Ictus cerebrale

Maurizio Paciaroni
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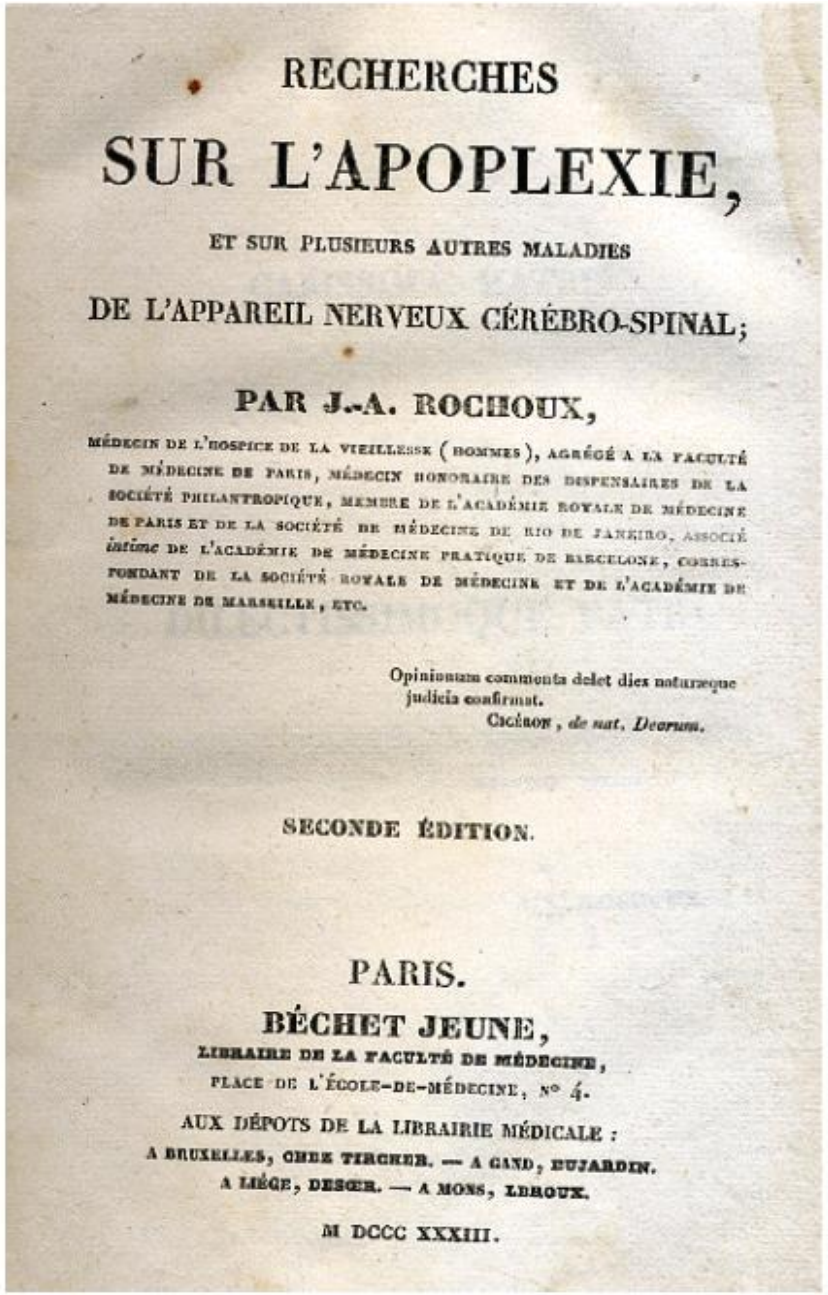
Università degli Studi di Perugia



RECHERCHES

SUR L'APOPLEXIE,

ET SUR PLUSIEURS AUTRES MALADIES

DE L'APPAREIL NERVEUX CÉRÉBRO-SPINAL;

PAR J.-A. ROCHOUX,

MÉDECIN DE L'HOSPICE DE LA VIEILLESSE (HOMMES), AGRÉGÉ A LA FACULTÉ DE MÉDECINE DE PARIS, MÉDECIN HONORAIRE DES DISPENSAIRES DE LA SOCIÉTÉ PHILANTROPIQUE, MEMBRE DE L'ACADÉMIE ROYALE DE MÉDECINE DE PARIS ET DE LA SOCIÉTÉ DE MÉDECINE DE RIO DE JANEIRO, ASSOCIÉ *intime* DE L'ACADÉMIE DE MÉDECINE PRATIQUE DE BARCELONE, CORRESPONDANT DE LA SOCIÉTÉ ROYALE DE MÉDECINE ET DE L'ACADÉMIE DE MÉDECINE DE MARSEILLE, ETC.

Opinionum commenta delet dies naturæque
judicia confirmat.
CICÉRON, *de nat. Deorum.*

SECONDE ÉDITION.

PARIS.

BÉCHET JEUNE,

LIBRAIRE DE LA FACULTÉ DE MÉDECINE,

PLACE DE L'ÉCOLE-DE-MÉDECINE, N° 4.

AUX DÉPOTS DE LA LIBRAIRIE MÉDICALE :

A BRUXELLES, CHEZ TIRCHER. — A GAND, BUJARDIN.

A LIÉGE, DESOER. — A MONS, LEROUX.

M DCCC XXXIII.

How did stroke become of interest to neurologists?

A slow 19th century saga

Maurizio Paciaroni, MD
Julien Bogousslavsky, MD

Neurology® 2009;73:724-728

BRAIN SOFTENING AND APOPLEXIA: EVOLVING CONCEPTS.

After Morgagni, Jean Andre´ Rochoux (1787–1852) claimed in 1814 that apoplexy was always the result of bleeding. The word apoplexy served to describe either the lesion (hemorrhage) or the symptom (loss of movement and sensation). In his monograph, Rochoux also introduced the term “ramollissement” (softening), mentioning that it had been in use for a few years in the medical community.

Second edition, 1833, Jean Andrè Rochoux

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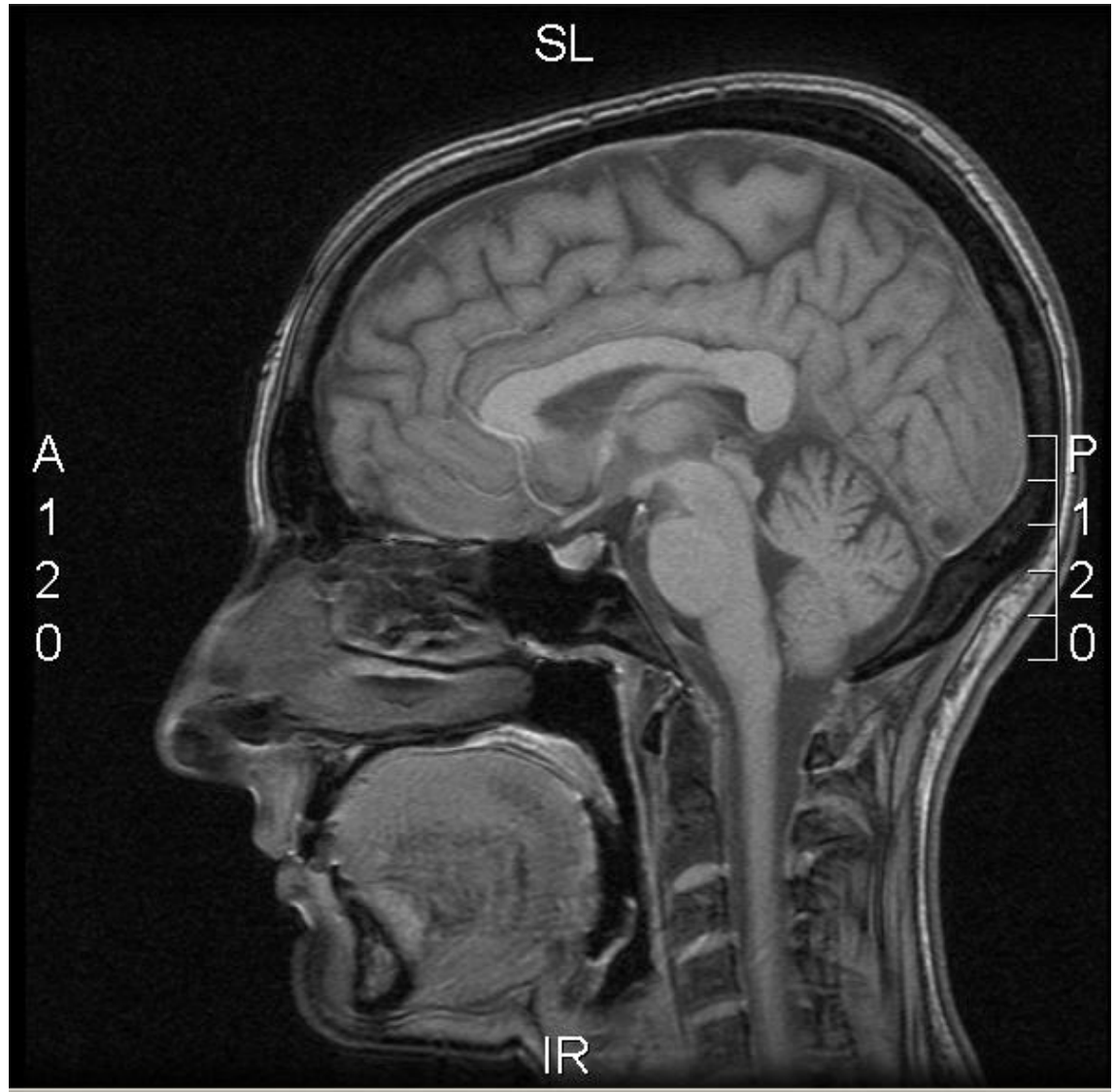
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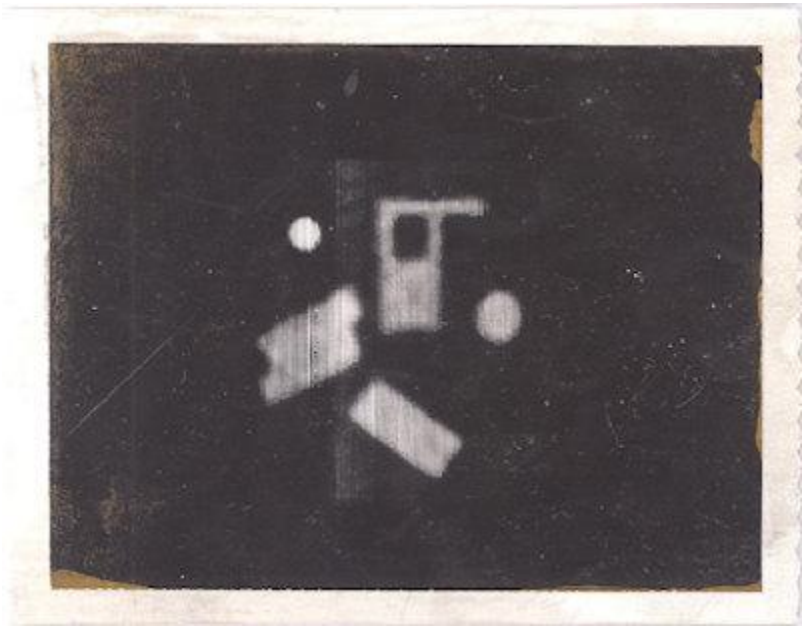
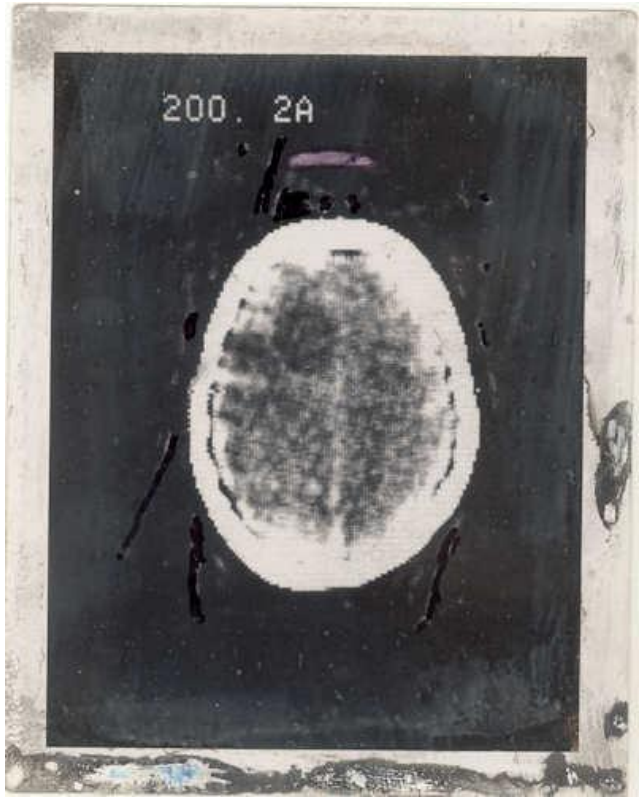
Allan M. Cormack and Godfrey N. Hounsfield:
Nobel Prize in Physiology or Medicine 1979
"for the development of computer assisted tomography"

Paciaroni M, Bogousslavsky J.
The history of stroke and cerebrovascular disease.
Handb Clin Neurol. 2009;92:3-28

1967



Polaroid image of the first CT scan made by Godfrey Hounsfield of a Perspex phantom.



Polaroid image of the first clinical CT scan

Principali cause di morte nel mondo

1	Cardiopatia ischemica	6.260.000
2	Cerebrovasculopatie	4.381.000
3	Infezioni respiratorie	4.299.000
4	Patologie diarroiche	2.946.000
5	Patologie perinatali	2.443.000
6	BPCO	2.211.000
7	Tubercolosi	1.960.000
8	Malattie esantematiche	1.058.000
9	Incidenti della strada	999.000
10	Tumori vie respiratorie	945.000

Murray and Lopez, Lancet 1997

Che cosa è uno stroke?

Definizione

Sindrome clinica caratterizzata dal rapido sviluppo di sintomi e/o segni focali neurologici dovuti alla perdita di una o più funzioni cerebrali di origine vascolare. I sintomi devono durare almeno 24 ore (a meno che vi sia stato un intervento terapeutico o il decesso del paziente)

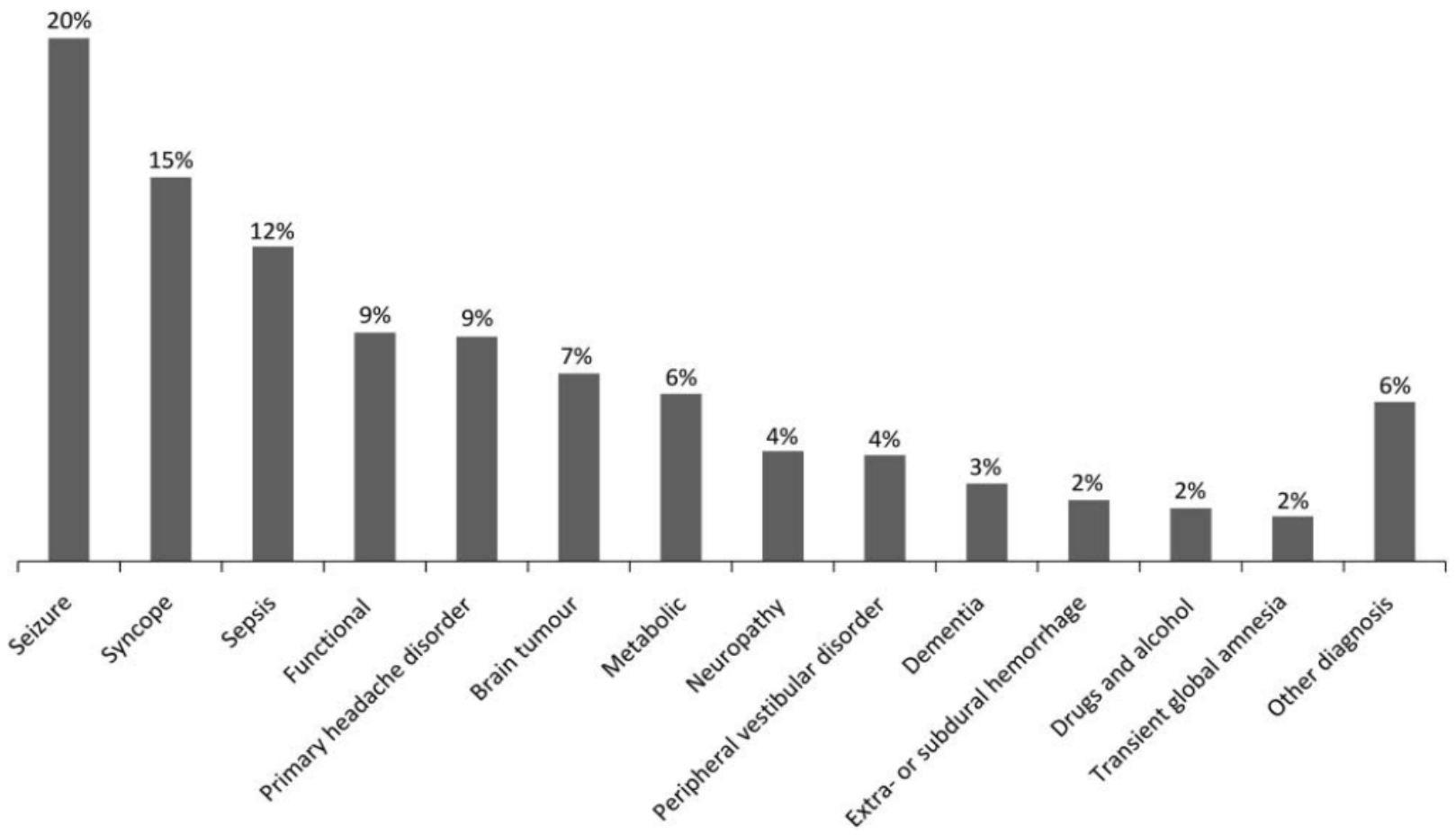
- **Anamnesi (modalità e ora di insorgenza, fattori di rischio)**
- **Esame neurologico (conferma la presenza dei segni neurologici focali anticipati dalla storia clinica)**

Segni neurologici focali

- **Sintomi motori:**
  - debolezza di una metà del corpo in toto o in una sua parte (es. monoparesi)
  - difficoltà nella deglutizione*
  - difficoltà nella coordinazione dei movimenti*
- **Disturbi del linguaggio:**
  - difficoltà nella comprensione e nell'espressione del linguaggio (afasia)
  - difficoltà nella lettura (alessia) e nella scrittura (agrafia)
  - difficoltà nel calcolo (acalculia)
  - difficoltà nell'articolazione del linguaggio (disartria)*
- **Sintomi sensitivi:**
  - negativi (ipoestesia) o positivi (parestesie e disestesie) in una metà del corpo in toto o in una sua parte
- **Sintomi visivi:**
  - perdita della visione completa o parziale in un occhio
  - perdita della visione in un quarto o in una metà del campo visivo (quadrantopsia o emianopasia)
  - cecità in entrambi gli occhi
  - diplopia*
- **Sintomi vestibolari:**
  - vertigini*
- **Disturbi cognitivi o comportamentali:**
  - difficoltà nel vestirsi, nel lavarsi (aprassia); disfunzioni percettive visuo-spaziali (es. emidisattenzione)
  - amnesia*

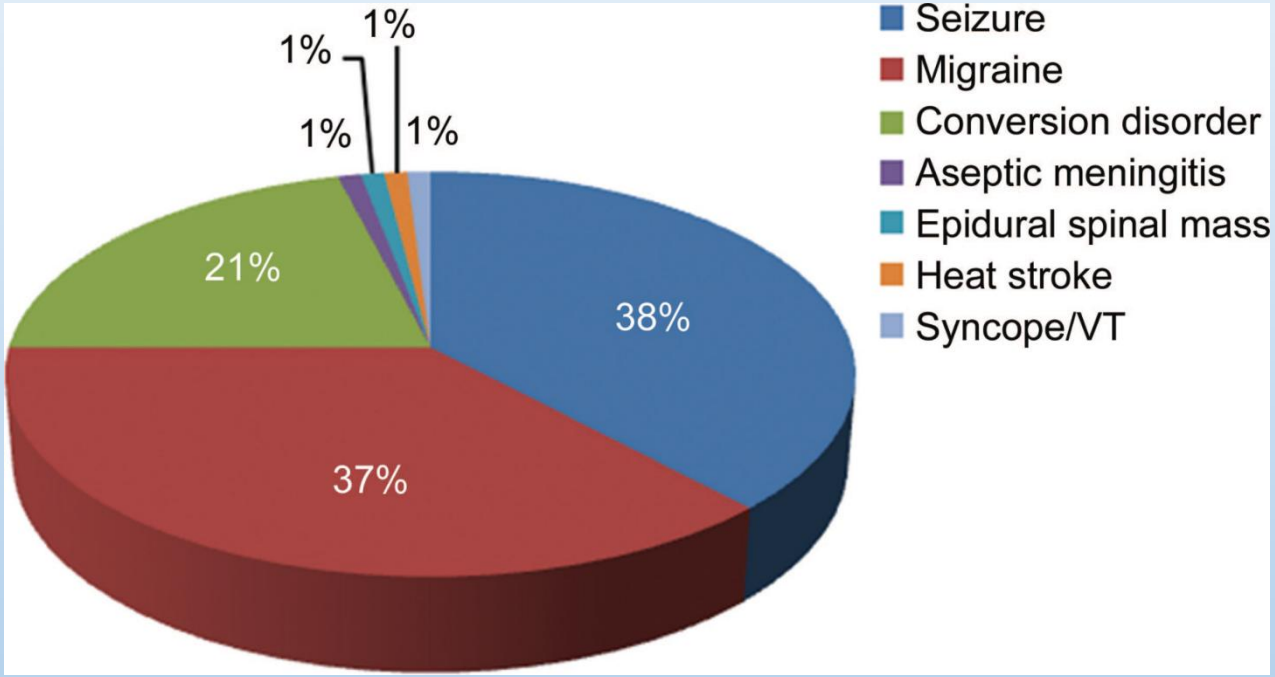
*isolati non possono essere considerati segni neurologici focali

