RHNe Réseau Hospitalier Neuchâtelois

L'AVC: perspectives sur la prise en charge

Pavel-Dan NANU

Neurologie FMH

Maladies cérébrovasculaires et électroencéphalographie

Médecin chef adjoint Neurologie RHNe

RHNe Réseau Hospitalier Neuchâtelois

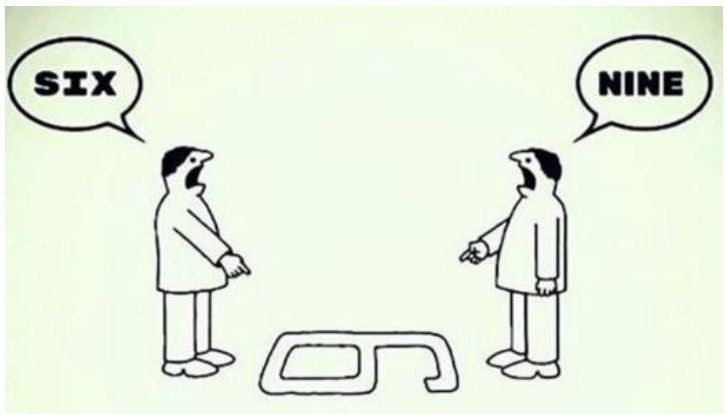
perspective

nom féminin

1. Vue qu'on a d'un lieu.
   Synonyme :
   coup d'œil, échappée, paysage, point de vue, site, vue.

2. Manière de voir.
   Synonyme :
   angle, aspect, attente, côté, éclairage, face, facette, jour, optique, pensée, point de vue, prévision, vision.

3. Ce qu'on pense possible.
   Synonyme :
   conjecture, débouché, espérance, éventualité, expectative, horizon, idée.



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Deaths from cardiovascular diseases

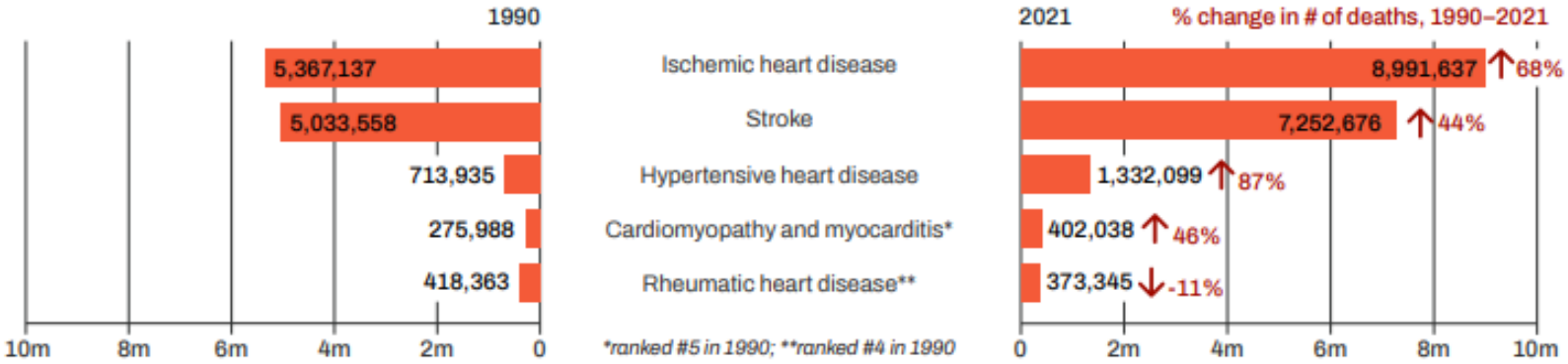
1990

12 330 009

2021

19 414 853

Top causes of cardiovascular disease deaths, 1990 and 2021[1]



[1]*For more information about cardiovascular diseases modeled in the Global Burden of Disease study, please visit healthdata.org/research-analysis/diseases-injuries/factsheets.*

Review

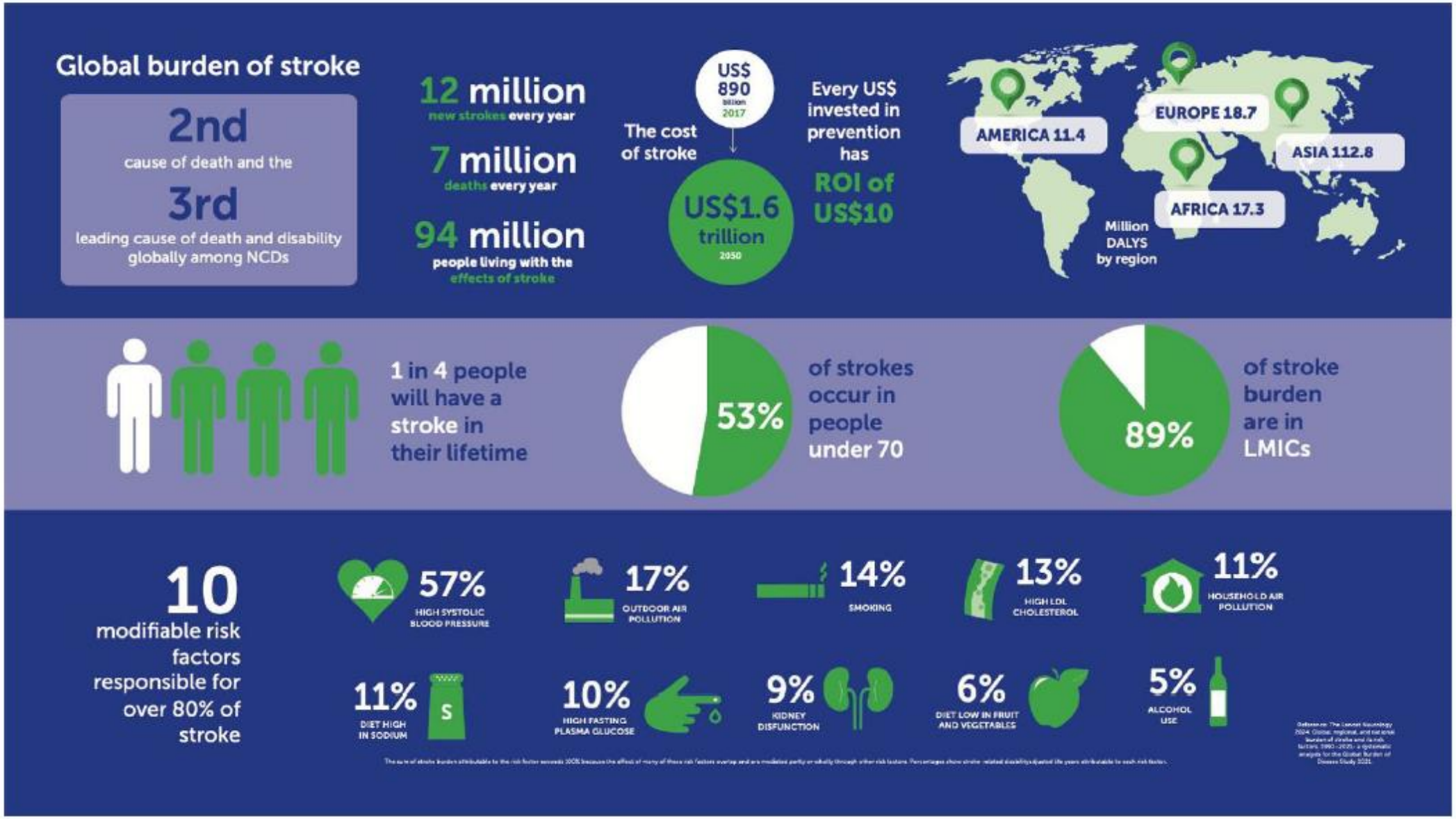
International Journal of Stroke WSO

International Journal of Stroke
2025, Vol. 20(2) 132–144

DOI: 10.1177/17474930241308142
journals.sagepub.com/home/wso
Sage

World Stroke Organization: Global Stroke Fact Sheet 2025

Valery L Feigin[1], Michael Brainin[2], Bo Norrving[3], Sheila O Martins[4], Jeyaraj Pandian[5], Patrice Lindsay[6], Maria F Grupper[7] and Ilari Rautalin[1,8]



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Perspectives sur…

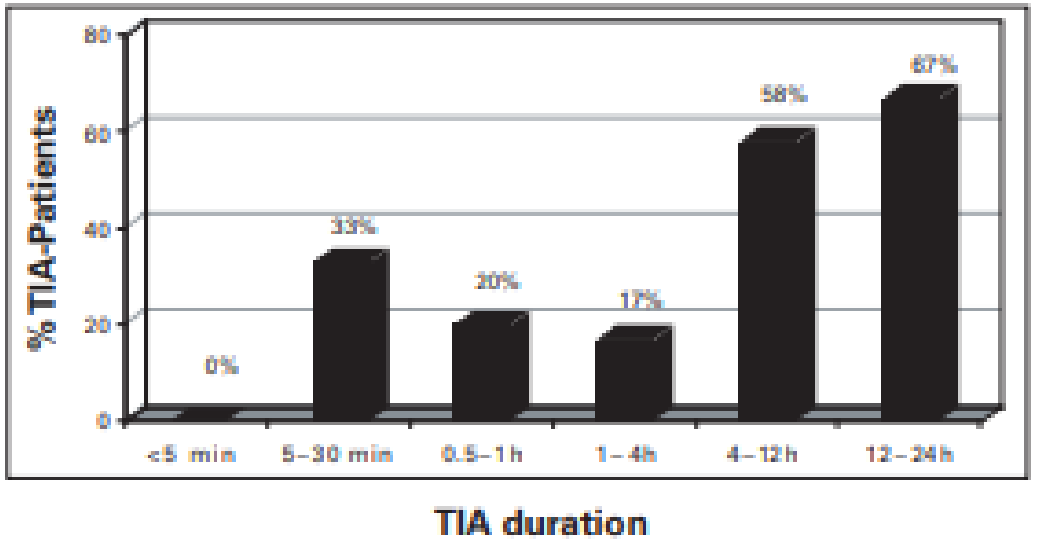
- L’accident ischémique transitoire
- L’accident vasculaire cérébral ischémique
- L’accident vasculaire cérébral hémorragique

L'accident ischémique transitoire (AIT) – définition

- épisode bref, début brutal et maximal de déficit neurologique focal cérébral, médullaire ou rétinien
- signes cliniques spécifiques
- durée - habituellement moins d'une heure, < 24h
- pas d'évidence de lésion ischémique à l'imagerie

Figure 1

Symptom duration correlates with the likelihood of visualised DWI lesion in TIA patients (adapted from [16]).



The clinical significance of diffusion-weighted MR imaging in stroke and TIA patients

Stefan T. Engelter, Stephan G. Wetzel, Leo H. Bonati, Felix Fluri, Philippe A. Lyrer
Neurological Clinic, University Hospital and Felix Platter-Spital Basel, Basel, Switzerland

SWISS MED WKLY 2008;138(49–50):729–740 · www.smw.ch

On a tous cette perspective de recevoir en consultation un patient qui…

- hier matin
- déficit moteur de 5 minutes du bras gauche, possiblement trainait la jambe gauche, en ayant remarqué des difficultés pour descendre les escaliers
- il n'a jamais eu ces symptômes auparavant
- n'est pas stressé car les symptômes étaient passagers

… et a décidé de nous le dire ce jour quand il a RDV pour son vaccin grippe et COVID…

- Examen neurologique sp
- TA 156/82 à gch connu pour une HTA

Qui de nous aurait envie de prescrire de l'Aspirine Cardio car il n'a pas de traitement anti thrombotique et puis organiser une IRM cérébrale, consultation neurologique… etc ?

Est-ce qu'un AIT est toujours un AIT?

- June 1, 2000, to August 31, 2014
- transient deficits (symptoms, signs, or both) from an ICH that had resolved on a repeated examination within 24 hours were eligible.
- 2137 patients had a spontaneous ICH
- **34 patients had transient deficits**, which were defined as clinical symptoms and signs that had resolved within 24 hours.

Table. Baseline Characteristics of the Patient Cohort

Sex/Age, y	Symptom	Initial Examination Findings	Duration	Hemorrhage Volume, mL	Site	Etiology
M/mid-20s	Right-sided paresthesias	BP 120/90 mm Hg; sensory loss; NIHSS score 2	<24 h	11.7	Left subinsular region	Cavernoma
M/mid-70s	Left foot numbness, arm clumsiness, slurred speech	BP 198/92 mm Hg; left pronator drift, extinction; NIHSS score 2	4 h	14.9	Right internal capsule	Hypertensive
F/mid-60s	Headache, left tongue and lip numbness	BP 200/100 mm Hg; normal NIHSS score 0	30 min	7	Right basal ganglia	Hypertensive
M/early 60s	Severe word-finding difficulties	BP 220/98 mm Hg; normal NIHSS score 0	30 min	29	Left temporal	Probable amyloid angiopathy
F/late 50s	Nausea, vomiting, left-sided weakness, dysarthria	BP 120/80 mm Hg; mild left facial droop, left pronator drift; NIHSS score 2	30 min	32.6	Right internal capsule	Possibly hypertensive
M/early 70s	Speech arrest	BP 201/89 mm Hg; mild anomia, right facial droop; NIHSS score 3	15 min	14.8	Left temporal	Hypertensive or amyloid angiopathy
M/early 70s	Dizziness, slurred speech, gait unsteadiness	BP 176/100 mm Hg; mild dysarthria, left pronator drift; NIHSS score 2	30 min	10.7	Left basal ganglia	Hypertensive
F/early 80s	Dizziness, near syncope	BP 140/97 mm Hg; mild lethargy; NIHSS score 1	5 min	8.5	Left internal capsule	Possible amyloid angiopathy
M/early 50s	Dizziness, gait unsteadiness, mild confusion	BP 100/58 mm Hg; mild inattention, pronator drift; NIHSS score 1	<1 h	9.8	Left basal ganglia	Unclear
F/mid-80s	Right leg numbness	BP 210/59 mm Hg; left leg sensory loss; NIHSS score 2	30 min	7.7	Left lateral thalamic	Possibly hypertensive
M/mid-60s	Right hemiparesis, dysarthria, gait unsteadiness	BP 178/95 mm Hg; dysarthria, right hemiparesis, dysmetria; NIHSS score 4	2-3 h	24	Left basal ganglia	Anticoagulation, possibly hypertensive
F/early 80s	Left leg weakness	BP 200/90 mm Hg; left leg drift; NIHSS score 1	30 min	18	Right internal capsule	Anticoagulation, possibly hypertensive
M/late 60s	Left arm clumsiness, facial droop, slurred speech	BP 182/70 mm Hg; mild left ataxia, dysarthria; NIHSS score 3	5 h	21	Right thalamus	Hypertensive
M/early 60s	Dysarthria, hand clumsiness	BP 141/93 mm Hg; mild dysarthria, limb ataxia; NIHSS score 2	Possibly 6-8 h	7	Left lentiform nucleus	Hypertensive
F/mid-60s	Dysarthria, headache	BP 197/93 mm Hg; left hemiparesis; NIHSS score 5	<12 h	9.4	Right basal ganglia	Hypertensive
M/late-40s	Left hemiparesis	BP 132/73 mm Hg; left hemiparesis; NIHSS score 4	<6 h	23	Right putamen	Moyamoya disease
M/mid-40s	Slurred speech, right leg weakness, gait difficulty	BP 220/120 mm Hg; mild right hemiparesis, aphasia, ataxia; NIHSS score 5	<8 h	40.4	Left putamen	Hypertensive

Abbreviations: BP, blood pressure; NIHSS, National Institutes of Health Stroke Scale.

Kumar S, Selim M, Marchina S, Caplan LR. Transient Neurological Symptoms in Patients With Intracerebral Hemorrhage. *JAMA Neurol.* 2016;73(3):316–320. doi:10.1001/jamaneurol.2015.4202

Cerebral Amyloid Angiopathy–Related Transient Focal Neurologic Episodes

Eric E. Smith, MD, MPH, Andreas Charidimou, MD, PhD, Cenk Ayata, MD, David J. Werring, MD, and Steven M. Greenberg, MD, PhD

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Dr. Smith
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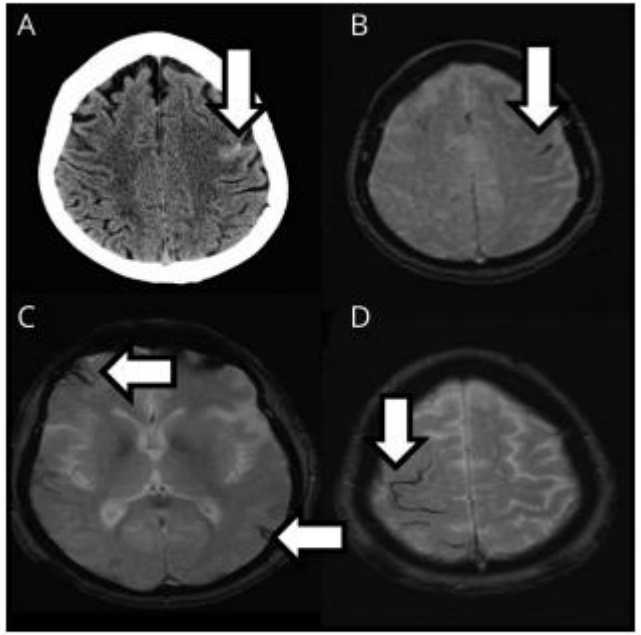
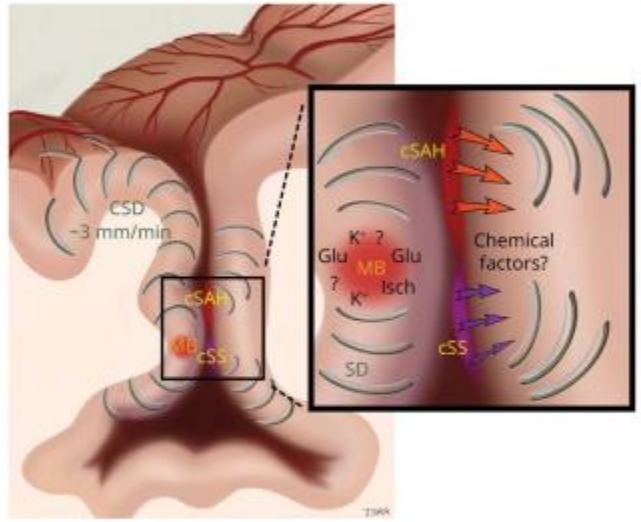
Neurology® 2021;97:231-238. doi:10.1212/WNL.0000000000012234

Figure 1 Convexity Sulcal Subarachnoid Hemorrhage (cSAH) and Cortical Superficial Siderosis (cSS)



A 71-year-old woman presented with paresthesias and weakness of the right hand. (A) CT showed acute cSAH in a left frontal sulcus, visible as a linear hypointensity on T2*-weighted gradient-recalled echo (GRE) MRI (B). MRI GRE also showed 3 areas of cSS (arrows, C and D) in sulci without acute cSAH. One year later, the patient had a left parietal lobar intracerebral hemorrhage.

Figure 2 Possible Mechanisms Triggering Spreading Depolarizations

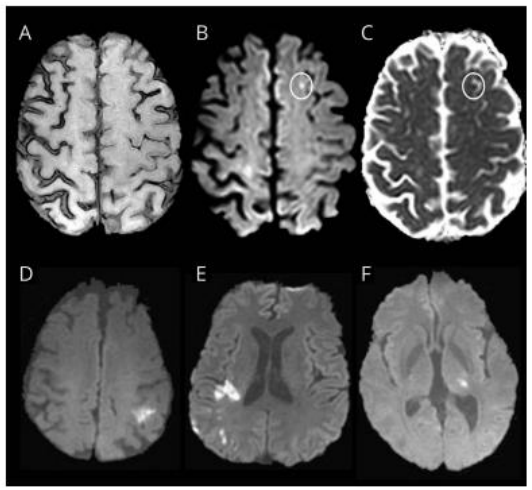


Schematic representation of hypotheses on the origin of spreading cortical depolarizations (CSDs) within a sulcus affected by cerebral amyloid angiopathy (CAA). Convexity subarachnoid hemorrhage (cSAH) and cortical superficial siderosis (cSS) could trigger CSD by releasing chemical factors that affect the brain tissue or pial vasculature. An acute cortical microbleed might also trigger CSD via ischemia (Isch) in the territory of the ruptured artery, via mechanical distortion of brain tissue by expanding microbleed, or by release of depolarizing factors from plasma leakage or hematoma lysis (e.g., potassium [K+] ions or glutamate [Glu]). Once initiated, CSDs propagate in cortical gray matter at a speed of ~3 mm/min for many centimeters, creating a TFNE.

Table 1 Competing Causes of Transient Neurologic Symptoms

TIA
Migraine with aura
Focal seizure
Structural lesions (e.g., tumor, vascular malformation, subdural hematoma)
Metabolic abnormalities (e.g., hypoglycemia, hyponatremia)
Syncope or presyncope
Functional neurologic disorder

Figure 3 MRI Diffusion-Weighted Imaging (DWI) in Cerebral Amyloid Angiopathy (CAA)–Related Transient Focal Neurologic Episodes (TFNEs) Compared With Ischemic Stroke



(A–C) From the same patient, disseminated cortical superficial siderosis (cSS) on MRI susceptibility-weighted imaging (A) with a 3 mm focus of bright signal on DWI in the left superior frontal gyrus adjacent to cSS (B, circled) with hypointensity on apparent diffusion coefficient image (C, circled), indicating restricted diffusion. Small (≤10 mm) DWI-positive lesions are often seen in CAA with convexity subarachnoid hemorrhage and TFNEs, usually adjacent to regions of cSS and sometimes multiple.[42] (D–F) DWI-positive lesion patterns seen in ischemic stroke but not CAA include single larger (>10 mm) DWI-positive infarcts (D), multiple DWI-positive infarcts restricted to a vascular perfusion territory (E), or DWI-positive small subcortical infarcts restricted to the territory of a single perforating artery (as in the thalamic recent subcortical infarct seen in F).

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+ / - ?

- Symptômes négatifs ?
- Symptômes positifs ?

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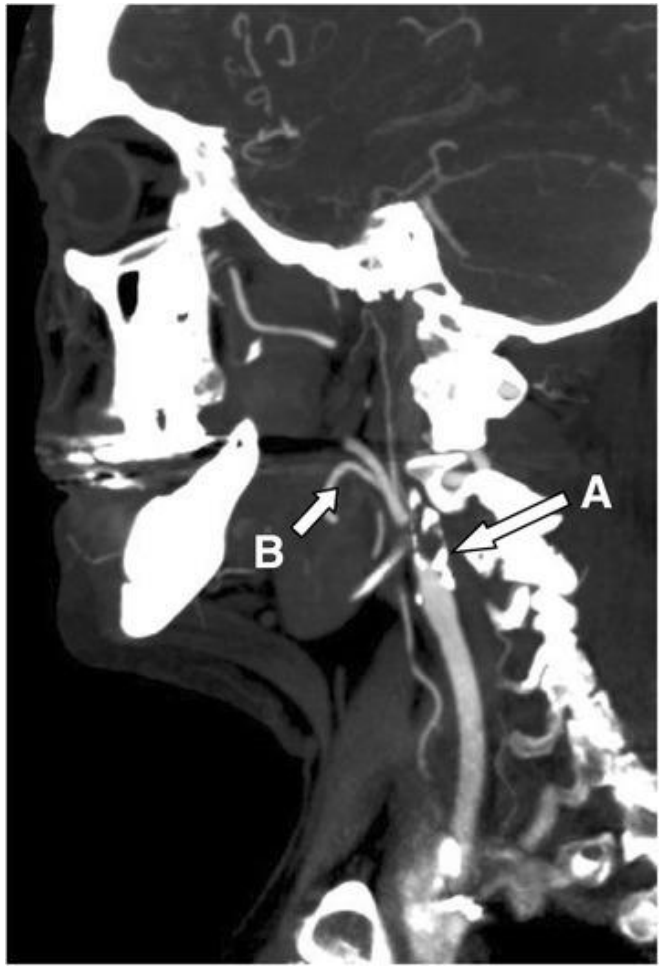
Crise d'épilepsie? AIT? Fonctionnel?



