
2024
13 et 14 avril
12ème
SÉMINAIRE de
ARDIOLOGIE
INTERVENTIONNELLE
de TROYES
PROGRAMME PRÉLIMINAIRE
PRÉSIDENTS DU SÉMINAIRE :
Dr. Alain Shqueir et Pr. Camille Brasselet,
Vice-président Pr. laurent Farroux
Ancien co-président : Pr. Thierry Folliguet
COMITÉ D'ORGANISATION - ASSOCIATIONS DE CARDIOLOGIE :
Seine et Marne, Champagne Aisne & Ardennes, Collège des Cardiologues de l'Est,
Formation Cardiologique Alsacienne Libérale,
Association des Specialistes en Cardiologie de la Côte d'Or
COMITÉ SCIENTIFIQUE :
Alain Shqueir, Camille Brasselet, Patrick Arnold, Mohamed Belhameche,
Eric Bergoend, Alain Bérribi, Christian Breton, Bernard Carette, Laurent Chapoutot,
Yves Cottin, Simon El Haddad, Laurent Faroux, Thierry Folliguet, Philippe Lang, Hugo Manzi,
Guillaume Lebreton, Nicolas Lellouche, Pascal Leprince, Bruno Mailler, Pierre Nazeyrollas,
Vito-Giovanni Ruggieri, Ahmed Salhi, Jérôme Schwartz, Emmanuel Teiger, Sylvain Rubin
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Bienvenue

BIENVENUE

DIMANCHE 14 AVRIL 2024 - SALON JACQUARD

09H00 - 10H30 L'INTERVENTIONNEL EFFICACE

Modérateurs: Dr. Bernard Carette, Dr. Philippe Lang (Mulhouse), Dr. Alexandre Kovalchuk (Jossigny)

- Ablation de la Fibrillation Atriale : quelle énergie choisir ? — **Dr. Ahmed Salhi (Jossigny)**, **Dr. Fatma Zouari (Jossigny)**
- Insuffisance Cardiaque : l'ablation de la FA améliore le pronostique — **Dr. Jérome Schwartz (Nancy)**, **Dr. Elon Zerah (Nancy)**
- Les AVC Ischémiques : la prise en charge optimale — **Pr. Hassan Hosseini (Crétei)**

10H30 - 11H00 PAUSE ET VISITE DE L'ÉXPOSITION - SALON JERSEY

11H00 - 12H00 L'INTERVENTION ADAPTÉE

Modérateurs: Dr. Christien Breton (Nancy), Dr.Philippe Jauffrion (Villejuif), Dr. Laurent Chapoutot (Troyes

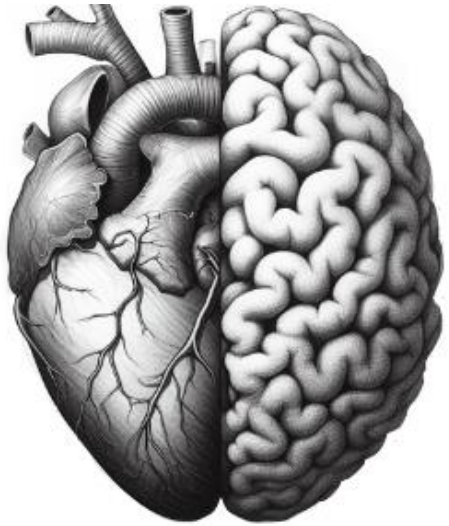
- La Maladie Coronaire Stable et les récommandations — **Pr. Yves Cottin (Dijon)**
- Lésions de Bifurcation Coronaire: quel traitement de référence ? **Pr. Camille Brasselet (Reims)**

12H00 - 12H30 DÉMONSTRATION ET FONCTIONALITÉ DES PROTHÈSES VALVULAIRES

Modérateur : Dr. Jean-Paul Bellefleur (Troyes) — **Pr.Thierry Folliguet (Créteil)**

12H30 - FIN DU SÉMINAIRE

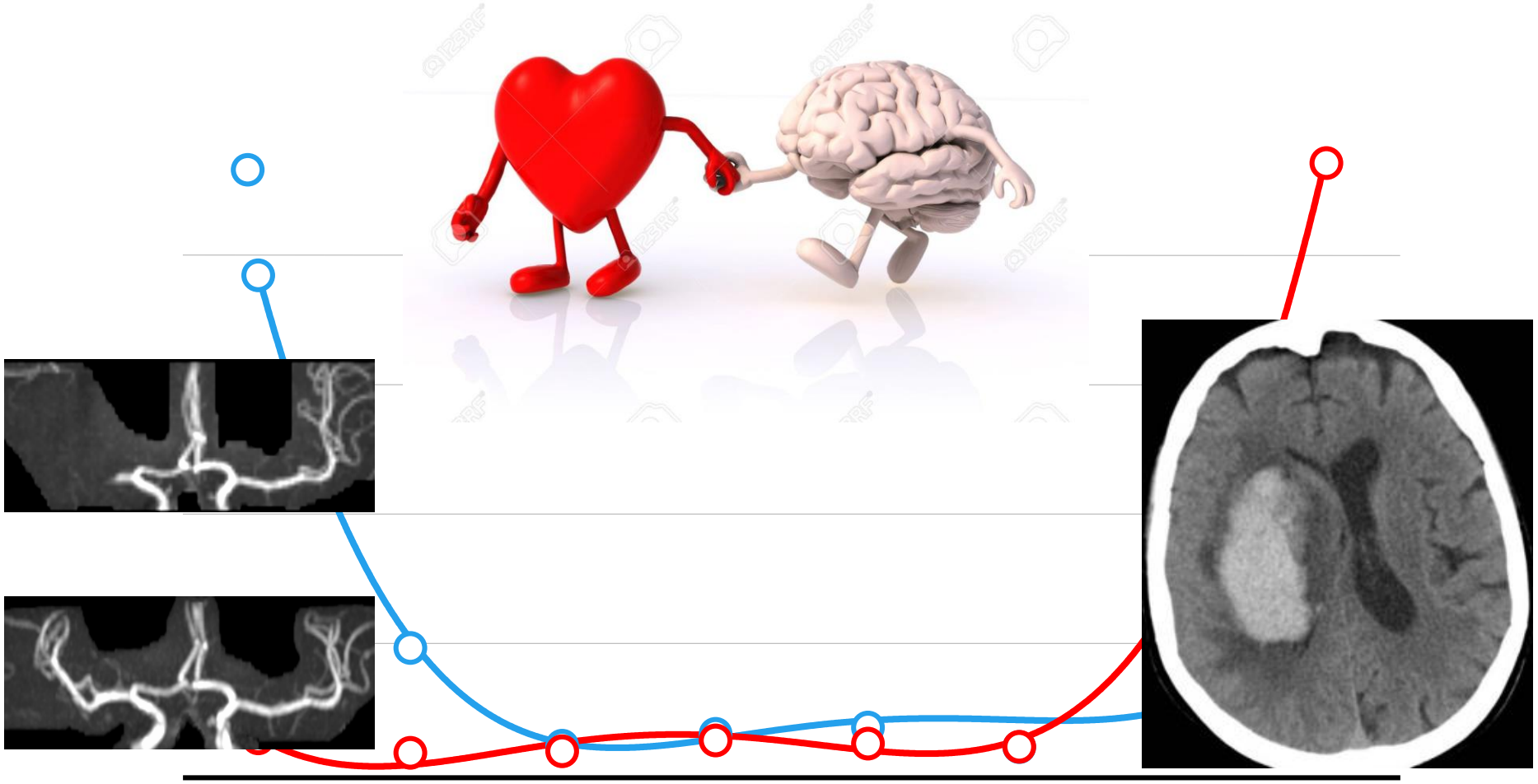
ICCE : INSTITUT COEUR ET CERVEAU DE L'EST PARISIEN



- Pr Hassan HOSSEINI
- Dr Stéphane COSSON
- Dr Ruxandra STANESCU

- HPPE
- Champigny-sur-Marne
- Secrétariat : 0752525555
- Email : consultation.hosseini@gmail.com

Yalta
Fév 1945



Cas clinique

Homme de 75 ans, hémiplégie G brutale

ATCD

- DNID
- HTA
- HC
- IDM, angioplastie stent
- Fibrillation atriale
- trt: anti hypertenseur, ADO, atorvastatine, AVK, aspirine

Hémiplégie G H3,

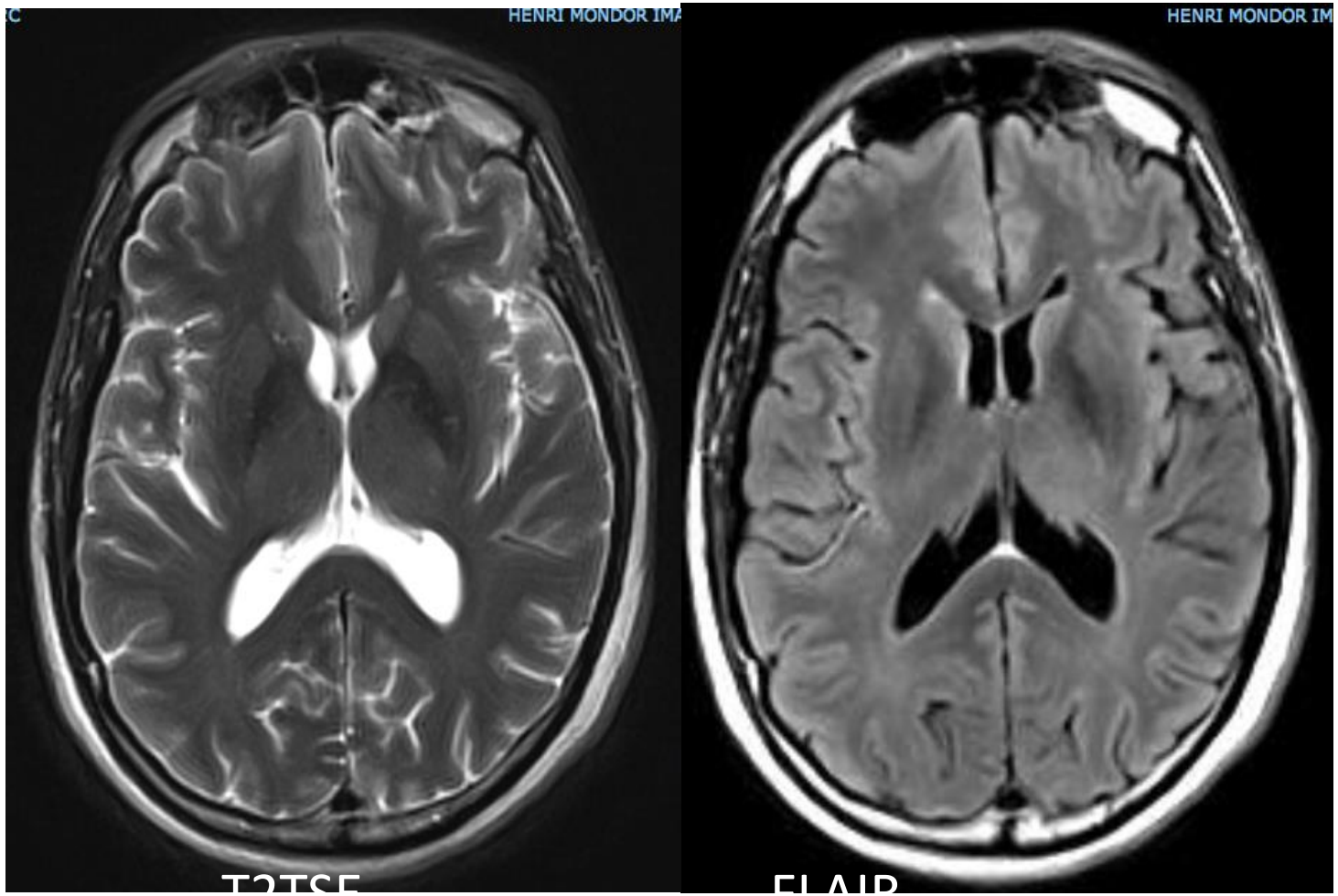
NIHSS 13


Votre CAT ?

IRM

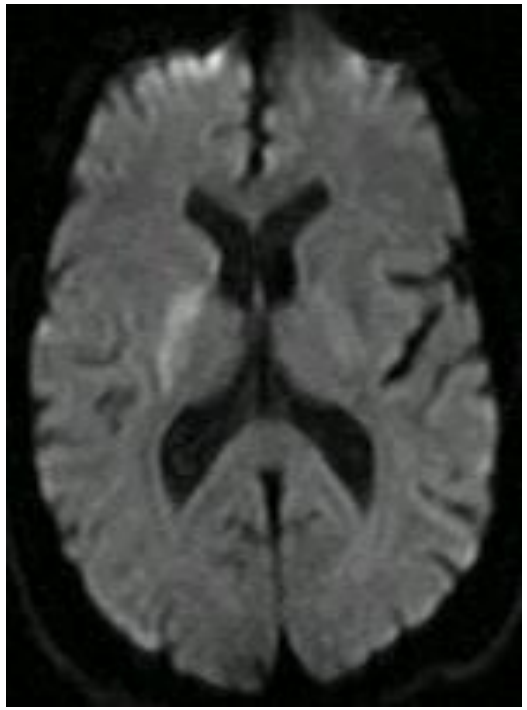
T2

FLAIR



IRM

DIFFUSION



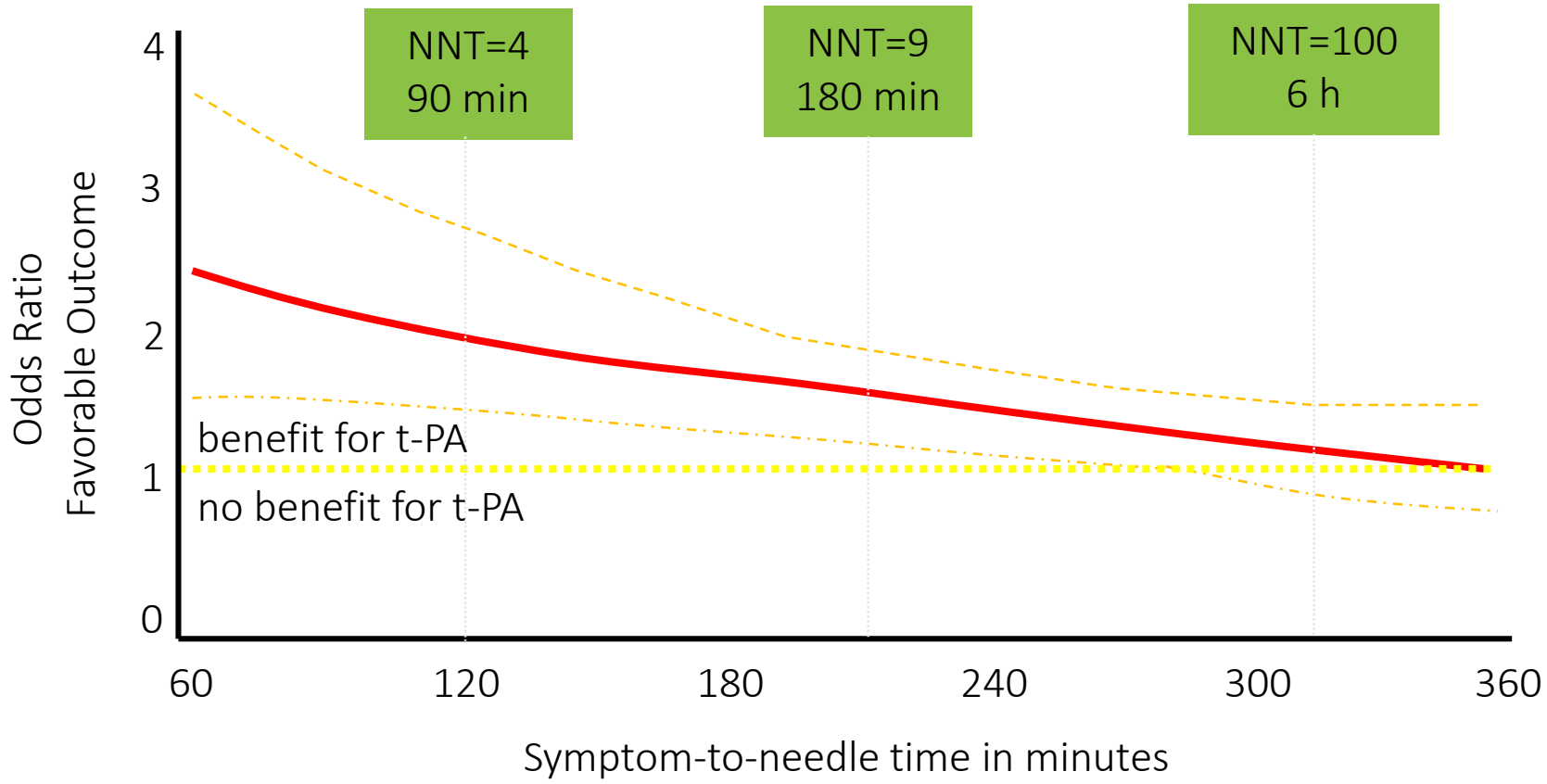
TOF

Autre donnée manquante ?

INR = 1.2

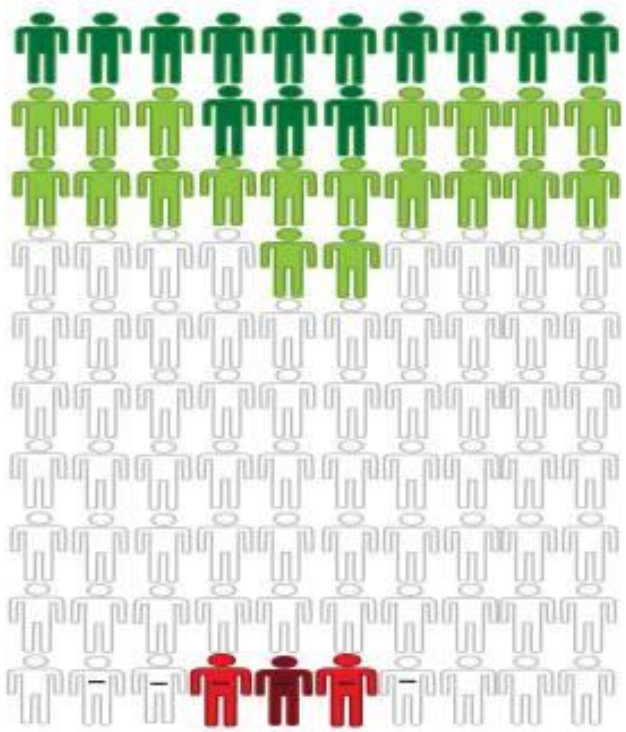
Traitement ?

TIV : Alteplase ou Tenecteplase



The ATLANTIS, ECASS, and NINDS rt-PA Study Group Investigators.
Lancet 2004; 363: 768

TPA for Cerebral Ischemia within 3 Hours of Onset-Changes in Outcome Due to Treatment



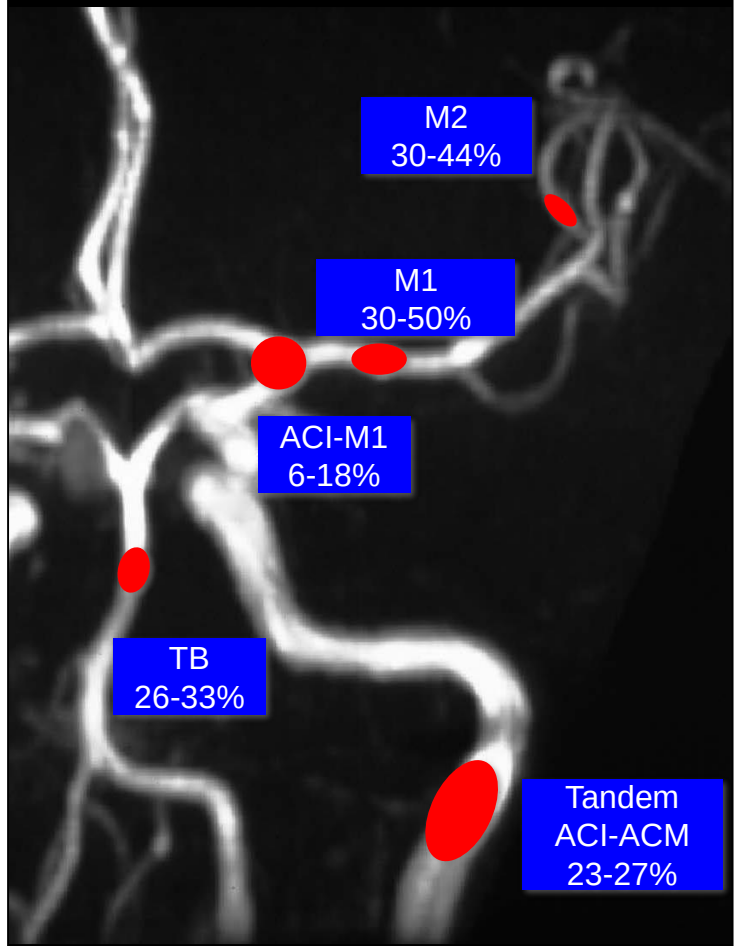
Changes in final outcome as a result of treatment:

- Normal or nearly normal
- Better
- No major change
- Worse
- Severely disabled or dead

Early course:

