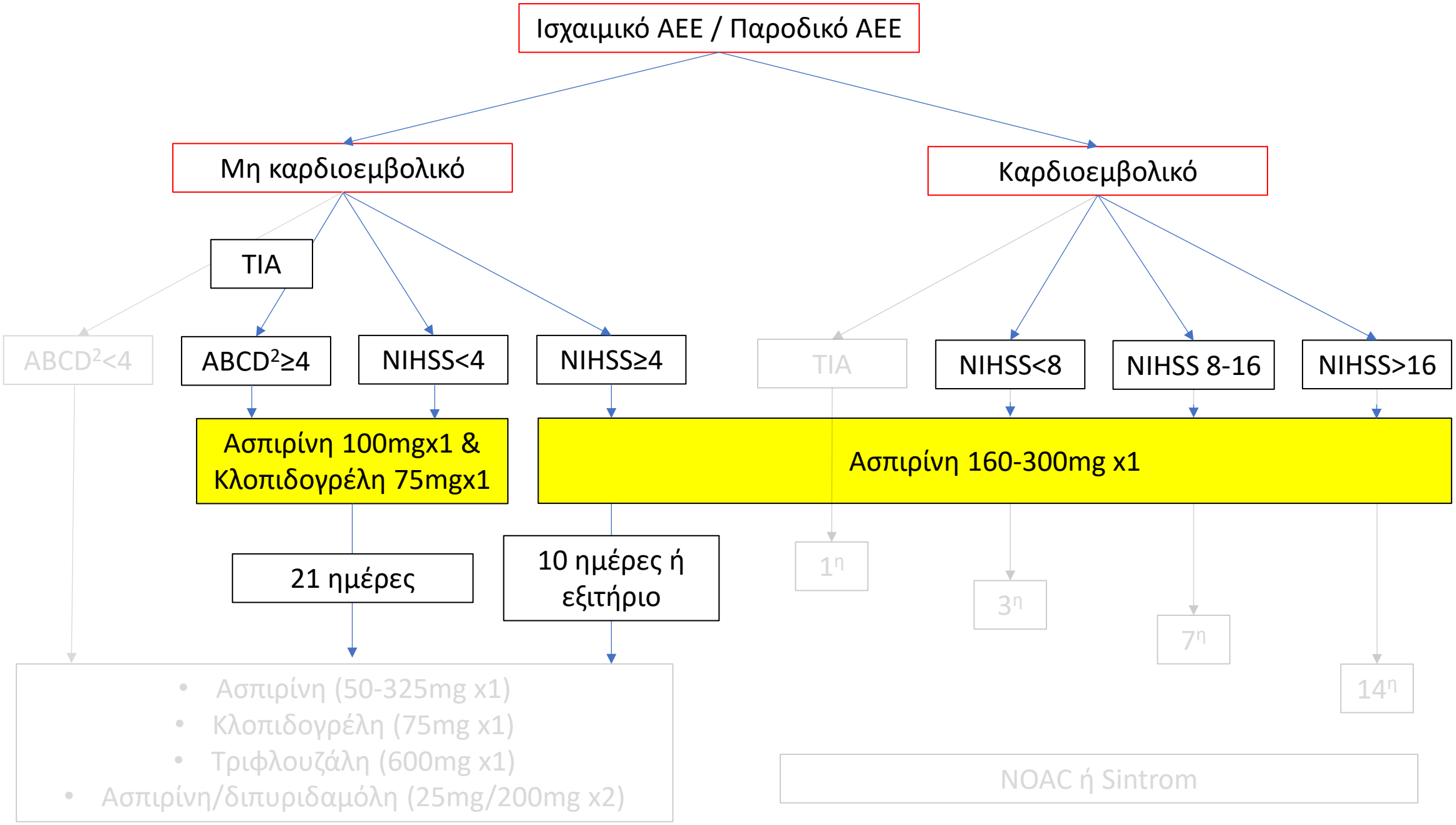
Quality of evidence: High ⊕⊕⊕⊕

Strength of recommendation: Strong for intervention ↑↑

Expert consensus statement

In people with acute non-cardioembolic low risk TIA or in whom the diagnosis is uncertain, 11/11 experts voted against use of dual antiplatelet therapy over monotherapy


Ισχαιμικό ΑΕΕ / Παροδικό ΑΕΕ
Μη καρδιοεμβολικό
Καρδιοεμβολικό
TIA
ABCD²<4
ABCD²≥4
NIHSS<4
NIHSS≥4
TIA
NIHSS<8
NIHSS 8-16
NIHSS>16
Ασπιρίνη 100mgx1 & Κλοπιδογρέλη 75mgx1
Ασπιρίνη 160-300mg x1
21 ημέρες
10 ημέρες ή εξιτήριο
1η
3η
7η
14η
• Ασπιρίνη (50-325mg x1)
• Κλοπιδογρέλη (75mg x1)
• Τριφλουζάλη (600mg x1)
• Ασπιρίνη/διπυριδαμόλη (25mg/200mg x2)
NOAC ή Sintrom


Ισχαιμικό ΑΕΕ / Παροδικό ΑΕΕ
Μη καρδιοεμβολικό
Καρδιοεμβολικό
TIA
ABCD²<4
ABCD²≥4
NIHSS<4
NIHSS≥4
TIA
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Ασπιρίνη 100mgx1 & Κλοπιδογρέλη 75mgx1
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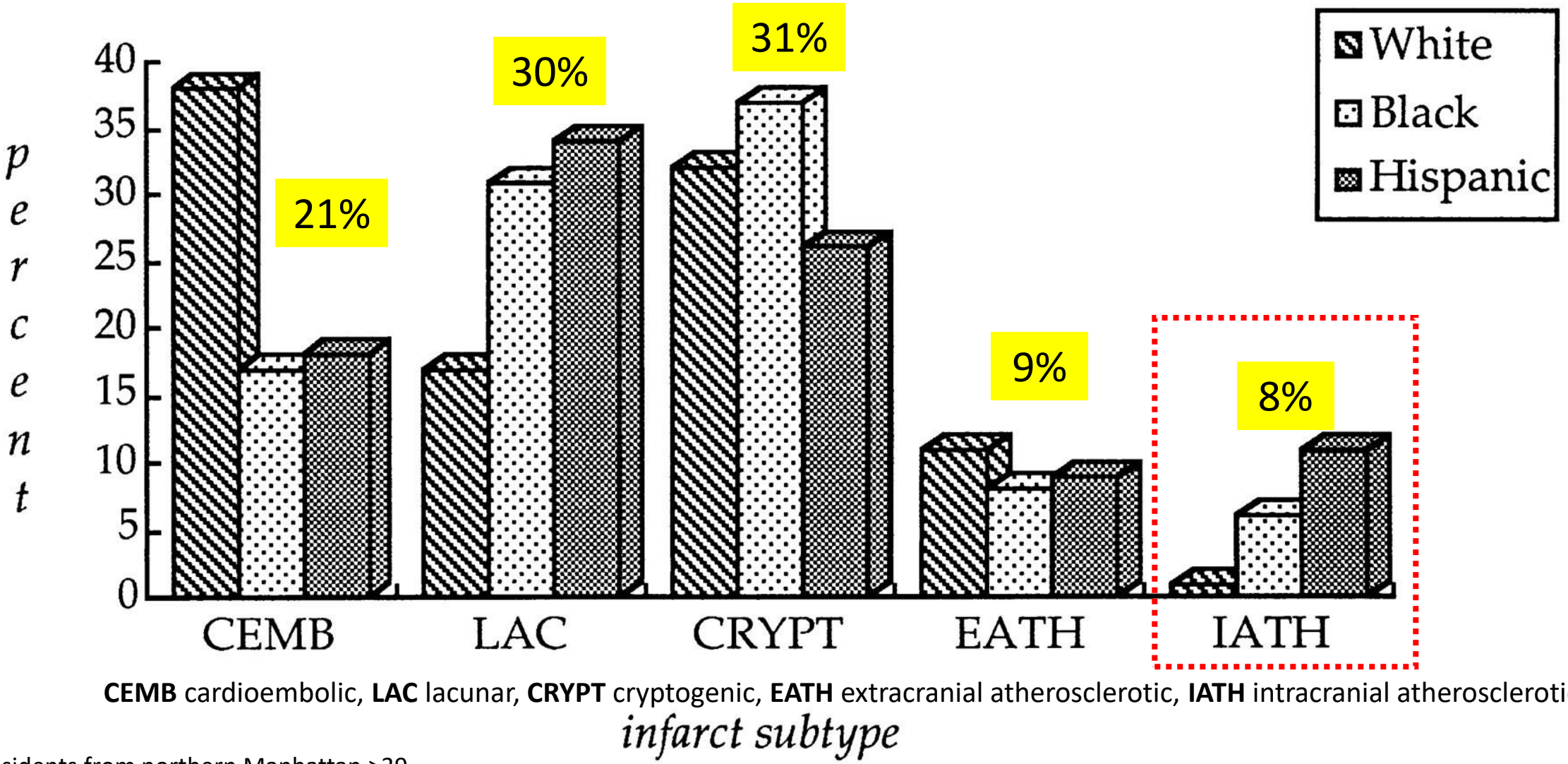
NOAC ή Sintrom

Race-ethnicity and determinants of intracranial atherosclerotic cerebral infarction. The Northern Manhattan Stroke Study



CEMB cardioembolic, **LAC** lacunar, **CRYPT** cryptogenic, **EATH** extracranial atherosclerotic, **IATH** intracranial atherosclerotic.

438 Residents from northern Manhattan >39 years hospitalized for acute ischemic stroke (black 35%, Hispanic 46%, white 19%)

Stroke 1995;26(1):14-20

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JULY 16, 2020 VOL. 383 NO. 3

Ticagrelor and Aspirin or Aspirin Alone in Acute Ischemic Stroke or TIA

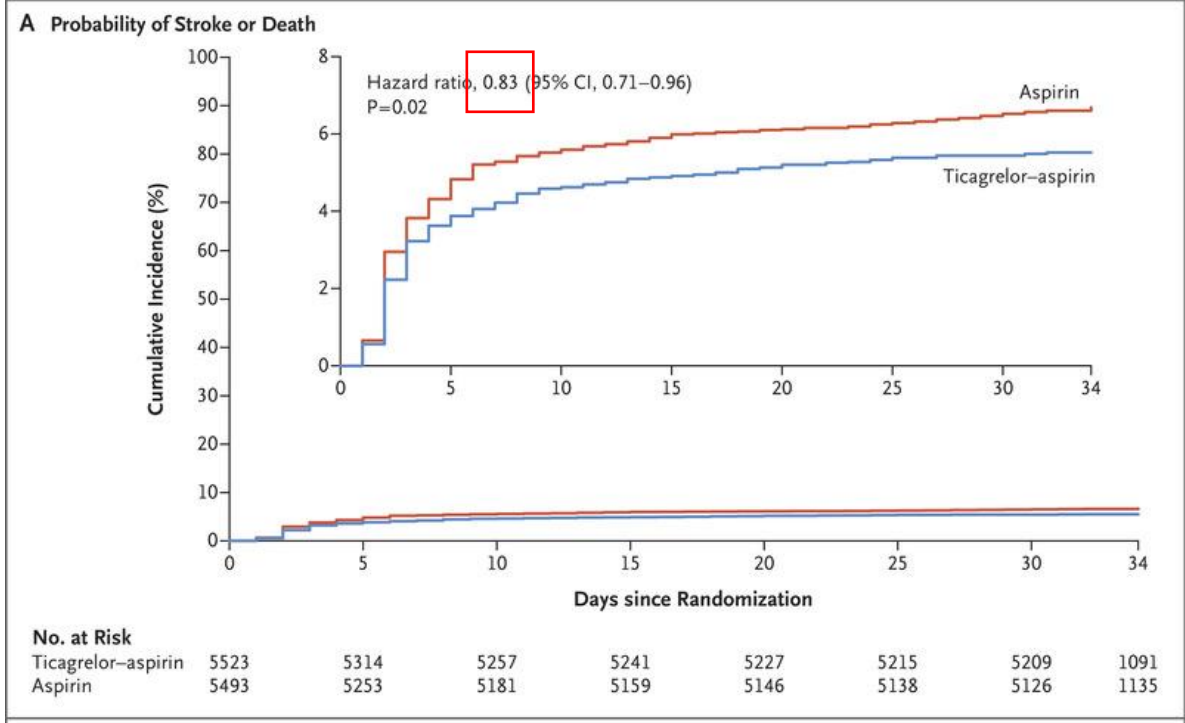
THALES

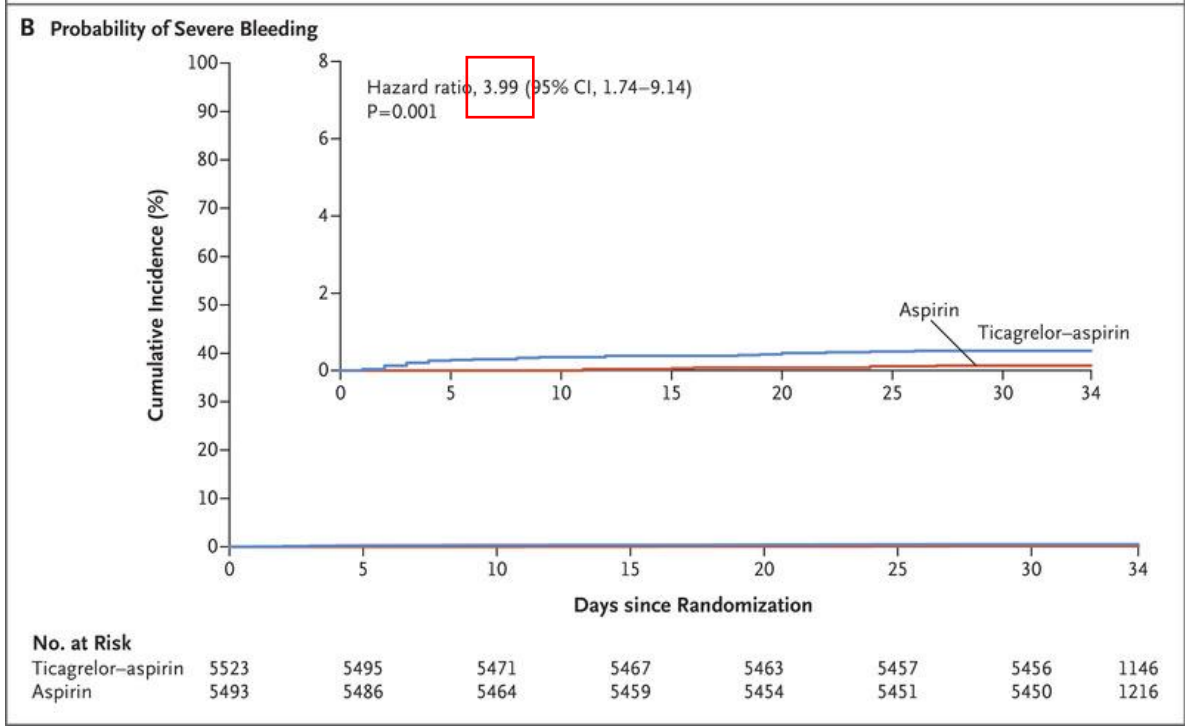
NIHSS ≤ 5 or
TIA +ABCD^2 ≥6 or
Intra-/ extracranial stenosis >50%

Table 2. Efficacy and Safety Outcomes.*

Outcome	Ticagrelor–Aspirin Group (N=5523)		Aspirin Group (N=5493)		Hazard Ratio (95% CI)	P Value
	Patients with Event	Event Rate†	Patients with Event	Event Rate†		
	no. (%)	%	no. (%)	%		
Primary outcome						
Stroke or death	303 (5.5)	5.4	362 (6.6)	6.5	0.83 (0.71–0.96)	0.02
Stroke	284 (5.1)	5.1	347 (6.3)	6.3	0.81 (0.69–0.95)	
Death	36 (0.7)	0.6	27 (0.5)	0.5	1.33 (0.81–2.19)	
Secondary outcomes						
Ischemic stroke	276 (5.0)	5.0	345 (6.3)	6.2	0.79 (0.68–0.93)	0.004
Overall disability‡	1282 (23.8)	NA	1284 (24.1)	NA	0.98 (0.89–1.07)	0.61
Safety outcomes						
Severe bleeding	28 (0.5)	0.5	7 (0.1)	0.1	3.99 (1.74–9.14)	0.001
Intracranial hemorrhage or fatal bleeding	22 (0.4)	0.4	6 (0.1)	0.1	3.66 (1.48–9.02)	0.005
Fatal bleeding	11 (0.2)		2 (<0.1)			
Intracranial hemorrhage	20 (0.4)	0.4	6 (0.1)	0.1	3.33 (1.34–8.28)	0.01
Hemorrhagic stroke	10 (0.2)		2 (<0.1)			
Moderate or severe bleeding	36 (0.7)	0.6	11 (0.2)	0.2	3.27 (1.67–6.43)	<0.001
Premature permanent discontinuation of trial treatment owing to bleeding	152 (2.8)	2.9	32 (0.6)	0.6	4.80 (3.28–7.02)	<0.001

* NA denotes not applicable.
† Event rates are Kaplan–Meier estimates of the percentage of patients with events.
‡ Overall disability was determined by a score greater than 1 on the modified Rankin scale. The odds ratio is shown rather than the hazard ratio (5386 patients in the ticagrelor–aspirin group and 5333 patients in the aspirin group).





European Stroke Organisation expedited recommendation for the use of short-term dual antiplatelet therapy early after minor stroke and high-risk TIA

EUROPEAN STROKE JOURNAL

European Stroke Journal
0(0) 1–5

Recommendation 2

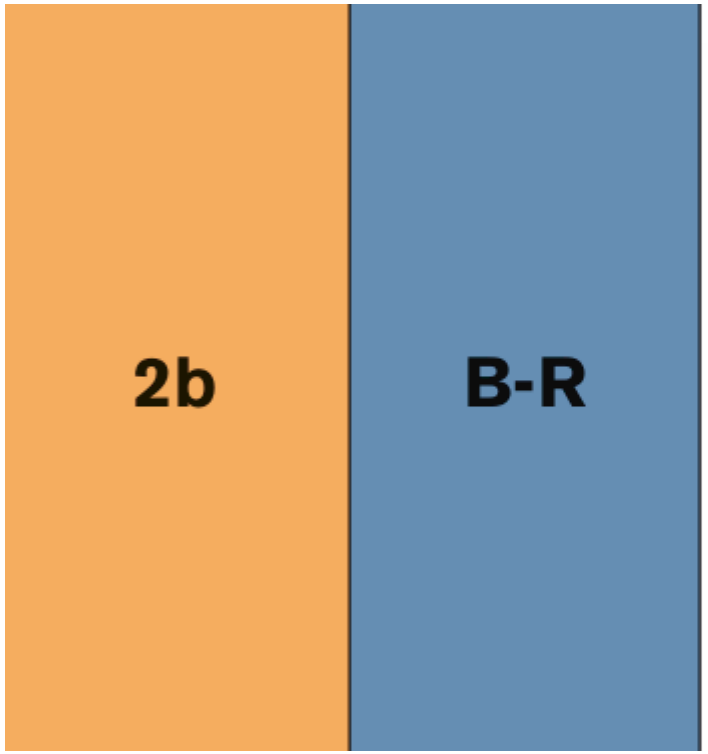
In people with non-cardioembolic mild to moderate ischaemic stroke (NIHSS of 5 or less) or high-risk TIA (ABCD2 score of 6 or more or other high-risk features*) in the past 24 hours, we suggest 30-days of dual antiplatelet therapy with aspirin and ticagrelor followed by antiplatelet monotherapy thereafter.

*defined as either intracranial atherosclerotic disease or at least 50% stenosis in an internal carotid artery that could account for the presentation.

Quality of evidence: Moderate ⊕⊕⊕

Strength of recommendation: Weak for intervention ↑?

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association



13. In patients with recent (<24 hours) minor (NIHSS score ≤5) noncardioembolic AIS or high-risk TIA (ABCD² score ≥6 or symptomatic intracranial or extracranial ≥50% stenosis of an artery that could account for TIA) who did not receive IVT, DAPT with ticagrelor (including loading dose) plus aspirin for 30 days may be considered to reduce the risk of 30-day recurrent stroke.[26]

Stroke. 2026 Jan 26. Epub ahead of print.

2021 Guideline for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack

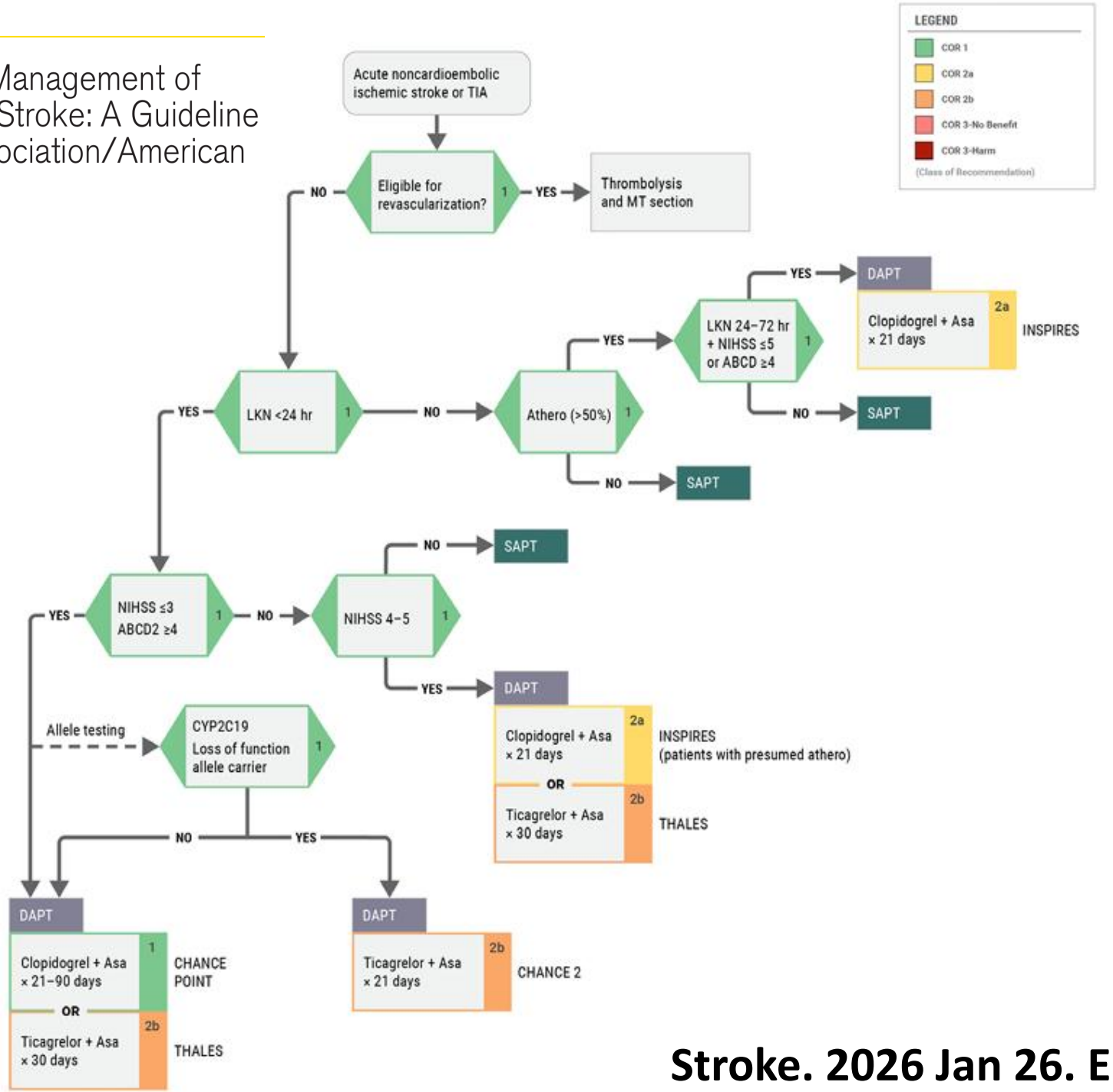
A Guideline From the American Heart Association/American Stroke Association

Recommendations for Intracranial Large Artery Atherosclerosis
Referenced studies that support recommendations are summarized in online Data Supplements 20–27.