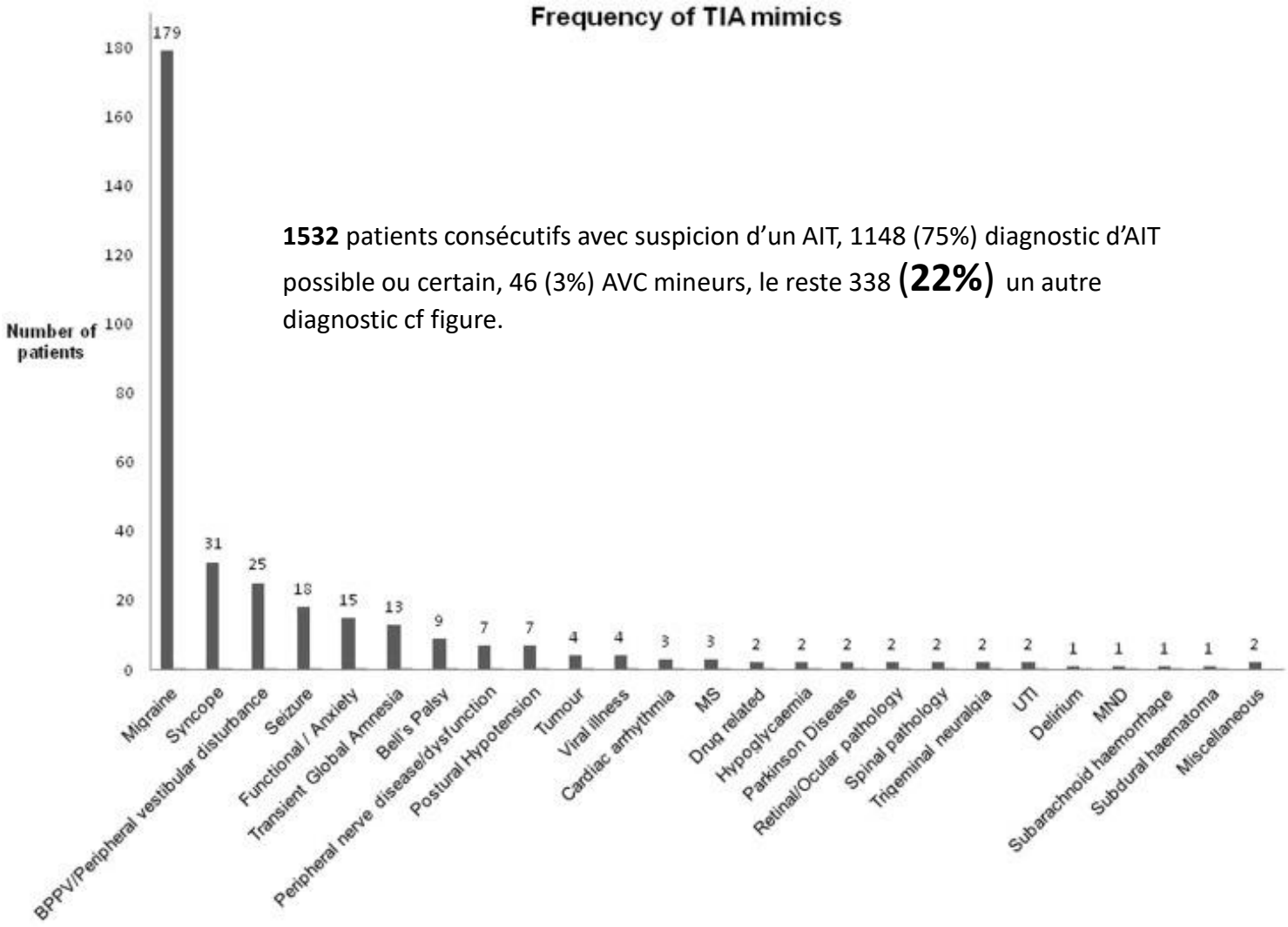
Limb shaking TIA

Rosenbaum *et al. BMC Neurology* (2016) 16:78
DOI 10.1186/s12883-016-0601-8

TIA mimics

- **Durée des symptômes:**
  - Migraine:
    - aura migraineuse < d'1h pour chaque symptôme
    - progression sur minutes… marche migraineuse
  - Crise d'épilepsie < 5 minutes
    - progression dans quelques secondes
    - symptômes négatifs post critiques (phénomène de Todd)
  - AIT <1h la majorité
    - d'emblé déficit maximal mais rémission graduelle possible
    - Symptômes concordants à un territoire vasculaire

Nadarajan V, *et al. Pract Neurol* 2014;**14**:23–31. doi:10.1136/practneurol-2013-000782



1532 patients consécutifs avec suspicion d'un AIT, 1148 (75%) diagnostic d'AIT possible ou certain, 46 (3%) AVC mineurs, le reste 338 **(22%)** un autre diagnostic cf figure.

Début brutal, maximal = très probablement AIT ?

Table 1 Clinical features of transient ischaemic attack (TIA) and some common mimics

	TIA	Migraine	Seizure	Syncope	Functional/ anxiety
Demographic	Older age Vascular risk factors More common in men	Younger age More common in women	Any age	Any age, often younger More common in women	Younger More common in women
Neurological symptoms	Negative symptoms, usually maximal at onset: for example, numbness, weakness, visual loss. Transient diplopia and monocular visual loss are often due to TIA Does not spread into other sensory modalities. Alteration or loss of consciousness almost never occur	Positive, spreading symptoms at onset. Visual the most common. May be followed by negative symptoms in the same domain Symptoms may evolve into another modality (eg, visual followed by somatosensory) True alteration or loss of consciousness almost never occur, though there may be 'confusion' or muddled thinking	Positive symptoms including painful sensory disturbance, limb jerking, head turning, dystonic posturing, lip smacking. Loss of awareness and amnesia for event unless simple partial seizures Postictal negative symptoms (eg, Todd's paresis) may persist for days	Faint or light headed (presyncopal). Vision may darken, or hearing becomes muffled. Loss of awareness	Isolated sensory symptoms common
Timing	Abrupt onset, gradual offset (minutes). Usually total duration minutes, nearly always <1 h Recur over days or weeks, usually not months or years.	Usually last 20–30 min, but may be much longer Can recur over years or decades.	Usually less than 2 min. Can recur over years	Seconds to less than a minute. Can recur over years	Tend to be recurrent and stereotyped
Associated symptoms	Headaches may occur, usually during the attacks	Headache usually afterwards with migrainous features (nausea, vomiting, photophobia, phonophobia, mechanosensitivity)	Tongue biting (especially lateral), incontinence, muscle pains, exhaustion or disorientation, headache follow	Sweating, pallor, nausea, rapid recovery to full alertness	May be preceded by emotional or psychosocial stressors Anxiety

Nadarajan V, *et al*. *Pract Neurol* 2014;**14**:23–31. doi:10.1136/practneurol-2013-000782

- le diagnostic d'un **évènement vasculaire cérébral ischémique** était considéré comme **définitif** lorsqu'une lésion ischémique aiguë appropriée était observée à l'imagerie cérébrale et comme **probable** lorsqu'il existait un **accord diagnostique entre deux neurologues** spécialisés en pathologie vasculaire cérébrale en l'absence de telles lésions à l'imagerie.

- données sur 100 patients consécutifs ayant présenté un ou plusieurs épisodes neurologiques focaux transitoires durant moins de 24 heures et chez lesquels le diagnostic initial était un AIT:
  - **60 un NI-TNA :**
- apparition **progressive** des symptômes (OR ajusté 6,7, p = 0,002),
- antécédents d'attaques neurologiques transitoires **inexpliquées** (OR ajusté 10,6, p = 0,031)
- présence de **symptômes non spécifiques** (OR ajusté 4,2, p = 0,008)

Misdiagnosis of Transient Ischemic Attacks in the Emergency Room

Shyam Prabhakaran Adam J. Silver Lakshmi Warrior Bethany McClenathan Vivien H. Lee

Department of Neurological Sciences, Rush University Medical Center, Chicago, Ill., USA

Cerebrovasc Dis 2008;26:630–635
DOI: 10.1159/000166839

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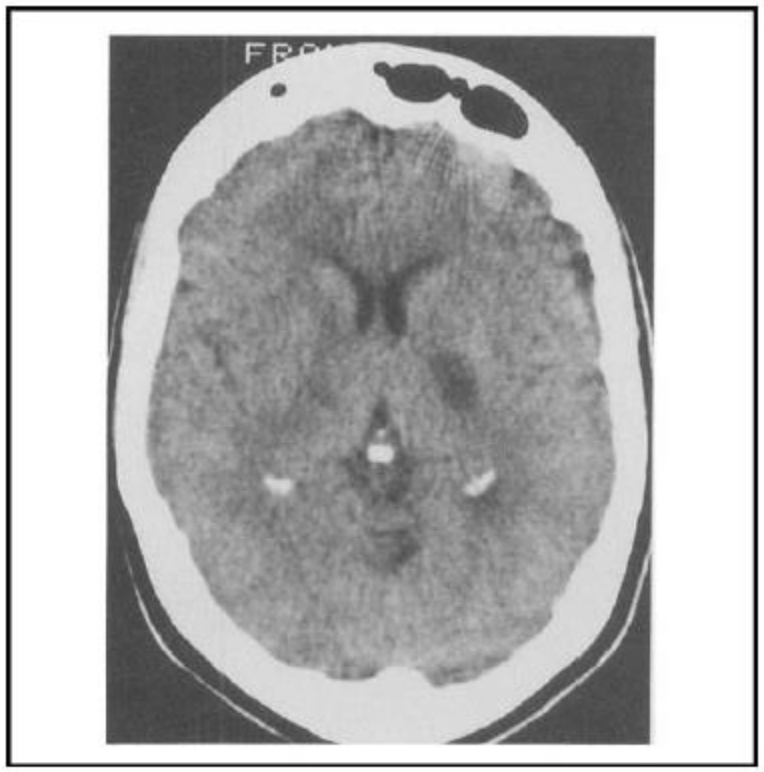


Figure 2. CT of a 56-year-old woman (pt 2) who presented with repetitive episodes of right hemiparesis and developed a capsular stroke. Scan shows an infarct involving more than one penetrator in the territory of the left anterior choroidal artery.

The capsular warning syndrome:
Pathogenesis and clinical features

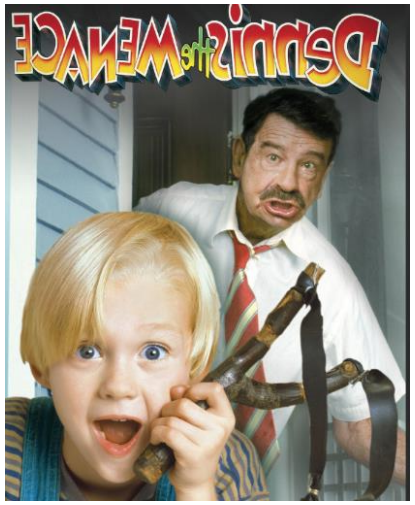
G.A. Donnan, MD, FRACP; H.M. O'Malley, RN; L. Quang, Grad. Dip. Sci.; S. Hurley, PhD; and P.F. Bladin, BSc, MD, FRACP

Article abstract—Transient ischemic attacks (TIAs) are not homogeneous and may consist of subsets with mechanisms as varied as their stroke counterparts. We describe a form of TIA in 50 patients where crescendo episodes of ischemia were restricted to the region of the internal capsule, usually causing symptoms affecting face, arm, and leg. These patients composed 4.5% of a consecutive series of patients admitted with TIAs over a 15-year period and 33% of all TIAs classified as subcortical. We believe that the ischemia was most often due to hemodynamic phenomena in diseased, single, small penetrating vessels. When cerebral infarction developed, it was usually lacunar and involved a single penetrating vessel, although occasionally striatocapsular or anterior choroidal artery territory infarction occurred. There was no evidence of artery-to-artery or heart-to-artery embolism. Resistance to various forms of therapy, including hemodiluting, anticoagulant, and thrombolytic agents, was common. Because of dramatic and easily recognizable clinical presentation, apparent specific pathophysiologic mechanism, and the development of early capsular stroke in a high proportion of cases (42%), we have termed this the "capsular warning syndrome."

NEUROLOGY 1993;43:957-962

Table. The distribution of motor and sensory symptoms in 50 cases of the capsular warning syndrome

Symptoms	No. of patients
Motor	
Face/Arm/Leg	16
Face/Arm	1
Arm/Leg	5
Sensory	
Face/Arm/Leg	1
Face/Arm	0
Arm/Leg	1
Sensory/Motor	
Face/Arm/Leg	14
Face/Arm	2
Arm/Leg	9
Dysarthria/Motor/Sens. Arm	1
Total	50



NEUROLOGY 1993;43:957-962

Risque de récidive?

Table 3. Comparison of ABCD², ABCD³, and ABCD³-I Scores

Components	ABCD² score	ABCD³ score	ABCD³-I score	ABCD³-I (d, c/i) score
Risk factor				
Age ≥60 y	1	1	1	1
Blood pressure ≥140/90 mm Hg	1	1	1	1
Diabetes	1	1	1	1
Clinical features				
Unilateral weakness	2	2	2	2
Language disturbance without weakness	1	1	1	1
Symptom duration, min				
≥60	2	2	2	2
10–59	1	1	1	1
<10	0	0	0	0
>10	N/A	N/A	0	0
Dual transient ischemic attack	N/A	2	2	2
Imaging				
Ipsilateral ≥50% stenosis of internal carotid artery	N/A	N/A	2	N/A
Ipsilateral ≥50% stenosis of internal carotid artery and major cerebral artery	N/A	N/A	N/A	2
Acute diffusion-weighted imaging hyperintensity	N/A	N/A	2	2
Total points	0–7	0–9	0–13	0–13
ABCD² score	2-d risk (%)	7-d risk (%)	90-d risk (%)	
Low (0–3)	1.0	1.2	3.1	
Moderate (4–5)	4.1	5.9	9.8	
High (6–7)	8.1	11.7	17.8	

ABCD² indicates age/blood pressure/clinical features of transient ischemic attack/duration/diabetes score; ABCD³, ABCD² plus Dual TIA; c, carotid stenosis; d, diffusion-weighted image; I, imaging; i, intracranial stenosis; and N/A, not applicable.

Stroke

AHA SCIENTIFIC STATEMENT

Diagnosis, Workup, Risk Reduction of Transient Ischemic Attack in the Emergency Department Setting: A Scientific Statement From the American Heart Association

Hardik P. Amin, MD, Chair; Tracy E. Madsen, MD, PhD, Vice Chair; Dawn M. Bravata, MD; Charles R. Wira, MD; S. Claiborne Johnston, MD, PhD; Susan Ashcraft, DNP; Tamika M. Burrus, MD; Peter D. Panagos, MD; Max Wintermark, MD, MAS; Charles Esenwa, MD, MS; on behalf of the American Heart Association Emergency Neurovascular Care Committee of the Stroke Council and Council on Peripheral Vascular Disease

Addition of brain and carotid imaging to the ABCD² score to identify patients at early risk of stroke after transient ischaemic attack: a multicentre observational study

Áine Merwick, Gregory W Albers, Pierre Amarenco, Ethem M Arsava, Hakan Ay, David Calvet, Shelagh B Coutts, Brett L Cucchiara, Andrew M Demchuk, Karen L Furie, Matthew F Giles, Julien Labreuche, Philippa C Lavallée, Jean-Louis Mas, Jean Marc Olivot, Francisco Purroy, Peter M Rothwell, Jeffrey L Saver, Órla C Sheehan, John P Stack, Cathal Walsh, Peter J Kelly

Stroke. 2023;54:e109–e121.

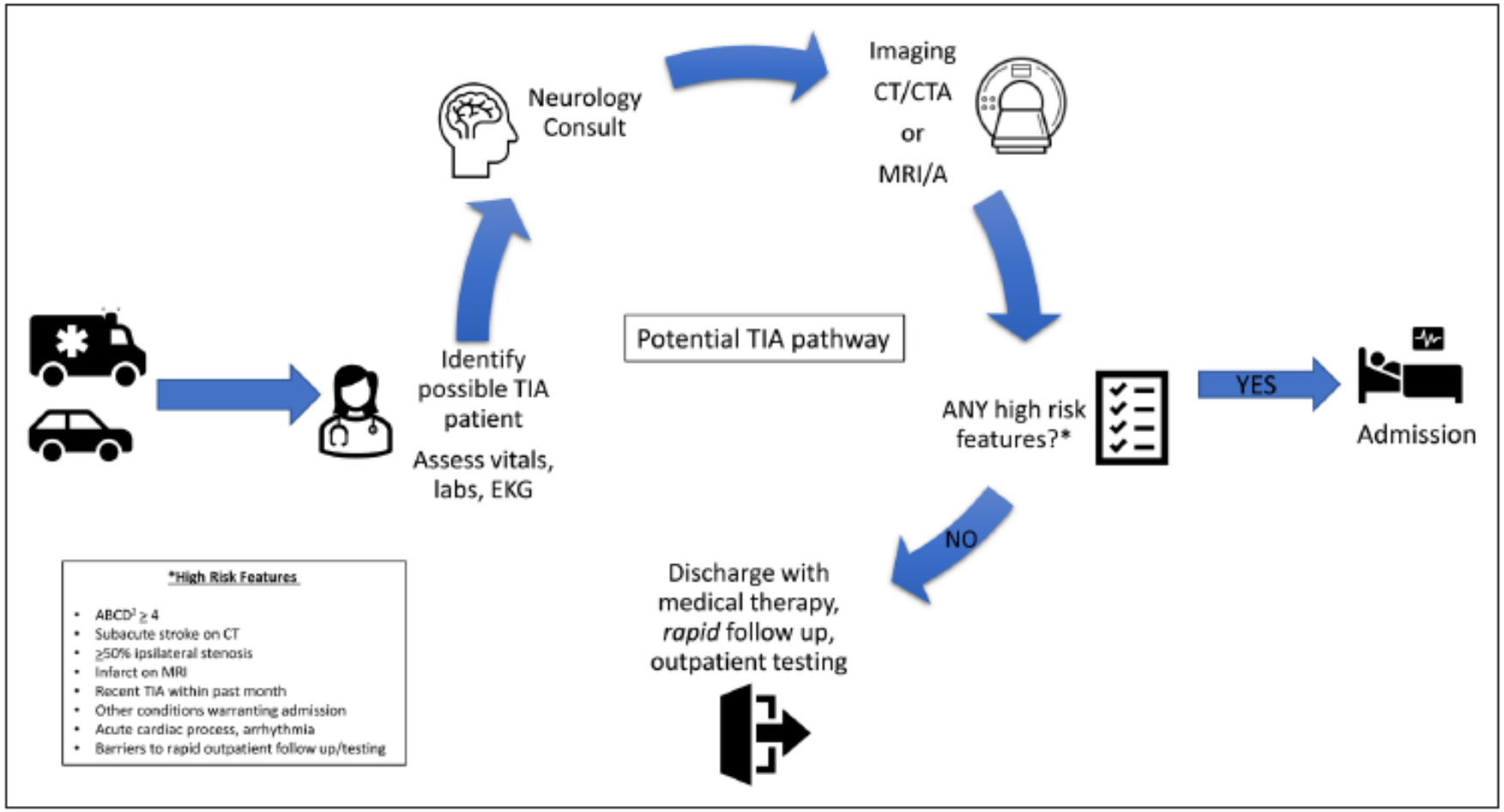


Figure. A potential TIA pathway that incorporates clinical evaluation, imaging, and risk stratification to guide disposition decisions.

Modifications are expected when rapid neurology consultation or MRI are not available. CT indicates computed tomography; CTA, computed tomography angiography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; and TIA, transient ischemic attack.

Amin et al. Transient Ischemic Attack in the Emergency Departement. Stroke 2023.54:e109-121.

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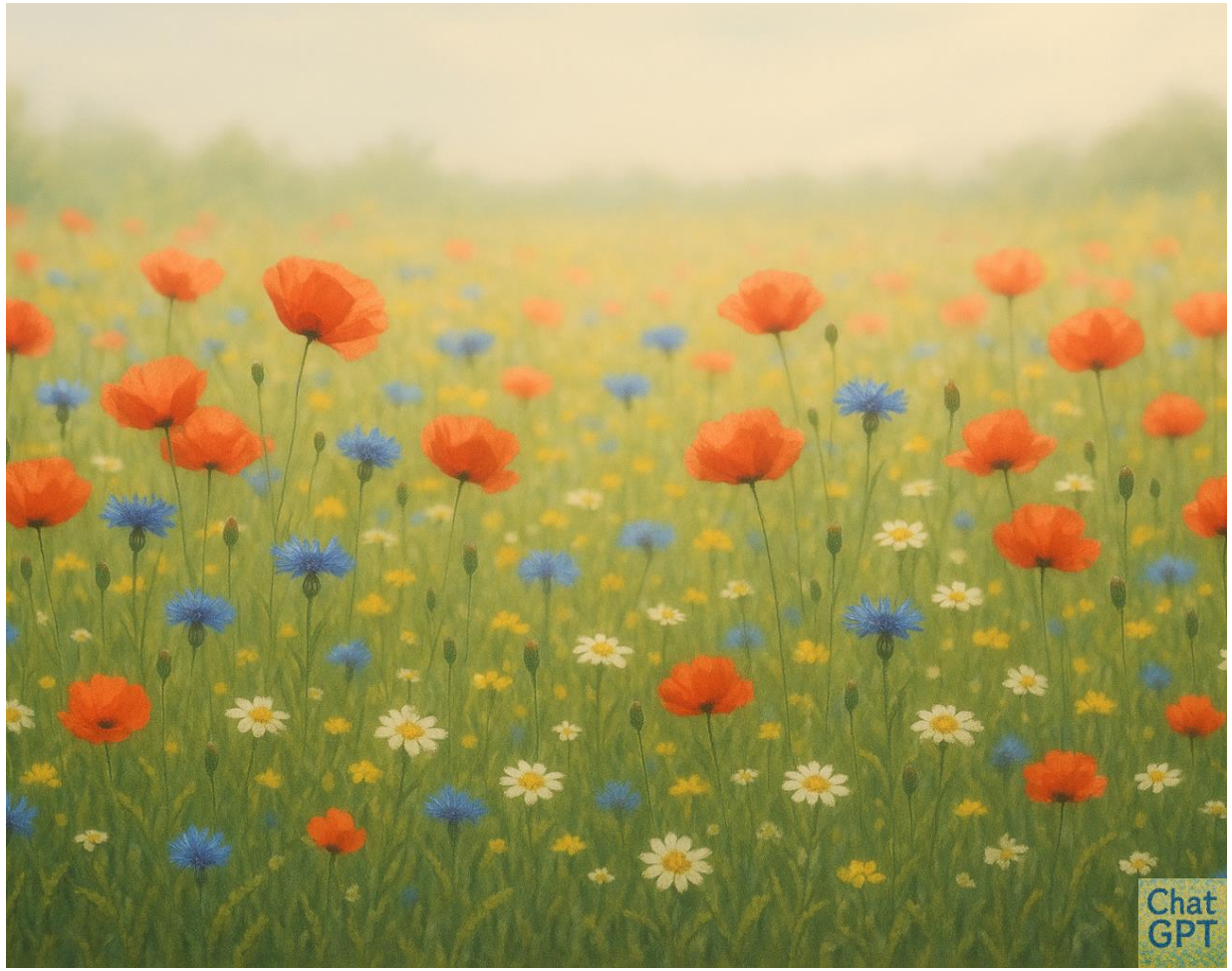
Peut-on prescrire l'Aspirine sur la base de la clinique?

NON

Monet et AI



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L’AVC ischémique

Stroke
Volume 26, Issue 7, July 1995; Pages 1205-1209
https://doi.org/10.1161/01.STR.26.7.1205

ARTICLE

Is It Clinically Possible to Distinguish Nonhemorrhagic Infarct From Hemorrhagic Stroke?

Gérard Besson, Claudine Robert, Marc Hommel, and Jean Perret

- (CT scan) comme référence standard
- 368 patients ont été inclus dans l'étude interne
- **score obtenu était :**
  (2 × consommation d'alcool) + (1,5 × réponse plantaire) + (3 × céphalée) + (3 × antécédents d'hypertension) – (5 × antécédents de déficit neurologique transitoire) – (2 × maladie artérielle périphérique) – (1,5 × antécédents d'hyperlipidémie) – (2,5 × fibrillation auriculaire à l'admission)
- Tous les patients avec un score inférieur à 1 (n = 123) avaient un infarctus non hémorragique (soit 40 % des 305 patients avec un infarctus non hémorragique)
- 43 % (IC à 95 %, 36 à 50) des patients présentant un infarctus non hémorragique pourraient recevoir un diagnostic au lit du malade. Le score est simple et peut être calculé à partir d'informations accessibles à tous les médecins.

