STROKE

RAHAYU G.

- CVA = Cerebrovascular Accident
- CVD = Cerebrovascular Dissease
- GPDO = Gangguan Pembuluh Darah Otak
- Brain Attact = Serangan Otak
- Apoplexy

Face

Arm

Learn these signs of stroke.

Speech

Be a hero. Save a life.

Time

Call 9-1-1

NSMC
NORTH SHORE MEDICAL CENTER
A Primary Stroke Service of the Department of Public Health
nsmcstroke@partners.org

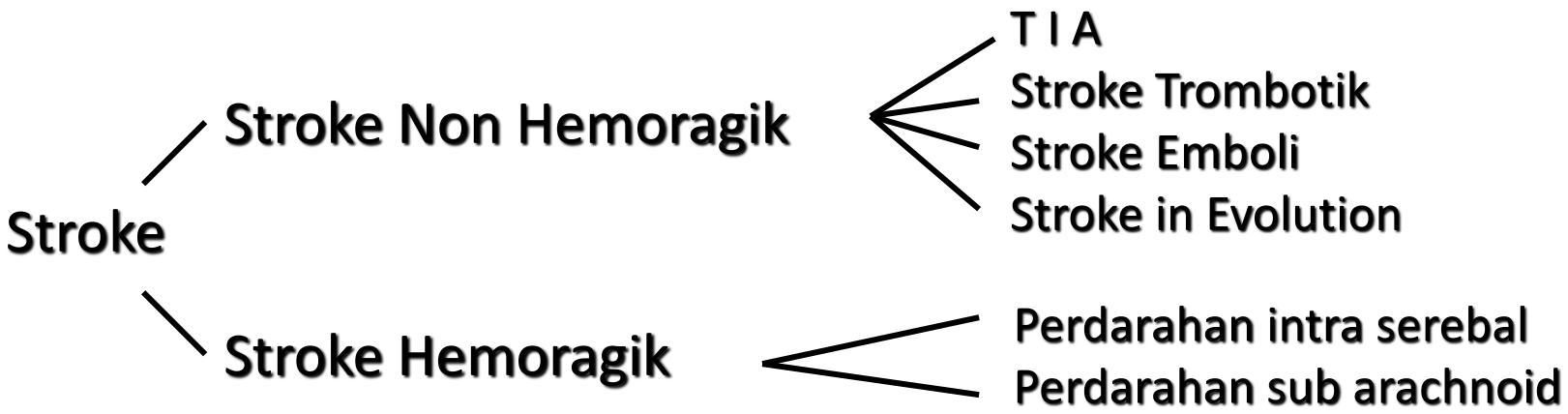
Massachusetts Department of Public Health — For more information call 1-800-467-1119 or email heart.stroke@state.ma.us

I. Definisi (WHO)

- Gangguan otak (Fokal, Global)
- Mendadak
- > 24 jam / berakhir dengan
- OK = gangguan vaskuler ≠

  tanpa penyebab lain (infeksi, trauma, psiko gen)