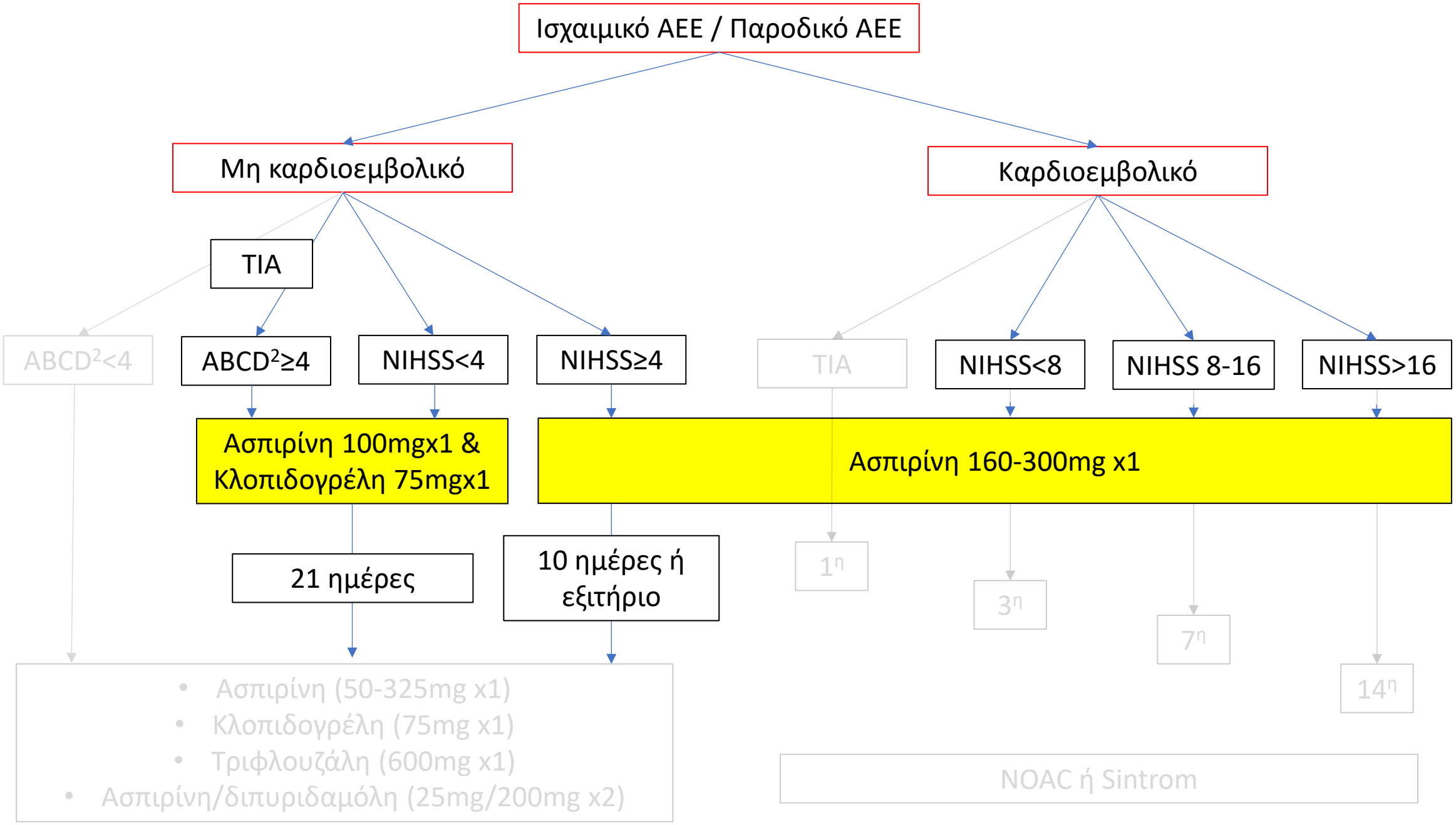
COR	LOE	Recommendations
		Antithrombotic Therapy
1	WASID B-R SAMMPRIS	1. In patients with a stroke or TIA caused by 50% to 99% stenosis of a major intracranial artery, aspirin 325 mg/d is recommended in preference to warfarin to reduce the risk of recurrent ischemic stroke and vascular death.[335,336]
2a	SAMMPRIS WASID B-NR CHANCE CLAIR	2. In patients with recent stroke or TIA (within 30 days) attributable to severe stenosis (70%–99%) of a major intracranial artery, the addition of clopidogrel 75 mg/d to aspirin for up to 90 days is reasonable to further reduce recurrent stroke risk.[336–339]
2b	THALES B-NR	3. In patients with recent (within 24 hours) minor stroke or high-risk TIA and concomitant ipsilateral >30% stenosis of a major intracranial artery, the addition of ticagrelor 90 mg twice a day to aspirin for up to 30 days might be considered to further reduce recurrent stroke risk.[340]
2b	TOSS-I C-LD TOSS-II	4. In patients with stroke or TIA attributable to 50% to 99% stenosis of a major intracranial artery, the addition of cilostazol 200 mg/day to aspirin or clopidogrel might be considered to reduce recurrent stroke risk.[341–344]
2b	C-EO	5. In patients with stroke or TIA attributable to 50% to 99% stenosis of a major intracranial artery, the usefulness of clopidogrel alone, the combination of aspirin and dipyridamole, ticagrelor alone, or cilostazol alone for secondary stroke prevention is not well established.

Stroke. 2021;52:e364–e467.

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association



Stroke. 2026 Jan 26. Epub ahead of print.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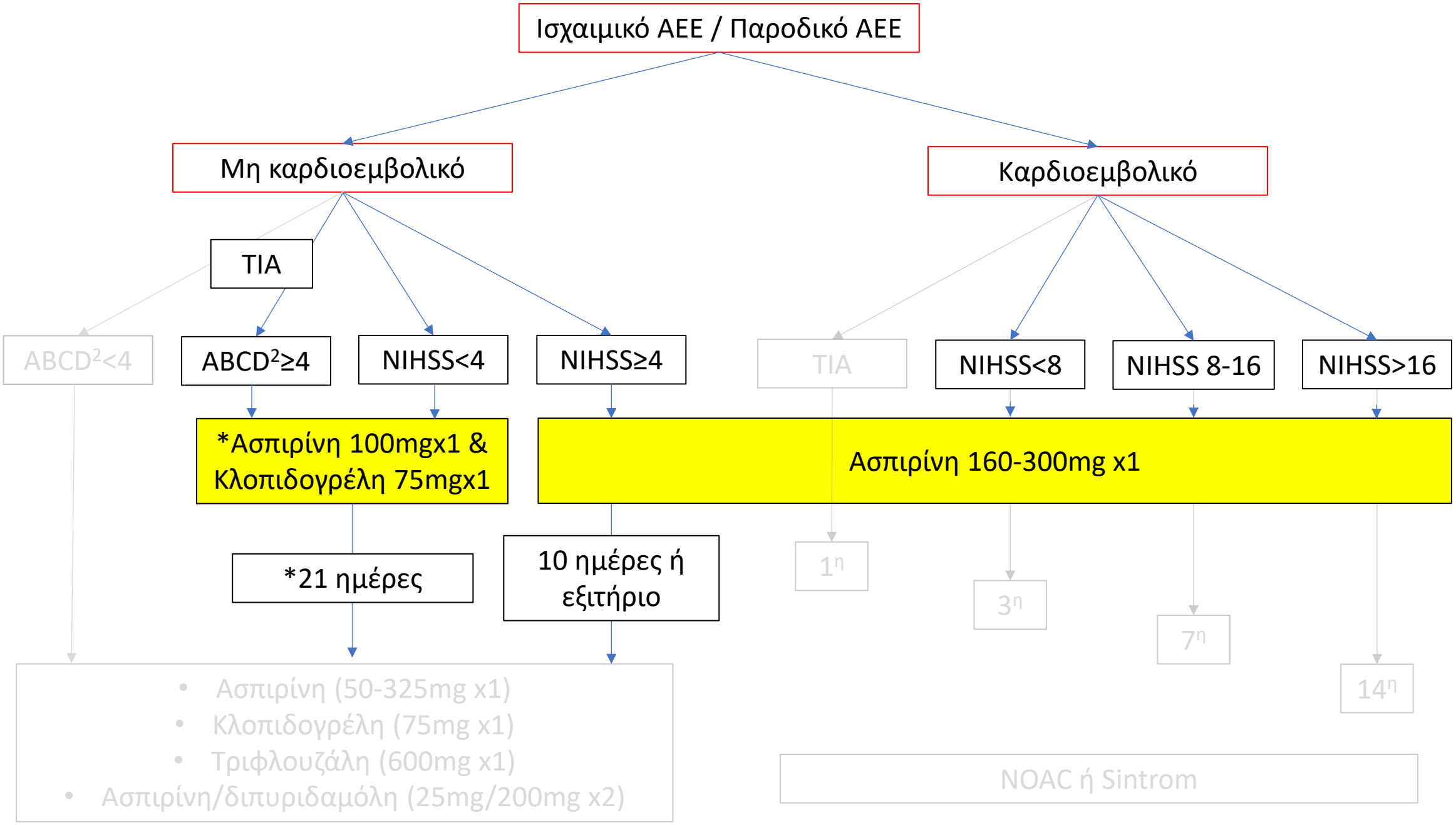
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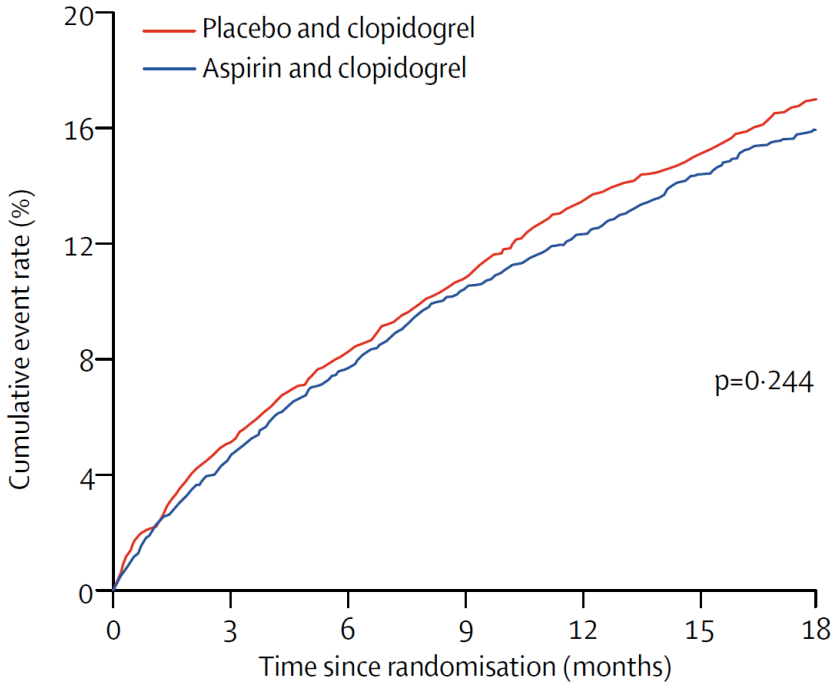
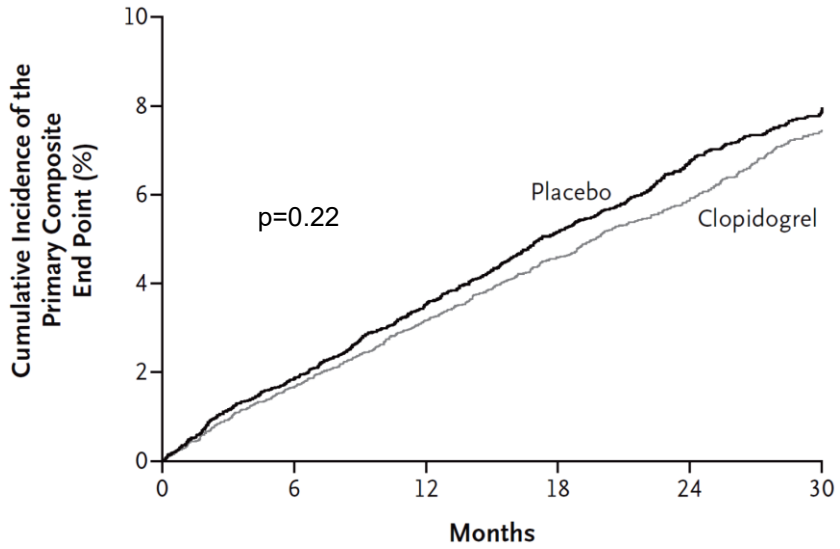
MATCH: Clopidogrel/aspirin vs clopidogrel / Major vascular event



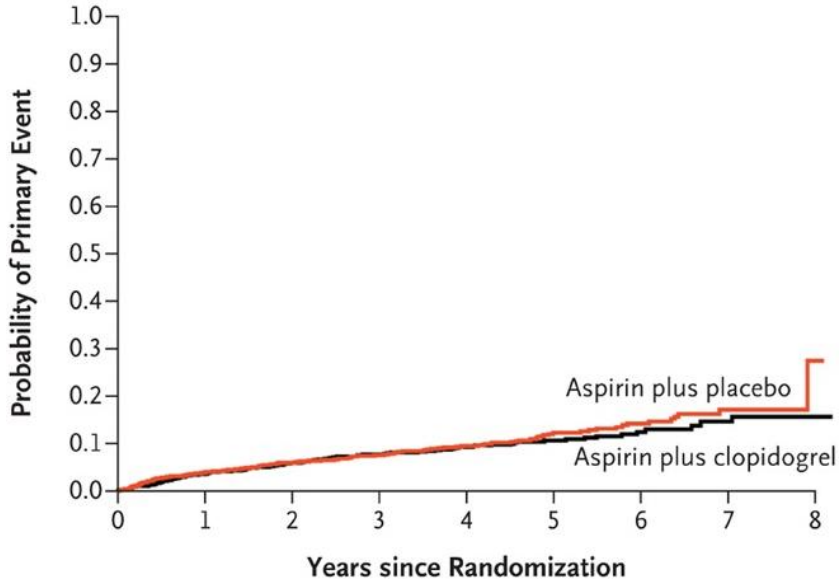
Lancet 2004; 364: 331–37

CHARISMA: Clopidogrel/aspirin vs aspirin / Major vascular event

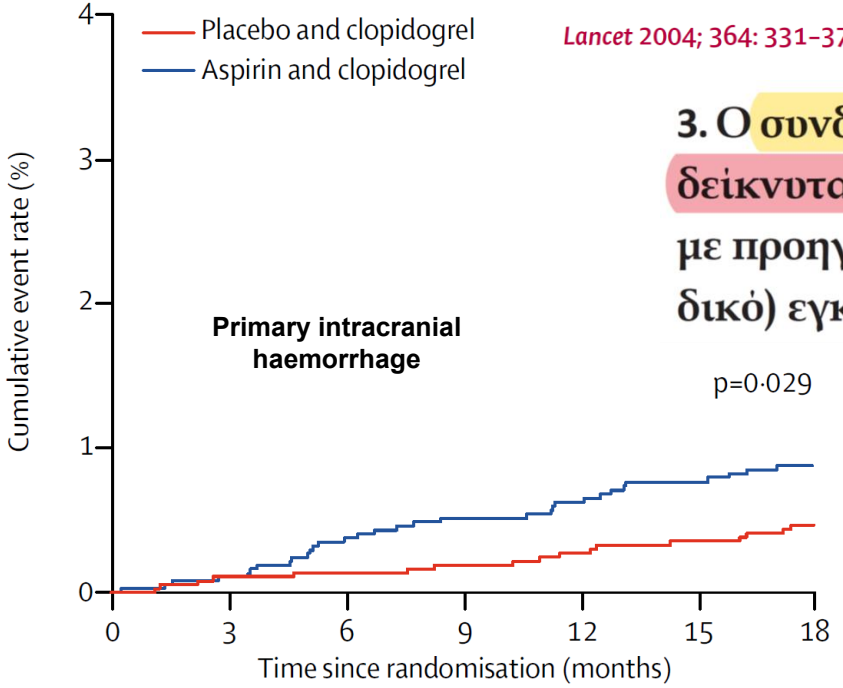


N Engl J Med 2006;354:1706-17

SPS3: Aspirin/clopidogrel vs aspirin / Stroke recurrence



N Engl J Med 2012;367:817-25



3. Ο συνδυασμός ασπιρίνης με κλοπιδογρέλη αντενδείκνυται για μακροχρόνια χορήγηση σε ασθενείς με προηγούμενο ισχαιμικό (εγκατεστημένο ή παροδικό) εγκεφαλικό επεισόδιο (1A).

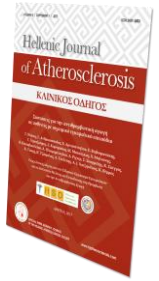


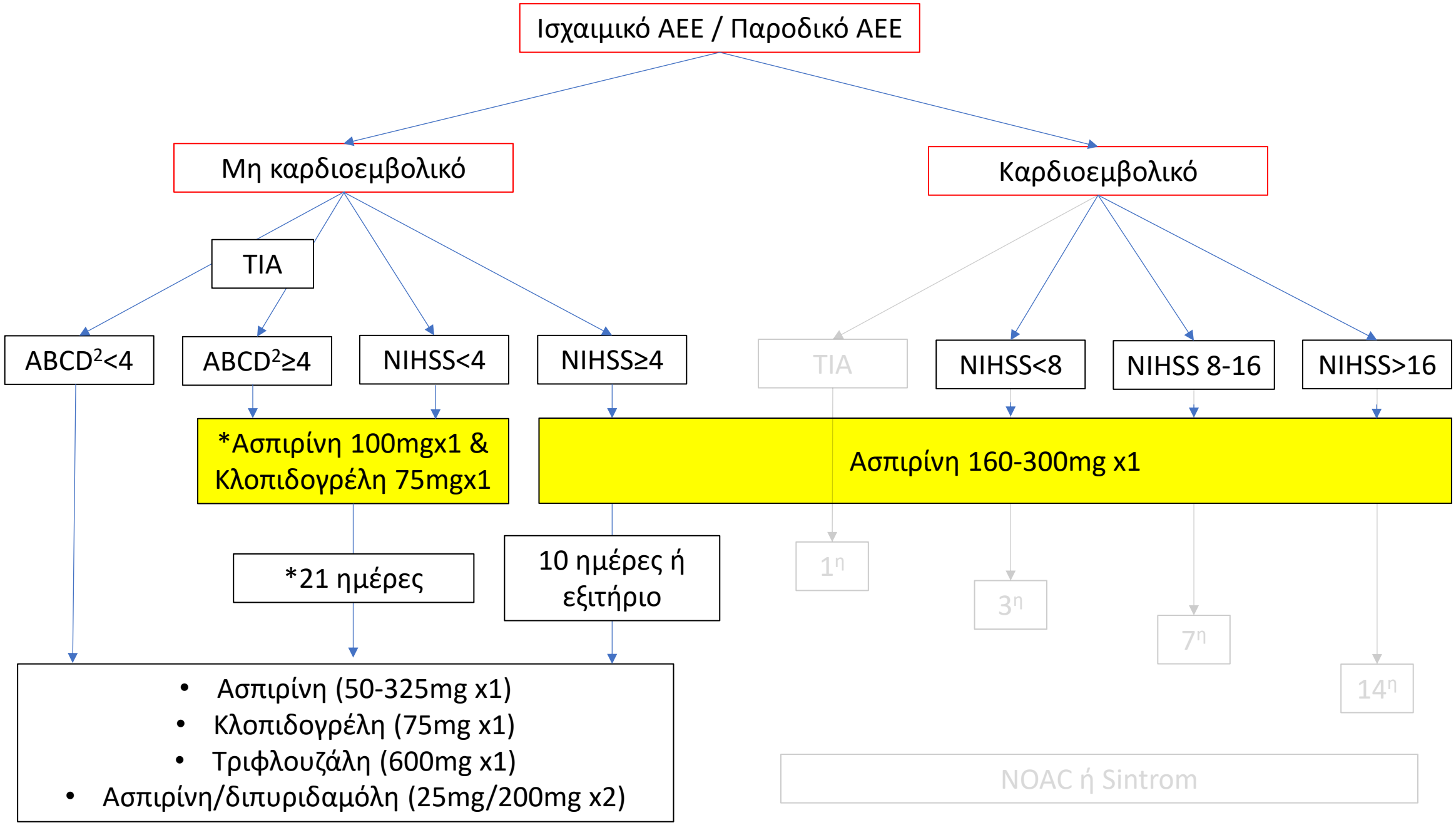
Table 2. Primary Efficacy Outcomes.*

Outcome	Aspirin plus Placebo (N=1503)		Aspirin plus Clopidogrel (N=1517)		Hazard Ratio (95% CI)	P Value
	no.	*rate (%/yr)*	*no.*	*rate (%/yr)*		
All strokes (ischemic and hemorrhagic)	138	2.7	125	2.5	0.92 (0.72–1.16)	0.48
Ischemic stroke	124	2.4	100	2.0	0.82 (0.63–1.09)	0.13
Intracranial hemorrhage	13	0.25	21	0.42	1.65 (0.83–3.31)	0.15
Unknown†	1	0.02	4	0.08	3.97 (0.44–35.47)	0.22
Disabling or fatal stroke‡	40	0.78	42	0.84	1.06 (0.69–1.64)	0.79
Transient ischemic attack without stroke	39	0.78	28	0.57	0.73 (0.45–1.18)	0.19
Myocardial infarction	38	0.71	31	0.59	0.84 (0.52–1.35)	0.47
Other thromboembolic events§	12	0.22	21	0.40	1.81 (0.89–3.68)	0.10
Major vascular event¶	174	3.4	153	3.1	0.89 (0.72–1.11)	0.29
All deaths	77	1.4	113	2.1	1.52 (1.14–2.04)	0.004
Vascular causes	19	0.35	27	0.51	1.46 (0.81–2.64	0.20
Cerebral	9	0.17	10	0.19	1.13 (0.46–2.78)	0.79
Noncerebral	10	0.18	17	0.32	1.77 (0.81–3.87)	0.15
Probable vascular causes	6	0.11	18	0.34	3.09 (1.23–7.80)	0.02
Nonvascular causes	31	0.57	39	0.73	1.31 (0.82–2.10)	0.26
Uncertain	21	0.39	29	0.55	1.41 (0.82–2.52)	0.21

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association

Early secondary prevention		
1	A	5. In patients with noncardioembolic AIS or TIA, antiplatelet therapy is indicated in preference to oral anticoagulation to reduce the risk of recurrent ischemic stroke and other cardiovascular events, while minimizing the risk of bleeding.[8,9]
1	C-EO	6. In patients with noncardioembolic AIS or TIA, the selection of an antiplatelet agent for early secondary stroke prevention should be individualized on the basis of patient risk factor profiles, cost, tolerance, relative known efficacy of the agents, and other clinical characteristics.

