STROKE EPIDEMIOLÓGIA

Dr. Fehér Gergely

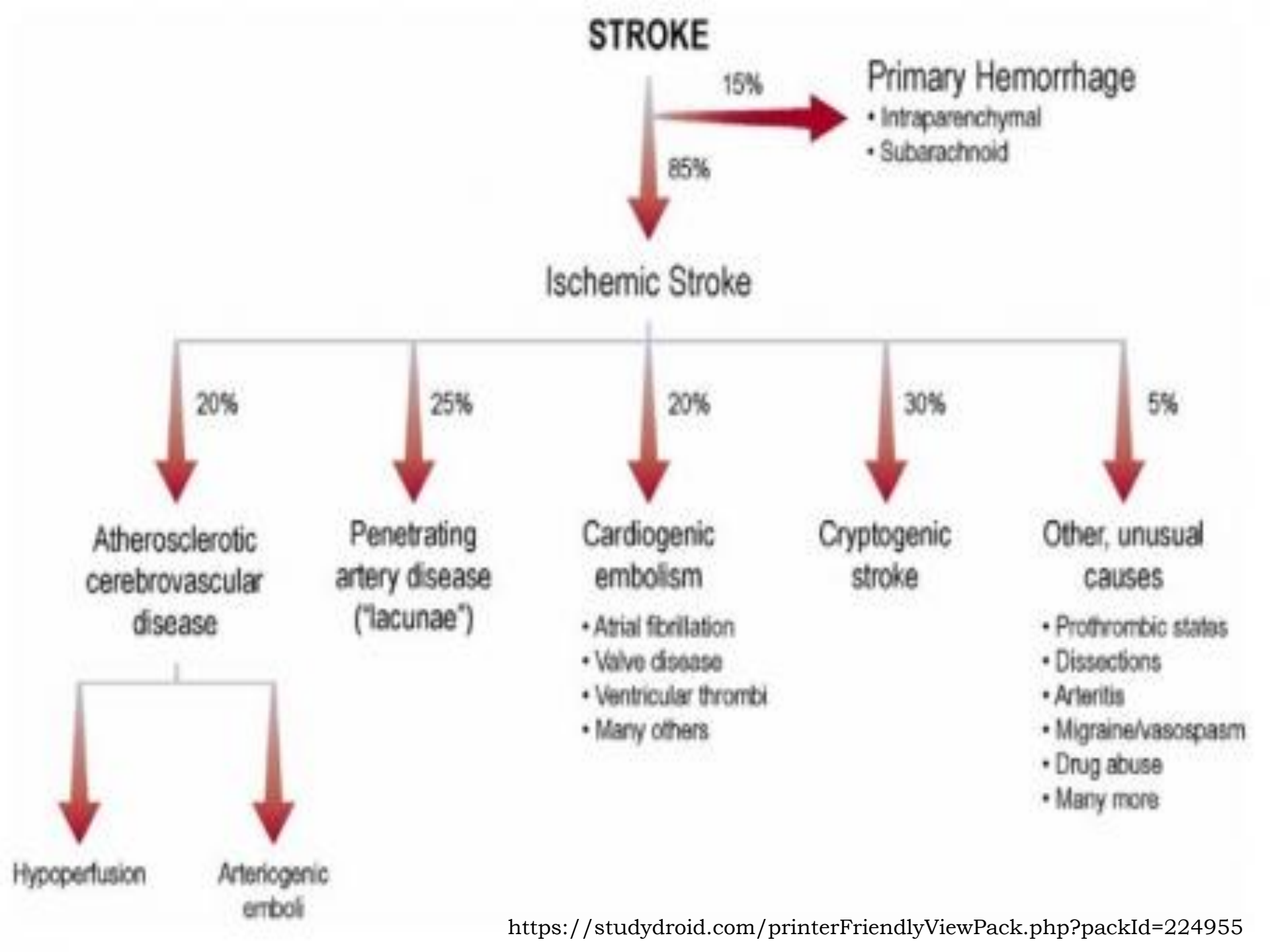
Szigetvári Kórház
Neurológiai Osztály

DEFINÍCIÓ

Vascularis esemény okozta
akut neurológiai deficit (>24h)

Többféle típus

1. Ischaemiás stroke
2. Haemorrhagiás stroke
3. Agyi vénás thrombosisok
4. Subarachnoidealis vérzés



DIFFERENCIÁL DIAGNÓZIS (Teljesség igénye nélkül)

Térfoglalás

Migrén

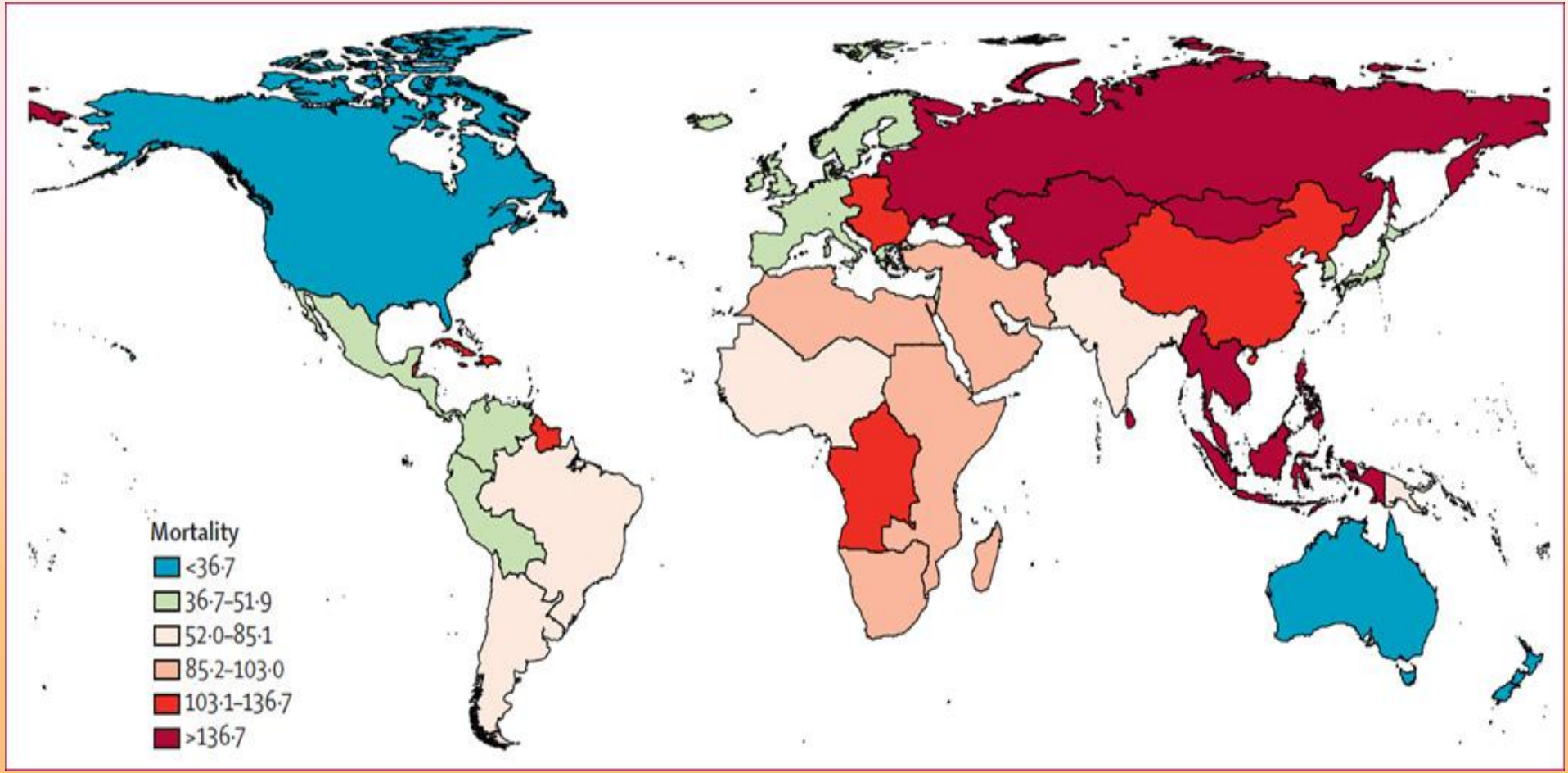
Demyelinisatio

Intoxicatio (egyéb metabolicus eltérések)

Gyulladások

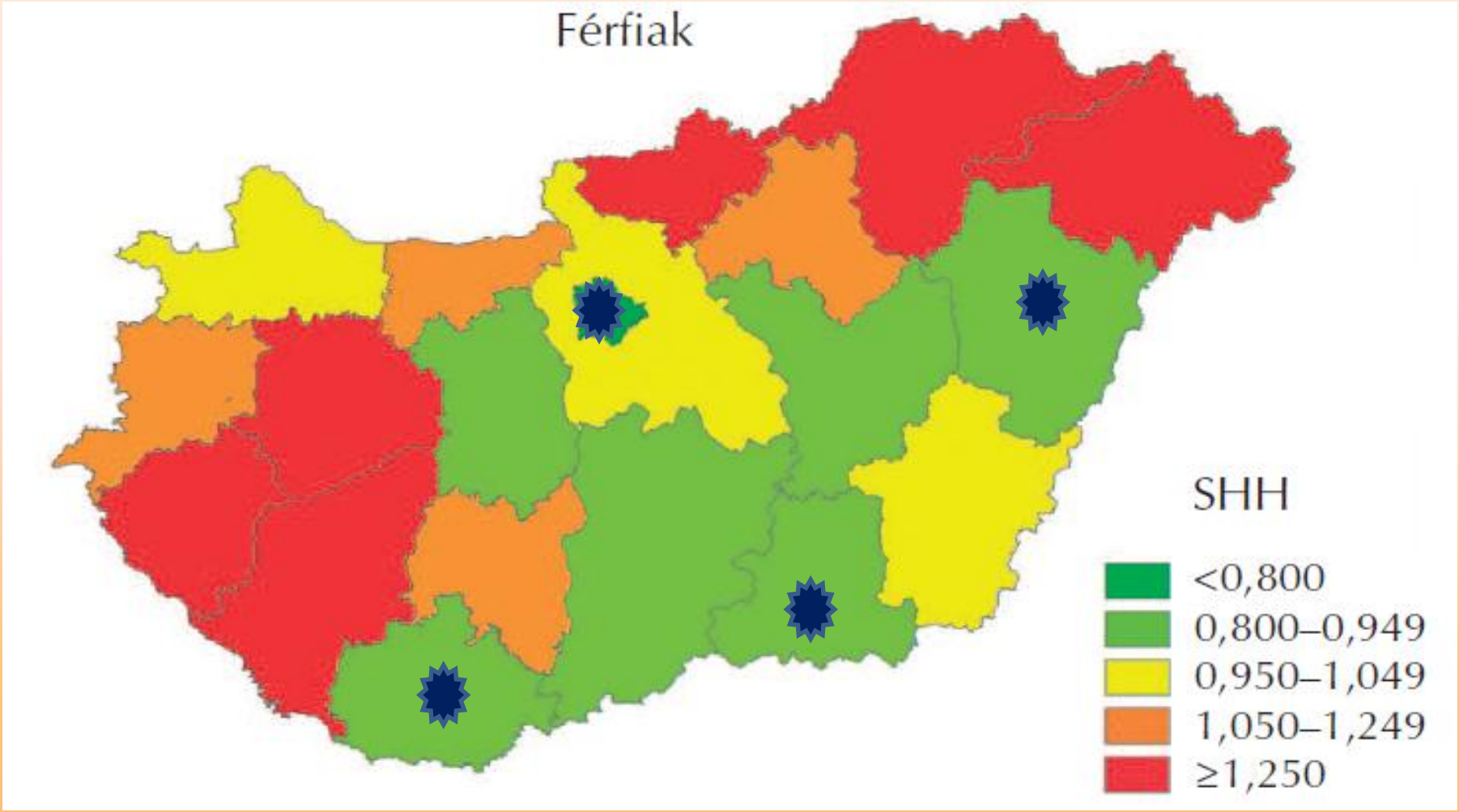
Sérülés

STROKE HALÁLOZÁS VILÁGSZERTE 2010-BEN

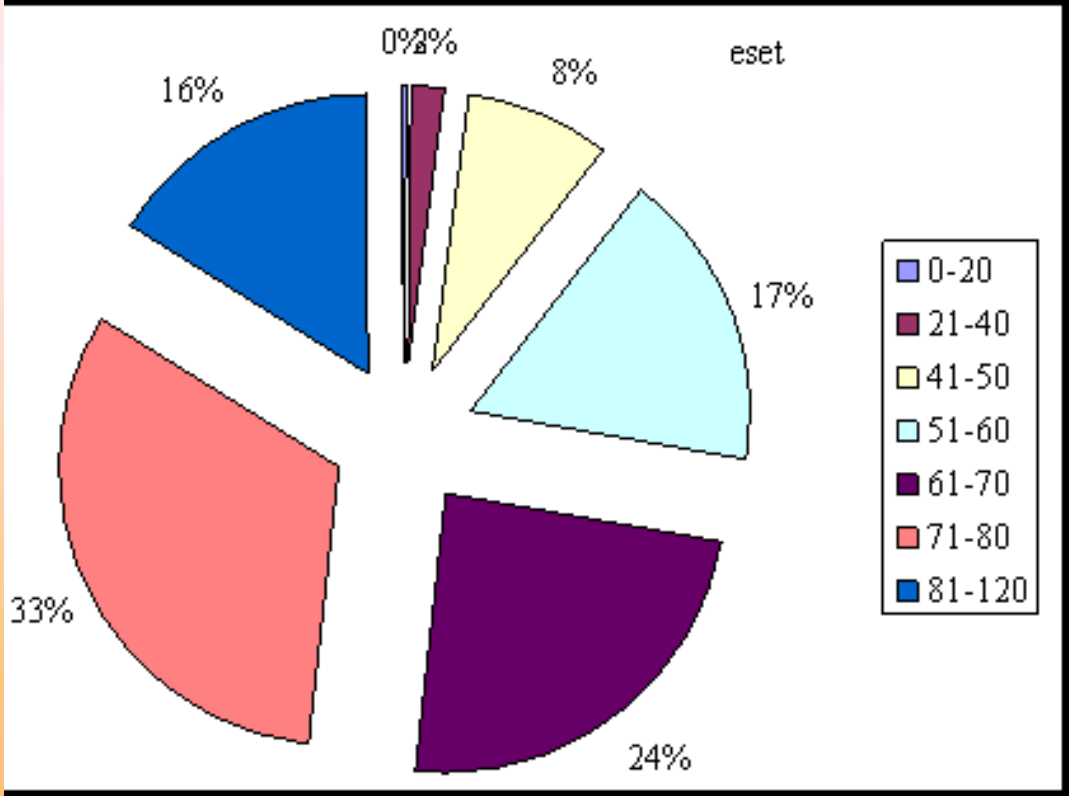


Feigh et al. Lancet 2014;383(9913):245-54

Stroke halálozás Magyarország különböző régióiban



Cerebrovascularis kórházi esetek: 2003



korcsop	eset	eset%
0-20	287	0,19
21-40	3 145	2,09
41-50	12 335	8,18
51-60	26 065	17,29
61-70	36 179	24,00
71-80	48 504	32,18
81-120	24 201	16,06
0-120	150 716	100,00

10%

OEP, 2005

A 60 ÉV ALATTIAK ARÁNYA 27%!!

I. RIZIKÓFAKTOROK: TIA/ISCHAEMIÁS STROKE

NEM BEFOLYÁSOLHATÓ RIZIKÓFAKTOROK

- **Életkor:** 55 éve felett évtizedenként megduplázódik a stroke rizikója
- **Nem:** 24-30 %-kal gyakoribb férfiakban, de a nők tovább élnek! (kiegyenlítődik)
- **Etnicitás:** 2-4x gyakoribb Afro-Amerikaiakban és egyre fiatalabb életkorban (hispán és kínai etnicitás esetében is)
- **Örökletes tényezők**: 9p21 kromoszóma?

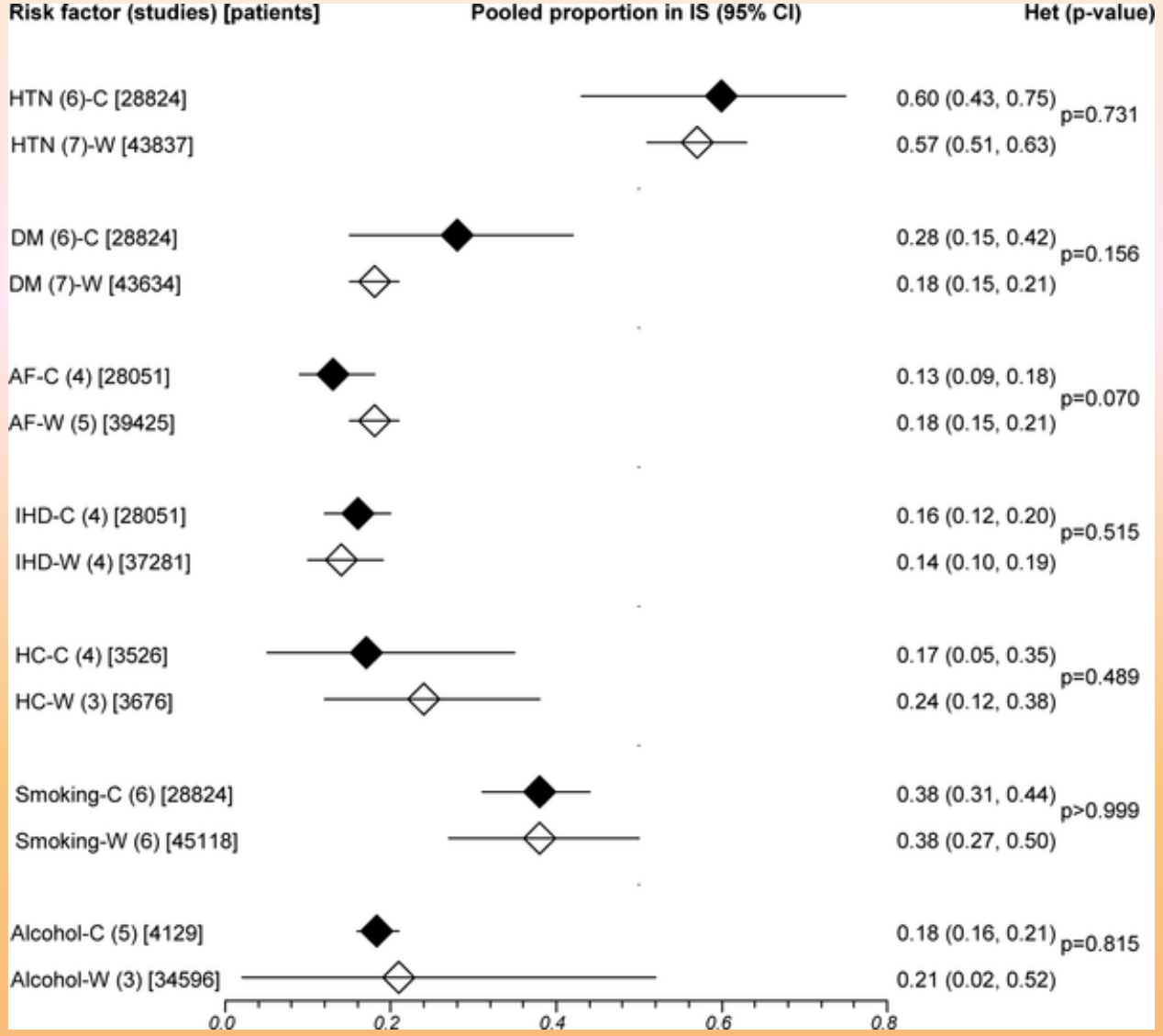
Ovbiagele B. et al Neurotherapeutics. 2011;8(3):319-29.