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Moving from CT to MRI paradigm in acute ischemic stroke: feasibility, effects on stroke diagnosis and long-term outcomes

Costanza M. Rapillo, Vincent Dunet, Silvia Pistocchi, Alexander Salerno, Vincent Darioli, Bruno Bartolini, Steven D. Hajdu, Patrik Michel and Davide Strambo

Starting May 2018, MRI became the first-line imaging modality for acute ischemic strokes at CHUV

MRI performed as first-line imaging in the MRI period

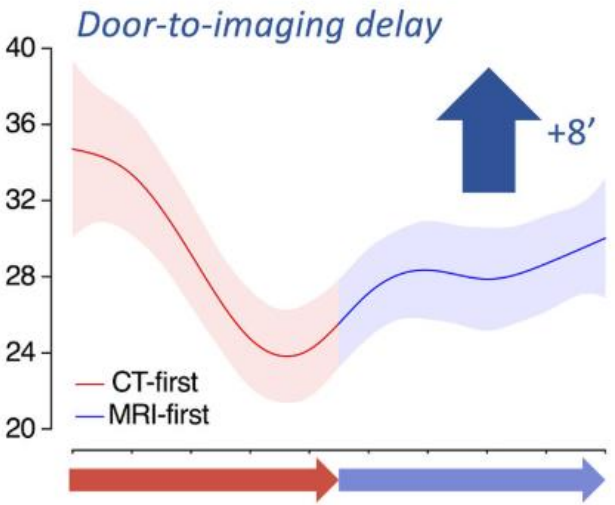
Ischemic lesion on acute MRI

Population

Consecutive AIS patients from the ASTRA registry.

CT period
2012-2018
N=1492

MRI period
2019-2022
N=1492



Stroke mimics treated with IVT

Repeated neuroimaging during stroke unit hospitalizatio

Stroke. 2024;55:1329–1338. DOI: 10.1161/STROKEAHA.123.045154

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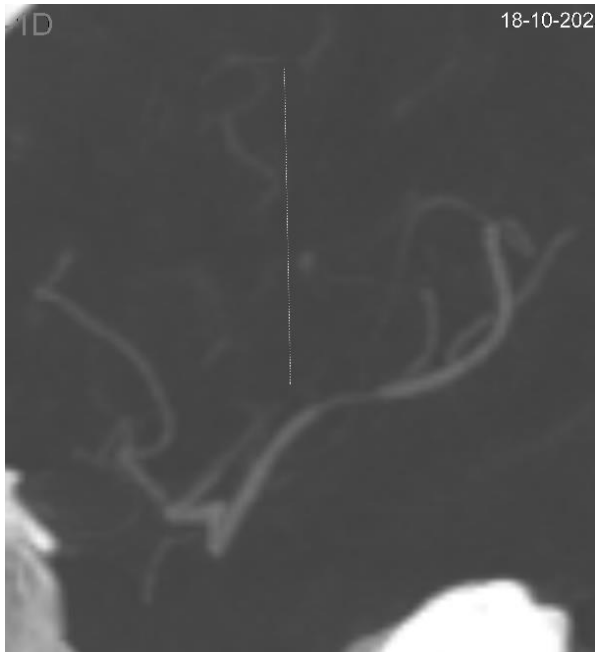
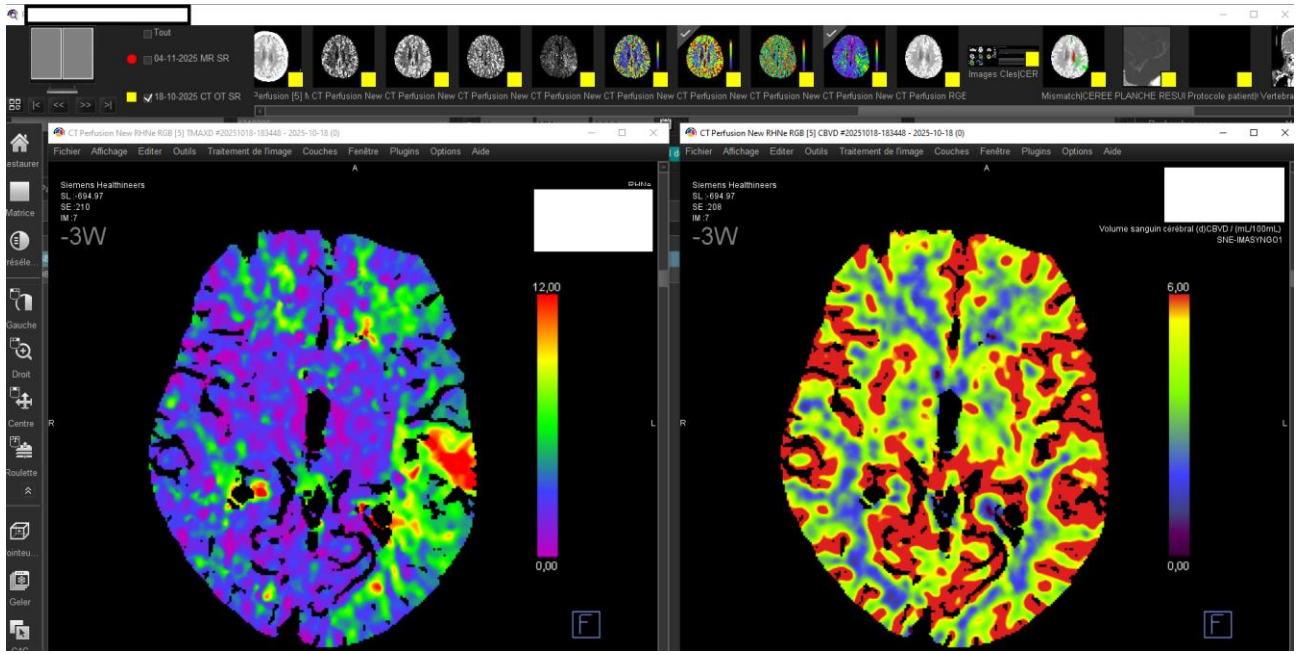
Diagnostic :

- **AVC ischémique sylvien gauche avec occlusion M2 et mismatch perfusionnel**
- Traitement par thrombolyse I.V. et transfert à l'Inselspital pour une thrombectomie
- - LTSW : 18.10.25 à 17:05
- - NIHSS initial : SMUR 9
- - DI : 18 : 15
- - CT scan : 18 :28
- - NIHSS avant la lyse : 2
- - Traitement : Actilyse 72mg à 18 :44 (99 min)
- - Bolus Lyse : 18 :44
- - NIHSS durant la lyse : 1
- - DO : 19 :02 direction InselSpital
- - Stat : OK





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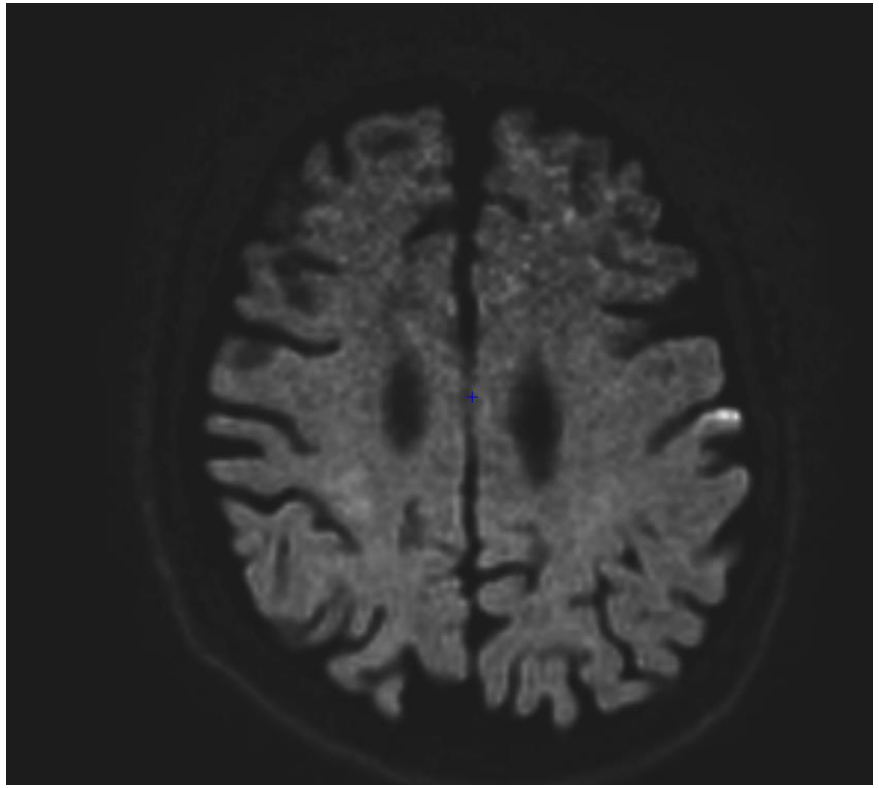

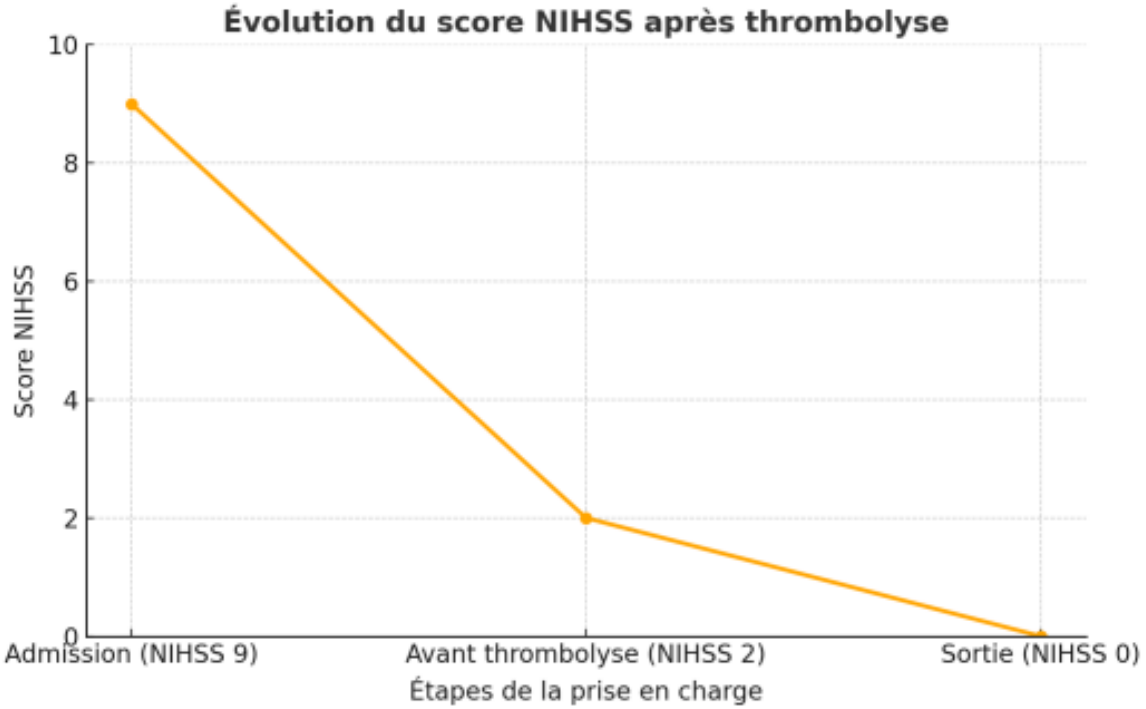

Status de sortie :

- NIHSS = 0/42
- mRS = 0/6
- Neurologique : superposable à l'examen d'entrée hormis une position debout actuellement stable.

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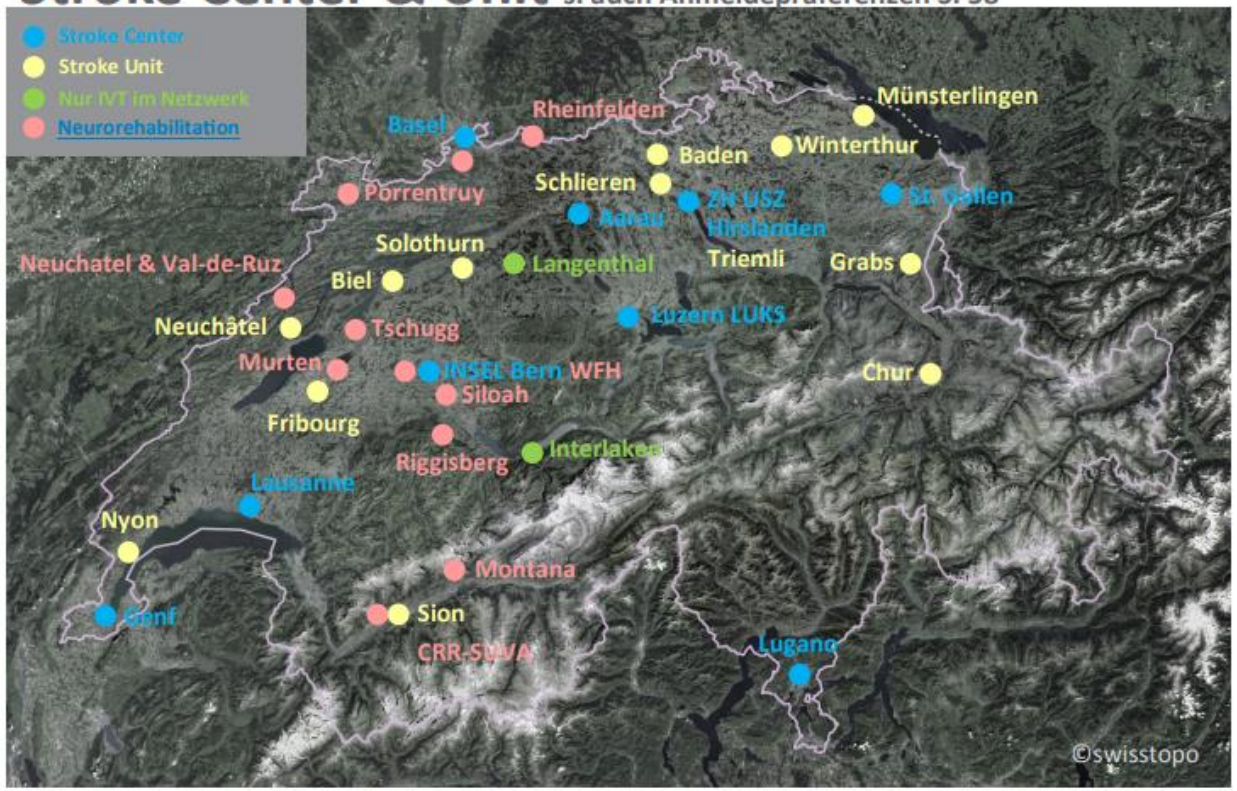
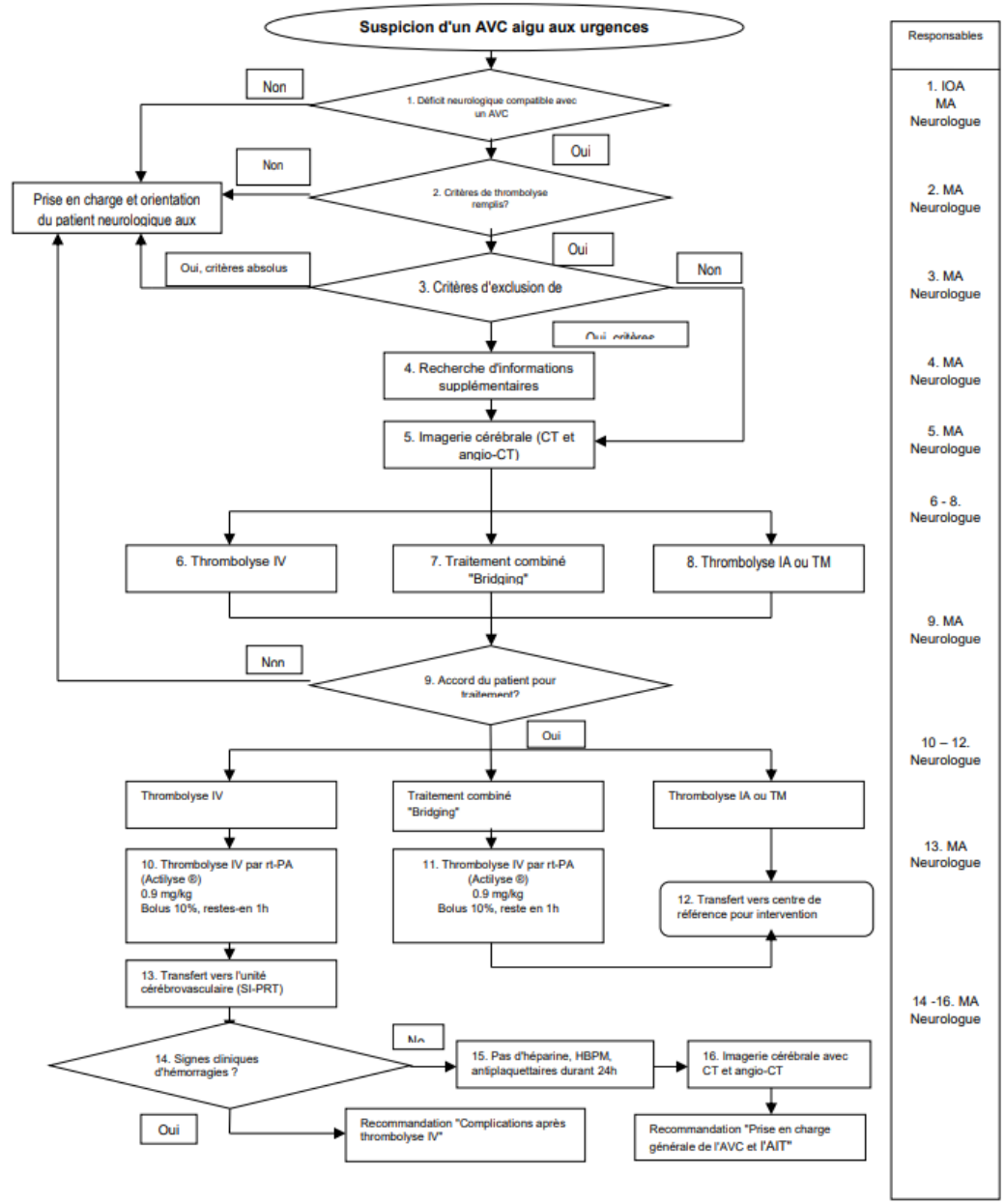
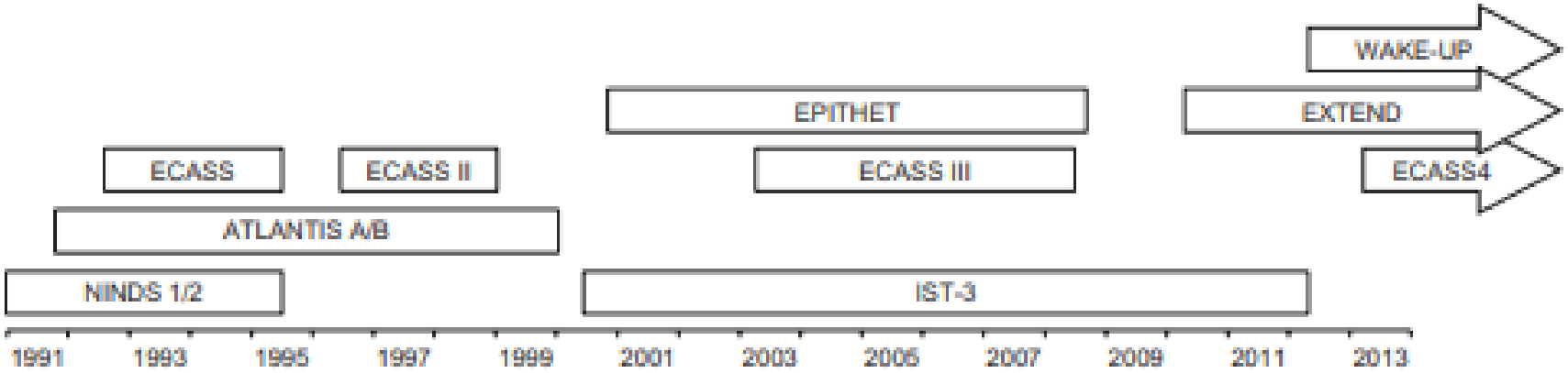
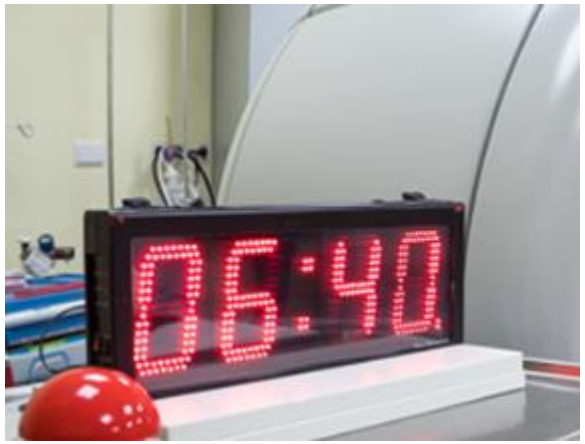
Stroke Center & Unit s. auch Anmeldepräferenzen S. 58

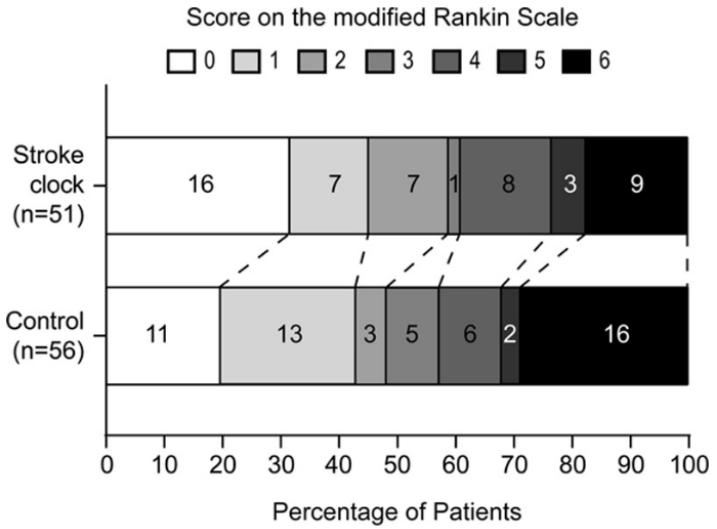




Le traitement de l'AVCi vu par la perspective du temps …







Mathias F et al. Effects of a Feedback-Demanding Stroke Clock on Acute Stroke Management: A Randomized Study. StrokeVolume 51, Issue 10, October 2020; Pages 2895-2900