II. Klasifikasi Stroke

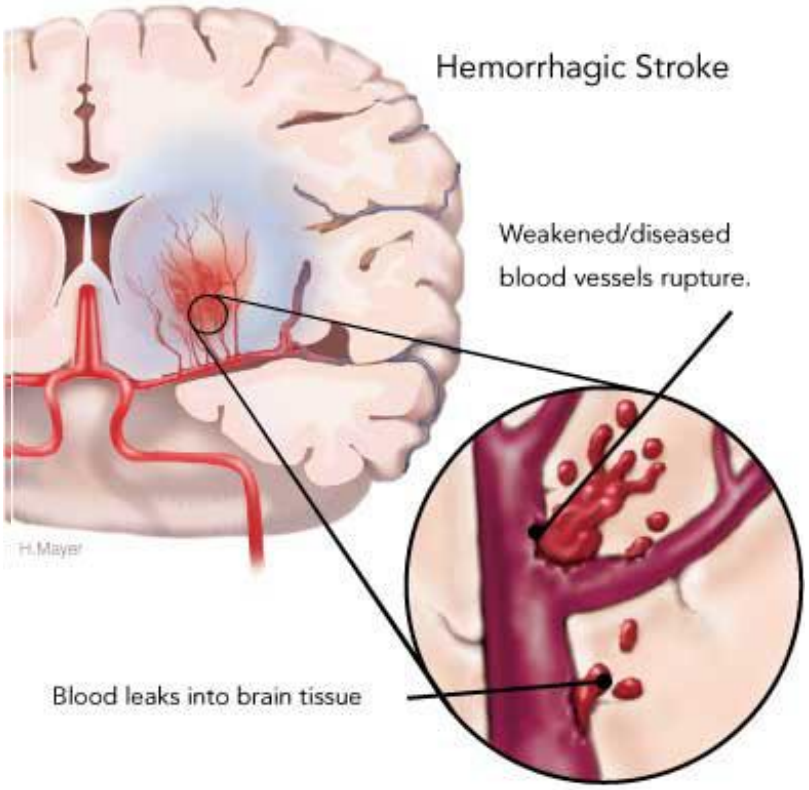


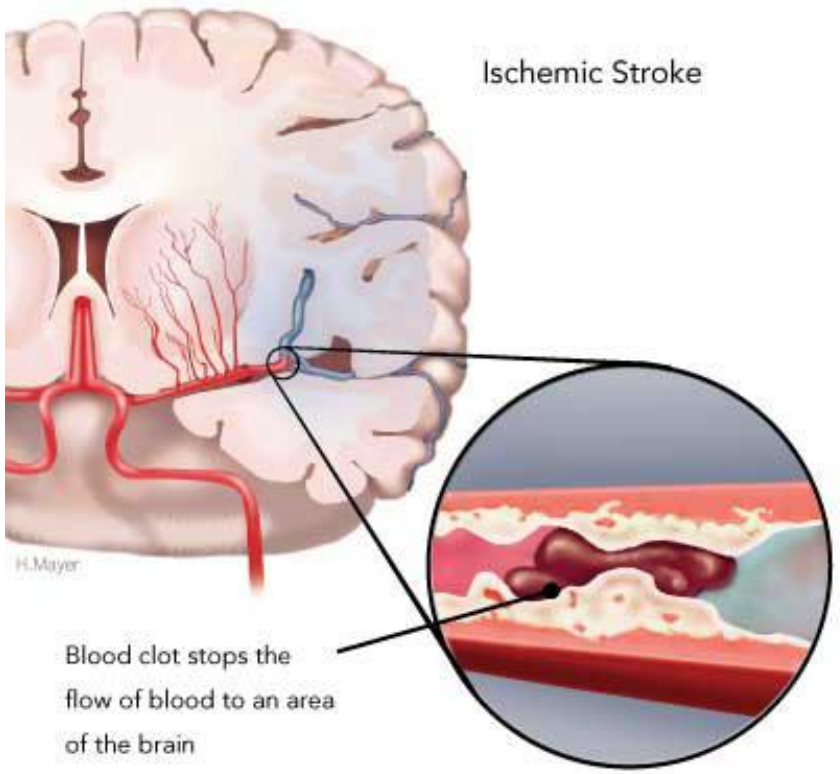
C V A Bleeding

= Stroke Hemoragik (Hemoragic Stroke)
= Stroke Perdarahan

I. PENDAHULUAN

- Stroke : masalah besar, tantangan $\rightarrow$ kesehatan
- Menyerang semua usia, >> $\rightarrow$ usia > 65 th
- Penyebab kematian tersering
- Perdarahan Intra Serebal (PIS / ICIt) 10% dari stroke 80% (Hemisfer) s 20% (Batang otak)
- Hemoragik (14%), Iskemik (86%) (Strven JI)





Usia	Stroke Iskemik	Perdarahan Intraserebral	Perdarahan Subarakhnoid
< 45 tahun	5	1	4
45 – 54	50	14	23
55 – 56	155	21	21
65 – 74	522	78	31
75 – 84	976	106	28
85 +	1357	158	13

(Dikutip dari Broderick JP, Phillips S.J, Whisnant JP and O'Fallon WM : Stroke Incidence rates in the eighties : The end of the decline in stroke ? Stroke 1989; 20:577).