BEFOLYÁSOLHATÓ RIZIKÓFAKTOROK

- **Hipertónia:** akár az esetek 50%-ában
- **Pitvarfibrilláció:** 80 év felett az összes ischaemiás stroke 25%-ában kimutatható
- **Dohányzás:** 1,5x-es rizikó
- **Alkoholfogyasztás**
- **Coronaria betegség**: akár 3-4x-es stroke rizikó
- **Diabetes, prediabetes, metabolicus syndroma**
- **Dyslipidaemia**
- **Fizikai aktivitás**: rendszeres mozgás esetén 27%-kal kisebb rizikó

Ovbiagele B. et al Neurotherapeutics. 2011;8(3):319-29.

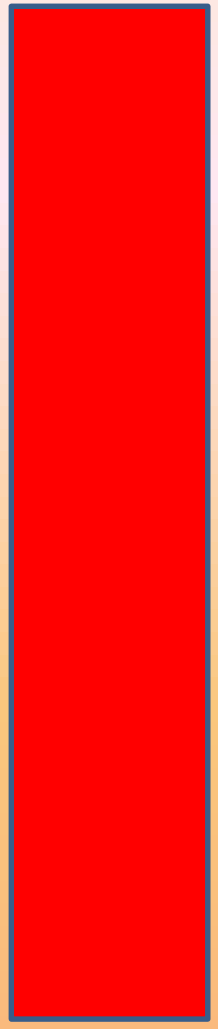
RIZIKÓFAKTOROK

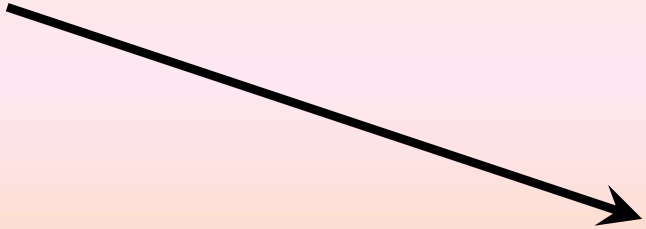


Tsai CF et al. PLoS ONE 2016;11(3): e0151743

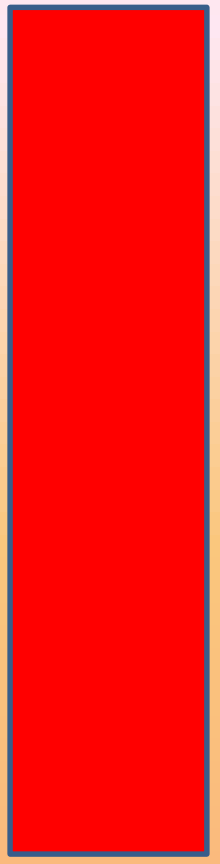
Hogyan csökkentsük 1/3-al a stroke-ot

30 000 stroke





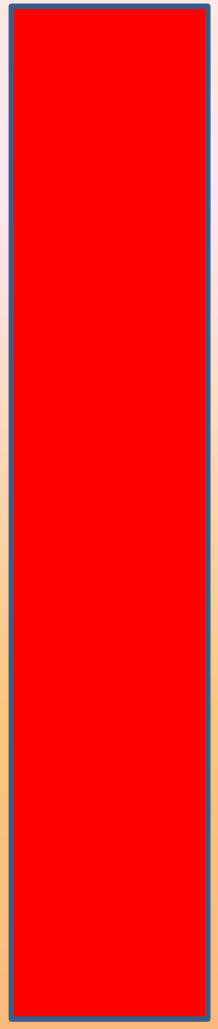
27 500 stroke

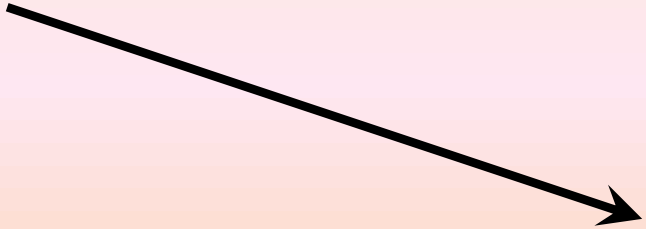


Ha a lusták fele mozogni kezd

Hogyan csökkentsük 1/3-al a stroke-ot

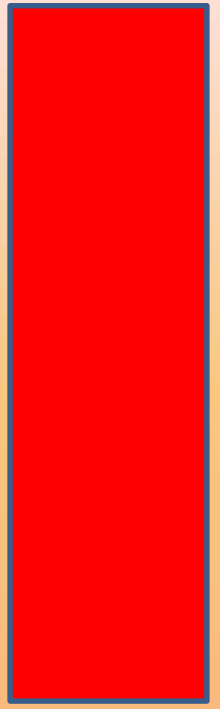
30 000 stroke





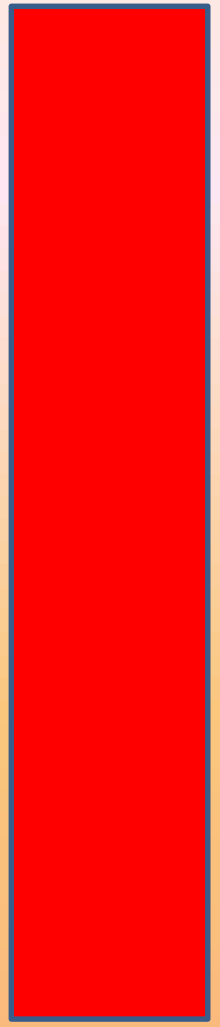
Ha a dohányzók fele abbahagyná

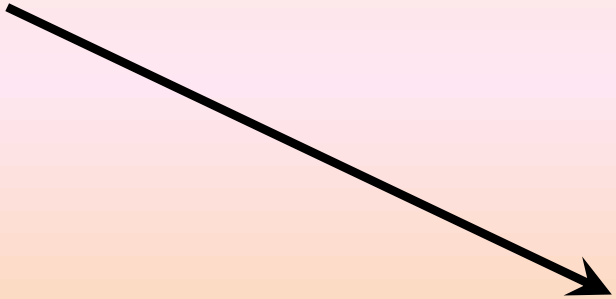
25 500 stroke



Hogyan csökkentsük 1/3-al a stroke-ot

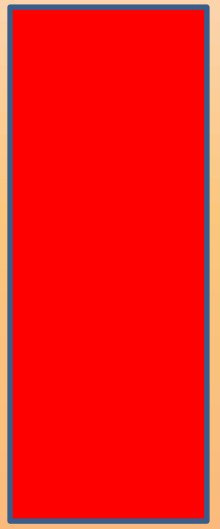
30 000 stroke





23 500 stroke

Ha a szivritmuszavar felét kezelnénk

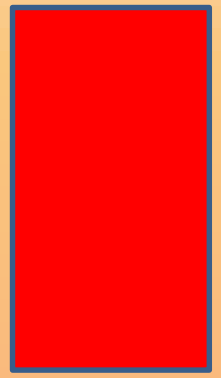


Hogyan csökkentsük 1/3-al a stroke-ot

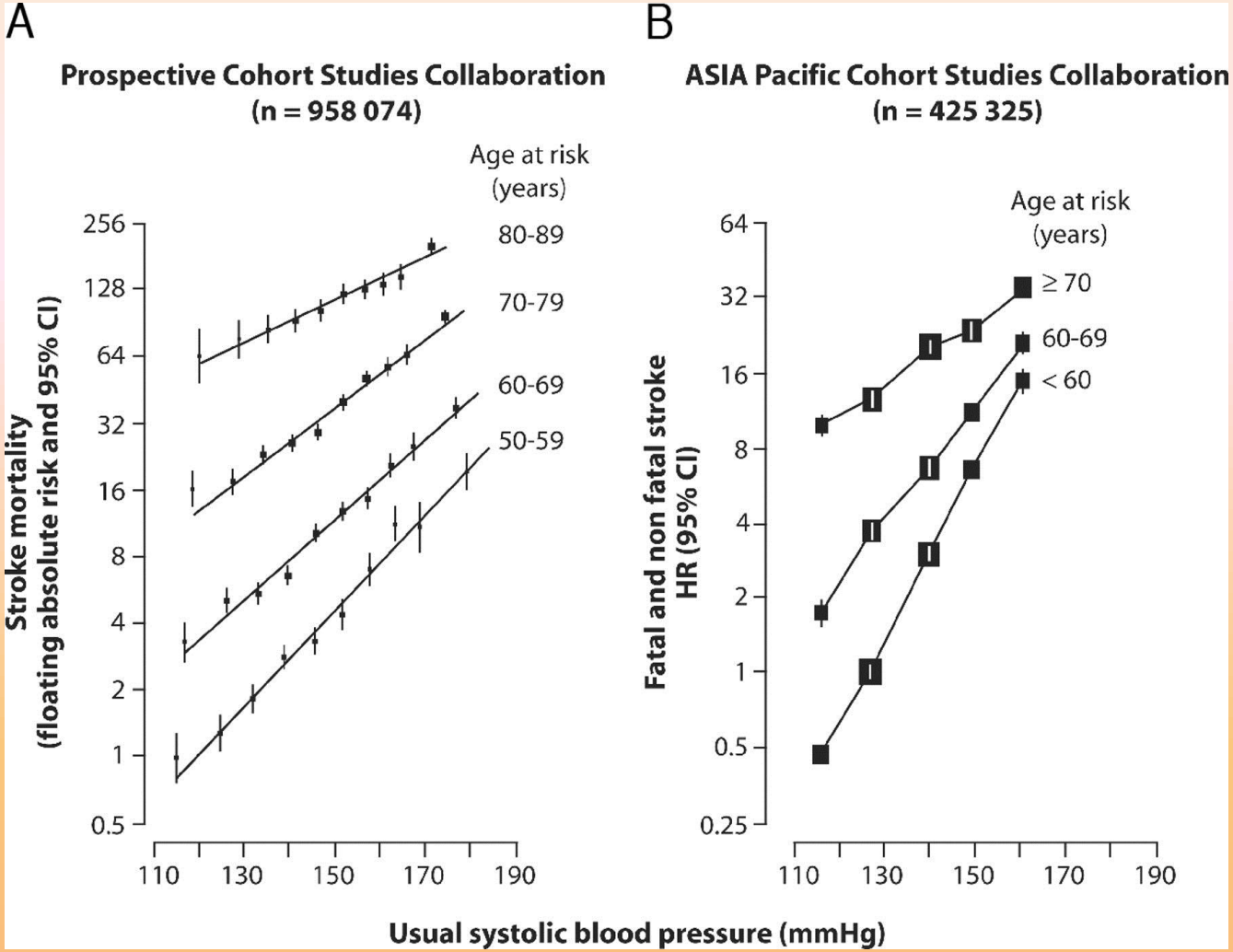
30 000 stroke

Ha a magasvérnyomás felét kezelnénk

20 000 stroke



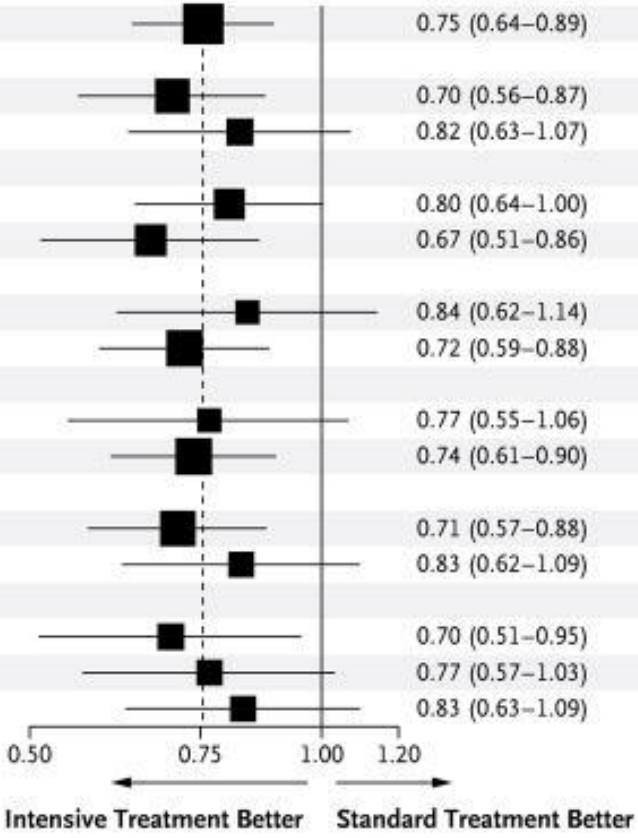
VÉRNYOMÁS ÉS STROKE



Haifeng Zhang et al. Hypertension. 2006;48:187-195

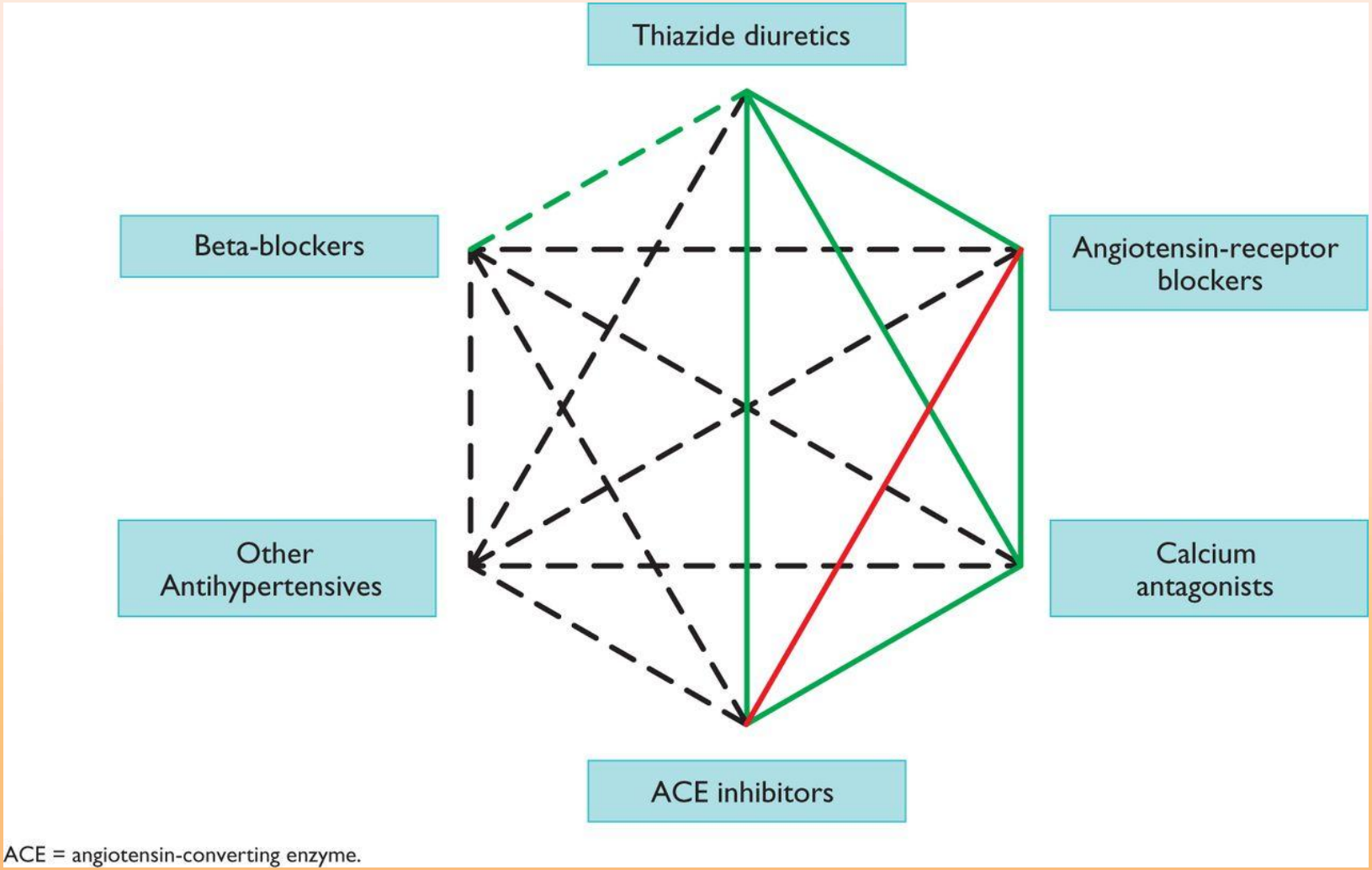
INTENZÍV VÉRNYOMÁSCSÖKKENTÉS

Subgroup	Intensive Treatment	Standard Treatment	Hazard Ratio (95% CI)	P Value for Interaction
	no. of patients with primary outcome/total no. (%)			
Overall	243/4678 (5.2)	319/4683 (6.8)	0.75 (0.64–0.89)	
Previous CKD				0.36
No	135/3348 (4.0)	193/3367 (5.7)	0.70 (0.56–0.87)	
Yes	108/1330 (8.1)	126/1316 (9.6)	0.82 (0.63–1.07)	
Age				0.32
<75 yr	142/3361 (4.2)	175/3364 (5.2)	0.80 (0.64–1.00)	
≥75 yr	101/1317 (7.7)	144/1319 (10.9)	0.67 (0.51–0.86)	
Sex				0.45
Female	77/1684 (4.6)	89/1648 (5.4)	0.84 (0.62–1.14)	
Male	166/2994 (5.5)	230/3035 (7.6)	0.72 (0.59–0.88)	
Race				0.83
Black	62/1454 (4.3)	85/1493 (5.7)	0.77 (0.55–1.06)	
Nonblack	181/3224 (5.6)	234/3190 (7.3)	0.74 (0.61–0.90)	
Previous cardiovascular disease				0.39
No	149/3738 (4.0)	208/3746 (5.6)	0.71 (0.57–0.88)	
Yes	94/940 (10.0)	111/937 (11.8)	0.83 (0.62–1.09)	
Systolic blood pressure				0.77
≤132 mm Hg	71/1583 (4.5)	98/1553 (6.3)	0.70 (0.51–0.95)	
>132 to <145 mm Hg	77/1489 (5.2)	106/1549 (6.8)	0.77 (0.57–1.03)	
≥145 mm Hg	95/1606 (5.9)	115/1581 (7.3)	0.83 (0.63–1.09)	