The 20 most common stroke mimics



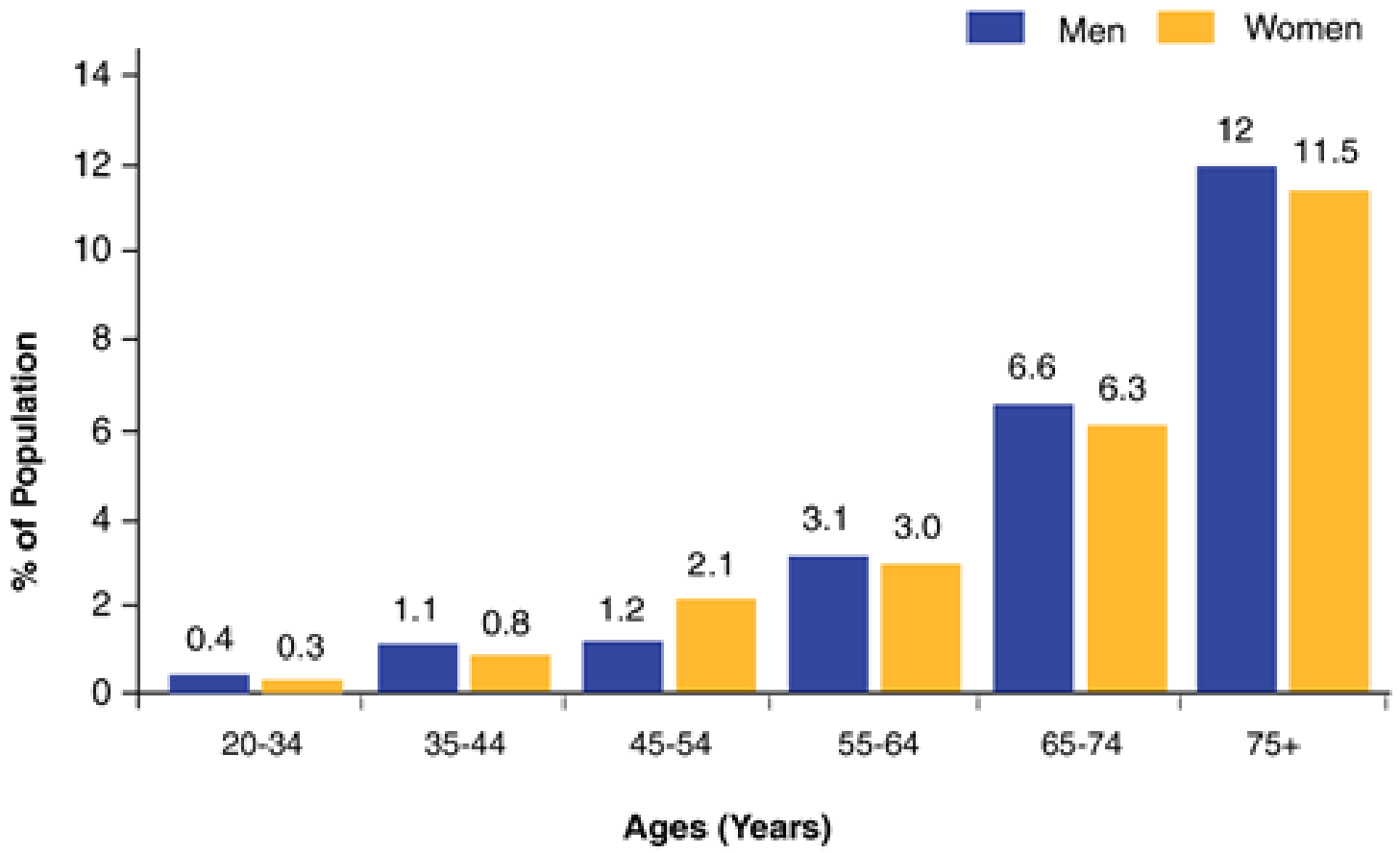
Whiteley et al, unpublished data

The most common stroke mimics: acute phase



Neurology® 2010;74:1340 –1345

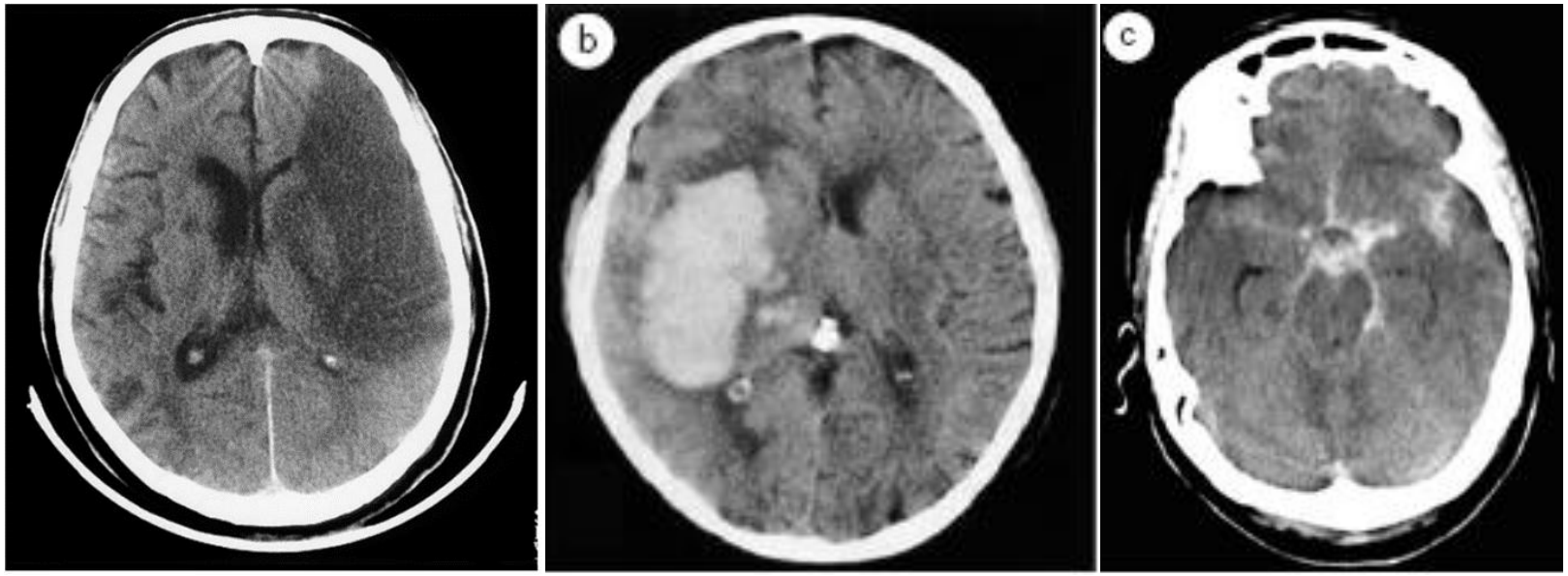
Prevalence of Stroke by Age and Sex: NHANES 1999-2002



Source: CDC/NCHS and NHLBI

Distribuzione ictus cerebri

Ictus ischemico	**67.3-80.5%**
Emorragia intraparenchimale	**6.5%-19.6%**
Emorragia subaracnoidea	**0.8-7.0%**
Forme indeterminate	**2.0-17.5%**



Feigin et al, Lancet Neurol 2003

Incidenza dei diversi sottotipi di stroke

Tassi grezzi di d'incidenza (per 1·000 per anno) dei diversi tipi di ictus nei registri italiani

registro	ischemia cerebrale	emorragia cerebrale	emorragia subaracnoidea	eventi mal definiti
Perugia	2,94	0,40	0,17	0,23
Aosta	1,50	0,30	0,05	0,40
Belluno	1,50	0,43	0,05	0,23
Aosta II	2,38	0,30	0,12	0,08
Vibo Valentia	1,31	0,35	0,06	0,07
L'Aquila	2,42	0,37	0,08	0,04

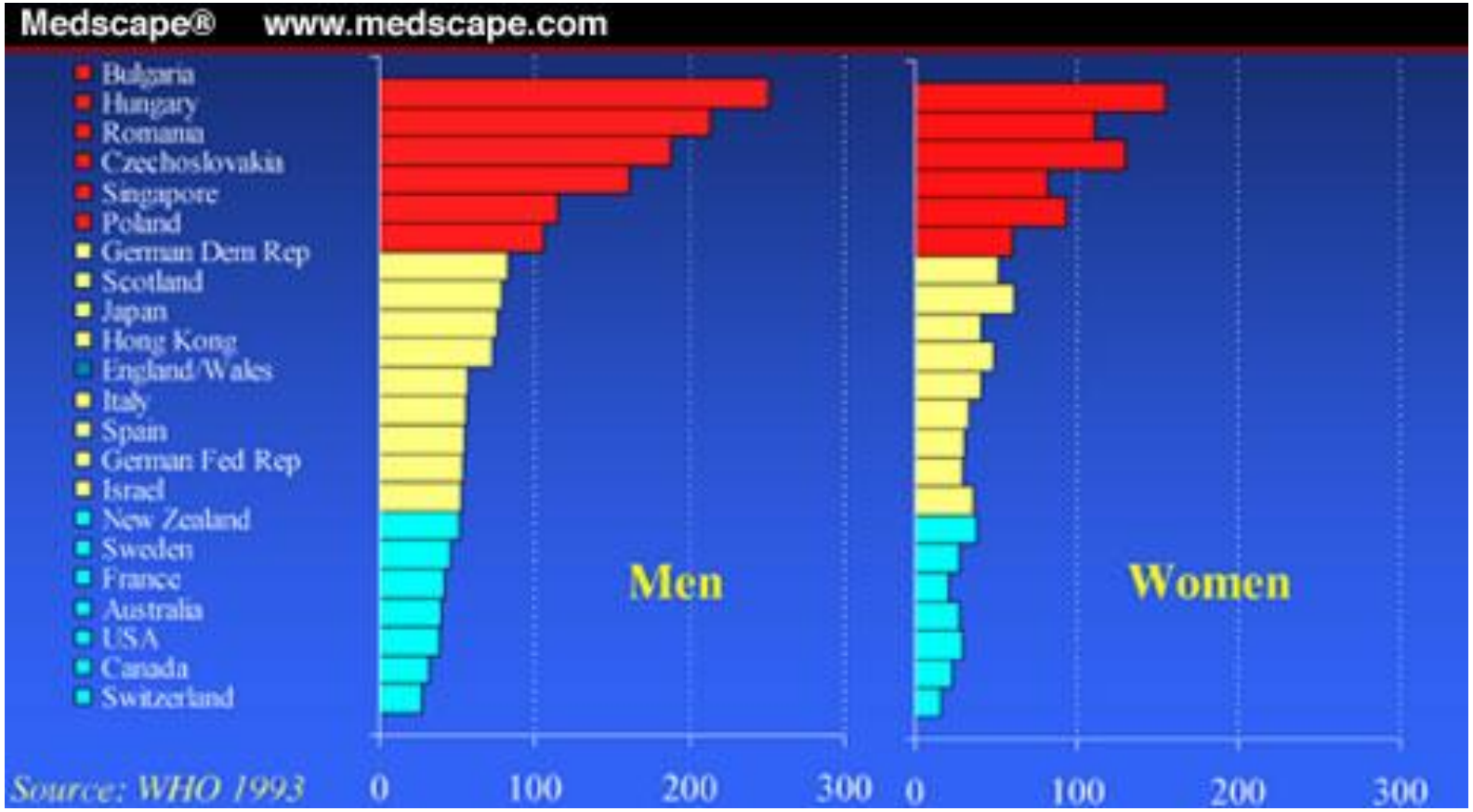
SPREAD 2007

Mortalità a 30 giorni dei diversi sottotipi di stroke

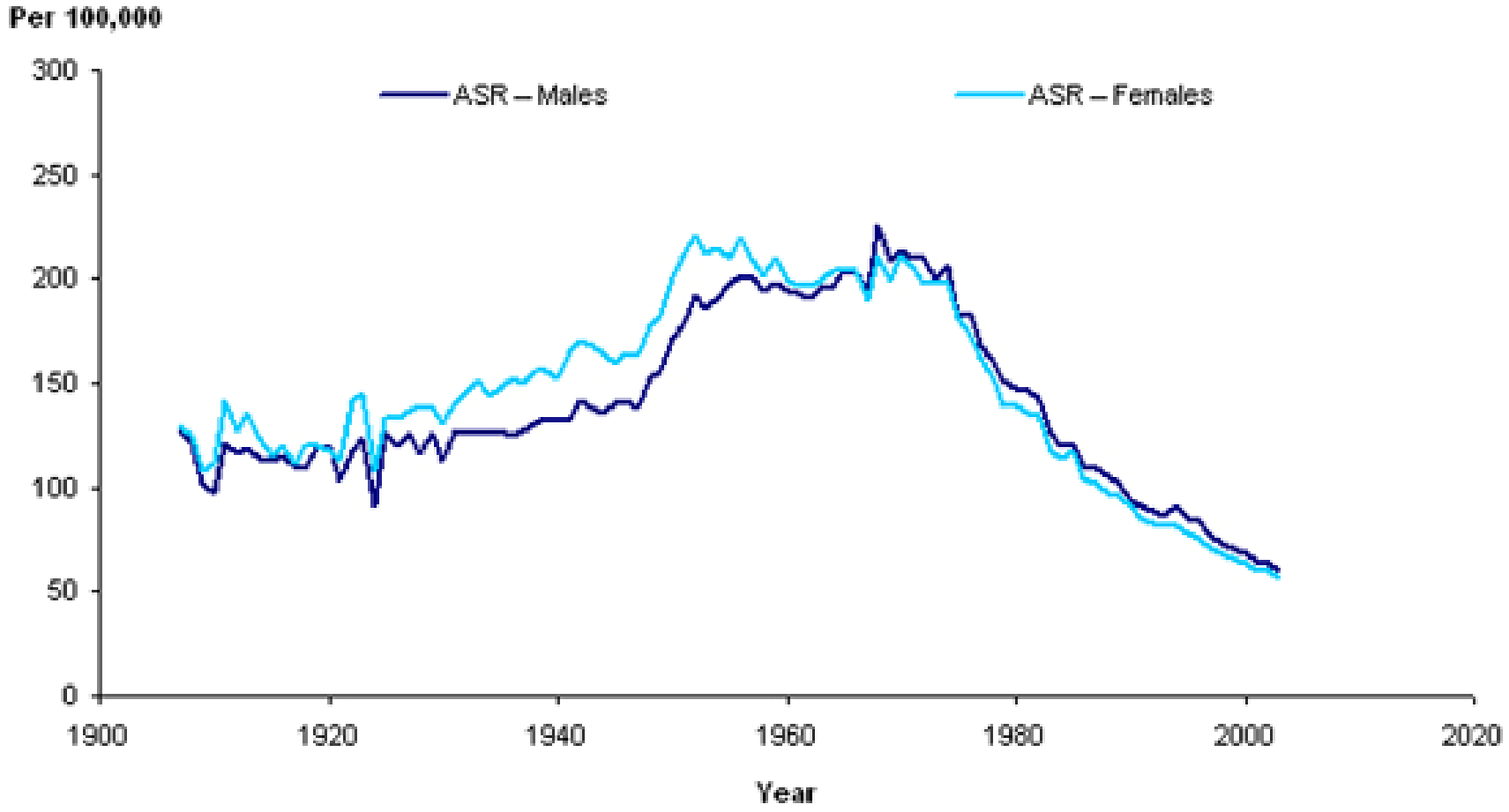
tipo di ictus	numero di casi	decessi	tasso di letalità	IC_{95}
emorragia subaracnoidea	118	41	34,7%	26,2-43,3
emorragia cerebrale	588	283	48,1%	44,1-52,2
ischemia cerebrale	3·594	863	21,2%	19,9-22,6
eventi mal definiti	53	40	75,5%	63,9-87,1
totale	4·353	1·127	25,9%	24,6-27,2

Sacco S et al, Riv Ital Neurobiol 2006

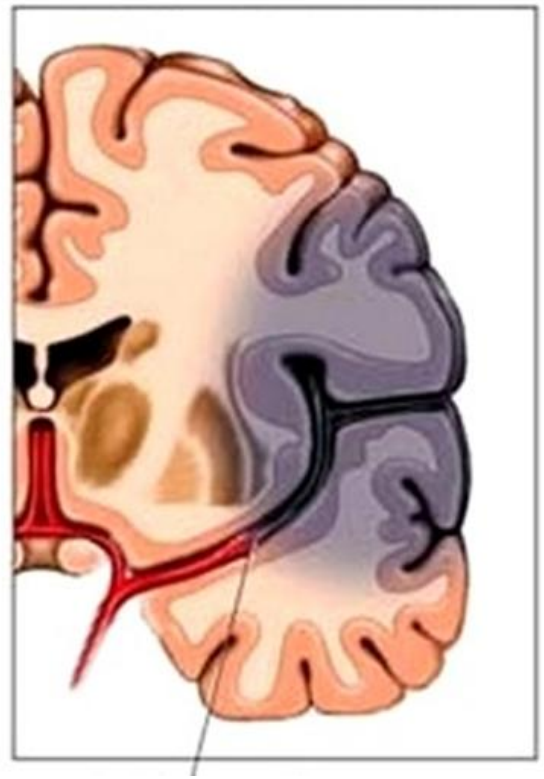
Mortalità per ictus



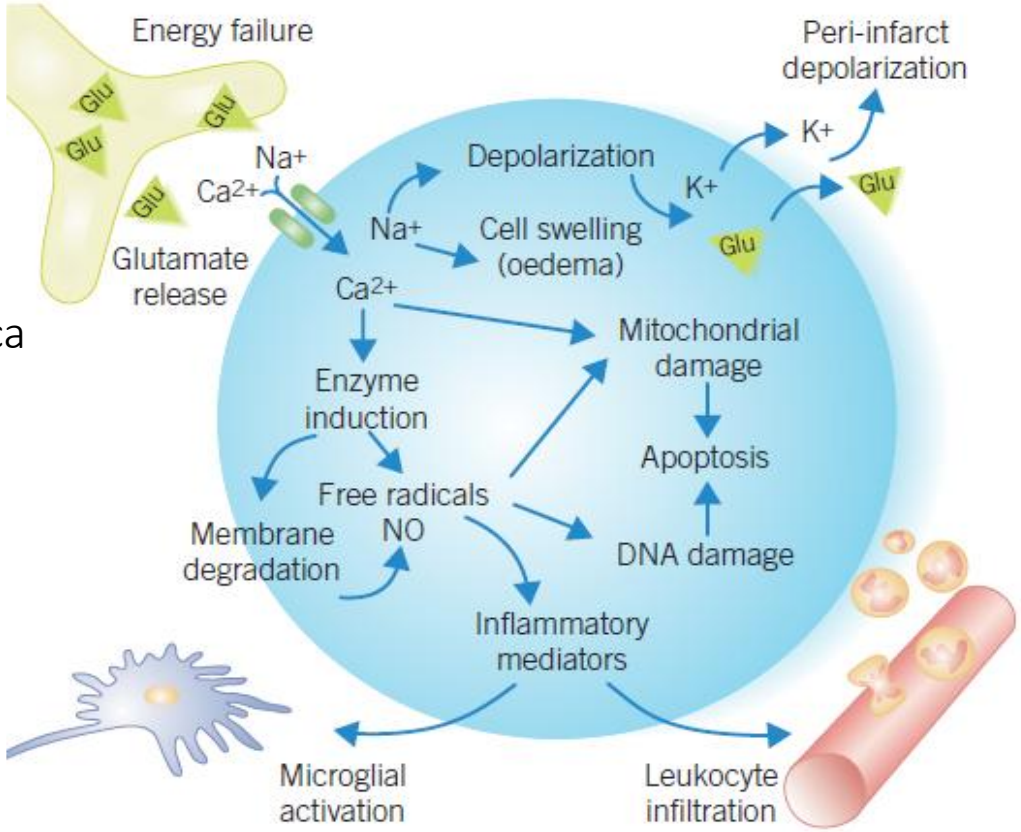
Mortalità nel corso degli anni



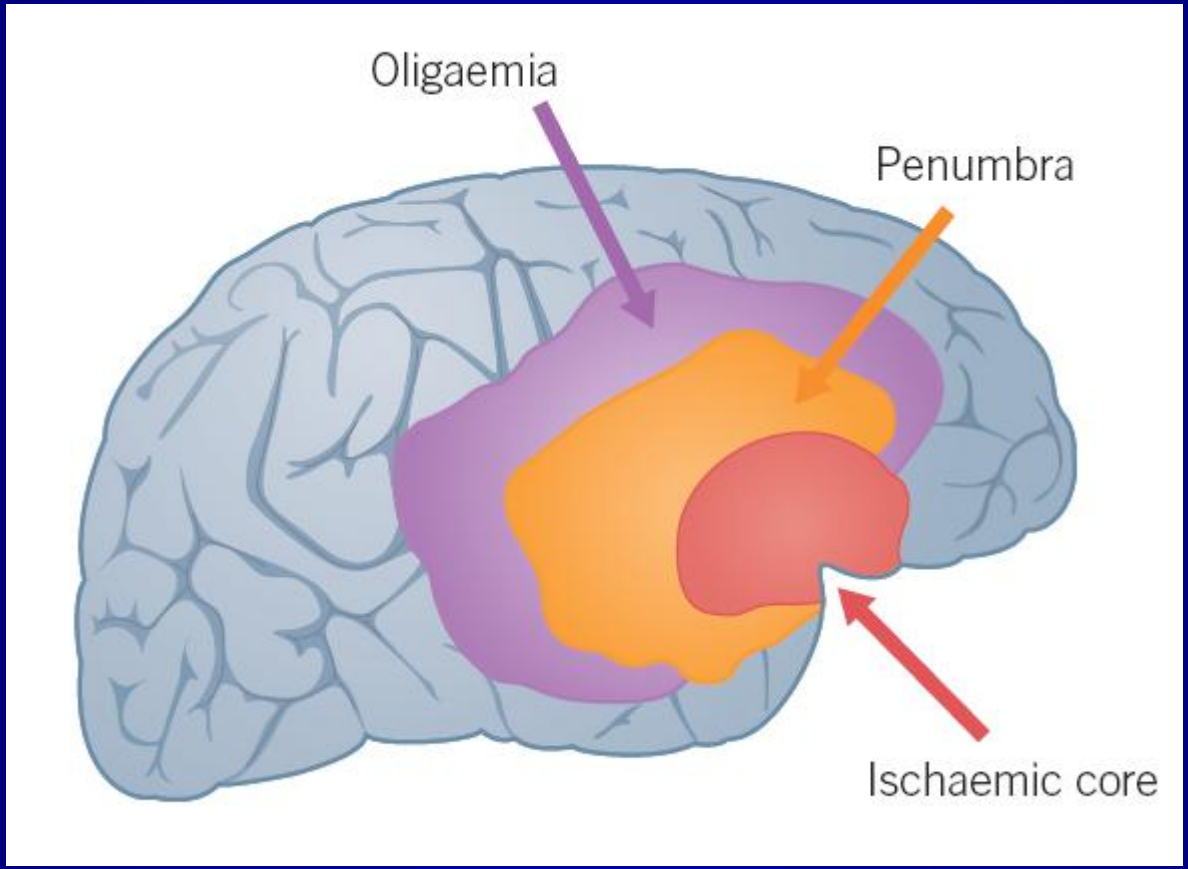
Ictus ischemico



Ictus ischemico:
La cascata ischemica



- Acidosi
- Rilascio di neurotrasmettitori
- Ingresso di Calcio nelle cellule
- Attivazione di proteasi
- Produzione di radicali liberi
- Depolarizzazione peri-infartuale
- Produzione di molecole pro-infiammatorie
- Attivazione di 2° messaggeri intracellulari
- Induzione di geni che promuovono la morte cellulare