- No early worsening with brain bleeding
- Early worsening with brain bleeding

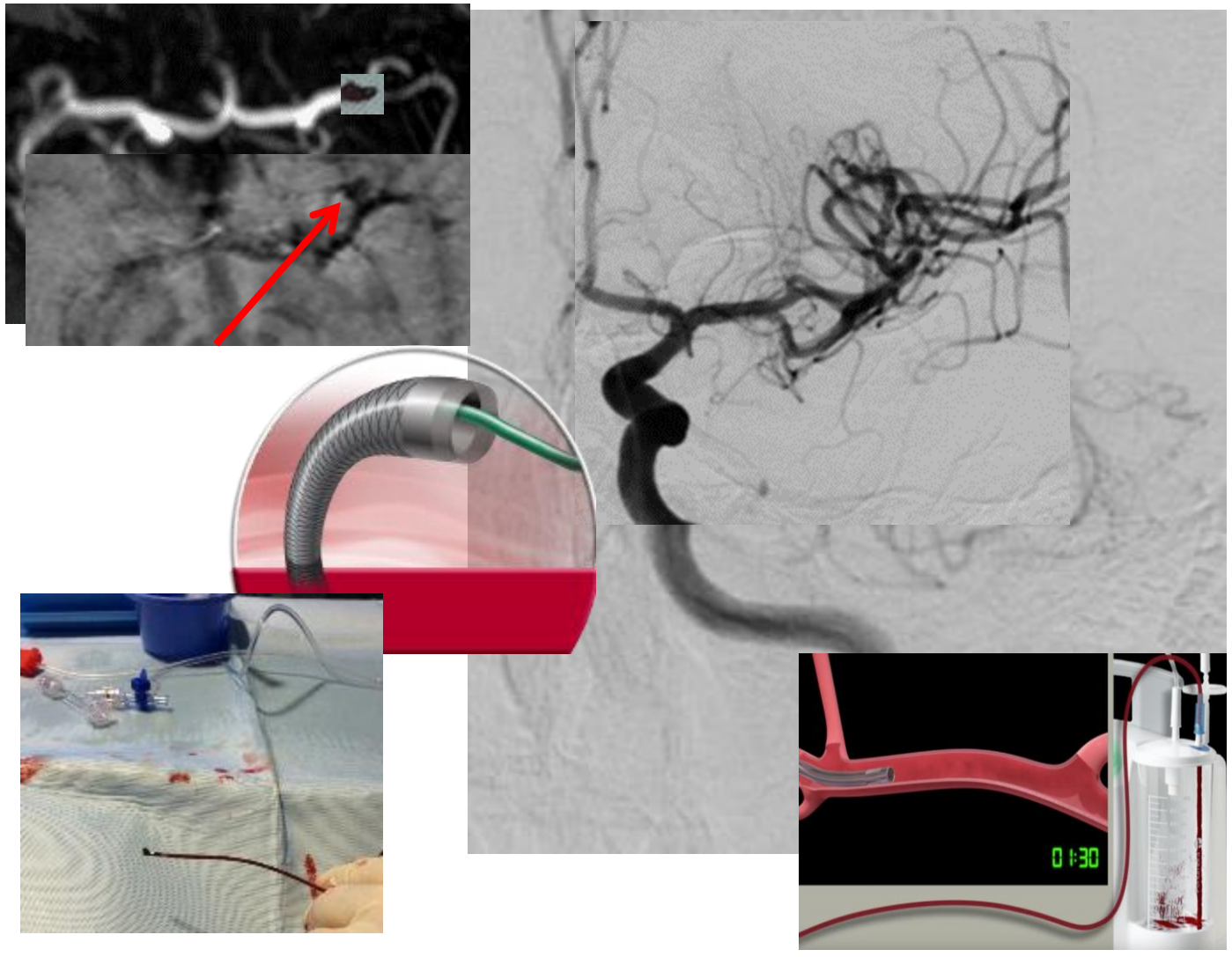
Rates of recanalization at 2 hours according to occlusion site

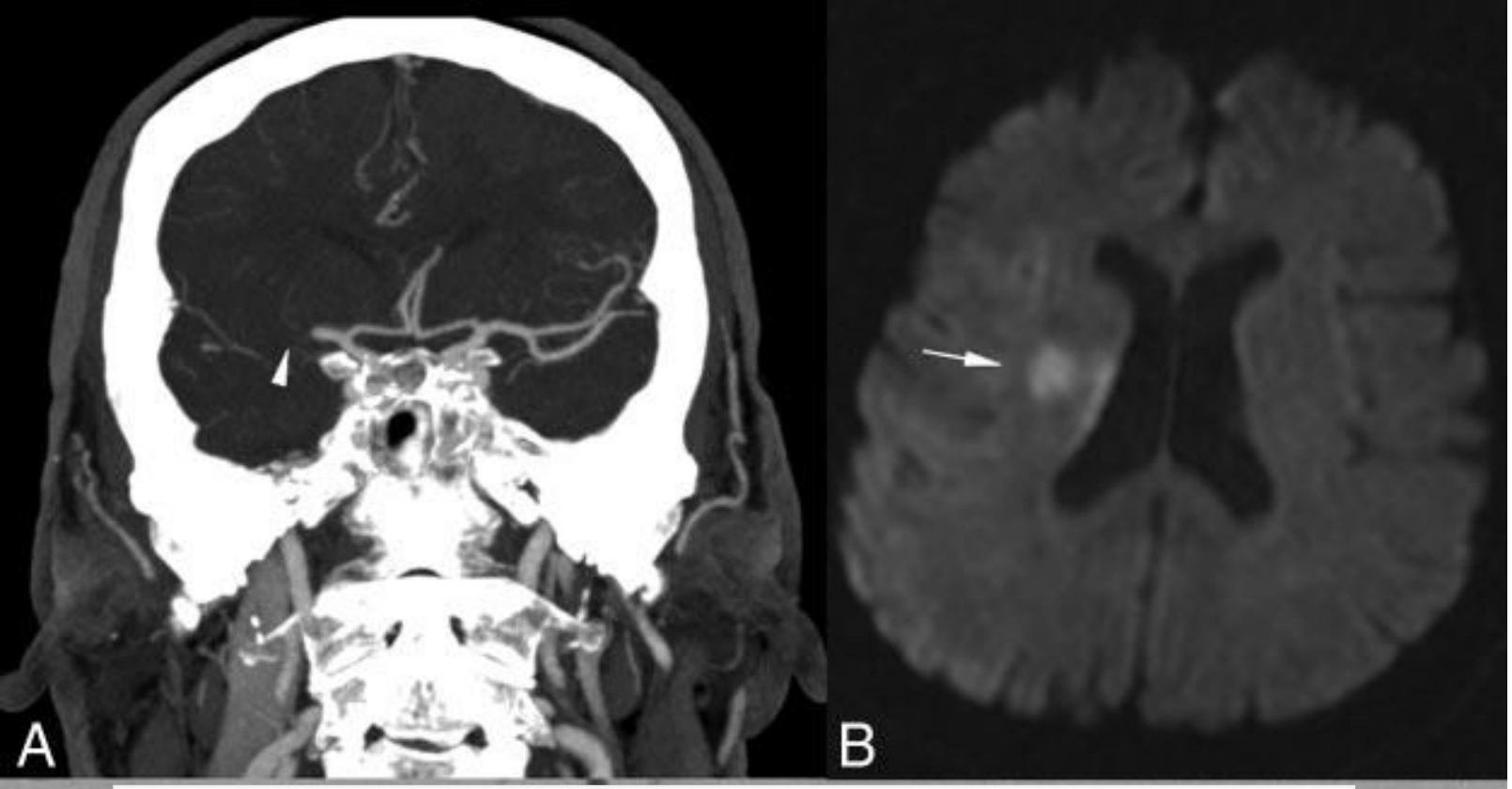


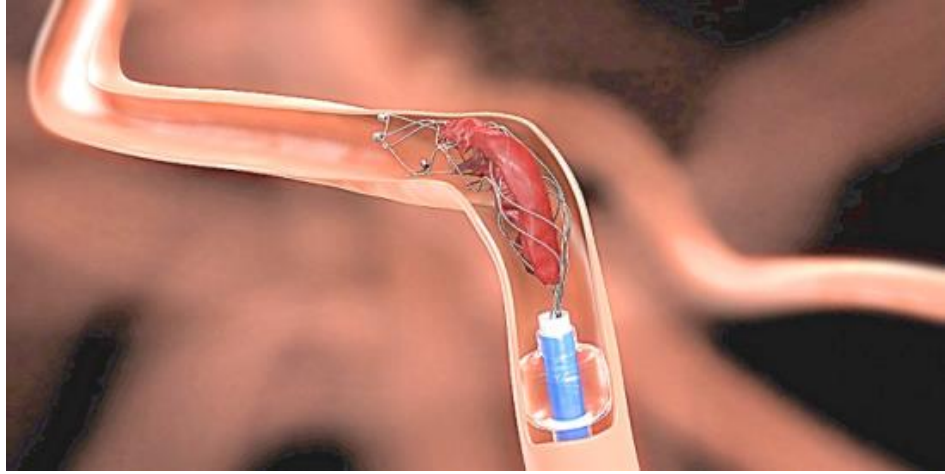
Rubiera et al. Stroke 2006;37:2301-5 – Saqqur et al. Stroke 2007;38:948-54 – Zangerle et al. Neurology 2007;68:39-44.
Mendonça et al. Stroke 2012;43:417-21 – Kimura et al. Stroke 2011;42:3150-5 – Alexandrov. J Int Med 2010;267:209-19.

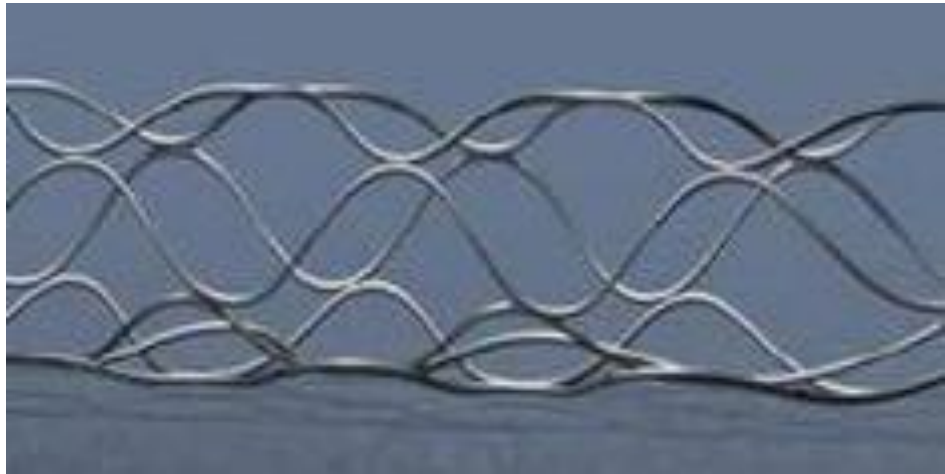
Autre traitement ?

TM


0:30



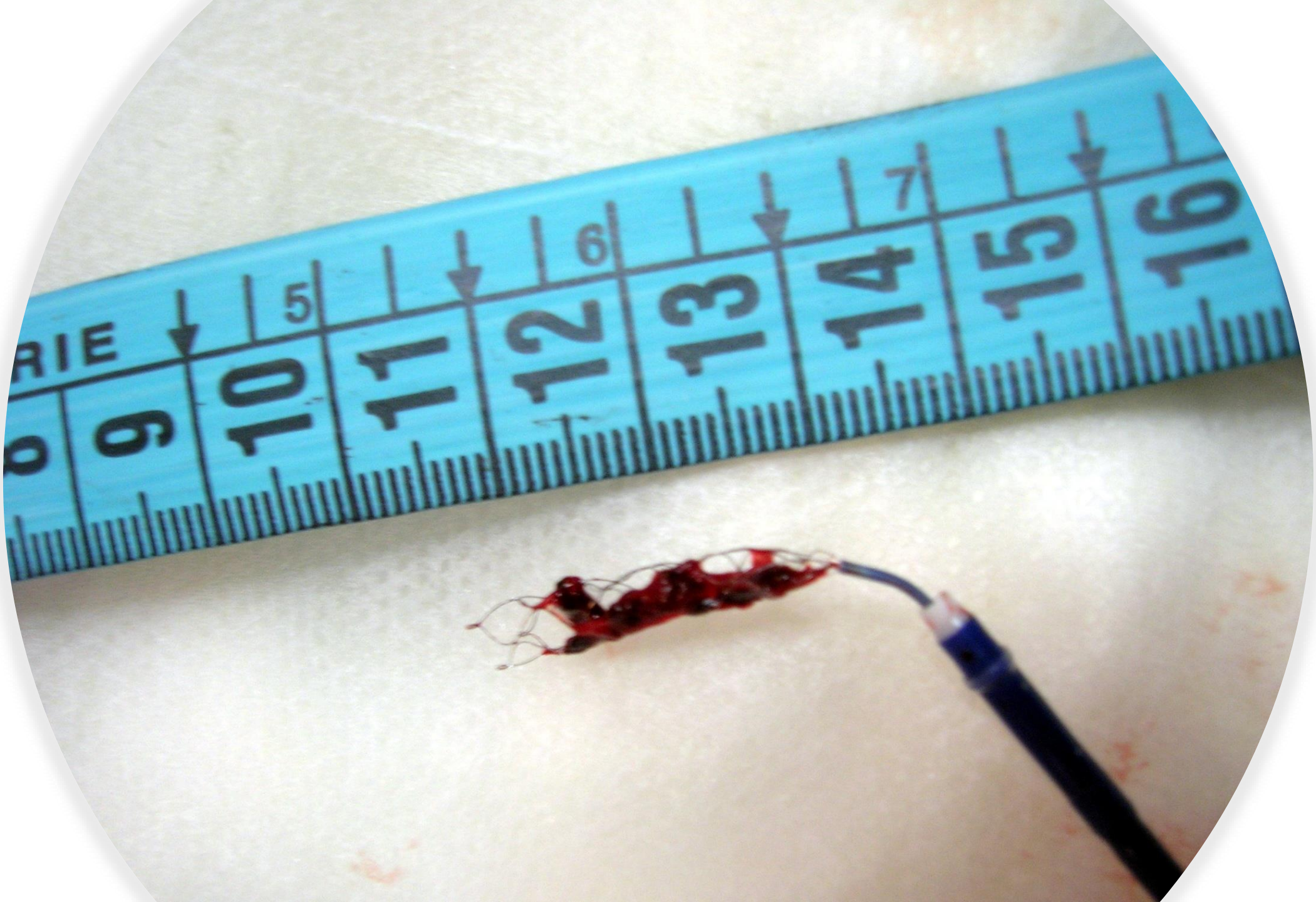




- TM


Carotide Interne Droite


Carotide Interne Droite


RIE
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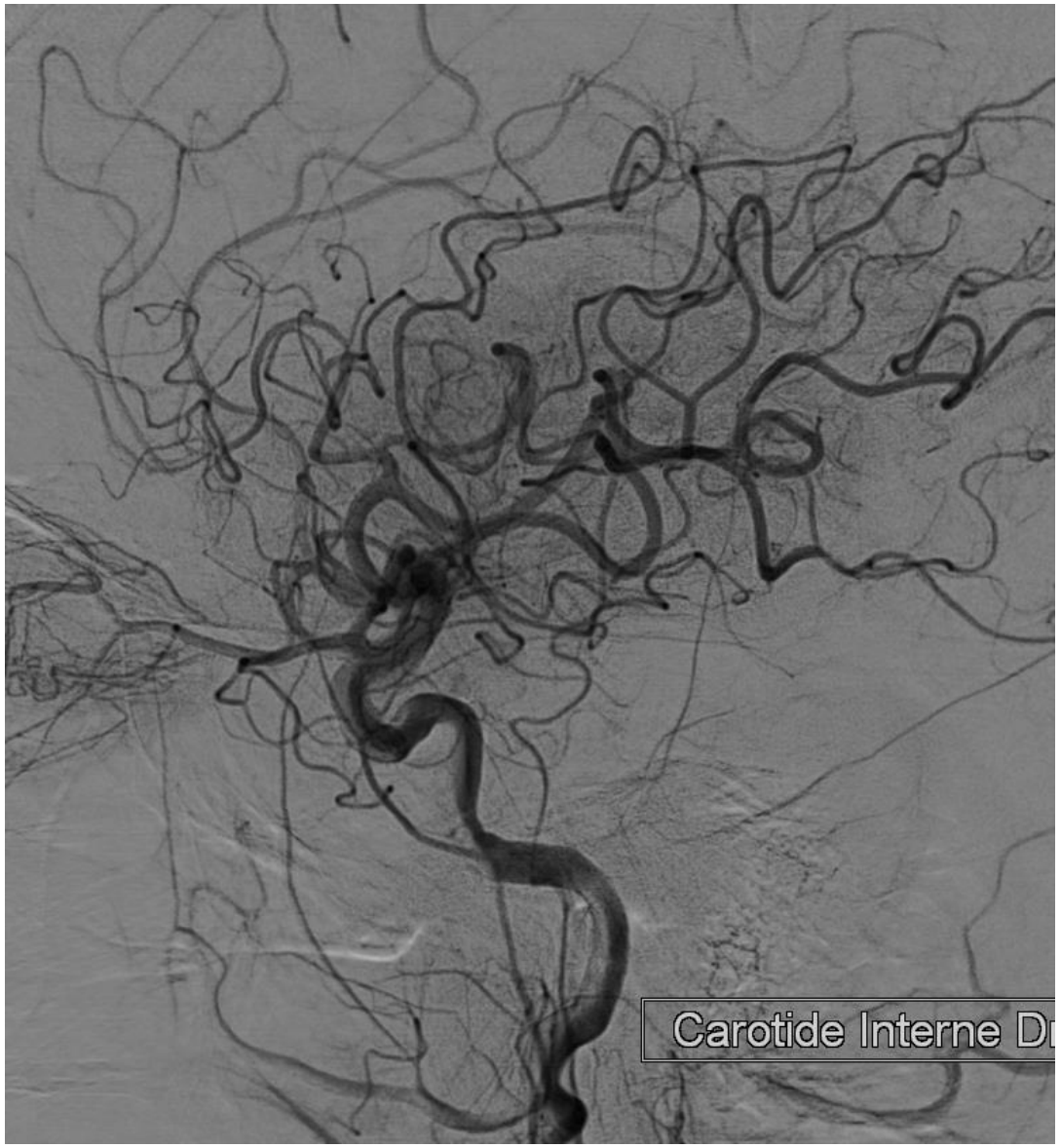
16

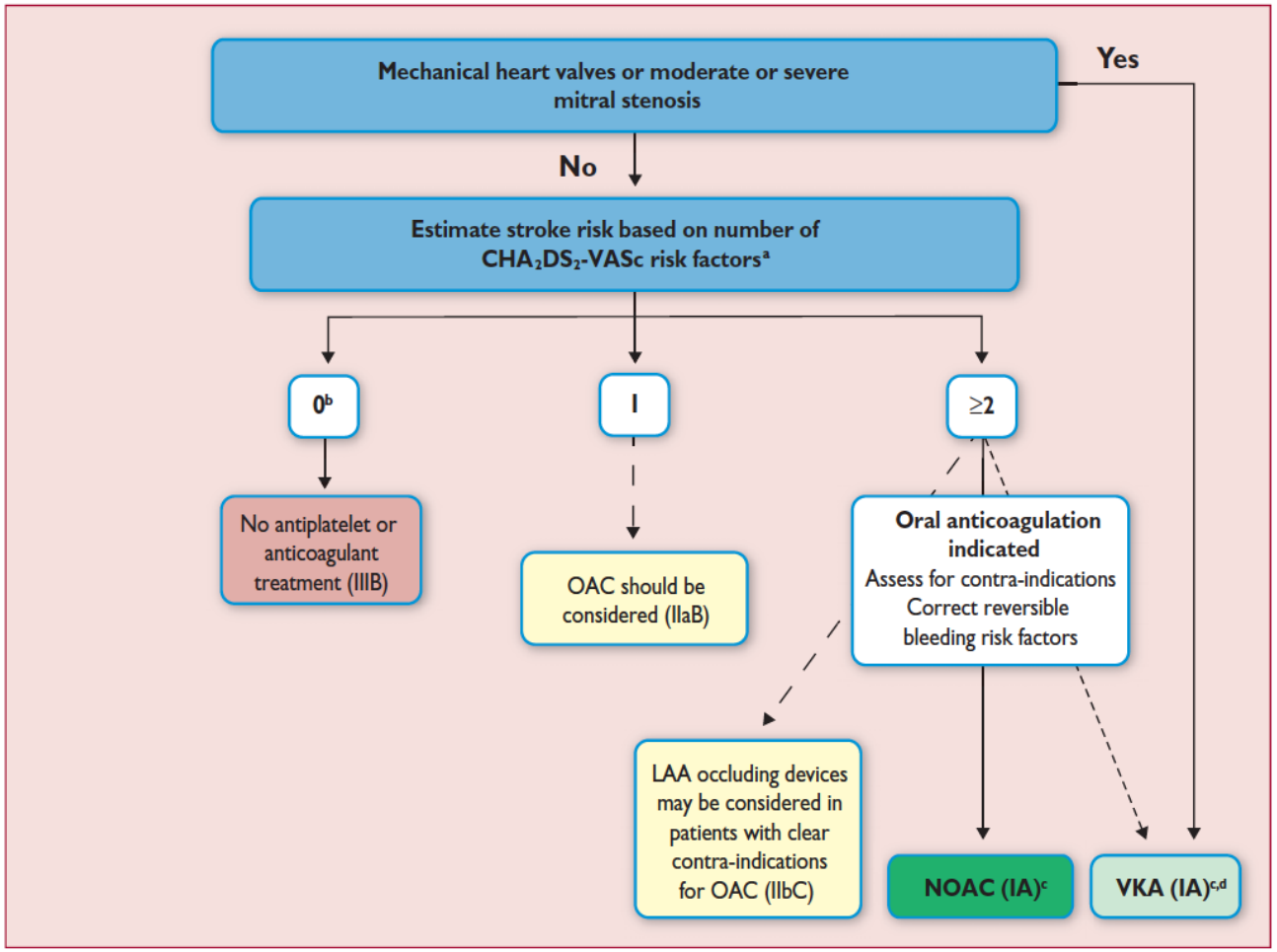

Carotide Int


Carotide Interne D

Si INR >2

Conduite à tenir ?

Prévention secondaire ?

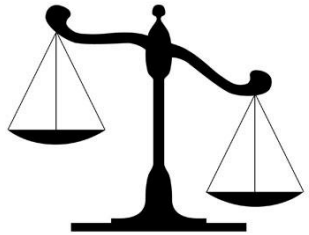


AF = atrial fibrillation; LAA = left atrial appendage; NOAC = non-vitamin K antagonist oral anticoagulant; OAC = oral anticoagulation; VKA = vitamin K antagonist.
[a]Congestive heart failure, Hypertension, Age ≥75 years (2 points), Diabetes, prior Sstroke/TIA/embolus (2 points), Vascular disease, age 65–74 years, female Sex.
[b]Includes women without other stroke risk factors.
[c]IIaB for women with only one additional stroke risk factor.
[d]IB for patients with mechanical heart valves or mitral stenosis.

http://eurheartj.oxfordjournals.org/

Quand débuter un traitement anticoagulant après un AVC ischémique lié à la FA?

Récidive précoce d'infarctus cérébral



Transformation hémorragique symptomatique

Le délai optimal de mise en route d'un traitement anticoagulant après un infarctus cérébral lié à la FA est incertain.