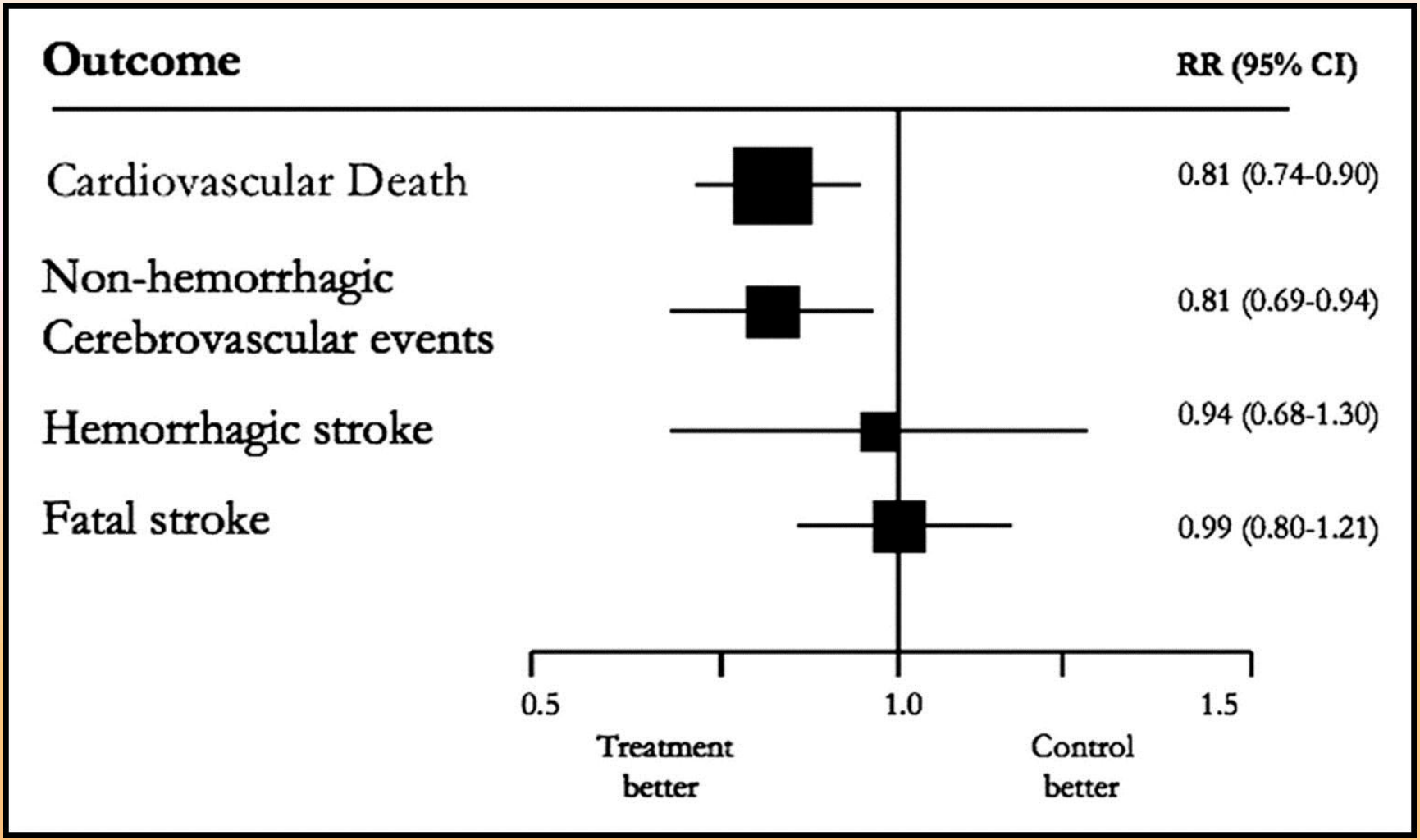
SPRINT Research Group N Engl J Med. 2015;373(22):2103-16

TENZIÓCSÖKKENTÉS



Authors/Task Force Members et al. Eur Heart J 2013;34:2159-2219

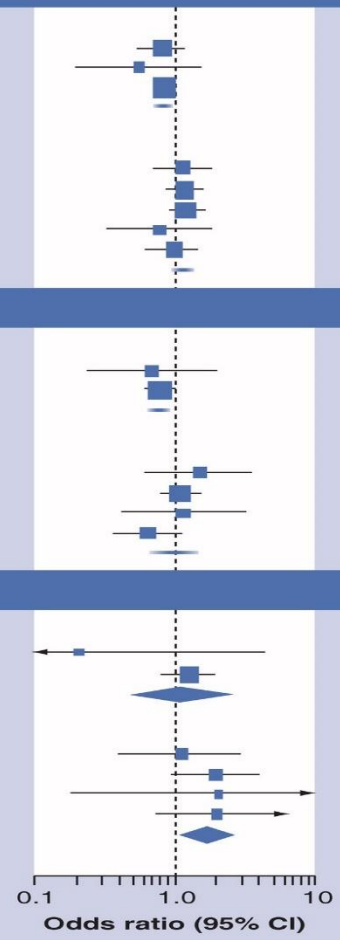
STATINKEZELÉS



O'Regan C. et al. Am J Med. 2008;121(1):24-33.

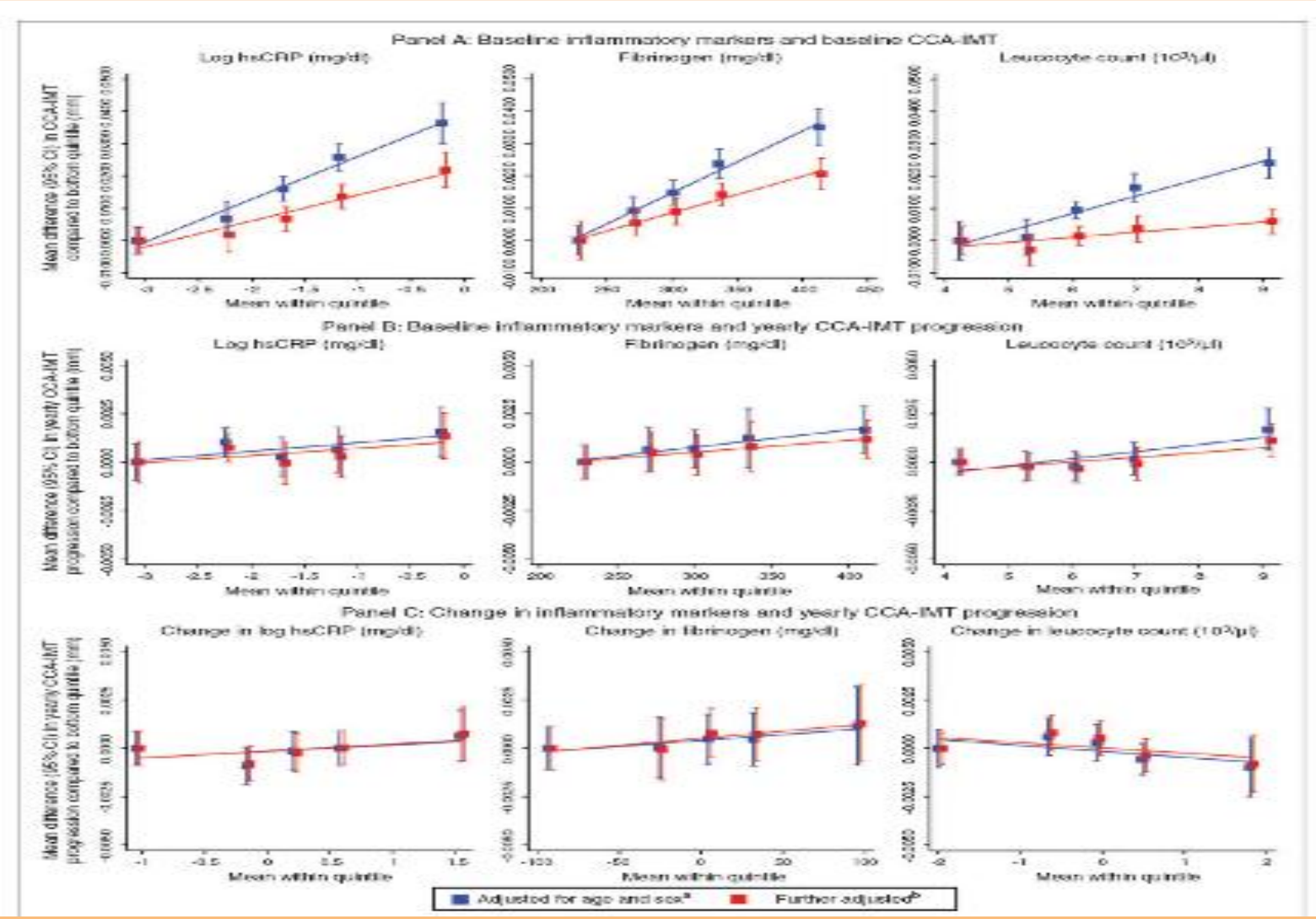
ASPIRIN PRIMER PREVENCIÓS SZEREPE

Study (year)	Events, n/total: Aspirin	Events, n/total: Control/placebo	Odds ratio (95% CI)
Stroke			
Women			
HOT (1996)	54/4437	67/4446	0.81 (0.56–1.16)
PPP (2001)	6/1277	11/1306	0.56 (0.21–1.51)
WHS (2005)	221/19,934	255/19,942	0.84 (0.70–1.01)
Total	281/25,648	344/25,694	0.83 (0.70–0.97)
Men			
BDT (1988)	61/3429	27/1710	1.13 (0.72–1.78)
HOT (1998)	94/4962	80/4945	1.17 (0.87–1.57)
PHS (1989)	119/11037	95/11,034	1.22 (0.93–1.59)
PPP (2001)	10/949	13/963	0.78 (0.34–1.78)
TPT (1998)	47/2545	48/2540	0.98 (0.65–1.47)
Total	331/22,922	266/21192	1.13 (0.96–1.33)
Ischemic stroke			
Women			
PPP (2001)	6/1277	9/1306	0.68 (0.24–1.92)
WHS (2005)	179/19,934	221/19,942	0.77 (0.63–0.94)
Total	179/21,211	230/21,248	0.76 (0.63–0.93)
Men			
BDT (1988)	61/3429	27/1710	1.50 (0.64–3.53)
PHS (1989)	119/11,037	95/11034	1.11 (0.82–1.50)
PPP (2001)	10/949	13/963	1.16 (0.42–3.22)
TPT (1998)	47/2545	48/2540	0.64 (0.37–1.11)
Total	331/17,960	266/16,247	1.00 (0.72–1.41)
Hemorrhagic stroke			
Women			
PPP (2001)	8/1277	2/1306	0.20 (0.01–4.23)
WHS (2005)	51/19,934	41/19,942	1.25 (0.83–1.88)
Total	51/21,211	43/21,248	1.07 (0.42–2.69)
Men			
BDT (1988)	13/3429	5/1710	1.08 (0.41–2.85)
PHS (1989)	23/11,037	12/11,034	1.92 (0.95–3.86)
PPP (2001)	2/949	1/963	2.03 (0.18–22.44)
TPT (1998)	12/2545	6/2540	2.00 (0.75–5.34)
Total	50/17,960	25/16,247	1.69 (1.04–2.73)



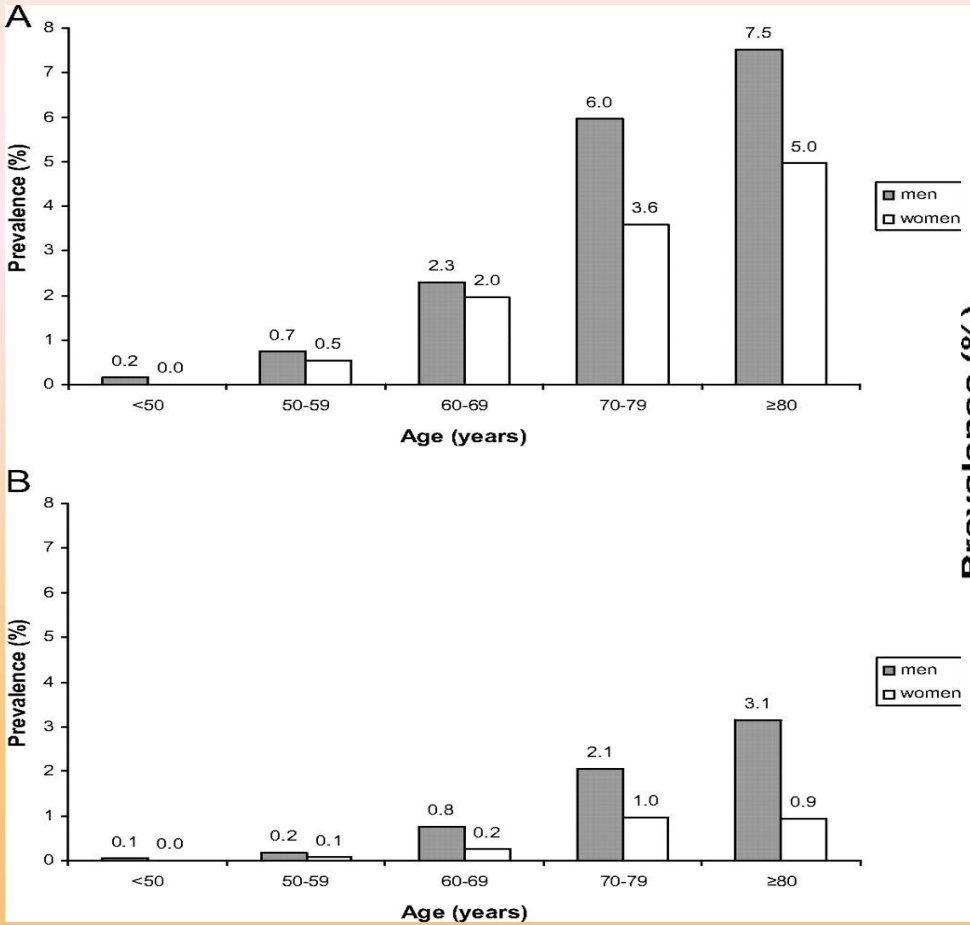
Adelmann EE. et al. Womens Health (Lond). 2011;7(3):341-52