Oligaemia
Penumbra
Ischaemic core

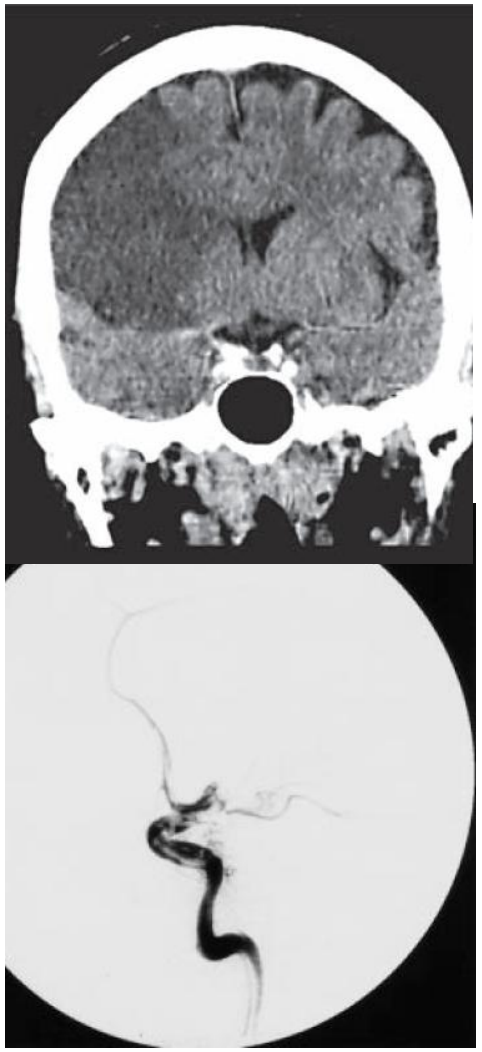
Internal Carotid Artery

Cervical Segment

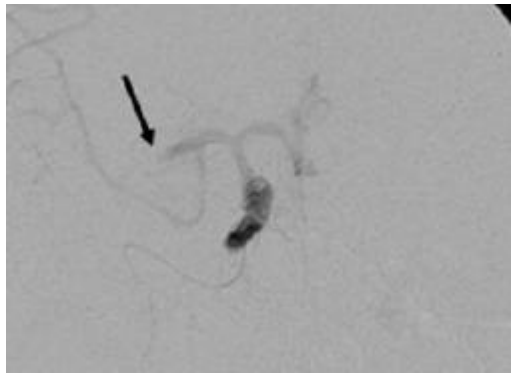
- 2 subsegments:
  - **CAROTID BULB**: focal dilatation of ICA at its origin.
  - **ASCENDING CERVICAL SEGMENT**: extending to the base of the skull
- No collateral branches

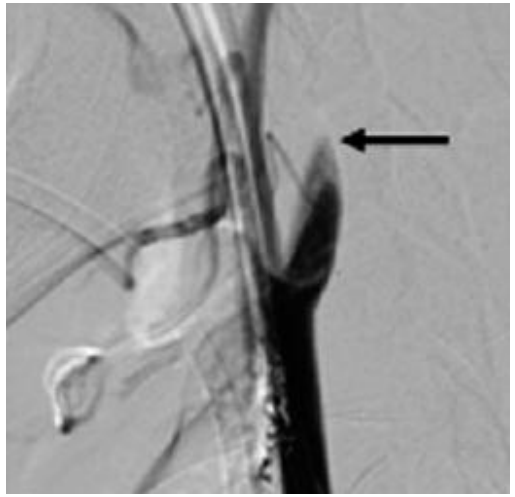
Different types of carotid occlusion

Proximal

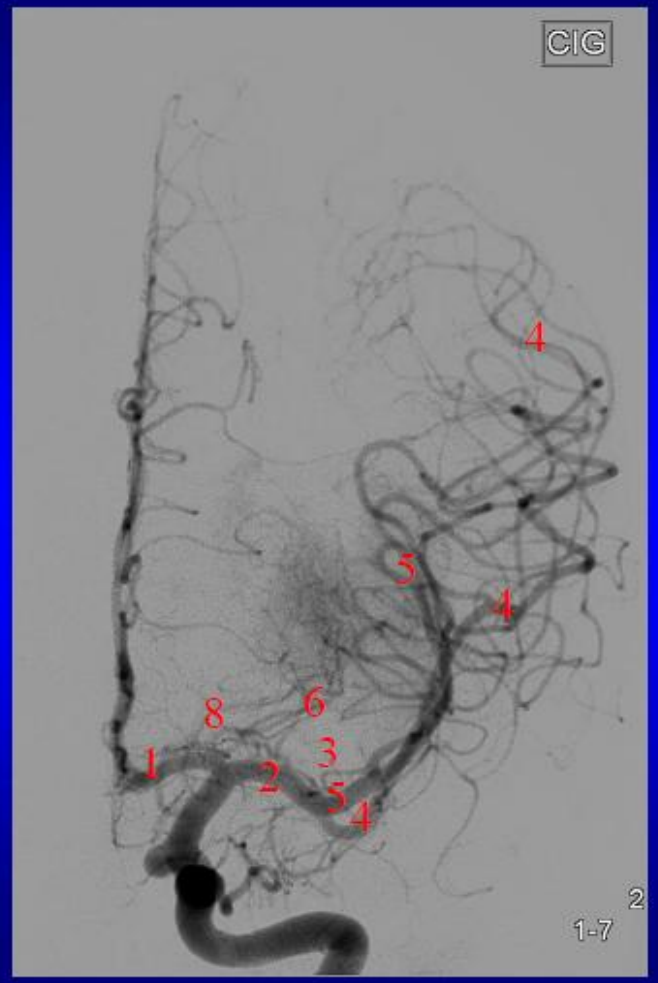


Distal (T)



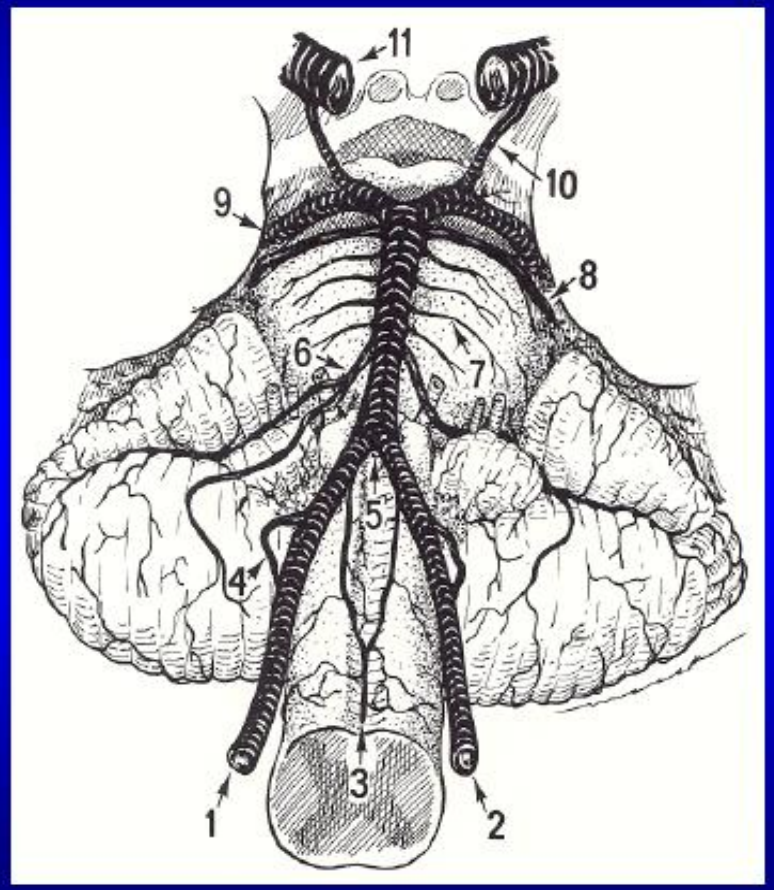


Tandem

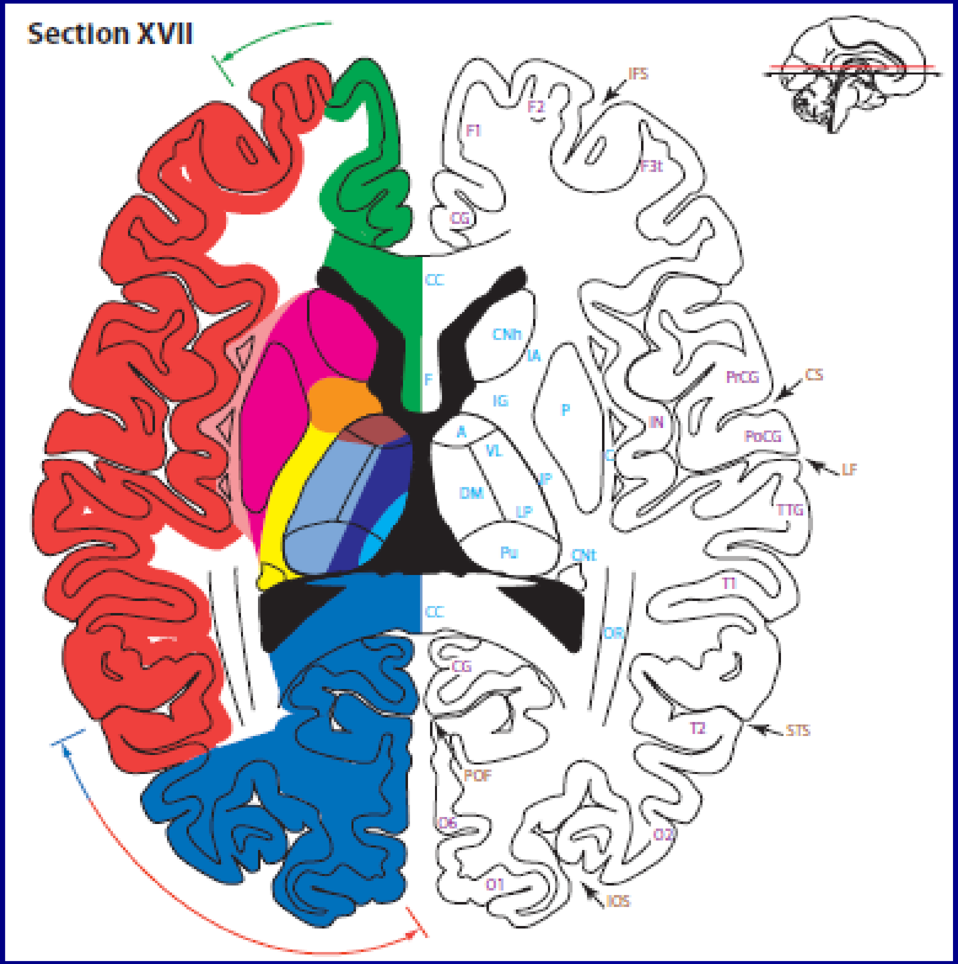


1. A1 segment
2. Prebifurcation M1 segm. (origin of anterior temporal art.)
3. Postbifurcation M1 segm.
4. Inferior Division MCA (M2)
5. Superior Division MCA (M2)
6. Lenticulostriate arteries
8. Recurrent artery of Heubner

Vertebro-Basilar Junction



1. Right Vertebral Artery
2. Left Vertebral Artery
3. Anterior Spinal Artery
4. PICA
5. Junction of VAs ⇨ basilar artery
6. AICA
7. Lateral circumferential branches
8. SCA
9. PCA
10. PComA
11. ICA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Quale parte dell'encefalo è coinvolta?
Quale è il territorio arterioso coinvolto?

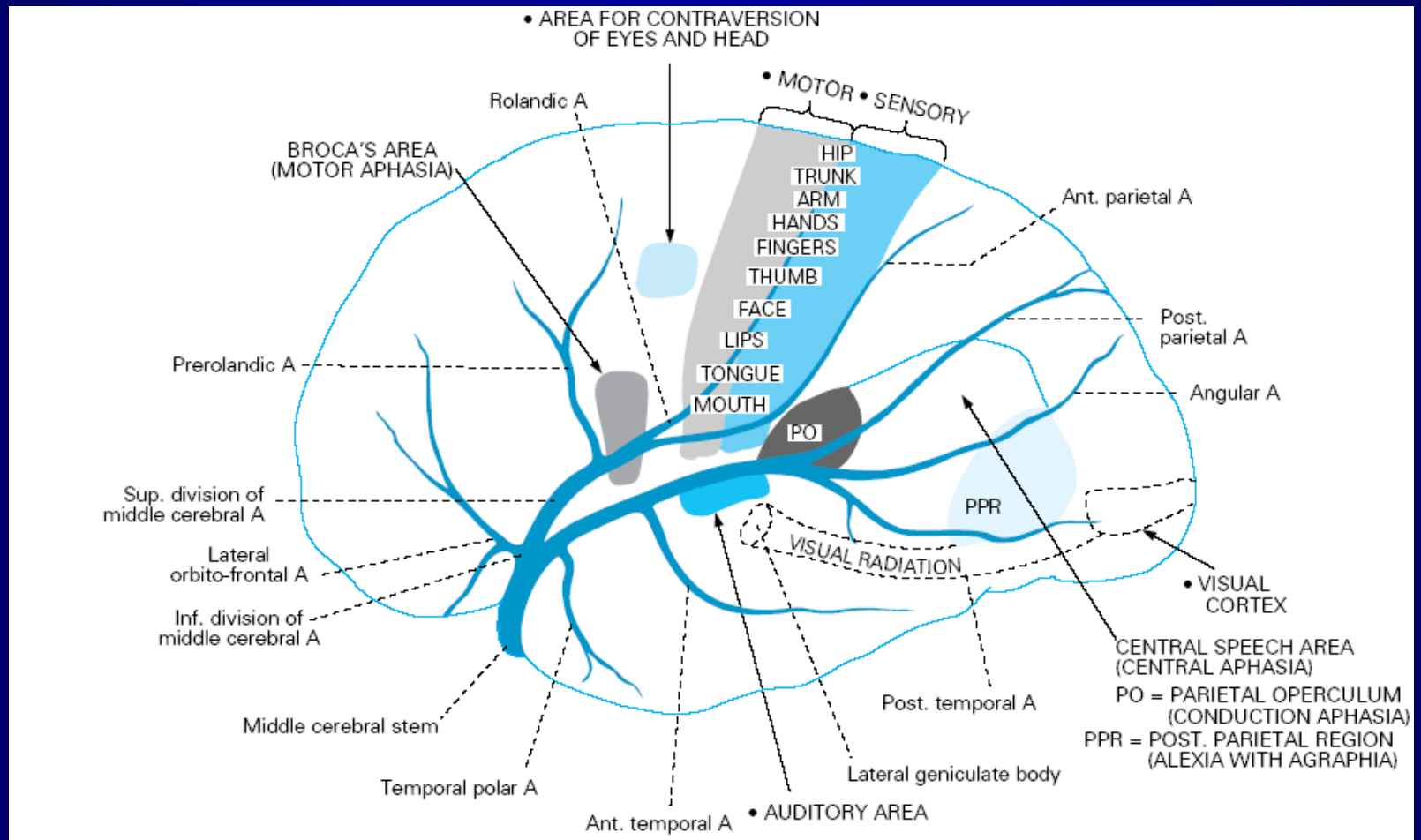
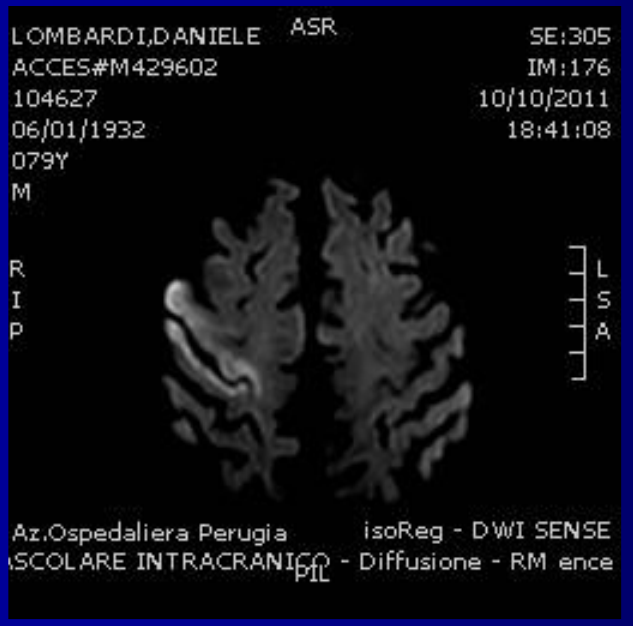


Figure 34-4. Diagram of a cerebral hemisphere, lateral aspect, showing the branches and distribution of the **middle cerebral artery** and the principal regions of cerebral localization. Below is a list of the clinical manifestations of infarction in the territory of this artery and the corresponding regions of cerebral damage.

ACM completa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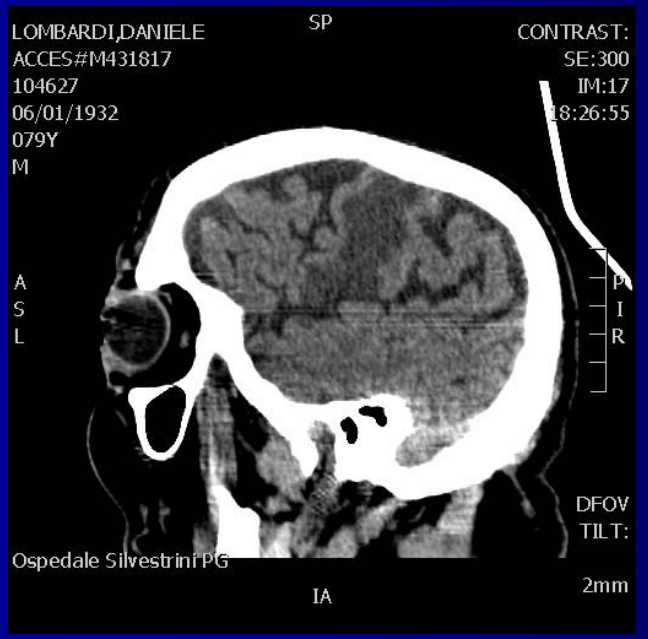
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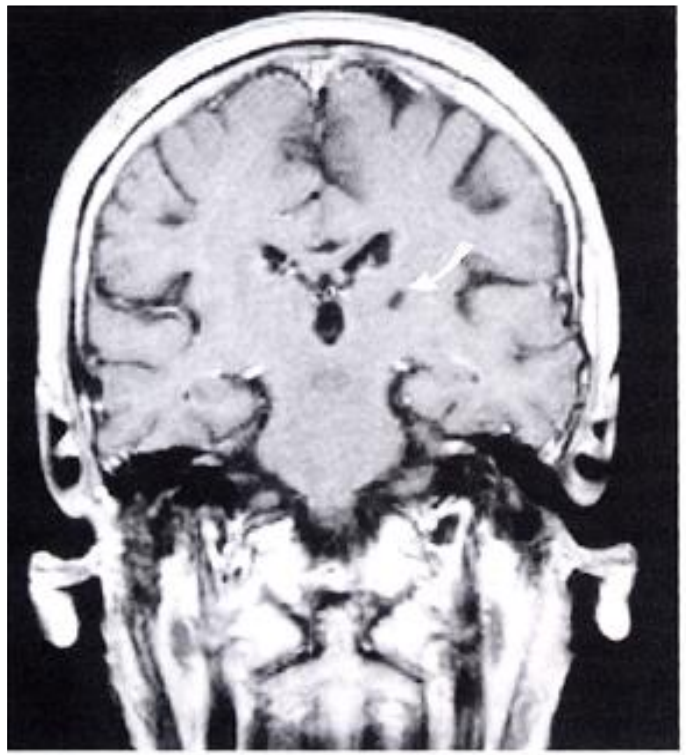
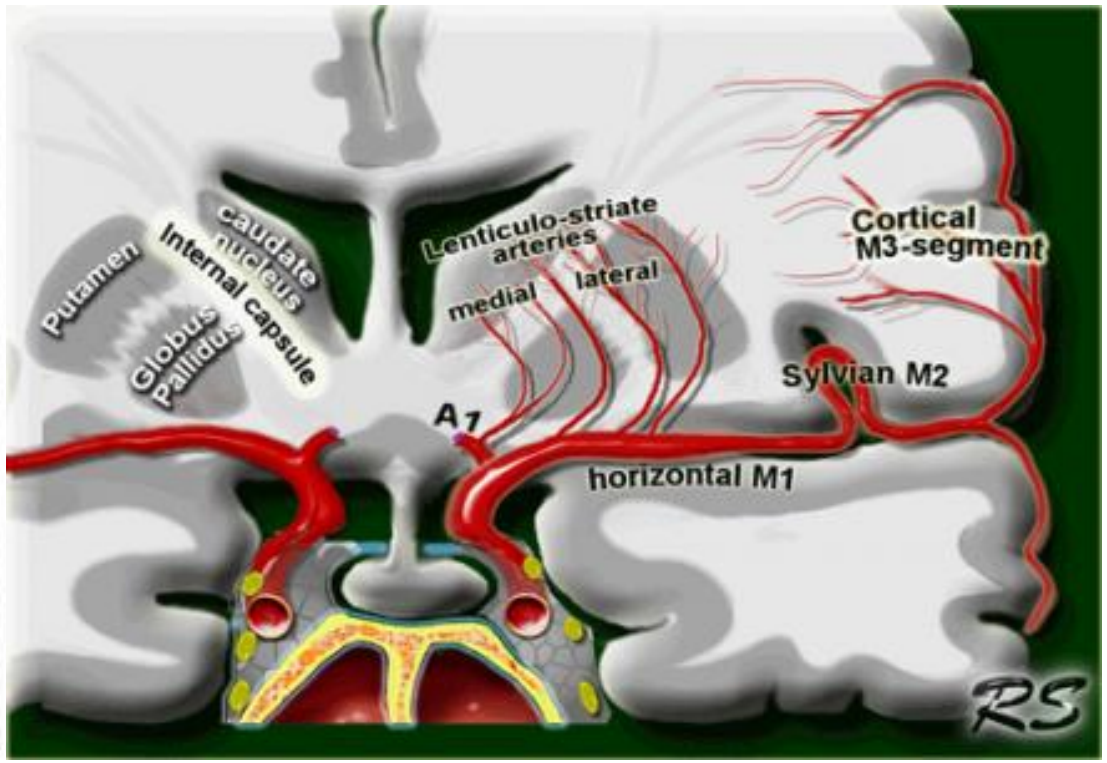
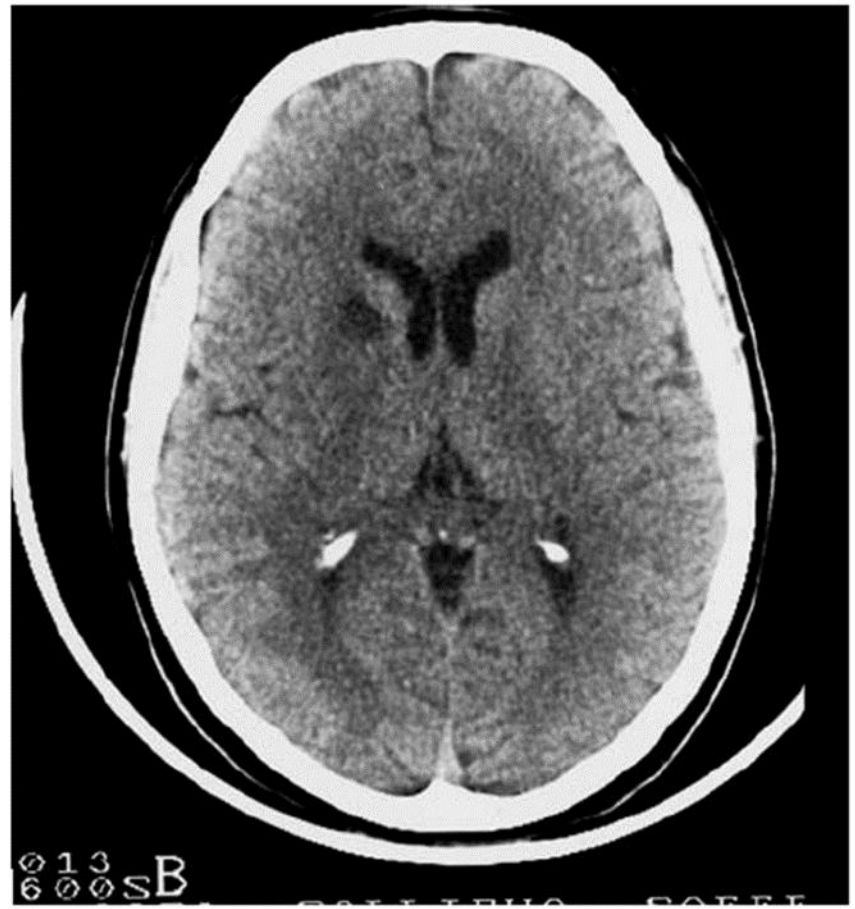
arteria perforante: ictus lacunare