Penyebab Stroke Perdarahan

- **Faktor Anatomik**
- **Faktor Hemodinamik**
- **Faktor Hemostatik**
- **Faktor Lain**

Faktor Anatomik

- AVM (Arterivenous malformation
- Lipohialinosis
- Angiopati amiloid
- Aneurisma
- Trombosis venus intrakranial
- Diseksi arteri
- Fistula karosiko kavernosa

Faktor Hemodinamik

- **Hipertensi**
- **Migren**

Faktor Hemostatik

- **Antikoagulan**
- **Antiplalelet**
- **Trombolitik**
- **Hemofilia**
- **Leukemia**
- **Trombositopeni**

Faktor Lain

- **Tumor intrakranial**
- **Vaskulitis**
- **Drug abuse (Amfetamin, Alkohol, Kokain)**

III. Klasifikasi

A. Perdarahan IntraSerebral (PIS)
   = Intracranial Hematoma (ICH)

B. Perdarahan Subarachnoid (PSA)
   = SubArachnoid Hematoma (SAH)

A. Perdarahan Intraserebral (PIS)

Insiden
PIS = 10% dari seluruh kasus stroke

- **80% (hemisfer), 20 % (batang otak & serebellum)**
- **Penyebab hipertensi : 50% → putamen & cap. Interna**
- **Angka kematian PIS : 30 hr serangan → 35 – 52%**

Patofisiologi PIS

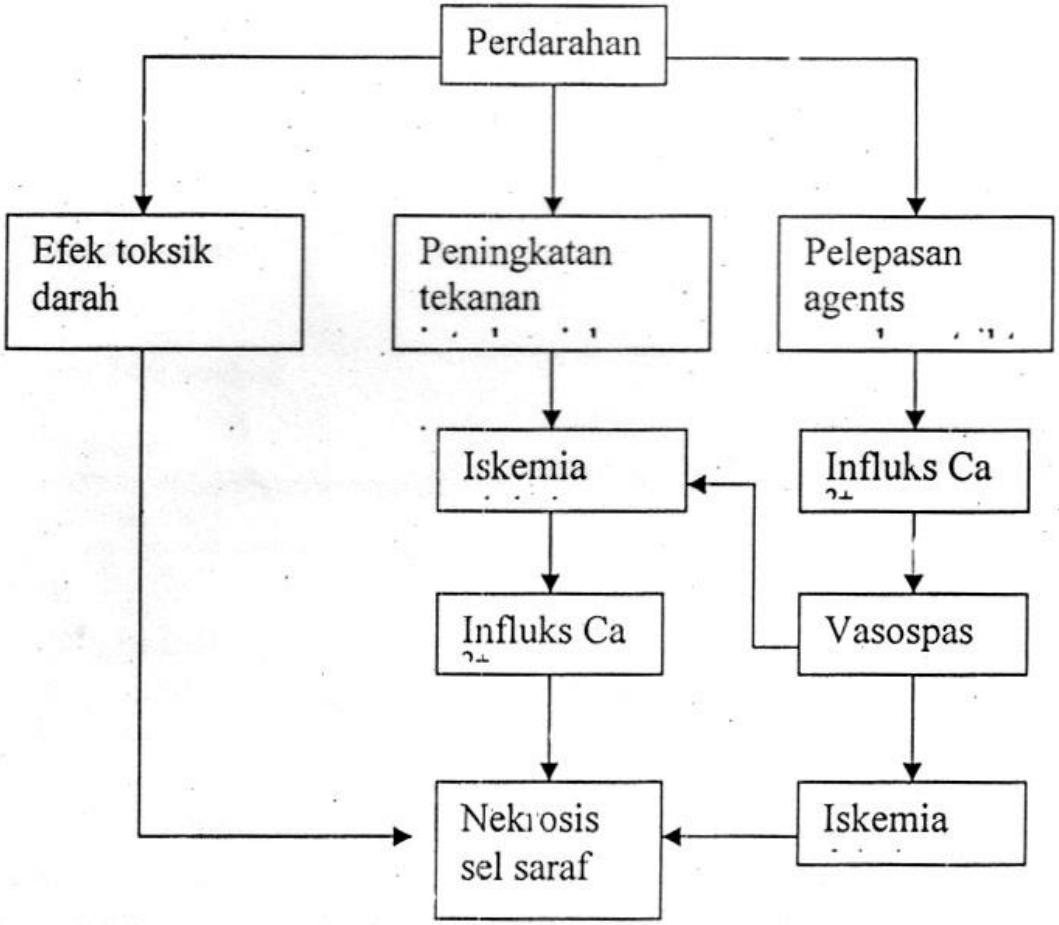
1. Konsep I $\rightarrow$ Hematoma Growth
2. Konsep II $\rightarrow$ Brain injury dan Swelling ^{ok}/
   trombin dan coagulatin and products