Bruce CV et al. Stroke. 2015;46:2341-2346

Un shift de paradigme: sélection sur l'imagerie

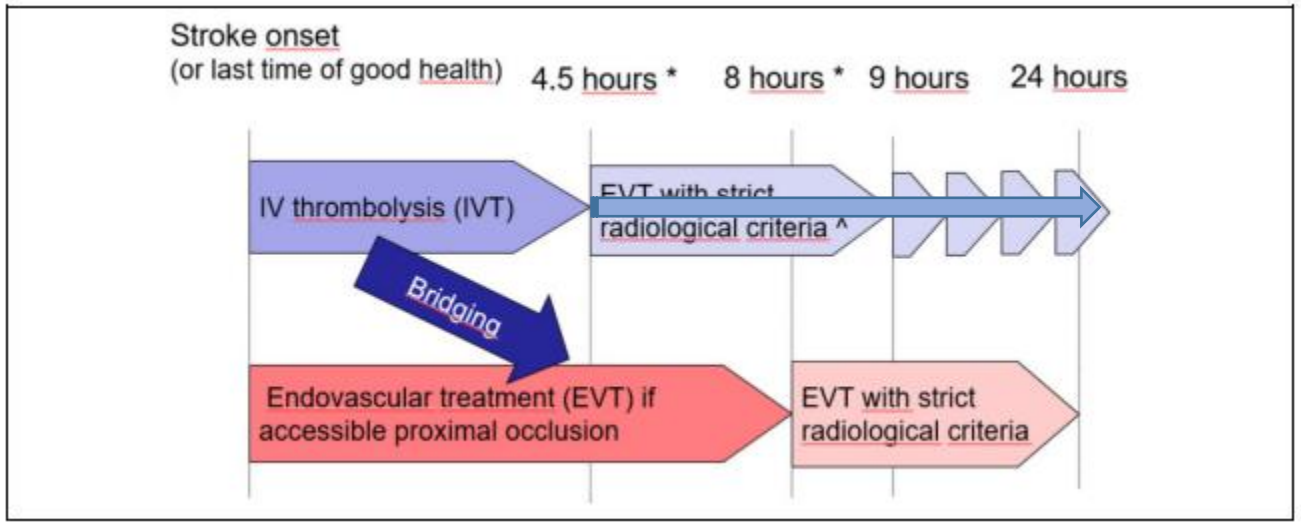
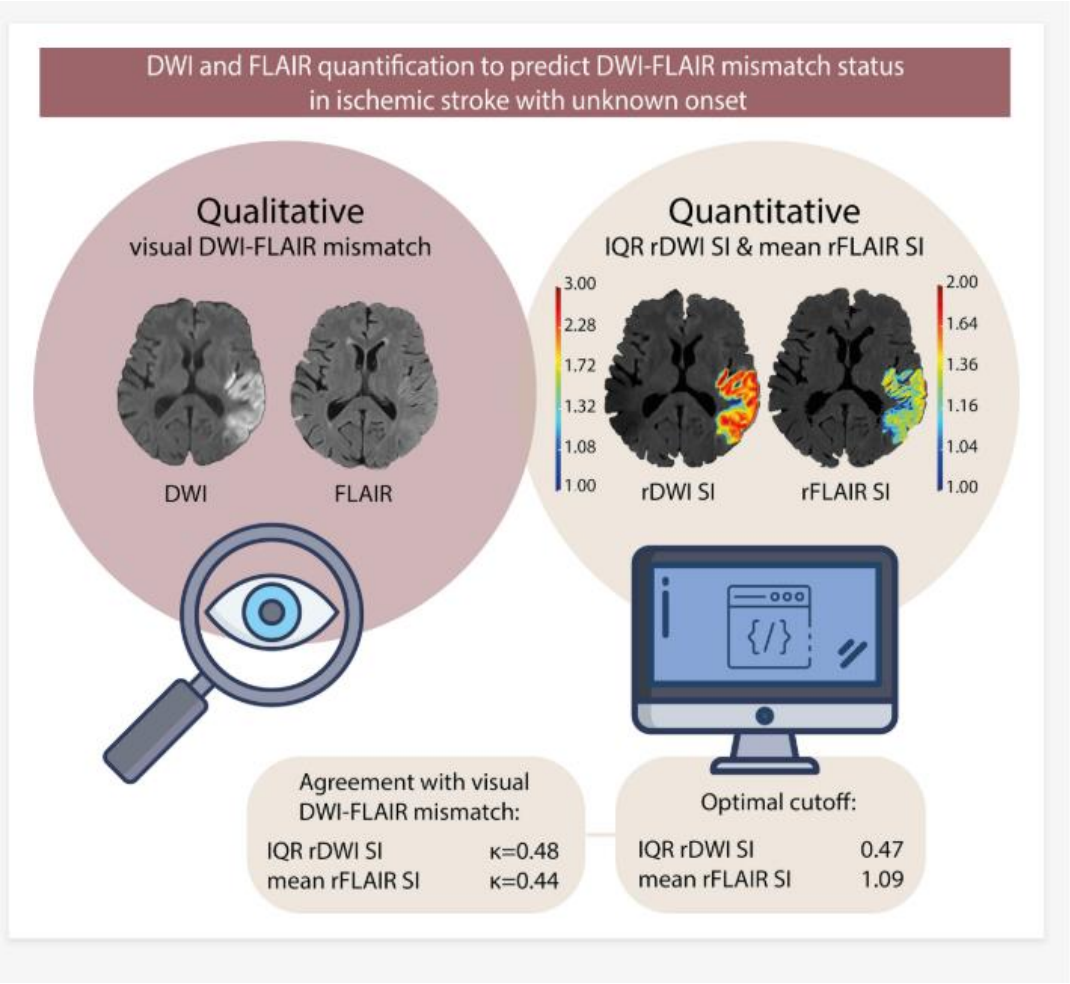


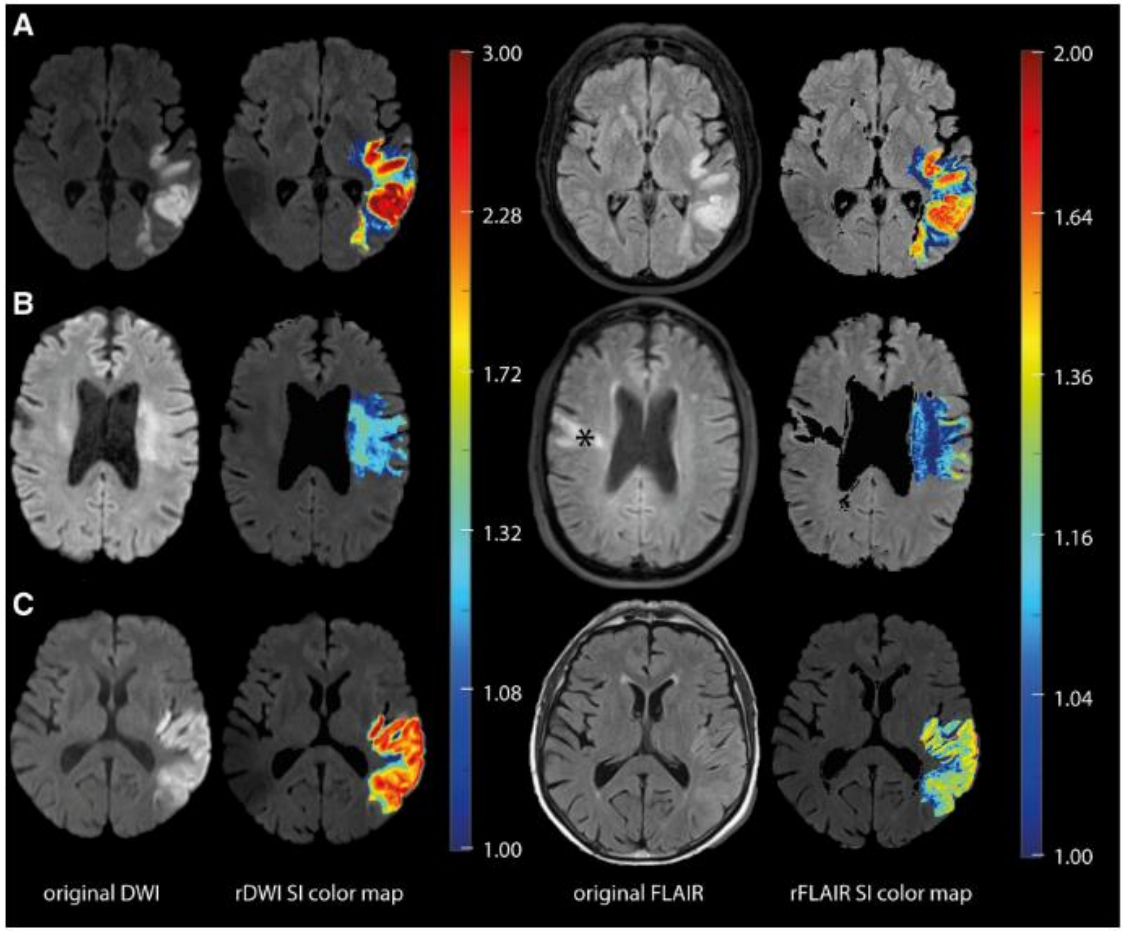
Table 1. Mismatch definitions as used in RCTs.

Mismatch type and examples of RCTs	Mismatch definition in trials	Comments
Pure neuroradiological mismatch (PCT or MRI)[20,21]	Mismatch ratio ≥ 1.8 (in some studies > 1.2)[58,63]	Thresholds not validated for posterior circulation. Penumbra estimation is clinical rather than neuroradiological.
Clinical–neuroradiological mismatch (PCT or MRI)[18]	NIHSS ≥10 and core ≤30 ml	Thresholds not validated for posterior circulation.
	NIHSS ≥20 and core 31–50 ml	Use of contrast medium not mandatory if using MRI.
FLAIR-DWI mismatch (MRI)[23]	Acute DWI lesion that is not visible on FLAIR sequences	Corresponds to stroke onset <4–6 h. Also applicable for posterior circulation. Use of contrast medium not mandatory if using MRI.

Patrick M et al. Acute revascularization in ischemic stroke: Updated Swiss guidelines. Clinical & Translational Neuroscience January-June 2021: 1–11

Mismatch DWI/FLAIR





Lauranne Scheldeman et al. Diffusion-Weighted Imaging and Fluid-Attenuated Inversion Recovery Quantification to Predict Diffusion-Weighted Imaging-Fluid-Attenuated Inversion Recovery Mismatch Status in Ischemic Stroke With Unknown Onset. Stroke Volume 53, Number 5. https://doi.org/10.1161/STROKEAHA.121.03687

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Thrombolysis Guided by Perfusion Imaging up to 9 Hours after Onset of Stroke

H. Ma, B.C.V. Campbell, M.W. Parsons, L. Churilov, C.R. Levi, C. Hsu, T.J. Kleinig, T. Wijeratne, S. Curtze, H.M. Dewey, F. Miteff, C.-H. Tsai, J.-T. Lee, T.G. Phan, N. Mahant, M.-C. Sun, M. Krause, J. Sturm, R. Grimley, C.-H. Chen, C.-J. Hu, A.A. Wong, D. Field, Y. Sun, P.A. Barber, A. Sabet, J. Jannes, J.-S. Jeng, B. Clissold, R. Markus, C.-H. Lin, L.-M. Lien, C.F. Bladin, S. Christensen, N. Yassi, G. Sharma, A. Bivard, P.M. Desmond, B. Yan, P.J. Mitchell, V. Thijs, L. Carey, A. Meretoja, S.M. Davis, and G.A. Donnan, for the EXTEND Investigators*

Critères d'inclusion EXTEND (Thrombolysis Guided by Perfusion Imaging up to 9 Hours) :

Âge adulte (patients majeurs).

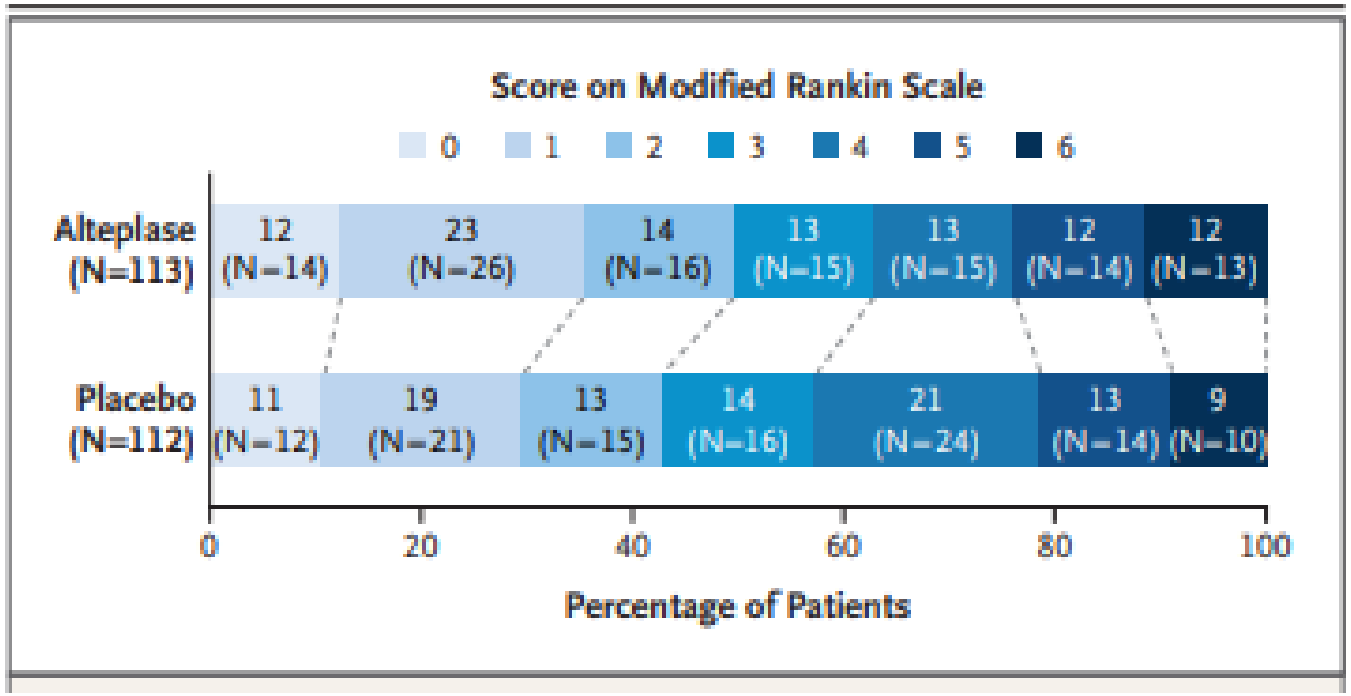
Bon état fonctionnel prémorbide : **mRS < 2** (0–1) avant l'AVC.

Fenêtre temporelle tardive : présentation **entre 4,5 et 9 heures** après le début des symptômes **ou AVC au réveil** (heure d'apparition estimée au **milieu du sommeil**).

Sévérité clinique : **NIHSS 4–26** à l'admission.

Profil d'imagerie sur perfusion CT/MRI:

- **Mismatch perfusion / noyau ischémique** avec **ratio > 1,2** (volume hypoperfusé / volume du noyau),
- **Différence absolue > 10 mL**,
- **Volume du noyau ischémique < 70 mL**



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MRI-Guided Thrombolysis for Stroke with Unknown Time of Onset

G. Thomalla, C.Z. Simonsen, F. Boutitie, G. Andersen, Y. Berthezene, B. Cheng, B. Cheripelli, T.-H. Cho, F. Fazekas, J. Fiehler, I. Ford, I. Galinovic, S. Gellissen, A. Golsari, J. Gregori, M. Günther, J. Guibernau, K.G. Häusler, M. Hennerici, A. Kemmling, J. Marstrand, B. Modrau, L. Neeb, N. Perez de la Ossa, J. Puig, P. Ringleb, P. Roy, E. Scheel, W. Schonewille, J. Serena, S. Sunaert, K. Villringer, A. Wouters, V. Thijs, M. Ebinger, M. Endres, J.B. Fiebach, R. Lemmens, K.W. Muir, N. Nighoghossian, S. Pedraza, and C. Gerloff, for the WAKE-UP Investigators*

Âge	**≥ 18 ans**
Diagnostic clinique	AVC ischémique aigu avec déficit neurologique mesurable (NIHSS compatible avec un AVC)
Heure de début	Heure exacte inconnue (souvent AVC au réveil) et dernière fois vu normal > 4,5 h avant
IRM de sélection (DWI-FLAIR mismatch)	Présence d'une lésion visible en DWI (diffusion) sans hypersignal correspondant en FLAIR, suggérant un AVC récent (moins de 4,5 h)
Sévérité clinique	NIHSS compatible avec un déficit cliniquement significatif, mais sans comas ni déficit massif (critères locaux d'éligibilité à la thrombolyse IV)
Délai	IRM et début du traitement possibles dans les 4,5 h suivant l'imagerie
Autres conditions générales	Consentement obtenu (du patient ou d'un représentant légal) et absence de contre-indication standard à l'altéplase IV

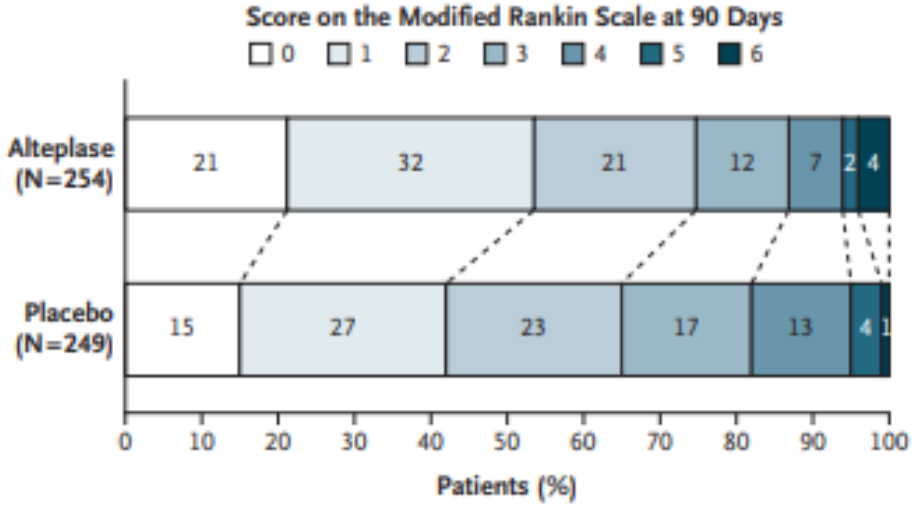

Figure 2. Distribution of Scores on the Modified Rankin Scale at 90 Days (Intention-to-Treat Population).

Shown are the differences in the scores on the modified Rankin scale among patients in the alteplase group and the placebo group at 90 days. Scores range from 0 to 6, with 0 indicating no symptoms, 1 no clinically significant disability, 2 slight disability, 3 moderate disability, 4 moderately severe disability, 5 severe disability, and 6 death. Numbers indicate rounded proportions. There was a significant difference favoring the alteplase group over the placebo group in the overall distribution of scores (adjusted common odds ratio, 1.62; 95% confidence interval, 1.17 to 2.23; P=0.003).

N Engl J Med 2018;379:611-622

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Risque d'hémorragie intracrânienne symptomatique EXTEND & WAKE UP TRIALS

EXTEND Trial, NEJM 2019 ; WAKE-UP Trial, NEJM 2018.

Étude	Type d'hémorragie	Groupe traité (Alteplase)	Groupe contrôle (Placebo/Standard)	Source
EXTEND (NEJM 2019)	Hémorragie intracrânienne symptomatique	6,2 %	0,9 %	NEJM 2019
WAKE-UP (NEJM 2018)	Hémorragie intracrânienne symptomatique	2,0 %	0,4 %	NEJM 2018

Une perspective sur la thrombectomie …

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Critères d'inclusion – étude DAWN

Résumé des critères d'inclusion du trial DAWN (Thrombectomy **6–24 Hours** after Stroke with a Mismatch between Deficit and Infarct).
DAWN Trial, NEJM 2018.

Critère	Description
Âge	≥ 18 ans
Diagnostic	AVC ischémique aigu causé par une occlusion intracrânienne de l'artère cérébrale moyenne (M1) ou de la carotide interne (ACI)
Heure de début	Dernière fois vu normal entre 6 et 24 heures avant l'évaluation
Imagerie de sélection	Mismatch clinique–imagerie défini par la combinaison du volume de l'infarctus et de la sévérité clinique (NIHSS) : • ≥ 80 ans : NIHSS ≥ 10 et volume du noyau ischémique < 21 mL • < 80 ans : NIHSS ≥ 10 et volume du noyau < 31 mL • < 80 ans : NIHSS ≥ 20 et volume du noyau entre 31 et 51 mL
État fonctionnel préalable	mRS ≤ 1 (indépendant avant l'AVC)
Traitement envisagé	Thrombectomie mécanique possible dans la fenêtre de 6 à 24 heures

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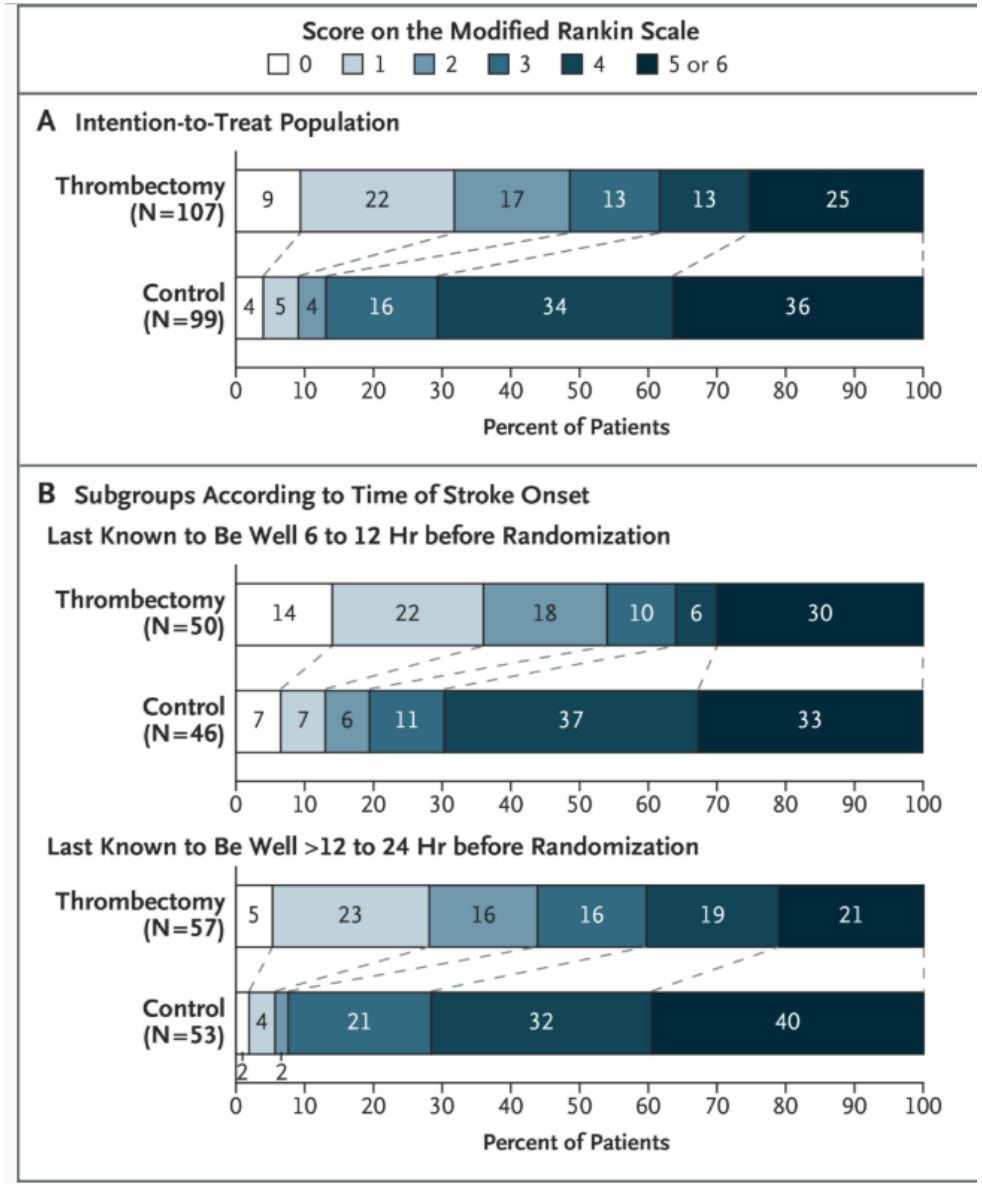


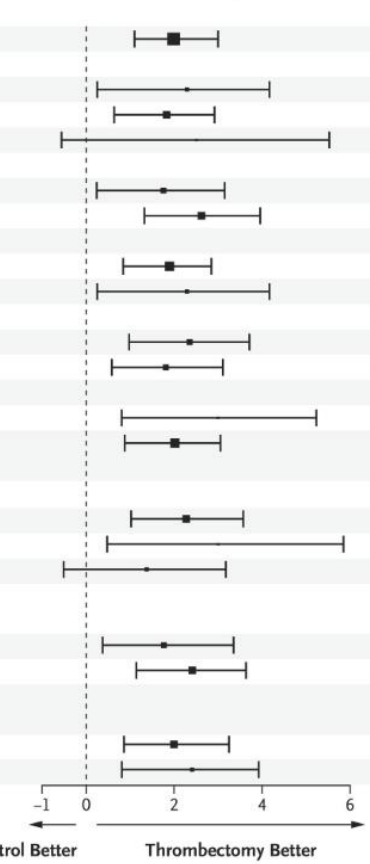
ORIGINAL ARTICLE

Thrombectomy 6 to 24 Hours after Stroke with a Mismatch between Deficit and Infarct

Authors: Raul G. Nogueira, M.D., Ashutosh P. Jadhav, M.D., Ph.D., Diogo C. Haussen, M.D., Alain Bonafe, M.D., Ronald F. Budzik, M.D., Parita Bhuva, M.D., Dileep R. Yavagal, M.D., +40, for the DAWN Trial Investigators* Author Info & Affiliations

Published November 11, 2017 | N Engl J Med 2018;378:11-21 | DOI: 10.1056/NEJMoa1706442 | VOL. 378 NO. 1

Subgroup	Adjusted Difference between Thrombectomy and Control (95% Credible Interval)	Posterior Probability Benefit	Posterior Probability Heterogeneity
Overall	2.0 (1.1 to 3.0)	>0.99	
Mismatch criteria			0.47
Group A	2.3 (0.3 to 4.2)	0.99	
Group B	1.8 (0.6 to 2.9)	>0.99	
Group C	2.5 (−0.6 to 5.5)	0.95	
Sex			0.14
Male	1.8 (0.2 to 3.2)	0.99	
Female	2.6 (1.3 to 4.0)	>0.99	
Age			0.42
<80 yr	1.9 (0.8 to 2.8)	>0.99	
≥80 yr	2.3 (0.3 to 4.2)	0.99	
Baseline NIHSS score			0.71
10 to 17	2.4 (1.0 to 3.7)	>0.99	
>17	1.8 (0.6 to 3.1)	>0.99	
Occlusion site			0.77
Intracranial internal carotid artery	3.0 (0.8 to 5.2)	>0.99	
First segment of the middle cerebral artery	2.0 (0.9 to 3.1)	>0.99	
Type of stroke onset			0.21
On awakening	2.3 (1.0 to 3.6)	>0.99	
Witnessed stroke	3.0 (0.5 to 5.9)	0.99	
Unwitnessed stroke	1.4 (−0.5 to 3.2)	0.93	
Interval between time that patient was last known to be well and randomization			0.22
6 to 12 hr	1.8 (0.4 to 3.4)	>0.99	
>12 to 24 hr	2.4 (1.1 to 3.6)	>0.99	
Time from first observation of symptoms to randomization			0.70
0 to 6 hr	2.0 (0.9 to 3.2)	>0.99	
>6 hr	2.4 (0.8 to 3.9)	>0.99	



Quelles perspectives de traitement aigu pour les patients anticoagulés?