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Esplorazione della forza muscolare (distribuzione)

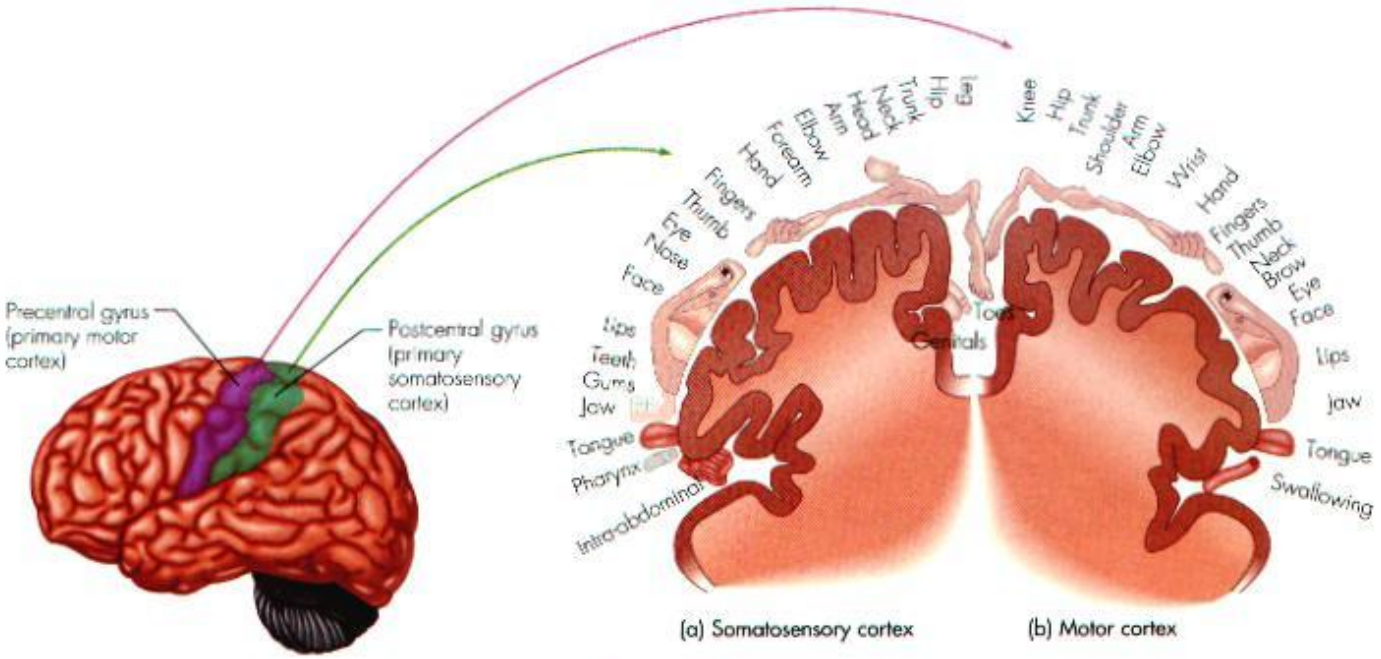
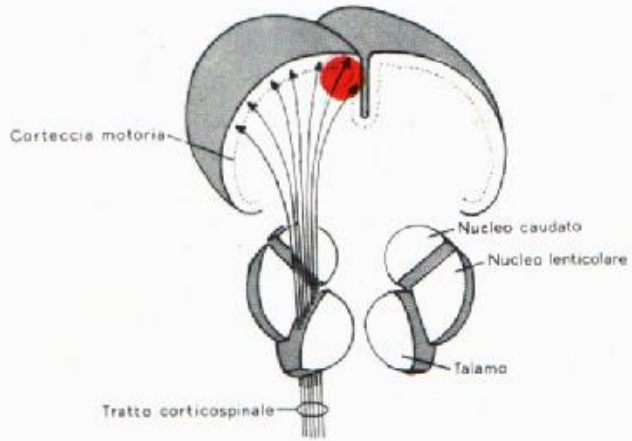


Figure 4.21 Approximate representation of sensory and motor information in the cortex
(**a**) Each location in the somatosensory cortex represents sensation from a different body part. (**b**) Each location in the motor cortex regulates movement of a different body part. *(Source: After Penfield & Rasmussen, 1950)*

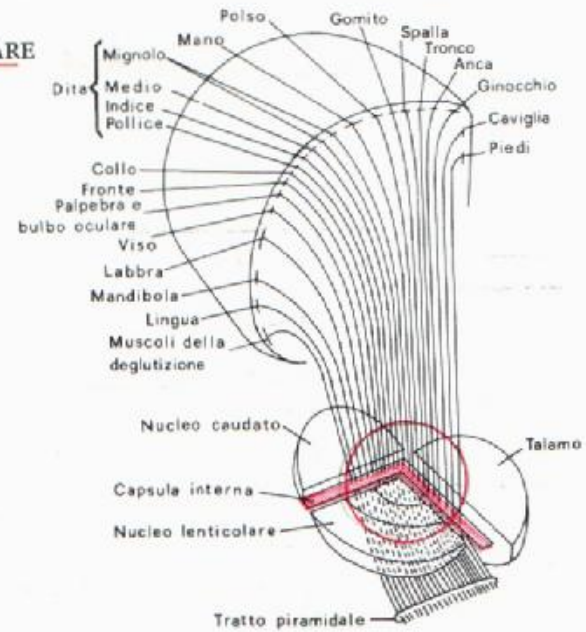
LESIONE CORTICALE

monoplegia



LESIONE CAPSULARE

emiplegia



E’ riconoscibile una sindrome clinica?

Bamford Stroke Classification

Lancet 337:1521-1526, 1991

TACS

PACS

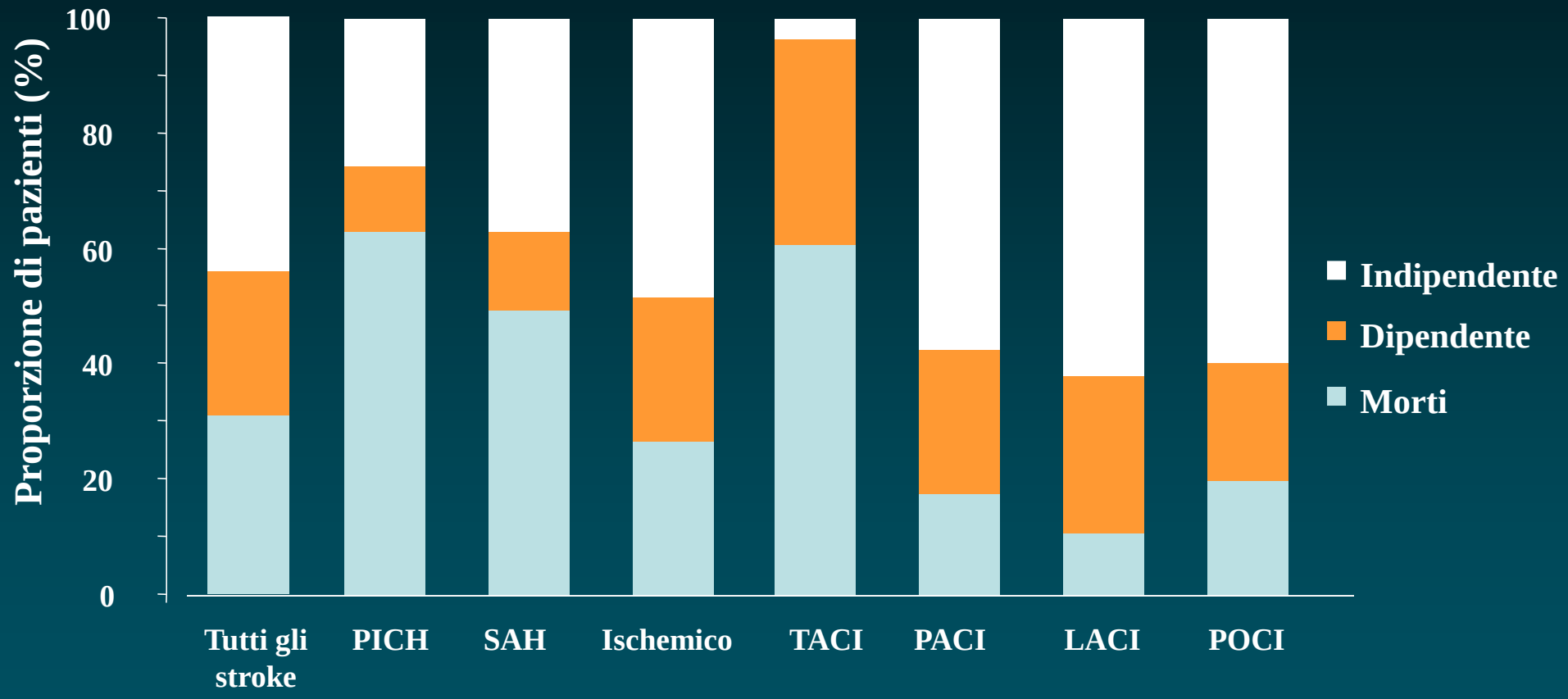
POCS

LACS

Table II. Stroke subtyping based on maximum deficit[4].

Total anterior circulation infarction (TACI)
- motor and sensory deficit, ipsilateral hemianopia and disturbance of higher cerebral function
Partial anterior circulation infarction (PACI)
- any two of the above
- or isolated disturbances of higher cerebral function
Posterior circulation infarct (POCI)
- unequivocal signs of brain stem disturbance
- or isolated hemianopia
Lacunar infarction (LACI)
- pure motor stroke
- or pure sensory stroke
- or pure sensorimotor stroke
- or ataxic hemiparesis

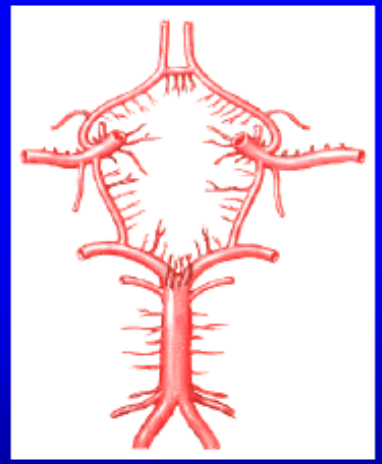
Proporzione di pazienti morti o dipendenti ad un anno dall'ictus



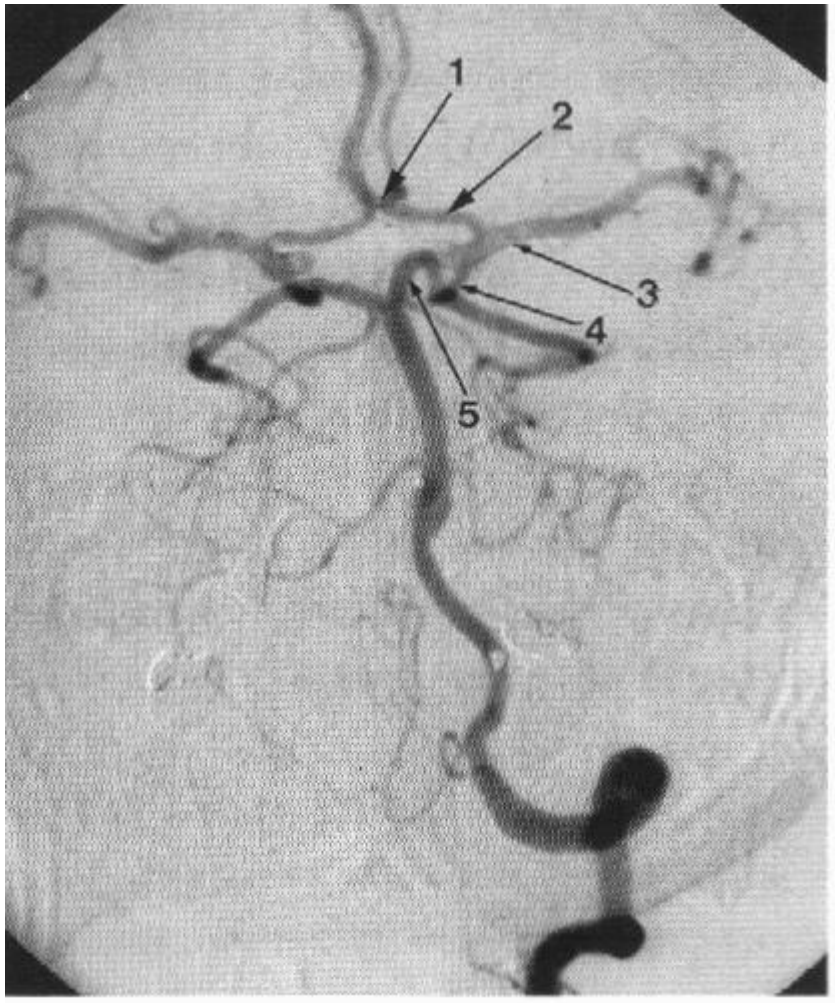
Warlow et al, Lancet 2003

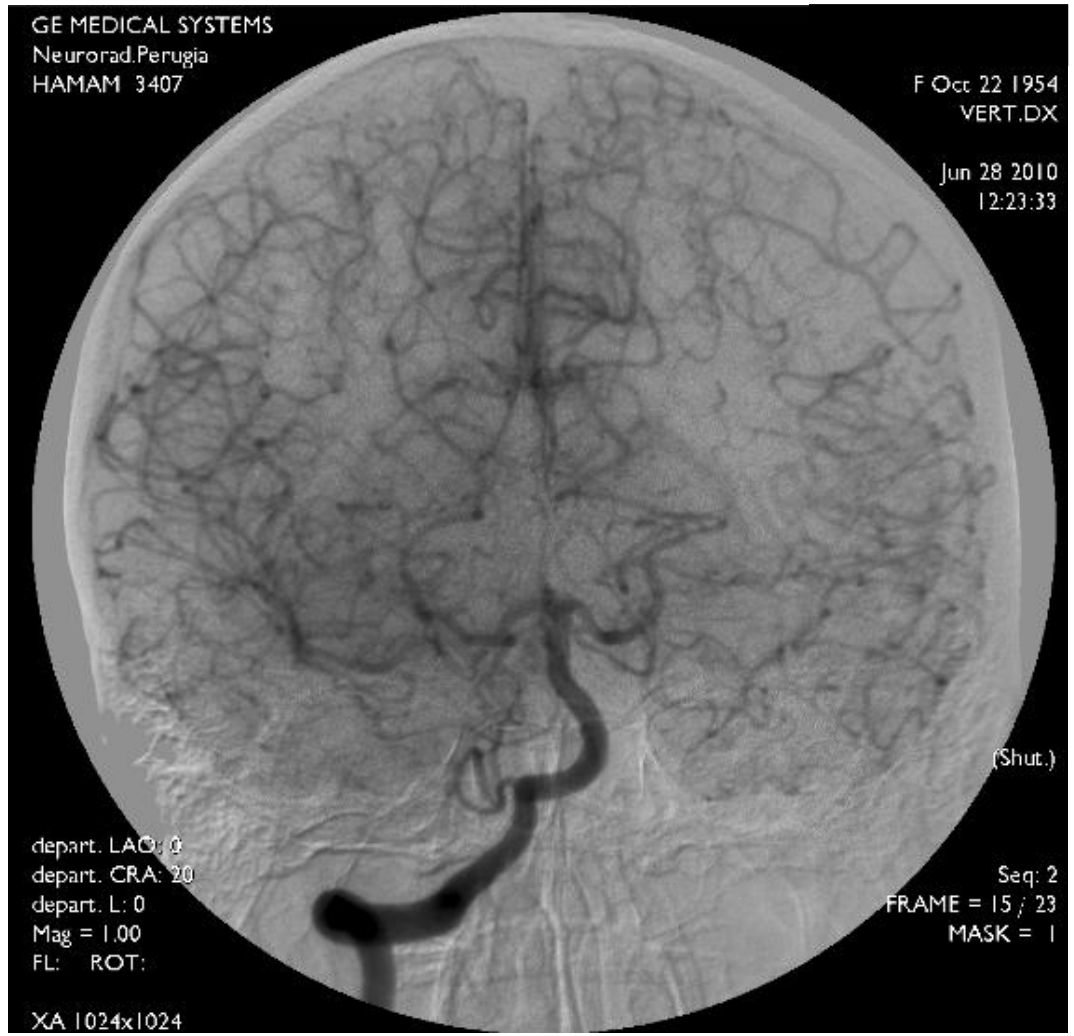
Circle of Willis

- Vascular ring located between the mesencephalon and the anterior perforated substance
- Connection between carotid circulations with vertebrobasilar circulation.
- Formed by 10 arteries:
  - 2 ICA
  - 2 PCom
  - 1 AComAnt
  - 2 A1 segments of ACAs
  - 2 P1 segments of PCAs
  - 1 basilar artery
- Complete circle only in 50% of cases,
- Frequent variants (hypoplasia or absence of segments).