Stroke. 2026 Jan 26. Epub ahead of print.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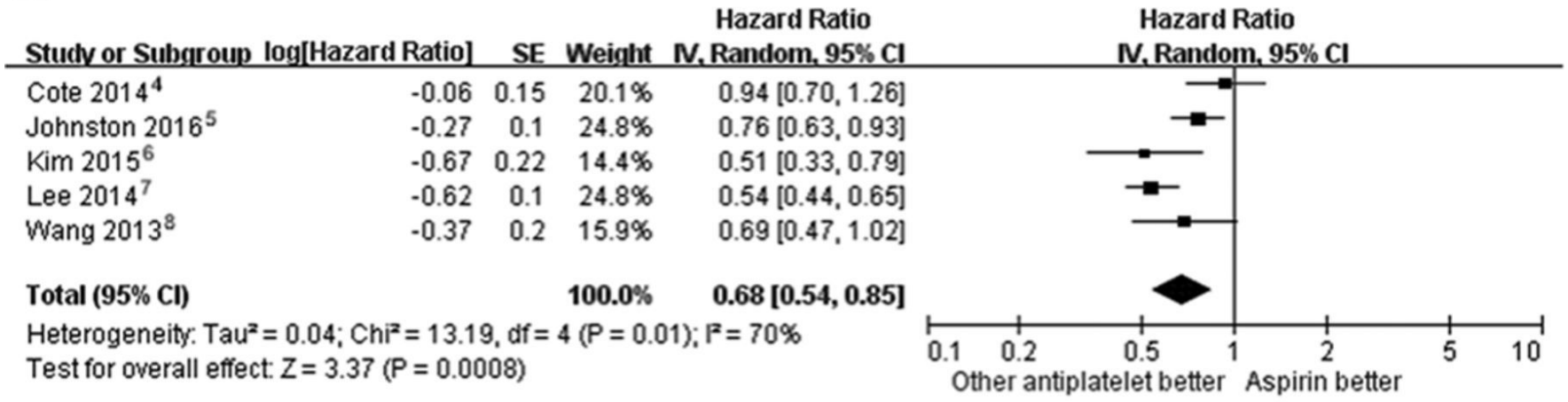
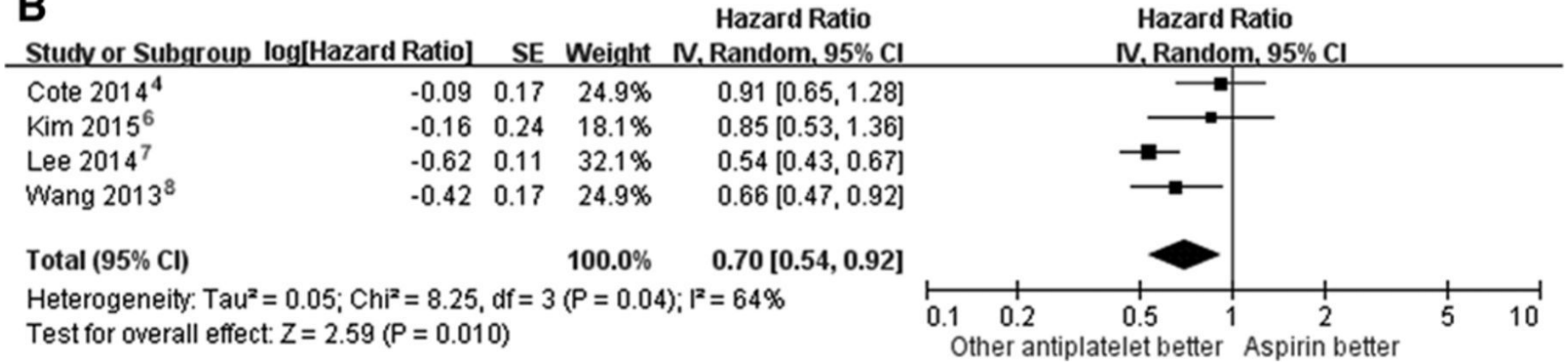
NOAC ή Sintrom

Lee M, Saver JL, Hong KS, Rao NM, Wu YL, Ovbiagele B.
Antiplatelet Regimen for Patients With Breakthrough Strokes While on Aspirin:
A Systematic Review and Meta-Analysis.
Stroke. 2017 Sep;48(9):2610-2613

A



B



Hazard ratio with 95% confidence interval (CI) estimates for (A) major adverse cardiovascular events and (B) recurrent stroke (active treatment vs control) by individual study and pooled

Conclusions

Among patients who experience an ischemic stroke or transient ischemic attack while on aspirin monotherapy, the addition of or a switch to another antiplatelet agent, especially in the first days after index event, is associated with fewer future vascular events, including stroke.

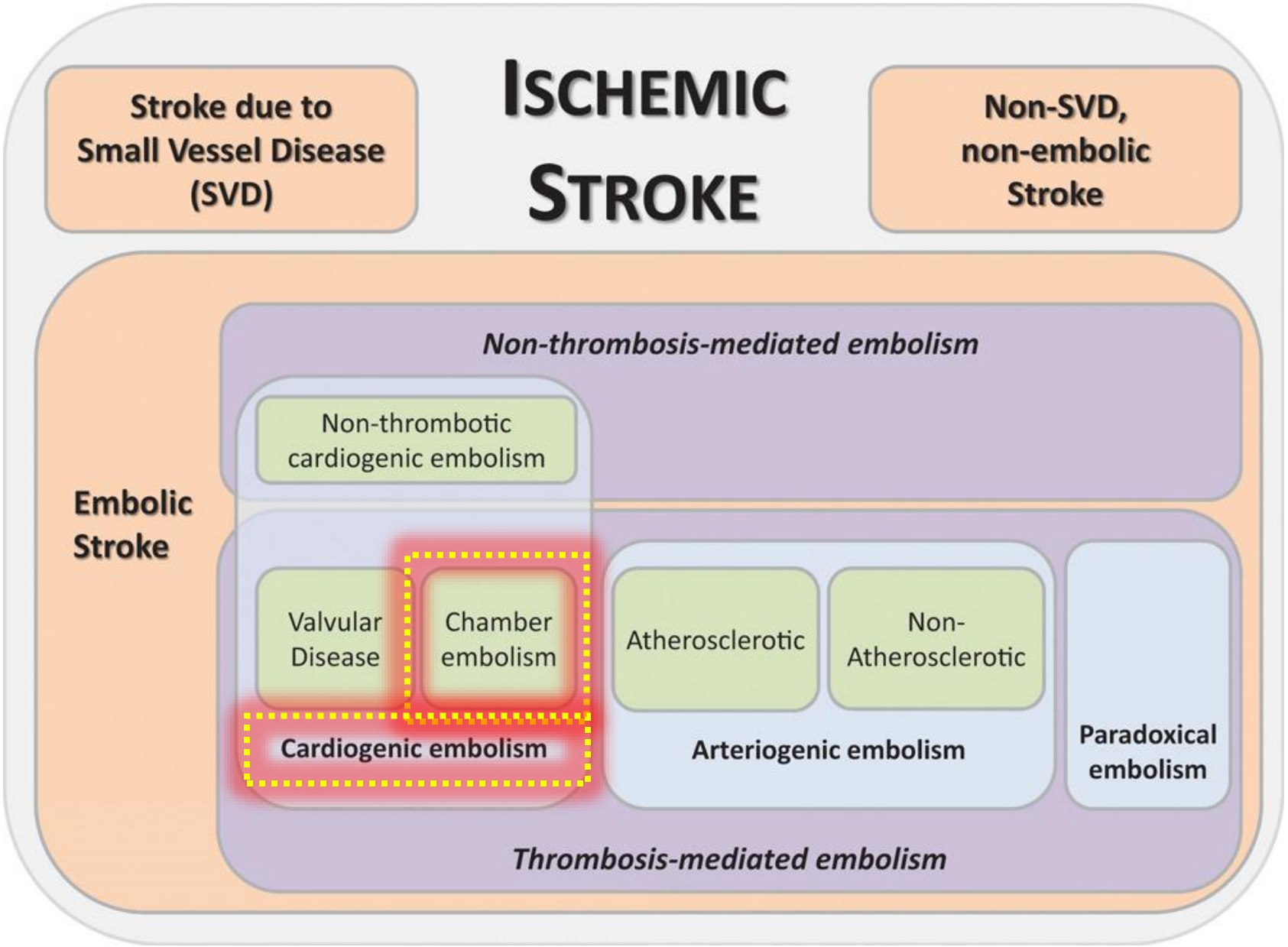
*The studies analyzed included **large registries** in addition to **randomized trials**.*
It was possible that the pooled results were biased because of confounding by indication.
*Therefore, these observational data can only be seen **as suggestive** rather than evidence-based guide for clinical practice.*

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association

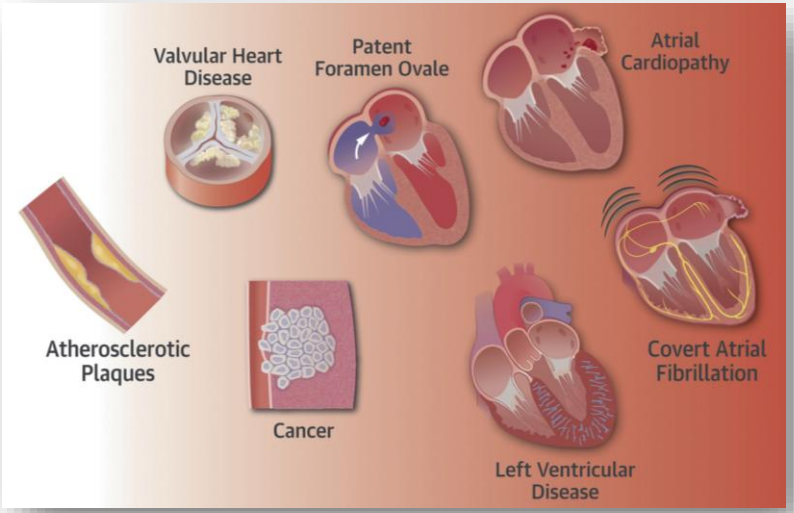
2b	**B-NR**	8. For patients already taking aspirin at the time of noncardioembolic ischemic stroke or TIA, the effectiveness of increasing the dose of aspirin or changing to another antiplatelet medication is not well established.[13,14]

Stroke. 2026 Jan 26. Epub ahead of print.

Ischemic stroke is an etiologically heterogeneous syndrome



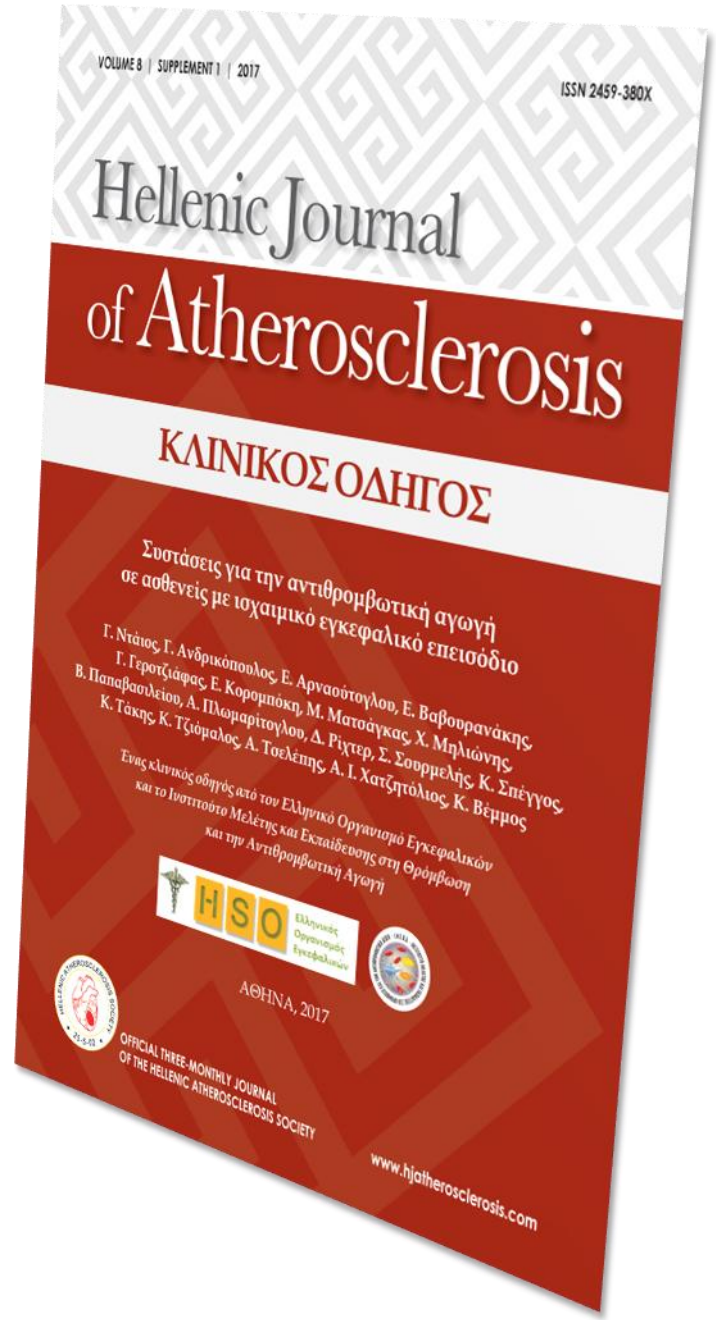
Ntaios G, Hart RG. Embolic stroke. Circulation. 2017;136:2403e2405.



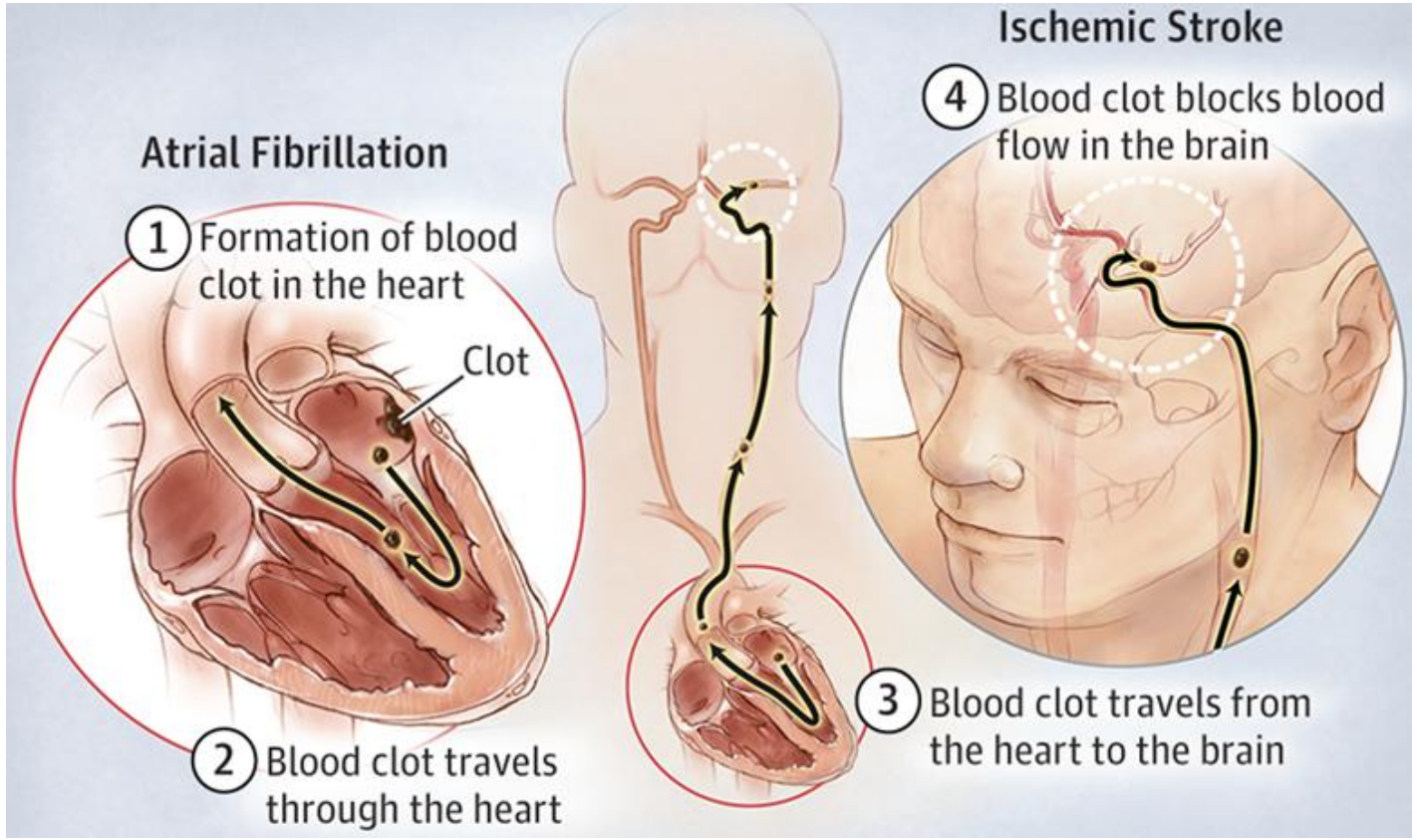
Ntaios G. Embolic Stroke of Undetermined Source: JACC Review Topic of the Week. J Am Coll Cardiol. 2020 Jan 28;75(3):333-340.

In order to optimize the secondary prevention strategy in a patient with ischemic stroke, it is rational to identify the underlying etiologic pathology.

6. Ασθενείς με ισχαιμικό (εγκατεστημένο ή παροδικό) εγκεφαλικό επεισόδιο και παροξυσμική, εμμένουσα ή χρόνια κολπική μαρμαρυγή πρέπει να λαμβάνουν αντιπηκτική αγωγή (1A). Η αντιαιμοπεταλιακή αγωγή σε ασθενείς με ισχαιμικό (εγκατεστημένο ή παροδικό) εγκεφαλικό επεισόδιο και παροξυσμική, εμμένουσα ή χρόνια κολπική μαρμαρυγή είναι σαφώς υποδεέστερη της αντιπηκτικής αγωγής και πρέπει να μην προτιμάται έναντι της αντιπηκτικής (1A). Σε ασθενείς στους οποίους δεν δύναται να χορηγηθεί αντιπηκτική αγωγή, μπορεί να χορηγηθεί ασπιρίνη (1A).



2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)

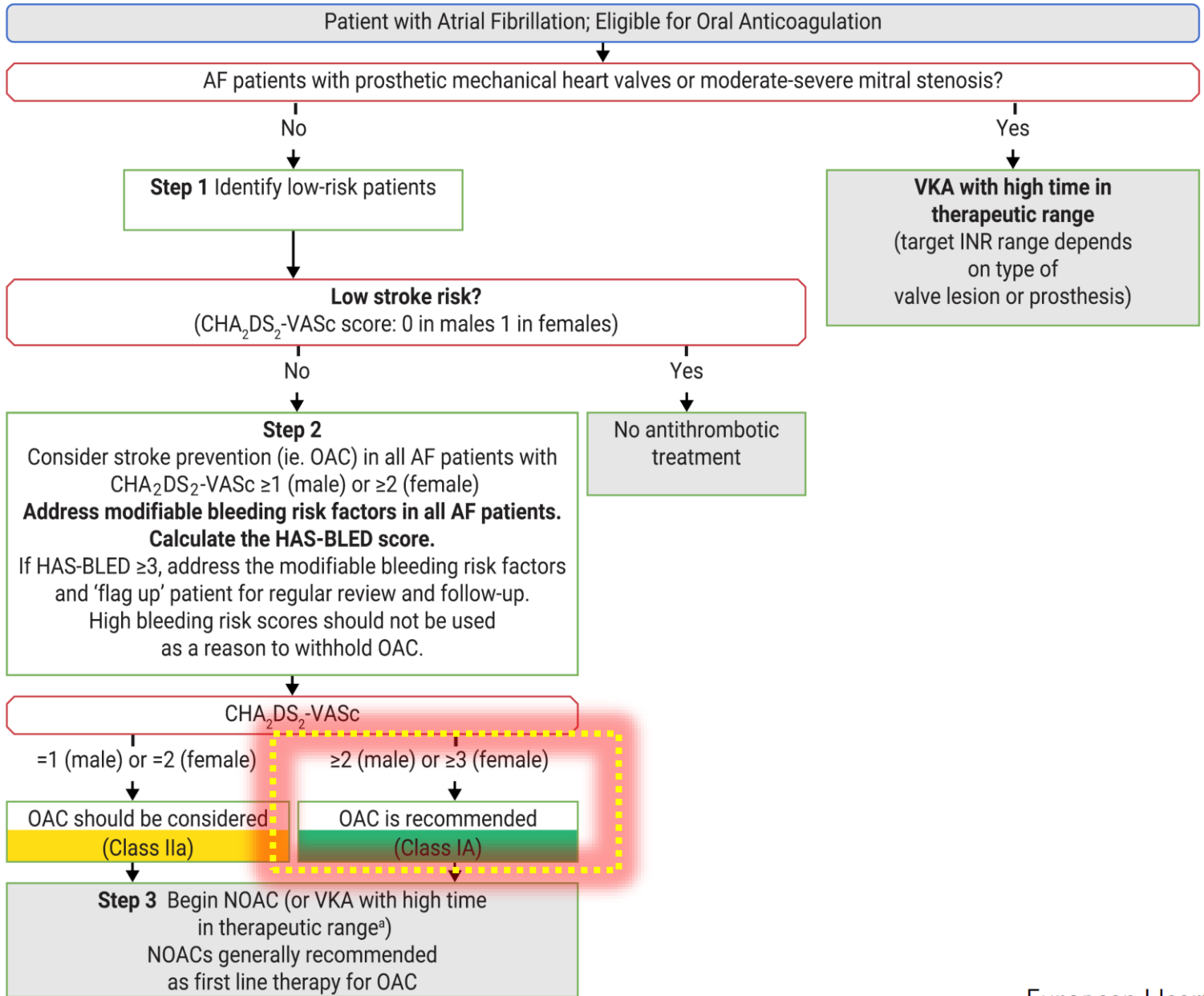


CHA_2DS_2-VASc score Risk factors and definitions		Points awarded
C	**Congestive heart failure** Clinical HF, or objective evidence of moderate to severe LV dysfunction, or HCM	1
H	**Hypertension** or on antihypertensive therapy	1
A	**Age 75 years or older**	2
D	**Diabetes mellitus** Treatment with oral hypoglycaemic drugs and/or insulin or fasting blood glucose >125 mg/dL (7 mmol/L)	1
S	**Stroke**Previous stroke, TIA, or thromboembolism	2
V	**Vascular disease** Angiographically significant CAD, previous myocardial infarction, PAD, or aortic plaque	1
A	**Age 65 – 74 years**	1
Sc	**Sex category (female)**	1
Maximum score		**9**