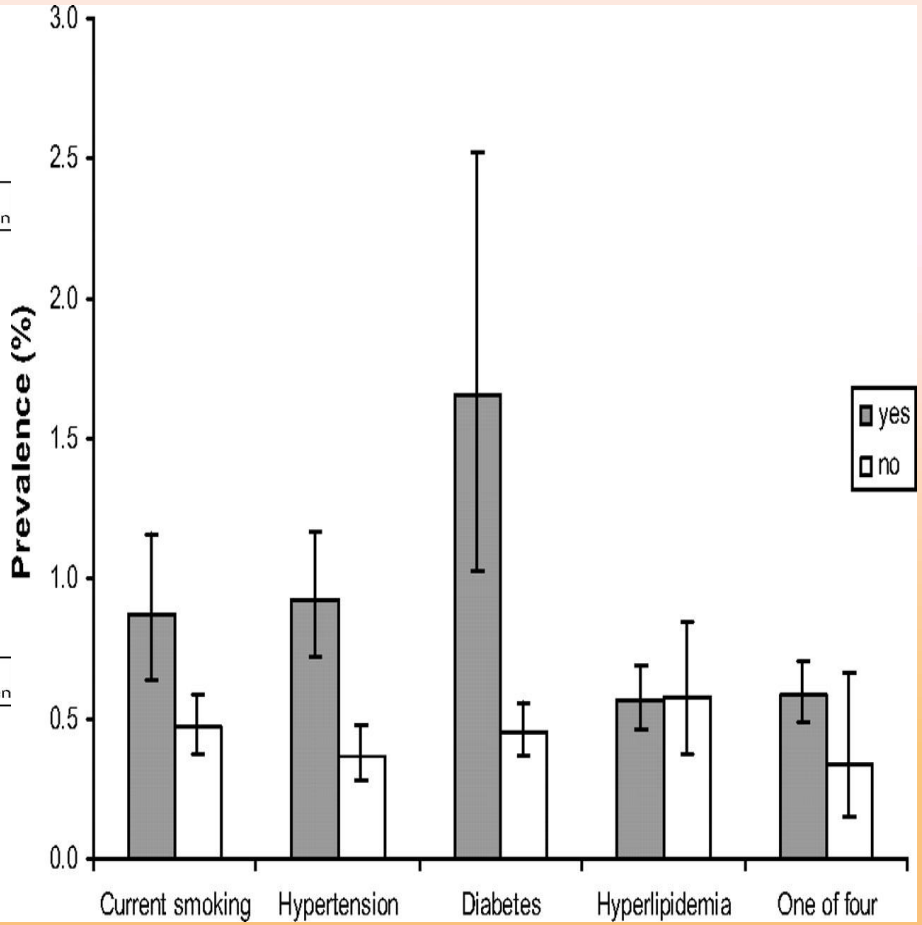
CAROTIS IMT ÉS ÉRBETEGSÉG



Villeit P. et al. Eur J Prev Cardiol. 2016;23(2):194-205

CAROTIS STENOSIS AZ ÁTLAGPOPULÁCIÓBAN



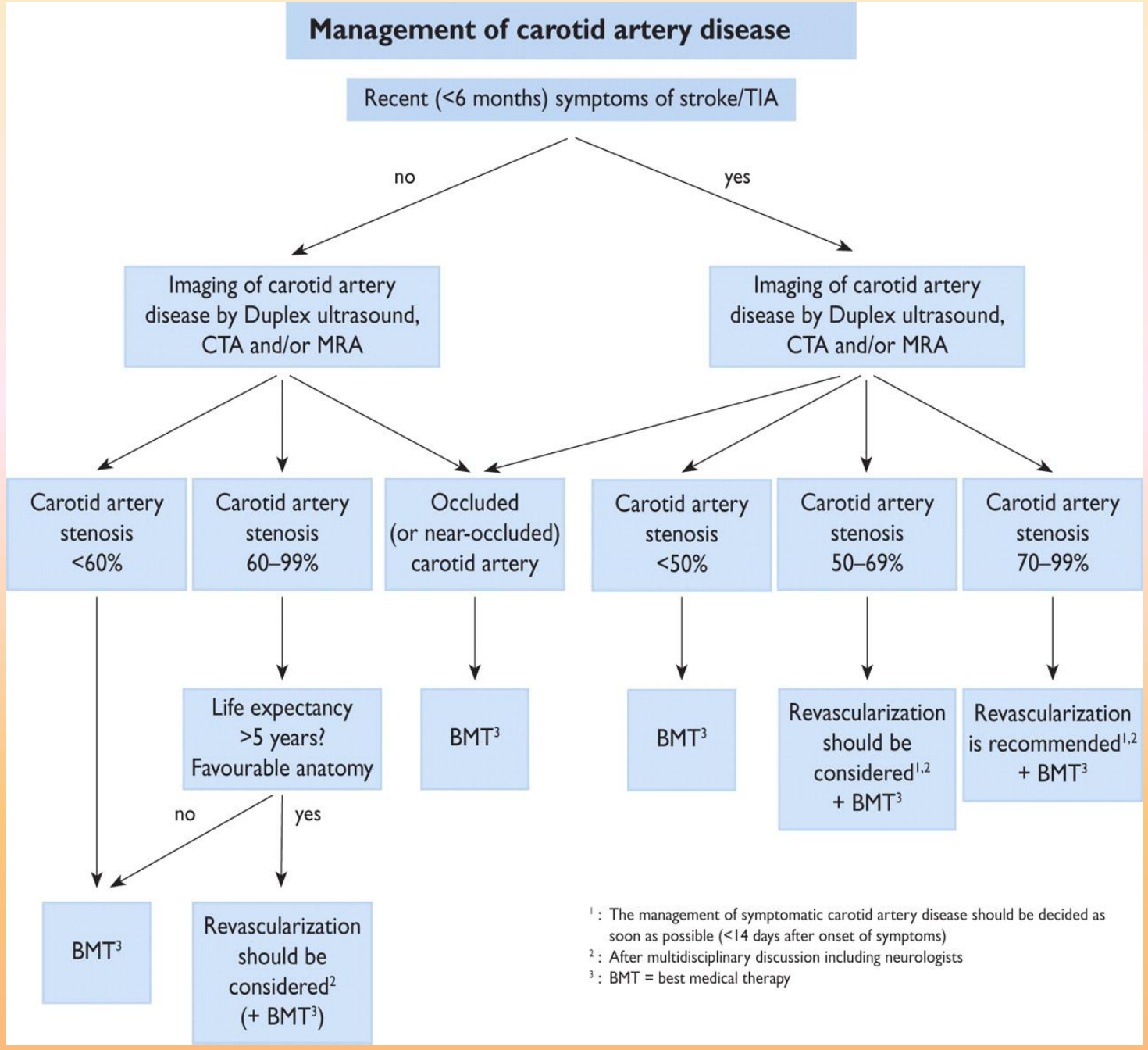


Marjolein de Weerd et al. Stroke. 2010;41:1294-1297

SZÉDÜLÉS ÉS CAROTIS STENOSIS

- Szédülés (különös tekintettel vertigo hiányára) carotis scan nem indokolt!
- 114 perifériás szédülős és 42 nem szédülős beteg összehasonlítása (átlagéletkor 62 év)
- Carotis plakkok aránya 19,8 vs 26,7 %
- Aktuális ajánlások sem támogatják CDU elvégzését szédülés esetében
- Leggyakoribb felesleges vizsgálat, melyet háziorvosok kérnek

Salvaggio G. et al. J Ultrasound. 2010;13(3):98-103.
Landry BA. et al. Can Fam Physician. 2011,57(8):e299-304



ESC Guidelines on the diagnosis and treatment of peripheral artery diseases Eur Heart J. 2011;32(22):2851-906.

Recommendations	Class[a]	Level[b]	Ref[c]
All patients with asymptomatic carotid artery stenosis should be treated with long-term antiplatelet therapy.	I	B	52, 54, 66
All patients with asymptomatic carotid artery stenosis should be treated with long-term statin therapy.	I	C	-
In asymptomatic patients with carotid artery stenosis ≥60%, CEA should be considered as long as the perioperative stroke and death rate for procedures performed by the surgical team is <3% and the patient's life expectancy exceeds 5 years.	IIa	A	52, 54, 66
In asymptomatic patients with an indication for carotid revascularization, CAS may be considered as an alternative to CEA in high-volume centres with documented death or stroke rate <3%.	IIb	B	79, 99

[a]Class of recommendation.
[b]Level of evidence.
[c]References.
CAS = carotid artery stenting; CEA = carotid endarterectomy.

ASYMPTOMATICUS CAROTIS SZŰKÜLET

The USPSTF recommends against screening for asymptomatic carotid artery stenosis in the general adult population.

ESC Guidelines on the diagnosis and treatment of peripheral artery diseases
Eur Heart J. 2011;32(22):2851-906..

Annals of Internal Medicine
Established in 1927 by the American College of Physicians

From: Management Strategies for Asymptomatic Carotid Stenosis: A Systematic Review and Meta-analysis

Ann Intern Med. 2013;158(9):676-685. doi:10.7326/0003-4819-158-9-201305070-00007

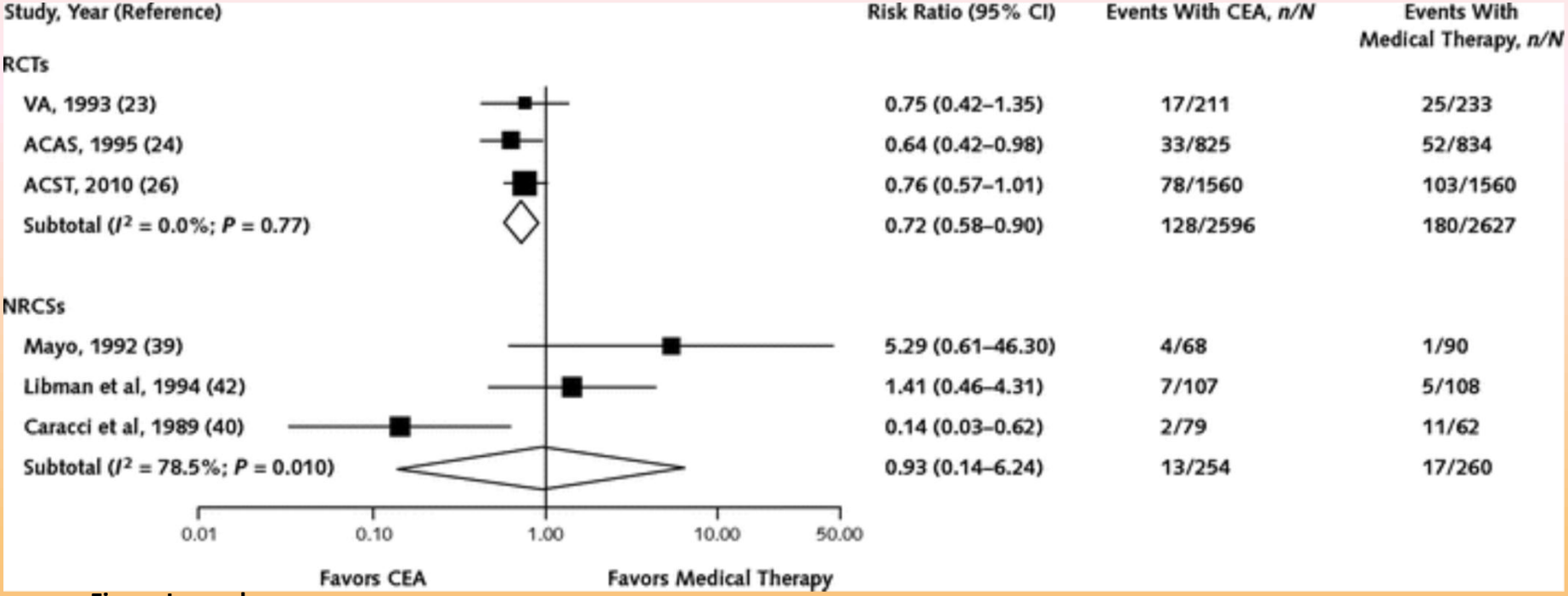
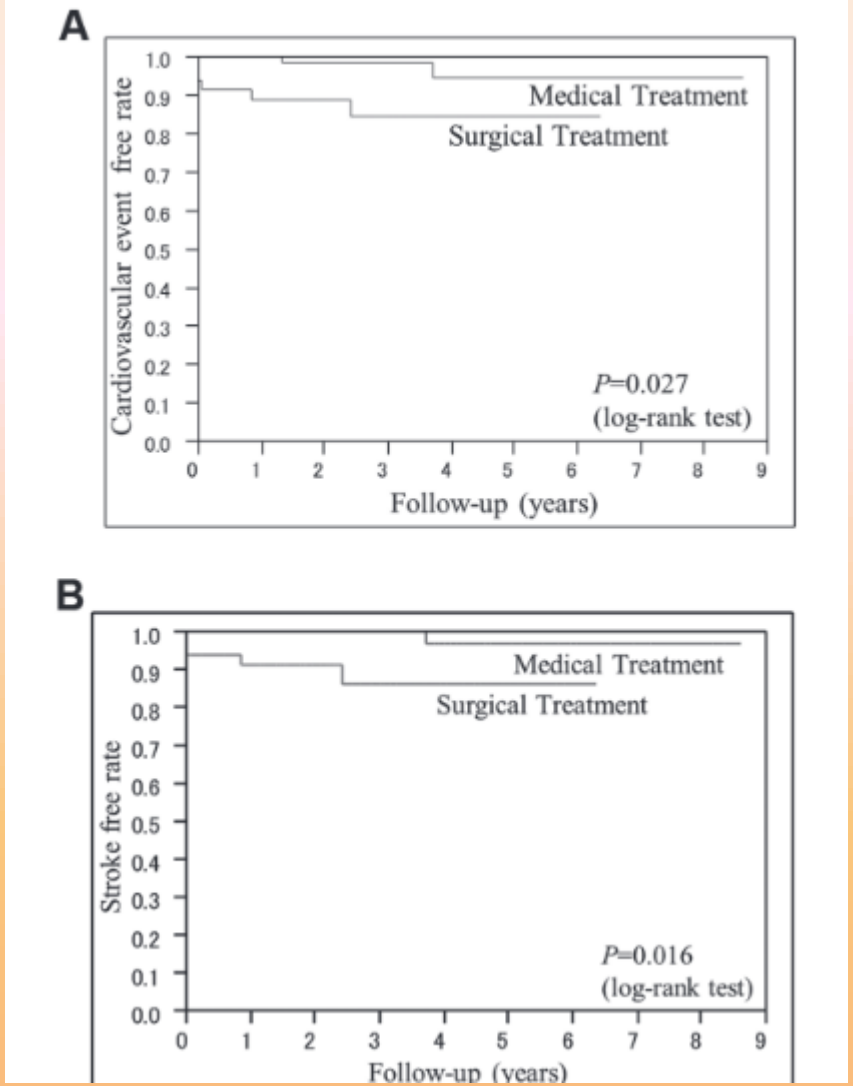


Figure Legend:

Meta-analysis of ipsilateral stroke (including any stroke within 30 days) in RCTs and NRCSs of CEA versus medical therapy.

ACAS = Asymptomatic Carotid Atherosclerosis Study; ACST = Asymptomatic Carotid Surgery Trial; CEA = carotid endarterectomy; NRCS = nonrandomized, comparative study; RCT = randomized, controlled trial; VA = Veterans Affairs.

VASCULARIS KIMENETEL



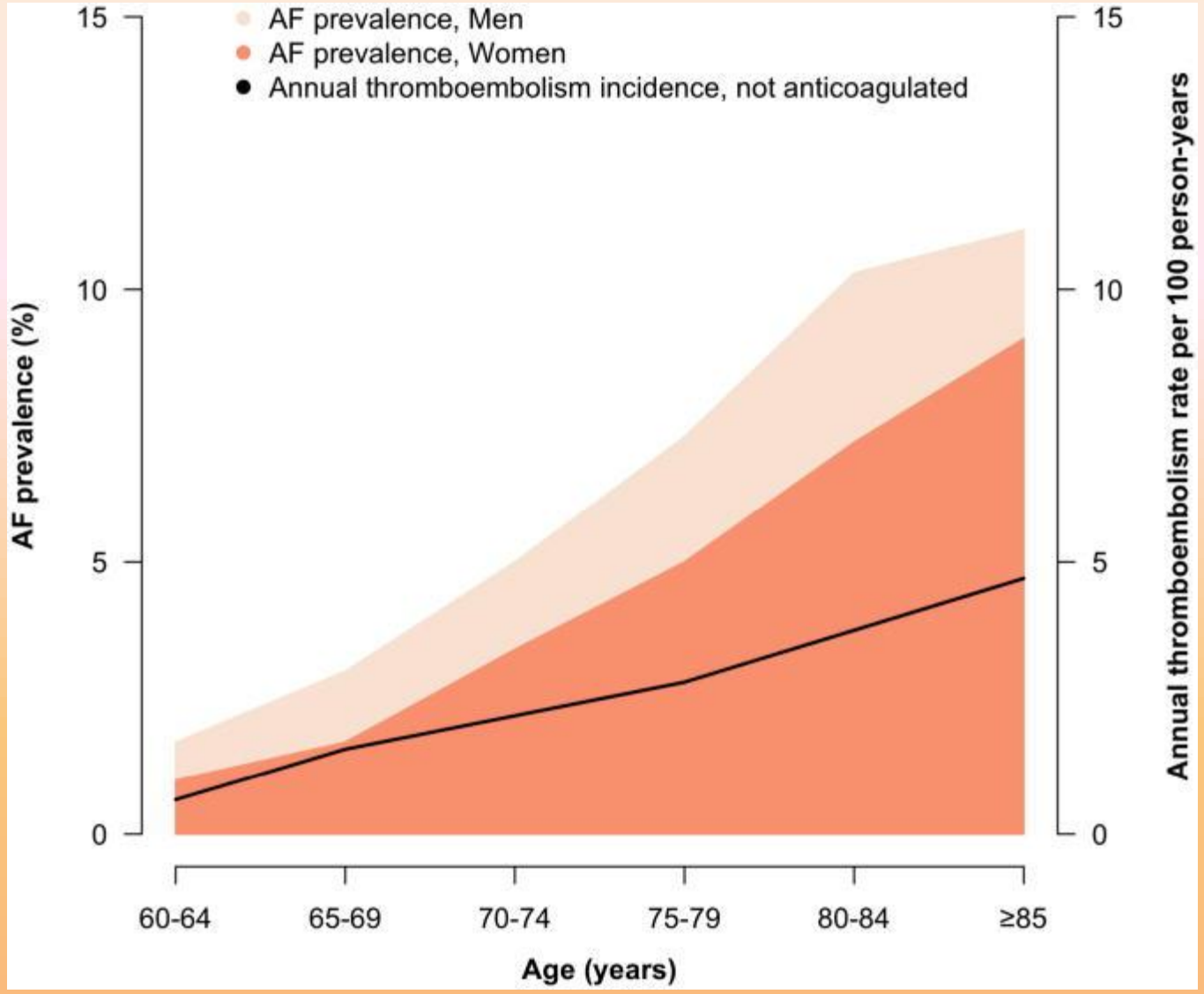
11391 asymptomaticus carotis stenosisos beteg utánkövetési meta-analízise

Összhalálozás **62,9 %**-a cardialis!!!

Stroke rizikó < 1%/év
Cardialis rizikó 2,9%/év

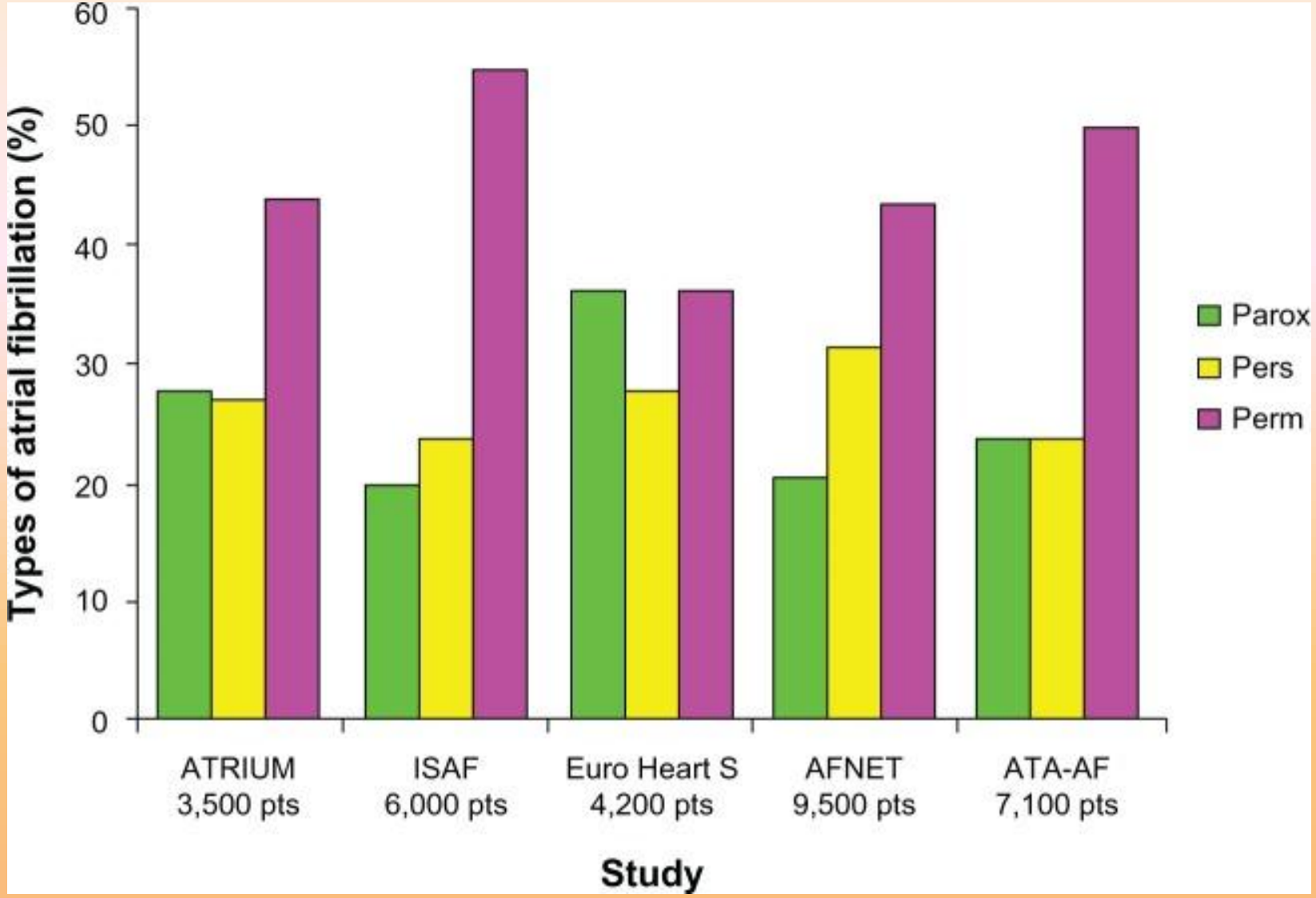
Sato K. et al. Int Heart J. 2016;57(1):80-6.
Eur J Vasc Endovasc Surg. 2015,50(5):573-82

PITVARFIBRILLÁCIÓ JELENTŐSÉGE



Lubitz SA. Et al. J Am Geriatr Soc. 2013;61(10):1798-803

PITVARFIBRILLÁCIÓ TÍPUSAI



Massimo Z. et al. Clin Epidemiol. 2014; 6: 213–220.