Poligono Willis




GE MEDICAL SYSTEMS
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Jun 28 2010
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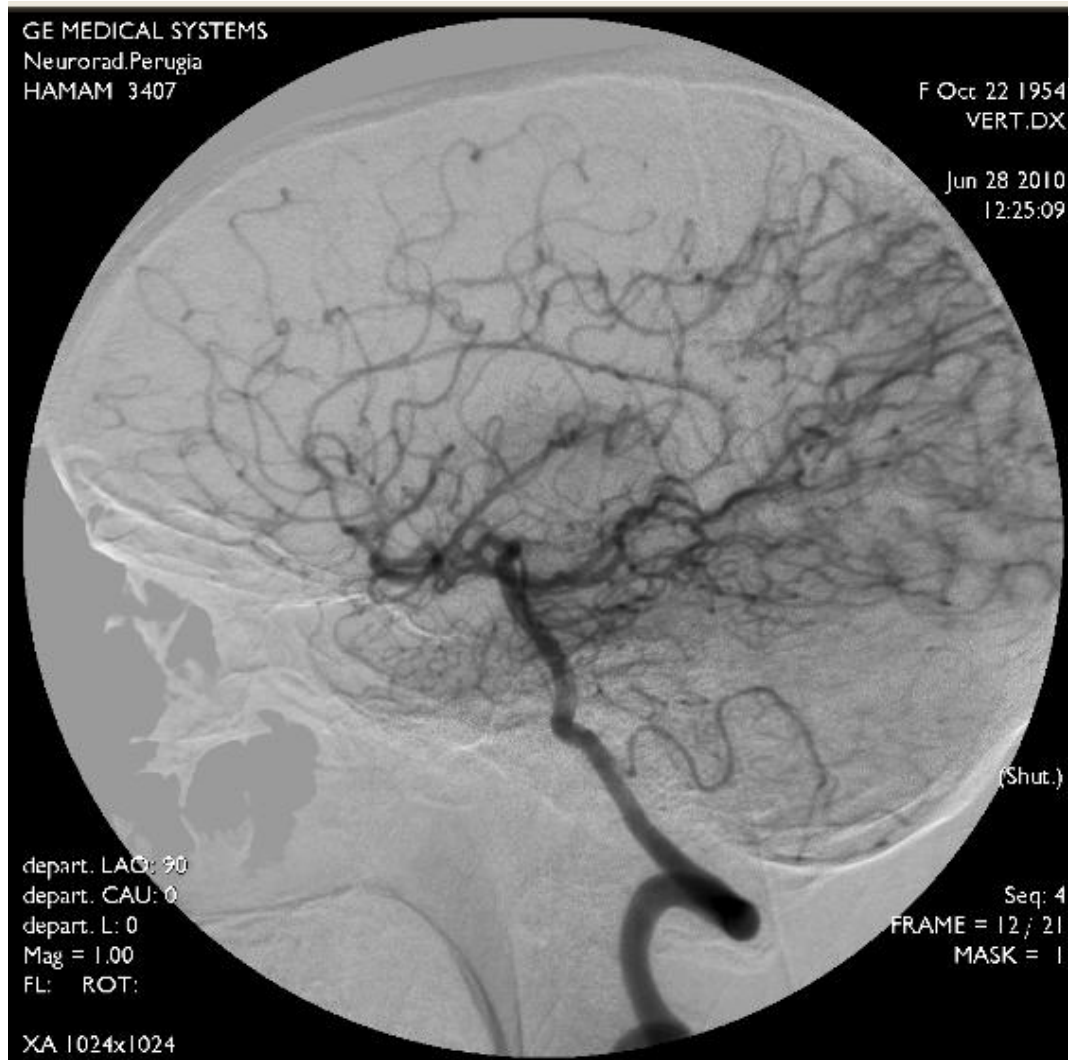
GE MEDICAL SYSTEMS
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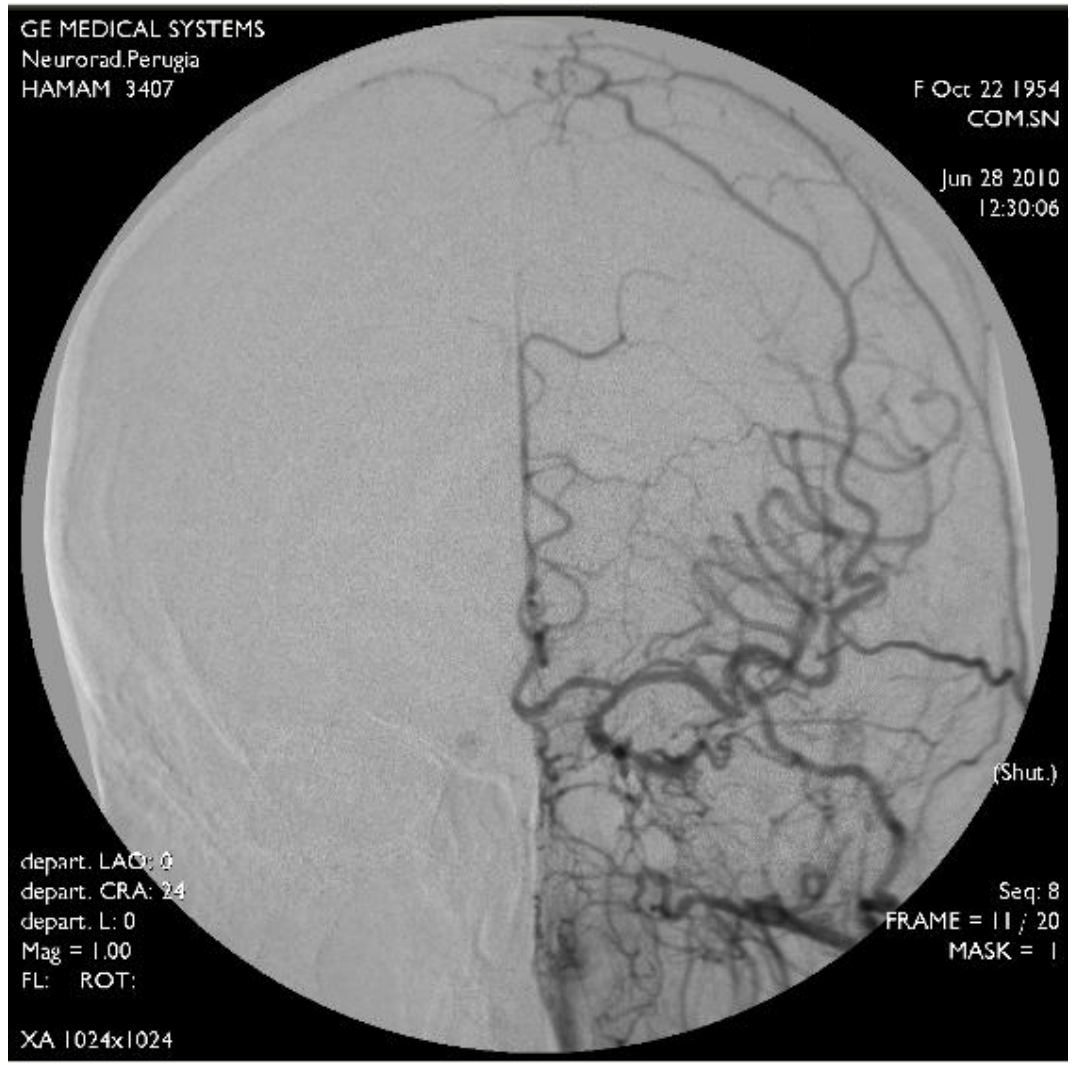
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depart. L: 0
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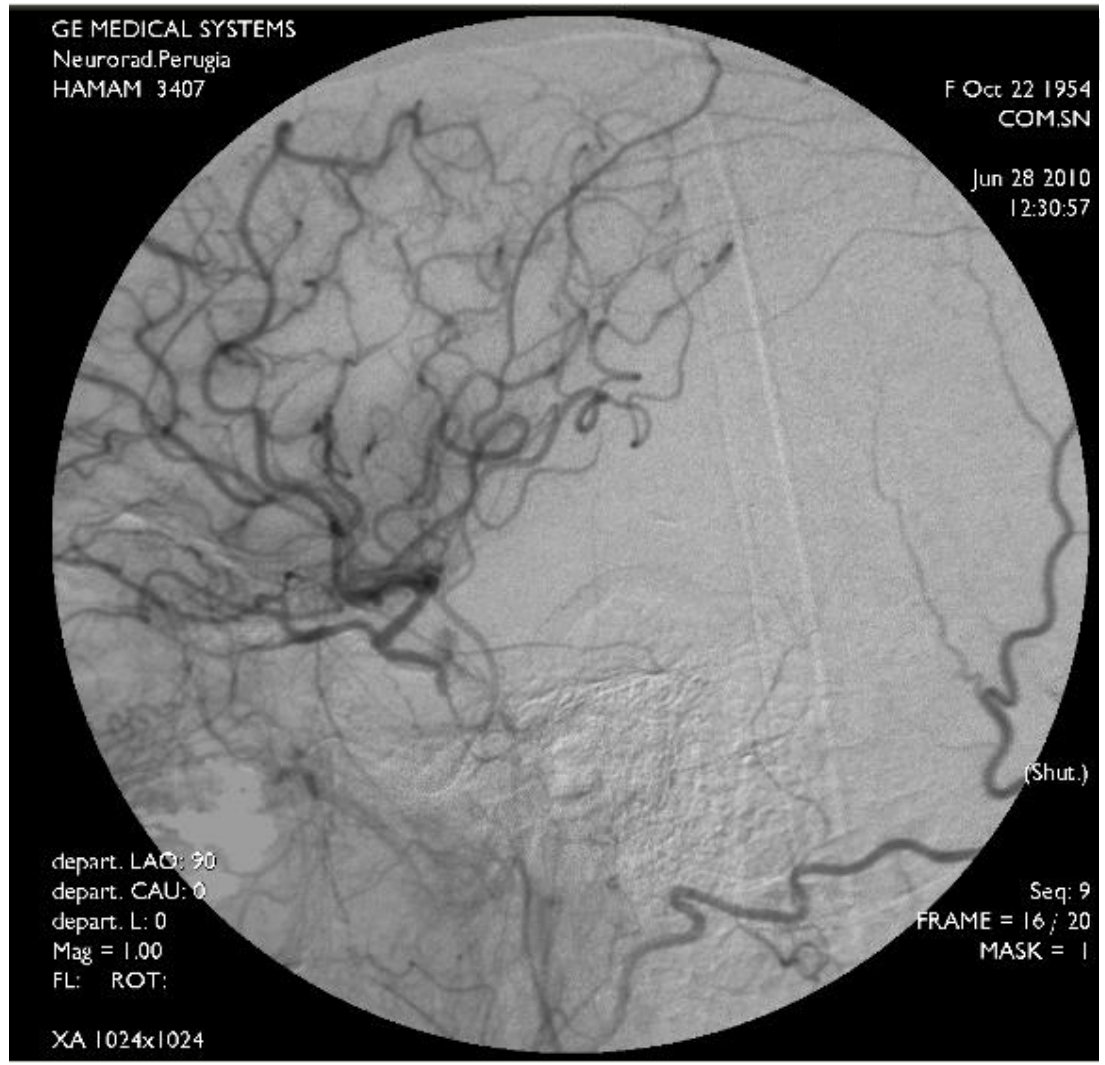
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Terapia dello stroke acuto

- Terapia di riperfusione
- Profilassi secondaria precoce
- Terapia delle complicanze precoci e tardive (es. antiedemigena, anti trombosi venose profonde)

Table 4. Prehospital Evaluation and Management of Potential Stroke Patients

Recommended	Not Recommended
Assess and manage ABCs	Do not initiate interventions for hypertension unless directed by medical command
Initiate cardiac monitoring	
Provide supplemental oxygen to maintain O_2 saturation >94%	
Establish IV access per local protocol	Do not administer excessive IV fluids
Determine blood glucose and treat accordingly	Do not administer dextrose-containing fluids in nonhypoglycemic patients
	Do not administer medications by mouth (maintain NPO)
Determine time of symptom onset or last known normal, and obtain family contact information, preferably a cell phone	
Triage and rapidly transport patient to nearest most appropriate stroke hospital	Do not delay transport for prehospital interventions
Notify hospital of pending stroke patient arrival	

ABCs indicates airway, breathing, and circulation; IV, intravenous; and NPO, nothing by mouth.

Table 8. Immediate Diagnostic Studies: Evaluation of a Patient With Suspected Acute Ischemic Stroke

All patients
Noncontrast brain CT or brain MRI
Blood glucose
Oxygen saturation
Serum electrolytes/renal function tests*
Complete blood count, including platelet count*
Markers of cardiac ischemia*
Prothrombin time/INR*
Activated partial thromboplastin time*
ECG*

Stroke Unit

- ✓ ***Struttura dedicata***
- ✓ ***Team multidisciplinare competente e dedicato alle malattie cerebrovascolari***

  - medici
  - infermieri
  - terapisti della riabilitazione

aggiornamento continuo del team

Stroke Unit

- ✓ ***Possibilità di monitorizzare:***
  - ***pressione arteriosa***
  - ***attività cardiaca***
  - ***saturazione arteriosa dell'ossigeno***
  - ***temperatura corporea***
- ✓ ***Disponibilità di presidi per prevenire complicanze secondarie***

Stroke Unit

- ✓ *Disponibilità di TC encefalo 24/7*
- ✓ *Disponibilità di laboratorio analisi 24/7*
- ✓ *Disponibilità di ecocolordoppler per studio vasi epiaortici*

Stroke Unit

- ✓ *Acute Stroke Unit*
- ✓ *Acute and Rehabilitation Stroke Unit*
- ✓ *Rehabilitation Stroke Unit*
- ✓ *Mobile Stroke Team*

bmj.com

BMJ 1997; 314: 1151-1159.

A collaborative systematic review of the randomized trials of organized inpatient (stroke unit) care after stroke

N = 3149

mortalità ↓ ***18%***

morte/dipendenza ↓ ***29%***

morte/necessità di istituzionalizzazione ↓ ***25%***

Stroke Unit Trialist's Collaboration

Cardiac care - Concepts

- Cardiac complications more frequent in ICH and SAH than in ischemic stroke (Oppenheimer, Neurol Clin N Am 1992;10:167)
- 15-40% of stroke patients may experience (Bamford, JNNP 1990;53:824; Broderick, Stroke 1992;23:1250; Vingerhoets, Stroke 1993;24:26)
  - AMI
  - congestive heart failure
  - arrhytmias, particularly AF
  - sudden death
- Significant correlation between infarcts of the insular cortex and cardiac complications (Kaste, Cerebrovasc Dis 2000;10(suppl 3):1)

Respiratory care – Concepts

- Adequate oxygenation is important to preserve the penumbra
- No data favour routine O_2 administration to all stroke patients

  (Ronning OM, Stroke 1999;30:2033)
- Most common causes of hypoxia in stroke
  - previous pulmonary diseases
  - airway obstruction (vomiting, oropharingeal muscular hypotonia)
  - acute aspiration (brainstem stroke; reduced vigilance)
  - hypoventilation due to
    - large hemispheric infarct or hemorrhage
    - brainstem infarct or hemorrhage
    - hearth failure
    - pulmonary embolism
    - status epilepticus

Body temperature – Concepts - 1

- Experimentally fever increases infarct size (Fukuda H, Acta Neurol Scand 1999;100:385)
- Body temperature increases in up to 50% of patients (Corbett D, Brain Pathol 2000;10:145)
- High body temperature may favor stroke progression (Castillo J, Cerebrovasc Dis 1999;9:22) and long term bad outcome (Hajat C, Stroke 2000;31:410)

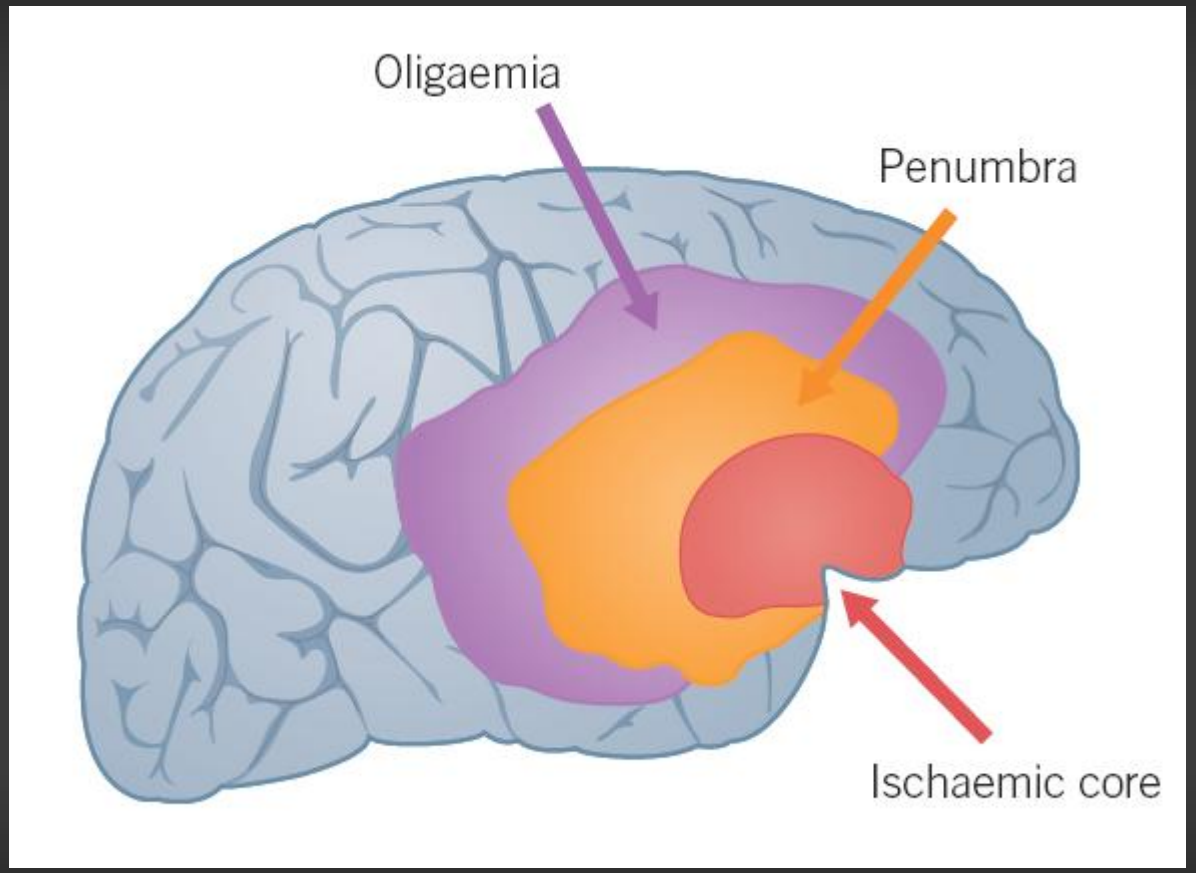
Dysphagia

- Present in up to 50% of patients
- Predictor of poor prognosis
- Risk for aspiration & pneumonia
- Malnourishion
- Dehydration with plasma volume contraction

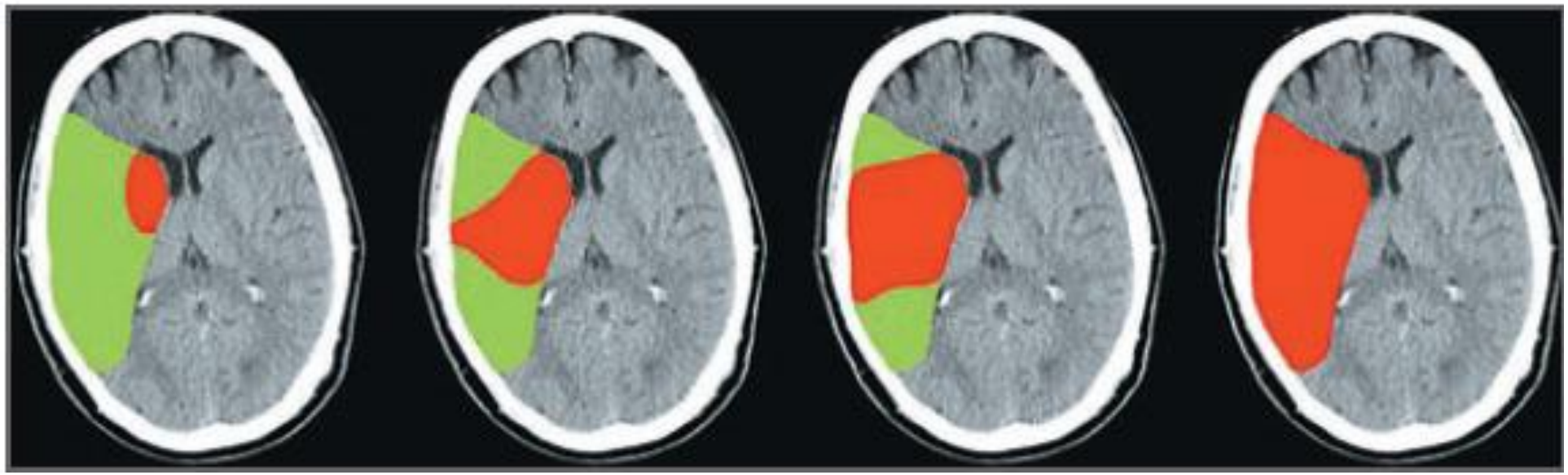
(Martino R, Stroke 2005;36:2756)