Picasso’s style

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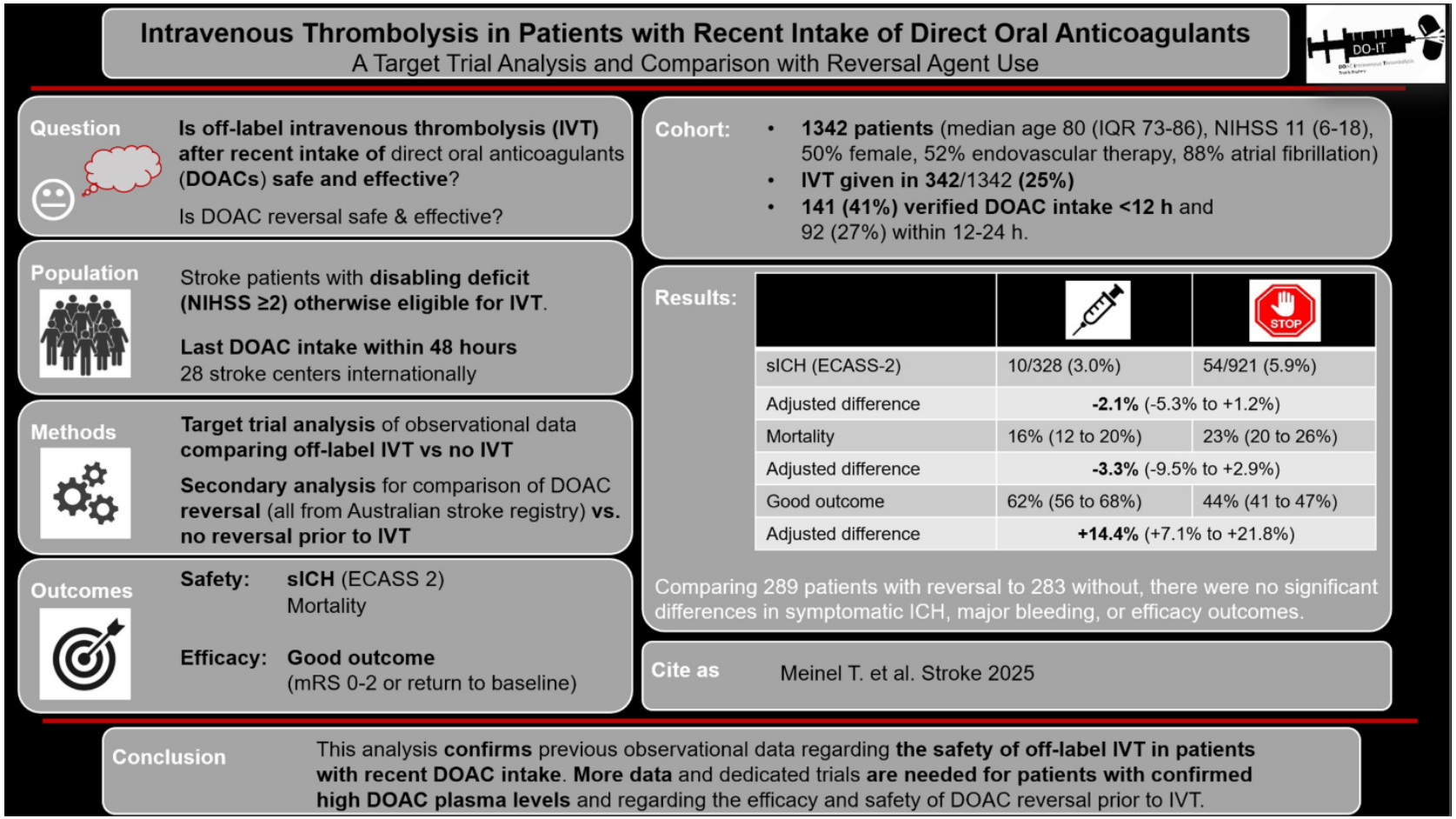
Stroke
Volume 56, Issue 10, October 2025; Pages 2836-2845
https://doi.org/10.1161/STROKEAHA.125.051384

American Heart Association.

CLINICAL TRIALS

Intravenous Thrombolysis in Patients With Recent Intake of Direct Oral Anticoagulants: A Target Trial Analysis and Comparison With Reversal Agent Use

DO-IT
DOAC Intravenous Thrombolysis Trial & Registry




DE LOREAN

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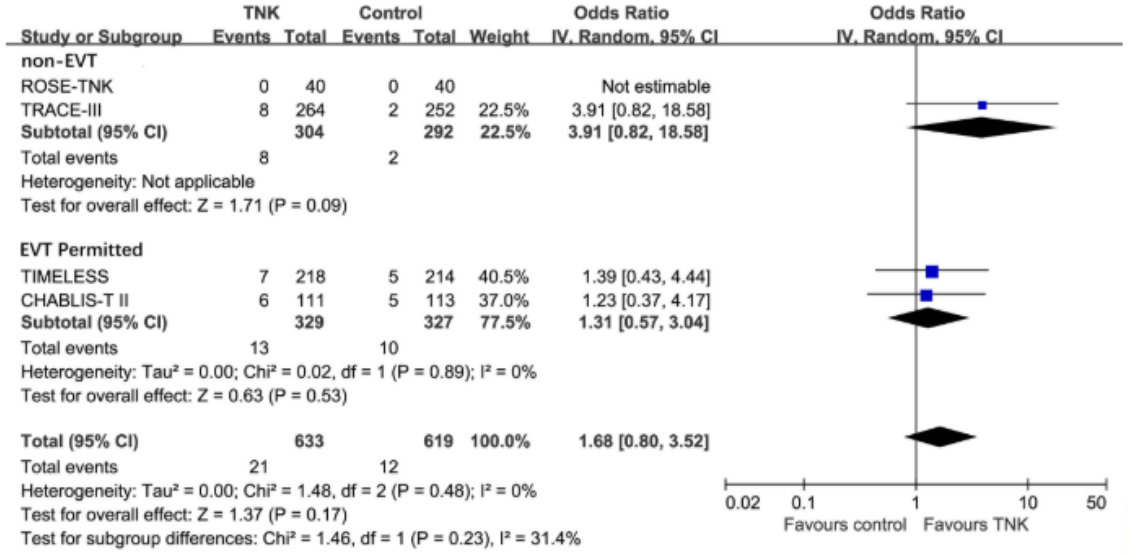
Tenecteplase for Acute Ischemic Stroke at 4.5 to 24 Hours: A Meta-Analysis of Randomized Controlled Trials

Zixin Wang, MM; Jiamin Li, MM; Xinyi Wang, MBBS; Boyi Yuan, MM; Jiameng Li, MBBS; Qingfeng Ma, MD



Tenecteplase for acute ischemic stroke at 4.5 to 24 hours: A meta-analysis of randomized controlled trials

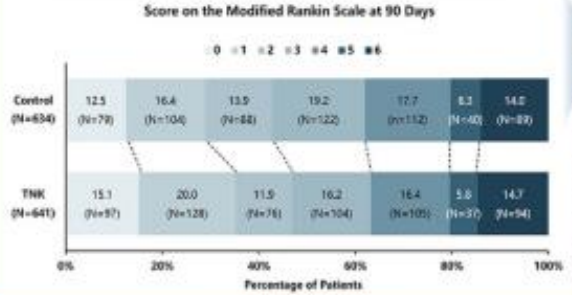
OBJECTIVE

To meta-analyze all RCTs comparing TNK with standard care or placebo in adults within 4.5-24 hours after AIS onset to determine the safety and efficacy of ultra-window TNK therapy.

RESULTS

4 RCTs, 1,278 patients

TNK, n=643 *vs.* Control, n=635



Overall (Favours TNK)

- Excellent functional outcome (OR 1.34, 95% CI 1.06-1.71, p=0.02)
- Recanalization (OR 3.30, 95% CI 1.59-6.84, p=0.001)
- sICH (OR 1.68; 95% CI 0.80-3.52; p=0.17)
- Death within 90 days (OR 1.04; 95% CI 0.76-1.43; p=0.81)

Non-EVT *vs.* EVT-Permitted Subgroups (Favours TNK)

Non-EVT	Excellent functional outcome (OR 1.46; 95% CI 1.02-2.08; p=0.04)	Recanalization (OR 6.17; 95% CI 3.36-11.33; p<0.00001)
	Good functional outcome (OR 1.50; 95% CI 1.07-2.09; p=0.02)	ENI (OR 3.21; 95% CI 1.82-5.66; p<0.0001)
EVT-permitted	Recanalization (OR 2.36; 95% CI 1.34-4.17; p=0.003	

CONCLUSION: TNK improves excellent functional outcomes and recanalization in AIS patients treated within 4.5-24 hours, without increasing the risks of sICH or mortality. Notably, TNK provides greater additional benefits when EVT is inaccessible.

Stroke. 2026;57:00–00. DOI: 10.1161/STROKEAHA.125.053256

Quelles perspectives thérapeutiques pour les patients qui ont des déficits neurologiques peu handicapants et/ou qui ne souhaitent pas une thrombolyse iv ?

AVC mineur: NIHSS ≤ 5

JAMA

QUESTION Is dual antiplatelet therapy (DAPT) noninferior to intravenous thrombolysis in patients with minor nondisabling acute ischemic stroke?

CONCLUSION Among patients with minor nondisabling acute ischemic stroke presenting within 4.5 hours of symptom onset, DAPT, compared with intravenous alteplase, met the criteria for noninferiority with regard to excellent functional outcome at 90 days.

POPULATION

496 Women
223 Men

Adults with acute minor nondisabling stroke (National Institutes of Health Stroke Scale score ≤5)

Median age: 64 years

LOCATIONS

38 Hospitals in China

INTERVENTION

760 Patients randomized
719 Patients analyzed

393 **DAPT**
Loading doses of clopidogrel and aspirin, followed by daily doses, and guideline-based antiplatelet treatment

367 **Alteplase**
Intravenous alteplase (0.9 mg/kg; maximum dose, 90 mg) followed by guideline-based antiplatelet treatment

PRIMARY OUTCOME

Excellent functional outcome, defined as a modified Rankin scale score (range, 0 [no symptoms] to 6 [death]) of 0 or 1, at 90 days

FINDINGS

Patients with excellent functional outcome at 90 days

DAPT	Alteplase
93.8% (346 of 369 patients)	91.4% (320 of 350 patients)

DAPT was noninferior to intravenous alteplase:

Risk difference of having excellent outcome at 90 days,

2.3% (unadjusted 95% CI, −1.5% to 6.2%);
P value for noninferiority < .001

Chen H, Cui Y, Zhou Z, et al; for the ARAMIS Investigators. Dual antiplatelet therapy vs alteplase for patients with minor nondisabling acute ischemic stroke: the ARAMIS randomized clinical trial. *JAMA*. Published June 27, 2023. doi:10.1001/jama.2023.7827

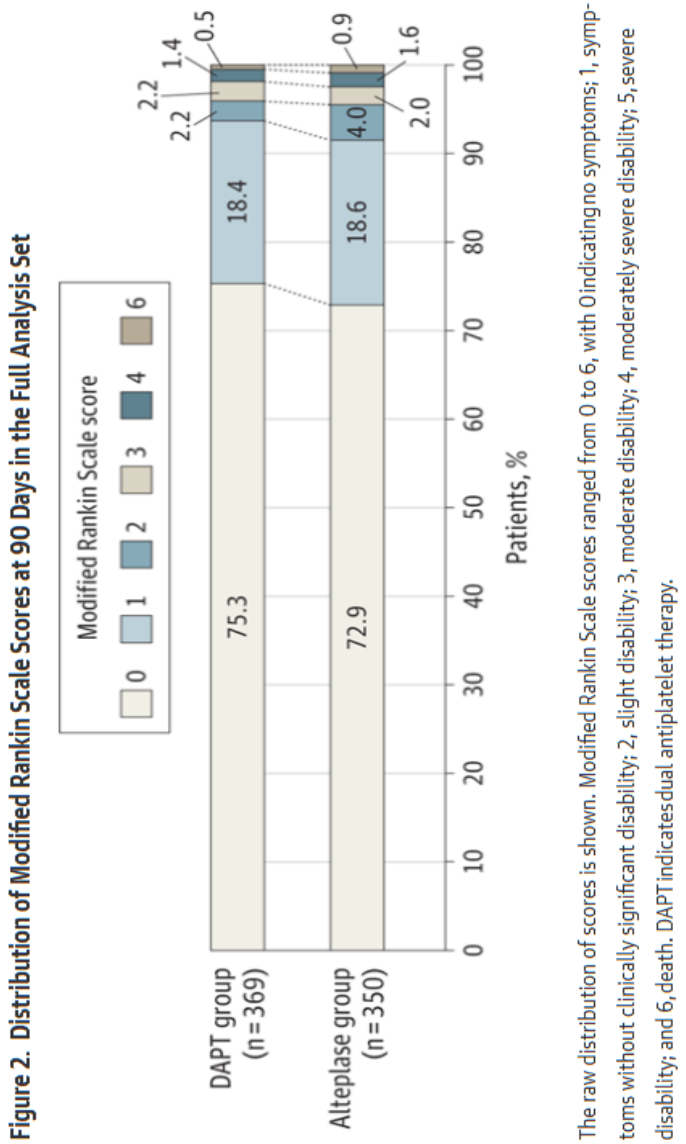


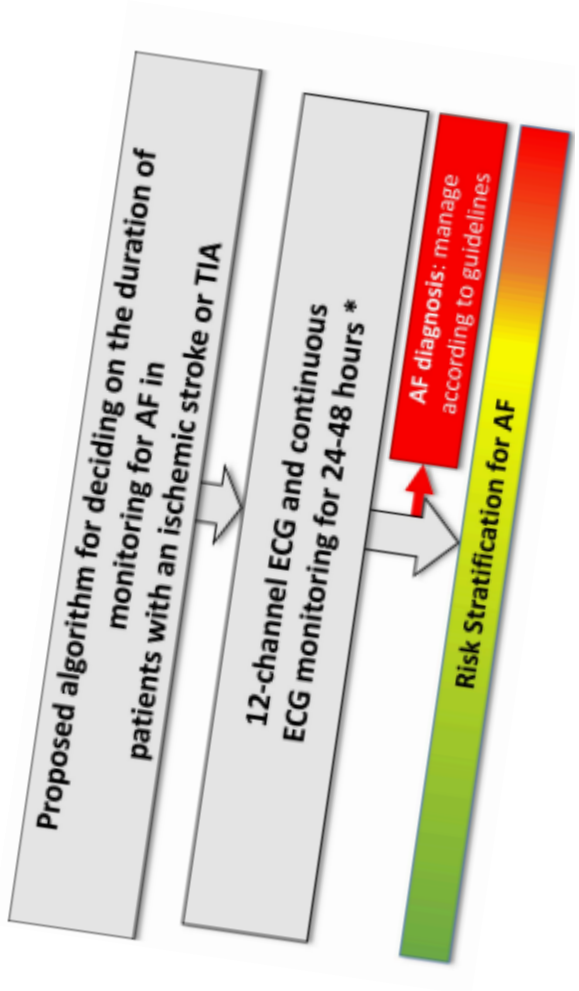
Figure 2. Distribution of Modified Rankin Scale Scores at 90 Days in the Full Analysis Set

The raw distribution of scores is shown. Modified Rankin Scale scores ranged from 0 to 6, with 0 indicating no symptoms; 1, symptoms without clinically significant disability; 2, slight disability; 3, moderate disability; 4, moderately severe disability; 5, severe disability; and 6, death. DAPT indicates dual antiplatelet therapy.

ARAMIS trial

Y-a-t-il des nouvelles perspectives sur la recherche de l’étiologie de l’AVC ischémique ?

Swiss Stroke Society position paper on atrial fibrillation monitoring and management after ischaemic stroke: a shift from understanding the index stroke to preventing the next one



Risk factors #	Comparatively low	Comparatively high
Patient characteristics		
Age, years	<60	≥75
Manifest heart failure, peripheral or coronary artery disease	Absent	Present
Echo and ECG biomarkers		
Left ventricular ejection fraction	Normal	<40%
Left atrial dilatation	Diameter <40 mm	>45mm LAVI >42 ml/m²
Monitoring on the stroke unit	SVES <120/24h Atrial run <5 beats	SVES ≥500/24 hours Atrial runs >20 successive beats/24h
Blood Biomarkers		
MRproANP (validated for all stroke subtypes)	<92 pmol/L	>200 pmol/L
NTproBNP (no validated cut off*)	<200 pg/mL	>400 pg/mL

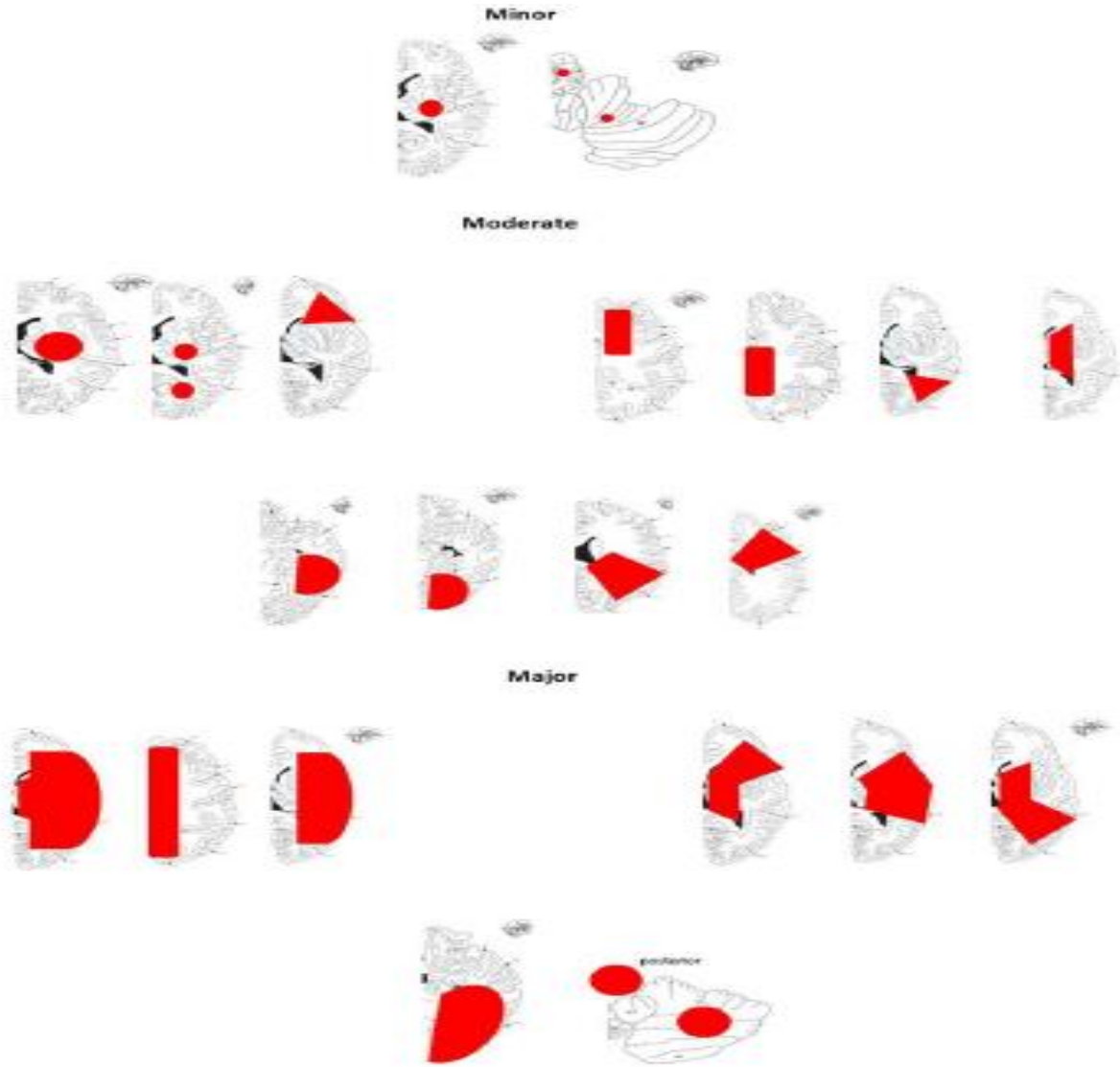
Low risk	Moderate risk	High risk
0 high risk factors AND ≥2 low risk factors	All between low and high risk	≥2 high risk factors
No further monitoring	Rhythm monitoring for a total of 7 – 30 days	Prolonged rhythm monitoring ≥30 days either by implantable cardiac device or repeat external loop-recorders (Holter)

- Les patients présentant un **AVC ischémique** ou un **accident ischémique transitoire (AIT)** doivent bénéficier d'une **surveillance ECG continue pendant 24 à 48 heures** (soit en unité neurovasculaire, soit par télémétrie par la suite).
- Une **surveillance cardiaque prolongée**, incluant les **moniteurs cardiaques implantables**, doit être envisagée chez les patients à **haut risque de fibrillation auriculaire**, quelle que soit l'étiologie de l'AVC.
- Une **fibrillation auriculaire diagnostiquée sur un tracé à une seule dérivation** (par exemple via un dispositif portable) doit être **confirmée par un médecin expérimenté** en analyse du rythme cardiaque.
  En cas de doute ou si la qualité du tracé est insuffisante pour un diagnostic définitif, **le diagnostic de FA ne doit pas être retenu**.
- Chez les patients présentant une **probabilité intermédiaire d'AVC lié à un foramen ovale perméable (FOP)**, pour lesquels une **fermeture percutanée du FOP** est envisagée, il est recommandé d'effectuer **au moins un Holter ECG de 7 jours** avant de conclure.

Thomas Meinel et al. Swiss Stroke Society position paper on atrial fibrillation monitoring and management after ischaemic stroke: a shift from understanding the index stroke to preventing the next one. Swiss Med Wkly. 2025;155:4170

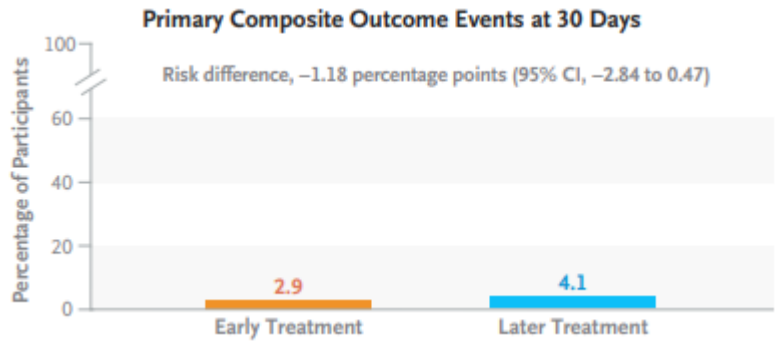
Early versus Later Anticoagulation for Stroke with Atrial Fibrillation

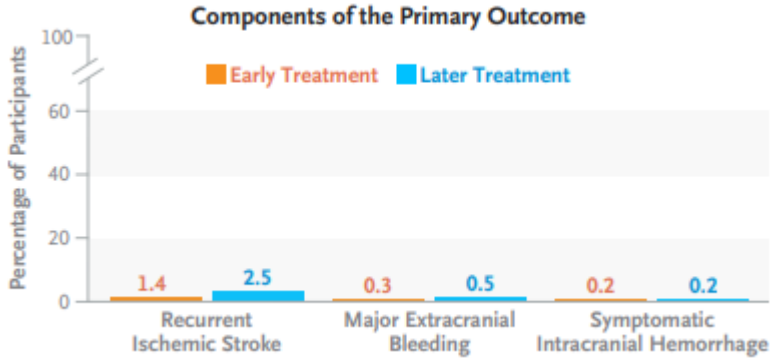
Fischer U et al. DOI: 10.1056/NEJMoa2303048

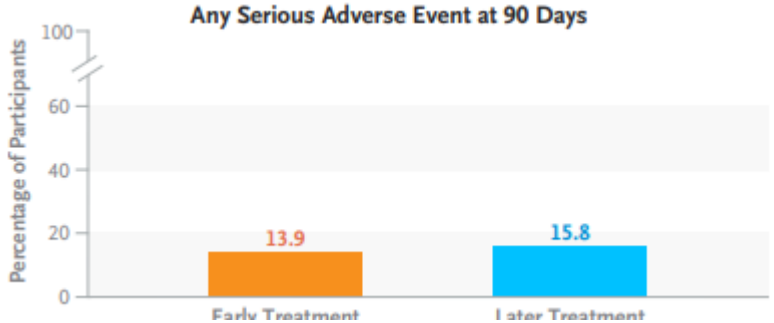


Early versus Later Anticoagulation for Stroke with Atrial Fibrillation

Fischer U et al. DOI: 10.1056/NEJMoa2303048







- 2013 participants atteints de fibrillation auriculaire et d'un accident vasculaire cérébral ischémique confirmé par imagerie ont été répartis aléatoirement pour recevoir soit une initiation d'un DOAC :
  - **précoce** :
    - à ≤ 48 heures après le début de l'AVC chez les patients présentant un AVC mineur ou modéré;
    - Le 6e ou 7e jour chez ceux présentant un AVC majeur.
  - **tardive** :
    - le 3e ou 4e jour pour les AVC mineurs;
    - le 6e ou 7e jour pour les AVC modérés;
    - Le 12e, 13e ou 14e jour pour les AVC majeurs.
- Le critère principal d'évaluation était un composite comprenant la récidive d'un AVC ischémique, une embolie systémique, une hémorragie majeure extracrânienne, une hémorragie intracrânienne symptomatique ou un décès d'origine vasculaire survenant à 30 jours suivant la randomisation.