Prolonged Ambulatory Cardiac Monitoring Improves the Detection and Treatment of Atrial Fibrillation in Patients with Cryptogenic Stroke: Primary Results from the EMBRACE Multicenter Randomized Trial

Background: **Detecting** atrial fibrillation (AF) in stroke/TIA patients can result in therapy to prevent recurrent strokes. However, standard short duration monitoring (24-48 h) for atrial fibrillation may not detect AF.

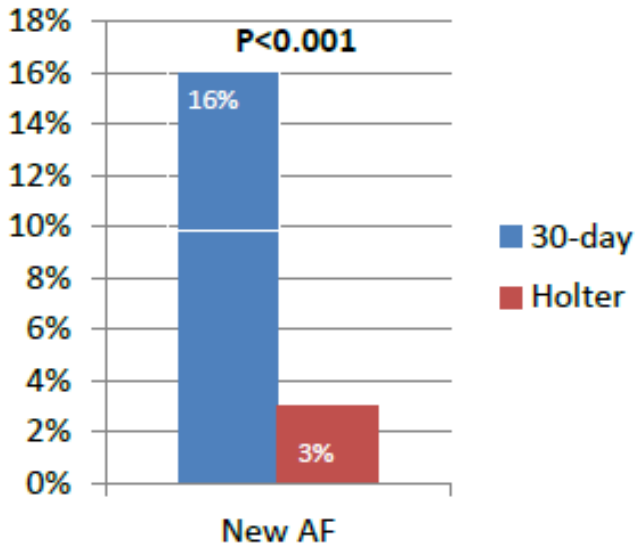
Purpose: This study is the first randomized trial to evaluate whether longer non-invasive ECG monitoring after stroke/TIA would produce beneficial results.

Methods: n=572 (age 73±9yrs;); recent ischemic stroke/TIA, no known AF; 16 stroke centers; Randomized to wear either an event-triggered cardiac monitor up to 30 days or a repeat 24 h Holter. AF events automatically recorded.

Primary Outcome: ≥1 episodes of AF of at least 30 seconds within 90 days of randomization

Secondary Outcomes: monitoring adherence ; anticoagulation status

Results: New AF detected among 16% of 30-day monitoring group, vs. 3% in the Holter group (p<0.001). In the 30 day group three quarters of AF events occurred within the first 2 weeks. 71% of all patients were anticoagulated; anticoagulant use at 90 days > 30 day group (49/280; 18%) vs. Holter group (28/279; 10%; p=0.01).



Conclusion: Paroxysmal AF is undiagnosed and untreated in many stroke/TIA patients ; in the post-stroke setting it is under-detected by the Holter monitor. Prolonged continuous monitoring for 30 days is "feasible, more effective, and leads to clinically meaningful changes in patient management. "

Presented by: Gladstone DJ, International Stroke Conference, Honolulu, HI

VÉRZÉSES SZÖVŐDMÉNYEK ASPIRIN VS WARFARIN MELLETT

TABLE 3. ADVERSE EVENTS ACCORDING TO TREATMENT ASSIGNMENT.*

EVENT	WARFARIN (N=1103)	ASPIRIN (N=1103)	ODDS RATIO (95% CI)	P VALUE†
	no. (%)			
Death	47 (4.3)	53 (4.8)	0.88 (0.58–1.32)	0.61
Related to hemorrhage	7 (0.6)	5 (0.4)	1.40 (0.42–5.13)	0.77
First hemorrhage‡				
Major	38 (3.4)	30 (2.7)	1.28 (0.78–2.10)	0.39
Minor	261 (23.7)	188 (17.0)	1.51 (1.22–1.87)	<0.001
			RATE RATIO (95% CI)	P VALUE§
	no. of events (rate/100 patient-yr)			
All hemorrhages¶				
Major	44 (2.2)	30 (1.5)	1.48 (0.93–2.44)	0.10
Minor	413 (20.8)	259 (12.9)	1.61 (1.38–1.89)	<0.001

*Maximal follow-up was 25 months. Hemorrhages occurring on the day of the primary event (death or recurrent ischemic stroke) are included. CI denotes confidence interval.

†P values were calculated by the exact test of two independent proportions.

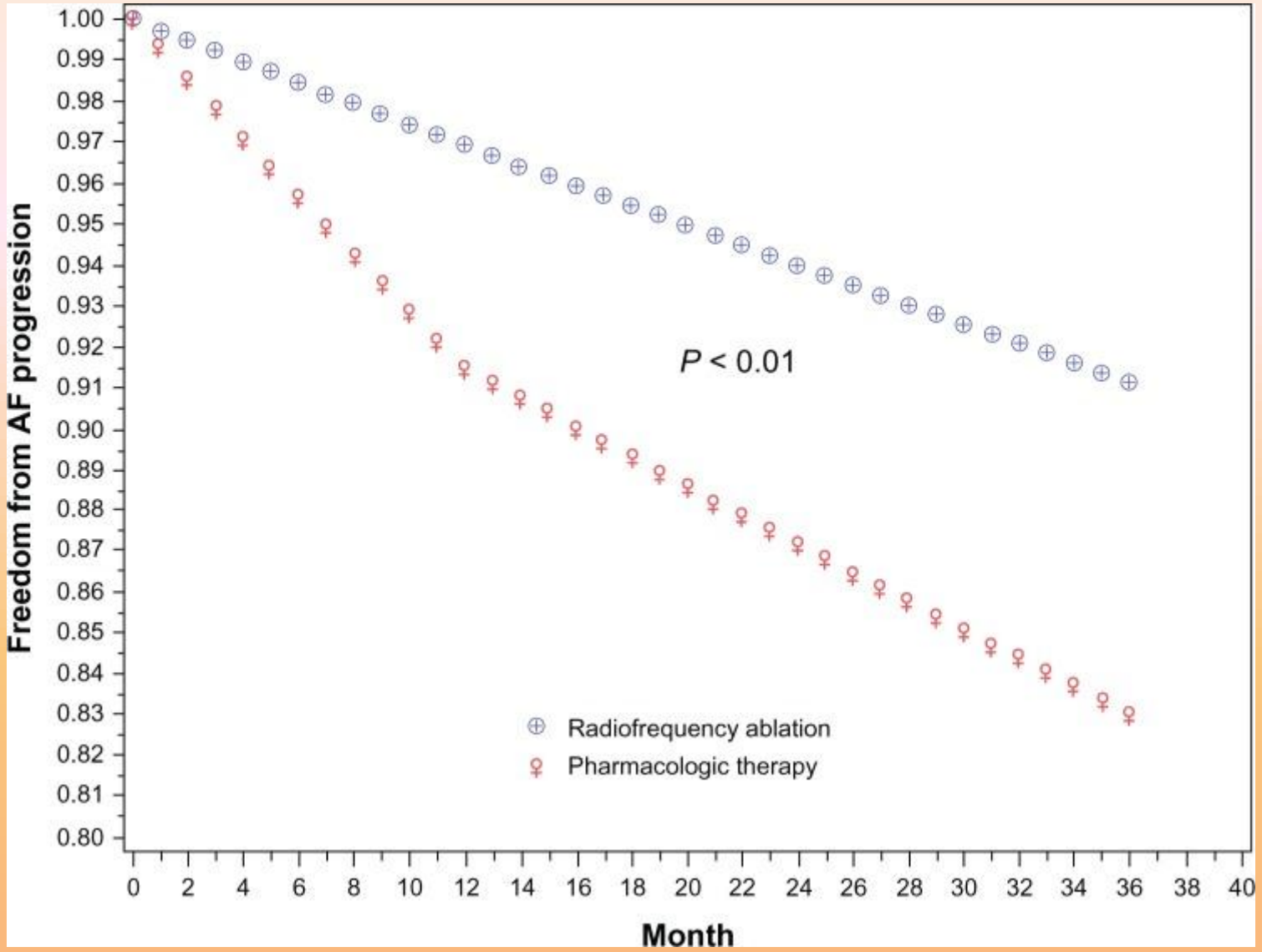
‡The first hemorrhage is the first or only hemorrhage for each patient.

§P values were calculated by the exact conditional binomial test for two independent Poisson processes.

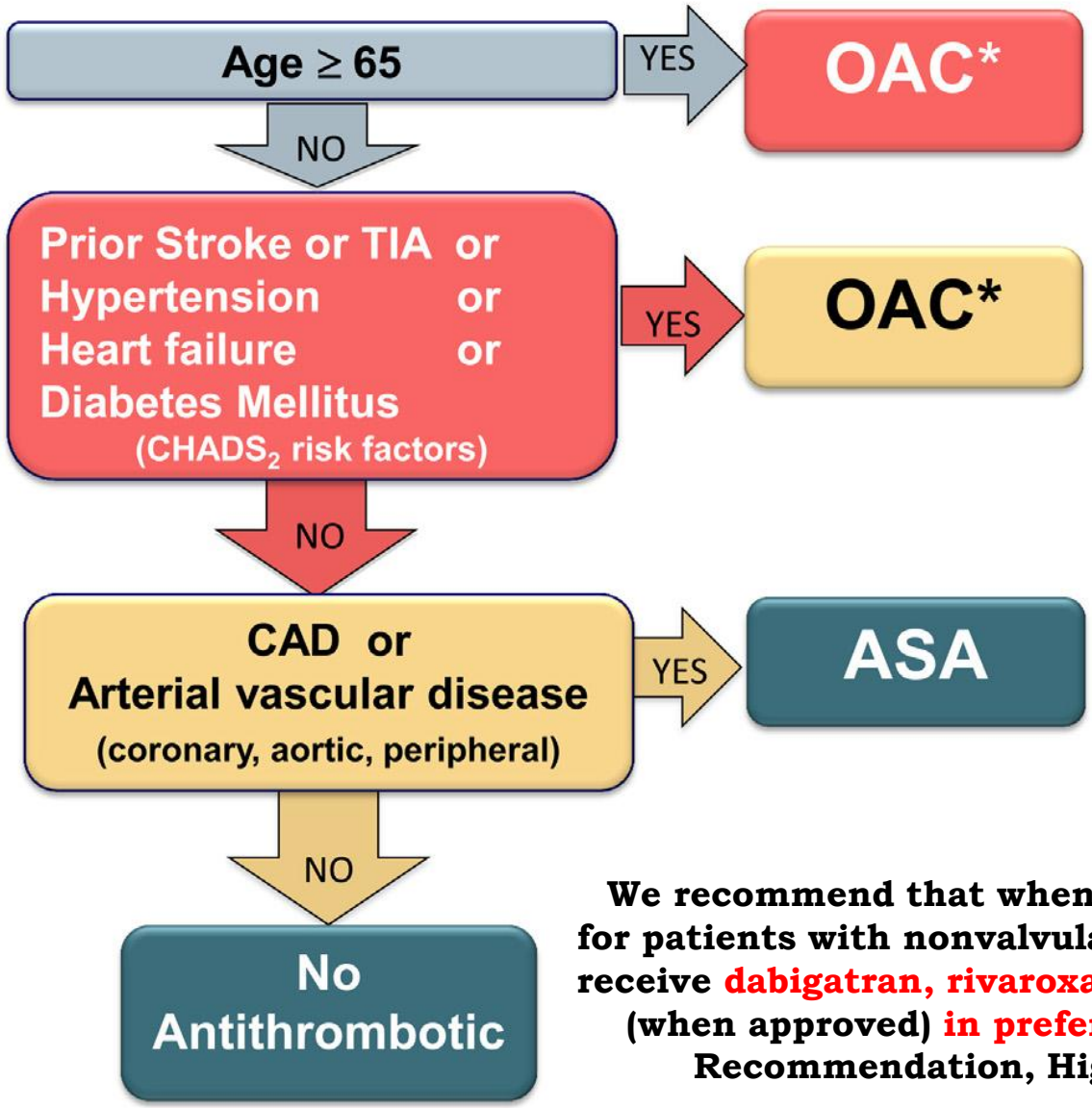
¶All hemorrhages include all hemorrhages in any patient.

Mohr JP et al. N Engl J Med. 2001;345(20):1444-51

CARDIOVERSIO HATÉKONYSÁGA



The "CCS Algorithm" for OAC Therapy in AF



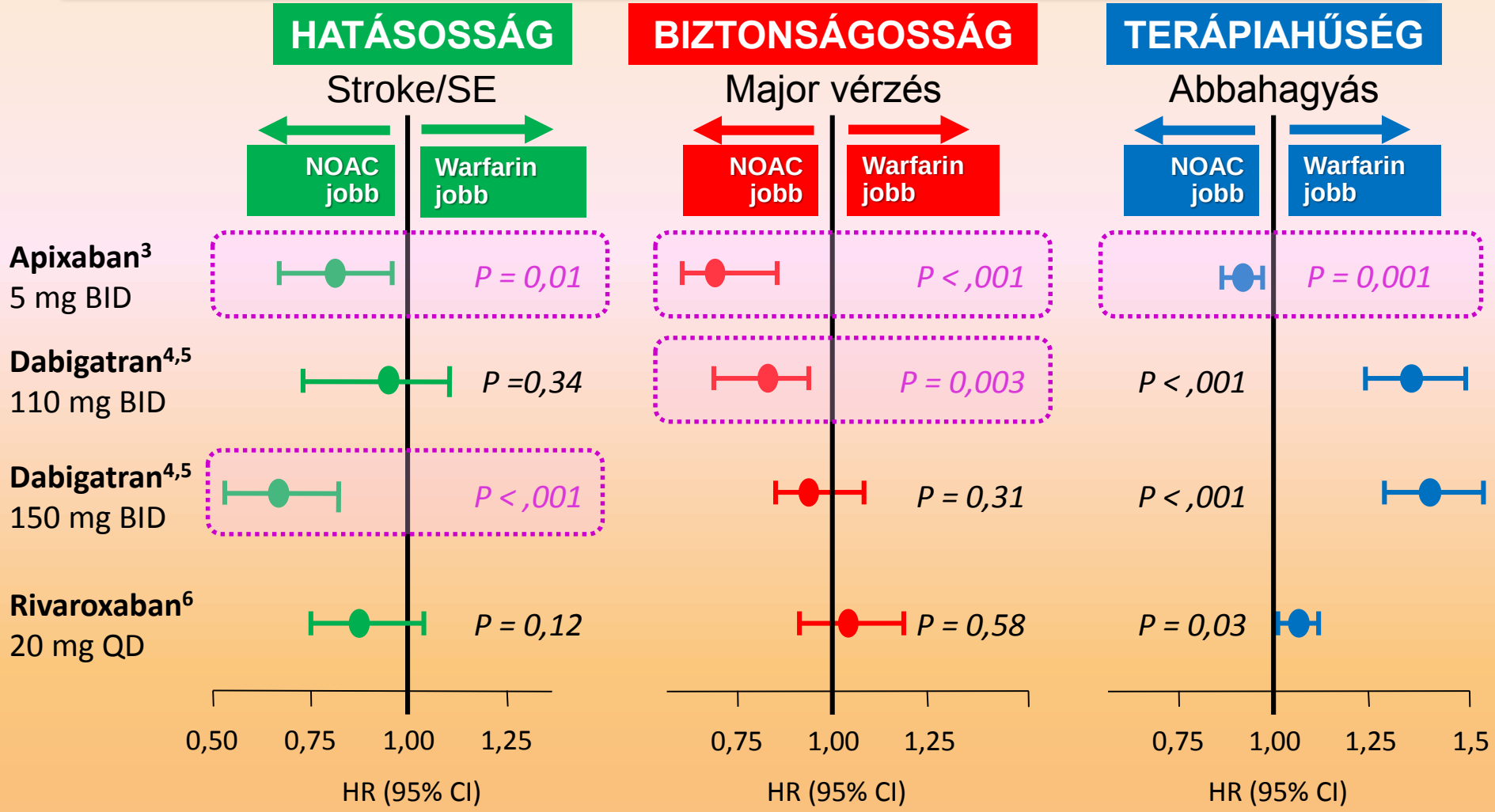
Consider and modify (if possible) all factors influencing risk of bleeding during OAC treatment (hypertension, antiplatelet drugs, NSAIDs, excessive alcohol, labile INRs) and specifically bleeding risks for NOACs (low eGFR, age ≥ 75, low body weight).†

We recommend that when OAC therapy is indicated for patients with nonvalvular AF, most patients should receive dabigatran, rivaroxaban, apixaban, or edoxaban (when approved) in preference to warfarin (Strong Recommendation, High-Quality Evidence).

Verma A et al. Can J Cardiol. 2014;30(10):1114-30.

A NOAC-ok klinikai profilja[1,2] (vs. warfarin):

Hatásosság ↑ Biztonságosság↑ Terápiahűség↑



Nincsenek a NOAC-okat közvetlen összehasonlító vizsgálatok

1. Mitchell SA, et al. Clin Appl Thromb Hemostas. 2013; 9(6) 619-631. 2. Pengo V, et al. J Thromb Haemost. 2012; 10: 1979–87.1. 3. Granger C, et al. N Engl J Med 2011;365:981–92; 4. Connolly SJ, et al. N Engl J Med 2009;361:1139–51; 5. Connolly SJ et al. N Engl J Med 2010;363:1875–6; 6. Patel MR, et al. N Engl J Med 2011;365:883–91;

432HU14PR04326-01, Pfizer ELI_2014_07_01, Lezárás dátuma: 2014. 07. 14.