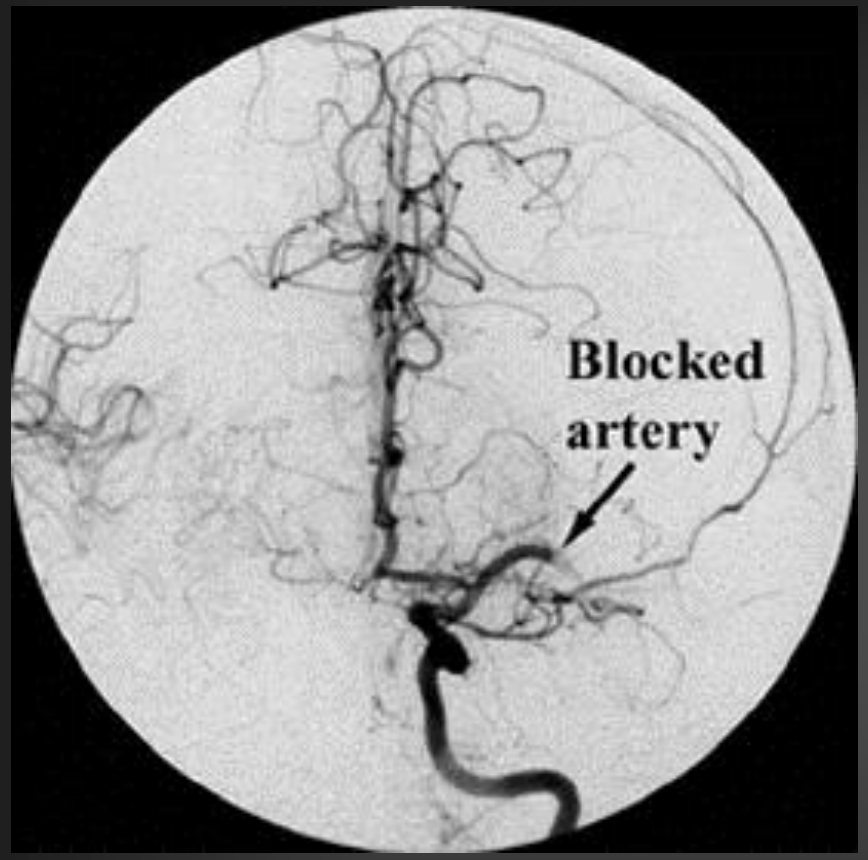
Terapia di riperfusione

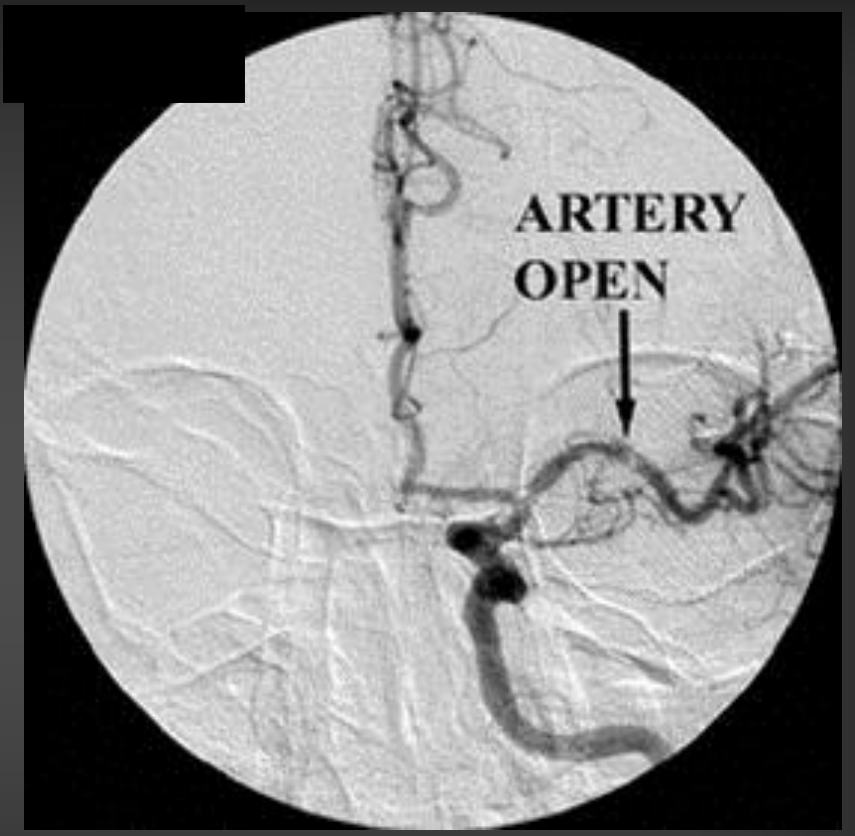
La terapia trombolitica


Oligaemia
Penumbra
Ischaemic core

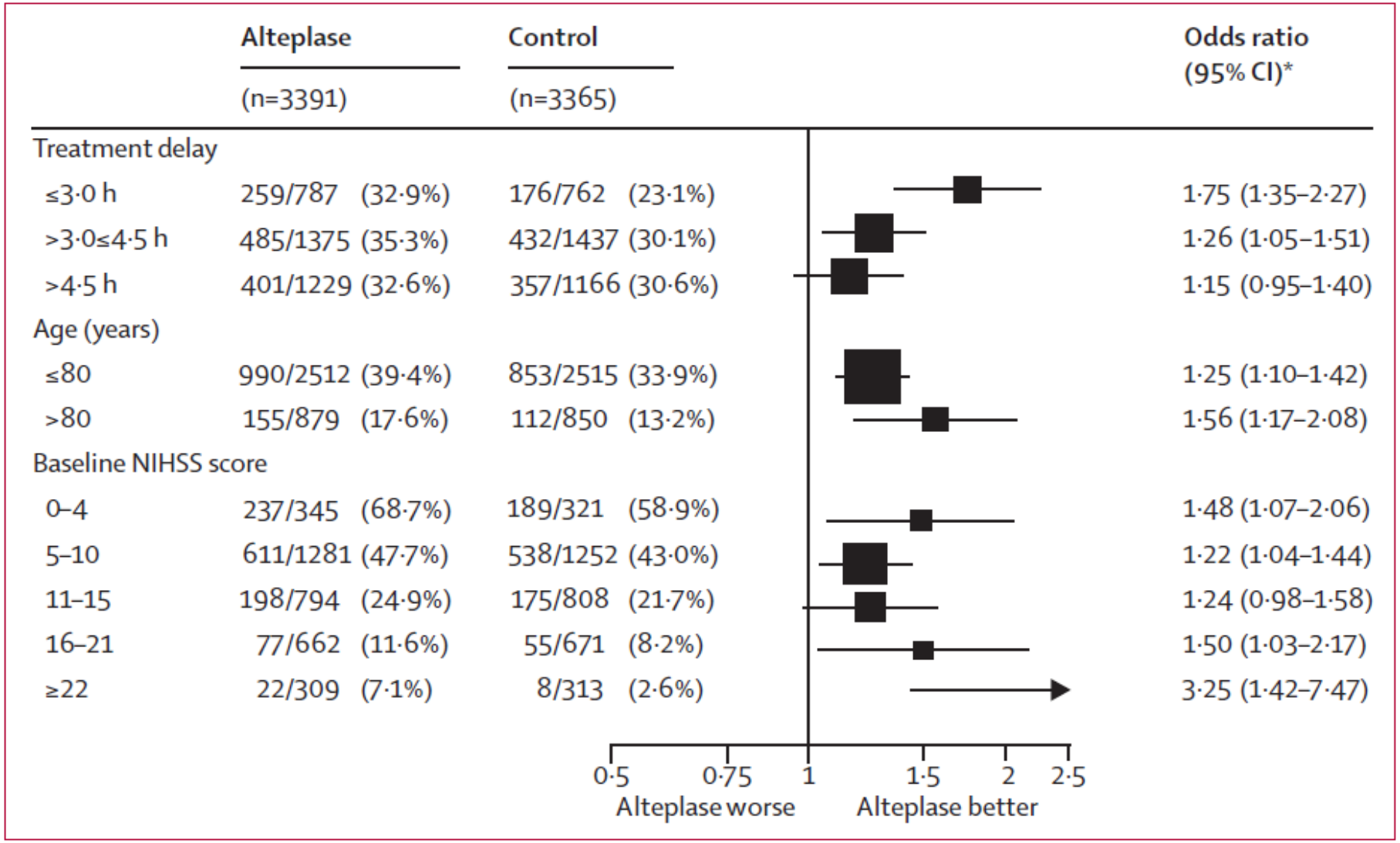
Reperfusion therapy




Blocked
artery

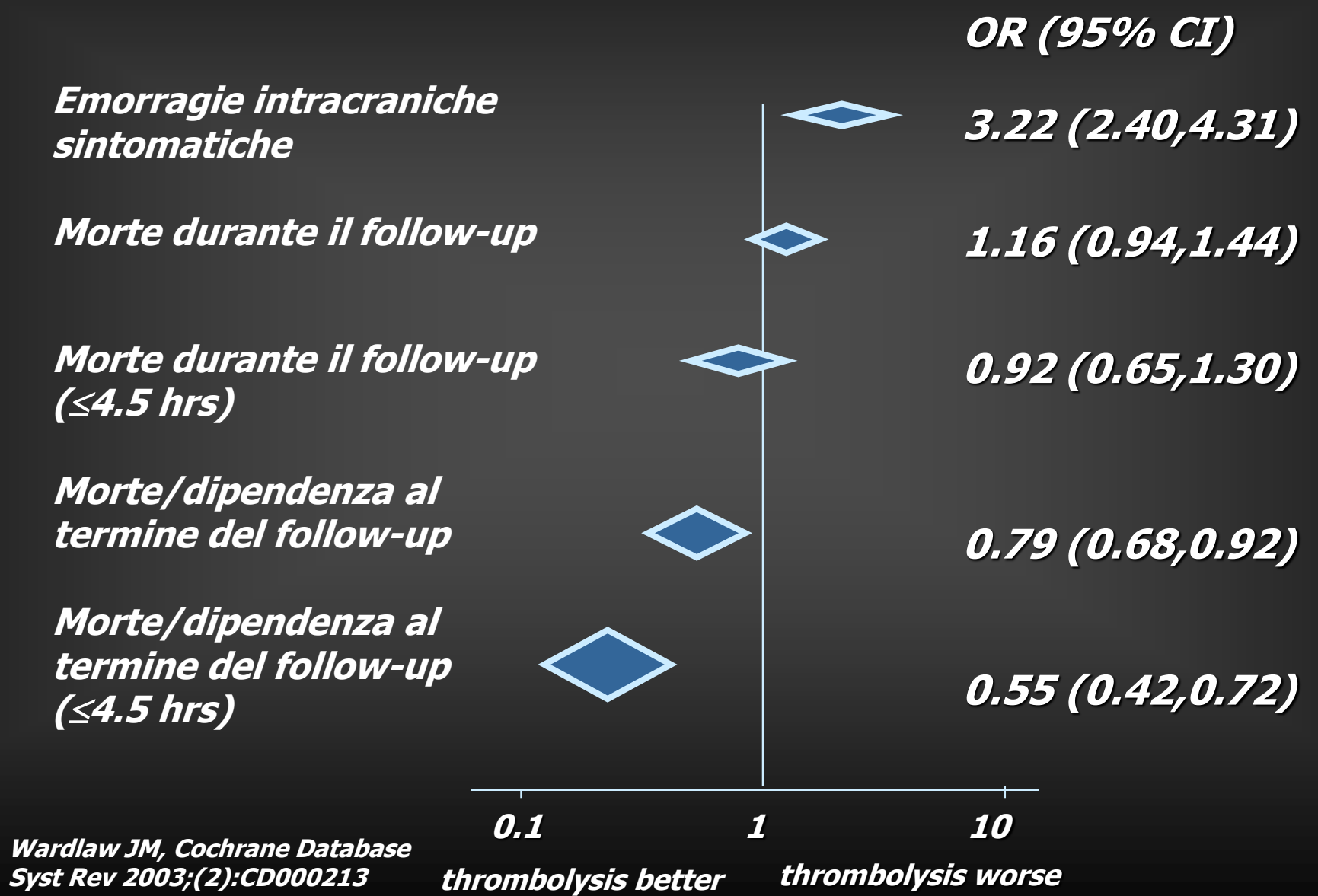

ARTERY
OPEN

Effect of rtPA on good stroke outcome (mRS 0–1)



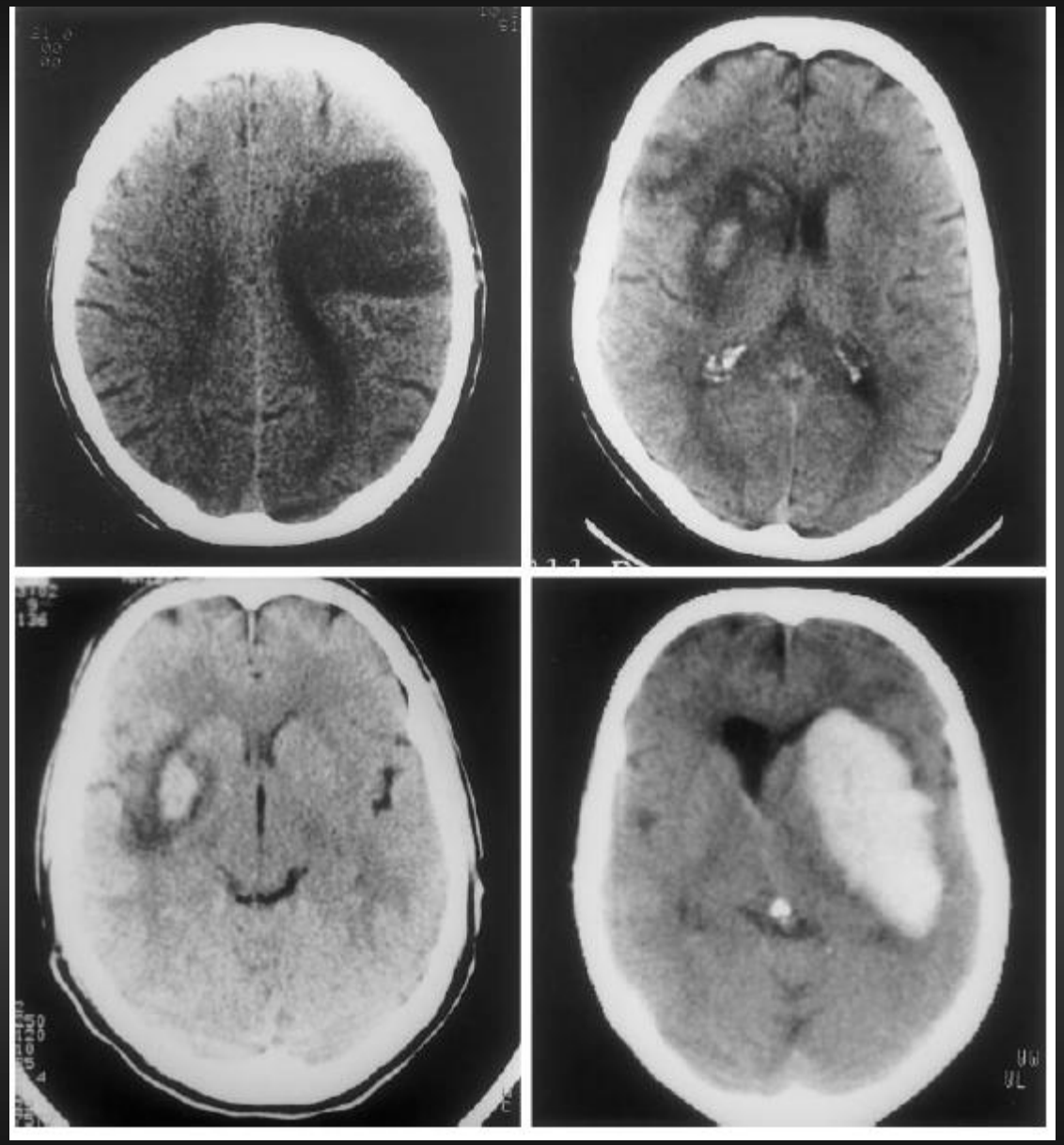
Lancet. 2014 Aug 5.

Trials con rt-PA (N=2955)



Wardlaw JM, Cochrane Database Syst Rev 2003;(2):CD000213

Infarcimento emorragico



Raccomandazione ***Grado A***

L'rt-PA e.v. (0,9 mg/kg, dose massima 90 mg, il 10% della dose in bolo, il rimanente in infusione di 60 minuti) è indicato entro 4,5 ore dall'esordio di un ictus ischemico

Criteri di esclusione

- Diatesi emorragica nota
- Pz. in TAO
- Sanguinamento in atto o recente grave
- Storia o sospetto di emorragia intracranica in atto
- Storia di patologie del SNC (neoplasia, aneurisma, intervento chirurgico cerebrale o midollare)
- recenti (<10 giorni) massaggio cardiaco esterno traumatico, parto, puntura di vaso sanguigno non comprimibile (es. vena succlavia o giugulare).

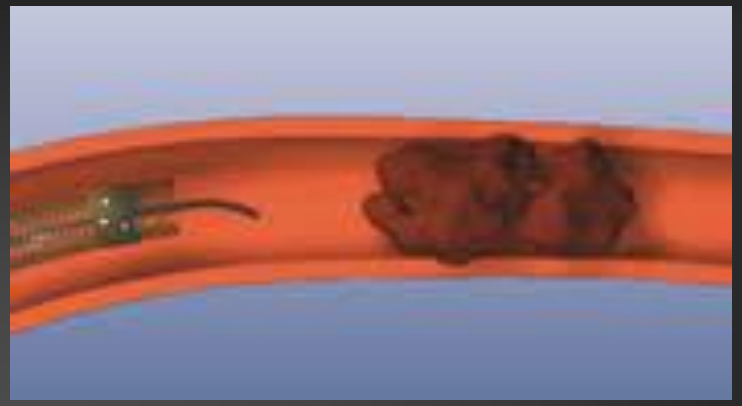
Altri criteri di esclusione

- Endocardite batterica, pericardite, pancreatite acuta malattia ulcerosa del tratto gastroenterico (< 3mesi)
- Aneurisma arterioso, malformazione artero-venosa
- Neoplasia con aumentato rischio emorragica
- Grave epatopatia, compresa insufficienza epatica, cirrosi, ipertensione portale (varici esofagee), epatite attiva
- Intervento chirurgico maggiore o grave trauma (<3 mesi)
- Insorgenza dell'ictus > 4,5 ore o ora di insorgenza non nota
- Deficit lieve o rapido miglioramento dei sintomi
- Ictus grave clinicamente (es. NIHSS >25) e/o sulla base di adeguate tecniche di neuroimmagini

Table 12. Treatment of Acute Ischemic Stroke: Intravenous Administration of rtPA

Infuse 0.9 mg/kg (maximum dose 90 mg) over 60 minutes, with 10% of the dose given as a bolus over 1 minute.
Admit the patient to an intensive care or stroke unit for monitoring.
If the patient develops severe headache, acute hypertension, nausea, or vomiting or has a worsening neurological examination, discontinue the infusion (if IV rtPA is being administered) and obtain emergent CT scan.
Measure blood pressure and perform neurological assessments every 15 minutes during and after IV rtPA infusion for 2 hours, then every 30 minutes for 6 hours, then hourly until 24 hours after IV rtPA treatment.
Increase the frequency of blood pressure measurements if systolic blood pressure is >180 mm Hg or if diastolic blood pressure is >105 mm Hg; administer antihypertensive medications to maintain blood pressure at or below these levels (Table 8).
Delay placement of nasogastric tubes, indwelling bladder catheters, or intra-arterial pressure catheters if the patient can be safely managed without them.
Obtain a follow-up CT or MRI scan at 24 hours after IV rtPA before starting anticoagulants or antiplatelet agents.

CT indicates computed tomography; IV, intravenous; MRI, magnetic resonance imaging; and rtPA, recombinant tissue plasminogen activator.



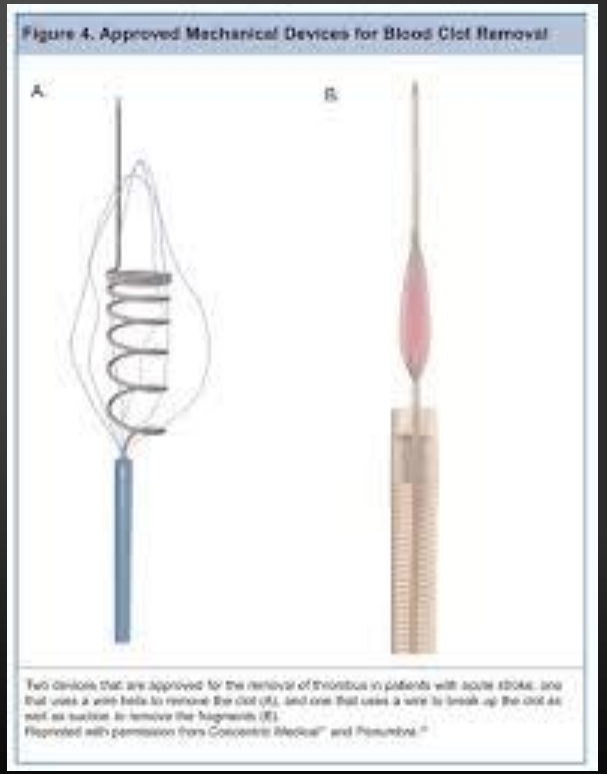
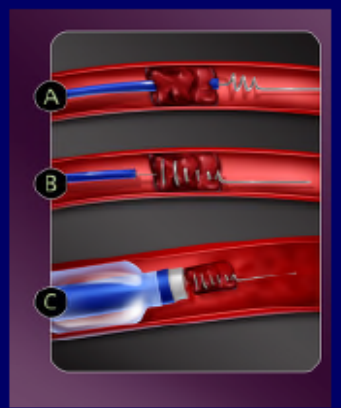


Figure 4. Approved Mechanical Devices for Blood Clot Removal
A.
B.
Two devices that are approved for the removal of thrombus in patients with acute stroke, one that uses a wire helix to remove the clot (A), and one that uses a wire to break up the clot as well as suction to remove the fragments (B).
Reprinted with permission from Concentric Medical™ and Penumbra.™

Mechanical Recanalization

- Thrombectomy, clot-retrieval devices
- Thromboaspiration
- Thrombus obliteration devices, angioplasty
- Laser thrombolysis
- Ultrasound thrombolysis