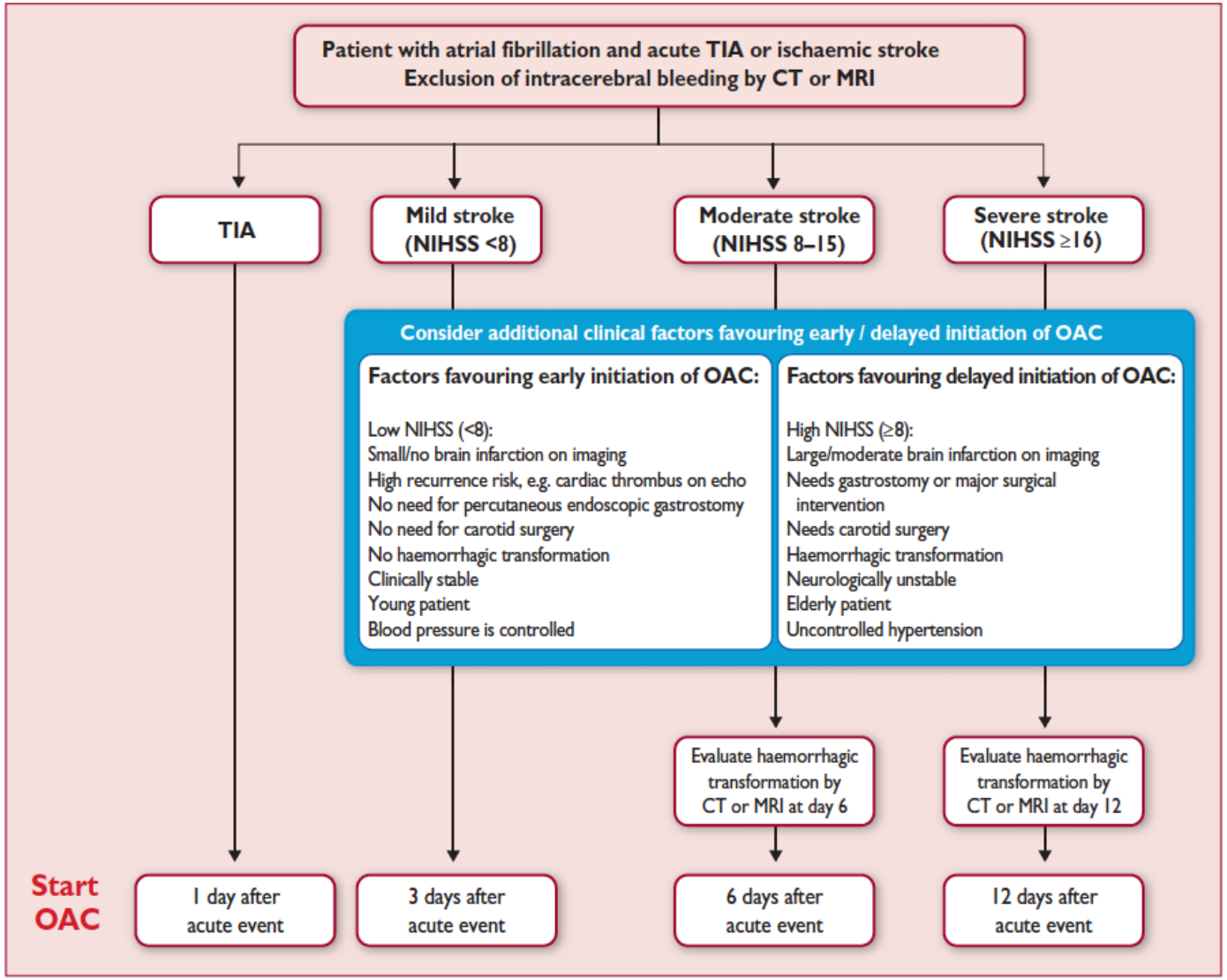
Une méta-analyse de 7 ECR évaluant traitement anticoagulant (par héparine non fractionnée, HBPM ou héparinoides) versus aspirine ou placebo, instaurés dans les 48 heures d'un infarctus cardio-embolique (principalement FA) n'a pas montré de bénéfice du traitement anticoagulant en termes de récidive d'AVC (AVC ischémiques et hémorragiques confondus) ou ou décès ou handicap (Paciaroni et al, Stroke 2007;38:423).

La décision est à prendre au cas par cas en mettant en balance le risque de récidive précoce d'infarctus cérébral en l'absence de traitement anticoagulant à celui d'aggravation par les anticoagulants d'une transformation hémorragique spontanée de l'infarctus cérébral.

La majorité des transformations hémorragiques spontanées surviennent dans la première semaine et particulièrement dans les 4 premiers jours (Lodder et al, Stroke 1988;19:1482).

Le risque de transformation hémorragique est plus important en cas d'infarctus de grande taille et d'hypertension artérielle non contrôlée.

Il faut vérifier l'absence de transformation hémorragique significative avant de mettre en route un traitement anticoagulant.



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

http://eurheartj.oxfordjournals.org/

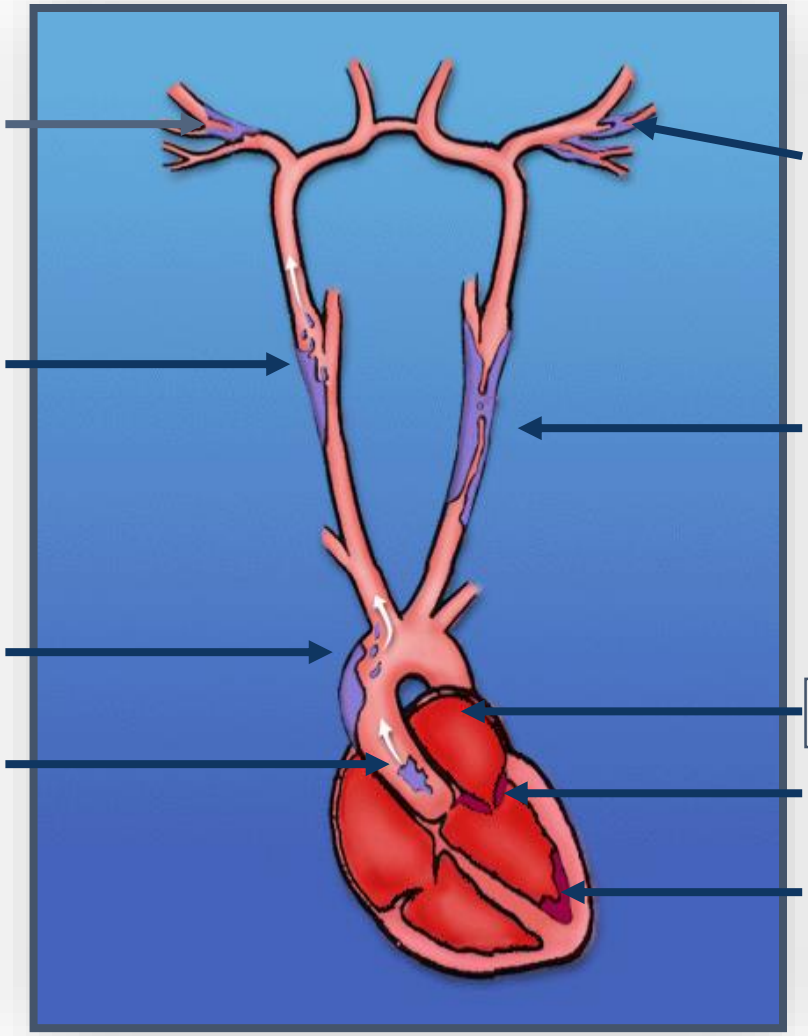
Evolution

- AVK , INR entre 2-3
- CMT droite et déficit G

Que faites vous ?

- Vérifier INR
- Augmenter les doses des AVK
- Mettre sous héparine
- Passer aux AOD
- Ajouter de l’aspirine
- Appeler un ami

Causes des AIC



Atherosclérose Intracrânienne

Plaque carotide avec embolie

Plaque de la crosse de l'aorte

Embolie cardiaque

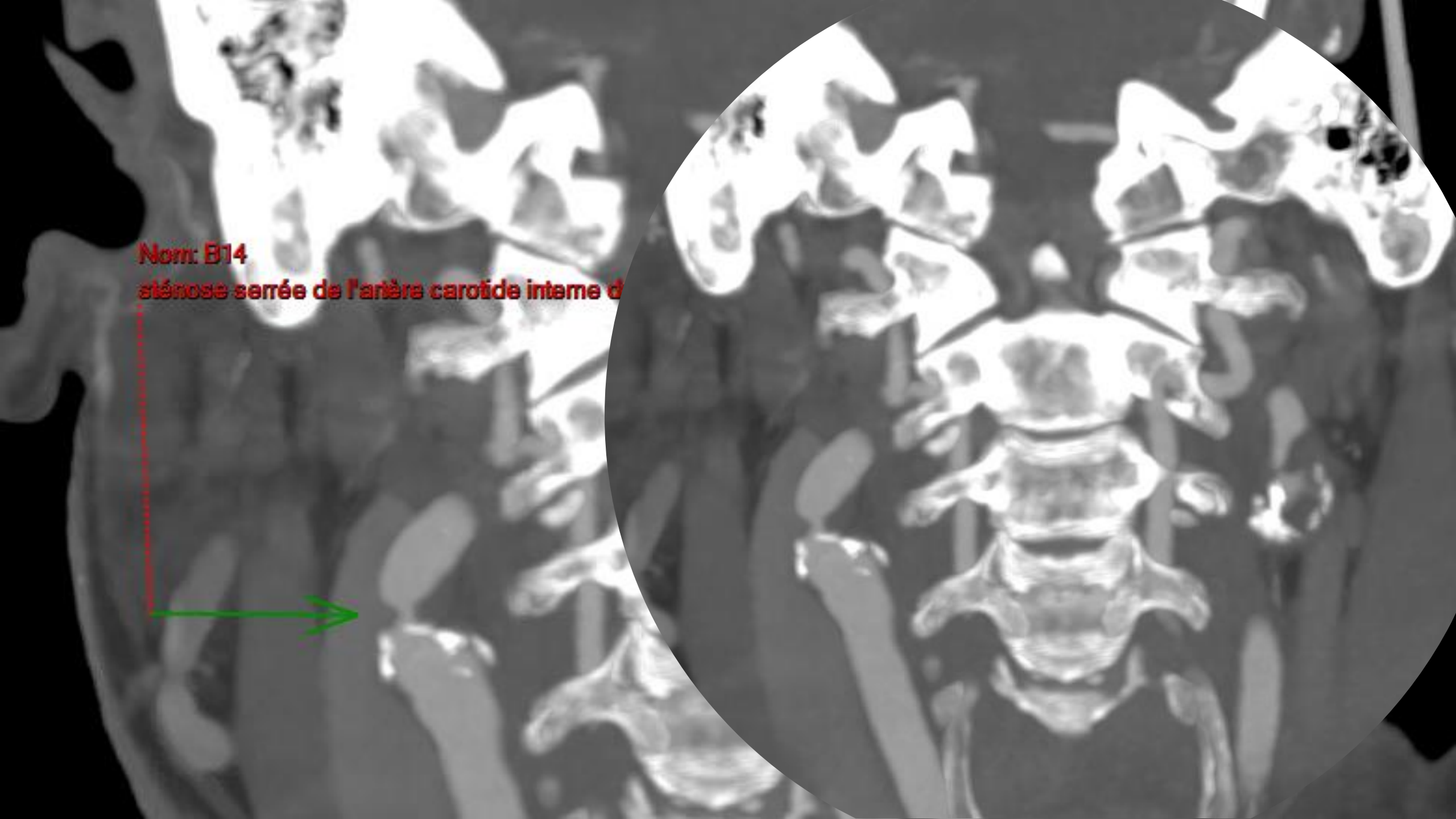
Maladie des artères perforantes

Sténose carotide

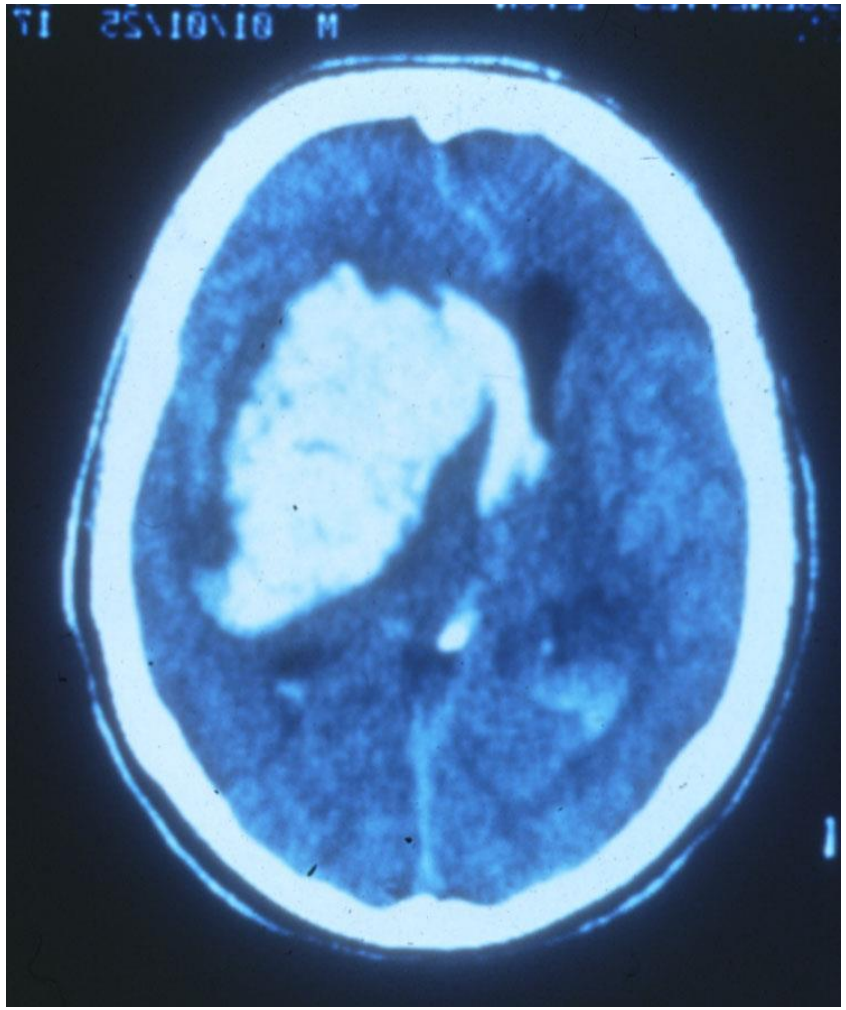
Fibrillation Atriale

Maladie Valvulaire

Thrombus du Ventricule gauche

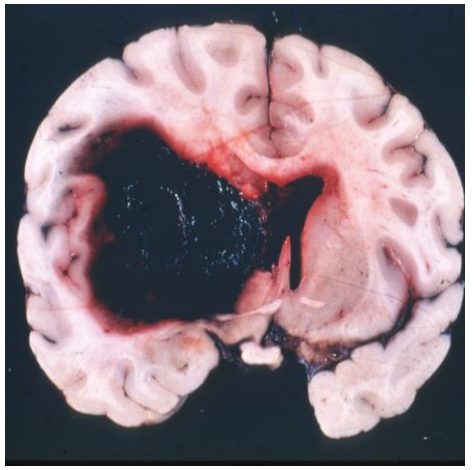

Nom: B14
sténose serrée de l'artère carotide interne d

INR à 3



Conduite à tenir ?
Prévention secondaire ?

HIC sous AC

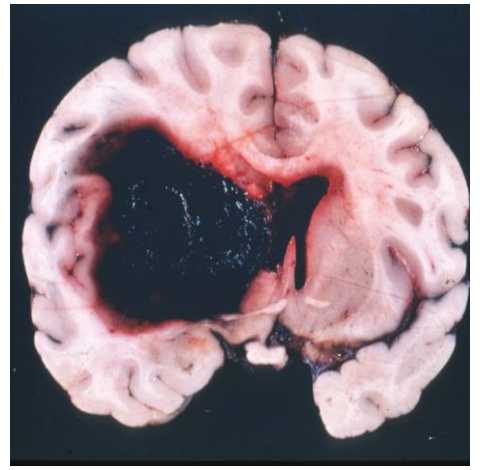


- 2 essais randomisés 2021 Lancet Neurol, ACO vs AAP
  - So Start (anglaise)
  - Apache-AF (hollandaise)
  - Pas de différence significative