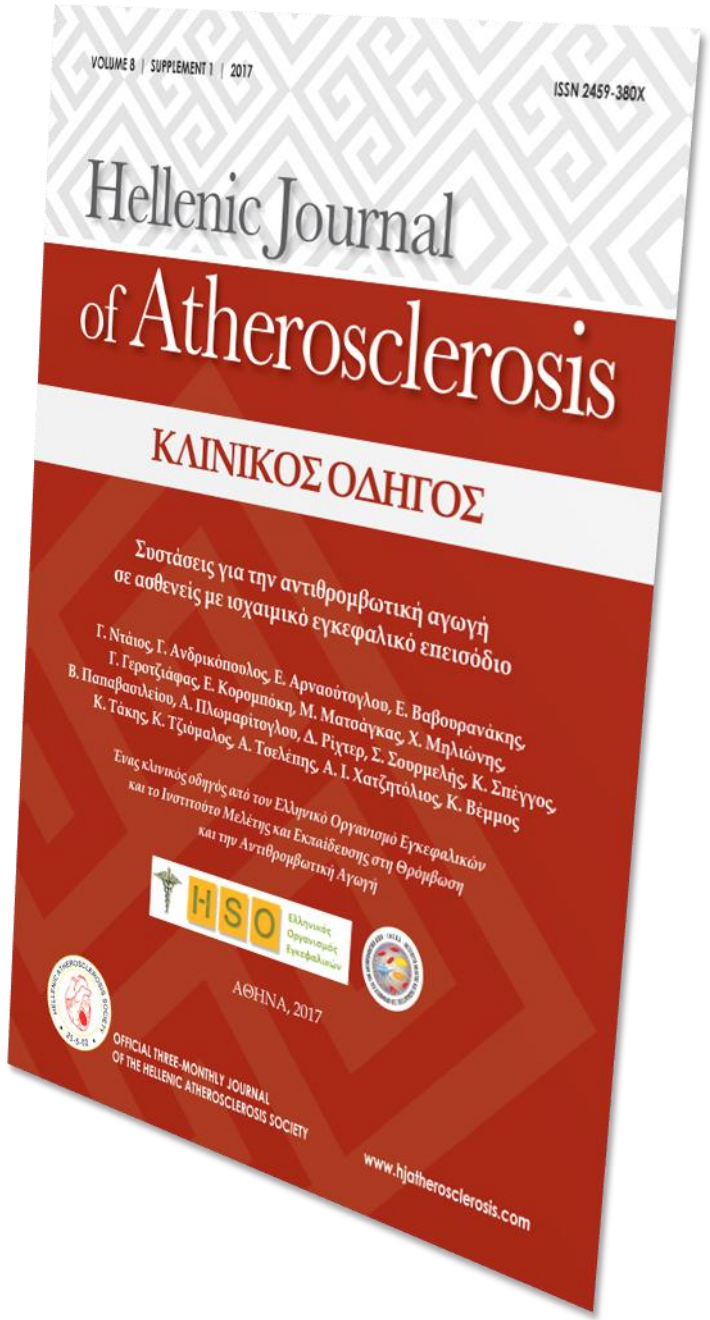
European Heart Journal (2020) **42**, 373–498

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)

ESC European Society of Cardiology



7. Οι επιλογές αντιπηκτικής αγωγής σε ασθενείς με ισχαιμικό (εγκατεστημένο ή παροδικό) εγκεφαλικό επεισόδιο και παροξυσμική, εμμένουσα ή χρόνια κολπική μαρμαρυγή μη βαλβιδικής αιτιολογίας είναι οι ανταγωνιστές της βιταμίνης Κ (ασενοκουμαρόλη ή βαρφαρίνη) (1Α), οι άμεσοι ανταγωνιστές της θρομβίνης (νταμπιγκαντράνη) (1Α) και οι αναστολείς του παράγοντα Χa (ριβαροξαμπάνη, απιξαμπάνη ή εντοξαμπάνη ή) (1Α).



ROCKET AF

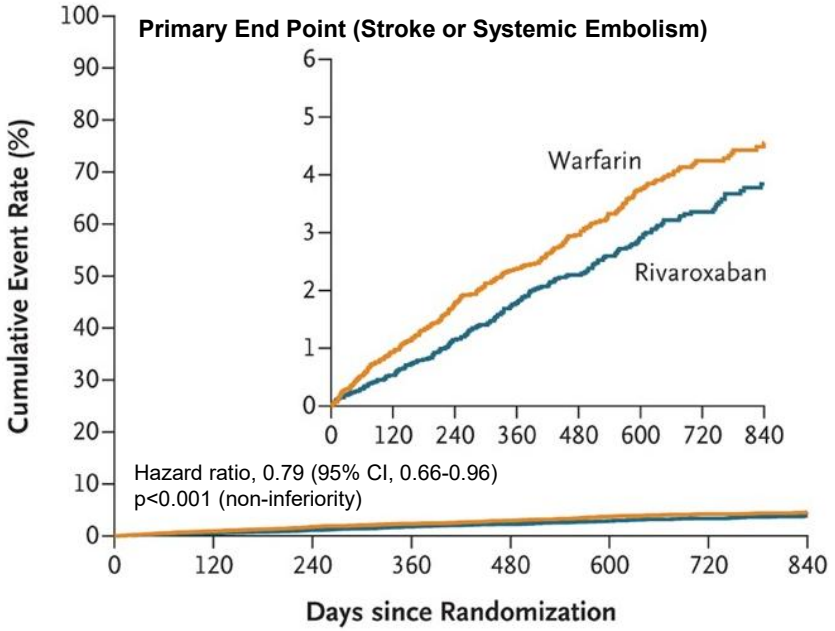
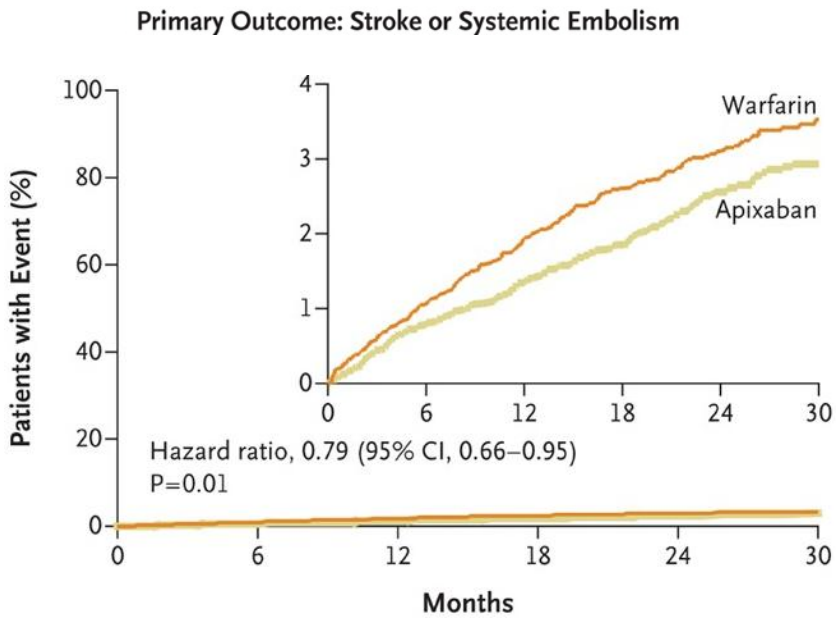


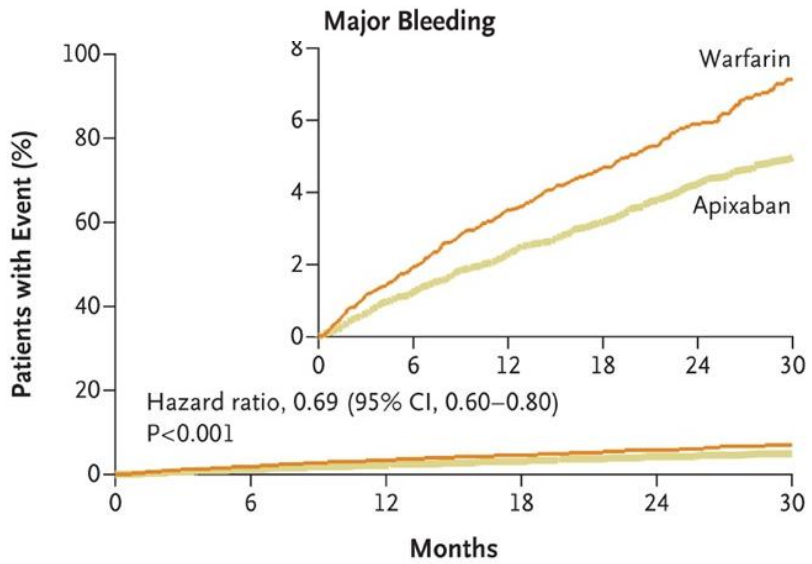
Table 3. Rates of Bleeding Events.*

Variable	Rivaroxaban (N=7111) Events no. (%)	Rivaroxaban (N=7111) Event Rate no./100 patient-yr	Warfarin (N=7125) Events no. (%)	Warfarin (N=7125) Event Rate no./100 patient-yr	Hazard Ratio (95% CI)†	P Value‡
Principal safety end point: major and nonmajor clinically relevant bleeding§	1475 (20.7)	14.9	1449 (20.3)	14.5	1.03 (0.96–1.11)	0.44
Major bleeding						
Any	395 (5.6)	3.6	386 (5.4)	3.4	1.04 (0.90–1.20)	0.58
Decrease in hemoglobin ≥2 g/dl	305 (4.3)	2.8	254 (3.6)	2.3	1.22 (1.03–1.44)	0.02
Transfusion	183 (2.6)	1.6	149 (2.1)	1.3	1.25 (1.01–1.55)	0.04
Critical bleeding¶	91 (1.3)	0.8	133 (1.9)	1.2	0.69 (0.53–0.91)	0.007
Fatal bleeding	27 (0.4)	0.2	55 (0.8)	0.5	0.50 (0.31–0.79)	0.003
Intracranial hemorrhage	55 (0.8)	0.5	84 (1.2)	0.7	0.67 (0.47–0.93)	0.02
Nonmajor clinically relevant bleeding	1185 (16.7)	11.8	1151 (16.2)	11.4	1.04 (0.96–1.13)	0.35

N Engl J Med 2011;365:883-91

ARISTOTLE





N Engl J Med 2011;365:981-92

RE-LY

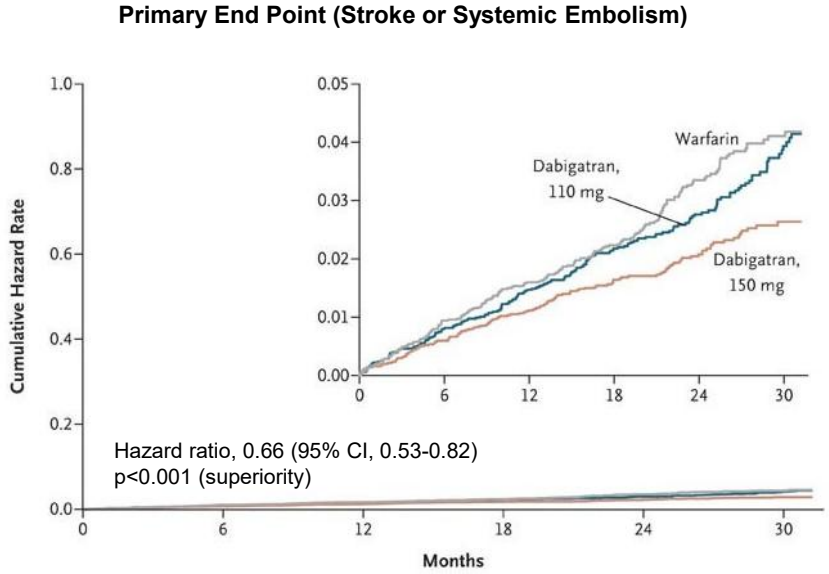


Table 3. Safety Outcomes, According to Treatment Group.*

Event	Dabigatran, 110 mg no. of patients	Dabigatran, 110 mg %/yr	Dabigatran, 150 mg no. of patients	Dabigatran, 150 mg %/yr	Warfarin no. of patients	Warfarin %/yr	Dabigatran, 110 mg, vs. Warfarin Relative Risk (95% CI)	Dabigatran, 110 mg, vs. Warfarin P Value	Dabigatran, 150 mg, vs. Warfarin Relative Risk (95% CI)	Dabigatran, 150 mg, vs. Warfarin P Value
Major bleeding	322	2.71	375	3.11	397	3.36	0.80 (0.69–0.93)	0.003	0.93 (0.81–1.07)	0.31
Life threatening	145	1.22	175	1.45	212	1.80	0.68 (0.55–0.83)	<0.001	0.81 (0.66–0.99)	0.04
Non–life threatening	198	1.66	226	1.88	208	1.76	0.94 (0.78–1.15)	0.56	1.07 (0.89–1.29)	0.47
Gastrointestinal†	133	1.12	182	1.51	120	1.02	1.10 (0.86–1.41)	0.43	1.50 (1.19–1.89)	<0.001
Minor bleeding	1566	13.16	1787	14.84	1931	16.37	0.79 (0.74–0.84)	<0.001	0.91 (0.85–0.97)	0.005
Major or minor bleeding	1740	14.62	1977	16.42	2142	18.15	0.78 (0.74–0.83)	<0.001	0.91 (0.86–0.97)	0.002
Intracranial bleeding	27	0.23	36	0.30	87	0.74	0.31 (0.20–0.47)	<0.001	0.40 (0.27–0.60)	<0.001
Extracranial bleeding	299	2.51	342	2.84	315	2.67	0.94 (0.80–1.10)	0.45	1.07 (0.92–1.25)	0.38
Net clinical benefit outcome‡	844	7.09	832	6.91	901	7.64	0.92 (0.84–1.02)	0.10	0.91 (0.82–1.00)	0.04

N Engl J Med 2009;361:1139-51

Nonvitamin-K-antagonist oral anticoagulants versus warfarin in patients with atrial fibrillation and previous stroke or transient ischemic attack: An updated systematic review and meta-analysis of randomized controlled trials

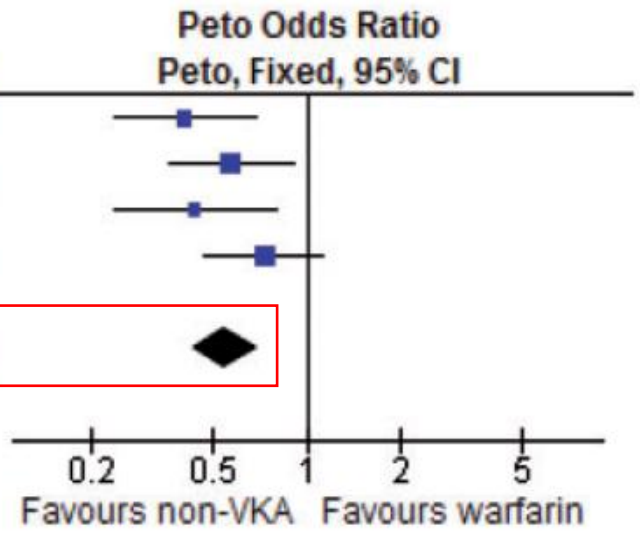
George Ntaios[1], Vasileios Papavasileiou[2], Hans-Chris Diener[3], Konstantinos Makaritsis[1] and Patrik Michel[4]

Forest plot of the effects of non-VKAs versus warfarin on safety outcomes: ***intracranial bleeding***

Study or Subgroup	non-VKA Events	non-VKA Total	Warfarin Events	Warfarin Total	Weight	Peto Odds Ratio Peto, Fixed, 95% CI
ARISTOTLE	15	1694	41	1742	22.0%	0.40 [0.24, 0.68]
ENGAGE AF-TIMI 48 (60mg)	27	1976	48	1991	29.4%	0.57 [0.36, 0.90]
RELY (150mg)	13	1233	30	1195	16.9%	0.43 [0.24, 0.79]
ROCKET-AF	34	3754	46	3714	31.6%	0.73 [0.47, 1.13]
Total (95% CI)		**8657**		**8642**	**100.0%**	**0.54 [0.42, 0.70]**
Total events	89		165			

Heterogeneity: $Chi^2 = 3.60$, $df = 3$ ($P = 0.31$); $I^2 = 17\%$

Test for overall effect: $Z = 4.81$ ($P < 0.00001$)



International Journal of Stroke
2017, Vol. 12(6) 589–596

ESC
European Society of Cardiology

The Task Force for the diagnosis and 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

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)

Recommendations for secondary stroke prevention in AF patients after acute ischaemic stroke	Class[a]	Level[b]
In AF patients with an ischaemic stroke or TIA, long-term secondary prevention of stroke using OAC is recommended if there is no strict contraindication to OAC use, with a preference for NOACs over VKAs in NOAC-eligible patients.[1125–1130]	I	A
In AF patients presenting with acute ischaemic stroke, very early anticoagulation (<48 h) using UFH, LMWH, or VKAs is not recommended.[1095]	III	B

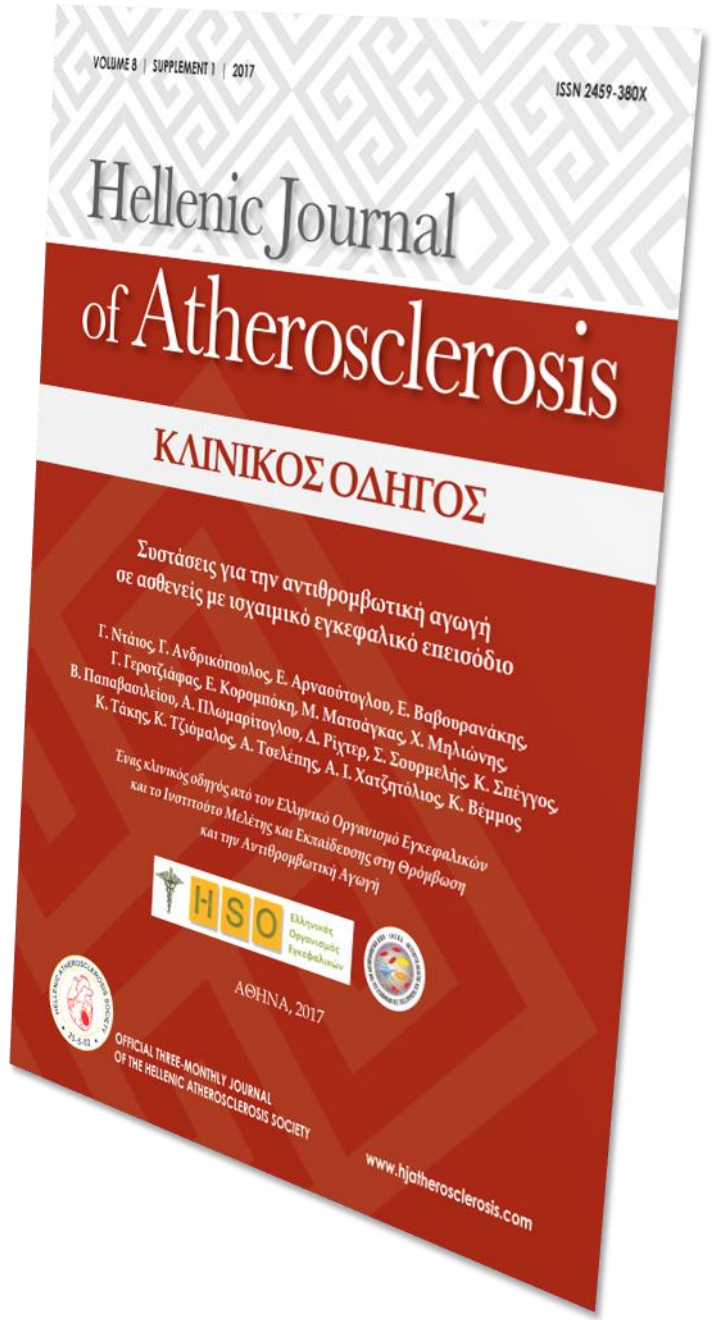
European Heart Journal (2020) **42**, 373–498

10. Οι αλληλεπιδράσεις της νταμπιγκαντράνης, ριβαροξαμπάνης, απιξαμπάνης ή εντοξαμπάνης και με φάρμακα και τροφές είναι πολύ λιγότερες σε σχέση με τους ανταγωνιστές της βιταμίνης Κ (1Γ).

11. Σε ασθενείς υπό νταμπιγκαντράνη, ριβαροξαμπάνη απιξαμπάνη ή εντοξαμπάνη, δεν υπάρχει λόγος ελέγχου του INR και πρέπει να αποφεύγεται (1Γ).

13. Η αντιθρομβωτική αγωγή σε ασθενείς με κολπικό πτερυγισμό πρέπει να ακολουθεί τις ίδιες αρχές με την κολπική μαρμαρυγή (1Γ).