Principal Investigators

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Bern, Switzerland

ELAPSE elapse-trial.ch

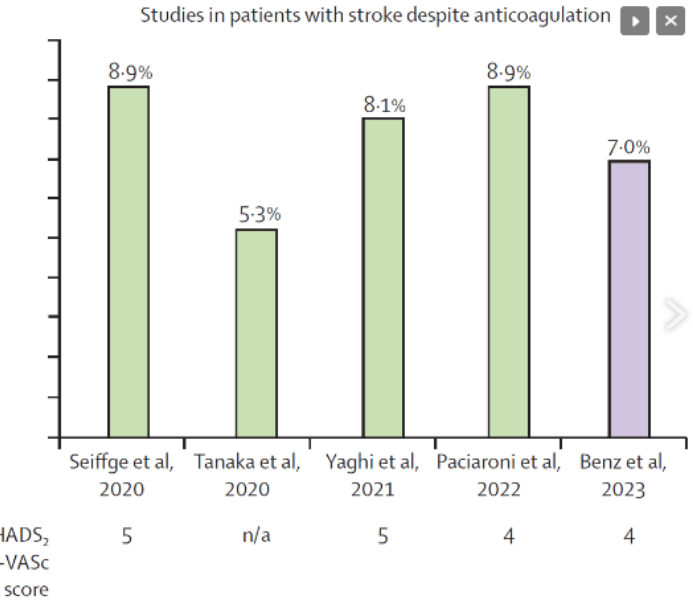


Table 1: Annualized rate of recurrent ischemic stroke despite anticoagulant therapy in patients with AF (Seiffge et al Lancet Neurology, 2024)

Item 2 of 3

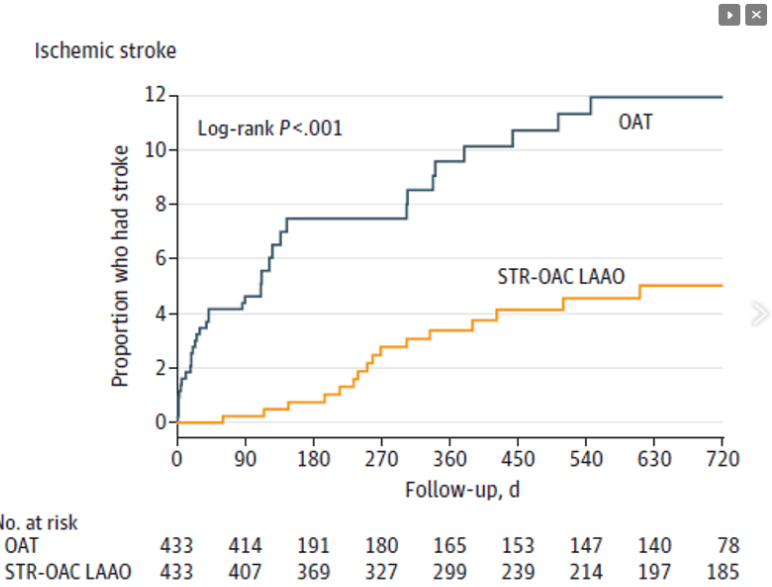
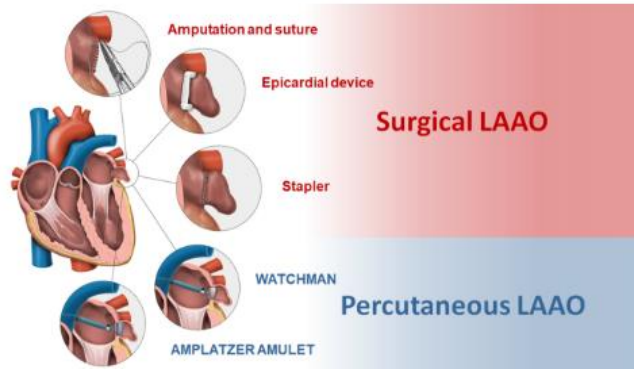


Figure 3: Data from a matched observational study using propensity score matching to compare outcomes in patients with AF and ischemic stroke despite anticoagulant therapy who receive DOAC therapy alone after the event compared to LAAO+DOAC (Maarse et al., 2024)

Item 3 of 3

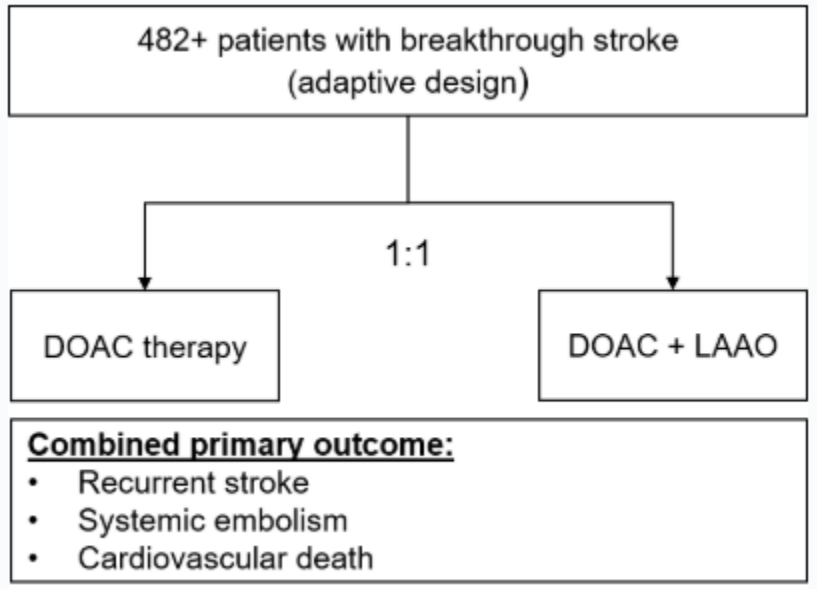


Stroke
Volume 56, Issue 7, July 2025; Pages 1928-1937
https://doi.org/10.1161/STROKEAHA.124.043867

TOPICAL REVIEWS

Left Atrial Appendage Occlusion and Its Role in Stroke Prevention

David J. Seiffge, MD, Maurizio Paciaroni, MD, Elias Auer, MD, Jacqueline Saw, MD, Michelle C. Johansen, MD, PhD, and Alexander P. Benz, MD



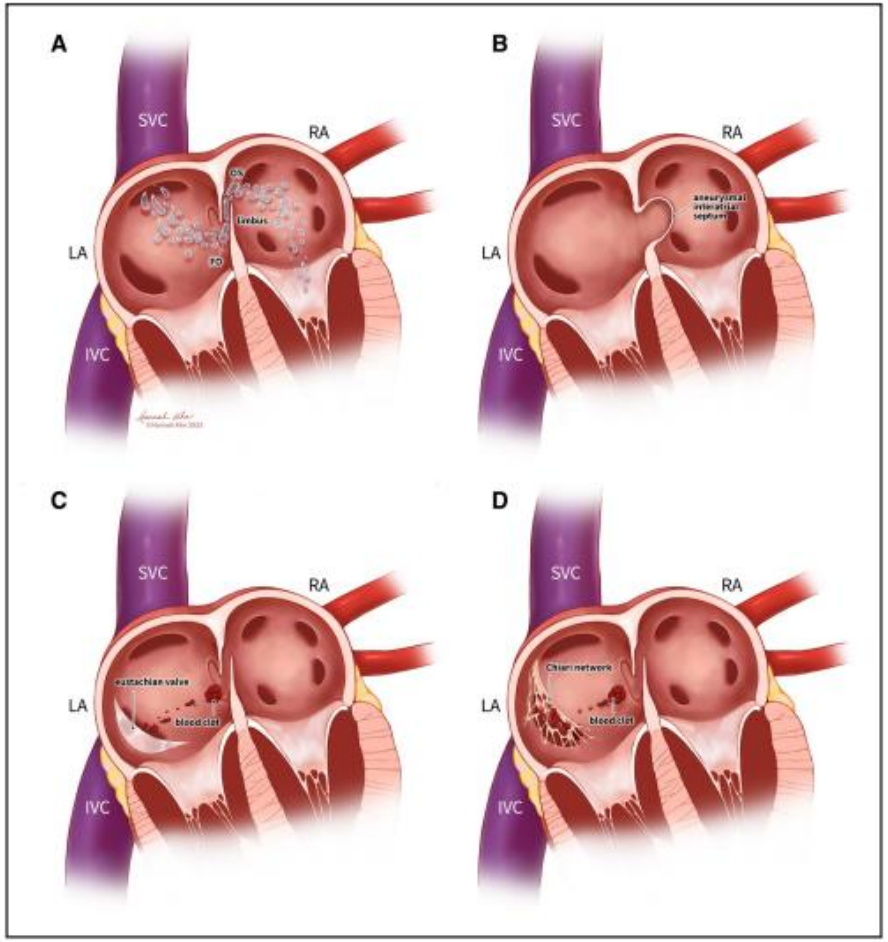


Figure 2. High-risk patent foramen ovale (PFO) features.
A, Number of bubbles used to quantify the size of the PFO during an echocardiogram with agitated bubble solution. **B**, Aneurysmal and mobile interatrial septum. **C**, Eustachian valve directing the venous thrombi to the PFO. **D**, Chiari network facilitating the development of thrombi. FO indicates fossa ovalis; IVC, inferior vena cava; LA, left atrium; OS, ostium secundum; RA, right atrium; and SVC, superior vena cava.

Luciano A. Sposato et al. Patent Foramen Ovale Management for Secondary Stroke Prevention: State-of-the-Art Appraisal of Current Evidence. Stroke. 2024;55:236–247. DOI: 10.1161/STROKEAHA.123.040546

Dans la perspective d'un prochain cas d'AVCi du à un FOP…

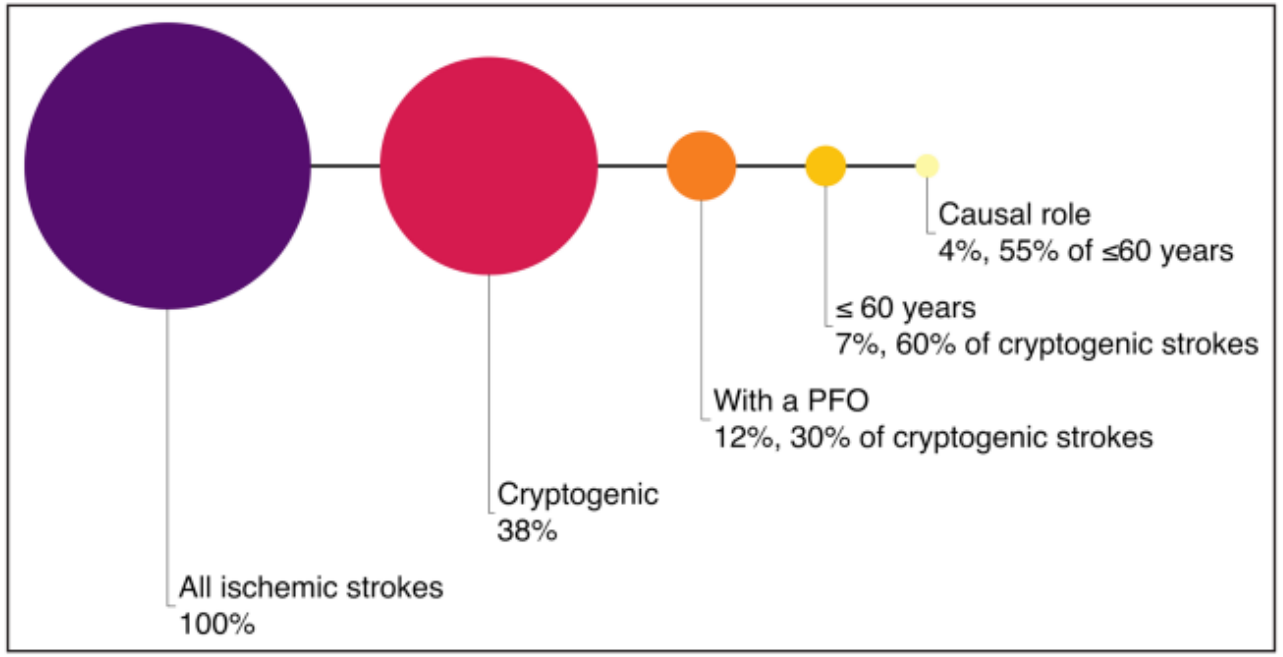


Figure 1. Proportion of ischemic stroke with patent foramen ovale (PFO) as a potential cause.
Proportions are derived from a cohort of 15 239 patients with ischemic stroke from the London Ontario Stroke Registry with complete echocardiograms (unpublished).

Luciano A. Sposato et al. Patent Foramen Ovale Management for Secondary Stroke Prevention: State-of-the-Art Appraisal of Current Evidence. Stroke. 2024;55:236–247. DOI: 10.1161/STROKEAHA.123.040546

EUROPEAN STROKE JOURNAL

European Stroke Organisation (ESO) Guidelines on the diagnosis and management of patent foramen ovale (PFO) after stroke

What are the ESO recommendations for diagnosing, treating, and long-term managing patients with ischemic stroke and PFO?

Methods

ESO EUROPEAN STROKE ORGANISATION — THE VOICE OF STROKE

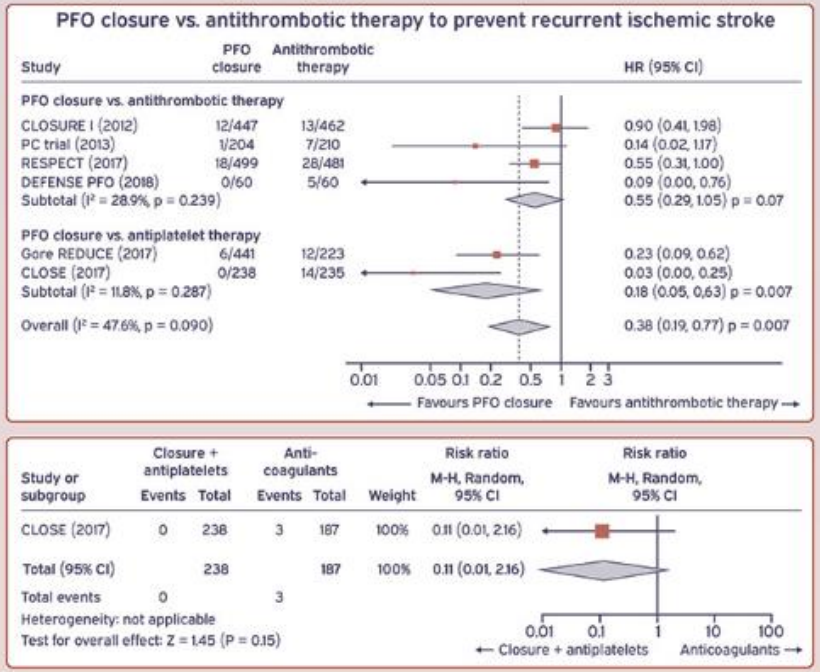
Develop PICO questions

Systematic searches of databases: MEDLINE, EMBASE, CINAHL and SCOPUS

Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) framework

Decision by consensus on the PICO questions

Results



Conclusion

Stroke with no cause other than PFO, age 18–60:
- Recommend PFO closure plus antiplatelets
- Evidence is strong

Stroke with PFO plus other possible cause(s), age 18–60:
- Prefer PFO closure plus antiplatelets to anticoagulants, due to superior RCT outcomes and lower risk of major bleeds
- Evidence is low quality

Stroke with PFO in age > 60:
- Enrol in trials or registries
- Evidence is insufficient

Caso, V., et al. European Stroke Journal, 2024 vcaso@hotmail.com doi.org/10.1177_23969873241247978

RoPE & PASCAL

Table 1 The RoPE Score and PASCAL Classifications

RoPE score calculator [a]	
Characteristic	**Points**
No history of hypertension	1
No history of diabetes	1
No history of stroke or transient ischemic attack	1
Nonsmoker	1
Cortical infarct on imaging	1
Age, y	
18-29	5
30-39	4
40-49	3
50-59	2
60-69	1
≥70	0
Total RoPE score (sum of individual points) = _____	

PASCAL classification system [b]		
High RoPE score (≥7)	**High-risk PFO feature (LS and/or ASA)[c]**	**PFO-related stroke**
Absent	Absent	Unlikely
Absent	Present	Possible
Present	Absent	
Present	Present	Probable

FOP

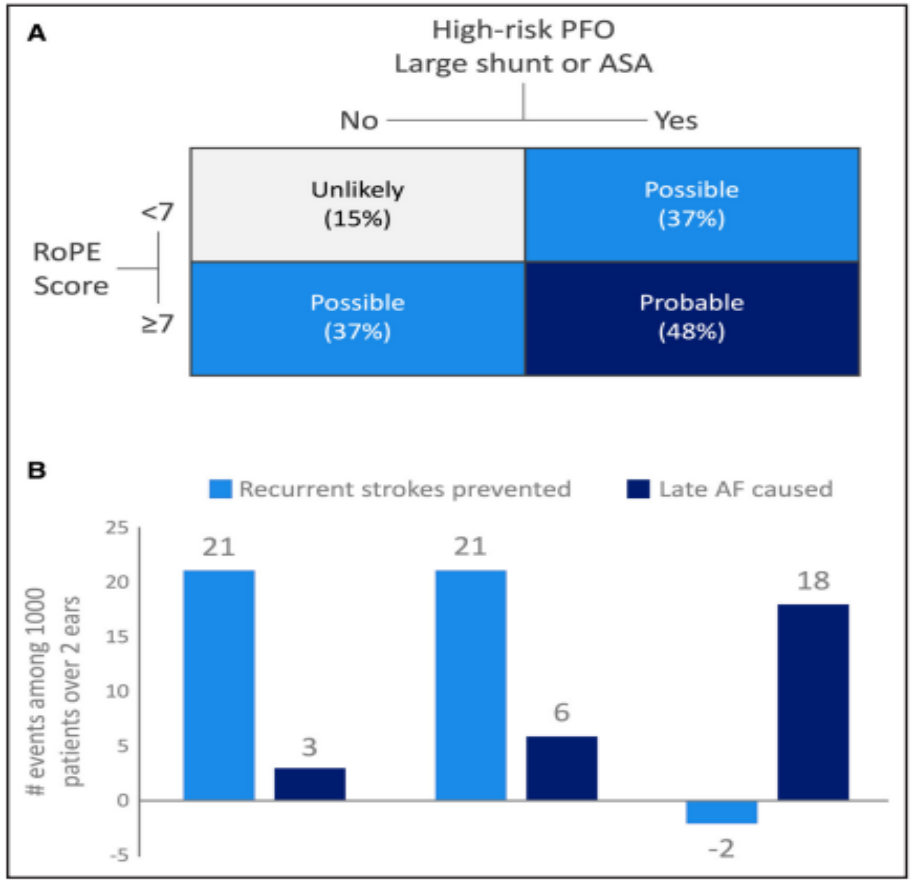


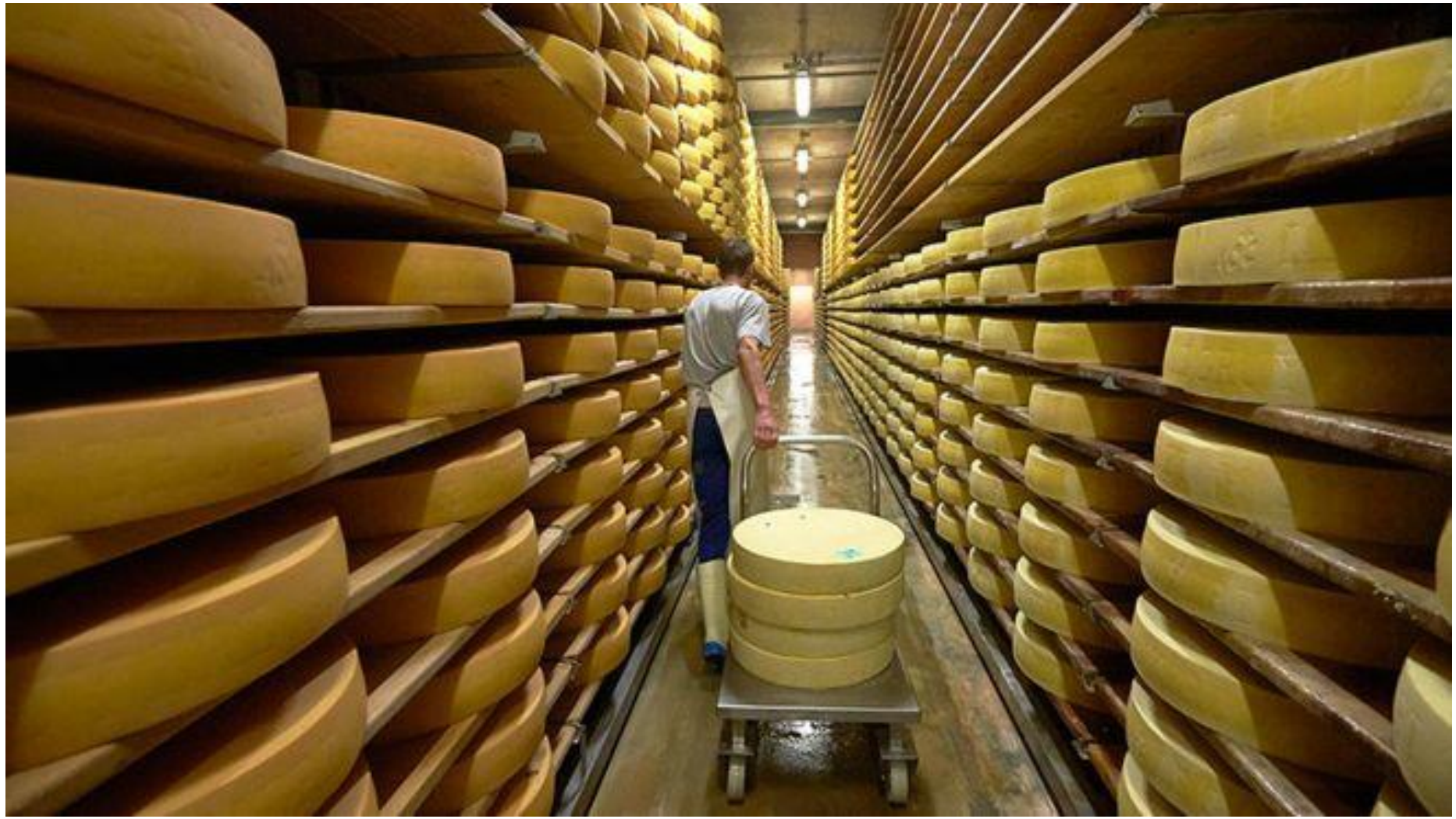
Figure 3. Risque d'AVC récurrent selon la probabilité causale d'un AVC lié au foramen ovale perméable (FOP) (classification PASCAL).

A. Chaque case représente une catégorie PASCAL. Les nombres entre parenthèses indiquent la proportion de patients dans chaque catégorie.

B. Nombre d'AVC ischémiques récurrents évités et de fibrillations auriculaires (FA) tardives induites par la fermeture du FOP chez 1000 patients sur une période de 2 ans de suivi, pour chaque catégorie PASCAL.