All techniques can be combined with pharmacological thrombolysis

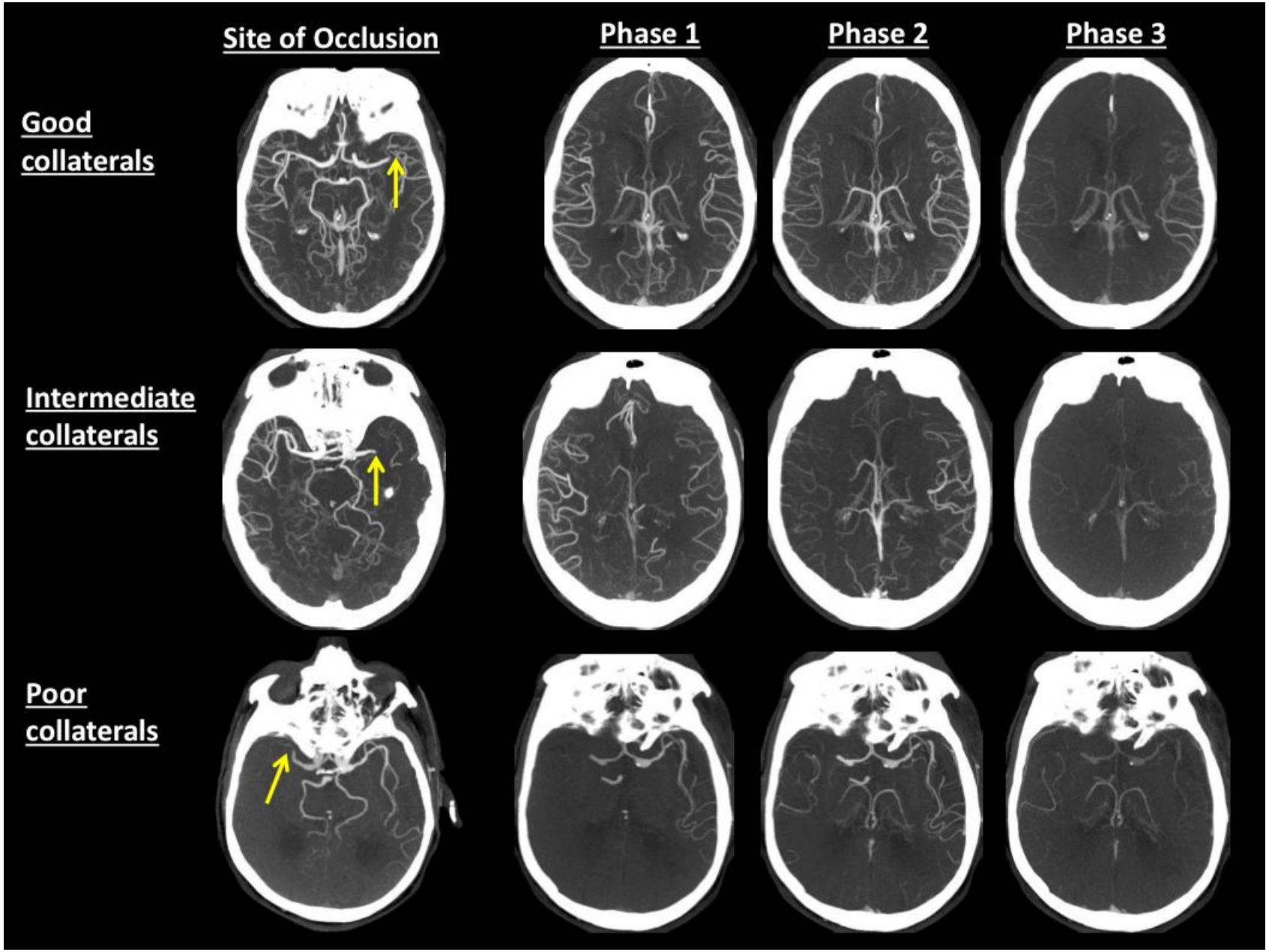


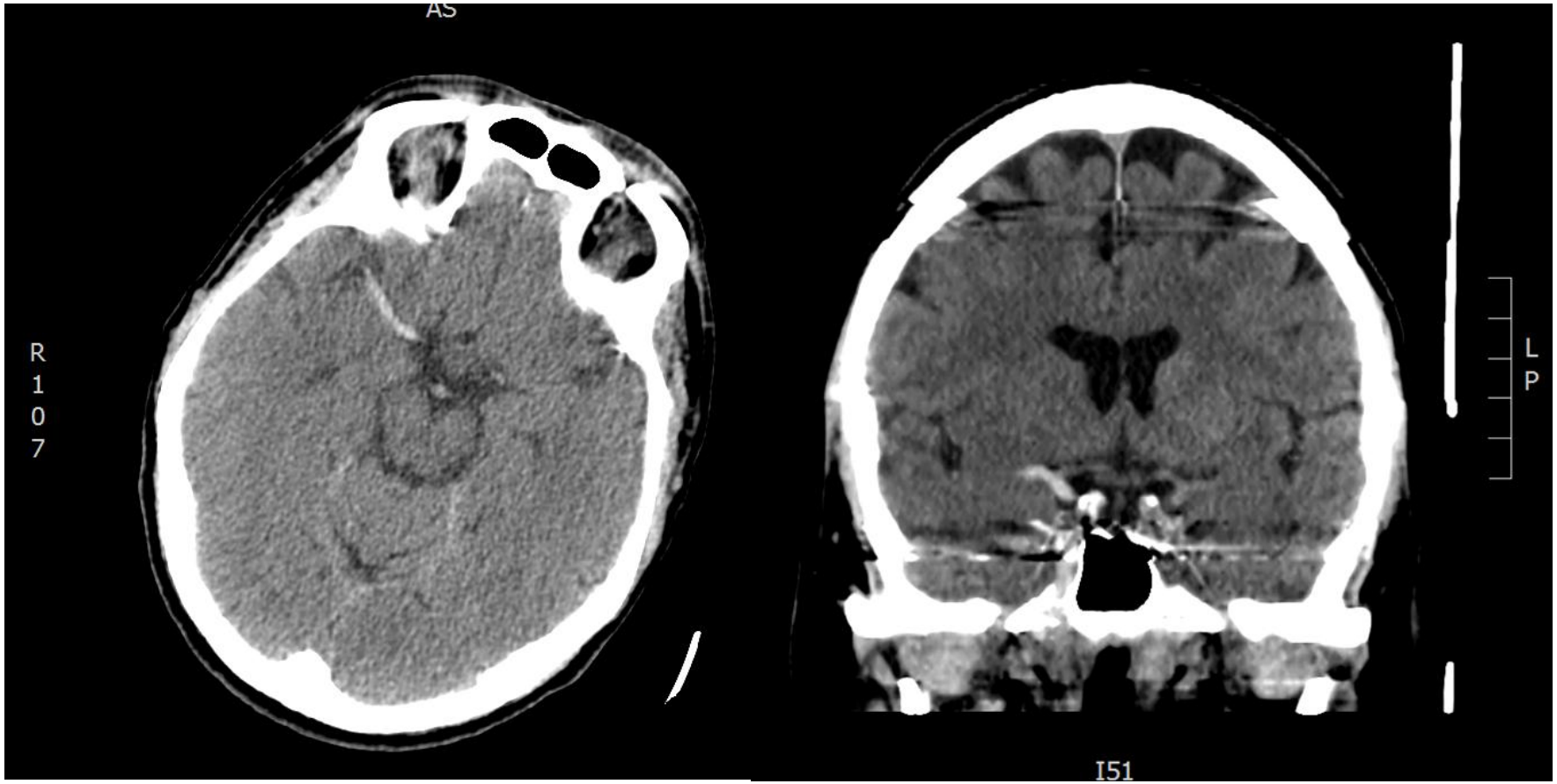
Overview of thrombectomy trials published in 2015:
Main inclusion criteria and baseline characteristics

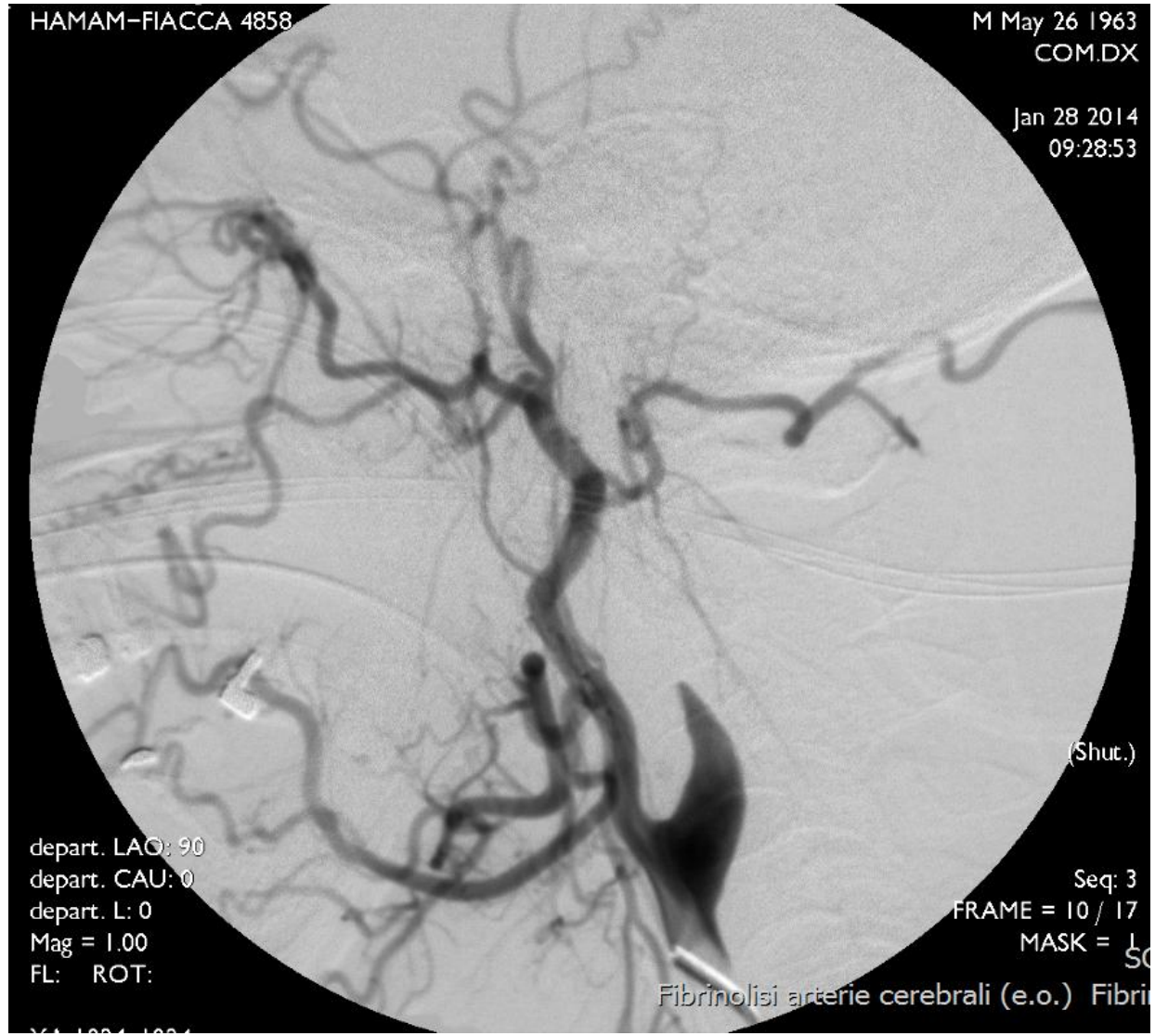
Study	Age	Time	Imaging	Inclusion NIHSS	Pat (n)	Median NIHSS T/C	IVT (%)
MR CLEAN (The Netherlands)	≥ 18	6 h	CT/CTA MR/MRA	≥ 2	500	17/18	89%
ESCAPE (US, Canada, UK, South Korea)	≥ 18	12 h	CT/CTA/ multiphase CTA	> 5	316	17/16	72.7%
EXTEND-IA (Australia, New Zealand)	≥ 18	6 h	CT/MR CTA/ CTP	0-42	70	17/13	100%
SWIFT PRIME (USA/EU)	18-85	6 h	CT MR DWI/PWI (Rapid) CTA/MRA	8-29	196	17/17	100%
REVASCAT (Catalonia)	18-80	8 h	CT/MR DWI CTA/MRA	6	206	17/17	72.8%

Patient selections

- Onset 6 h
- Occlusion of a proximal vessel (ICA T-L, M1) by angio-CT
- Collateral circulations


Site of Occlusion
Phase 1
Phase 2
Phase 3
Good collaterals
Intermediate collaterals
Poor collaterals


AS
R
1
0
7
L
P
I51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depart. L: 0
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Fibrinolisi arterie cerebrali (e.o.) Fibrin


Neurorad Perugia
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P119692
M May 26 1963
INT.SN
Jan 28 2014
09:42:58
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depart. CAU: 2
depart. L: 0
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CONTROLLO
Jan 28 2014
10:33:46
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depart. L: 0
Mag = 1.00
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FRAME = 9 / 10
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HAMAM-FIACCA 4858
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CONTROLLO
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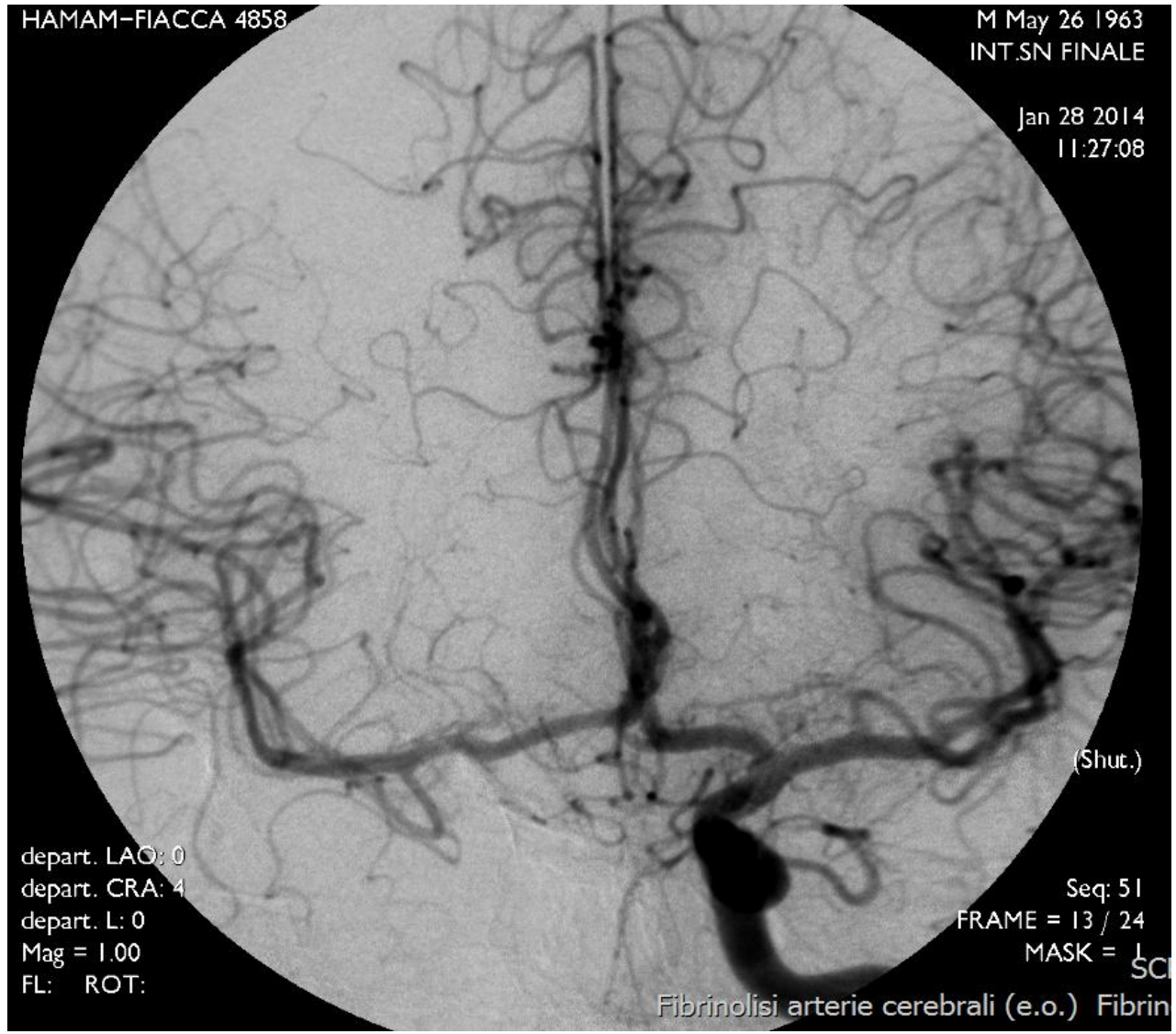
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depart. CRA: 4
depart. L: 0
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FL: ROT:
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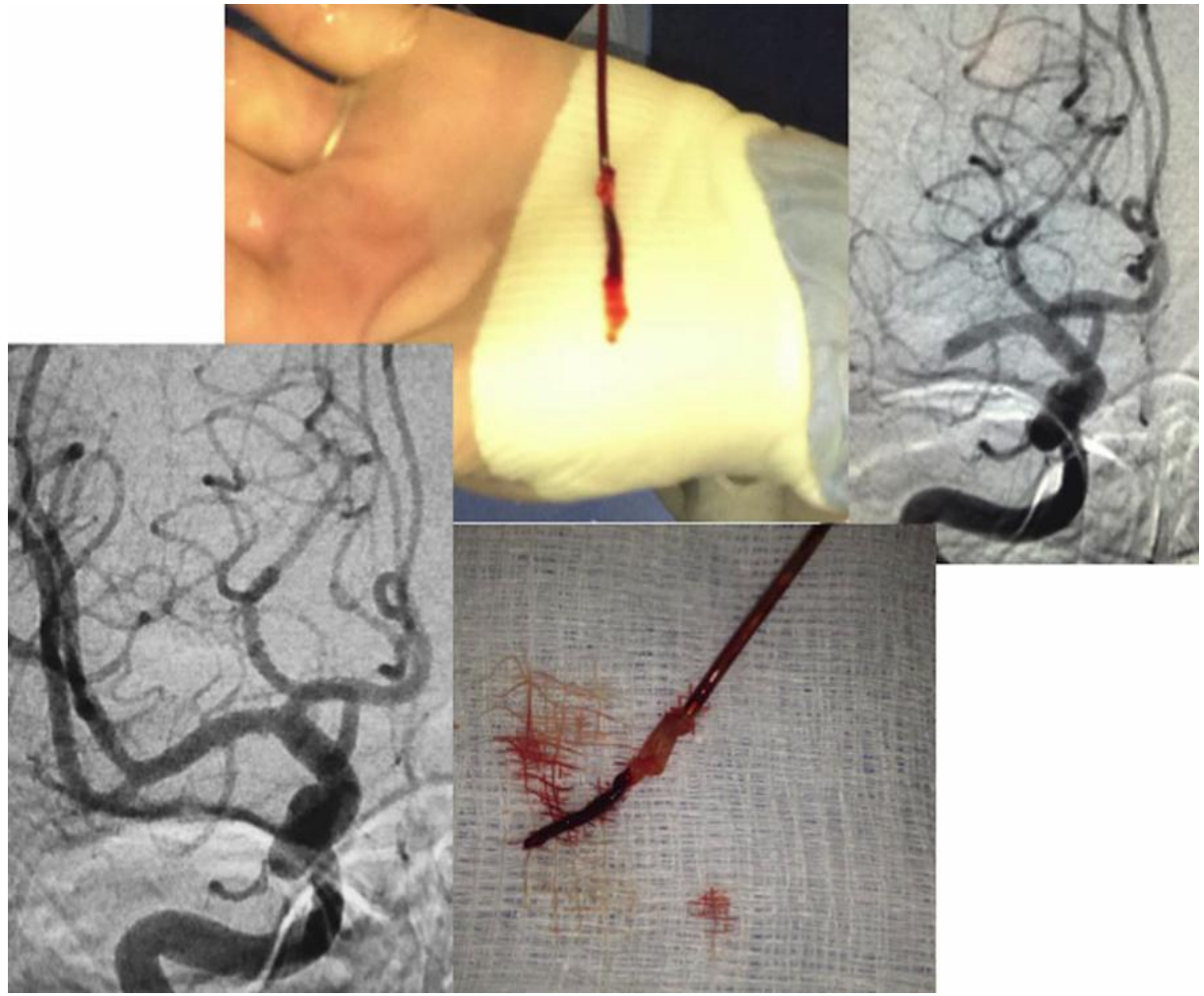
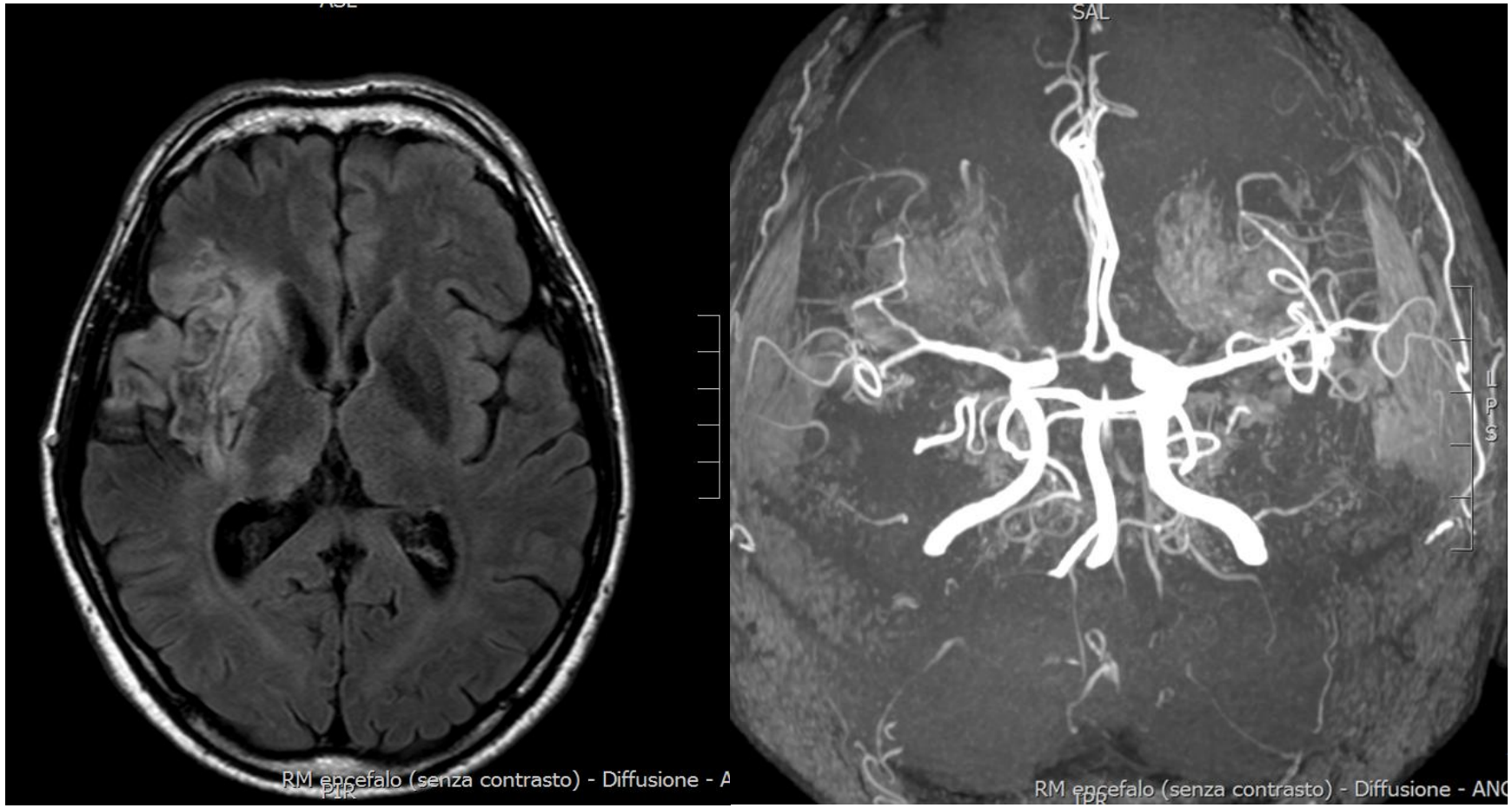
FRAME = 13 / 24
MASK = 1
Fibrinolisi arterie cerebrali (e.o.) Fibrin