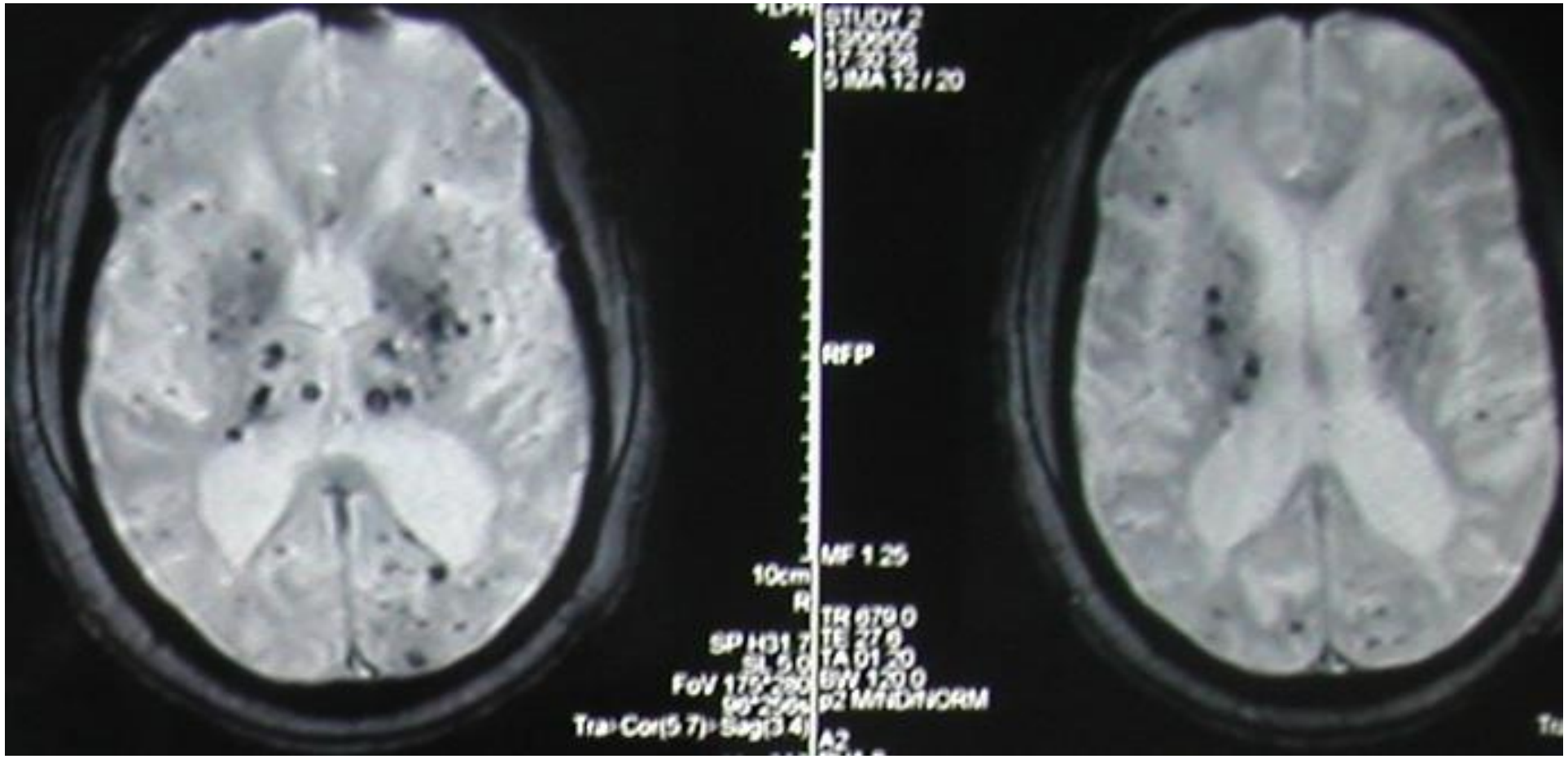
- FLAAC

- A3ICH
  - 3 bras
  - FAG/AAP/AOD

HIC sous AC



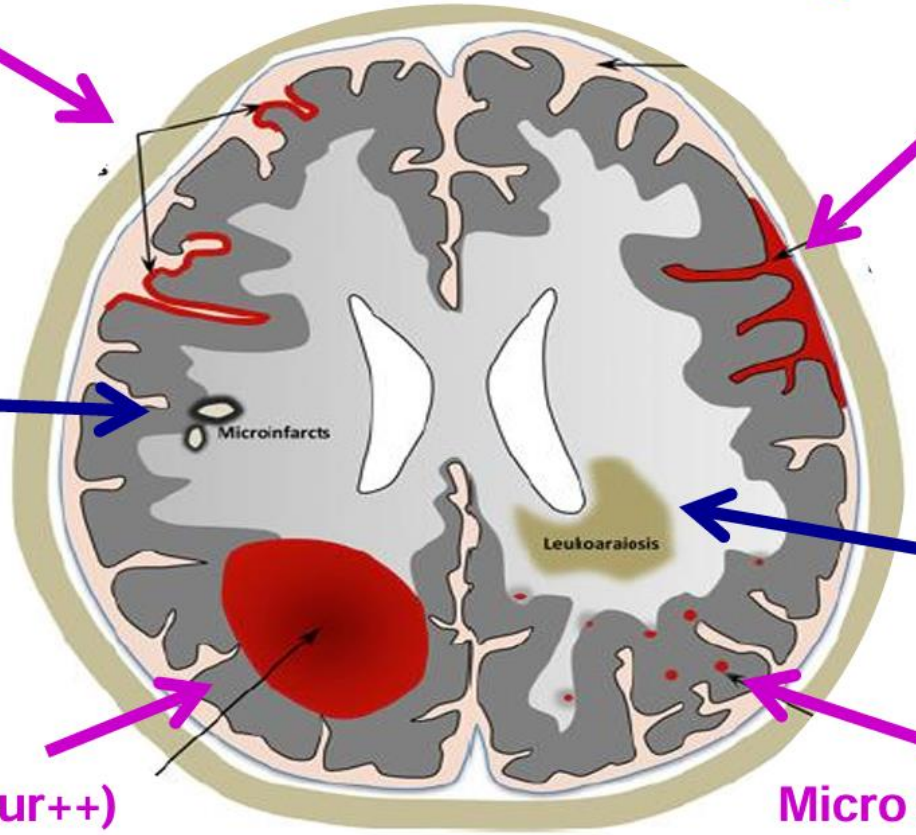
Microbleed/ HTA
IRM : SWI >T2*



AAC : angiopathie amyloide cérébrale

Hémosidérose corticale superficielle

Hémorragie sous-arachnoïdienne aigüe



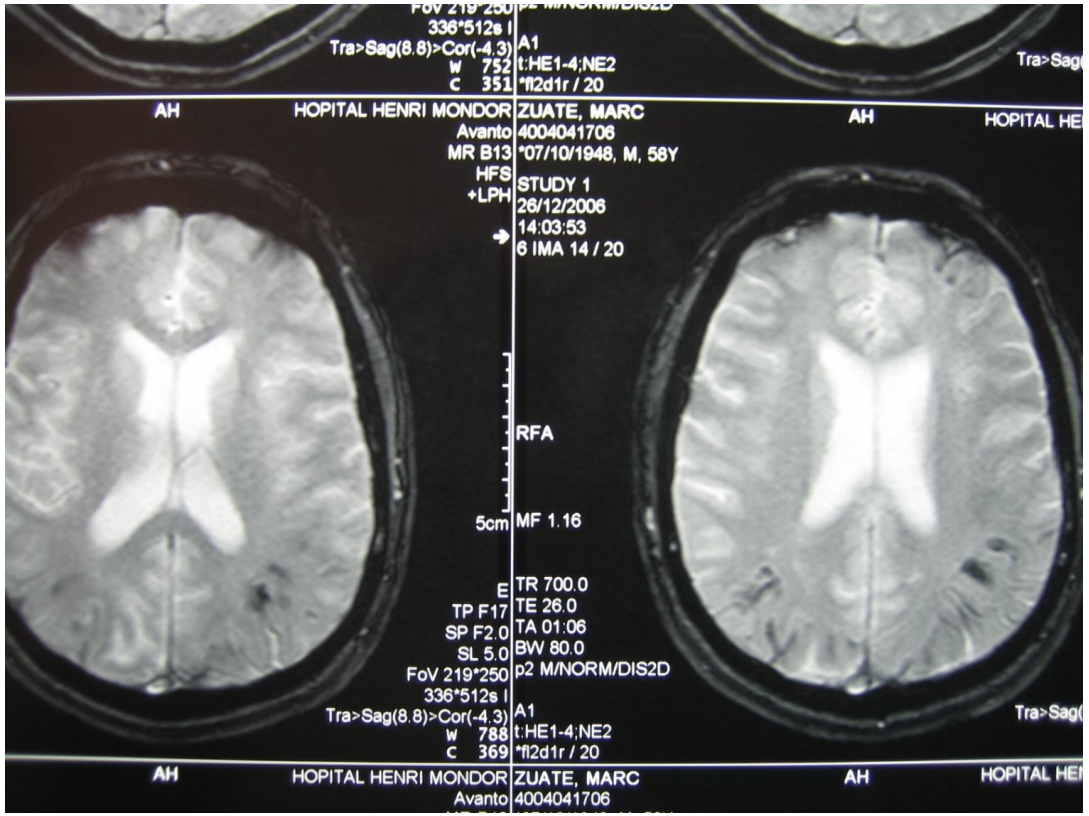
Micro infarctus

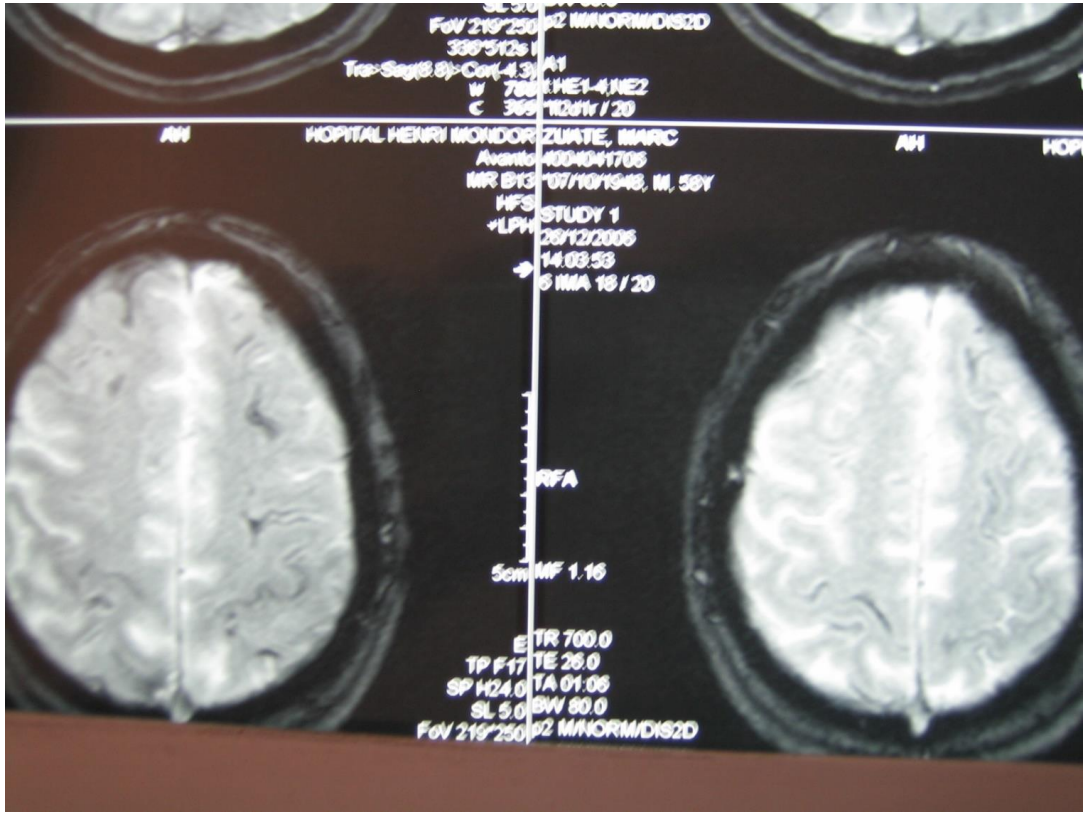
Leucopathie
- Prédominance postérieure
- Pattern « multi spot »

Hématome lobaire (postérieur++)

Micro saignements strictement lobaires

AAC


336*512s I
Tra>Sag(8.8)>Cor(-4.3)
W 752
C 351
A1
t:HE1-4;NE2
*fl2d1r / 20
AH
HOPITAL HENRI MONDOR
ZUATE, MARC
Avanto
4004041706
MR B13
*07/10/1948, M, 58Y
HFS
+LPH
STUDY 1
26/12/2006
14:03:53
6 IMA 14 / 20
RFA
5cm
MF 1.16
E
TR 700.0
TP F17
TE 26.0
SP F2.0
TA 01:06
SL 5.0
BW 80.0
FoV 219*250
p2 M/NORM/DIS2D
336*512s I
Tra>Sag(8.8)>Cor(-4.3)
W 788
C 369
A1
t:HE1-4;NE2
*fl2d1r / 20
AH
HOPITAL HENRI MONDOR
ZUATE, MARC
Avanto
4004041706
AH



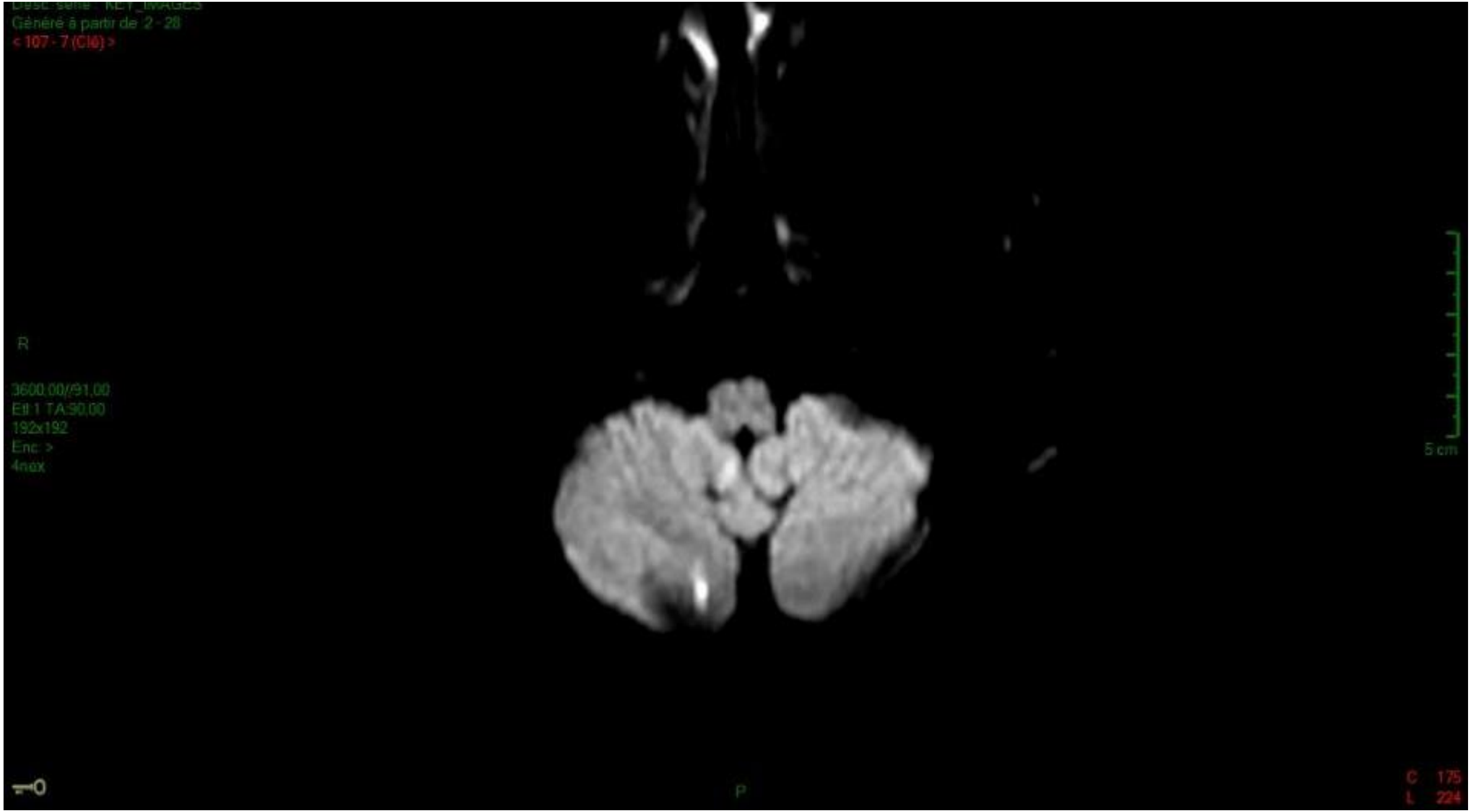
Cas Clinique M. A.

M. A.

- Homme de 74 ans, vit seul, autonome
- Antécédents :
  - Tabagisme sevré depuis < 1 mois
  - HTA
  - Dyslipidémie
  - Sténoses carotidiennes bilatérales

Histoire de la maladie

- Syndrome coronnarien aigu ST+ troponine+ avec angioplastie et stent sur l'inter ventriculaire antérieure
- +J14 :
  - Vertige et diplopie
  - hémiparésie G
  - Scanner cérébral : normal
- Que faites-vous ?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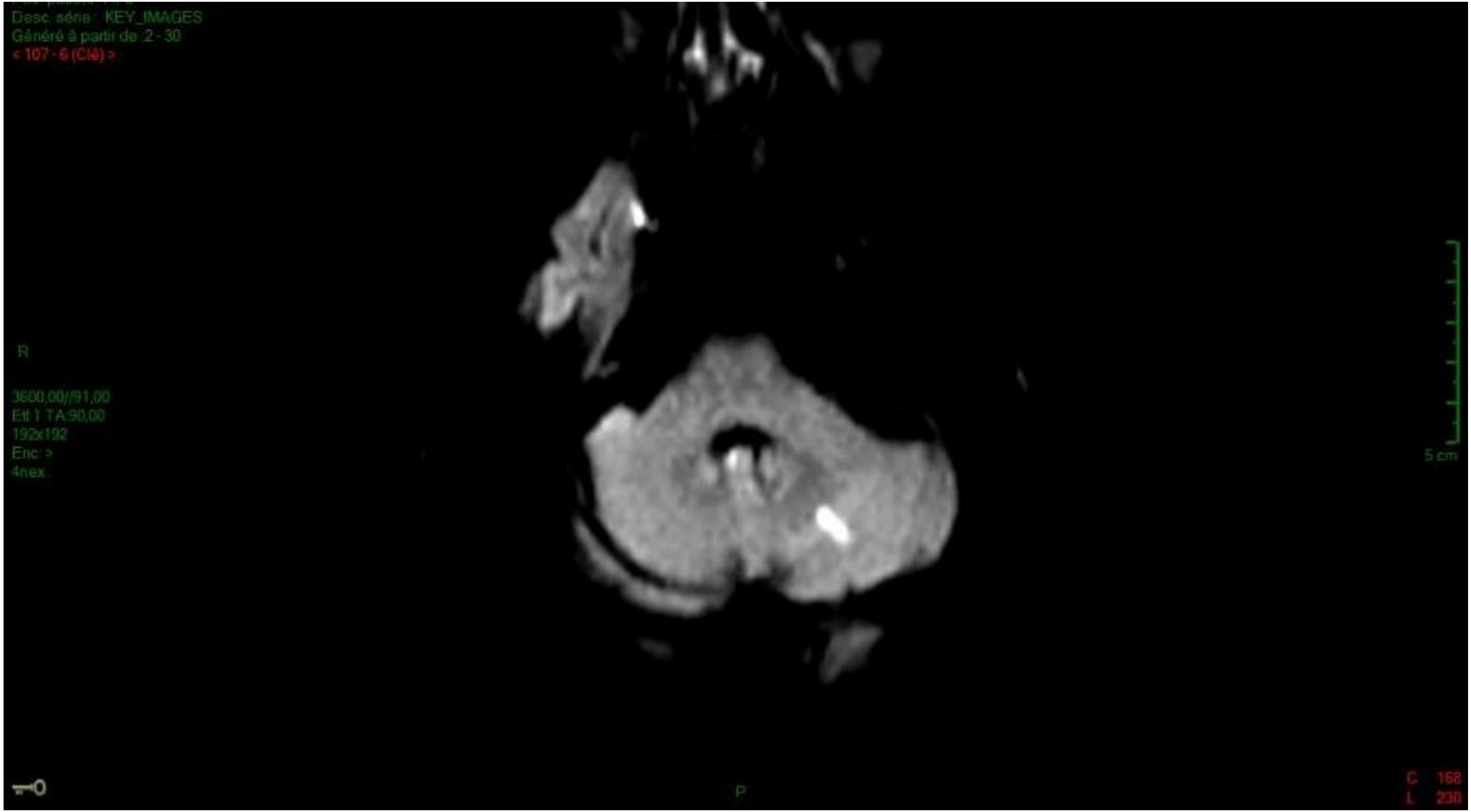
3600.00/91.00
Eff 1 TA:90.00
192x192
Enc >
4nex
5 cm
P
C 175
L 224


Desc. série : KEY_IMAGES
Généré à partir de :2 - 30
< 107 - 6 (Clé) >
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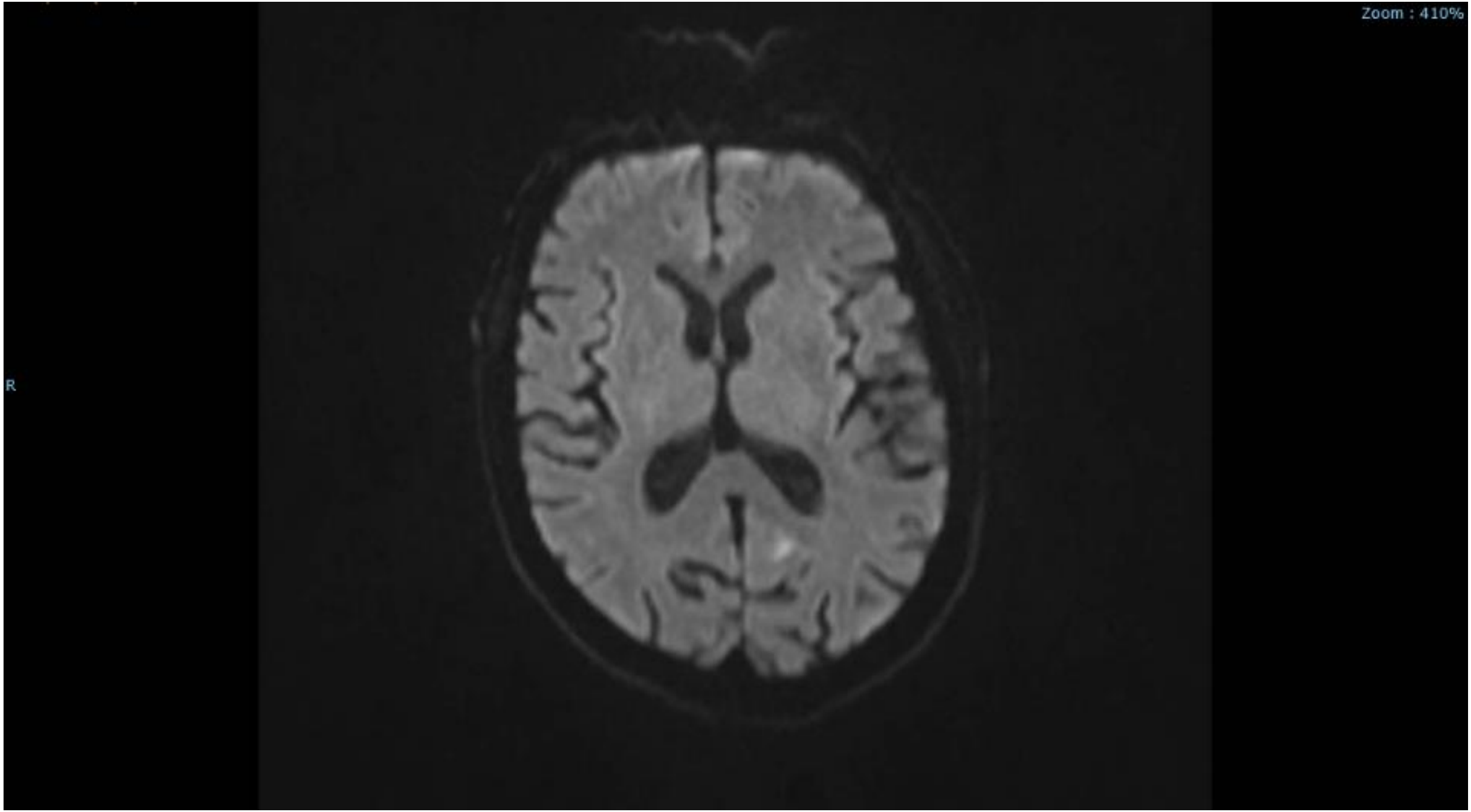
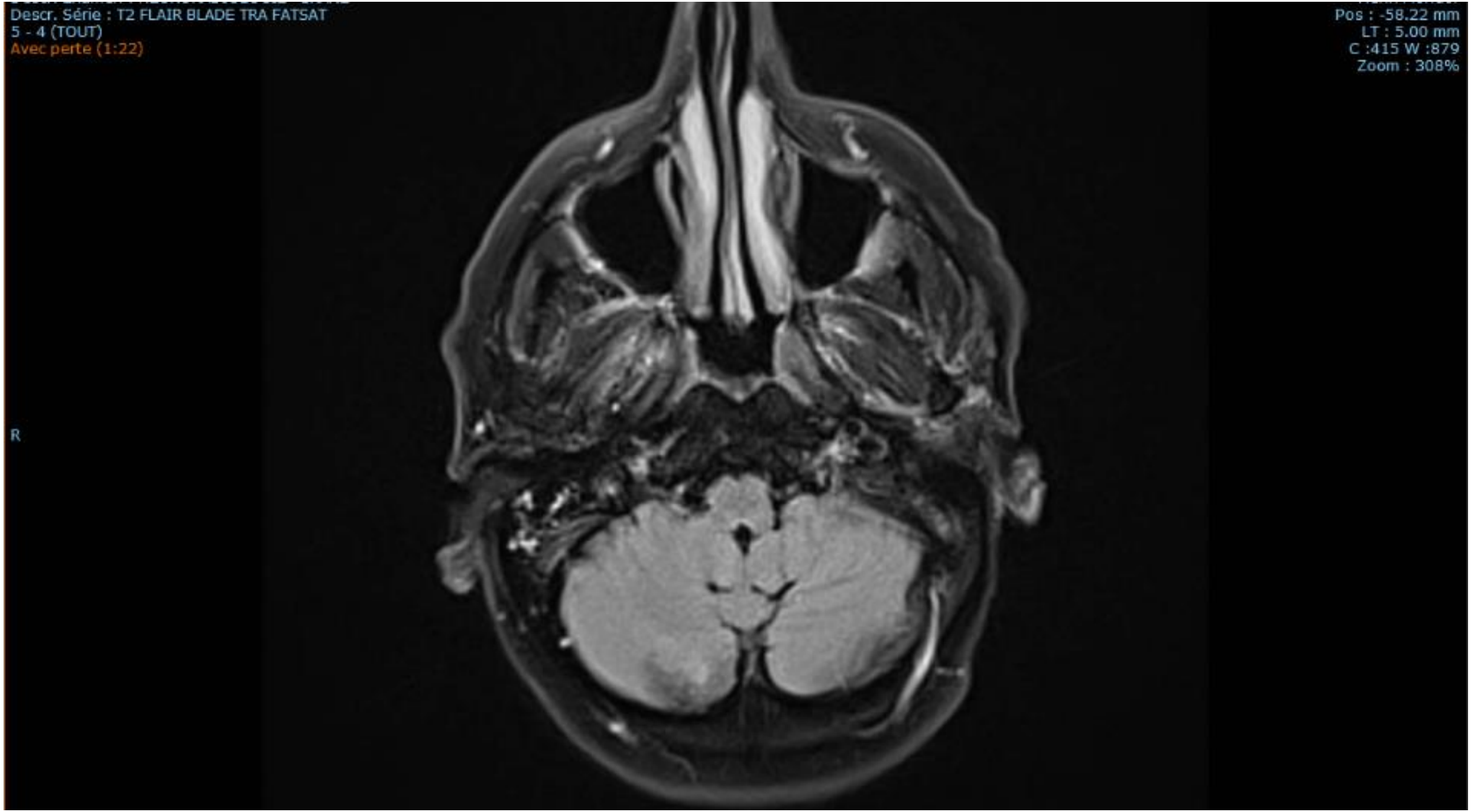
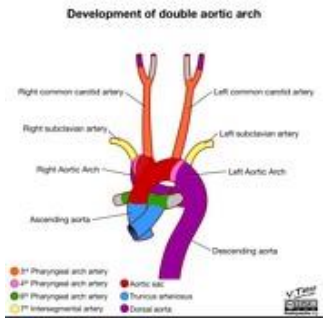
3600,00/91,00
Et| 1 TA:90,00
192x192
Enc: >
4nex
5 cm
P
C 168
L 230


Zoom : 410%
R


Descr. Série : T2 FLAIR BLADE TRA FATSAT
5 - 4 (TOUT)
Avec perte (1:22)
Pos : -58.22 mm
LT : 5.00 mm
C :415 W :879
Zoom : 308%
R

Etiologies ?