Catégorie de probabilité		RoPE < 7	RoPE ≥ 7
Élevée	Embolie pulmonaire ou TVP concomitante + FOP avec ASA ou shunt important	Probable	Très probable
Modérée	FOP avec shunt important ou anévrisme du septum interauriculaire	Possible	Probable
Faible	Petit FOP sans anévrisme du septum interauriculaire	Improbable	Possible

Luciano A. Sposato et al. Patent Foramen Ovale Management for Secondary Stroke Prevention: State-of-the-Art Appraisal of Current Evidence. Stroke. 2024;55:236–247. DOI: 10.1161/STROKEAHA.123.040546



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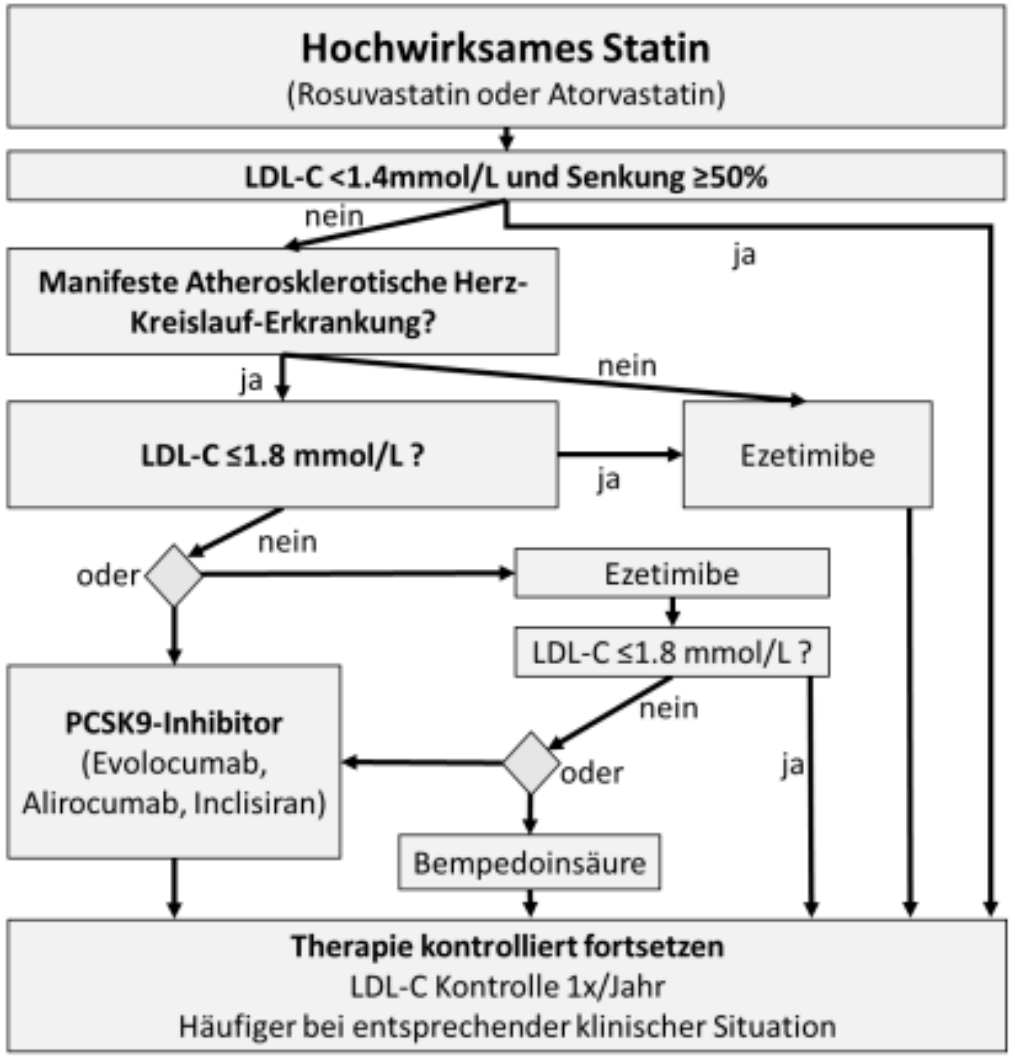
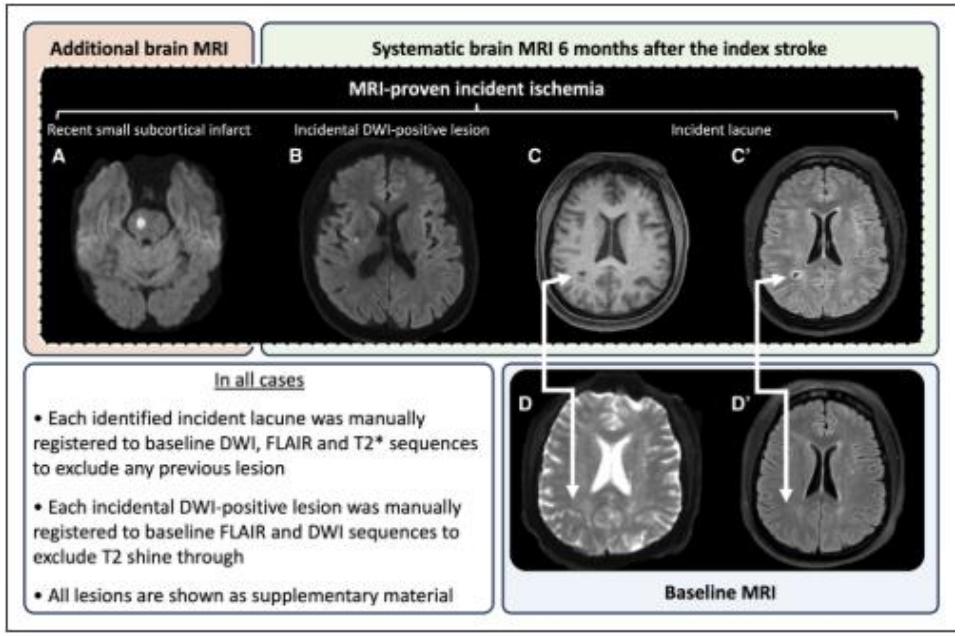
Objectifs lipidiques selon le risque vasculaire

Risque vasculaire	Faible	Modéré	Élevé ou preuve d'athérosclérose	Très élevé ou sténose symptomatique
LDL	< 3 mmol/l	< 2,6 mmol/l	Réduction > 50 % et < 1,8 mmol/l	Réduction > 50 % et < 1,4 mmol/l
Cholestérol non-HDL (TG - HDL)	—	< 3,4 mmol/l	< 2,6 mmol/l	< 2,2 mmol/l
Triglycérides (TG)	—	—	< 1,7 mmol/l	—



Lésions ischémique aigues de découverte fortuite



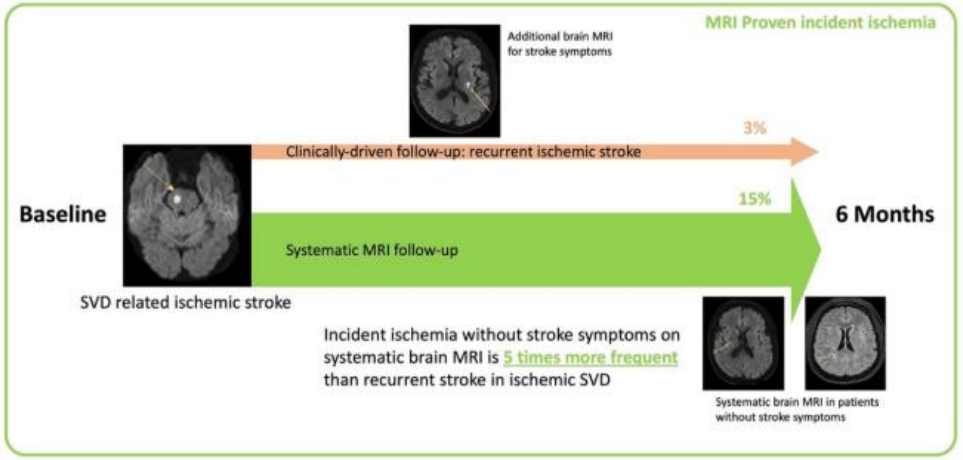


Table 2. Group Comparisons Between Patients With and Without MRI-Proven Incident Ischemia During Follow-Up (Table view)

	No MRI-proven incident ischemia (n=141)*	MRI-proven incident ischemia (n=31)*	*P* value†
At inclusion			
Female	40 (28%)	7 (23%)	0.5
Age, y	62 (56–70)	64 (57–71)	0.6
Current smoking	34 (24%)	7 (23%)	0.6
Hypertension	81 (58%)	29 (94%)	<0.001‡
Dyslipidemia	59 (43%)	21 (68%)	0.012‡
Diabetes	34 (24%)	10 (32%)	0.4
History of stroke	9 (6.4%)	4 (13%)	0.4
After 6 mo of follow-up			
Fazekas score			0.016‡
0–1	80 (61%)	10 (32%)	
2–3	51 (39%)	21 (68%)	
No. of lacunes	0 (0–2)	2 (1–7)	<0.001‡
No. of microbleeds	0 (0–1)	1 (0–8)	0.002‡
Antiplatelets	128 (91%)	30 (97%)	0.4
Antihypertensive	107 (77%)	27 (96%)	0.077
Statins	114 (82%)	28 (100%)	0.051
Anticoagulants for atrial fibrillation	9 (7%)	1 (3%)	<0.001‡
Anticoagulants for other reasons	4 (3%)	0	<0.001‡

Lina Grosset et al. MRI-Proven Incident Ischemia: A New Marker of Disease Progression in Small Vessel Diseases. Stroke Volume 56, Issue 1, January 2025; Pages 39-45

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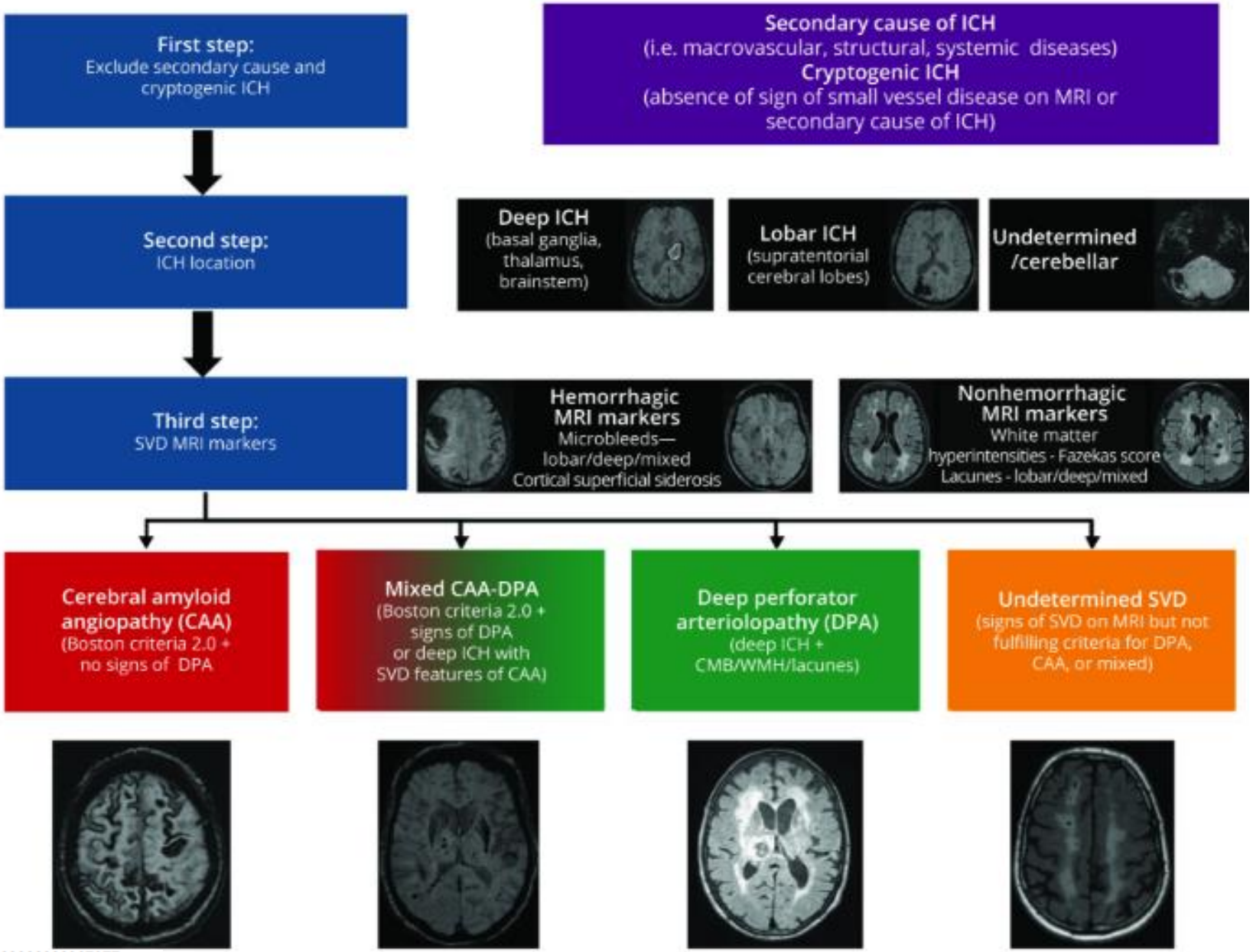
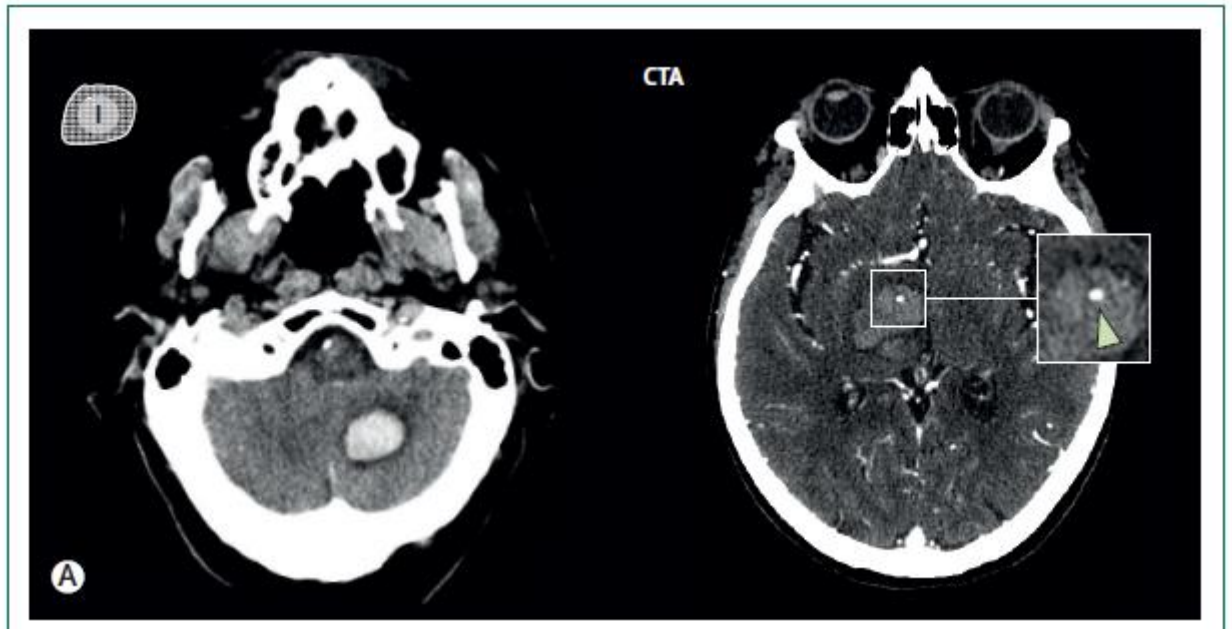
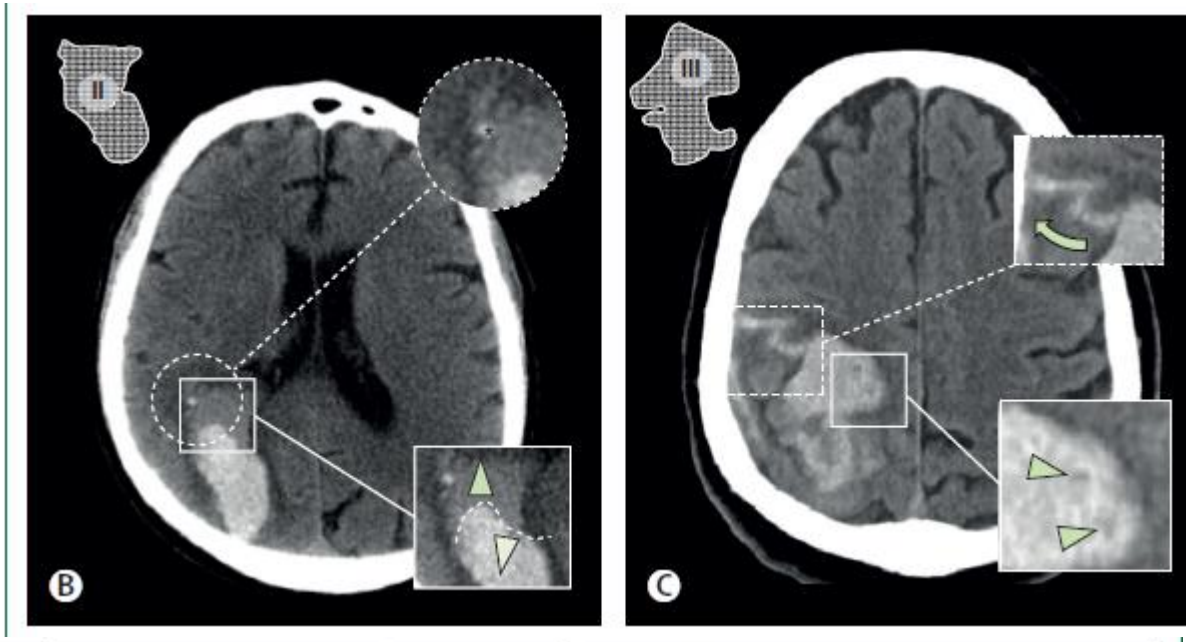
L’AVC hémorragique

CADMUS

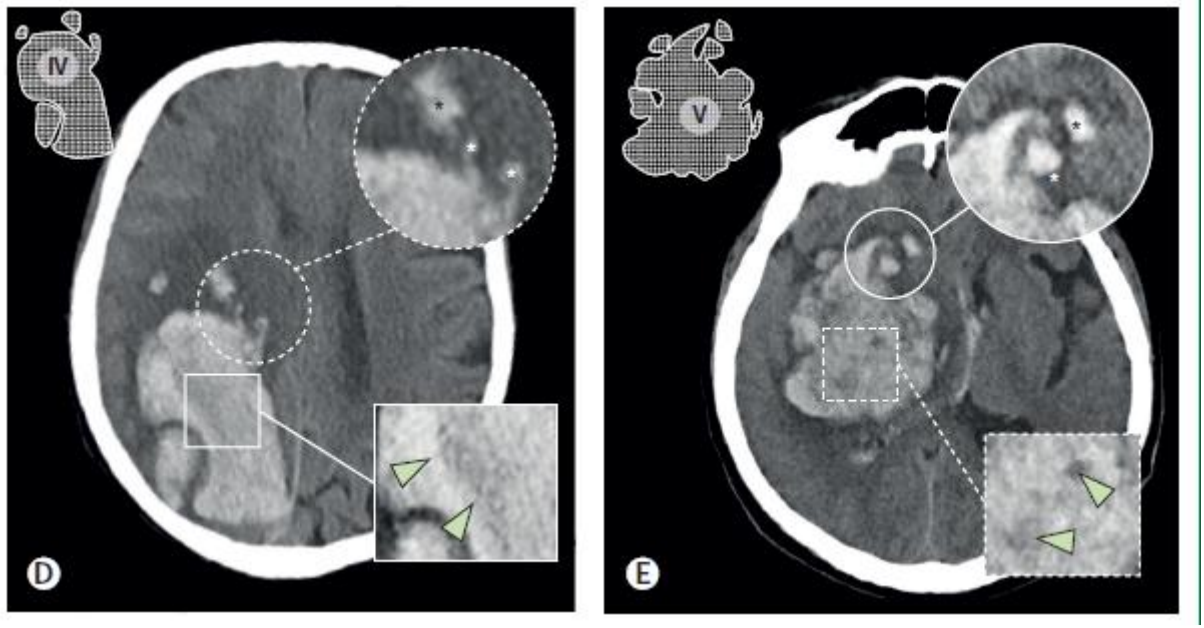
A Novel MRI-Based Classification of Spontaneous Intracerebral Hemorrhage Associated With Cerebral Small Vessel Disease







- Island sign
- Satellite sign
- Blend sign
- Swirl sign
- Spot sign



L'AVC hémorragique - bundle of care

The third Intensive Care Bundle with Blood Pressure Reduction in Acute Cerebral Haemorrhage Trial (INTERACT3): an international, stepped wedge cluster randomised controlled trial

- TA < **140 mmHg dans 30 – 60 minutes**
- **glycémie 6,1–7,8 mmol/L** chez les patients sans diabète et **7,8–10,0 mmol/L** chez les patients diabétiques
- **température corporelle ≤ 37,5°C**
- **correction rapide de l'anticoagulation** dans l'heure suivant le traitement

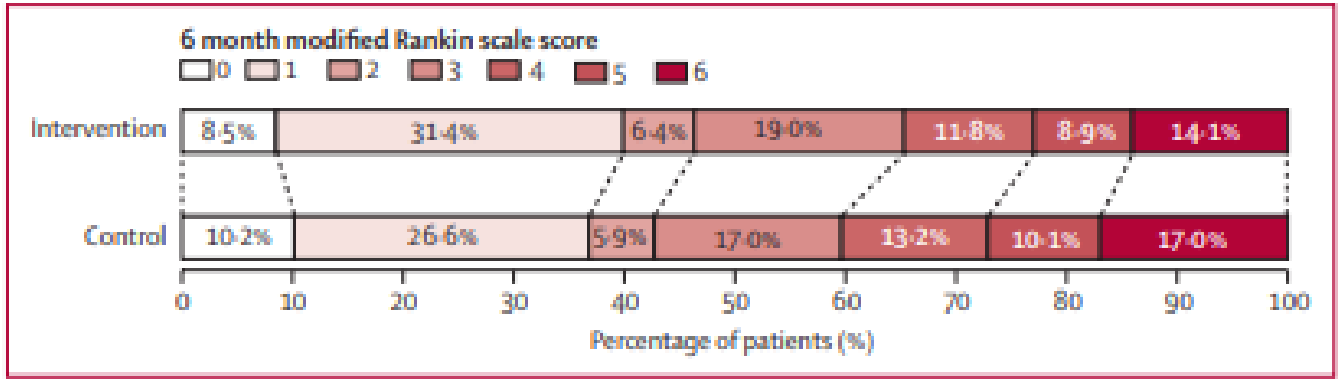


Figure 3: Functional outcome at 90 days in the care bundle and usual care groups, according to scores on the mRS

Ma, LuRobinson, Thompson et al.
The Lancet, Volume 402, Issue 10395, 27 - 40

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EUROPEAN STROKE JOURNAL

Acute care bundles should be used for patients with intracerebral haemorrhage: an expert consensus statement

ICH care bundles reduce morbidity and mortality.
We review current evidence and make practical recommendations for implementation.

Methods

Neurology

Neurosurgery

Critical Care

Emergency Medicine

Consensus Meeting May 2023

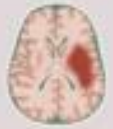
Consensus statement agreed

Results

We recommend:

Door	Stabilise patient, rapid imaging Coagulation tests
< 30 min	Reverse anticoagulant Start intensive BP lowering
< 60 min	SBP < 140, Consult Neurosurgery Achieve T < 37.5°C
7 days	Maintain SBP < 140; T < 37.5°C Maintain normoglycaemia

Conclusion



Multiple simultaneous interventions improve functional outcome

Rapid bundled care should be introduced

Quality improvement will help achieve **ambitious process targets**

Parry-Jones, A., et al. European Stroke Journal, 2023 adrian.parry-jones@manchester.ac.uk doi.org/10.1177_23969873231220235

	und auch KEIN PCC (keine Wirksamkeit bei DOAC-Blutung)	
II-Inhibitor Dabigatran	Idarucizumab (Praxbind® 2x2.5g) als spezifisches Antidot vorhanden **Kriterien**: Symptombeginn <6h, NIHSS <36, GCS>6, ICH-Volumen 1-60ml, letzte DOAC Dosis <15h oder wenn unbekannt Plasmalevel >100ng/ml, prestroke mRS <3. Falls Kritierien nicht erfüllt, **keine** Gabe von Praxbind und auch KEIN PCC (keine Wirksamkeit bei DOAC-Blutung)	Thrombinzeit und anti-IIa Aktivität bei Eintritt bestimmen
Thrombozy-	Keine spezifischen Massnahmen	TC-Infusionen potentiell schäd-

Andexanet (Ondexxya)- apixaban, rivaroxaban, edoxaban

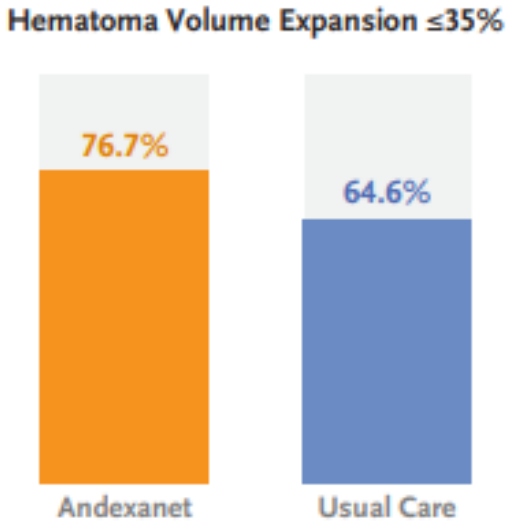


Table 2. Efficacy End Points.