VÉRZÉS NOAC MELLETT

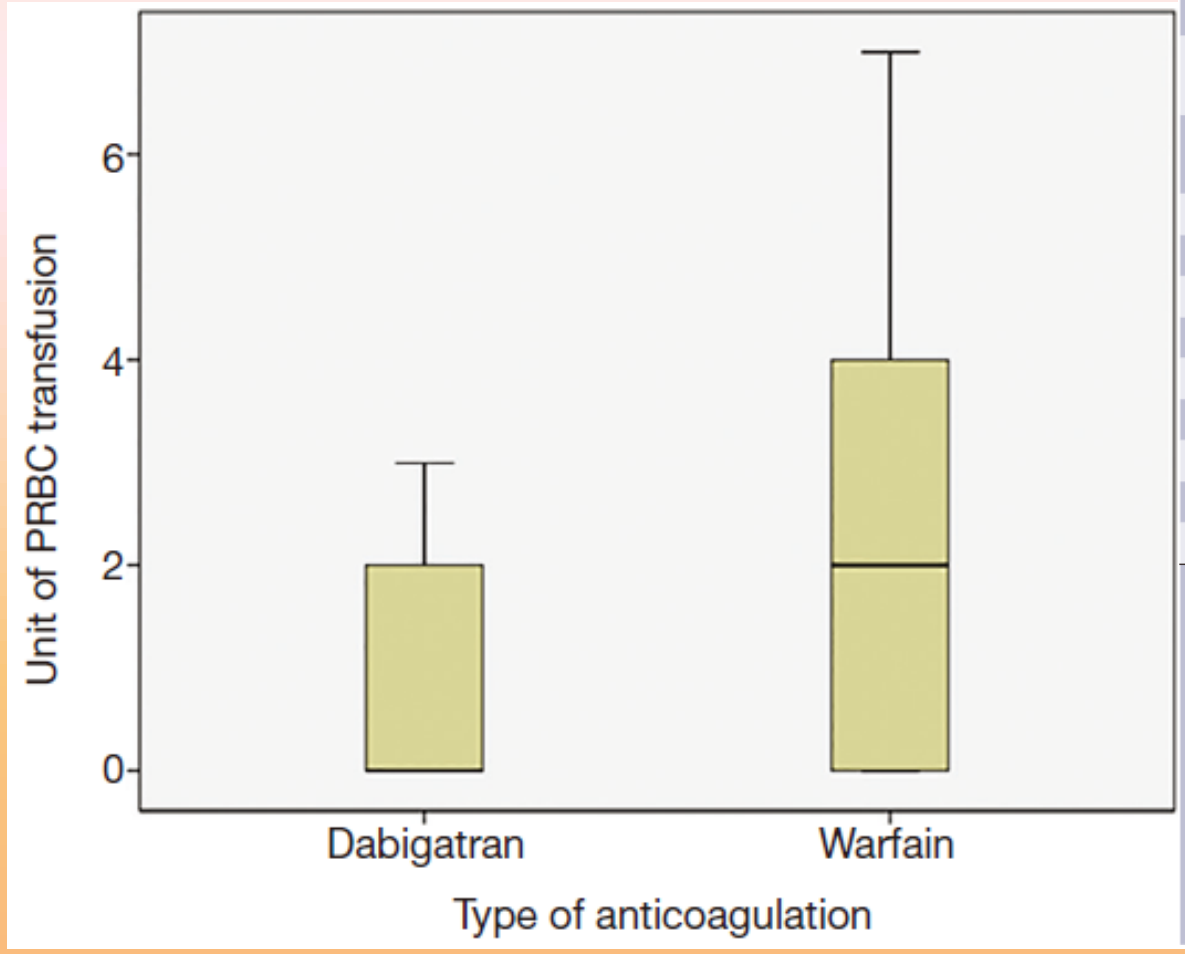


Table 2 Initial clinical presentation, initial laboratory results, and clinical outcomes of patients who presented with GI bleed in both groups

	Dabigatran	Warfarin	P value
Initial presentation			
UGIB n, (%)	0	1 (3.8)	–
LGIB n, (%)	11 (84.6)	20 (76.9)	0.61
Symptomatic anemia n, (%)	2 (15.4)	5 (19.2)	0.81
Hypotension n, (%)	1(7.7)	8(30.8)	0.11
Tachycardia n, (%)	3 (23.0)	5 (19.0)	0.78
Initial Hb at presentation (mg/dL)	10.4±2.1	9.6±2.6	0.34
Second Hb within 24 hr (mg/dL)	9.64±1.3	9.01±2.4	0.31
Platelet count (10^3/mm^3)	189±60	240±89	0.045
Creatinine (mg/dL)	1.35±1	1.35±0.8	0.99
INR	1.81±0.9	2.54±0.3	0.01
AKI n, (%)	4 (31.0)	5 (19.0)	0.42
PRBC transfusion (units)	0.69±1.1	1.92±2.2	0.024*
Length of stay (days)	5.6±4.9	5.9±4	0.86
ICU n, (%)	1 (7.7)	1 (3.8)	0.61
Death n, (%)	1 (7.7)	1 (3.8)	0.61
Endoscopy n, (%)	6 (46.0)	15 (58.0)	0.50

*, after multiple regression analysis correcting for history of CKD, and hemoglobin level at presentation, there is significant association between initial hemoglobin level at presentation, type of anticoagulation, and the quantity of PRBC transfusion with the higher amount of transfused PRBCs in the warfarin group. GI, gastrointestinal; UGIB, upper gastrointestinal bleeding; LGIB, lower gastrointestinal bleeding; Hb, hemoglobin; INR, international normalized ratio; AKI, acute kidney injury; PRBC, packed red blood cell; ICU, intensive care unit; CKD, chronic kidney disease.

Manatsathit W et al. Cardiovasc Diagn Ther 2014;4(3):224-231

II. HAEMORRHAGIÁS STROKE

SPONTÁN INCRACEREBRALIS VÉRZÉSEK

- Összes stroke 9-27%-a
- Ok lehet kisérbetegség, nagyérbetegség, vénás eredet, malformatio, coagulopathia
- 1 hónapos mortalitás akár 40%, egy éves mortalitás akár 54%

Tanaka E et al. Stroke. 2014;45:2275-2279

LEGFONTOSABB RIZIKÓFAKTOROK

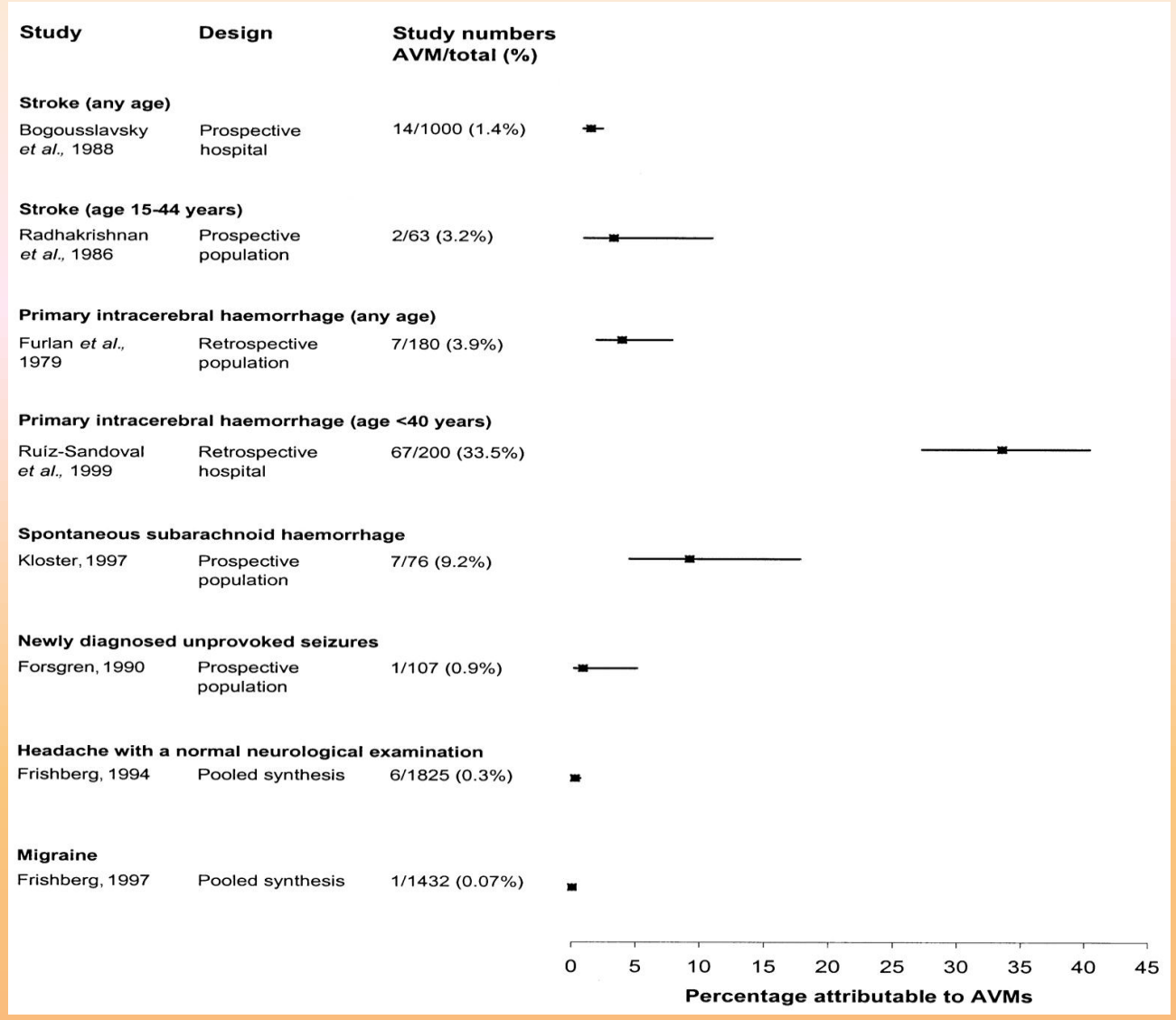
Table 3 Major risk factors for spontaneous intracerebral hemorrhage[a]

Risk Factor	Effect Size (95% Confidence Interval) [3]
Hypertension	Odds Ratio: 3.68 (2.52-5.38)
Age (every 10 year rise)	Risk Ratio: 1.97 (1.79-2.16)
Current smoking	Odds Ratio: 1.31 (1.09-1.58)
Diabetes Mellitus	Odds Ratio: 1.30 (1.02-1.67)
High alcohol intake (>56 grams/day)	Odds Ratio: 4.11 (2.54-6.65)
Moderate alcohol intake (<56 grams/day)	Odds Ratio: 2.05 (1.35-3.11)

[a]Other risk factors include drug abuse (e.g., cocaine, amphetamines), excessive anticoagulation, trauma, low serum cholesterol, thrombolytic therapy, and revascularization procedures

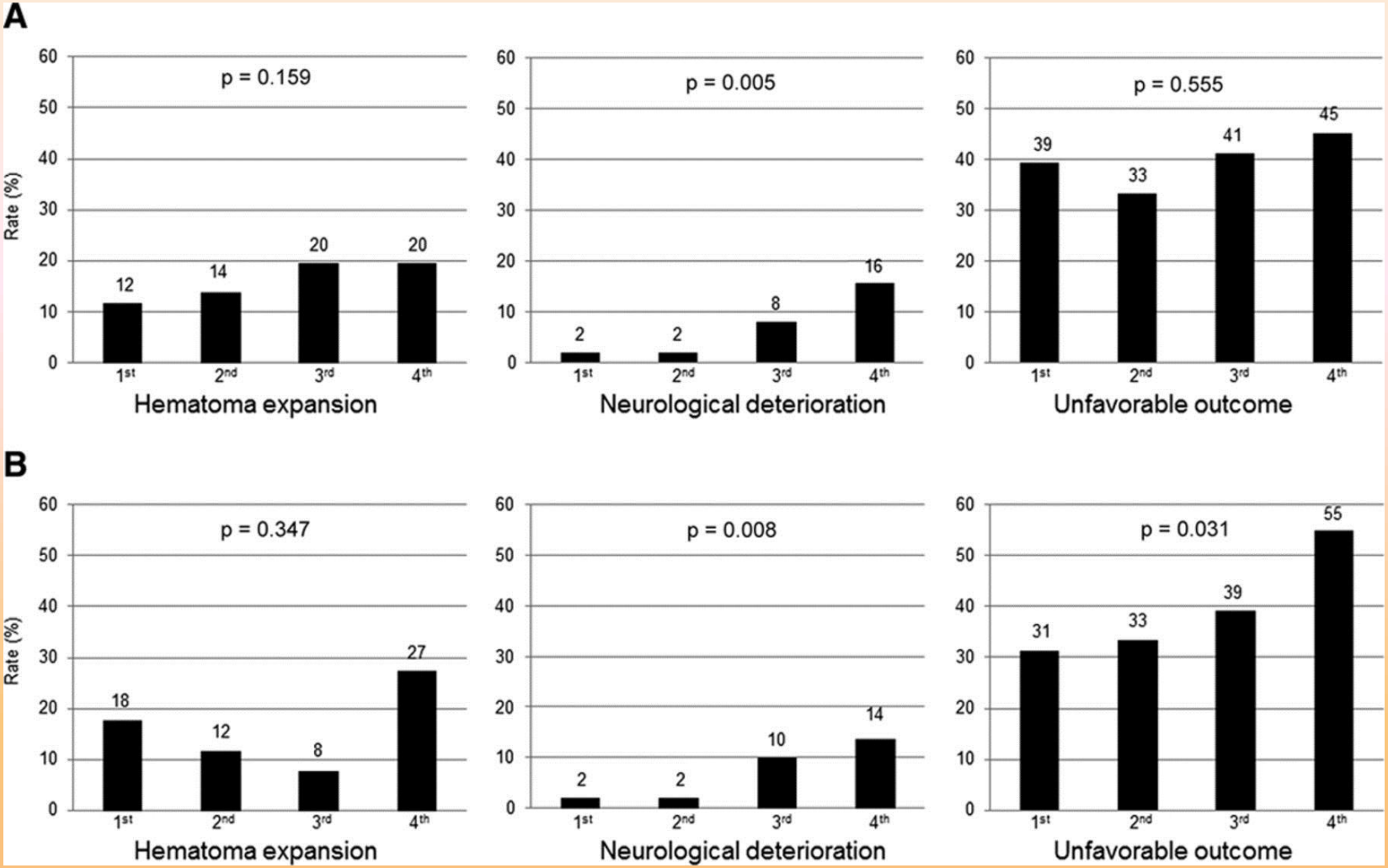
Ariesen MJ et al. Stroke. 2003;34:2060-2065
Ovbiagele B. et al Neurotherapeutics. 2011;8(3):319-29.

AV MALFORMATIO



Al-Shahi R t al. Brain 2001;124:1900-1926

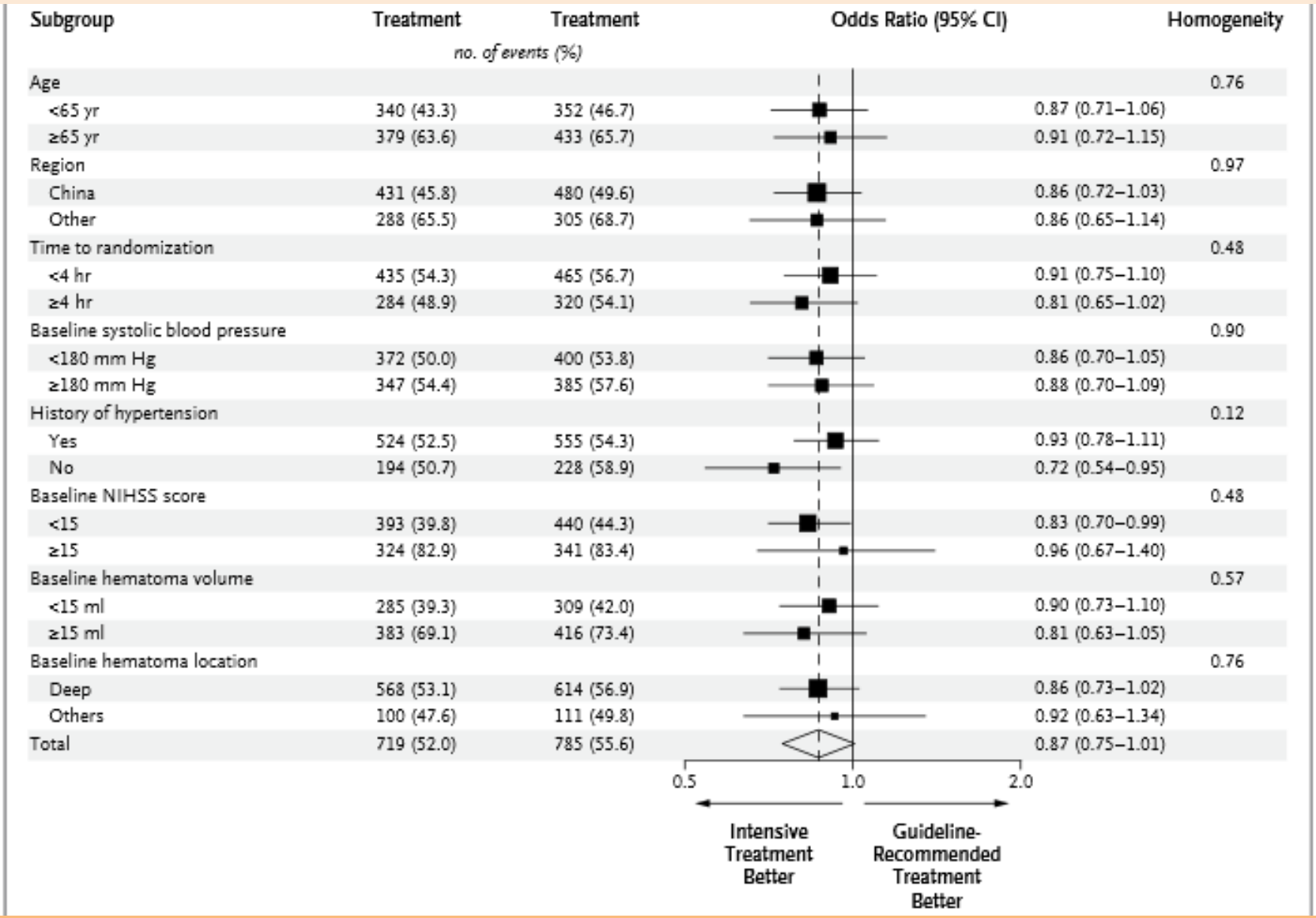
KORAI VÉRNYOMÁSCSÖKKENTÉS



Tanaka E et al. Stroke. 2014;45:2275-2279

American Heart Association.