Etiologies ?



artères

Athérome

Dissection post coronarographie.

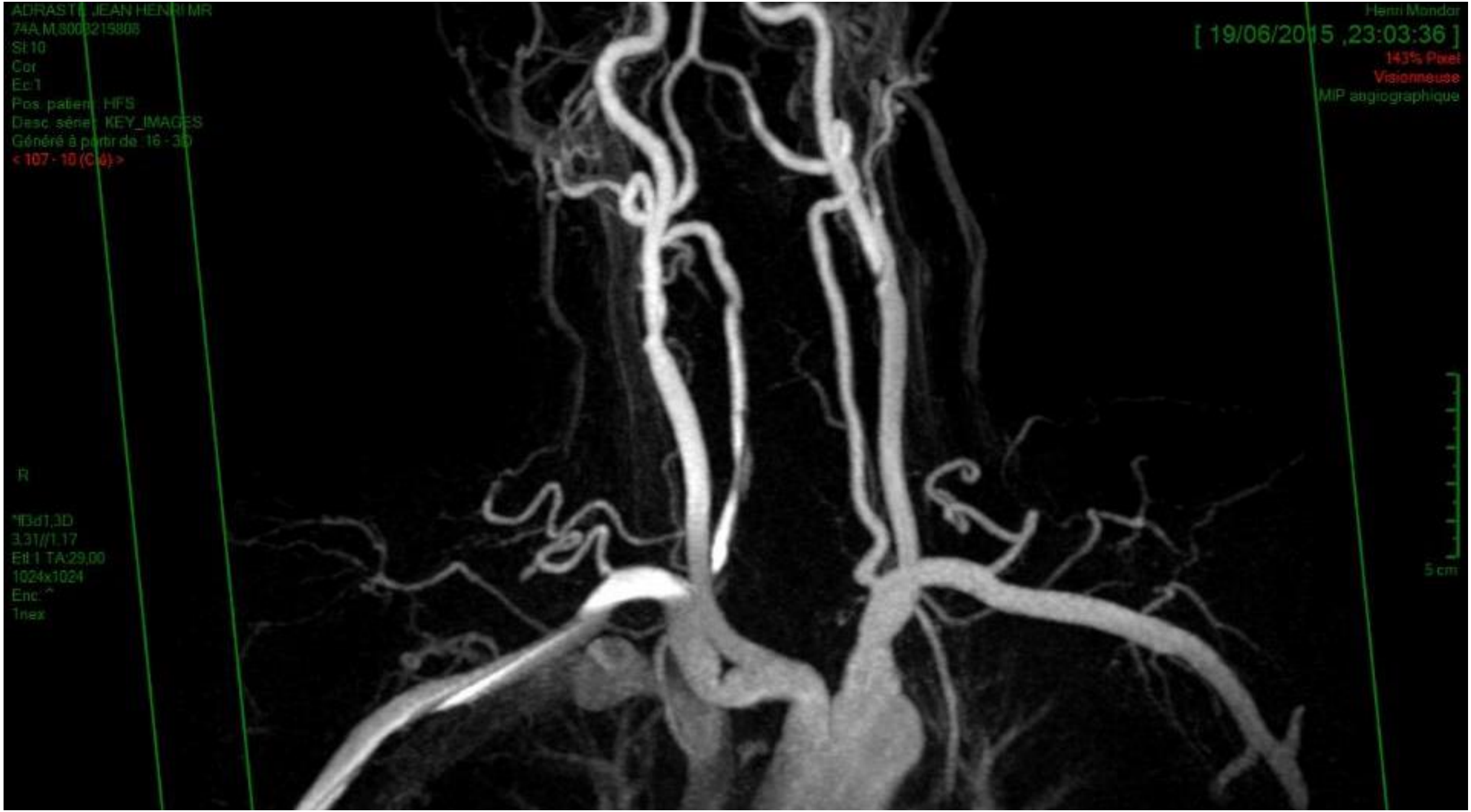
Cardiaque :

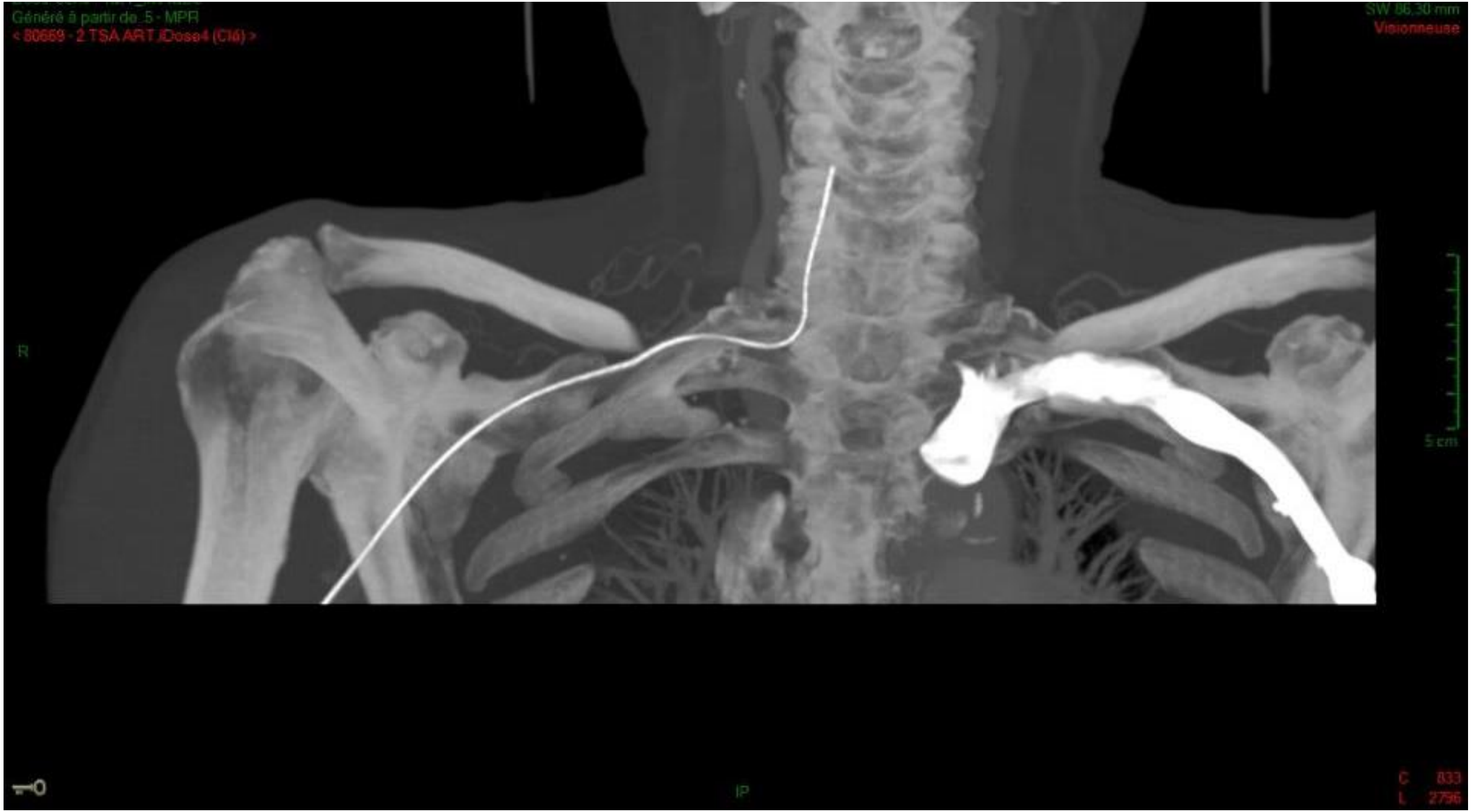
Nouvel IDM

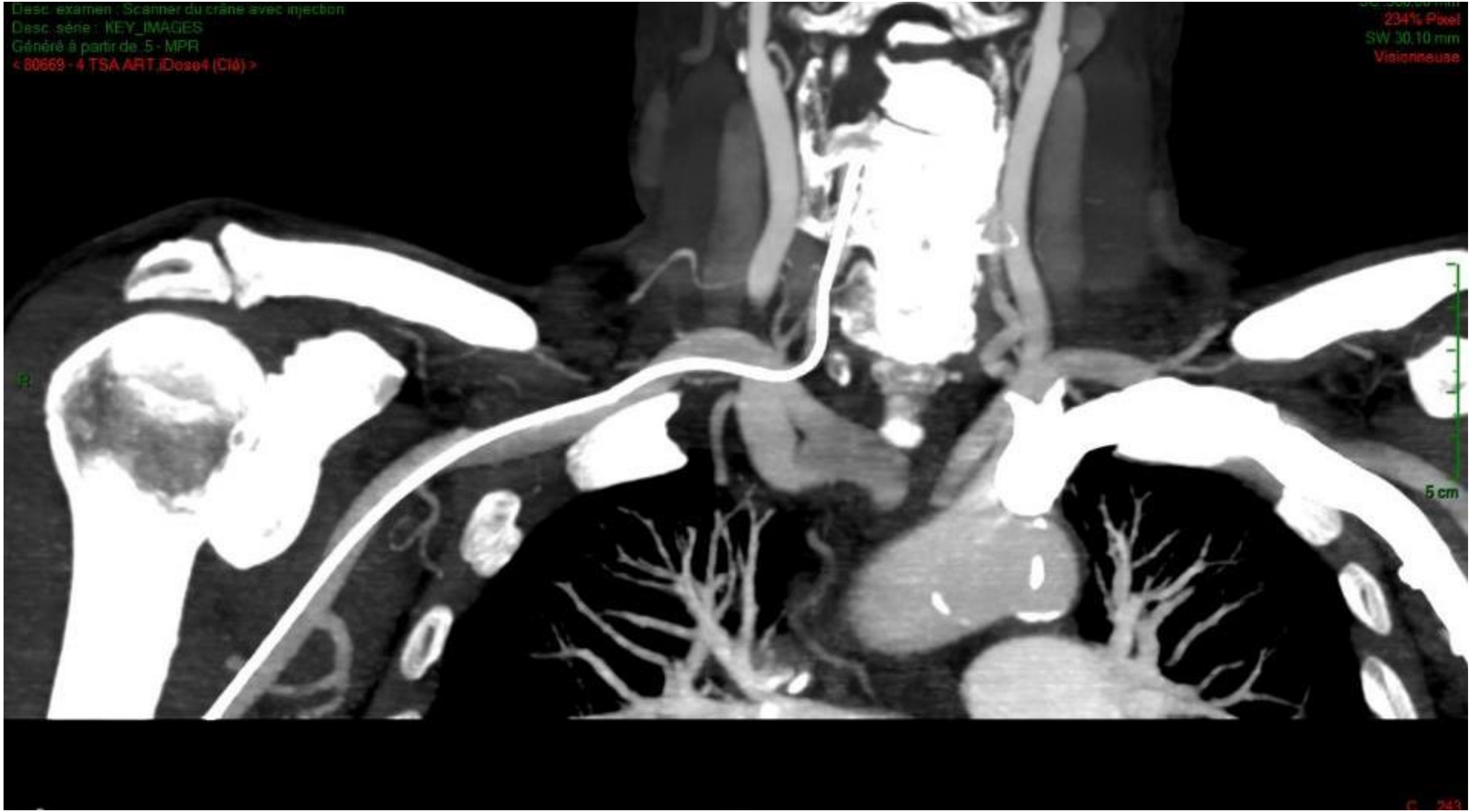
Insuffisance cardiaque

Anévrysme apex

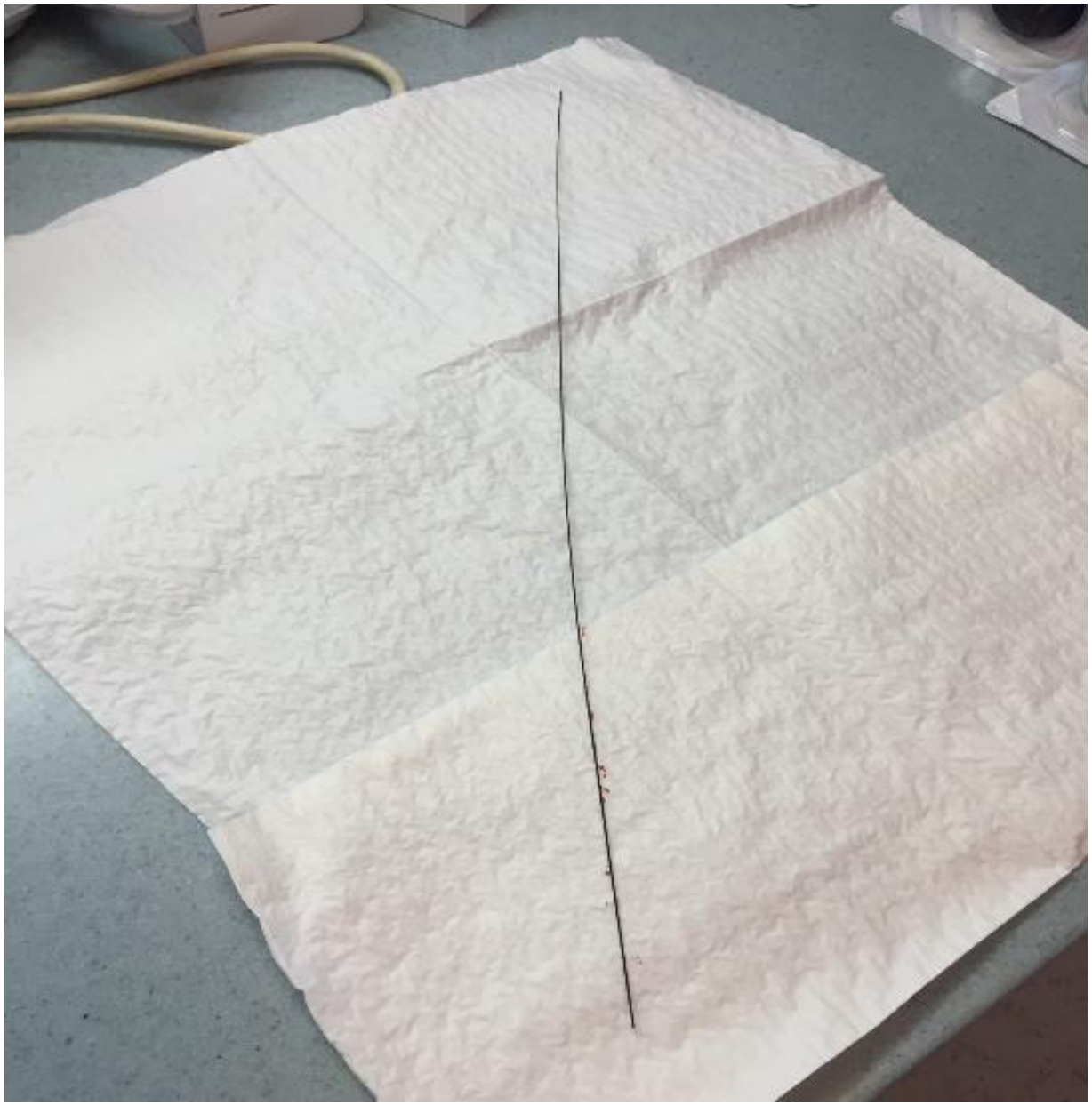
AC/FA


ADRAST JEAN HENRI MR
74A M,80
Sl 10
Cor
Ec 1
Pos patient HFS
Desc série KEY_IMAGES
Généré à partir de 16 - 35
< 107 - 10 (C 0) >
Henri Mondor
[19/06/20 5 ,23:03:36]
143% Pixel
Visionneuse
MIP angiographique
R
*fl3d1,3D
3,31//1,17
Etl 1 TA:29,00
1024x1024
Enc. ^
1nex
5 cm

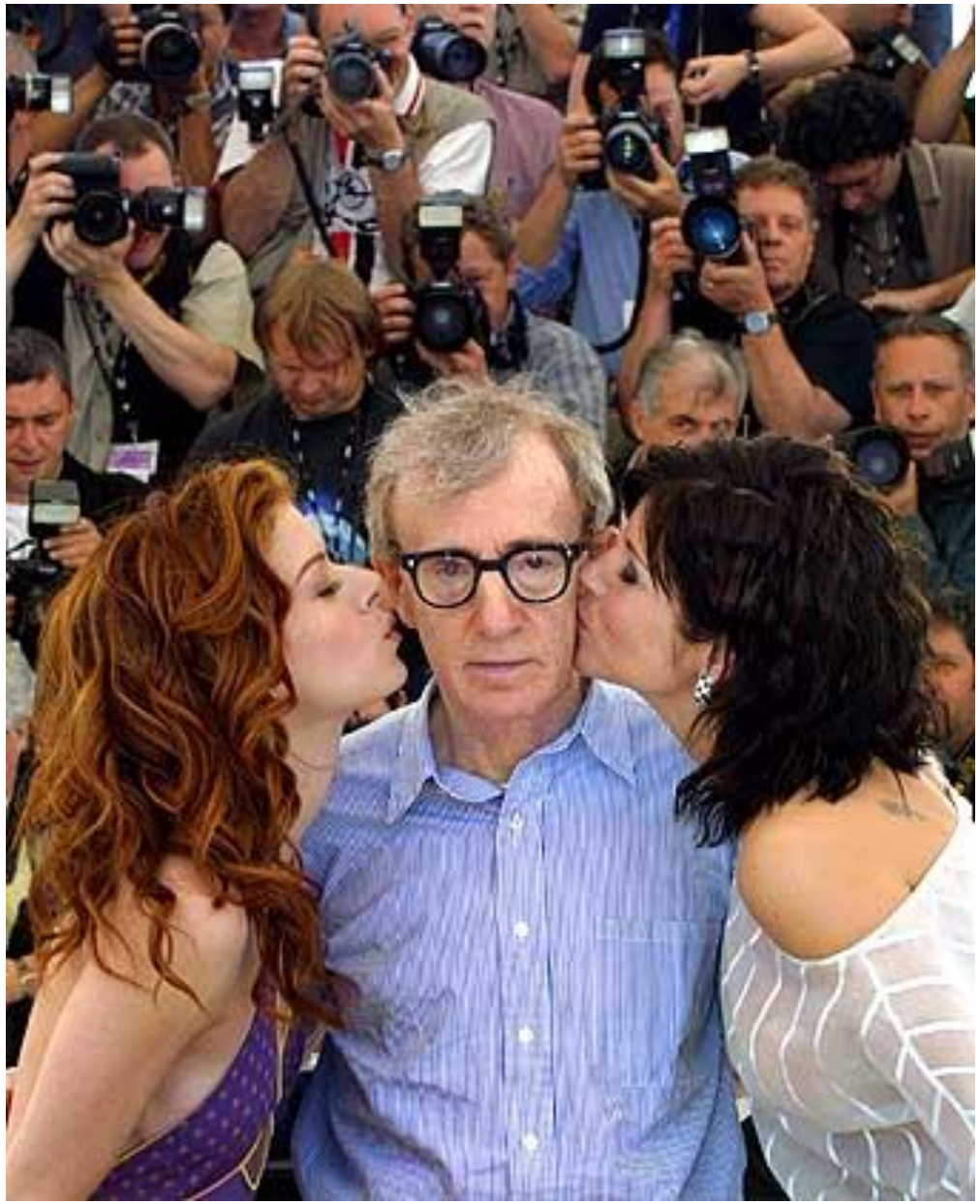

Généré à partir de :5 - MPR
< 80669 - 2 TSA ART JDose4 (CI6) >
SW 86.30 mm
Visionneuse
R
5 cm
IP
C 833
L 2756

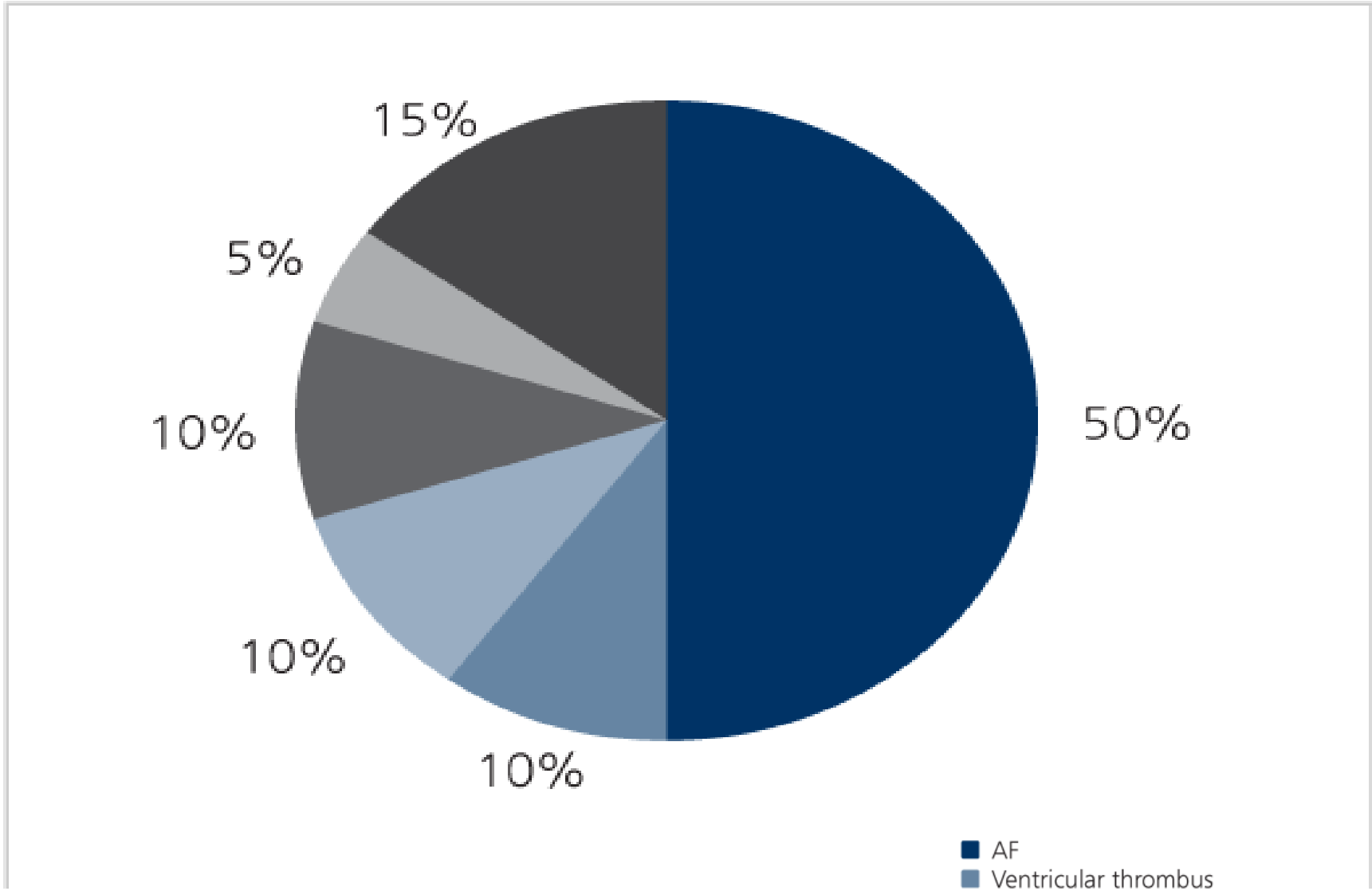

Desc. examen : Scanner du crâne avec injection
Desc. série : KEY_IMAGES
Généré à partir de .5 - MPR
< 80669 - 4 TSA ART iDose4 (Clé) >
234% Pixel
SW 30,10 mm
Visionneuse
R
5 cm