Diagnosis PIS

Anamnesis

- **mendadak'**
- **Saat aktivitas**
- **Kesadaran menurun**
- **Muntah, sakit kepala**
- **Riwayat hipertensi**
- **Kejang**
- **Pusing (vertiqo)**
- **Pencetus (emosi, ……..trauma**

Proses Nekrosis pada Perdarahan

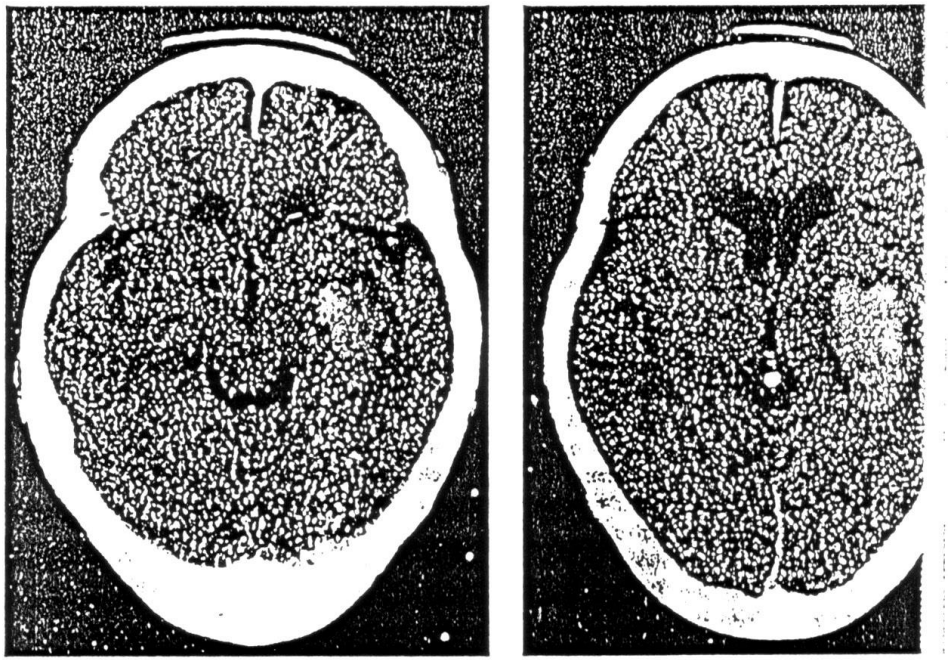


Pemeriksaan Klinis

- Hipertensi
- GCS menurun
- Defisit neurologis fokal :
  - **Gangguan FKL (Fungsi Kortikal Luhur)**
  - **Gangguan N Cranialis**
  - **Gangguan koordinasi, dismetri**
  - **Hemiparese alternan / tipikal**
  - **Gangguan sensorik**
  - **Gangguan otonom**
  - **Funduscopy : papilodema**