KORAI VÉRNYOMÁSCSÖKKENTÉS



Subgroup	Treatment	Treatment	Odds Ratio (95% CI)	Homogeneity
	no. of events (%)			
Age				0.76
<65 yr	340 (43.3)	352 (46.7)	0.87 (0.71–1.06)	
≥65 yr	379 (63.6)	433 (65.7)	0.91 (0.72–1.15)	
Region				0.97
China	431 (45.8)	480 (49.6)	0.86 (0.72–1.03)	
Other	288 (65.5)	305 (68.7)	0.86 (0.65–1.14)	
Time to randomization				0.48
<4 hr	435 (54.3)	465 (56.7)	0.91 (0.75–1.10)	
≥4 hr	284 (48.9)	320 (54.1)	0.81 (0.65–1.02)	
Baseline systolic blood pressure				0.90
<180 mm Hg	372 (50.0)	400 (53.8)	0.86 (0.70–1.05)	
≥180 mm Hg	347 (54.4)	385 (57.6)	0.88 (0.70–1.09)	
History of hypertension				0.12
Yes	524 (52.5)	555 (54.3)	0.93 (0.78–1.11)	
No	194 (50.7)	228 (58.9)	0.72 (0.54–0.95)	
Baseline NIHSS score				0.48
<15	393 (39.8)	440 (44.3)	0.83 (0.70–0.99)	
≥15	324 (82.9)	341 (83.4)	0.96 (0.67–1.40)	
Baseline hematoma volume				0.57
<15 ml	285 (39.3)	309 (42.0)	0.90 (0.73–1.10)	
≥15 ml	383 (69.1)	416 (73.4)	0.81 (0.63–1.05)	
Baseline hematoma location				0.76
Deep	568 (53.1)	614 (56.9)	0.86 (0.73–1.02)	
Others	100 (47.6)	111 (49.8)	0.92 (0.63–1.34)	
Total	719 (52.0)	785 (55.6)	0.87 (0.75–1.01)	

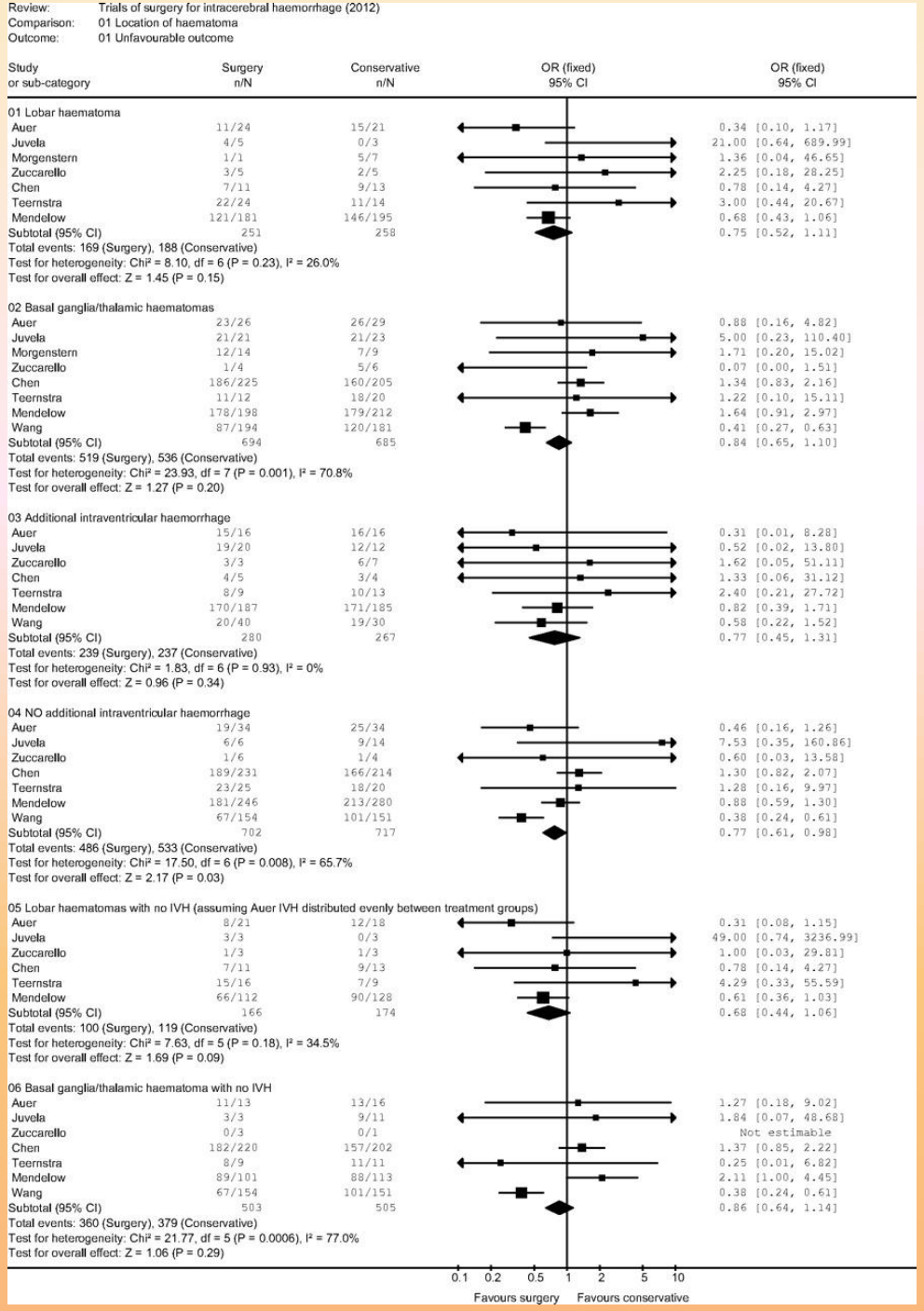
Anderson CS et al. N Engl J Med. 2013;368(25):2355-65.

VÉRZÉS OAC KEZELÉS MELLETT

- K-vitamin önmagában kevés – csak az orvosi lelkiismeret megnyugatása (legalább adtunk valamit).

- Friss fagyasztott plazma, vagy protromplex adása (favorizált – tudjuk a benne levő alvadási faktorok mennyiségét) is szükséges

Majeed A et al. Stroke 2010;41:2860–6.



MŰTÉTI KEZELÉS

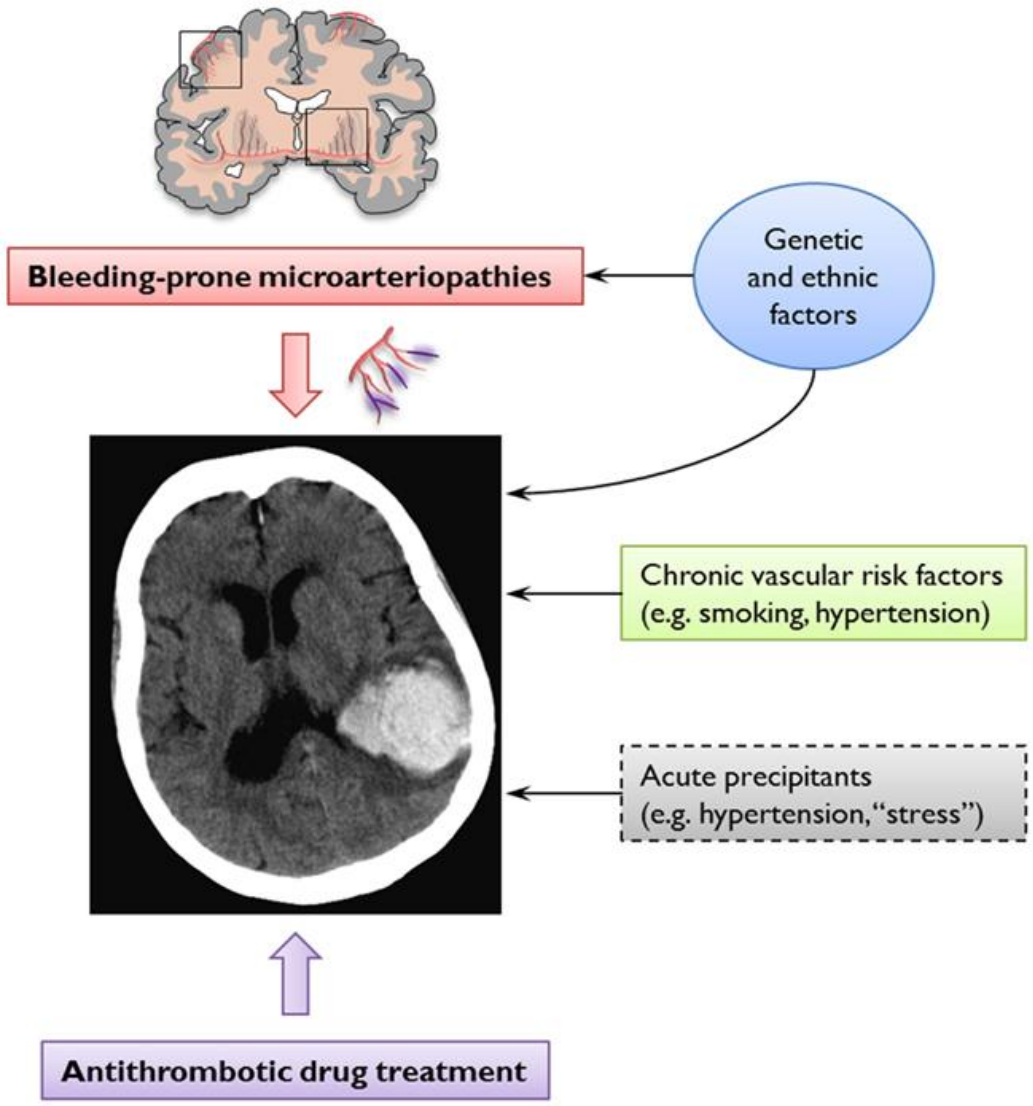
Rutinszerűen nem javasolt

Vannak bizonyos kivételek (pl IV. kamra kompresszió stb), de ehhez teammunka szükséges (radiológus-neurológus–idegsebész)

American Heart Association

Gregson B A et al. Stroke. 2012;43:1496-1504

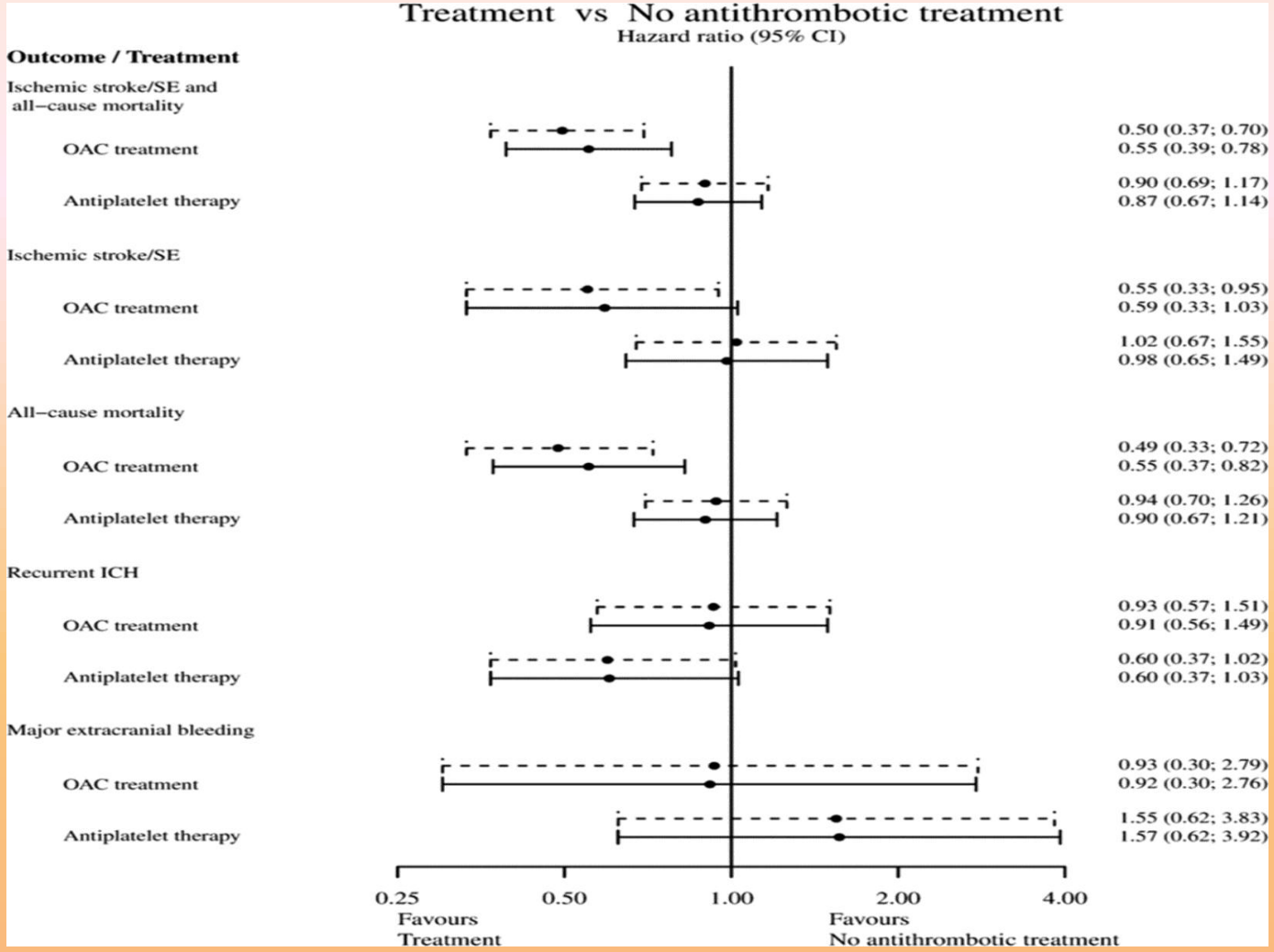
VÉRZÉS UTÁNI ANTIKOAGULÁLÁS



Ezen betegek gyakran nem kapnak újra sem antithromboticus, sem antikoaguláns kezelést – noha ischaemiás esemény sokkal gyakoribb, mint az újravérzés kockázata

Pennlert et al. Stroke. 2015;46:2094-2099.
Charidimou et al. Front Neurol. 2012;3:133.

VÉRZÉS UTÁNI ANTIKOAGULÁLÁS PITVARFIBRILLÁLÓ BETEGEKNÉL



Peter Brønnum Nielsen et al. Circulation. 2015;132:517-525

III. SUBARACHNOIDEALIS VÉRZÉS

SUBARACHNOIDEALIS VÉRZÉS

- Ütésszerű fejfájás, „életem legrosszabb fejfájása”
- 12 órán belül elvégzett koponya CT sensitivitása ~98%
- Harmadik nap után 73%-ra csökken az érzékenység
- Magnetic resonance imaging (MRI) szekvenciától függően jóval érzékenyebb (T2 gradiens echo 94-100%, FLAIR 81-87%) konzekvesen
- Legkisebb gyanú esetén is lumbalpunctio elvégzése kötelező (esetlegesen MR kiválthatja)

Schattlo B et al. Swiss Med Wkly. 2014;144:w13934.

RIZIKÓFAKTOROK

Nem befolyásolható rizikófaktorok:

1. Nem
2. Kor
3. Elhelyezkedés
4. Családi halmozódás

Befolyásolható tényezők:

1. Dohányzás
2. Hipertónia
3. Jelentős alkoholbevitel

De novo kialakulás rizikófaktorai:

1. Nem
2. Dohányzás
3. Hipertónia
4. Életkor
5. Pozitív családi anamnesis

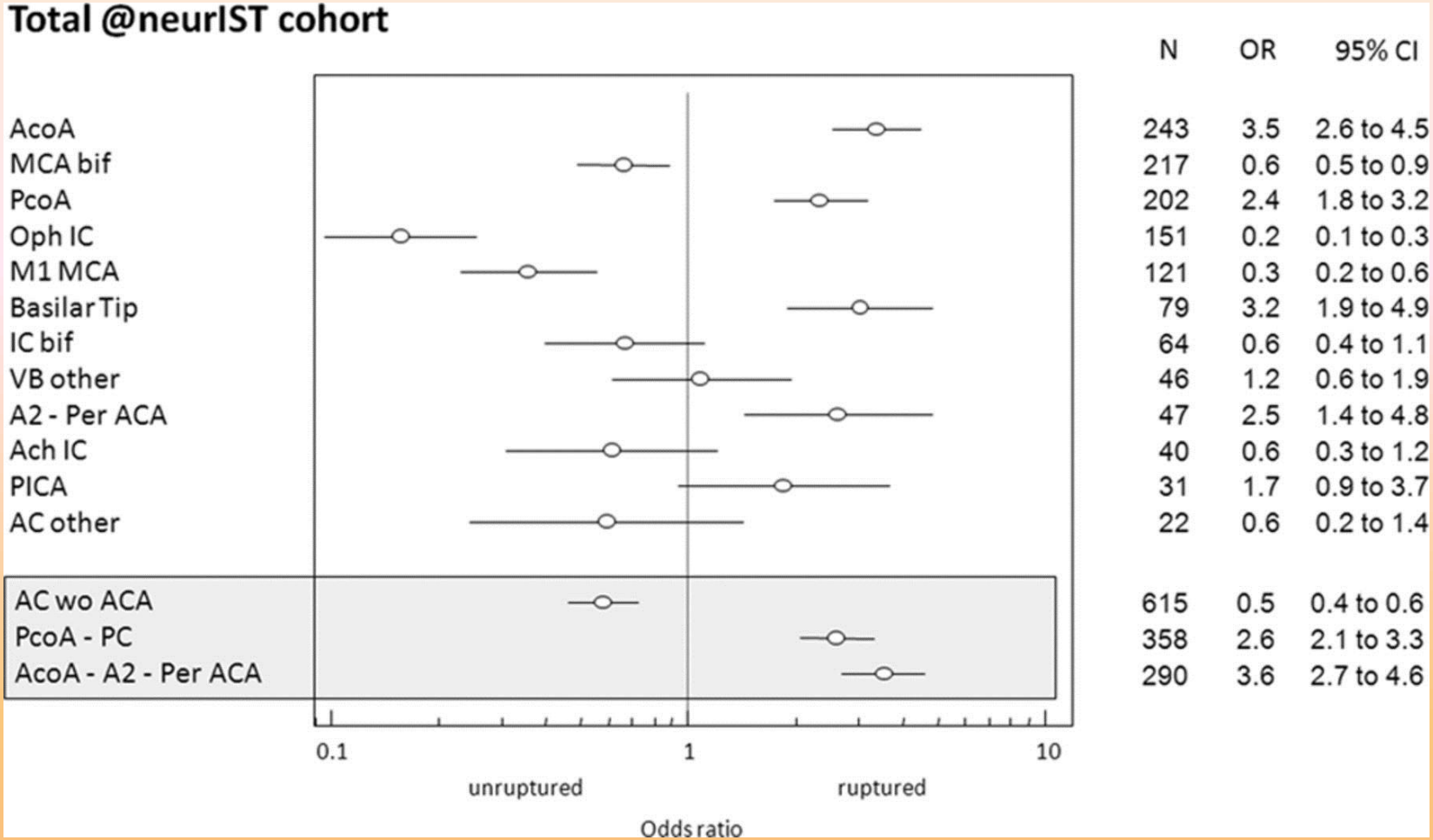
Növekedés egyetlen bizonyított tényezője a dohányzás!!!