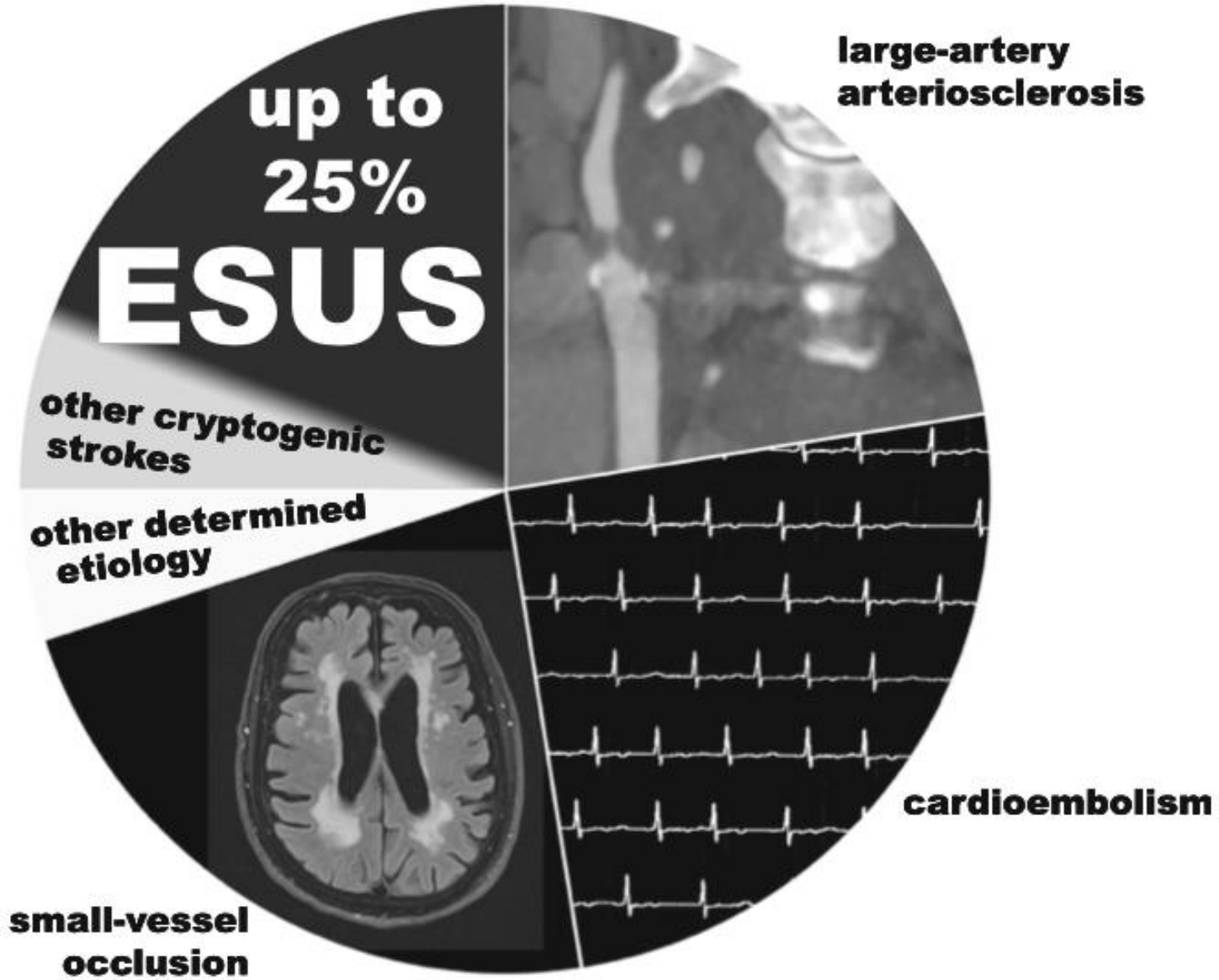
Traitement



“Don’t damage my brain it is my second favorite organ” *W.A.*




15%
5%
10%
10%
10%
50%
AF
Ventricular thrombus
Rheumatic heart disease
Acute myocardial infarction
Replacement valves
Other causes



Geisler et al., Drugs 2018

- Artériopathies non inflammatoires (dissection ++), inflammatoires ou infectieuses
- Affections hématologiques, coagulopathies
- Autres causes

- Toxicomanie, alcool
- Traumatisme
- Hypotension systémique
- Migraine
- Iatrogène (médicaments, cathétérisme)

- Embolie de matériel non thrombotique (ex: tumeur)
- Maladies métaboliques rares (ex: MELAS), pulmonaires (ex: Rendu-Osler) ...
- Etats pro-thrombotiques (CO, grossesse, cancers...)

- Nonatherosclerotic arteriopathies
  - Dissection
  - Vasculitis
  - Moya-moya
  - Sickle cell disease
  - Dolichoectasia
  - Migraine

- Vasospasm
  - After subarachnoid hemorrhage
  - Reversible vasoconstriction syndrome
- Fibromuscular dysplasia, stenosis ≥ 50%
- CADASIL
- Fabry Disease
- Susac Disease
- Hypercoagulable States
  - Arterial thrombosis
  - Antiphospholipid antibodies≥100 GPL units, or lupus anticoagulant
  - Hyperhomocysteinemia
  - Thrombocytosis with platelets ≥800,000
  - Disseminated intravascular coagulation
  - Malignancy

- 30%
- ETO +++
  - oreillette et auricule gauches
  - SIA
  - aorte thoracique

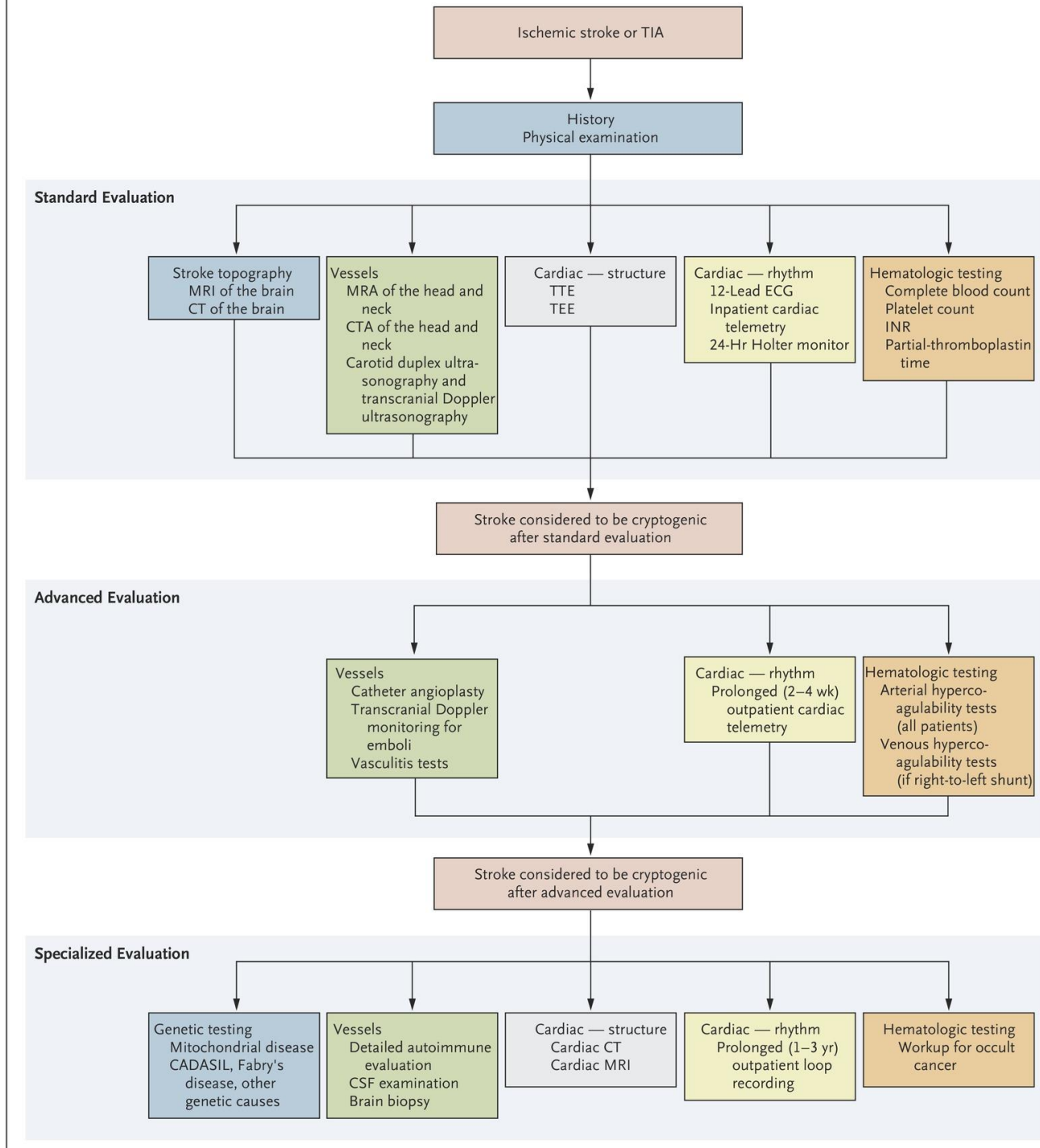
- 40 % en 1970
- 10-15 % actuellement dans les centres spécialisés

- Moins sévère
- Moins de mortalité
- Moins de récidive
  - Sous aspirine
    - 1,9 % /an la première année
    - 0,8% /an 2 à 4 ans

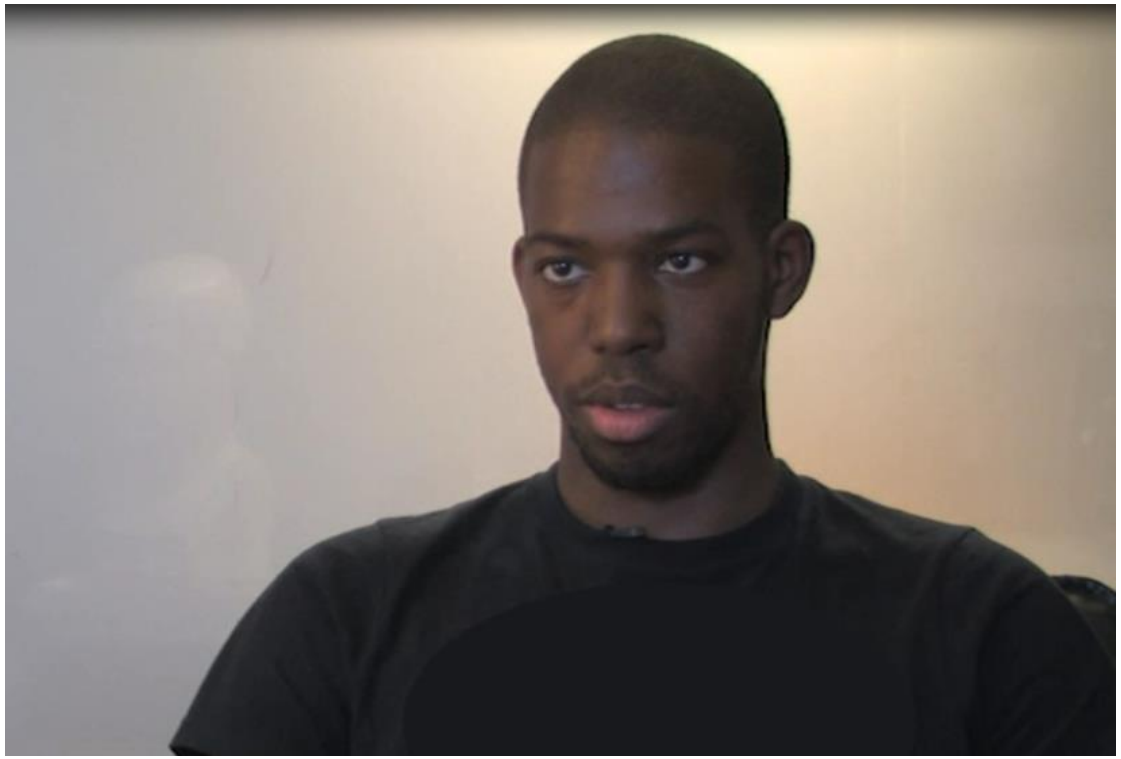
Table 1. Suggestive Findings on History and Physical Examination in Patients with Cryptogenic Stroke.*

Variable	Potential Clinical Implication
Historical feature	
Neck trauma or manipulation	Carotid or vertebral artery dissection
Migraine	Migrainous infarction or CADASIL
Intravenous drug use	Endocarditis, HIV infection, vasculitides, paradoxical emboli, or vasospasm
Dental procedure or systemic bacterial infection	Endocarditis, septic emboli, or coagulopathy
Airplane travel or Valsalva maneuver at stroke onset	Paradoxical embolism
Family history of early myocardial infarction or ischemic stroke	Genetic accelerated atherosclerosis
Pregnancy and peripartum	Cerebral venous thrombosis or eclampsia
Sickle-cell disease	Secondary moyamoya disease
Physical finding	
Asymmetric arm pressures	Coarctation of aorta, aortic dissection, Takayasu's disease, or premature atherosclerosis
Skin	
Needle tracks	Intravenous drug use or HIV infection
Livedo reticularis	Sneddon's syndrome, antiphospholipid antibody syndrome, or systemic lupus erythematosus
Xanthoma or xanthelasma	Hyperlipidemia
Adenopathy	HIV infection, sarcoid, or Tangier disease
Heart murmur	Endocarditis, ventral septal defect, or myxoma
Vessels	
Diminished pulses	Premature atherosclerosis, coarctation of aorta, aortic dissection, or Takayasu's disease
Bruit	Premature atherosclerosis, fibromuscular dysplasia, or arterial dissection
Venous thrombosis in the legs	Hypercoagulable state

* CADASIL denotes cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, and HIV human immunodeficiency virus.


Ischemic stroke or TIA
History
Physical examination
Standard Evaluation
Stroke topography
MRI of the brain
CT of the brain
Vessels
MRA of the head and neck
CTA of the head and neck
Carotid duplex ultrasonography and transcranial Doppler ultrasonography
Cardiac — structure
TTE
TEE
Cardiac — rhythm
12-Lead ECG
Inpatient cardiac telemetry
24-Hr Holter monitor
Hematologic testing
Complete blood count
Platelet count
INR
Partial-thromboplastin time
Stroke considered to be cryptogenic after standard evaluation
Advanced Evaluation
Vessels
Catheter angioplasty
Transcranial Doppler monitoring for emboli
Vasculitis tests
Cardiac — rhythm
Prolonged (2–4 wk) outpatient cardiac telemetry
Hematologic testing
Arterial hypercoagulability tests (all patients)
Venous hypercoagulability tests (if right-to-left shunt)
Stroke considered to be cryptogenic after advanced evaluation
Specialized Evaluation
Genetic testing
Mitochondrial disease
CADASIL, Fabry's disease, other genetic causes
Vessels
Detailed autoimmune evaluation
CSF examination
Brain biopsy
Cardiac — structure
Cardiac CT
Cardiac MRI
Cardiac — rhythm
Prolonged (1–3 yr) outpatient loop recording
Hematologic testing
Workup for occult cancer

Case 2: Man, 23 years

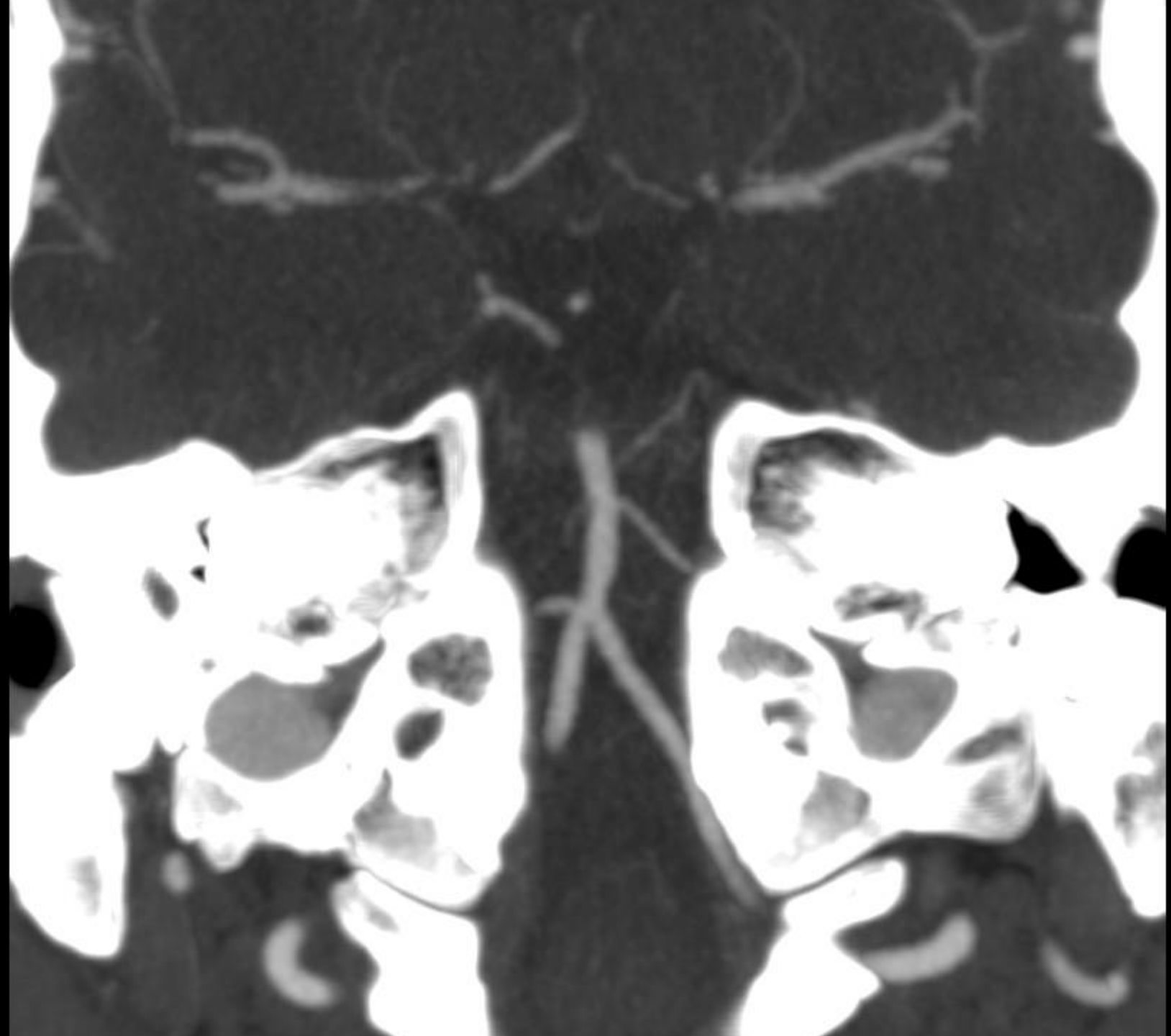


Case 2: Man, 23 years

- No medical history
- Onset of vertigo, vomiting
-


< 80221 - 6 MPR, AXIAL >
R

- Admitted for coma GCS 7, tetraplegic
- Bilateral mydriasis



Your diagnosis and Treatment ?