Primary site	Extension	Telltale signs
Caudate nucleus	Localized intraventricular hemorrhage	Headache, confusion, drowsiness-stupor, abul
	Capsule, putamen, diencephalon	Hemiparesis, eye deviation, Horner's syndrom
Putamen	Localized	Hemiparesis, eye deviation, global aphasia
	Posterior extension	Fluent aphasia
Thalamus	Localized	Paresthesia, hemineglect, nonfluent aphasia (often preserved repetition), disorientation t
	Mesencephalon	Marked bradykinesia
Cerebellum	Localized	Dysarthria, appendicular ataxia, headache
	Vermis	Deterioration in consciousness, marked gait a
Pons	Localized	Ataxic hemiparesis ophthalmoplegia, ocular b
	Mesencephalon	Hyperthermia, coma, pinpoint pupils

Adapted from Intracerebral hematoma. In: Wijdicks EFM, ed. *Neurologic catastrophies.* Boston: Butt Heineman, 2000:127.



A

FIG. 19-1. CT scan in a 92-year-old man with a putaminal (hypertensive) hemorrhage. Note sulci and fissures. Despite large volume of hemorrhage, no shift is seen and the additional is well-tolerated.

Pemeriksaan Tambahan

- CT scan : hiperdens
- Darah lengkap : lekositosis
- Kimia darah : glukosa
- LP (-)
- CT/BT, PTT, APTT

Prognosis :

- **Letak lesi**
- **Volume perdarahan**
- **Usia penderita**
- **Penyakit penyerta**

B. Perdarahan Subarachnoid (PSA)
= Subarachnoid Hemorrage (SAH)

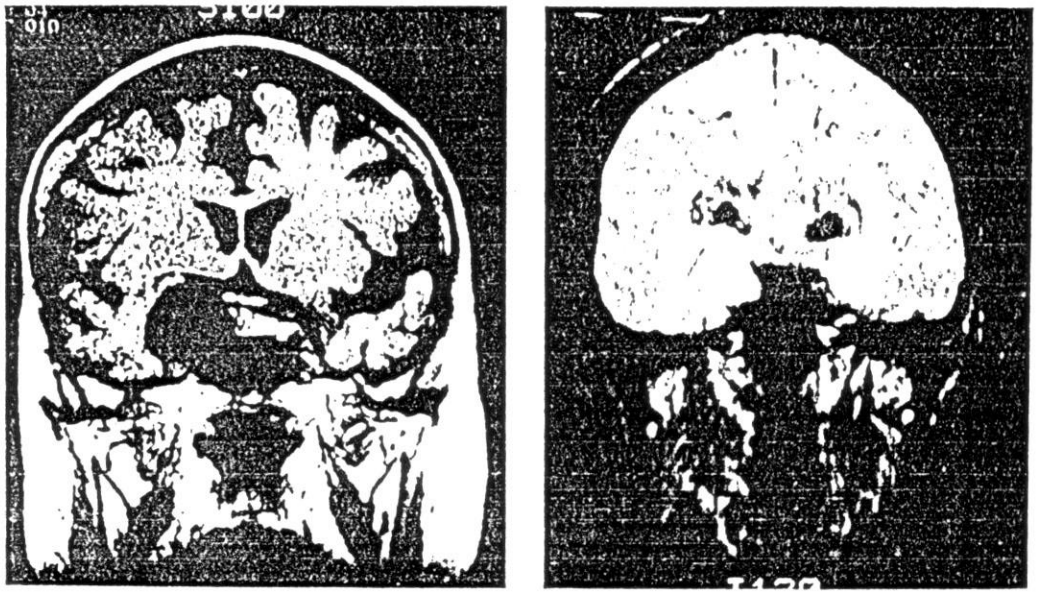
AS : 2500 kasus baru (dr 1 jt pdk) → 250 (SAH) → 50% (30 hr) (Gilroy)

Penyebab :

- Utama : aneurisma ($\phi \geq 10$ mm) / angiografi (Berry An)
  aneurisma sakuler (2 mm s/d 2/3 cm)
- AVM