Steiner T. et al. Cerebrovasc Dis. 2013;35(2):93-112

TÜNETMENTES ANEURYSMA

Lokalizáció, méret és pozitív anamnesis a ruptúra legfontosabb előrejelzői, különös tekintettel a **hátsó scalai** aneurysmákra, **mérettől függetlenül** és a **7mm-nél nagyobb elülső** keringésben elhelyezkedő aneurysmákra

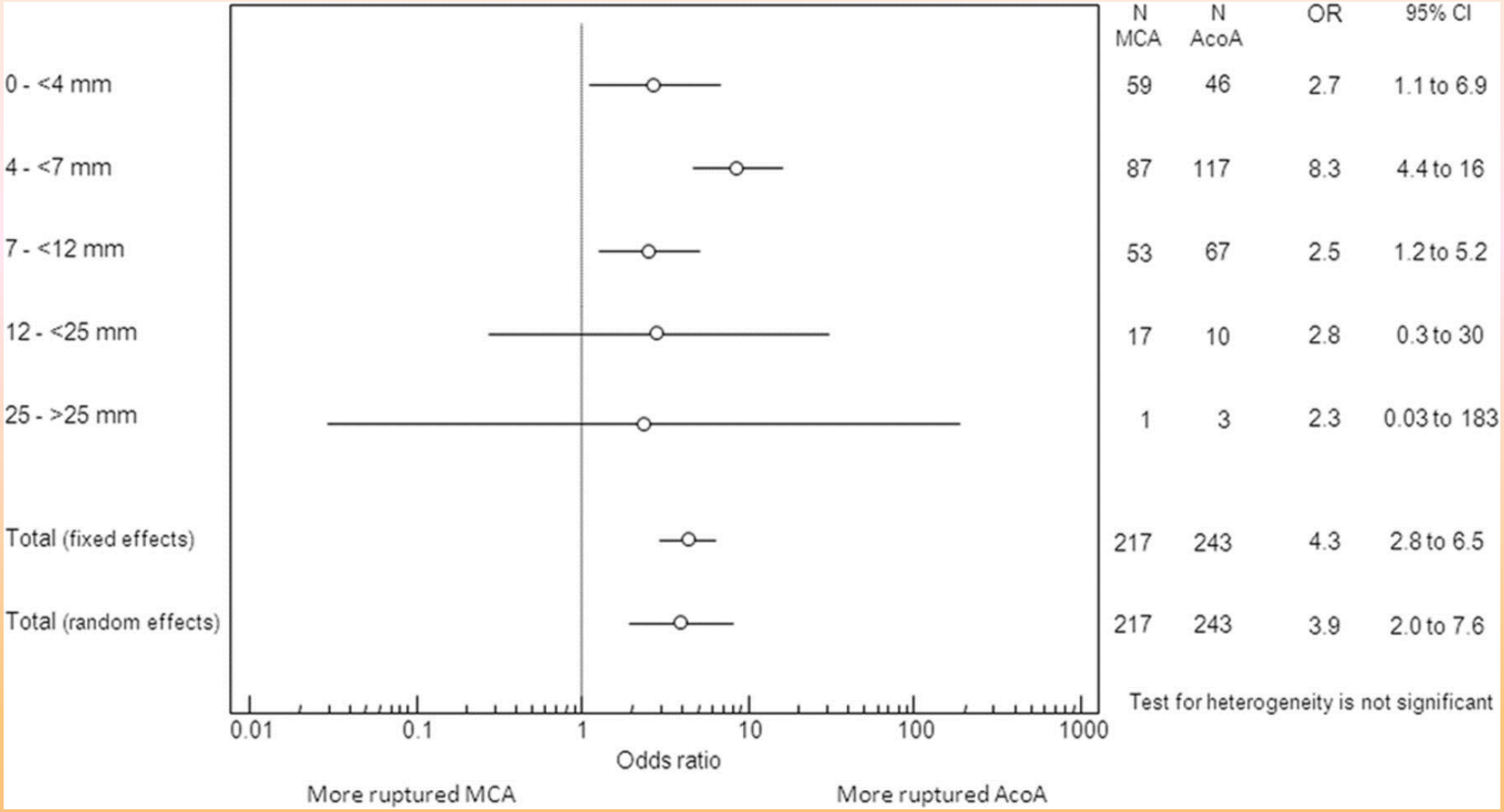
ÖSSZEVETÉS



Bijlenga P et al. Stroke. 2013;44:3018-3026

American Heart Association.

RUPTÚRARIZIKÓ



Bijlenga P et al. Stroke. 2013;44:3018-3026

American Heart Association.

IV. SINUS THROMBOSIS

ÜTÉSSZERŰ FEJFÁJÁS

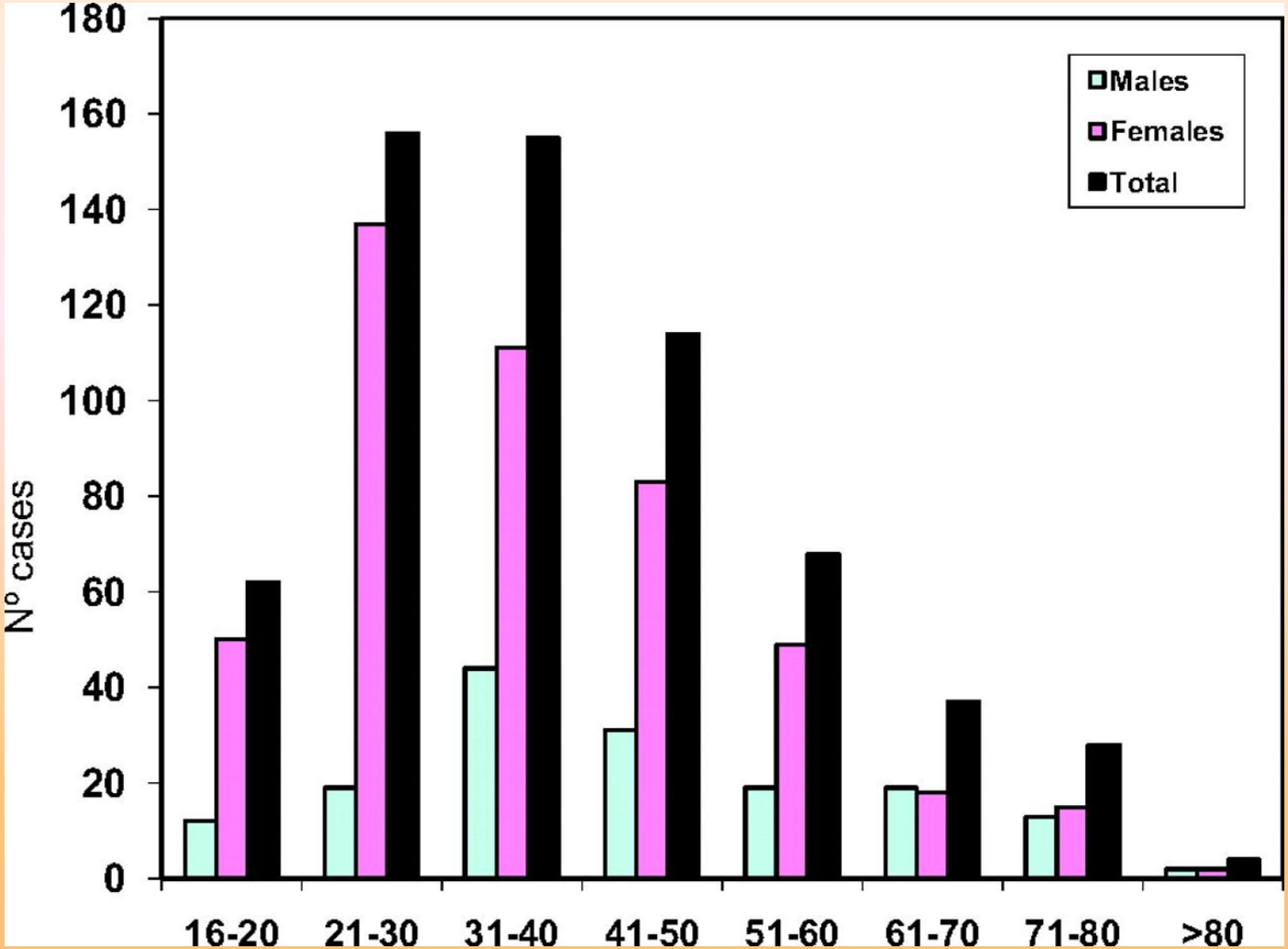
Table 1

Disorders Associated with Thunderclap Headache

Subarachnoid hemorrhage
Unruptured intracranial aneurysm
Cervical artery dissection
Ischemic stroke
Cerebral venous sinus thrombosis
Intraparenchymal hemorrhage
Spontaneous intracranial hypotension
Reversible cerebral vasoconstriction syndromes
Reversible posterior leukoencephalopathy
Infection: intracranial, sinusitis
Primary thunderclap headache

Ju YE. et al. Semin Neurol. 2010;30(2):192-200

ÉLETKORI ÉS NEMBELI MEGOSZLÁS



Saposnik G et al. Stroke. 2011;42:1158-1192

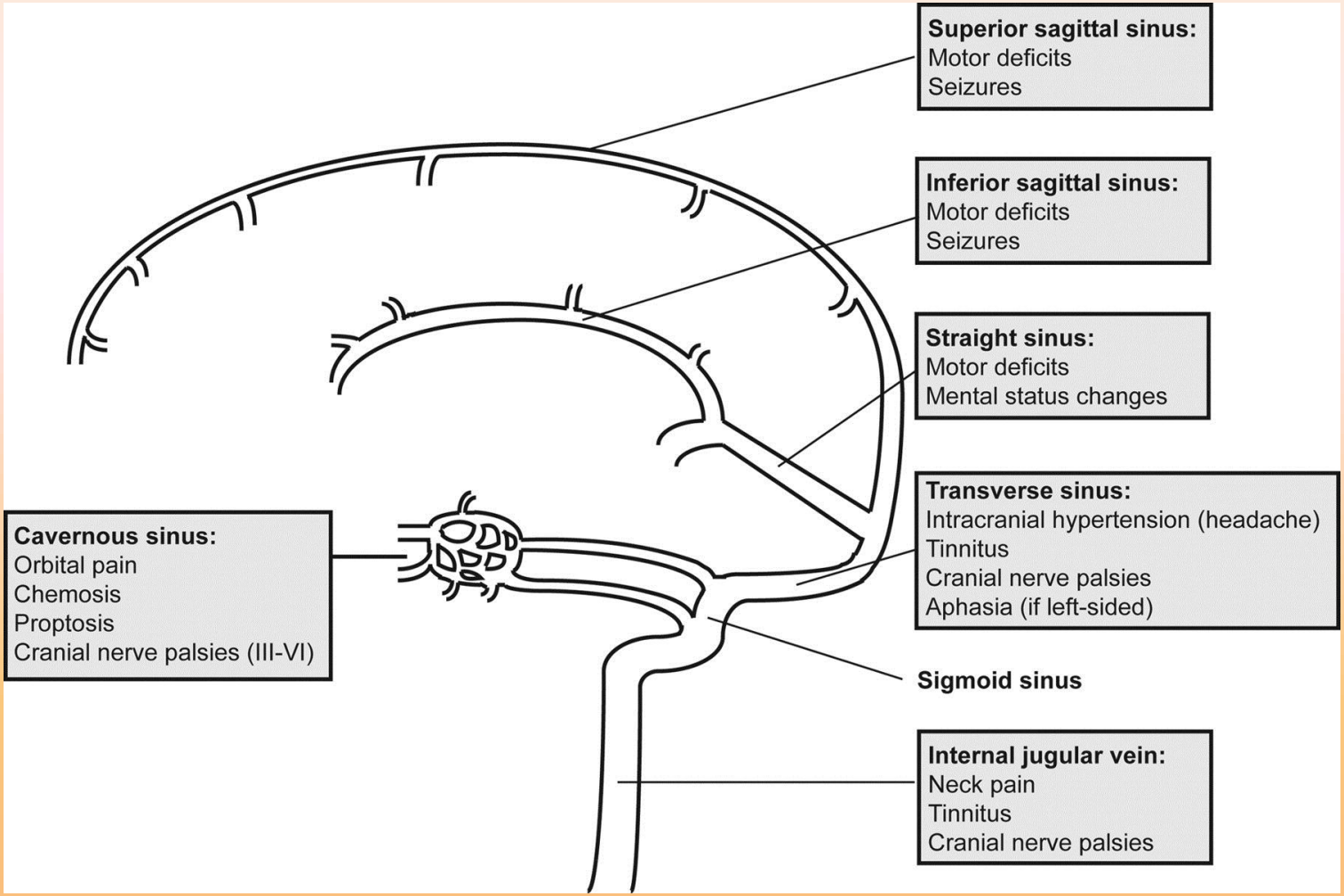
American Heart Association.

RIZIKÓFAKTOROK

• **Thrombophylia**	**34,5 %**
• Malignitás	7,4 %
• Központi idegrendszeri anomáliák	1,9 %
• **Hematológiai eltérések (anaemia, polycytaemia)**	**12%**
• Vasculitis	3%
• **Terhesség, puerperium**	**20,1 %**
• **Infectio (CNS, sinusitis)**	**12,3%**
• Mechanikai ok	4,5 %
• Gyógyszerek (~50 % OAC kezelés!)	7,5 %
• Sebészi beavatkozás	2,7 %
• Dehydratio	1,9 %

Ferro JM et al. Stroke. 2004;35(3):664-70

TÜNETTAN



Piazza G Circulation. 2012;125:1704-1709

Do we have to anticoagulate patients with cerebral venous thrombosis?

G. FEHER [1], Z. ILLES [2], D. HARGROVES [3], S. KOMOLY [4]

[1]Szigetvar Hospital, Szigetvár, Hungary; [2]University of Southern Denmark, Odense, Denmark; [3]William Harvey Hospital, Kent, UK; [4]University of Pecs, Pécs, Hungary

Cerebral venous thrombosis (CVT) is a rare form of venous thromboembolism (VTE). Although anticoagulation is recommended for the initial and long term treatment with regards to thrombotic risks for patients with CVT, the role of anticogalution has not been fully elucidated. The aim of our literature based review was collect articles showing the benefit of anticoagulation in CVT and gathering the data of follow-up studies focusing on the recurrence of CVT and other thrombotic events. We have identified 15 follow-up studies studies with 2422 patients. The mean duration of follow-up was 37.9 months. Death occured in 6.5% and 76.4% of the patients had favorable outcome; 85.5% received initial anticoagulation with ultrafractionated or low molecular weight heparin and 82.1% received long-term anticoagulation. Recurent CVT occured in 3.7% and other thrombotic event occured in 5.4%. The mentioned studies have led to incoclusive results with regards to the clinical outcome and the presence or absence of anticoagulation. The role of long term anticoagulation should be clarified in randomized multicentre studies as the recurrence rate seems to be low and the outcome of a second event as good as that of the first one irrespective of underlying risk factors.

[Int Angiol 2015;34:1-2]

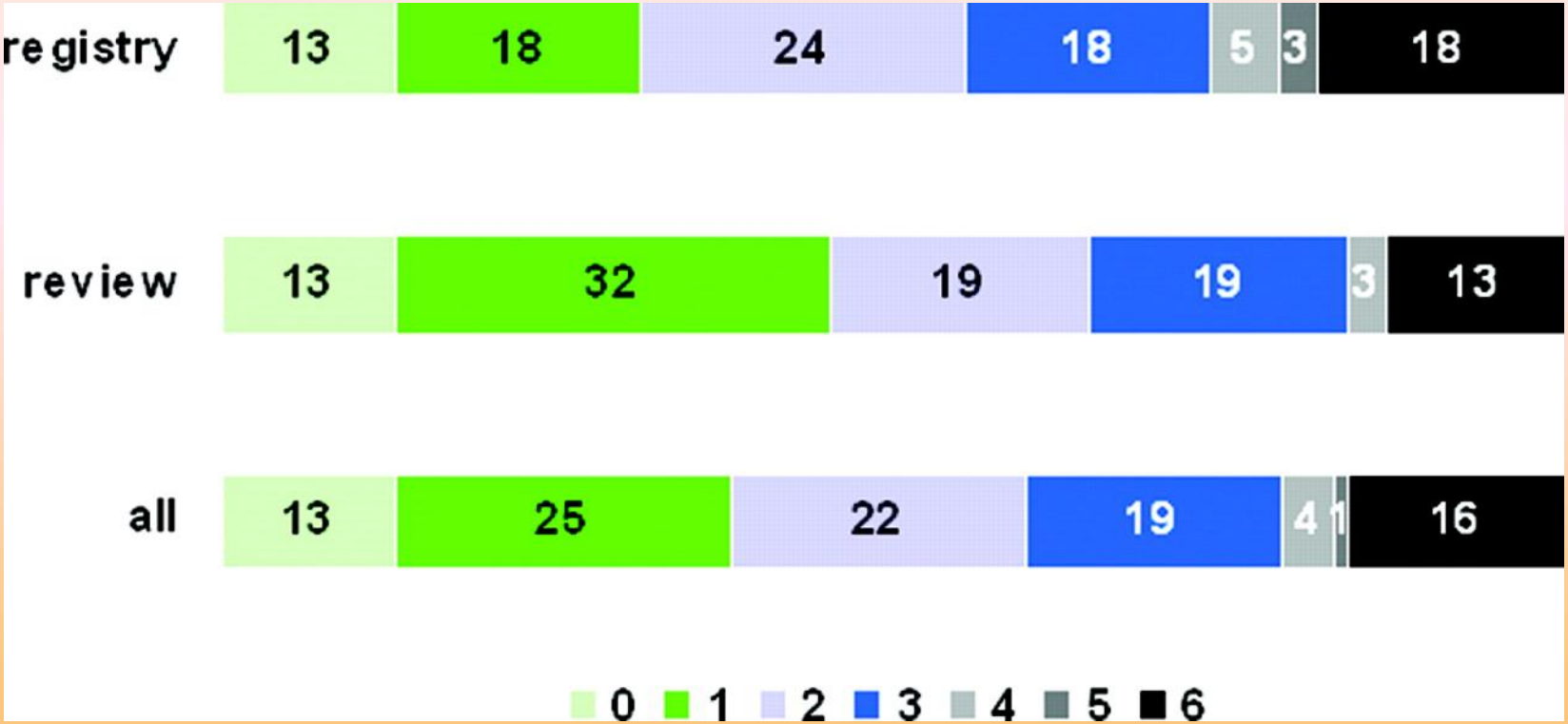
Key words: **Recurrence - Follow-up studies - Therapeutics.**

have completely modified our knowledge about the nature and clinical outcome of CVT showing that this is a relatively benign condition with favourable outcome for the vast majority of the patients.[5] Randomized studies have showed the overall benefit of early anticoagulation, so it has been included into the current guidelines, however, it is not supported by robust evidence.[6, 7]

The first large trial on CVT, the multicentre International Study on Cerebral Vein and Dural Sinus Thrombosis enrolling 624 consecutive patients showed that the recurrence of cerebral and other venous events are rare after a CVT (2.2% had a recurrent sinus thrombosis and 4.3% had other thrombotic events) and nearly half of these patients (41.5%) were on anticoagulants at the time of the event.[8] It raises the question whether routine anticoagulation in necessary or not.

The aim our review was to collect articles showing the benefit of anticoagulation in CVT and gathering the data of follow-up studies focusing on the recurrence of CVT and other

KIMENETEL MŰTÉTI BEAVATKOZÁS UTÁN



Ferro J M et al. Stroke. 2011;42:2825-2831

American Heart Association.

KÖSZÖNÖM A FIGYELMET!