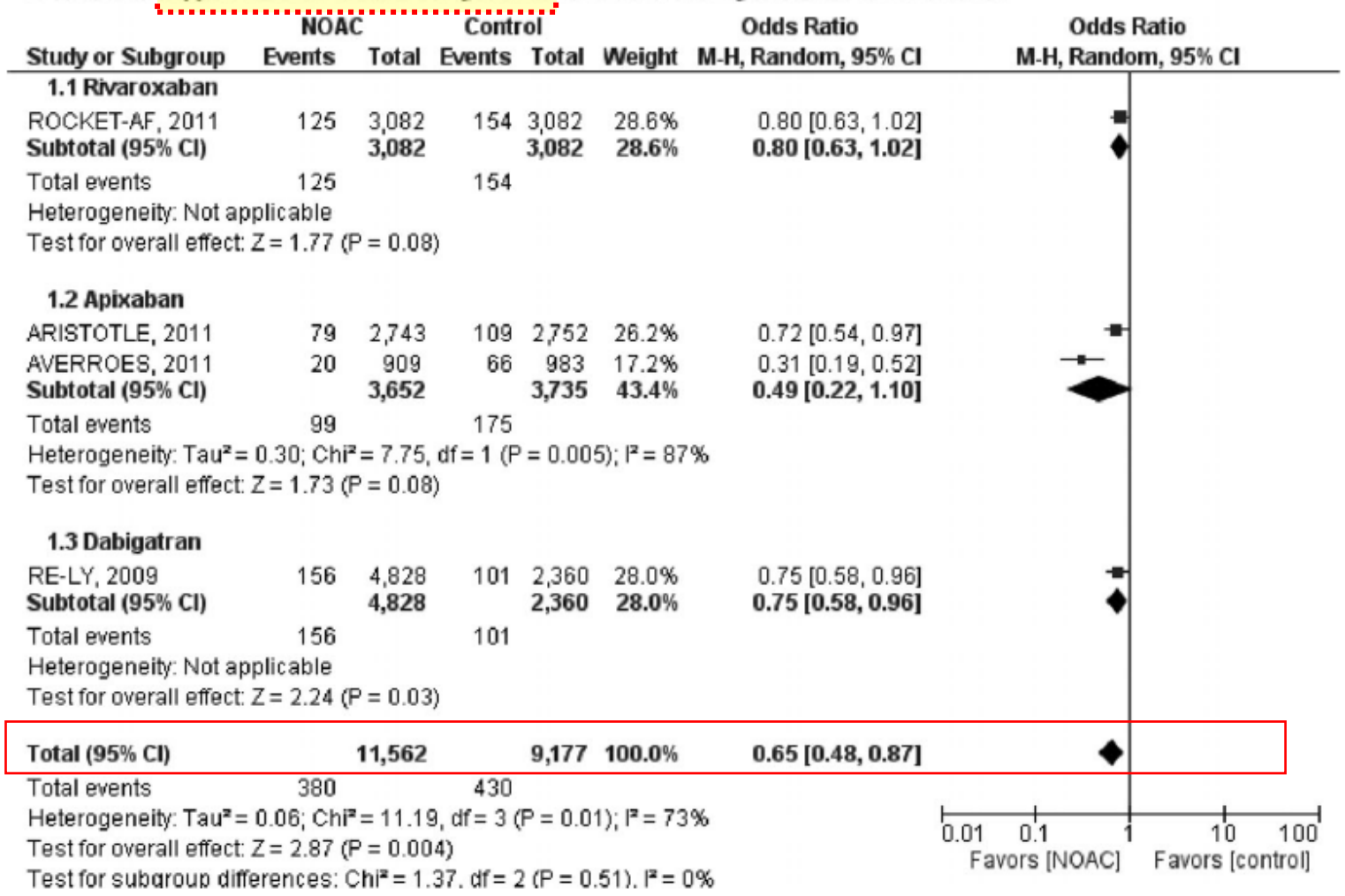
14. Η ηλικία του ασθενούς δεν αποτελεί αντένδειξη για την χορήγηση αντιπηκτικής αγωγής. Και οι ηλικιωμένοι ασθενείς με κολπική μαρμαρυγή πρέπει να λαμβάνουν αντιπηκτική αγωγή εάν δεν υπάρχει αντένδειξη (1Α).



New Oral Anticoagulants in Elderly Adults: Evidence from a Meta-Analysis of Randomized Trials

Partha Sardar, MD, Saurav Chatterjee, MD,† Shobhana Chaudhari, MD,* and Gregory Y. H. Lip, MD‡*

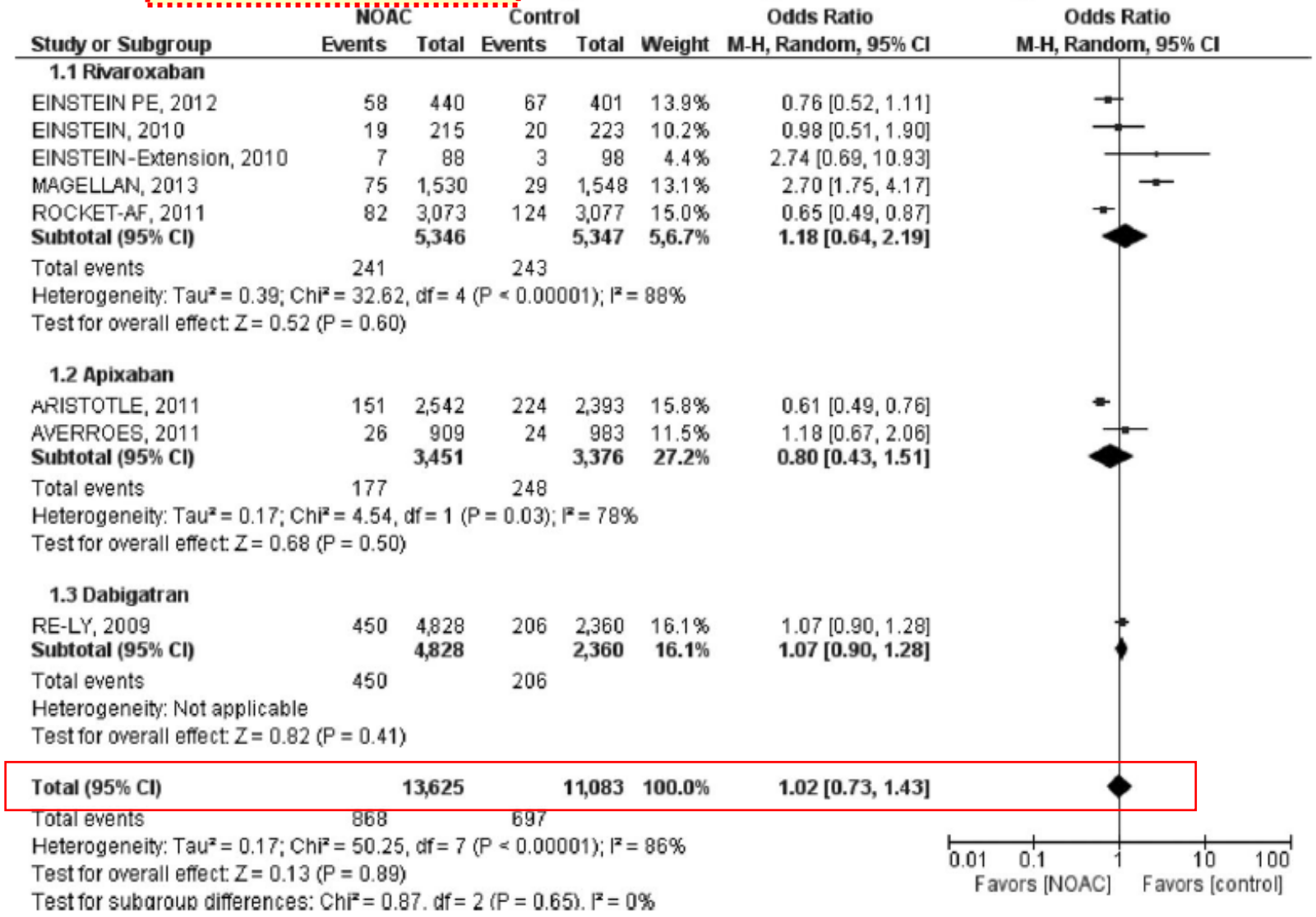
Patients aged more than 75 years: Stroke or systemic embolism



New Oral Anticoagulants in Elderly Adults: Evidence from a Meta-Analysis of Randomized Trials

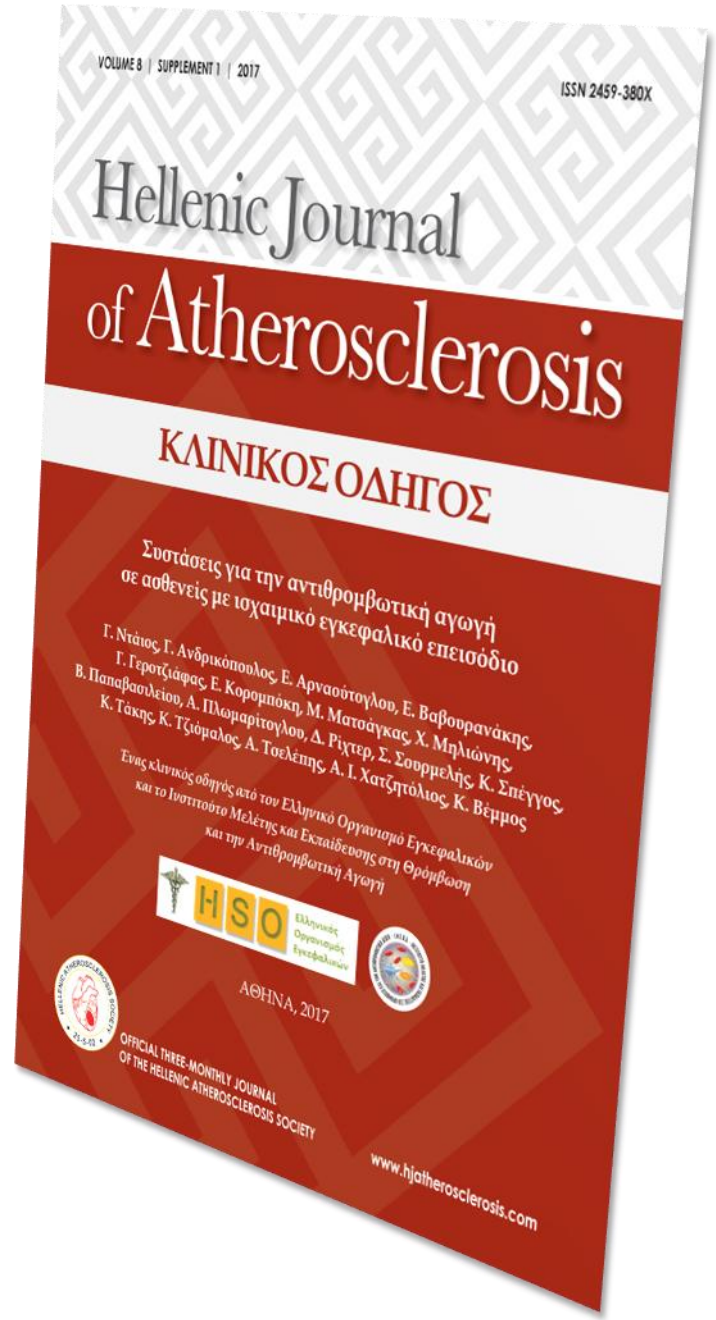
Partha Sardar, MD, Saurav Chatterjee, MD,† Shobhana Chaudhari, MD,* and Gregory Y. H. Lip,*

Patients aged more than 75 years: Major or clinically relevant bleeding

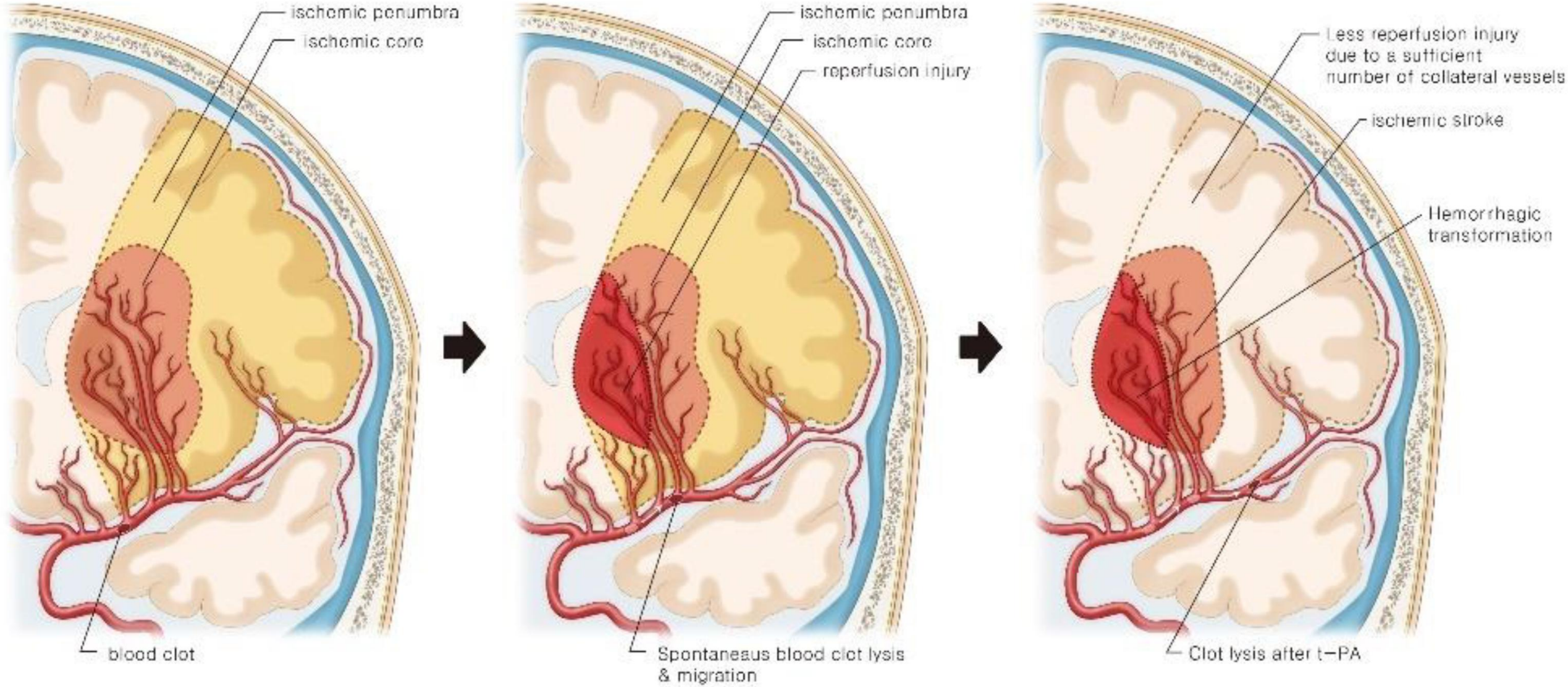


JAGS 62:857–864, 2014

9. Σε ασθενείς με οξύ ισχαιμικό εγκεφαλικό επεισόδιο και ένδειξη αντιπηκτικής αγωγής, η έναρξη της αντιπηκτικής αγωγής πρέπει να καθυστερεί για κάποιο χρονικό διάστημα ώστε να μειωθεί ο κίνδυνος αιμορραγικής μετατροπής του εγκεφαλικού εμφράκτου. Ο προτεινόμενος χρόνος έναρξης της αντιπηκτικής αγωγής είναι την 1η ημέρα σε ασθενείς με παροδικό ισχαιμικό εγκεφαλικό επεισόδιο, την 3η ημέρα σε ασθενείς με μικρής βαρύτητας ισχαιμικό εγκεφαλικό (NIHSS score κατά την εισαγωγή ≤ 8), την 6η ημέρα σε ασθενείς με μέτριας βαρύτητας ισχαιμικό εγκεφαλικό (NIHSS score κατά την εισαγωγή 8-16) και την 12η ημέρα σε ασθενείς με υψηλής βαρύτητας ισχαιμικό εγκεφαλικό (NIHSS score κατά την εισαγωγή ≥ 16) (2Γ). Στο μεσοδιάστημα ο ασθενής θα πρέπει να λαμβάνει ασπιρίνη.



Hemorrhagic Transformation of Infarction



<Before visiting the hospital>

<At the time of hospital visit>

<After using thrombolytic agent>

Timing of anticoagulation after recent ischaemic stroke in patients with atrial fibrillation

It is suspected (though not supported by evidence) that early initiation of anticoagulation might exacerbate or cause parenchymal haemorrhage, with potentially serious clinical consequences.[18] This concern has led clinicians to delay anticoagulation, although the independent contribution of haemorrhagic transformation of the infarct to clinical worsening remains uncertain[19] and evidence from randomised controlled trials is not available.

Lancet Neurol 2019; 18: 117–26

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

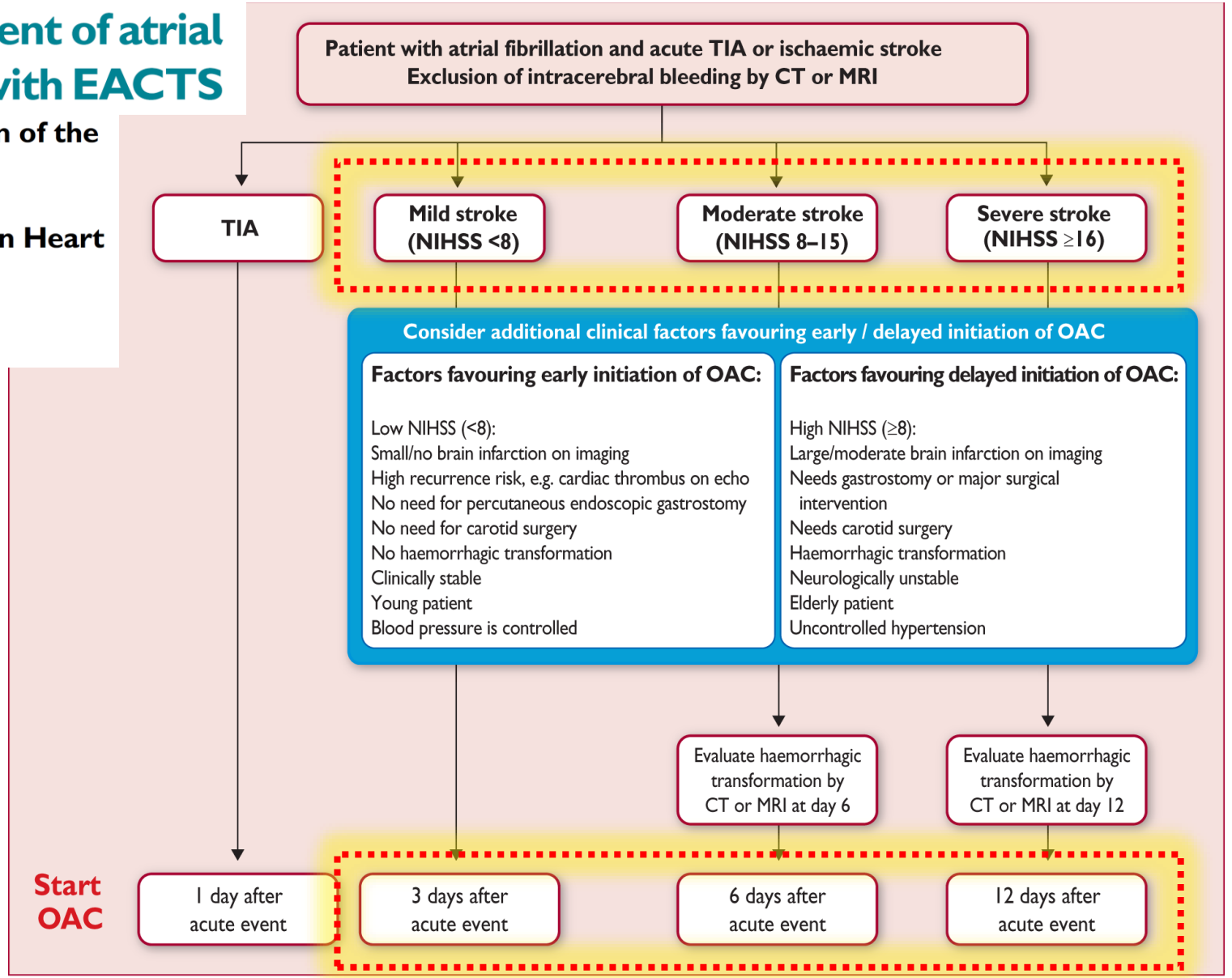
The Task Force for the management of atrial fibrillation of the European Society of Cardiology (ESC)

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

Endorsed by the European Stroke Organisation (ESO)

European Heart Journal (2016) **37**, 2893–2962

"1-3-6-12–day rule"



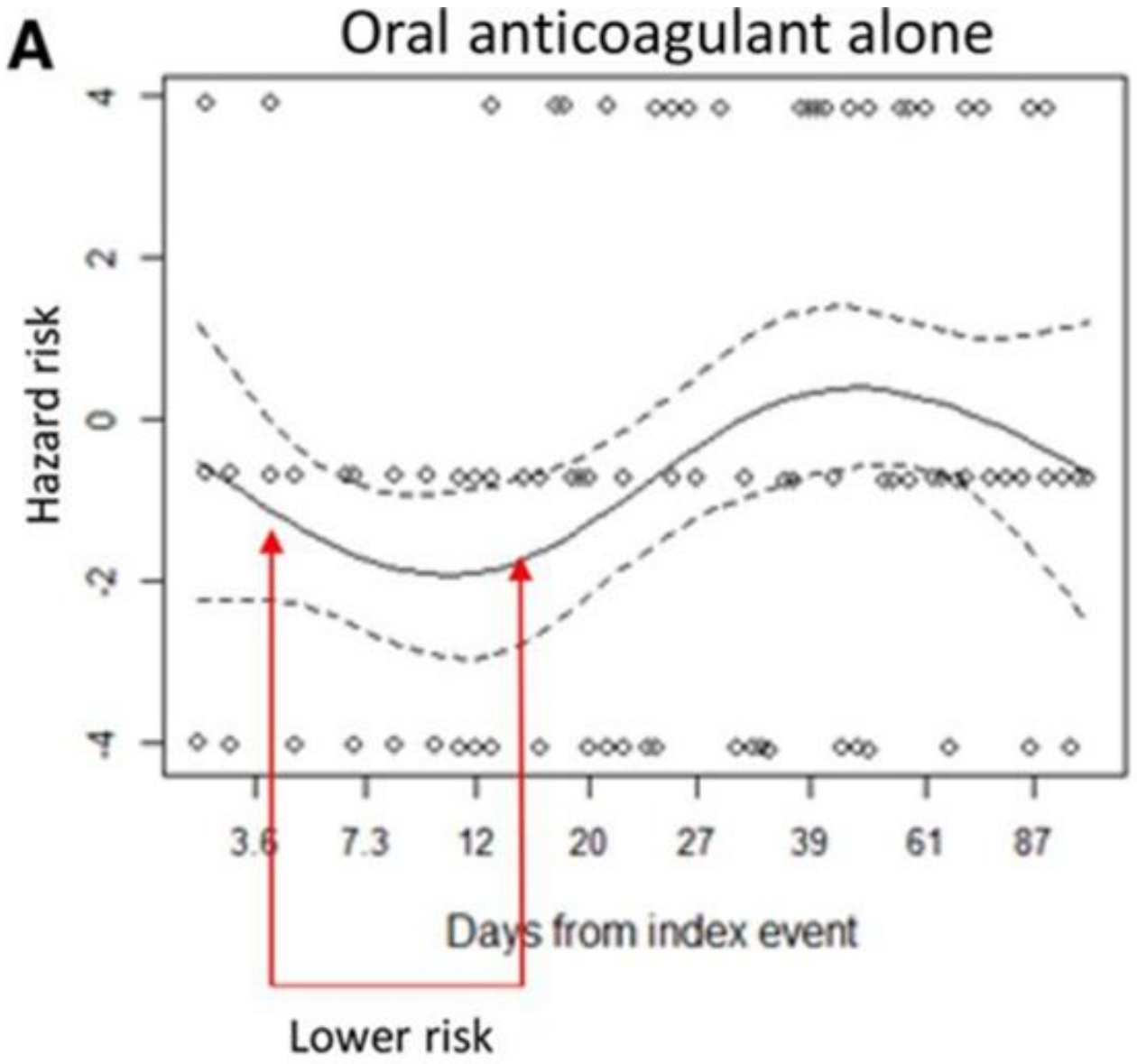
AF = atrial fibrillation; CT = computed tomography; NIHSS = National Institutes of Health stroke severity scale (available at http://www.strokecenter.org/wp-content/uploads/2011/08/NIH_Stroke_Scale.pdf); OAC = oral anticoagulation; TIA = transient ischaemic attack

*Initiation or continuation of anticoagulation in atrial fibrillation patients after a stroke or transient ischaemic attack. This approach is based on **consensus** rather than prospective data.*

Early Recurrence and Cerebral Bleeding in Patients With Acute Ischemic Stroke and Atrial Fibrillation

Effect of Anticoagulation and Its Timing: The RAF Study

Prospective ***Observational*** Study
1.029 patients



Time of Initiating Anticoagulant Treatment	All Outcome Events, HR (95% CI)	Ischemic Outcome Events, HR (95% CI)	Hemorrhagic Outcome Events, HR (95% CI)
Within 7 days	1.35 (0.82–2.22)	1.19 (0.76–1.81)	1.72 (0.75–4.00)
Within 14 days	0.71 (0.47–2.50)	0.61 (0.35–1.06)	1.81 (0.75–4.00)
Between 2 and 14 days	0.67 (0.39–1.14)	0.59 (0.27–1.29)	0.72 (0.29–1.78)
Between 3 and 14 days	0.58 (0.33–1.03)	0.50 (0.23–1.12)	0.51 (0.18–1.47)
Between 4 and 14 days	0.53 (0.30–0.93)	0.43 (0.19–0.97)	0.39 (0.12–1.19)
Between 5 and 14 days	0.47 (0.25–0.87)	0.40 (0.17–0.86)	0.33 (0.10–1.15)
Between 6 and 14 days	0.42 (0.22–0.81)	0.30 (0.11–0.80)	0.37 (0.10–1.37)
Between 7 and 14 days	0.43 (0.23–0.83)	0.25 (0.10–0.65)	0.42 (0.11–1.51)
Between 8 and 14 days	0.42 (0.21–0.87)	0.24 (0.08–0.69)	0.56 (0.15–2.12)
Between 9 and 14 days	0.43 (0.21–0.86)	0.22 (0.07–0.62)	0.48 (0.13–1.78)
Between 10 and 14 days	0.30 (0.13–0.71)	0.18 (0.05–0.63)	0.20 (0.02–1.75)
Between 11 and 14 days	0.29 (0.12–0.71)	0.16 (0.05–0.56)	0.24 (0.03–1.77)
Between 12 and 14 days	0.21 (0.08–0.57)	0.12 (0.03–0.45)	0.27 (0.03–2.17)
Between 13 and 14 days	0.38 (0.13–1.08)	0.21 (0.05–0.85)	0.36 (0.04–3.22)
Day 14	0.38 (0.13–1.11)	0.20 (0.05–0.85)	0.36 (0.04–3.22)

Stroke. 2015;46:2175-2182.