A. Common Causes
   1. Traumatic subarachnoid hemorrhage
   2. Spontaneous subarachnoid hemorrhage
      a. Intracerebral hemorrhage with rupture into the subarachnoid space
      b. Primary subarachnoid hemorrhage
         1. Ruptured berry aneurysm
         2. Bleeding arteriovenous malformation
         3. Ruptured mycotic aneurysm

B. Rare Causes
   1. Developmental defects including pseudoxanthoma elasticum, Ehlers-Danlos syndrome, Sturge-Weber disease, hereditary hemorrhagic telangiectasia, telangiectasia pontis
   2. Infections. *Herpes simplex* encephalitis, acute hemorrhagic leukoencephalitis, brain abscess, tuberculous meningitis, syphilitic vasculitis
   3. Neoplasm. Primary or metastatic brain tumor. Hemangioblastoma of the cerebellum or brain stem
   4. Blood dyscrasias. Leukemia, Hodgkin's disease, thrombocytopenia, sickle cell anemia, hemophilia, aplastic anemia, pernicious anemia, anticoagulant therapy
   5. Vasculitis. Polyarteritis nodosa, anaphylactic purpura
   6. Arteriosclerosis. Rupture of an arteriosclerotic vessel
   7. Subdural hematoma



A B

FIGURE 9-1. MR scan coronal sections, T1(A) and T2 (B) weighted images, showing a large aneurysm of the terminal portion of the right internal carotid artery.

Diagnosis PSA

ANAMNESA :

- Mendadak
- Aktivitas
- Pencetus
- Riwayat HT
- Sakit kepala hebat (Thunderclap Headache)
- Pusing
- Kejang, muntah
- Kesadaran ↓

PEMERIKSAAN KLINIS :

- **GCS menurun**
- **Meningeal sign (+) : KK, kerning sign, brudzinsky**
- **Defisit fokal neurologi**
- **Tanda TIK ↑**
- **Funduskopi : - papil edema**
  - perdarahan subhyaloid retina

PEMERIKSAAN PENUNJANG :

- CT scan
- LP Lumbal Punksi
  - **CT (kelainan (-)**
  - **hati-hati**
  - **10 – 15% PSA → < P -**
- **Angiografi serebral**
  - **Melihat aneurisma**
  - **Baku emas (dx PSA)**
- **MRI : - AVM**
- **TCD ((Trans Cranial Doppler)**
  CBF : Vasospasme
- **Lab : CT / BT, PTT / APTT : Agregasi trombosit**
  blood typing dan screening

Peringkat Klinis PSA (Hunt & Hess)

Tingkat 0 : Aneurisma (Unruptured)

I : Asimtomatik

II : - Nyeri kepala hebat

- Parese n Cranialis, KK (+)

- Def neurologi lain (-)

III : Somnolen, defisit ringan

IV : Slopor, defisit neurologis rigiditas awa

V : Coma, rigiditas deserebrasi ✝

Penatalaksanaan

- **Komplikasi SAH (Gilroy)**
  - Arterial Spasm (30%)
  - Rebleeding $\rightarrow \dagger$ (60%)
  - Intraserebral hematom $\rightarrow$ hemianopia homonim & hemiparesis
  - SIADHS (Syndrome of Inappropiate Anti diuretik Hormon Secretion)
  - Pneumoni, UTI, decubitus
- **Diferensial Diagnosis**
  - Migrain Headache
  - Infeksi Sistemik
  - Meningitis Akut
  - Hipertensi Ensefalopati

Penatalaksanaan Stroke Perdarahan

- **PIS**

  Umum : Prinsip = 5 B (Breathing, Blood, Brain, Bladder & Bowel) +, 1 B (Body & Skin)

Breath

Fungsi pernafasan : mayo, O_2

Blood

- **Pengendalian tekanan darah**

  Tensi diturunkan : S > 180 mmHg, D > 100 mmHg

  Penurunan tidak lebih 20% (tek. arterial rata-rata) = TAR

  TAR = MAP (Mean Arterial Pressure) = Mean BP

  = Teksistolik + 2 (tekanan Diastolik) / 3

- Balans cariran
- Elektrolit (K, Na, Ca, Mg, cl)
- Salin 0,9% : 1 ml/kg/jam
- Hindari Dextrosa