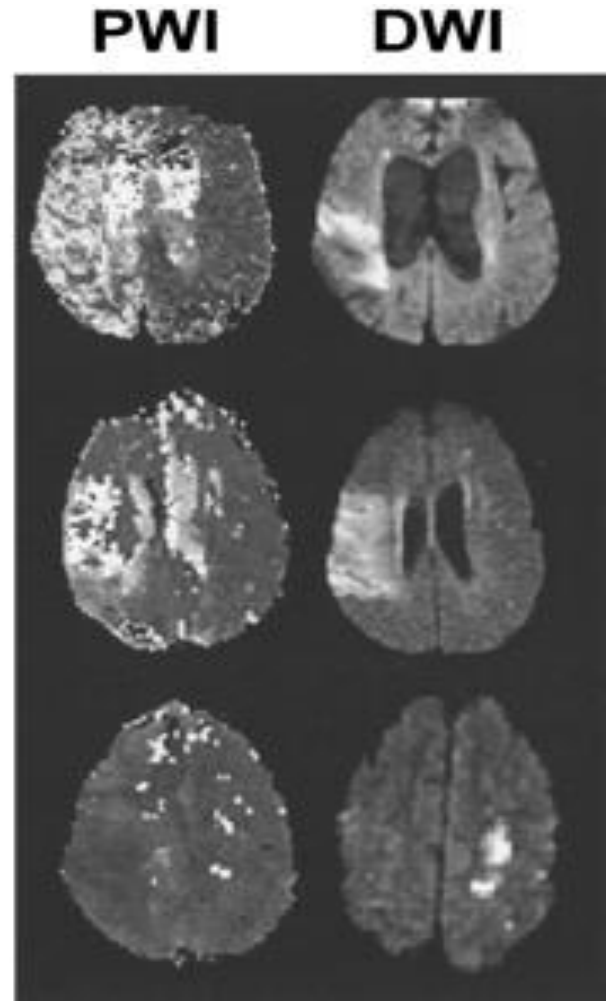



RM encefalo (senza contrasto) - Diffusione - A
RM encefalo (senza contrasto) - Diffusione - ANG

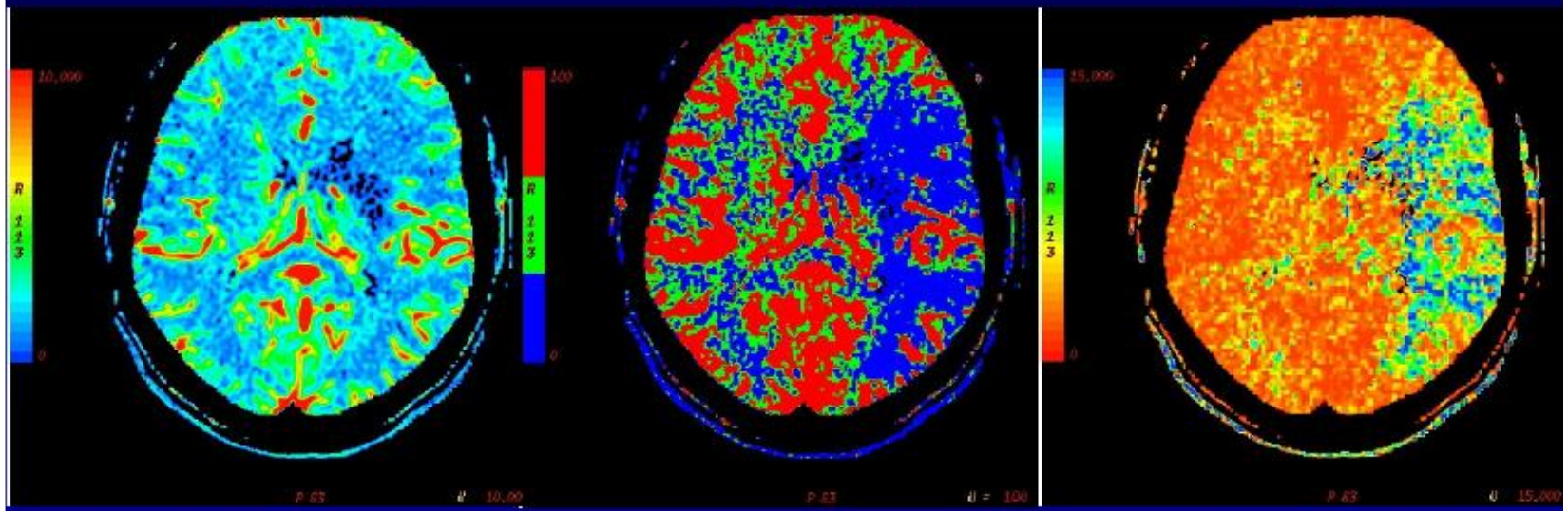
PWI lesion > DWI lesion

PWI lesion = DWI lesion

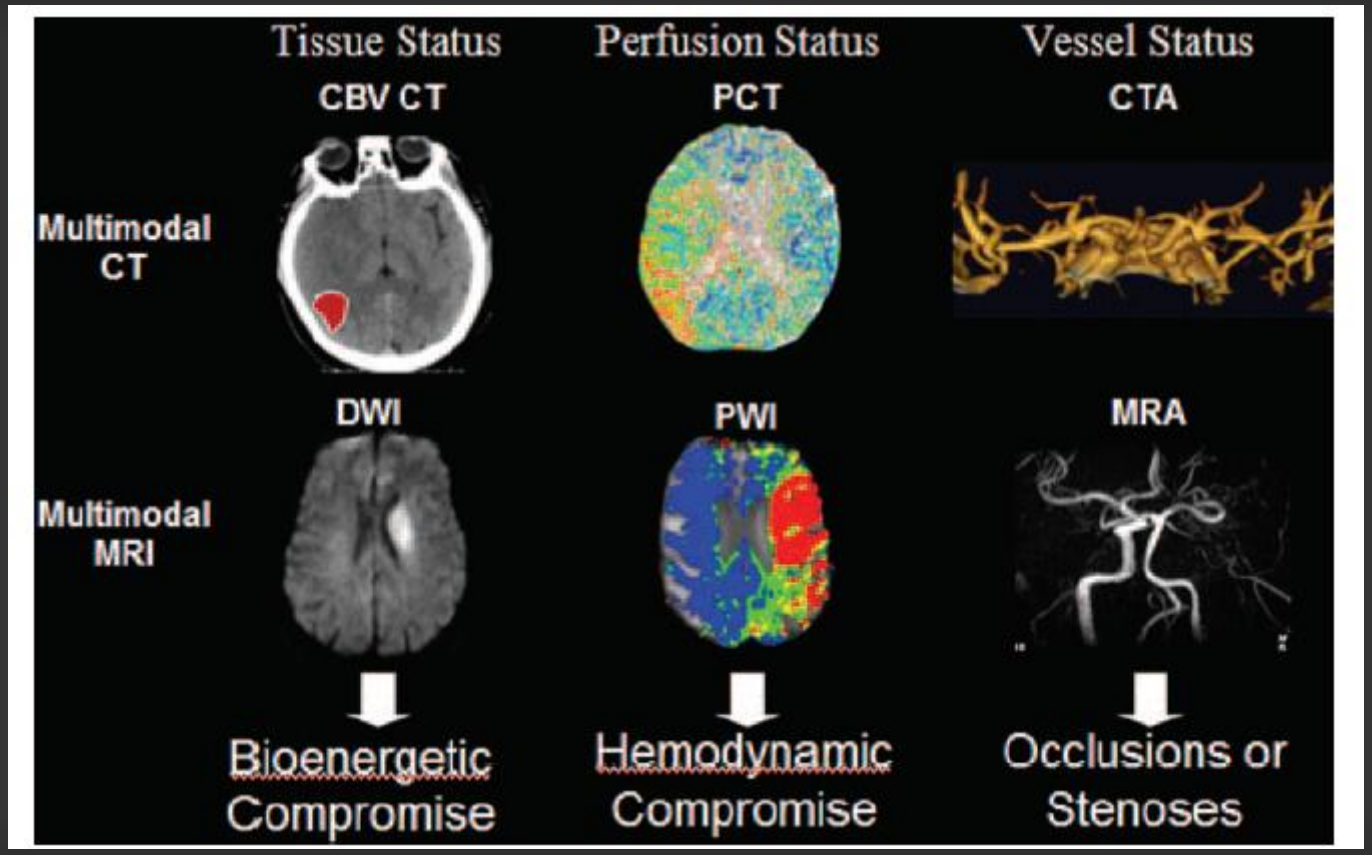
PWI lesion < DWI lesion



Perfusion Maps



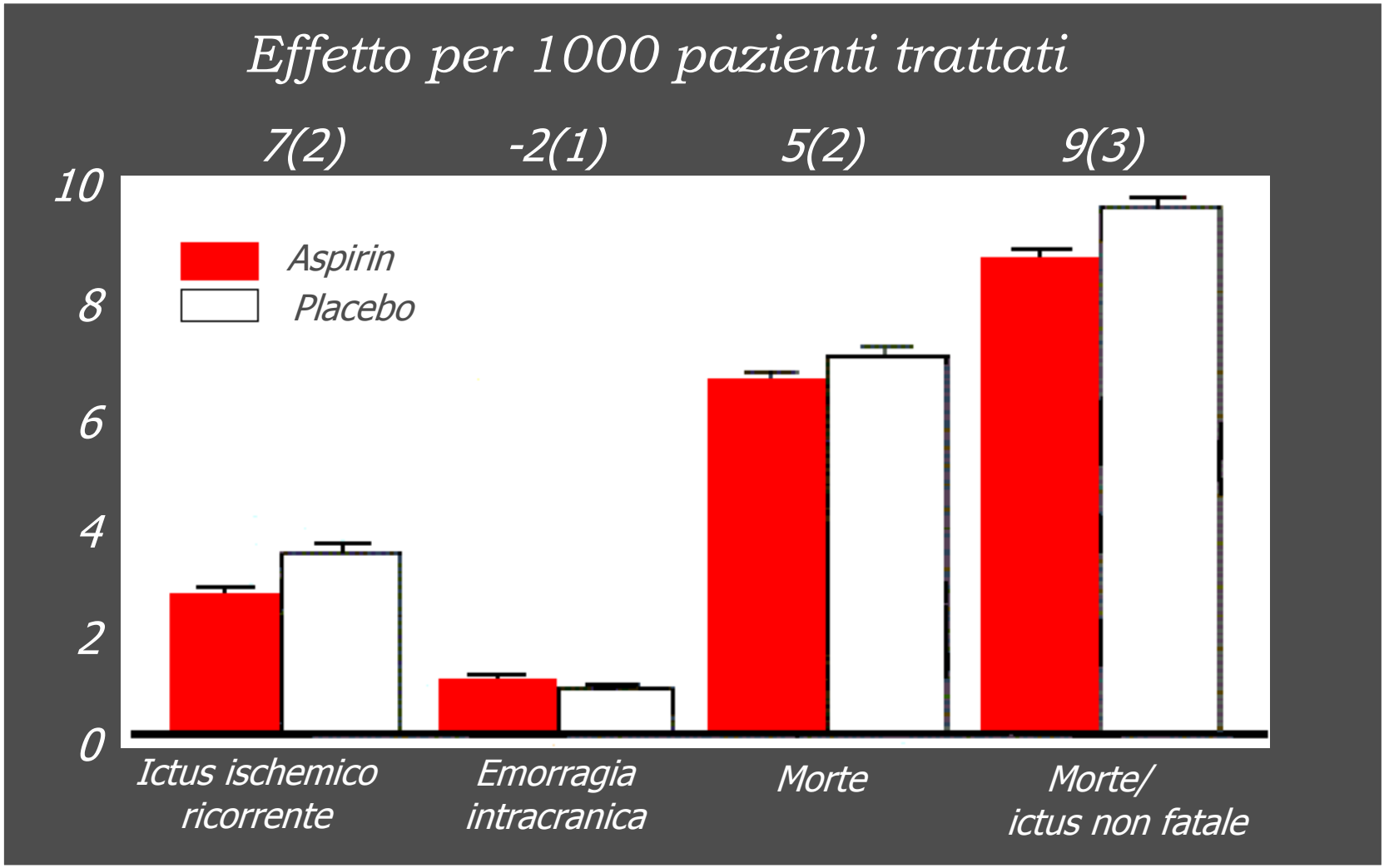
Cerebral Blood Volume	Cerebral Blood Flow	Mean Transit Time


Tissue Status
Perfusion Status
Vessel Status
CBV CT
PCT
CTA
Multimodal CT
DWI
PWI
MRA
Multimodal MRI
Bioenergetic Compromise
Hemodynamic Compromise
Occlusions or Stenoses

Se il paziente non ha effettuato trombolisi
o dopo la trombolisi?

Intanto se ha effettuato trombolisi, ripetere
TC dopo 24 ore per escludere trasformazione
emorragica

Profilassi secondaria precoce: Aspirina



The Lancet, 1997

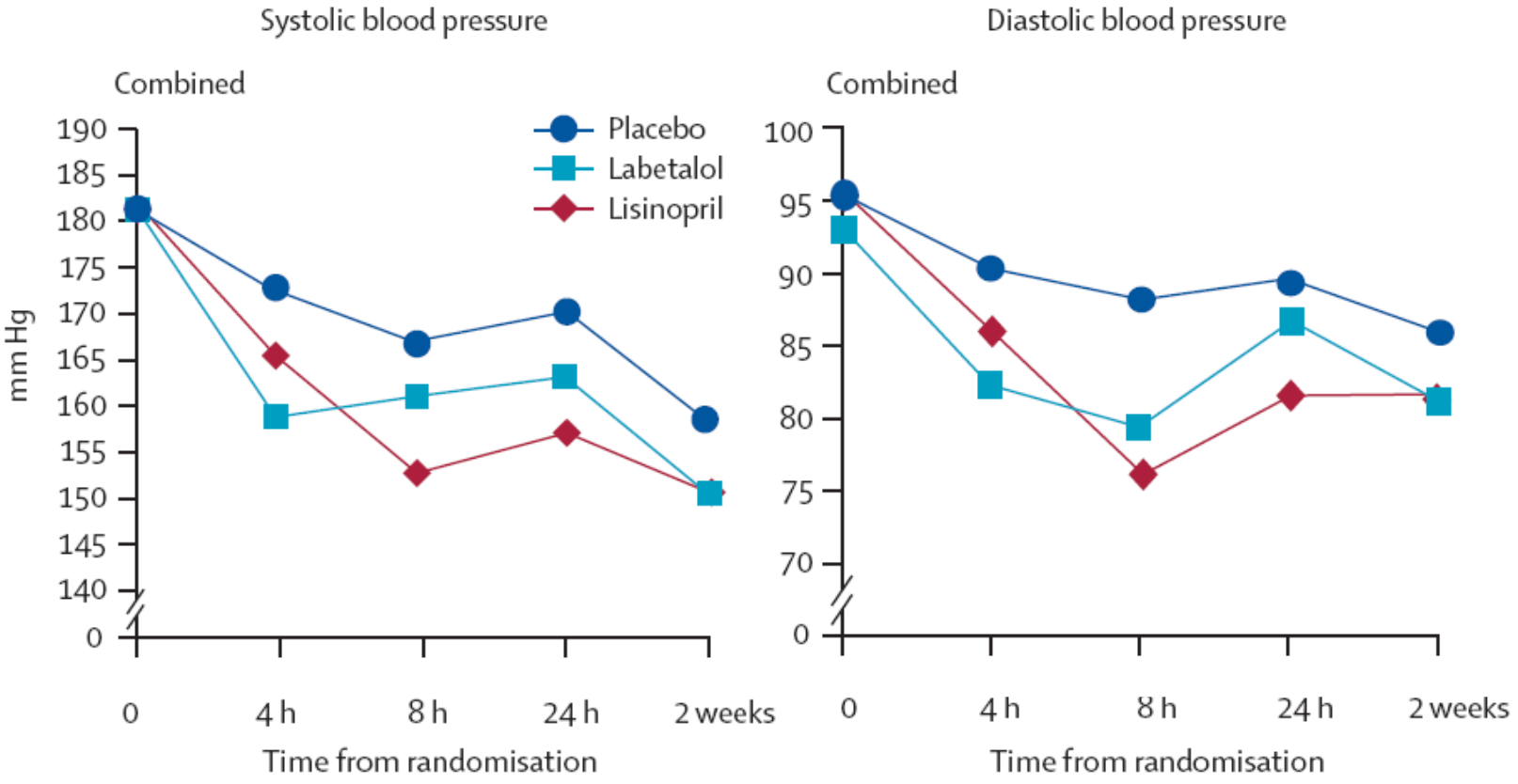
Profilassi secondaria precoce

- Aspirina 160-325 mg al giorno da subito

- Alternative all'aspirina:
  - Clopidogrel 75 mg/die
  - Aspirina + dipiradomolo

Complicanze precoci

The hypertensive response: self-limiting nature



Potter JF, Lancet Neurology 2009

Current Guidelines: when antihypertensive treatment is indicated

	Systolic BP	Diastolic BP
SPREAD	≥220	≥120
European Stroke Organization	≥220	≥120
American Heart Association	≥220	≥120
Stroke Foundation New Zeland	≥220	≥120
Hong Kong Consensus Statement	≥220	≥120
National Stroke Foundation	≥220	≥120
American Accademy of Neurology	≥220	≥140

Current Guidelines: when antihypertensive treatment is indicated

Patients candidates for iv rt-PA	Systolic BP	Diastolic BP
SPREAD	≥185	≥110
European Stroke Organization	≥185	≥110
American Heart Association	≥185	≥110
Stroke Foundation New Zeland	≥185	≥110
Hong Kong Consensus Statement	≥185	≥110
National Stroke Foundation	≥185	≥110
American Accademy of Neurology	≥185	≥110

Current Guidelines: when antihypertensive treatment is indicated

Indications to actively lower blood pressure

- ✓ hypertensive encephalopathy
- ✓ aortic dissection
- ✓ heart failure
- ✓ acute myocardial infarction
- ✓ acute renal failure
- ✓ preeclampsia and eclampsia

- urapidil (EBRANTIL)

- fiale da 50mg (10 cc) diluibili in soluzione fisiologica o glucosata

- somministrare con pompa di infusione: diluire 2 fiale da 10 cc (50 mg) + 30 cc di soluzione fisiologica. La concentrazione finale è 2 mg/ml. Iniziare l'infusione con 2mg/min (60 ml/h), riducendo quindi la velocità di infusione in base alla risposta clinica fino a valori medi di 0.15-0.20 mg/min (vedi tabella):

mg/minuto	ml/ora	dose/ora (in mg)
0.05	1.5	3
0.10	3	6
0.15	4.5	9
0.20	6	12
0.30	9	18
0.50	15	30
0.75	22.5	45
1	30	60
2	60	120

- labetalolo (TRANDATE)

  - fiale da 100 mg
  - 2 f. in 200 cc di soluzione fisiologica
  - infusa con pompa 2mg (2ml) per minuto (poi modificare in base alla risposta)
  - non utilizzare in caso di asma, insufficienza cardiaca, bradicardia, disturbi severi della conduzione.

Durante e dopo trattamento:

Monitorizzare la pressione arteriosa per 24h dall'inizio del trattamento

Ogni 15 minuti per due ore dopo che è iniziata l'infusione, poi

Ogni 30 minuti per sei ore, poi ogni ora per 18 ore.

Early worsening after an ischemic stroke

1-2 every 5 patients

- Causes related to arterial occlusion or cerebral infarct
- Systemic causes

Edema cerebrale

- ➔ *10-20% degli ictus*
- ➔ *Massima espressione 3-5 giorni dopo l'evento acuto*
- ➔ *Determina ↑ della pressione intracranica (PIC) condizionando la prognosi del paziente*

Hacke, 2004

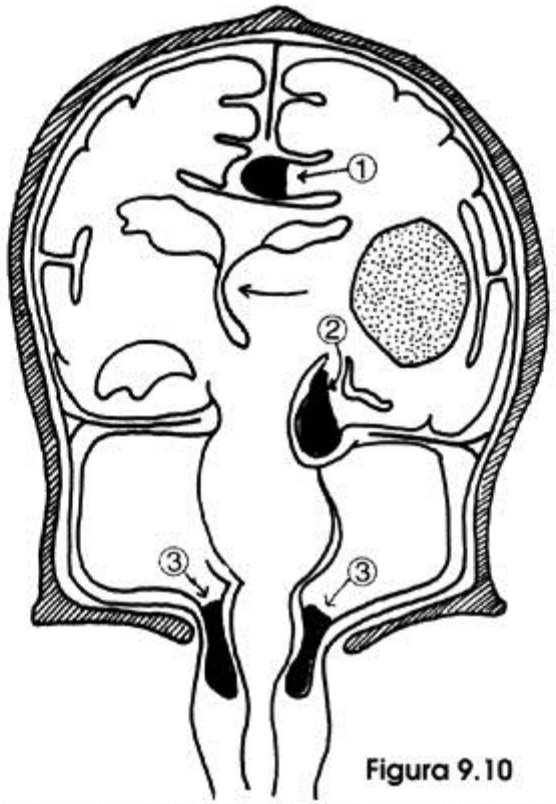


Figura 9.10

1: hernia del *gyrus cinguli*, **2**: hernia de la circunvolución parahipocampal, **3**: hernia de amígdalas cerebelosas. Punteado: masa que produce hipertensión endocraneana (según Hirano A (1983) Praktischer Leitfaden der Neuropathologie. Springer, Berlin Heidelberg New York; modificado)

Edema cerebrale

Strategia di trattamento:

- posizione del capo 20-30° rispetto al piano del letto
- diuretici osmotici
- iperventilazione
- derivazione liquorale
- craniotomia

American Heart Association®
Fighting Heart Disease and Stroke

Guidelines for the early management of patients with ischemic stroke: a scientific statement from the Stroke Council of the American Stroke Association , 2003

Edema cerebrale

Diuretici osmotici:

- Mannitolo

- Glicerolo

Edema cerebrale

Mannitolo *(0.25-0.5 g/Kg ev nell'arco di 20 min. ogni 6 ore per massimo 5 giorni)*

↓ *PIC*

Edema cerebrale

Glicerolo (250 ml al 10% ev in 30-60 min. ogni 6 ore)

↓ mortalità

(emolisi!)

Livello II

American Heart Association®
Fighting Heart Disease and Stroke

Guidelines for the early management of patients with ischemic stroke: a scientific statement from the Stroke Council of the American Stroke Association , 2003

Brain edema

- Mannitol is typically used at 0.25 to 0.5 g/kg IV administered over 20 minutes, lowers intracranial pressure, and can be given every 6 hours. The usual maximal dose is 2 g/kg.

- The effect of mannitol in patients with ischemic brain swelling is unknown, but it is often used especially in patients with reduced consciousness.

- Although aggressive medical measures, including osmotherapy, have been recommended for treatment of deteriorating patients with malignant brain edema after large cerebral infarction, these measures are unproven.

Edema cerebrale

CONTROINDICATI

Steroidi

Barbiturici

Livello I

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