AHA/ASA Guideline

Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke

A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association

Endorsed by the Society for Academic Emergency Medicine and The Neurocritical Care Society

American Stroke Association.
A division of the American Heart Association.

6.6.2. Atrial Fibrillation	COR	LOE	New, Revised, or Unchanged
1. For most patients with an AIS in the setting of atrial fibrillation, it is reasonable to initiate oral anticoagulation between 4 and 14 days after the onset of neurological symptoms.	IIa	B-NR	Recommendation revised from 2014 Secondary Prevention.

Antithrombotic treatment for secondary prevention of stroke and other thromboembolic events in patients with stroke or transient ischemic attack and non-valvular atrial fibrillation:

A European Stroke Organisation guideline

Recommendations. We cannot make recommendations about the optimal time for initiating anticoagulation treatment in patients with acute ischemic stroke based on randomised trials. We encourage inclusion of patients in ongoing randomised controlled trials testing the efficacy and safety of early anticoagulation to answer this question.

Quality of evidence: Low

Strength of recommendation: Weak

European Stroke Journal
2019, Vol. 4(3) 198–223

Antithrombotic treatment for secondary prevention of stroke and other thromboembolic events in patients with stroke or transient ischemic attack and non-valvular atrial fibrillation:

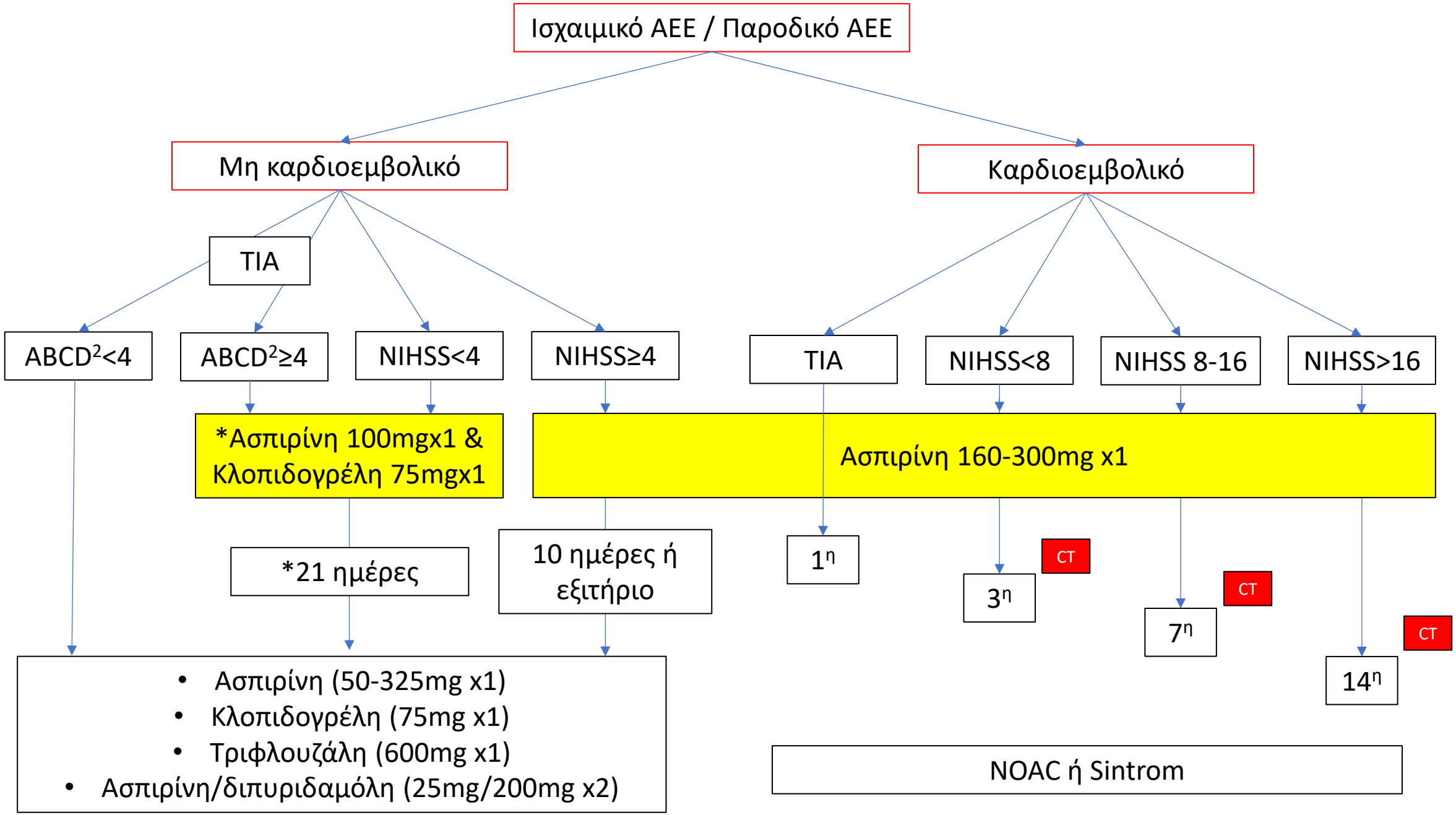
A European Stroke Organisation guideline

Expert opinion (Delphi vote: 6/7 agree, 1/7 disagree). We suggest antiplatelet therapy in the first 48 h after ischemic stroke associated with AF.

We consider it reasonable to start anticoagulant therapy at day 3 or 4 from the index stroke in patients with mild stroke and small infarcts (<1.5 cm) and at day 7 for moderate infarcts.

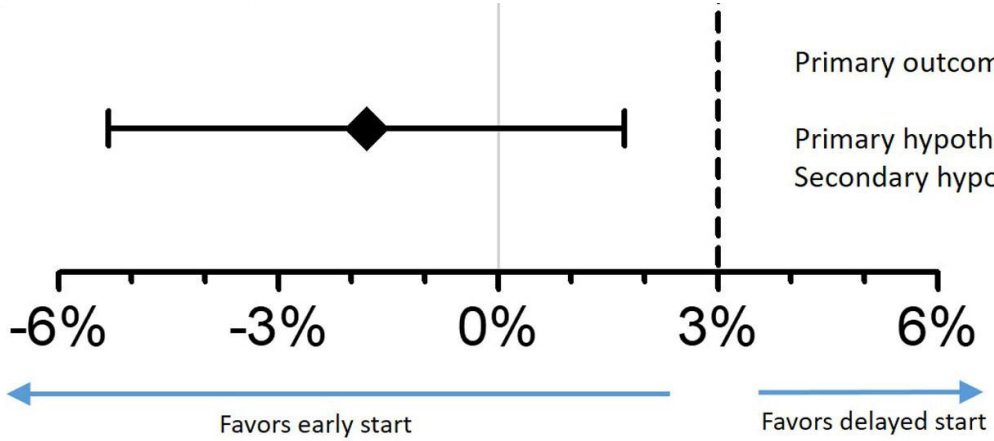
For large infarcts, anticoagulation treatment might be best delayed for 14 days after the index stroke.

European Stroke Journal
2019, Vol. 4(3) 198–223


Ισχαιμικό ΑΕΕ / Παροδικό ΑΕΕ
Μη καρδιοεμβολικό
Καρδιοεμβολικό
TIA
ABCD²<4
ABCD²≥4
NIHSS<4
NIHSS≥4
TIA
NIHSS<8
NIHSS 8-16
NIHSS>16
*Ασπιρίνη 100mgx1 & Κλοπιδογρέλη 75mgx1
Ασπιρίνη 160-300mg x1
*21 ημέρες
10 ημέρες ή εξιτήριο
1η
3η
CT
7η
CT
14η
CT
• Ασπιρίνη (50-325mg x1)
• Κλοπιδογρέλη (75mg x1)
• Τριφλουζάλη (600mg x1)
• Ασπιρίνη/διπυριδαμόλη (25mg/200mg x2)
NOAC ή Sintrom

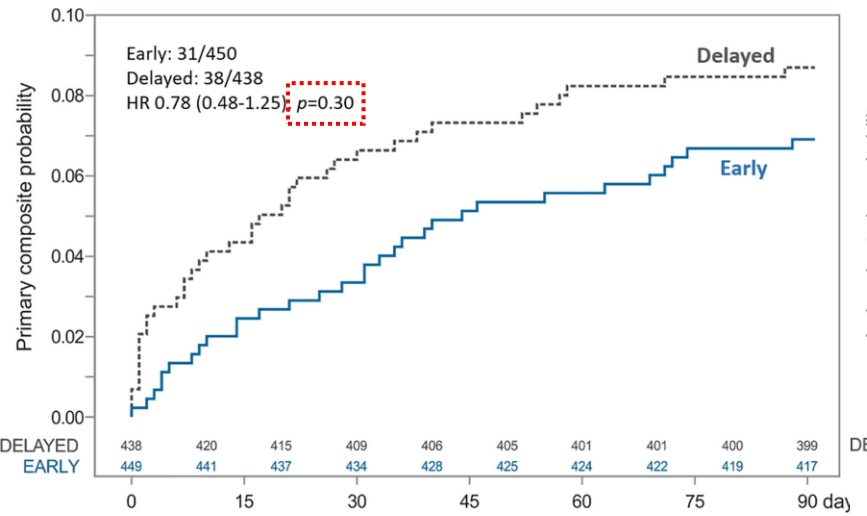
Early Versus Delayed Non–Vitamin K Antagonist Oral Anticoagulant Therapy After Acute Ischemic Stroke in Atrial Fibrillation (TIMING): A Registry-Based Randomized Controlled Noninferiority Study

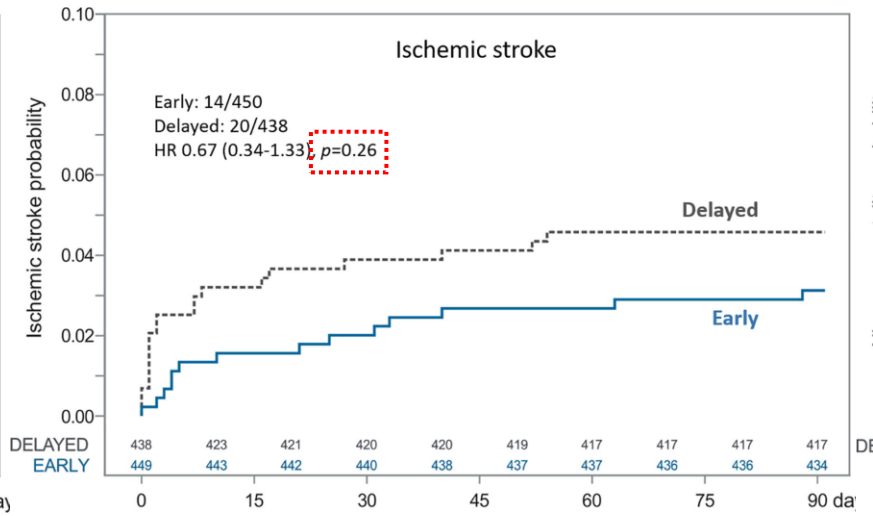
TIMING

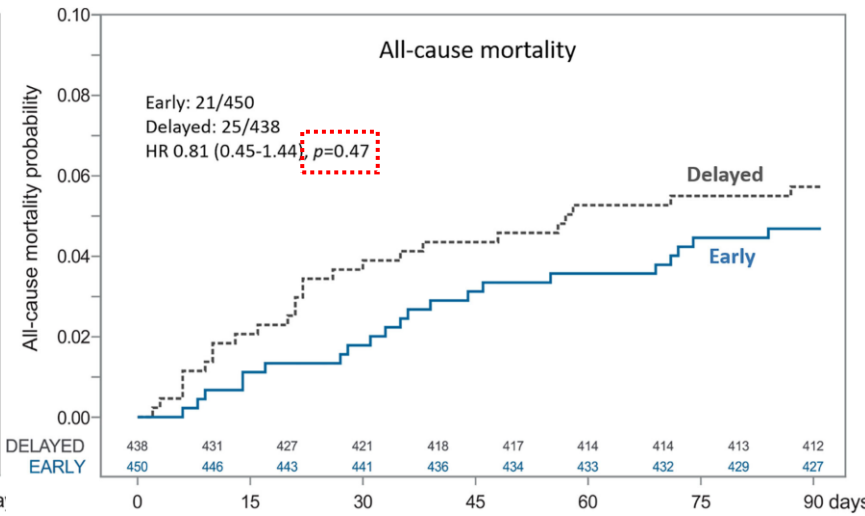


Primary outcome event difference -1.79%; 95% CI -5.31 to 1.74%

Primary hypothesis non-inferiority, p = 0.004
Secondary hypothesis superiority, p = 0.380







Circulation. 2022;146:1056–1066

Early versus Later Anticoagulation for Stroke with Atrial Fibrillation

ELAN

A

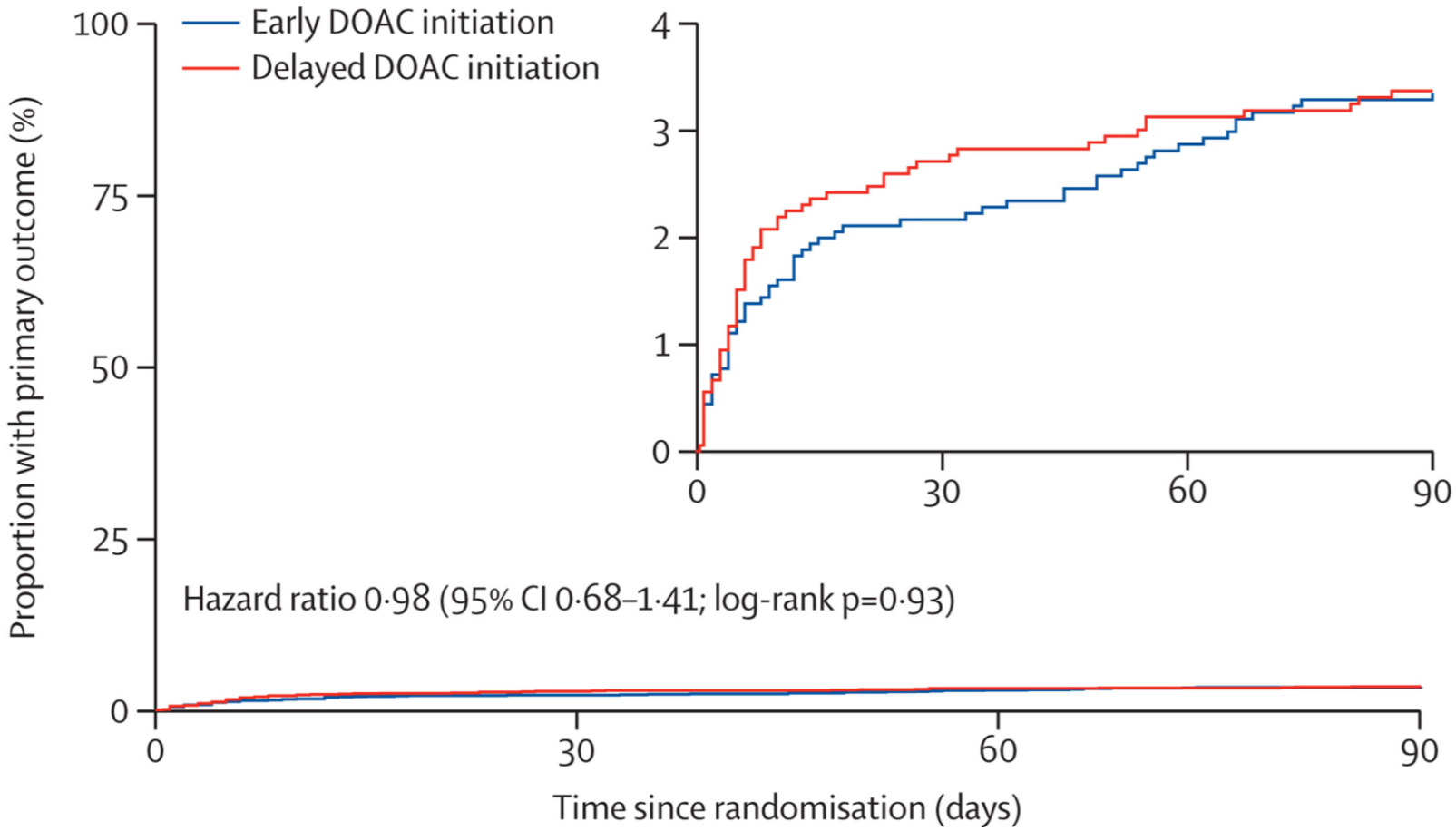
Outcome within 30 Days	Early-Treatment Group (N=984)	Later-Treatment Group (N=991)	Adjusted Risk Difference (95% CI)
	no. of events (%)		*percentage points*
Primary-outcome event	29 (2.9)	41 (4.1)	−1.18 (−2.84 to 0.47)
Major extracranial bleeding	3 (0.3)	5 (0.5)	−0.25 (−0.90 to 0.41)
Symptomatic intracranial hemorrhage	2 (0.2)	2 (0.2)	0.01 (−0.52 to 0.53)
Recurrent ischemic stroke	14 (1.4)	25 (2.5)	−1.14 (−2.41 to 0.13)
Systemic embolism	4 (0.4)	9 (0.9)	−0.55 (−1.34 to 0.23)
Death from vascular cause	11 (1.1)	10 (1.0)	0.13 (−0.84 to 1.09)

B

Outcome within 90 Days	Early-Treatment Group (N=968)	Later-Treatment Group (N=965)	Adjusted Risk Difference (95% CI)
	no. of events (%)		*percentage points*
Primary-outcome event	36 (3.7)	54 (5.6)	−1.92 (−3.82 to −0.02)
Major extracranial bleeding	3 (0.3)	8 (0.8)	−0.61 (−1.37 to 0.14)
Symptomatic intracranial hemorrhage	2 (0.2)	2 (0.2)	0.00 (−0.54 to 0.53)
Recurrent ischemic stroke	18 (1.9)	30 (3.1)	−1.29 (−2.72 to 0.13)
Systemic embolism	4 (0.4)	10 (1.0)	−0.70 (−1.53 to 0.13)
Death from vascular cause	17 (1.8)	16 (1.7)	0.07 (−1.13 to 1.27)

Optimal timing of anticoagulation after acute ischaemic stroke with atrial fibrillation (OPTIMAS): a multicentre, blinded-endpoint, phase 4, randomised controlled trial

OPTIMAS



Interpretation

Early DOAC initiation within 4 days after ischaemic stroke associated with AF was non-inferior to delayed initiation for the composite outcome of ischaemic stroke, intracranial haemorrhage, unclassifiable stroke, or systemic embolism at 90 days.

Our findings *do not support* the current common and guideline-supported practice *of delaying DOAC* initiation after ischaemic stroke with atrial fibrillation.

Lancet 2024; 404: 1731–41

Original Investigation

March 31, 2025

START

Optimal Delay Time to Initiate Anticoagulation After Ischemic Stroke in Atrial Fibrillation

A Pragmatic, Response-Adaptive Randomized Clinical Trial

Conclusions and Relevance

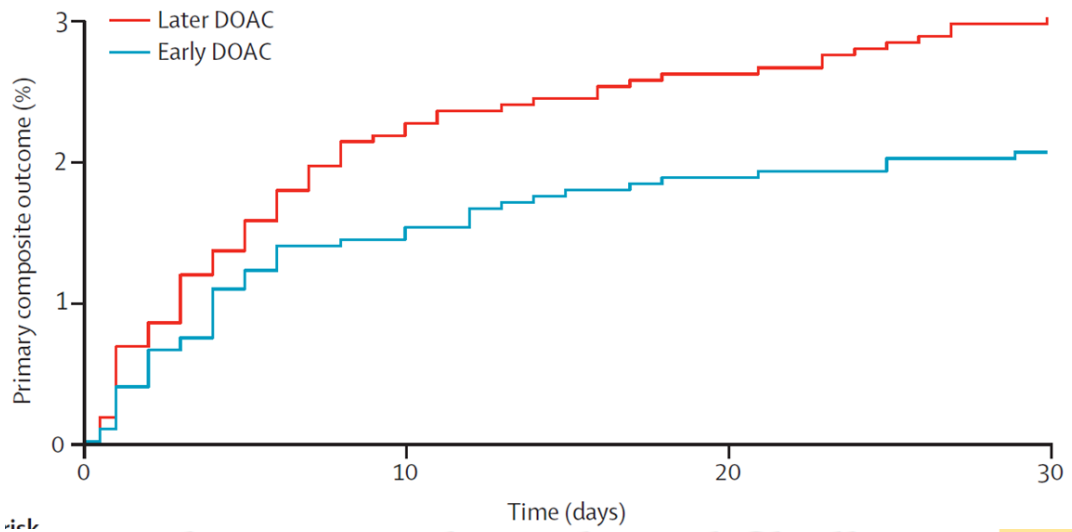
A clearly superior day to initiate use of a direct oral anticoagulant for secondary stroke prevention in patients with atrial fibrillation was not identified, but ***the evidence suggests that initiating use of a direct oral anticoagulant earlier is better*** than at later times within the first 2 weeks after stroke onset.