End Point	Andexanet (N=224)	Usual Care (N=228)	Adjusted Difference per 100 Patients (95% CI)*	P Value*
	no./total no. (%)		*percentage points*	
Hemostatic efficacy	150/224 (67.0)	121/228 (53.1)	13.4 (4.6 to 22.2)	0.003
Hematoma volume change ≤35%†	165/215 (76.7)	137/212 (64.6)	12.1 (3.6 to 20.5)	
NIHSS score change <7 points	188/214 (87.9)	181/218 (83.0)	4.6 (–2.0 to 11.2)	
No receipt of rescue therapy between 3 hr and 12 hr	218/224 (97.3)	213/228 (93.4)	3.8 (0.0 to 7.6)	
Hematoma volume increase ≥12.5 ml‡	24/216 (11.1)	36/214 (16.8)	–5.6 (–12.0 to 0.8)	
Hemostatic efficacy, excluding patients nonevaluable for administrative reasons	150/218 (68.8)	121/225 (53.8)	14.5 (5.7 to 23.4)	

Table 3. Thrombotic Events and Deaths at 30 Days.*

Event	Andexanet (N=263)	Usual Care (N=267)	Increase per 100 Patients (95% CI)†	P Value†
	no. of patients (%)		*percentage points*	
≥1 Thrombotic event	27 (10.3)	15 (5.6)	4.6 (0.1 to 9.2)	0.048
Transient ischemic attack	0	0	—	
Ischemic stroke	17 (6.5)	4 (1.5)	5.0 (1.5 to 8.8)	
Myocardial infarction	11 (4.2)	4 (1.5)	2.7 (–0.2 to 6.1)	
Deep-vein thrombosis	1 (0.4)	2 (0.7)	–0.4 (–2.4 to 1.5)	
Pulmonary embolism	1 (0.4)	6 (2.2)	–1.9 (–4.5 to 0.2)	
Arterial systemic embolism	3 (1.1)	2 (0.7)	0.4 (–1.7 to 2.7)	
Death	73 (27.8)	68 (25.5)	2.5 (–5.0 to 10.0)	0.51

S.J. Connolly. Andexanet for Factor Xa Inhibitor–Associated Acute Intracerebral Hemorrhage. N Engl J Med 2024;390:1745-55.

RHNe Réseau Hospitalier Neuchâtelois

Self-fulfilling prophecy

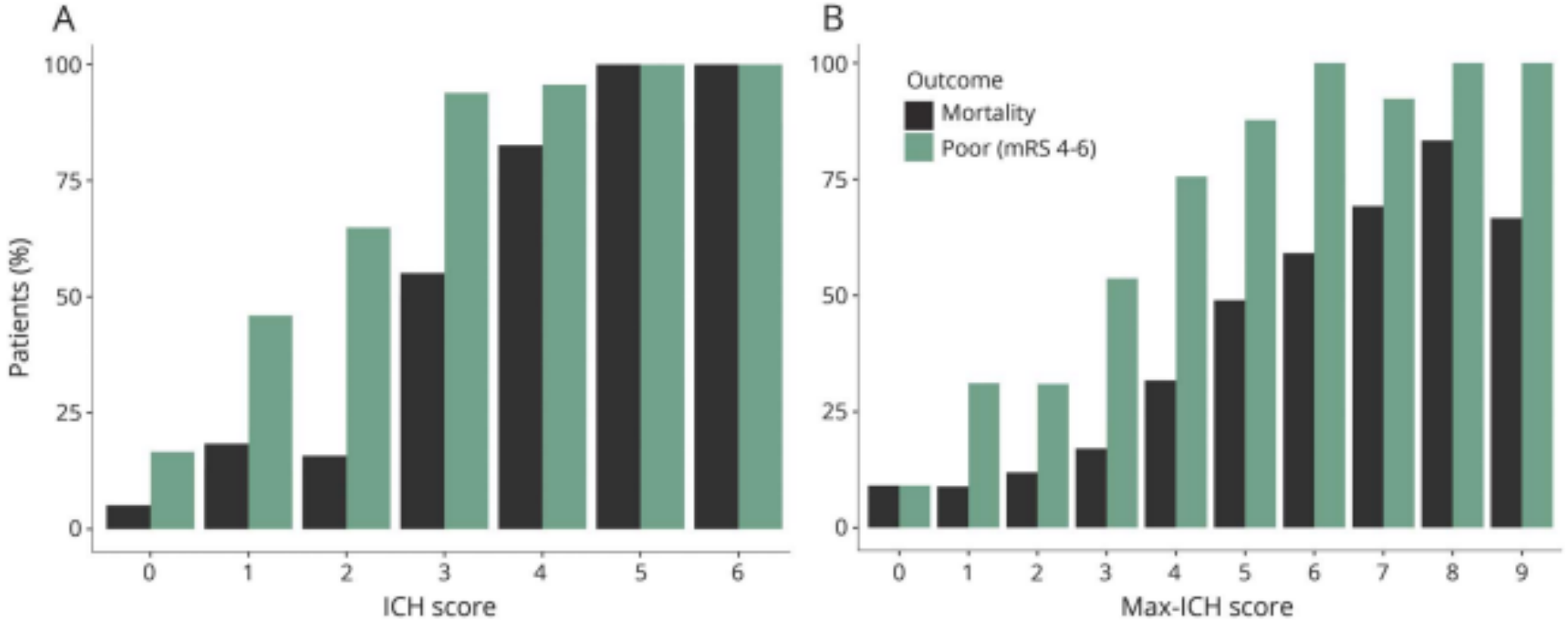
Table 1 ICH score and max-ICH score composition

ICH score variable	Points	Max-ICH score variable	Points
Glasgow Coma Scale		NIH Stroke Scale	
3–4	2	≥21	3
5–12	1	14–20	2
13–15	0	7–13	1
		0–6	0
Age, y		Age, y	
≥80	1	≥80	3
<80	0	75–79	2
		70–74	1
		≤69	0
Hematoma volume, mL		Hematoma volume, mL	
≥30	1	Lobar ≥30	1
<30	0	Lobar <30	0
		Nonlobar ≥10	1
		Nonlobar <10	0
Intraventricular hemorrhage		Intraventricular hemorrhage	
Yes	1	Yes	1
No	0	No	0
Infratentorial hemorrhage		Oral anticoagulation	
Yes	1	Yes	1
No	0	No	0

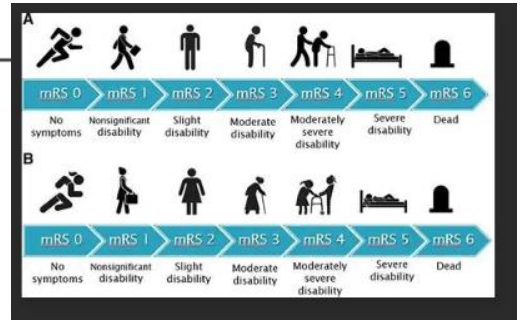
Felix A. Schmidt et al. Assessment and comparison of the max-ICH score and ICH score by external validation. Neurology® 2018;91:e939-e946. doi:10.1212/WNL.0000000000006117

Self-fulfilling prophecy (2)

Figure 1 Mortality and poor outcome by ICH score and max-ICH score rank



Observed mortality (black) and poor outcome (green) rates are shown for the ICH score (A) and max-ICH score (B) for each score rank in the maximally treated group. ICH = intracerebral hemorrhage; mRS = modified Rankin Scale.

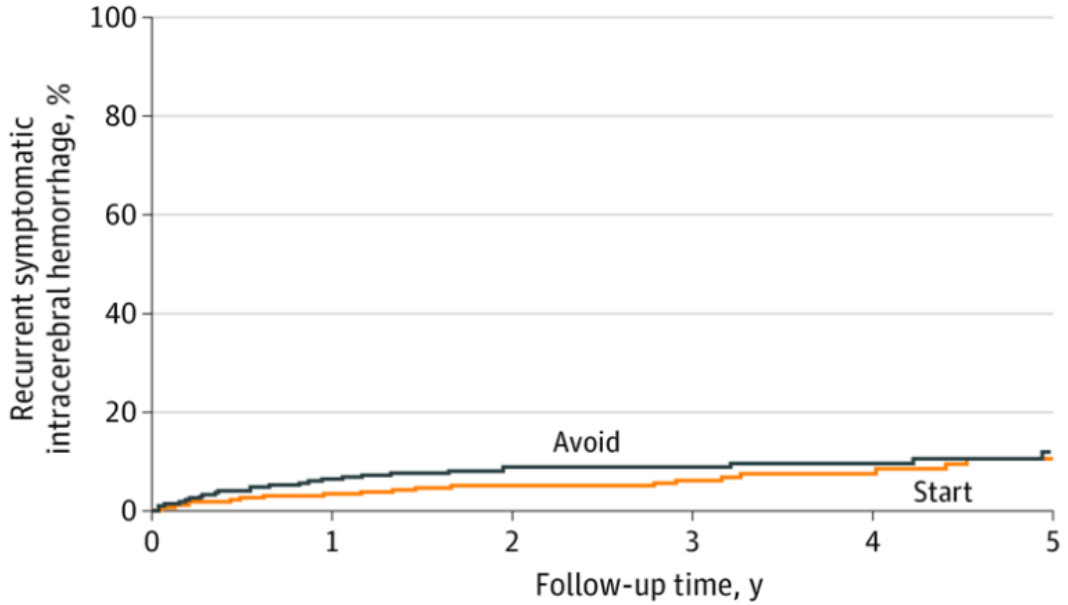


Felix A. Schmidt et al. Assessment and comparison of the max-ICH score and ICH score by external validation. Neurology® 2018;91:e939-e946. doi:10.1212/WNL.0000000000006117

Reprise du traitement anti thrombotique

RESTART (France)	NCT02966119	25	April 2022
ASPIRING external pilot phase (China and Australia)	NCT04522102	80	September 2023
STATICH (Nordic countries)	NCT03186729	69	December 2024

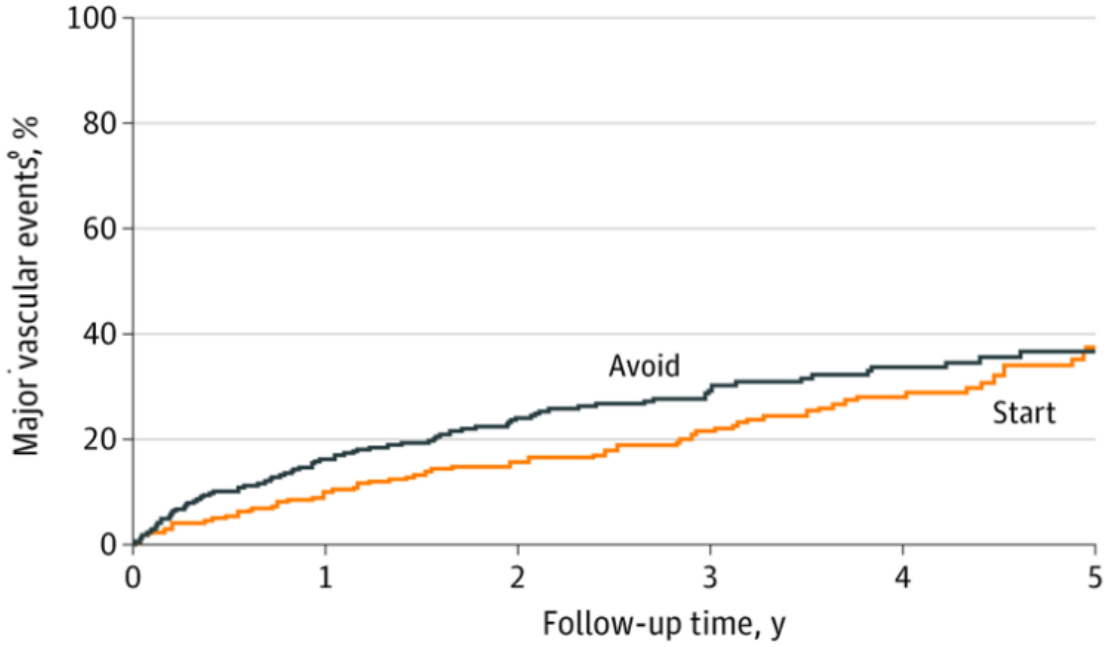
Figure 2. Risk of the First Occurrence of Recurrent Symptomatic Intracerebral Hemorrhage



No. at risk (No. of cumulative events)

Avoid	268 (0)	233 (17)	205 (23)	159 (23)	99 (24)	67 (25)
Start	268 (0)	239 (9)	211 (13)	161 (15)	111 (17)	65 (21)

Figure 3. Risk of the First Occurrence of a Major Vascular Event



Al-Shahi Salman R, Dennis MS, Sandercock PAG, et al. Effects of antiplatelet therapy after stroke caused by intracerebral hemorrhage: extended follow-up of the RESTART randomized clinical trial. *JAMA Neurol*. Published online September 3, 2021. doi:10.1001/jamaneurol.2021.2956

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Après un AVC hémorragique «l'homéostasie» est atteinte après environ 24h, quand le risque d'augmentation en volume de l'hématome diminue; ainsi, une reprise du traitement anti plaquettaire pourrait être prévue à 7-14 jours.

Table 3. Hazard of Intracranial Thrombotic and Hemorrhagic Events Within 30 Days of Follow-Up (Table view)

Variable	Unadjusted HR (95% CI) *P* value	Adjusted HR (95% CI)* *P* value
No resumption of anticoagulation vs early or late resumption		
Acute ischemic stroke	4.6 (1.2–17.0) *P*=0.0239	7.5 (1.3–45.5) *P*=0.0277
New ICH or symptomatic ICH expansion	4.0 (1.1–14.0) *P*=0.0328	3.9 (0.7–20.3) *P*=0.1076
Composite outcome	2.4 (0.8–6.7) *P*=0.1162	2.1 (0.5–9.5) *P*=0.35
Late vs early resumption of anticoagulation		
Acute ischemic stroke	0.8 (0.2–3.0) *P*=0.6999	1.3 (0.2–7.9) *P*=0.7772
New ICH or symptomatic ICH expansion	1.0 (0.3–2.9) *P*=0.9393	0.3 (0.1–1.3) *P*=0.112
Composite outcome	0.7 (0.3–1.5) *P*=0.3565	0.5 (0.2–1.3) *P*=0.1466

Pargol Balali et al. Timing of Antiplatelet Resumption After Intracerebral Hemorrhage: A Sophie's Choice. Stroke. 2023;54:546–548. DOI: 10.1161/STROKEAHA.122.041466

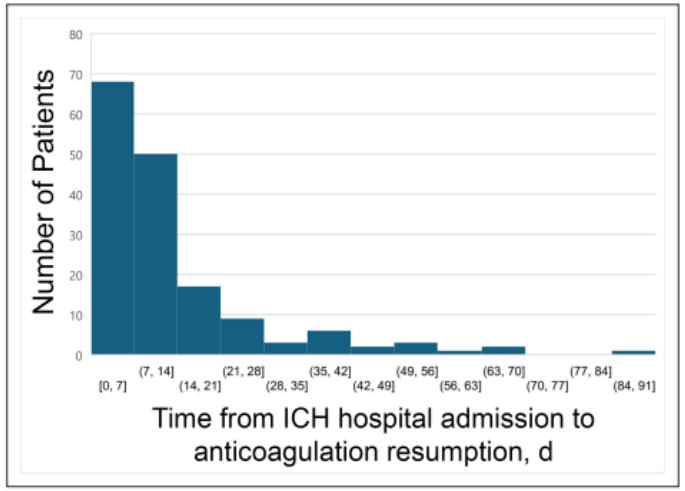


Figure 1. Distribution of time (days) from ICH hospital admission to therapeutic anticoagulation resumption. ICH indicates intracranial hemorrhage.

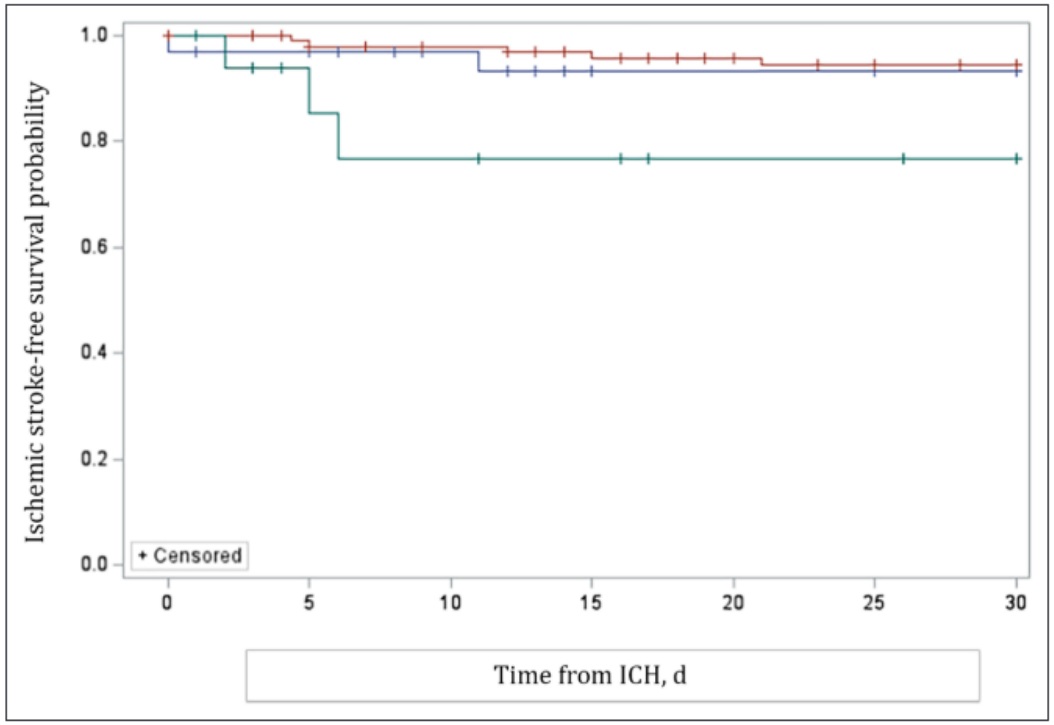


Figure 2. Unadjusted Kaplan–Meier curve for acute ischemic stroke within 30 days of the index ICH among patients with anticoagulation resumption within 7 days (blue), from 7 to 30 days (red), and beyond 30 days or never after the ICH. ICH indicates intracranial hemorrhage.

J Am Heart Assoc. 2024;13:e032094. DOI: 10.1161/JAHA.123.032094

Les microhémorragies et le traitement anti-thrombotique

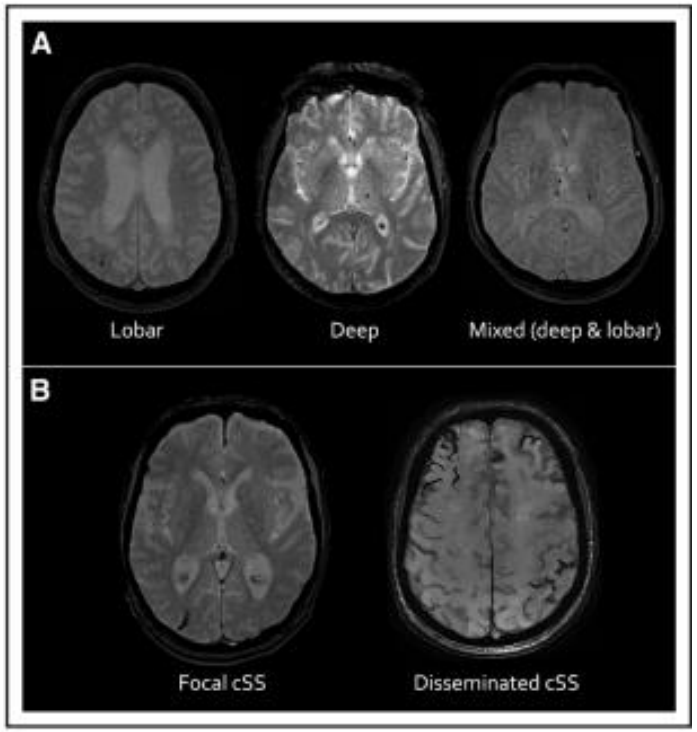
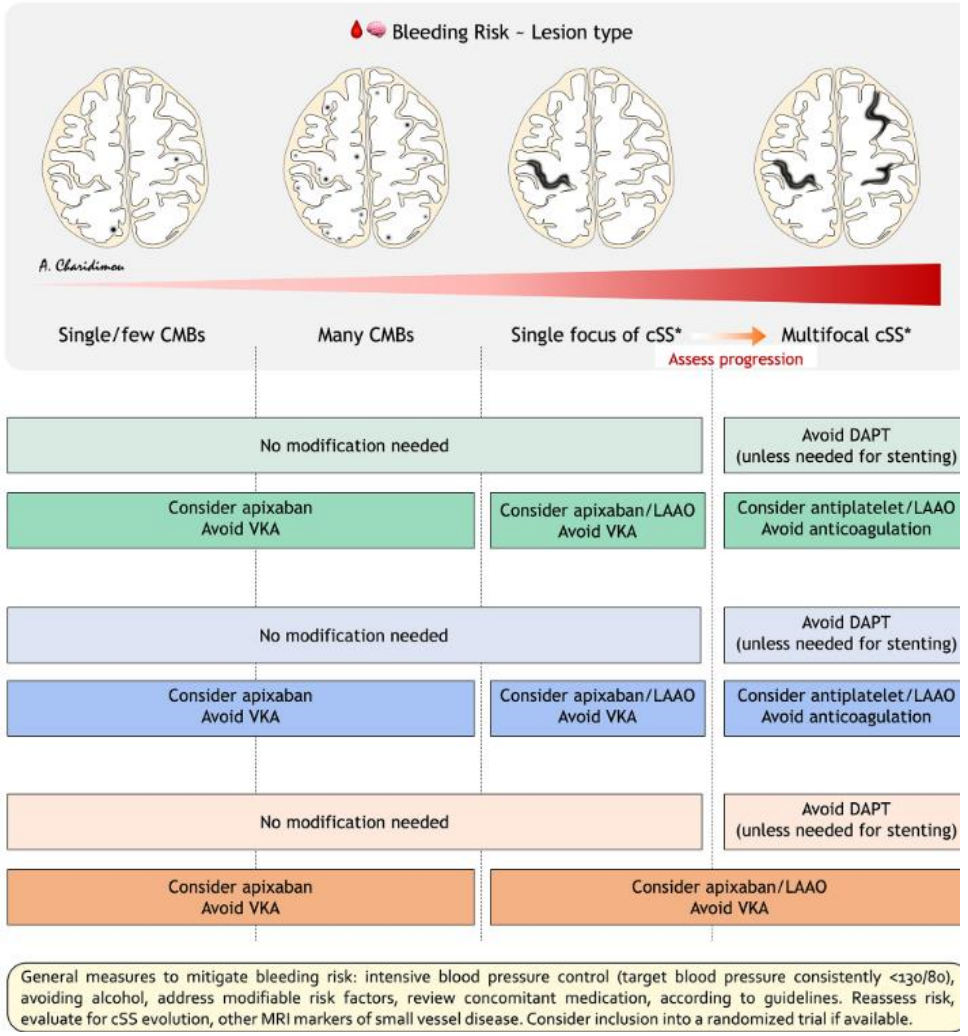


Figure 1. Anatomic patterns of cerebral microbleeds (CMBs) and cortical superficial siderosis (cSS) extent on blood-sensitive magnetic resonance imaging sequences.

Managing antithrombotics and anticoagulants in patients with asymptomatic cerebral microbleeds (CMBs) or cortical superficial siderosis (cSS)



Andreas Charidimou. Cardiovascular Management in Asymptomatic (Silent)

Cerebral Microbleeds and Suspected Cerebral Amyloid Angiopathy. Stroke. 2024;55:1101–1112.

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"No, no, no... the perspective is all wrong."