Brain

- Kesadaran (CrCS), posisi kepala 20 – 30^{o}
- Kejang
  - Diazepam (0,05 – 0,1 mm/kg)
  - Phenitoin (15 – 20 mg/kg)
  - Phenobarbital (15 – 20 mg/kg)
- Pe↑ TIK :
  - Posisi 20 – 30, hipotermi
  - Hiperventilasi Pa CO_2 30 – 35 mmHg
- Manitol 20 % (0,25 – 0,5 g / kg / BB / b)
  15 – 30, 4 – 6 x / b (Tapp of)
- Suhu tubuh

Bowel

- Nutrisi (enteral, stl 4 j)
- NGT

Bladder

- Urin (PU)
- Cateterisasi

Bones Body Skin

- Decubitus

II. KHUSUS

- Neuroprotektor (Piracetam, CDP cholin)
- Antifibrinolitik agent (Anti perdarahan)
- Tx Simtomatik lain

III. INDIKASI OPERASI

Rekomendasi Terapi Operatif pada Perdarahan Intraserebral
(Broderick,1999)

Kandidat yang tidak dioperasi

1. Perdarahan kecil (< 10 cm^3) atau dengan defisit neurologi minimal
2. GCS < 4. Tetapi penderita dengan GCS < 4 dengan perdarahan serebelum dan kompresi batang otak adalah kandidat operasi pada beberapa kasus

Kandidat yang dioperasi

1. Perdarahan serebelum dengan diameter > 3 cm dengan deteriorasi atau mengalami kompresi batang otak dan hidrosefalus akibat obstruksi ventrikel
2. Perdarahan intraserebral karena lesi struktural (aneurisma, malformasi arteriovenosa, angioma kavernosa), jika lesi terjangkau
3. Penderita muda dengan perdarahan lobar sedang atau besar (> 50 cm^3) yang mengalami deteriorasi

Table 52.8 **Recommendations for Surgical or Nonsurgical Treatment of Intracranial Hemorrhage**

ICH	*Clinical or CT features*	*Treatment*
Putamen	Alert, small ICH (<30 mL)	Nonsurgical
	Comatose, large ICH (>60 mL)	Nonsurgical
	Drowsy, intermediate ICH (30–60 mL)	Consider evacuation
Caudate	Alert or drowsy, with intraventricular hemorrhage and hydrocephalus	Consider ventriculostomy
Thalamus	Drowsy or lethargic, with blood in the 3rd ventricle and hydrocephalus	Consider ventriculostomy
Lobar white matter	Drowsy or lethargic, with intermediate ICH (20–60 mL), progressive decline in level of consciousness	Consider evacuation
Pons, midbrain, medulla	—	Nonsurgical
Cerebellum	Noncomatose, with ICH > 3 cm in diameter, and/or hydrocephalus, and/or effacement of quadrigeminal cistern	Evacuation recommended, preceded by ventriculostomy if status is actively deteriorating

CT, computed tomography; ICH, intracranial hemorrhage.

IV. REHAB MEDIK

PSA

I. Umum → PIS
II. Khusus → PIS
III. Indikasi Operasi → (derajat klinis / diatas derajat 3)

CVA TROMBOSIS (TROMBOSIS)