JAMA Neurol. Published online March 31, 2025. doi:10.1001/jamaneurol.2025.0285

Collaboration on the optimal timing of anticoagulation after ischaemic stroke and atrial fibrillation: a systematic review and prospective individual participant data meta-analysis of randomised controlled trials (CATALYST)

CATALYST

Findings We identified four eligible trials: TIMING (NCT02961348), ELAN (NCT03148457), OPTIMAS (NCT03759938), and START (NCT03021928). After excluding participants who opted out of data sharing or were not randomly assigned to DOAC initiation within 4 days or at day 5 or later, we included 5441 participants (mean age 77·7 years [SD 10·0], 2472 [45·4%] women, median National Institutes of Health Stroke Scale 5 [IQR 3–10]) in the individual patient data meta-analysis.



Interpretation For people with acute ischaemic stroke and atrial fibrillation, early DOAC initiation (within 4 days) reduced the risk of the composite outcome of recurrent ischaemic stroke, symptomatic intracerebral haemorrhage, or unclassified stroke within 30 days. These findings support early DOAC initiation in clinical practice.

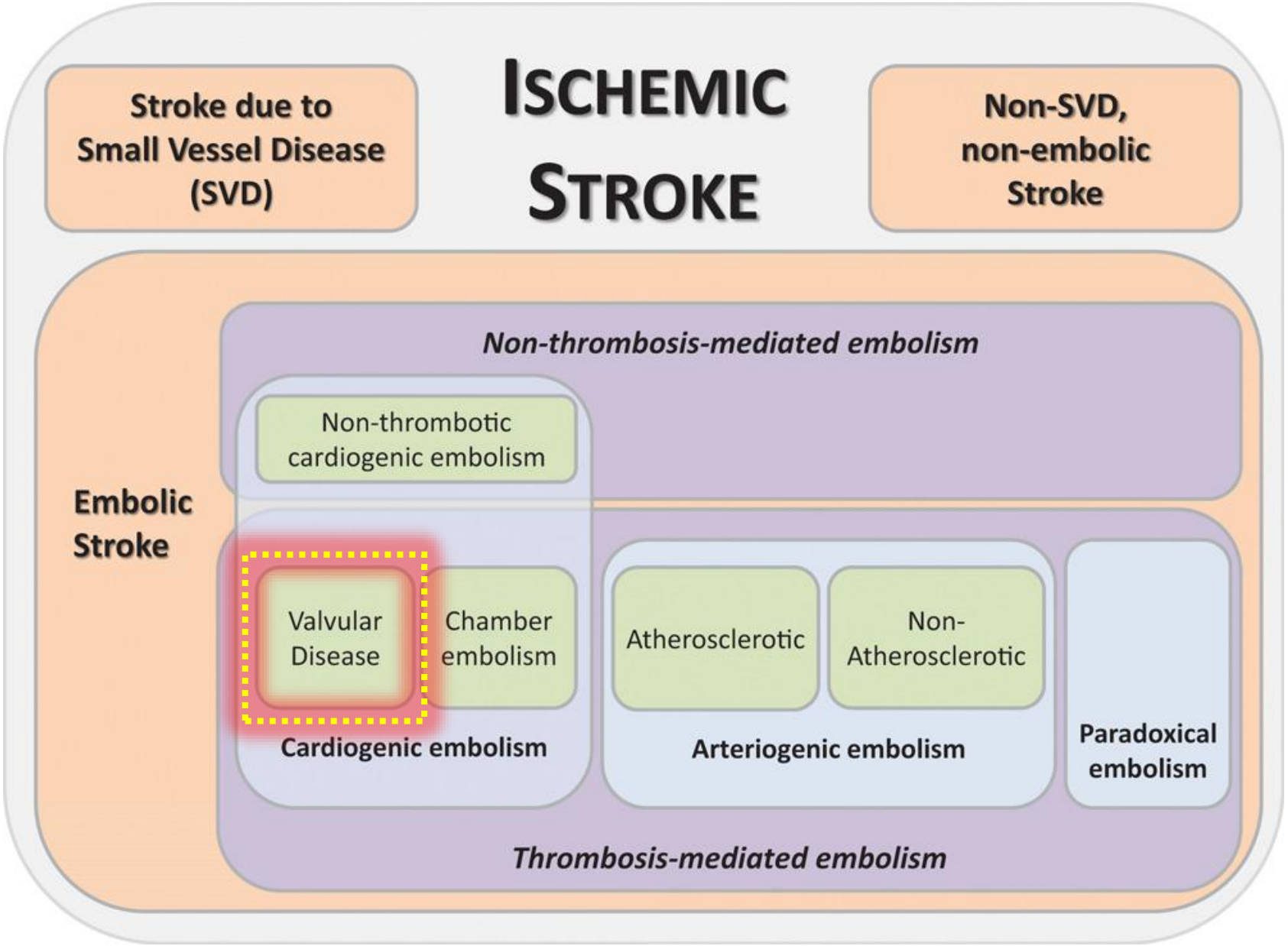
Lancet 2025; 406: 43–51

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association

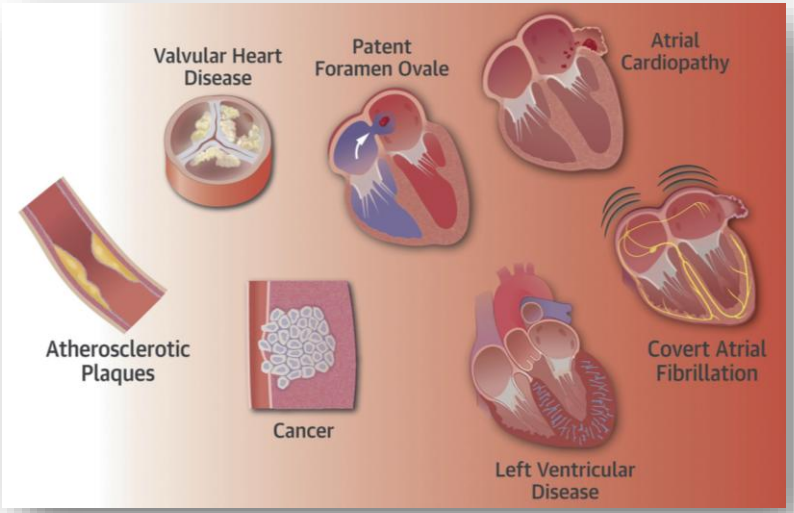
COR	LOE	Recommendations
2a	A	1. In carefully selected (eg, milder severity) patients with AIS with atrial fibrillation, a strategy of early oral anticoagulation poststroke is low risk and is reasonable compared with a strategy of delayed anticoagulation, although the efficacy of early anticoagulation for prevention of early recurrent stroke is not established.[1–3]

Stroke. 2026 Jan 26. Epub ahead of print.

Ischemic stroke is an etiologically heterogeneous syndrome



Ntaios G, Hart RG. Embolic stroke. Circulation. 2017;136:2403e2405.

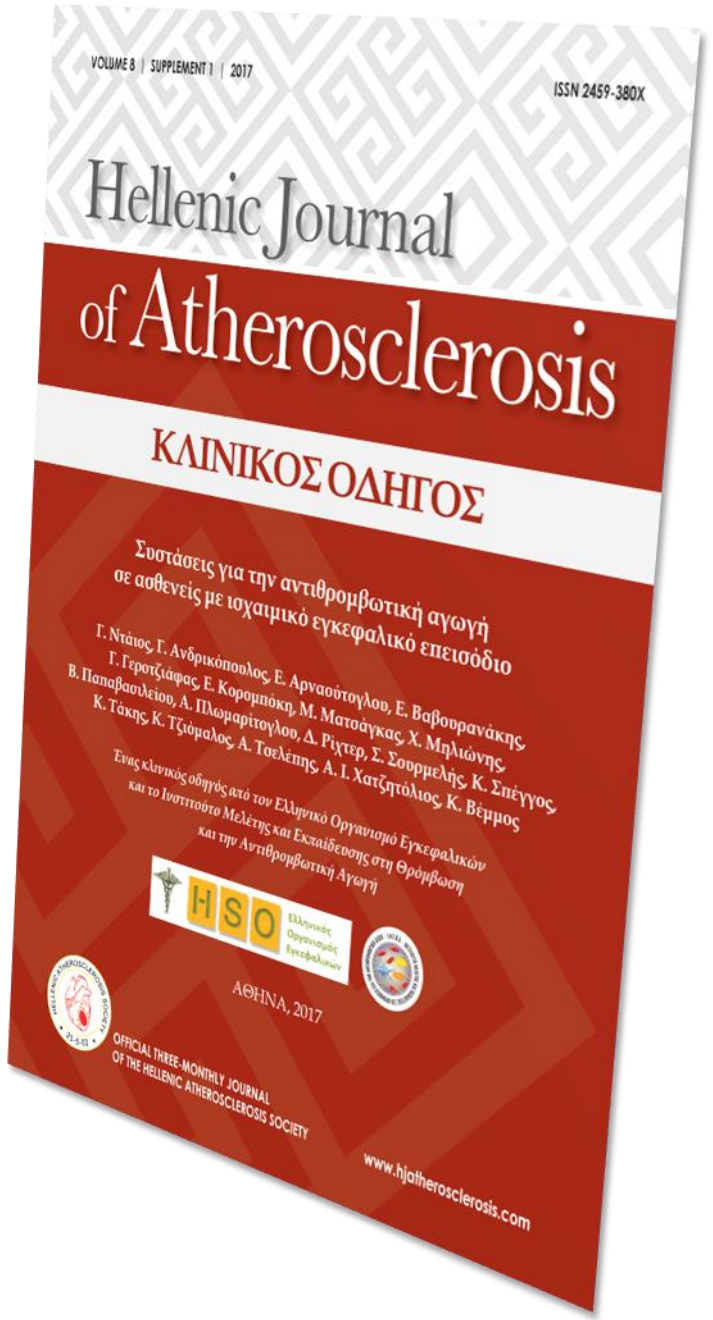


Ntaios G. Embolic Stroke of Undetermined Source: JACC Review Topic of the Week. J Am Coll Cardiol. 2020 Jan 28;75(3):333-340.

In order to optimize the ***secondary prevention strategy*** in a patient with ischemic stroke, it is rational to identify the underlying etiologic pathology.

12. Ασθενείς με ισχαιμικό (εγκατεστημένο ή παροδικό) εγκεφαλικό επεισόδιο και παροξυσμική, εμμένουσα ή χρόνια κολπική μαρμαρυγή σε έδαφος βαλβιδοπάθειας της μιτροειδούς ρευματικής αιτιολογίας πρέπει να λαμβάνουν ανταγωνιστές της βιταμίνης Κ (ασενοκουμαρόλη ή βαρφαρίνη) με στόχο εύρος INR 2,0-3,0 (1Γ).

15. Σε ασθενείς με ισχαιμικό (εγκατεστημένο ή παροδικό) εγκεφαλικό επεισόδιο και μεταλλική καρδιακή βαλβίδα πρέπει να χορηγείται ανταγωνιστής της βιταμίνης Κ (ασενοκουμαρόλη ή βαρφαρίνη). Οι ασθενείς πρέπει να προβαίνουν σε τακτικό έλεγχο του INR (τουλάχιστον άπαξ μηνιαίως) και τιτλοποίηση της δόσης με στόχο εύρος INR 2,5-3,5 ανάλογα με τον τύπο και τη θέση της βαλβίδας καθώς και την παρουσία προηγούμενου θρομβοεμβολικού επεισοδίου (1Α).



ESC
European Society of Cardiology

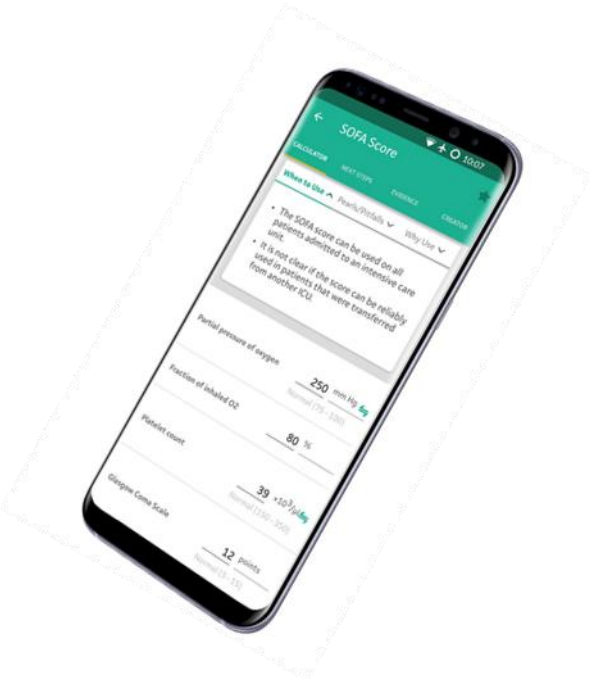
The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC)

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

2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)

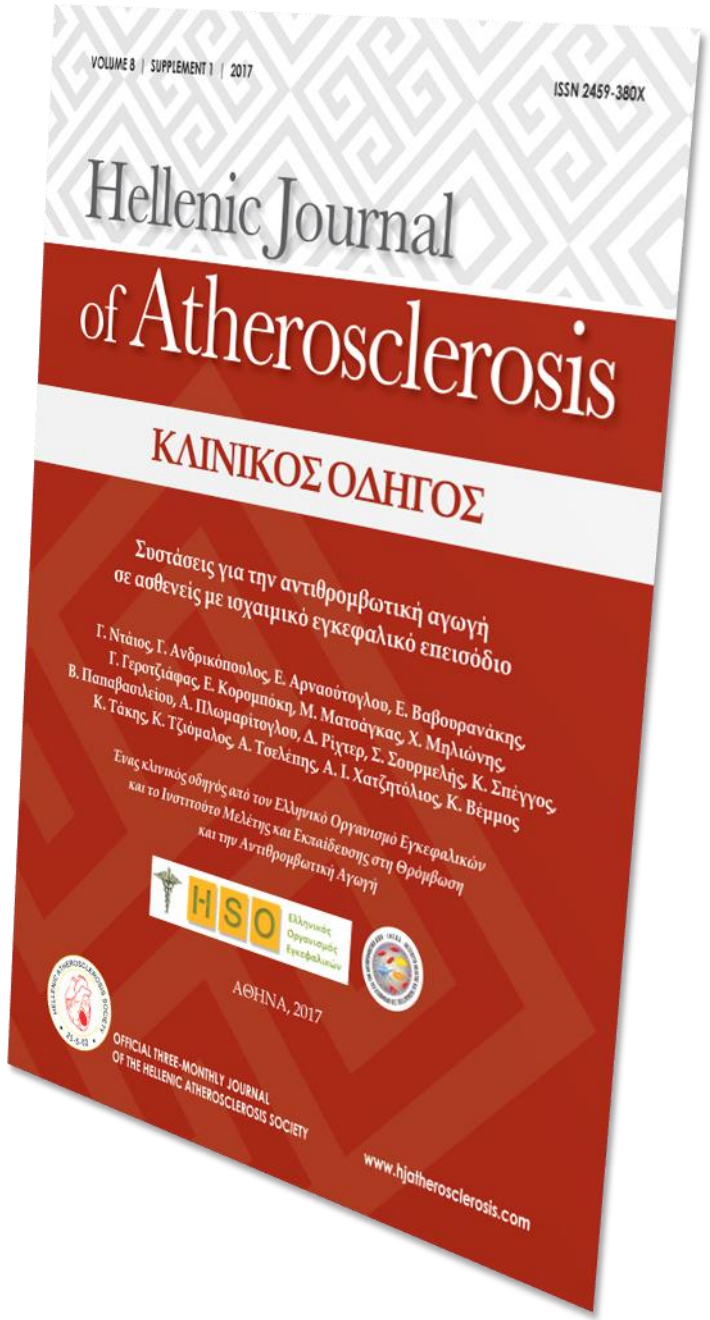
HAS-BLED score

Risk factors and definitions		Points awarded
H	**Uncontrolled hypertension** SBP >160 mmHg	1
A	**Abnormal renal and/or hepatic function** Dialysis, transplant, serum creatinine >200 µmol/L, cirrhosis, bilirubin > × 2 upper limit of normal, AST/ALT/ALP >3 × upper limit of normal	1 point for each
S	**Stroke** Previous ischaemic or haemorrhagic[a] stroke	1
B	**Bleeding history or predisposition** Previous major haemorrhage or anaemia or severe thrombocytopenia	1
L	**Labile INR[b]** TTR <60% in patient receiving VKA	1
E	**Elderly** Aged >65 years or extreme frailty	1
D	**Drugs or excessive alcohol drinking** Concomitant use of antiplatelet or NSAID; and/or excessive[c] alcohol per week	1 point for each
Maximum score		**9**



European Heart Journal (2020) **42**, 373–498

20. Συστήνεται η χορήγηση ηπαρίνης χαμηλού μοριακού βάρους σε προφυλακτική δόση σε ασθενείς με οξύ ισχαιμικό εγκεφαλικό επεισόδιο οι οποίοι δεν είναι περιπατητικοί και στους οποίους το όφελος της μείωσης του κινδύνου φλεβικής θρομβοεμβολής ξεπερνά την αύξηση του κινδύνου αιμορραγίας (1A).



European Stroke Organisation (ESO) guidelines for prophylaxis for venous thromboembolism in immobile patients with acute ischaemic stroke

EUROPEAN STROKE JOURNAL
European Stroke Journal
2016, Vol. 1(1) 6–19

Figure 3.4 The effect on prophylactic anticoagulants on PE during treatment

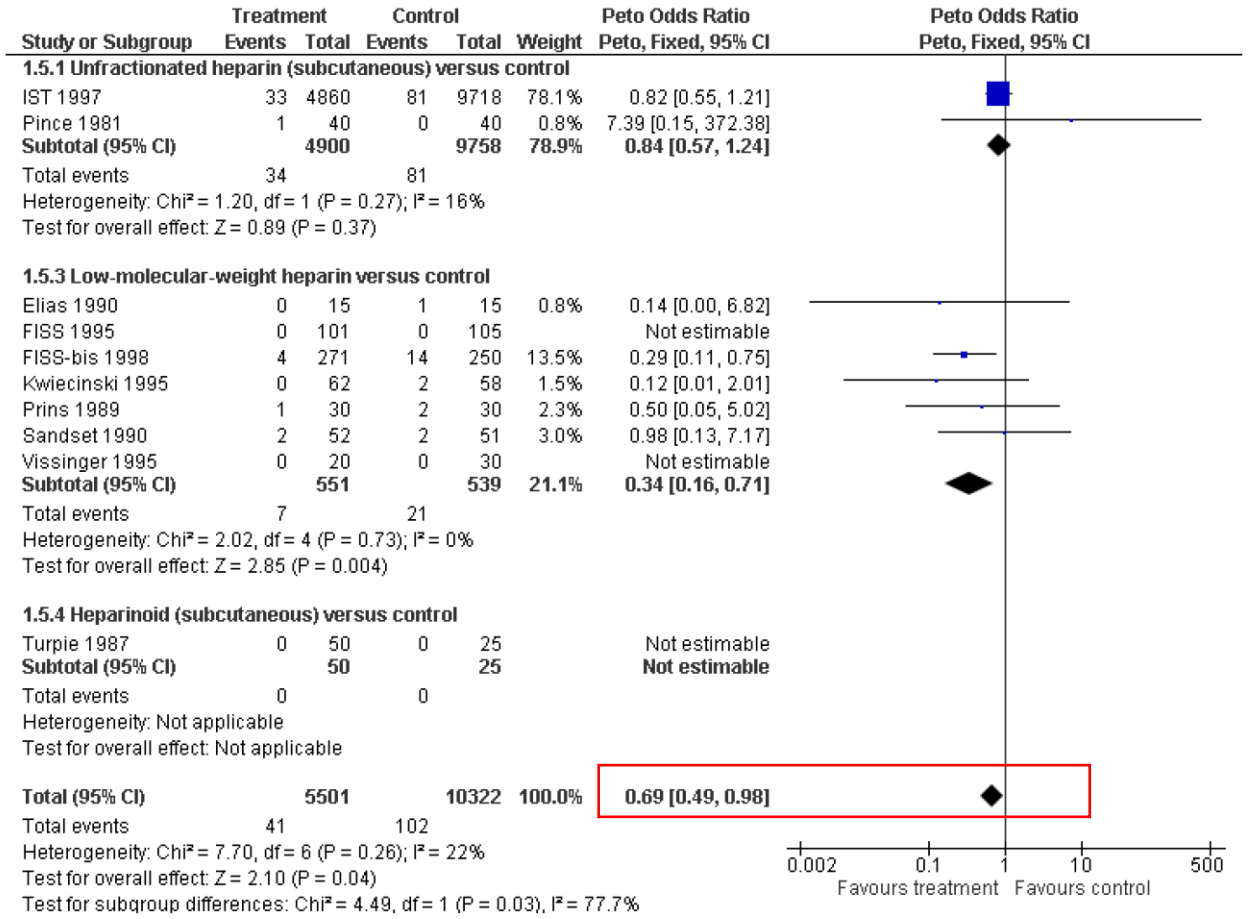
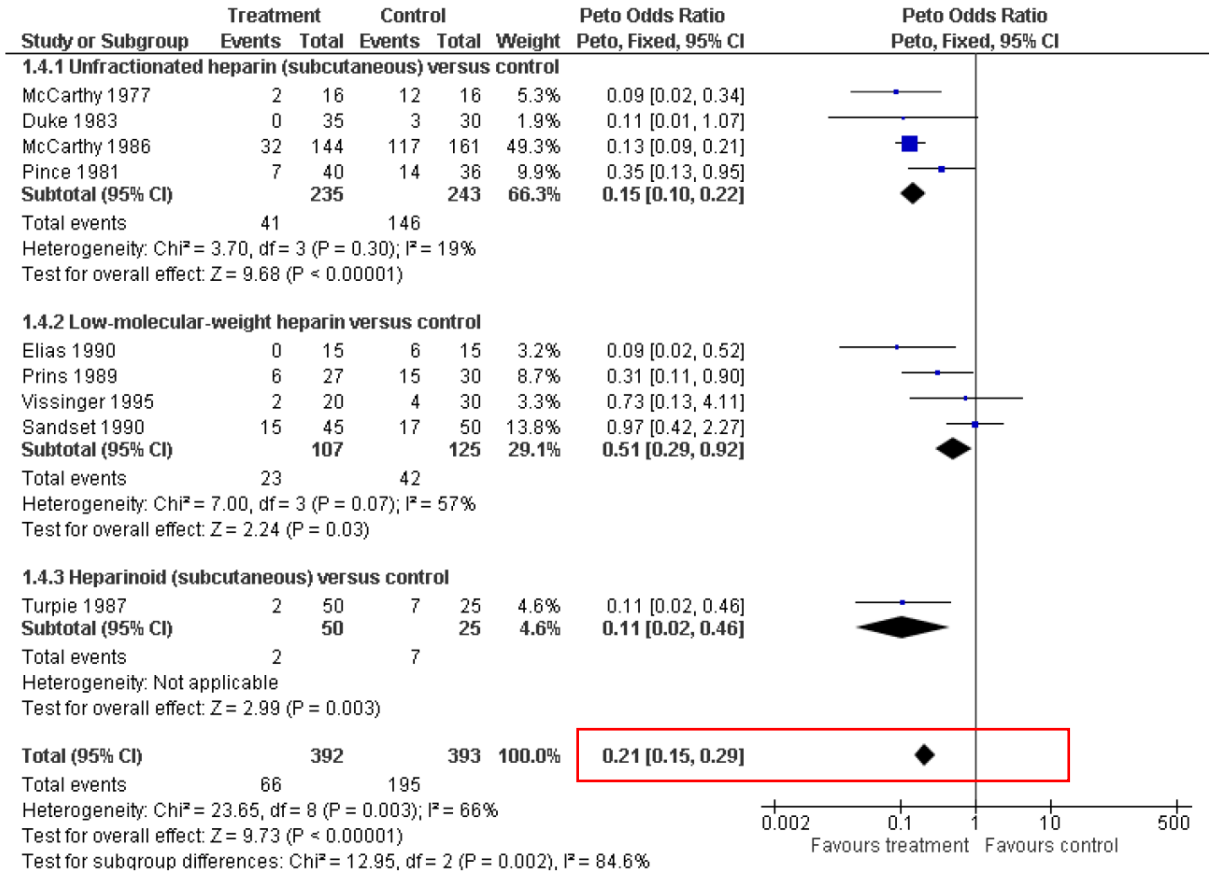