008
que:
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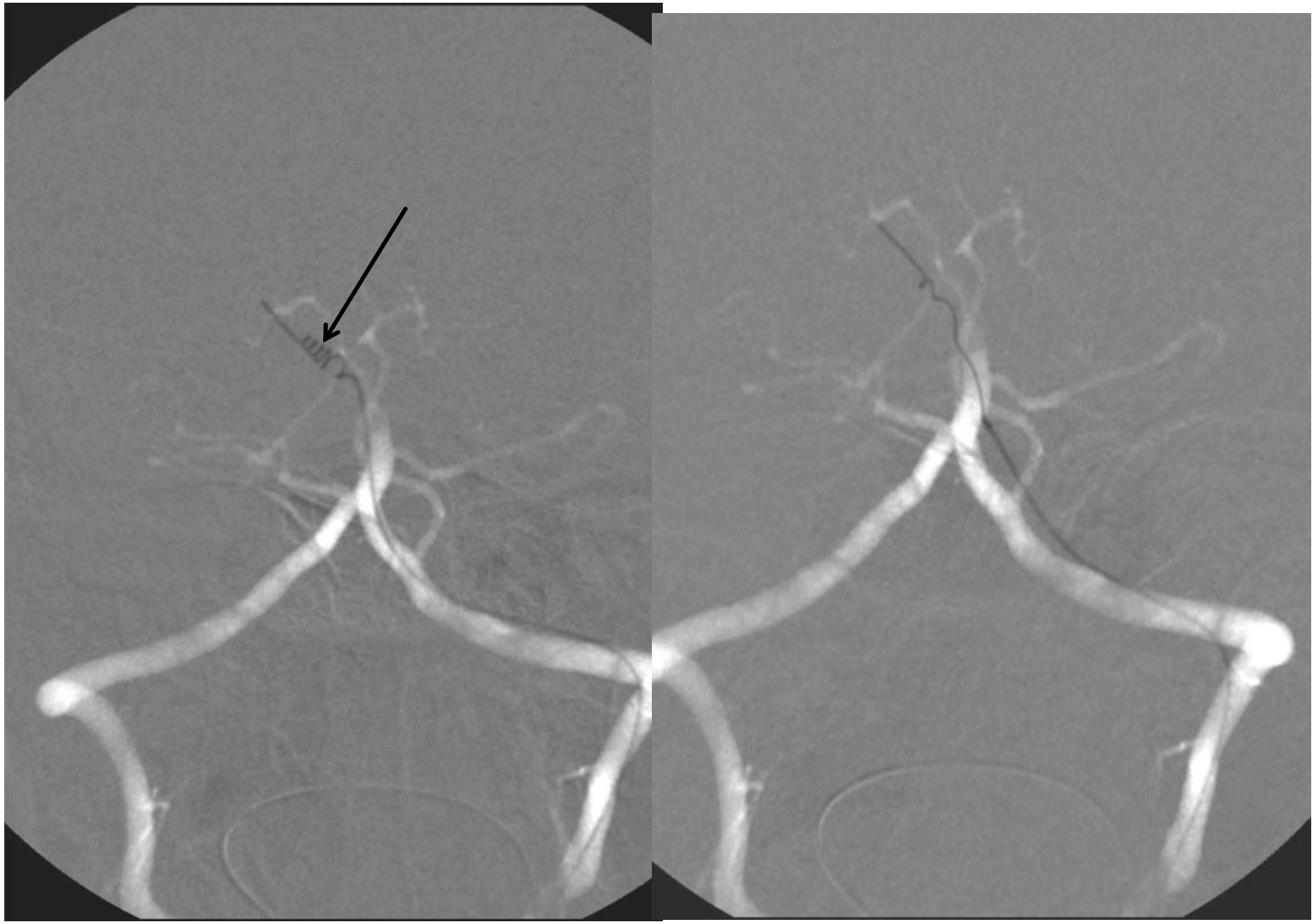
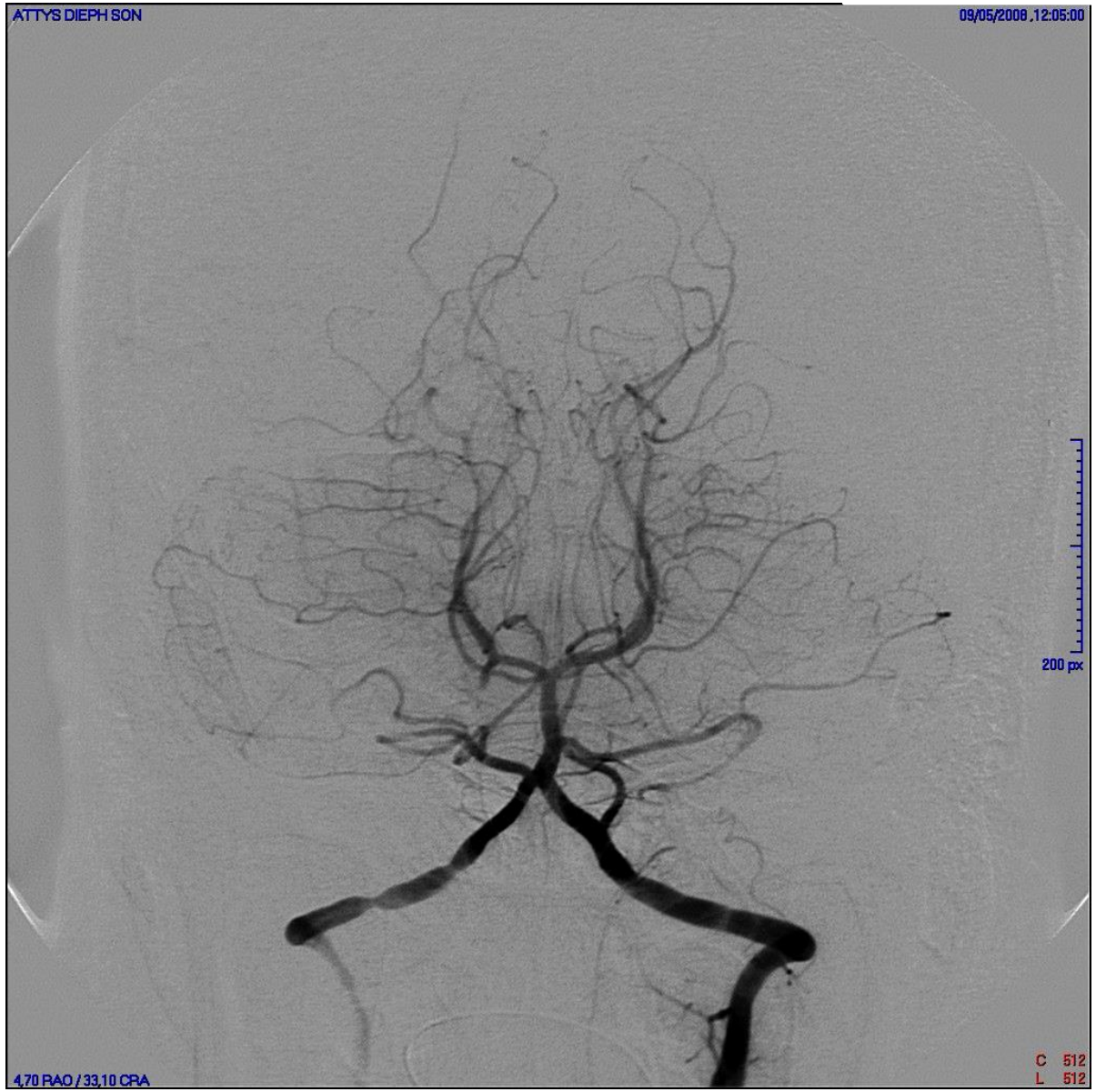
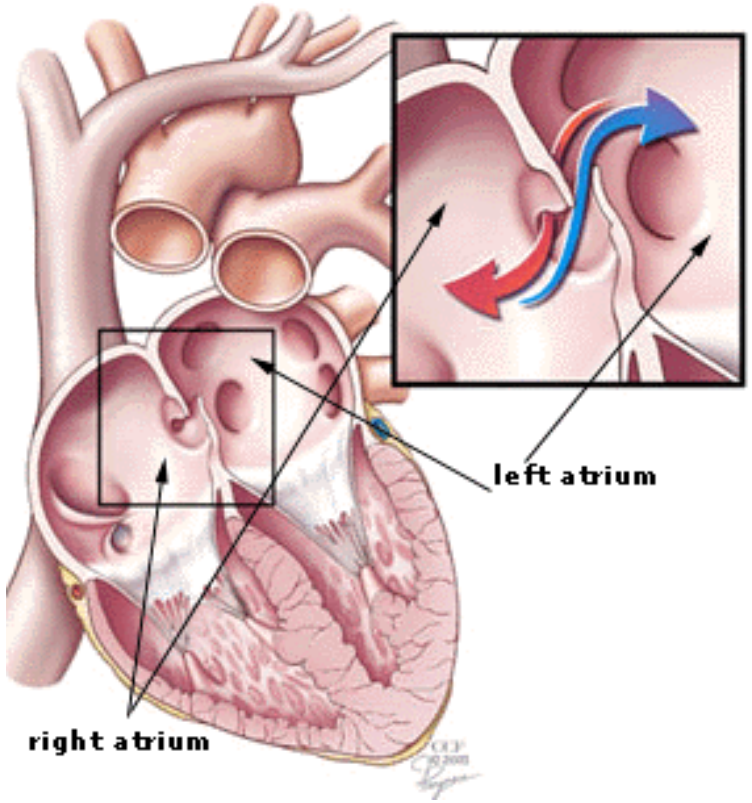
MF

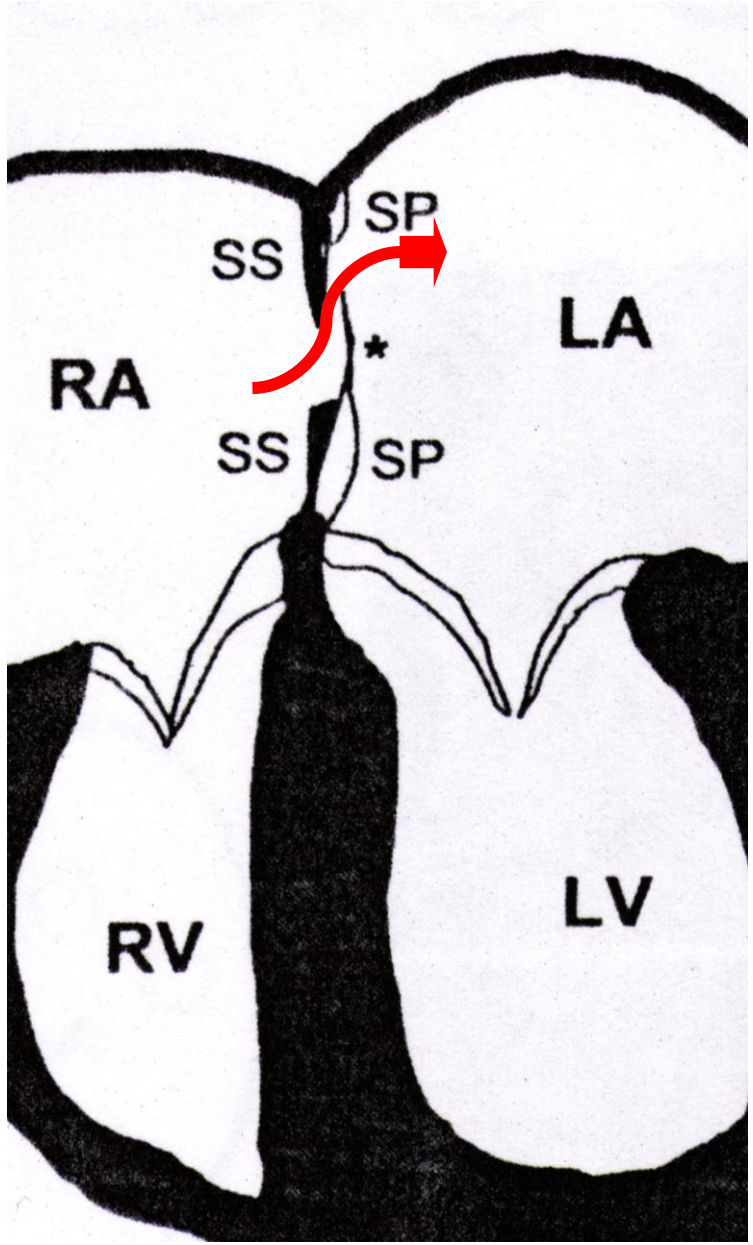



ATTYS DIEPH SON
09/05/2008 ,12:05:00
200 px
4,70 RAO / 33,10 CRA
C 512
L 512

FOP

- 25 à 35 % séries autopsiques
  - (Hagen, 1984)
- forte association avec AVC cryptogéniques (50%)
- surtout sujets jeunes





Sang désaturé
Embol

- Séries autopsiques dans population générale
  - Mayo clinic 1994. 965 coeurs normaux
    - 27.3%
    - Taille moyenne : 5mm (1 -19)
  - Thompson 1930. 1000 autopsies
    - 35%
    - Taille : 2 – 10mm

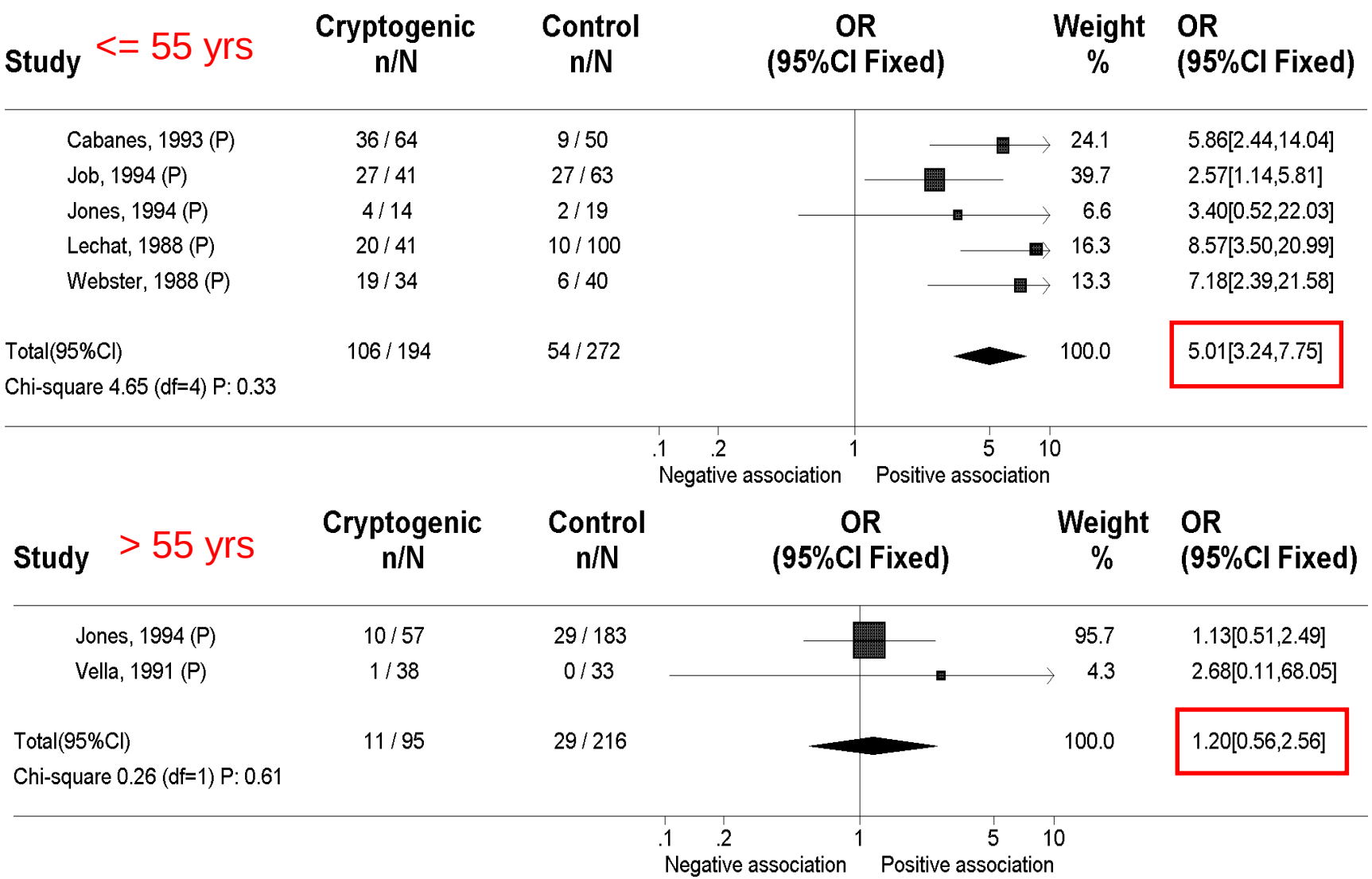
- Etudes cas-contrôles en échocardiographie, après accident vasculaire cérébral
  - 30 à 50% selon voie échographique

- 40% AVC/AIT du sujet <55 ans « idiopathiques »

- Liaison statistique forte
  - entre anomalie du SIA
  - et AVC

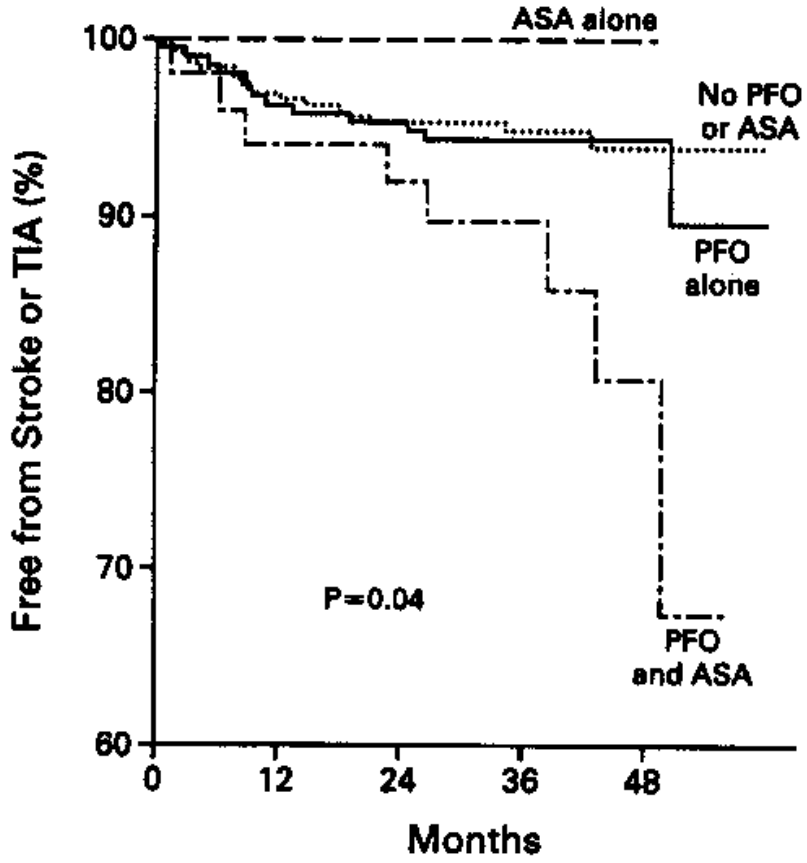
FOP ET AIC : ÉTUDES CAS - TÉMOINS

Overell et al, Neurology 2000



PFO and ASA Study. MAS JL et al. N.Engl.J.Med 2001

581 Patients, <55y, ischaemic stroke of unknown origin, Aspirin 300mg, FU 4y



No. at Risk					
No PFO or ASA	304	291	267	158	48
PFO alone	216	207	198	122	43
ASA alone	10	10	9	4	1
PFO and ASA	51	46	44	25	10

Risk of recurrence at 4 years

	Stroke	Stroke+TIA
No PFO or ASA	4.2	6.2
PFO alone	2.3	5.6
ASA alone	0	0
PFO + ASA*	15.2	19.2

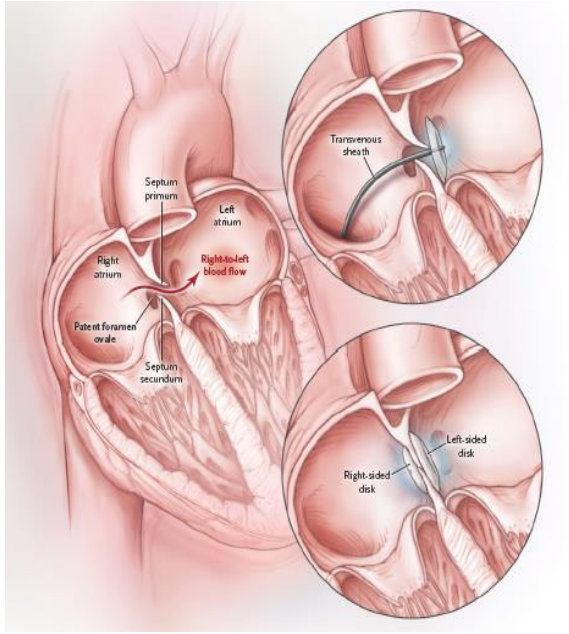
* Risk 4% / year

- Embolie paradoxale
- Thrombus développé dans FOP ou ASIA
- Arythmie paroxystique
- Autre cause (± en rapport avec FOP)
  - Anomalie de coagulation ou système fibrinolytique
- Association fortuite ?

- Antiplaquettaires
  - Bénéfice établi en prévention secondaire après un AIC
  - Bonne tolérance

- Anticoagulants oraux
  - Thrombus de stase dans cavité cardiaque ou système veineux ?
  - Risque hémorragique

- Fermeture du FOP
  - Peut seulement prévenir les embolies paradoxales
  - Risques à court et long terme mal connus



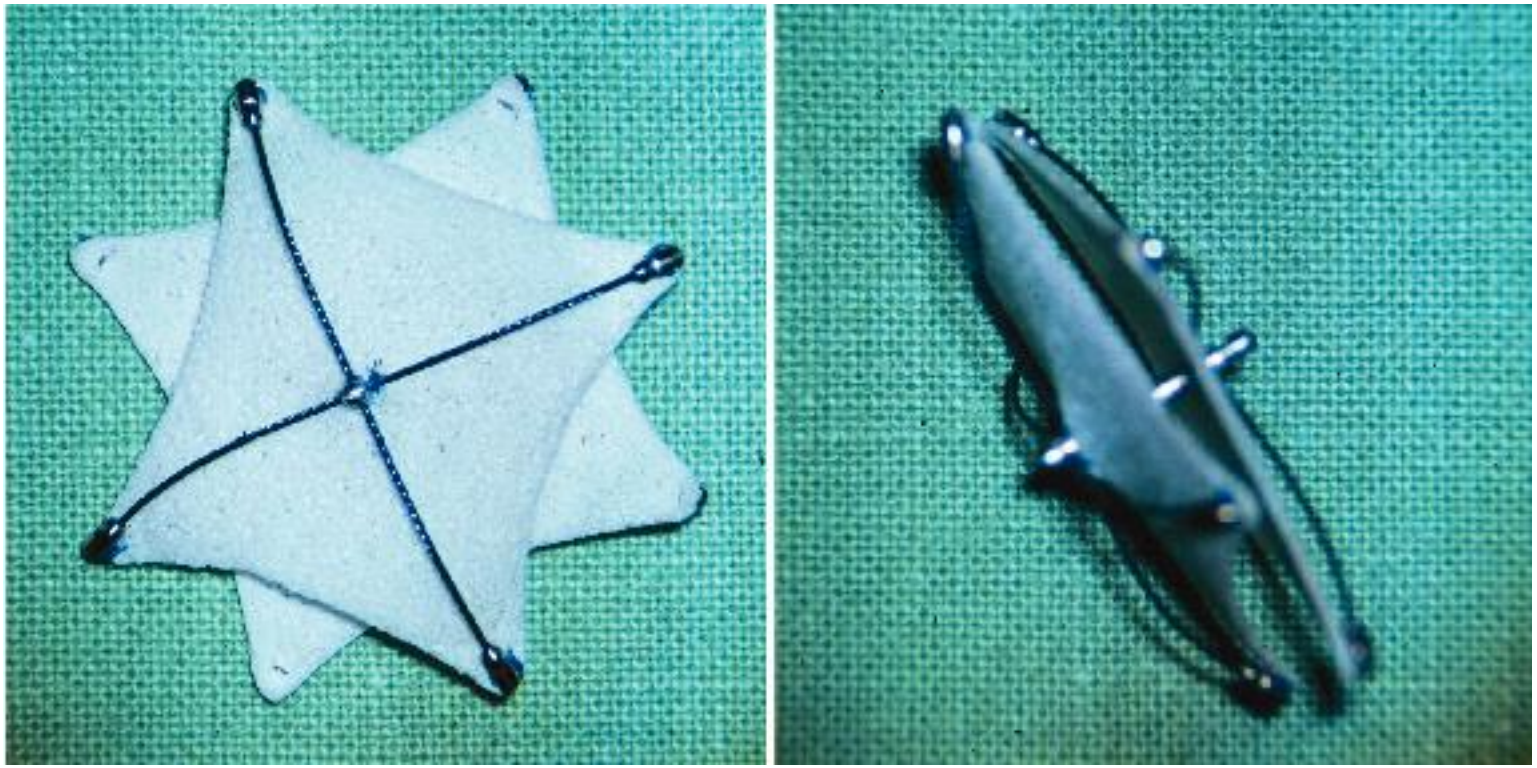
- Complications au site d'accès
- Perforation cardiaque, tamponnade
- Embolie gazeuse
- Migration du matériel
- Arythmies cardiaques
- Thrombus sur le matériel
- Endocardite infectieuse