Figure 3.6 The effect on prophylactic anticoagulants on any DVT (including Isotope scanning) only during treatment



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European Stroke Journal
2016, Vol. 1(1) 6–19

Figure 3.3 The effect of prophylactic anticoagulation on symptomatic intracranial bleeding during treatment period

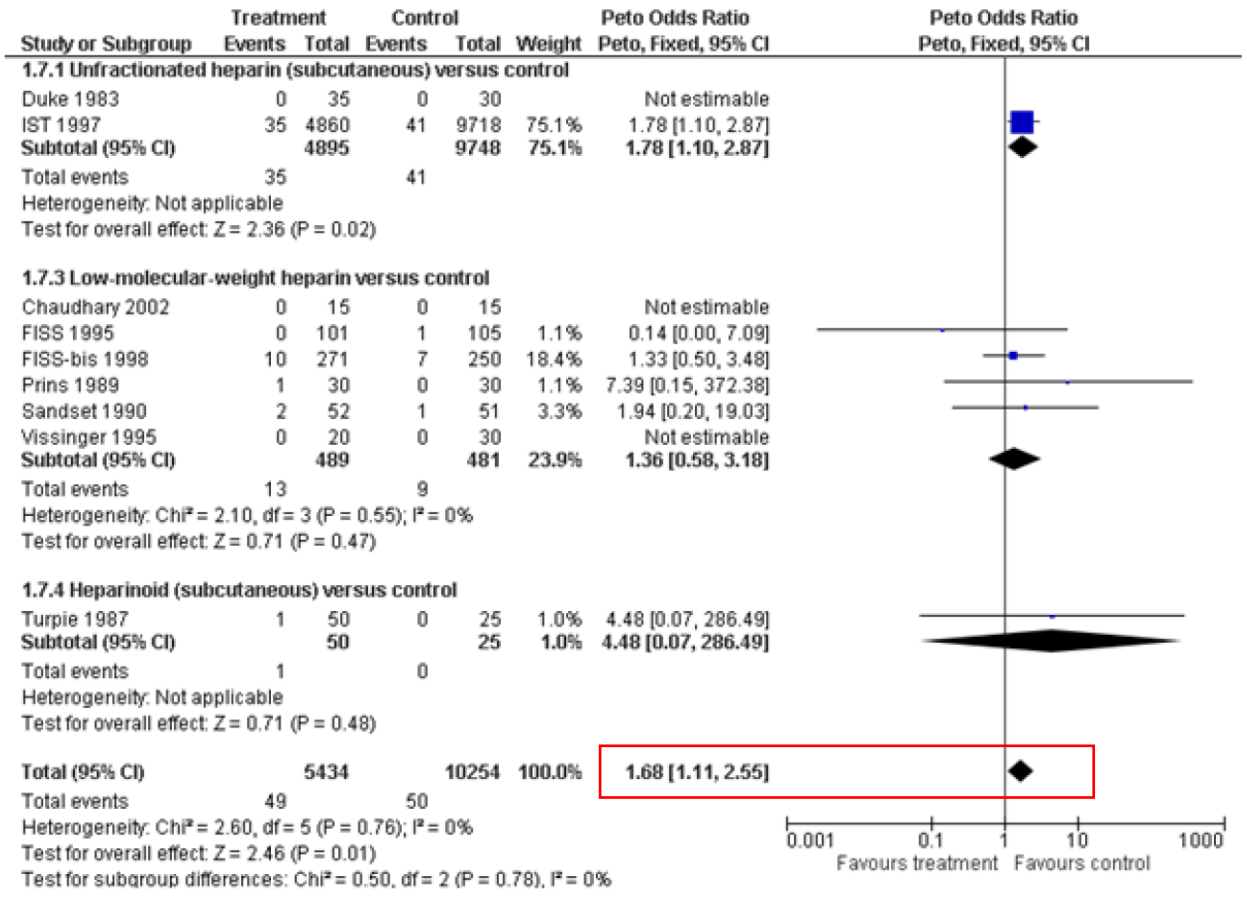
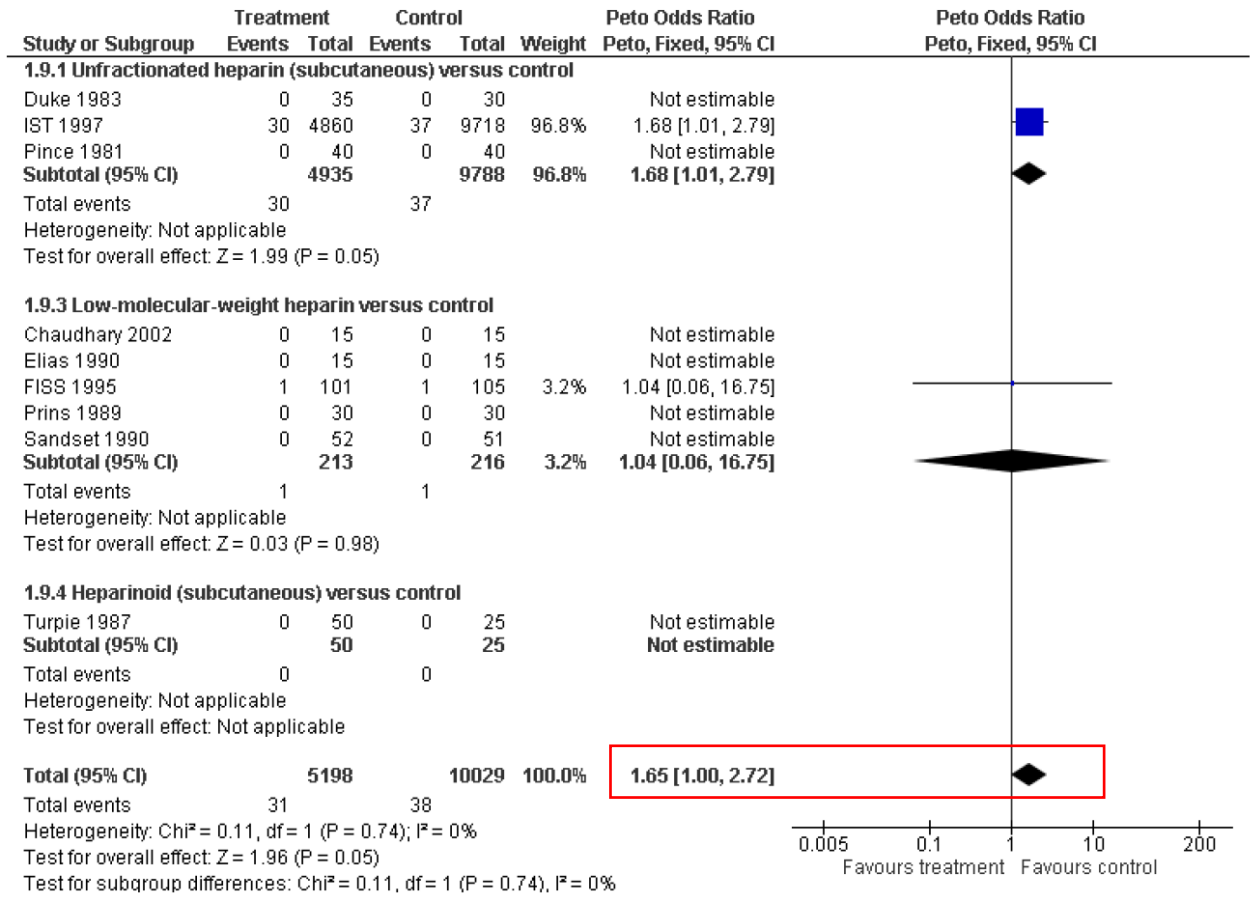


Figure 3.5 The effect of prophylactic anticoagulation on symptomatic extracranial bleeding during treatment period



European Stroke Organisation (ESO) guidelines for prophylaxis for venous thromboembolism in immobile patients with acute ischaemic stroke

Figure 3.2 The effect of prophylactic anticoagulants on death during treatment

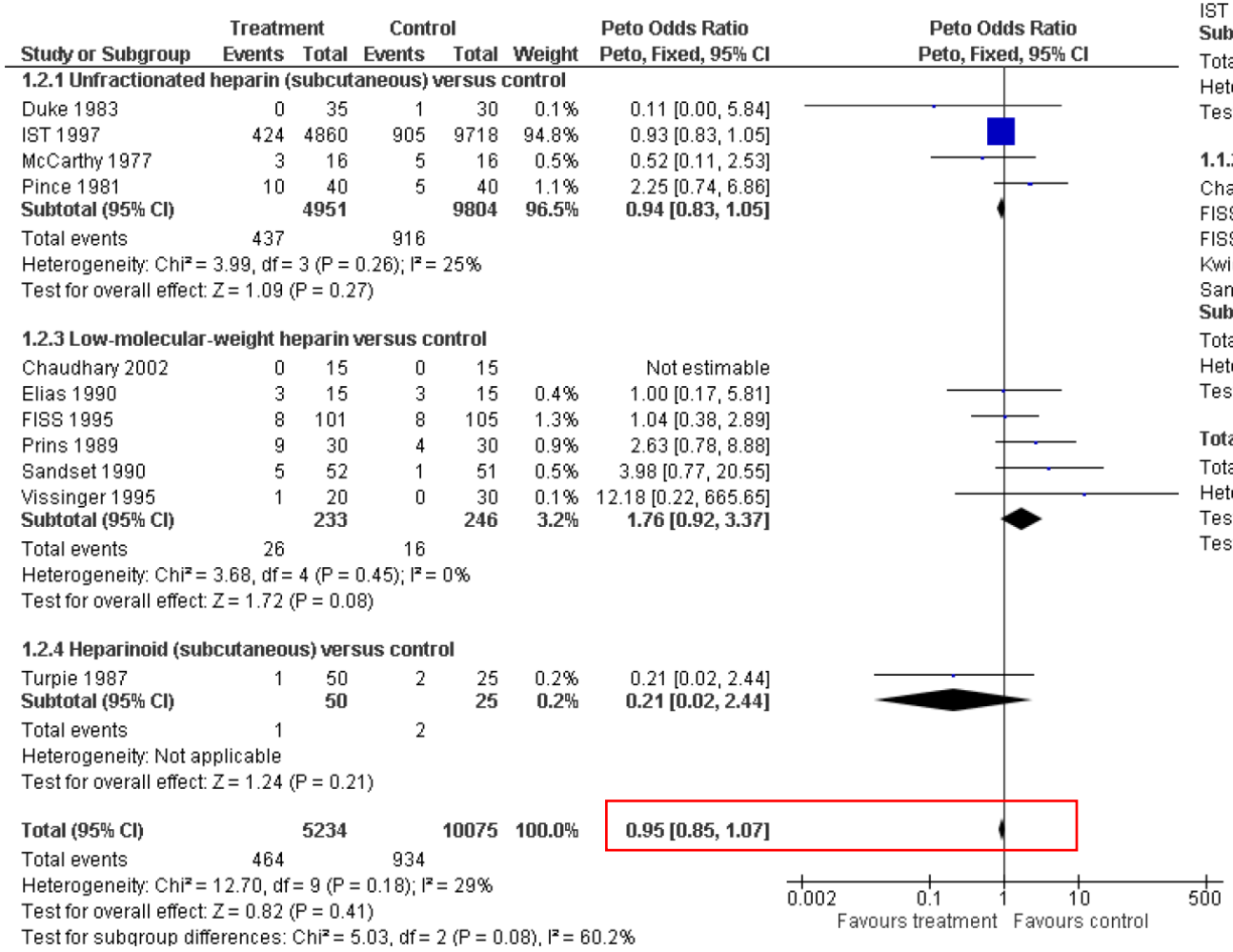
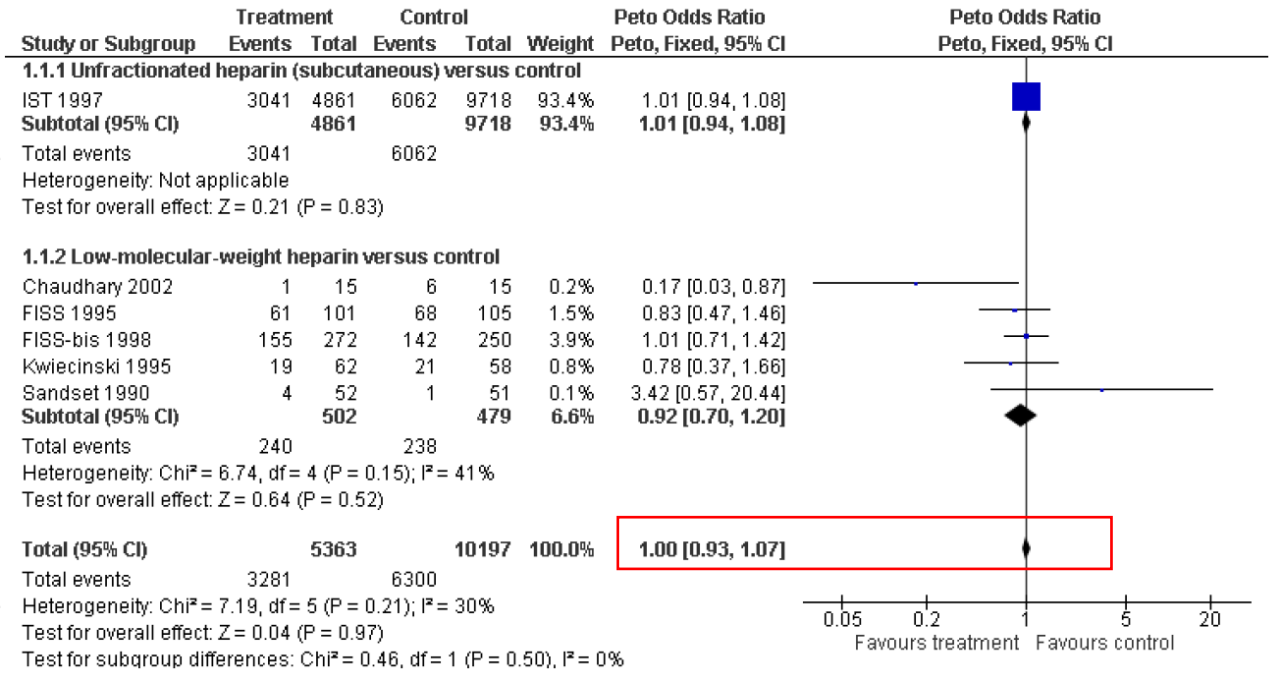


Figure 3.1 The effect on prophylactic anticoagulants of death or dependency at final follow up



EUROPEAN STROKE JOURNAL
European Stroke Journal
2016, Vol. 1(1) 6–19

European Stroke Organisation (ESO) guidelines for prophylaxis for venous thromboembolism in immobile patients with acute ischaemic stroke

Recommendation
Prophylactic anticoagulation with unfractionated heparin (UFH) (5000U ×2, or ×3 daily) or low molecular weight heparin (LMWH) or heparinoid should be considered in immobile patients with ischaemic stroke in whom the benefits of reducing the risk of venous thromboembolism is high enough to offset the increased risks of intracranial and extracranial bleeding associated with their use.
Quality of evidence: Moderate ⊕⊕⊕
Strength of recommendation: Weak for ↑?

Recommendation
Where a judgement has been made that prophylactic anticoagulation is indicated LMWH or heparinoid should be considered instead of UFH because of its greater reduction in risk of DVT, the greater convenience, reduced staff costs and patient comfort associated single daily dose vs. multiple daily injections but these advantages should be weighed against the higher risk of extracranial bleeding, higher drug costs and risks in elderly patients with poor renal function
Quality of evidence: Moderate ⊕⊕⊕
Strength of recommendation: Weak ↑?

EUROPEAN STROKE JOURNAL
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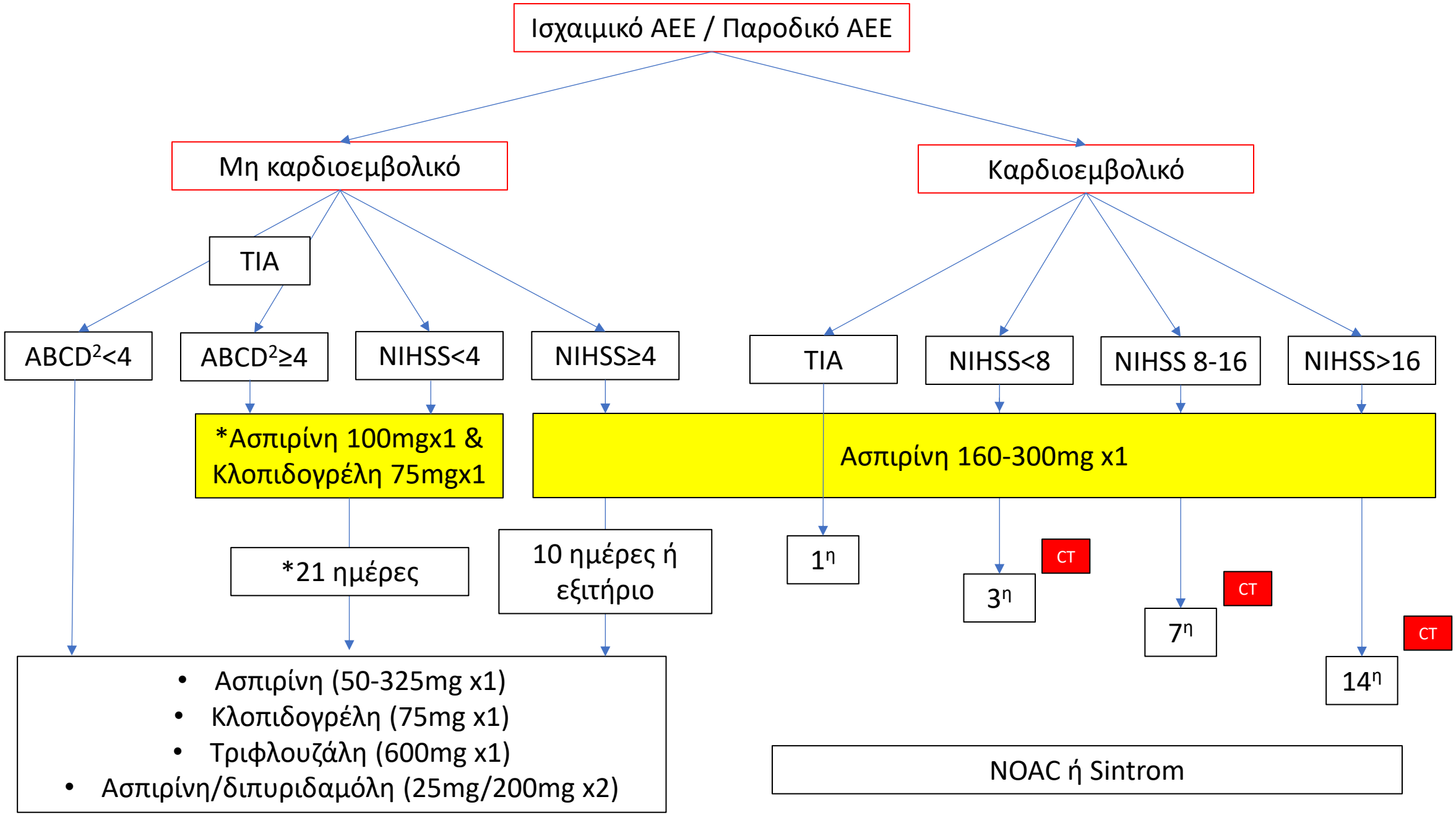
2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association

Recommendations for Deep Vein Thrombosis Prophylaxis
Referenced studies that support the recommendations are summarized in the online data supplement.

COR	LOE	Recommendations
1	B-R	1. In patients with AIS who have impaired mobility and do not have contraindications to intermittent pneumatic compression (IPC), IPC in addition to routine care is recommended over routine care alone to reduce the risk of deep vein thrombosis (DVT).[1,2]
2a	B-R	2. In patients with AIS who have impaired mobility, either prophylactic-dose subcutaneous heparin (UFH or LMWH) is reasonable to reduce the risk of VTE.[3]

Stroke. 2026 Jan 26. Epub ahead of print.


Ισχαιμικό ΑΕΕ / Παροδικό ΑΕΕ
Μη καρδιοεμβολικό
Καρδιοεμβολικό
TIA
ABCD²<4
ABCD²≥4
NIHSS<4
NIHSS≥4
TIA
NIHSS<8
NIHSS 8-16
NIHSS>16
*Ασπιρίνη 100mgx1 & Κλοπιδογρέλη 75mgx1
Ασπιρίνη 160-300mg x1
*21 ημέρες
10 ημέρες ή εξιτήριο
1η
3η
CT
7η
CT
14η
CT
• Ασπιρίνη (50-325mg x1)
• Κλοπιδογρέλη (75mg x1)
• Τριφλουζάλη (600mg x1)
• Ασπιρίνη/διπυριδαμόλη (25mg/200mg x2)
NOAC ή Sintrom

ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
ΙΔΡΥΘΕΝ ΤΟ 1837

ΠΡΟΓΡΑΜΜΑ ΜΕΤΑΠΤΥΧΙΑΚΩΝ ΣΠΟΥΔΩΝ
«ΚΑΡΔΙΟΜΕΤΑΒΟΛΙΚΗ ΙΑΤΡΙΚΗ»

ΑΕΕ και πρόληψη. Πρωτογενής Πρόληψη ΑΕΕ και η διαχείριση της αντιθρομβωτικής αγωγής στη Δευτερογενή Πρόληψη

Σας ευχαριστώ πολύ για την προσοχή σας!