- Réalisable en < 30 minutes
- Progrès techniques: prothèses biodégradables …
- coût

- Revue systématique : 10 études (n = 1355)
- Complications majeures: 1.5%
- Complications mineures: 7.9%

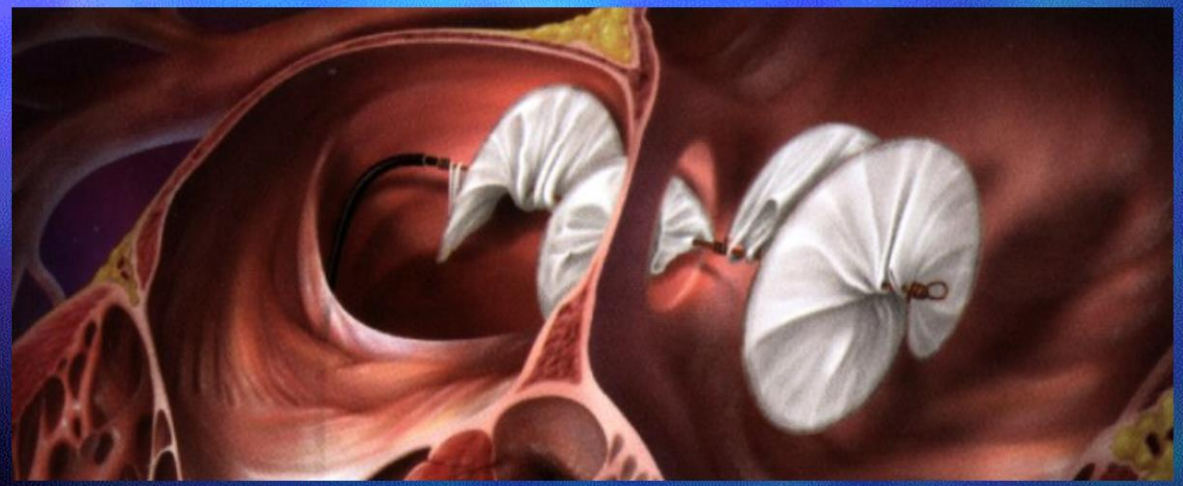
Khairy et al, Ann Intern Med 2003

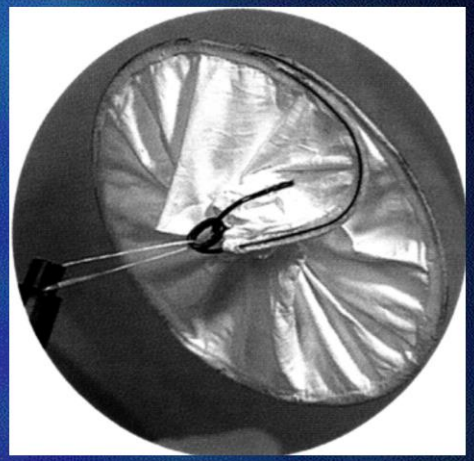
PFO STAR 1ère génération



Grebe O et al. Circulation. 2001;104:e117-e118

Helex septal occluder - Gore





- 16 – 60 ans
- Infarctus cérébral (confirmé par imagerie), cryptogénique, récent (<= 6 mois), Rankin <=3
- FOP > 30 microbulles ou FOP + ASIA

AVK
au long cours

AAP
au long cours

Fermeture du FOP
+ AAP au long cours

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The NEW ENGLAND JOURNAL of MEDICINE

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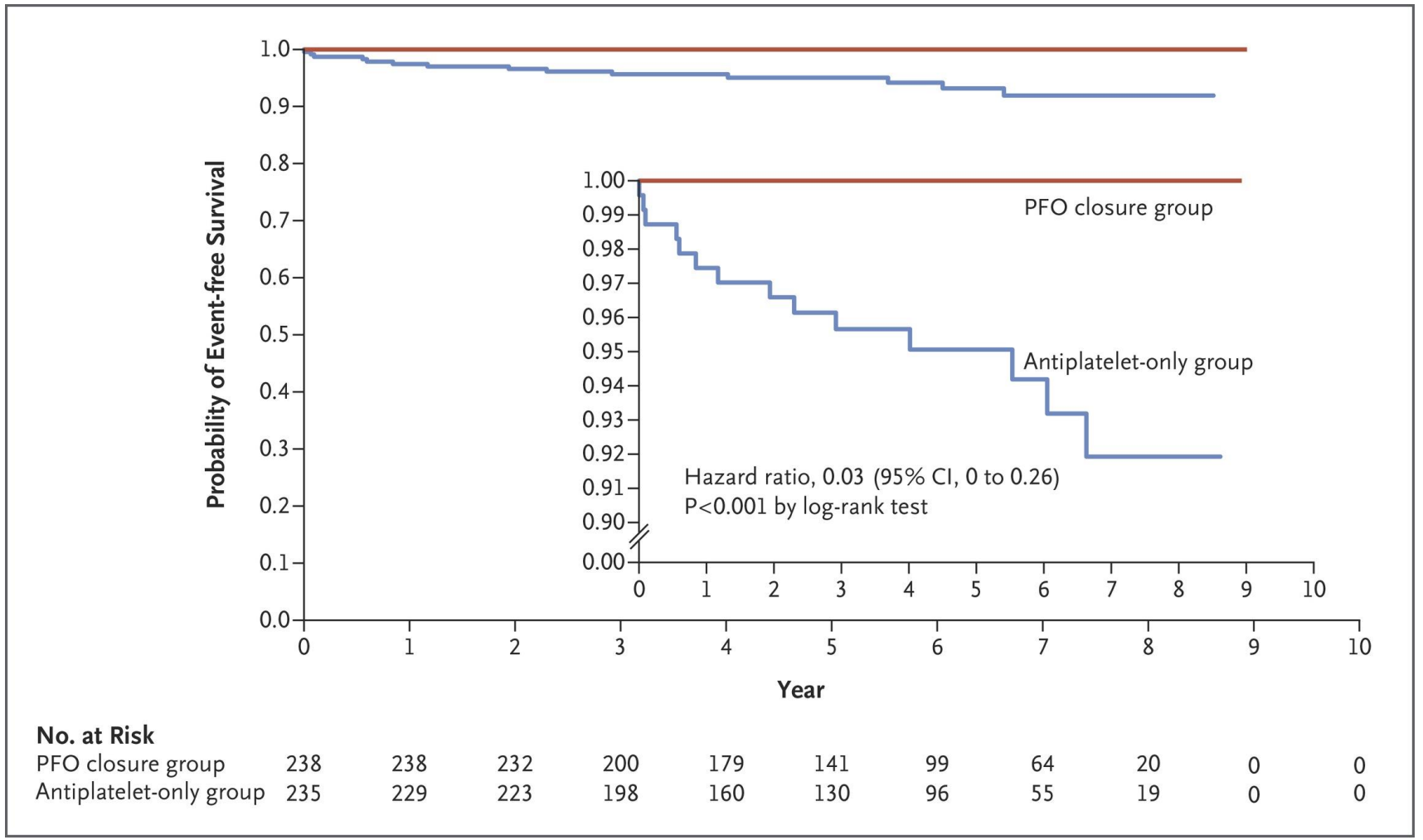
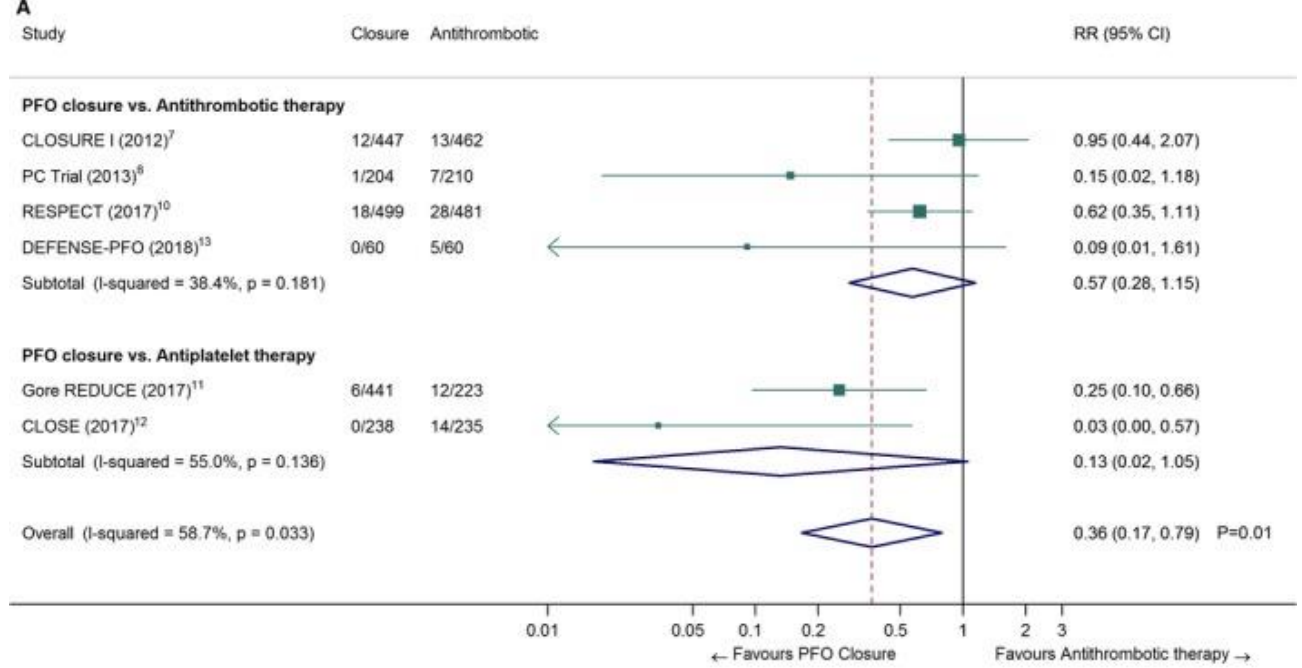
Patent Foramen Ovale Closure or Anticoagulation vs. Antiplatelets after Stroke

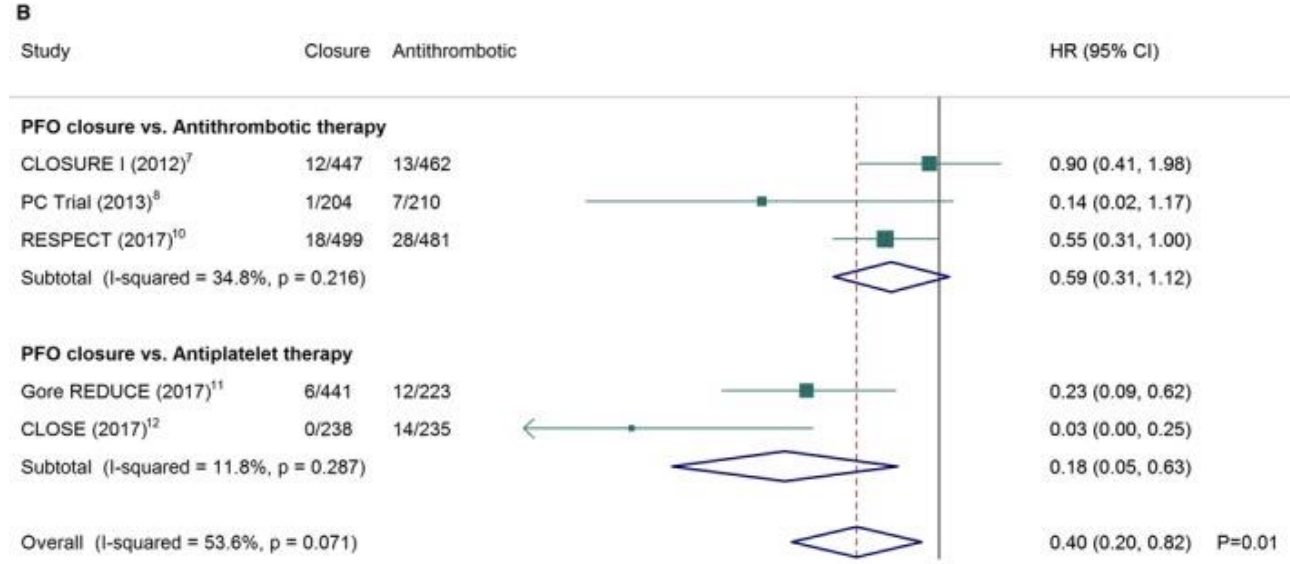
J.-L. Mas, G. Derumeaux, B. Guillon, E. Massardier, H. Hosseini, L. Mechtouff, C. Arquizan, Y. Béjot, F. Vuillier, O. Detante, C. Guidoux, S. Canaple, C. Vaduva, N. Dequatre-Ponchelle, I. Sibon, P. Garnier, A. Ferrier, S. Timsit, E. Robinet-Borgomano, D. Sablot, J.-C. Lacour, M. Zuber, P. Favrole, J.-F. Pinel, M. Apoil, P. Reiner, C. Lefebvre, P. Guérin, C. Piot, R. Rossi, J.-L. Dubois-Randé, J.-C. Eicher, N. Meneveau, J.-R. Lusson, B. Bertrand, J.-M. Schleich, F. Godart, J.-B. Thambo, L. Leborgne, P. Michel, L. Pierard, G. Turc, M. Barthelet, A. Charles-Nelson, C. Weimar, T. Moulin, J.-M. Juliard, and G. Chatellier, for the CLOSE Investigators*



No. at Risk

PFO closure group	238	238	232	200	179	141	99	64	20	0	0
Antiplatelet-only group	235	229	223	198	160	130	96	55	19	0	0


A
Study
Closure
Antithrombotic
RR (95% CI)
PFO closure vs. Antithrombotic therapy
CLOSURE I (2012)[7]
12/447
13/462
0.95 (0.44, 2.07)
PC Trial (2013)[8]
1/204
7/210
0.15 (0.02, 1.18)
RESPECT (2017)[10]
18/499
28/481
0.62 (0.35, 1.11)
DEFENSE-PFO (2018)[13]
0/60
5/60
0.09 (0.01, 1.61)
Subtotal (I-squared = 38.4%, p = 0.181)
0.57 (0.28, 1.15)
PFO closure vs. Antiplatelet therapy
Gore REDUCE (2017)[11]
6/441
12/223
0.25 (0.10, 0.66)
CLOSE (2017)[12]
0/238
14/235
0.03 (0.00, 0.57)
Subtotal (I-squared = 55.0%, p = 0.136)
0.13 (0.02, 1.05)
Overall (I-squared = 58.7%, p = 0.033)
0.36 (0.17, 0.79) P=0.01
0.01
0.05
0.1
0.2
0.5
1
2
3
← Favours PFO Closure
Favours Antithrombotic therapy →


B
Study
Closure
Antithrombotic
HR (95% CI)
PFO closure vs. Antithrombotic therapy
CLOSURE I (2012)[7]
12/447
13/462
0.90 (0.41, 1.98)
PC Trial (2013)[8]
1/204
7/210
0.14 (0.02, 1.17)
RESPECT (2017)[10]
18/499
28/481
0.55 (0.31, 1.00)
Subtotal (I-squared = 34.8%, p = 0.216)
0.59 (0.31, 1.12)
PFO closure vs. Antiplatelet therapy
Gore REDUCE (2017)[11]
6/441
12/223
0.23 (0.09, 0.62)
CLOSE (2017)[12]
0/238
14/235
0.03 (0.00, 0.25)
Subtotal (I-squared = 11.8%, p = 0.287)
0.18 (0.05, 0.63)
Overall (I-squared = 53.6%, p = 0.071)
0.40 (0.20, 0.82) P=0.01

- Fermeture incomplète
- Thrombus développé sur le site de fermeture (prothèse ou chirurgie)
- autre facteur causal?

FAG

- Les données de la littérature montrent que la FAG est une option utile chez les patients associant risque thrombo-embolique élevé (CHA_2DS_2-VASc ≥4) et contrindication aux anticoagulants

Recommendations for occlusion or exclusion of the LAA		
LAA occlusion may be considered for stroke prevention in patients with AF and contraindications for long-term anticoagulant treatment (e.g. intracranial bleeding without a reversible cause).[448,449,481,482]	IIb	B
Surgical occlusion or exclusion of the LAA may be considered for stroke prevention in patients with AF undergoing cardiac surgery.[459,483]	IIb	C

- La population concernée présente des comorbidités fréquentes qui doivent être prises en charge
- Un suivi cardiologique est indispensable
  - ETO ou scanner dans les 3 mois post-intervention
  - Echographie transthoracique à 12 et 24 mois

- Safety and efficacy of platelet glycoprotein VI inhibition in acute ischaemic stroke (ACTIMIS): a randomised, double-blind, placebo-controlled, phase 1b/2a trial

- *Lancet Neurol. 2024 Feb;23(2):157-167. doi: 10.1016/S1474-4422(23)00427-1*

Evidence-based Recommendation: Antithrombotics

PICO 9: In people with TIA and ischaemic stroke, does treatment with dual antiplatelet therapy for longer than 90 days with aspirin plus clopidogrel or aspirin plus dipyridamole, compared to a single antiplatelet, reduce the risk of recurrent stroke?

Evidence-based Recommendation

In people with previous ischaemic stroke or TIA, we recommend against use of dual antiplatelet therapy with aspirin and clopidogrel in the long-term and recommend use of single antiplatelet to reduce the risk of recurrent stroke.

Quality of evidence: **Very Low ⊕**

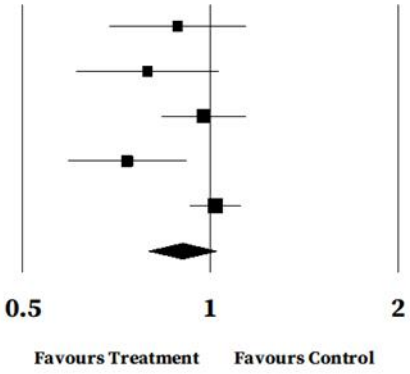
Strength of recommendation: **Weak against intervention ↓?**

ESO
EUROPEAN STROKE ORGANISATION

Supporting Information

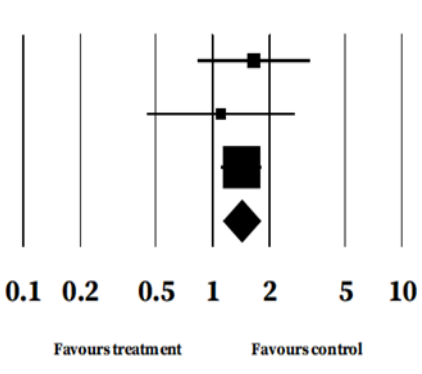
Recurrent Stroke

Study name	Odds ratio	Lower limit	Upper limit	Z-Value	p-Value
SPS3	0.888	0.689	1.144	-0.917	0.359
CHARISMA	0.794	0.610	1.033	-1.715	0.086
MATCH	0.976	0.834	1.142	-0.302	0.763
ESPS2 (aspirin)	0.737	0.591	0.918	-2.725	0.006
PROFESS	1.019	0.925	1.122	0.377	0.706
	0.903	0.797	1.022	-1.612	0.107



ICH

Study name	Hazard ratio	Lower limit	Upper limit	Z-Value	p-Value
SPS3	1.650	0.826	3.295	1.419	0.156
CHARISMA	1.110	0.450	2.739	0.226	0.821
PRoFESS	1.420	1.106	1.823	2.749	0.006
	1.421	1.132	1.784	3.026	0.002



Meta Analysis

- Non-significant reduction in recurrent stroke → NNT 8 per 1000
- Significant increase in intracerebral haemorrhage → NNH 4 per 1000

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EUROPEAN STROKE ORGANISATION

Alternative Strategies: NOACs

PICO 10 Expert Consensus Statement: Low dose NOAC + Antiplatelet

The use of antiplatelet therapy combined with a low-dose direct oral anticoagulant (rivaroxaban) can be considered to optimise treatment of coronary artery disease or peripheral arterial disease in people with a history of ischaemic stroke or TIA more than one month previously. It should not be considered in people with ischaemic stroke or TIA who do not have coronary artery disease or peripheral arterial disease.

PICO 11 Evidence-based Recommendation: NOAC vs Antiplatelet in ESUS

In people with an embolic stroke of undetermined source, we suggest use of antiplatelet therapy and not a DOAC to reduce the risk of recurrent stroke.

Quality of evidence: **Low** ⊕⊕

Strength of recommendation: **Weak against intervention** ↓?

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Supporting Information

- **PICO 10: Low dose NOAC + antiplatelet:**
  - Only trial is COMPASS in primary prevention, limited stroke patients, but more with carotid stenosis
  - Where stroke patients fulfil inclusion criteria for COMPASS, this approach is reasonable.

- **PICO 11: NOAC versus antiplatelet in ESUS:**

  - NAVIGATE and RESPECT (Atticus presented at ESOC 2022)
  - No significant benefit of treatment:
    - Any Stroke: OR 0.96, 95% CI 0.75 to 1.22,

EUROPEAN STROKE ORGANISATION

Expert Consensus Statements: Diabetes

PICO 12: In people with diabetes mellitus and ischaemic stroke or TIA, does intensive control of glycated haemoglobin level (HbA1c) compared to less intensive HbA1c control reduce the risk of recurrent stroke?

Expert Consensus Statement

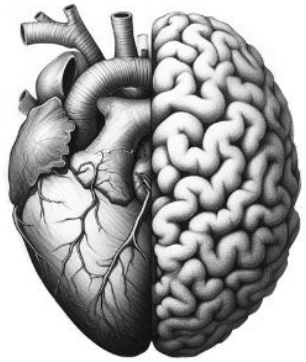
In people with ischaemic stroke or TIA and diabetes mellitus, we support aiming for an HbA1c level of <53mmol/mol (7%, 154 mg/dl) to reduce risk of microvascular and macrovascular complications. However, this target may need to be individualised based on duration of diabetes, age and comorbidities.

- No Secondary Prevention Evidence
- Based upon primary prevention guidance

ESO
EUROPEAN STROKE ORGANISATION

Merci de votre attention

ICCE



- Pr Hassan HOSSEINI
- Dr Stéphane COSSON
- Dr Ruxandra COSSON