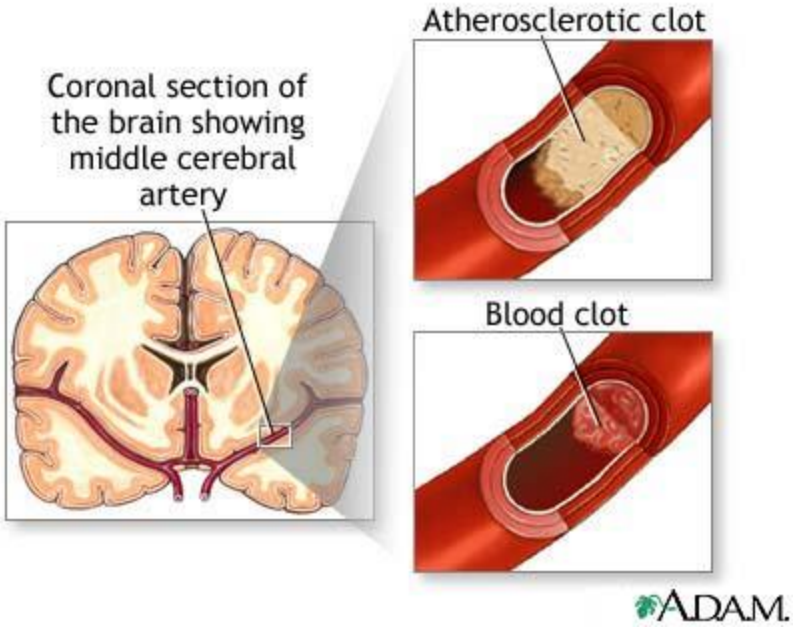
= Stroke Non Hemoragik (SNH)
= Non Hemorragic Stroke (NHS)
= Serebral Infarction
= Strok Iskemik

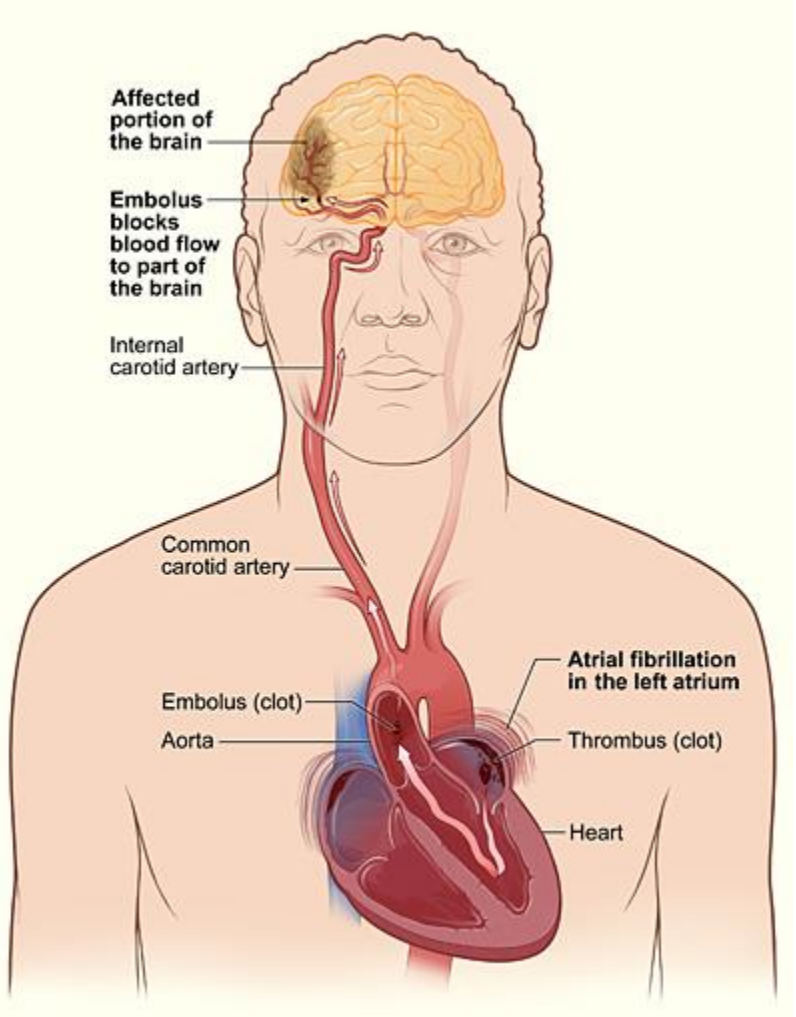
I. PENDAHULUAN

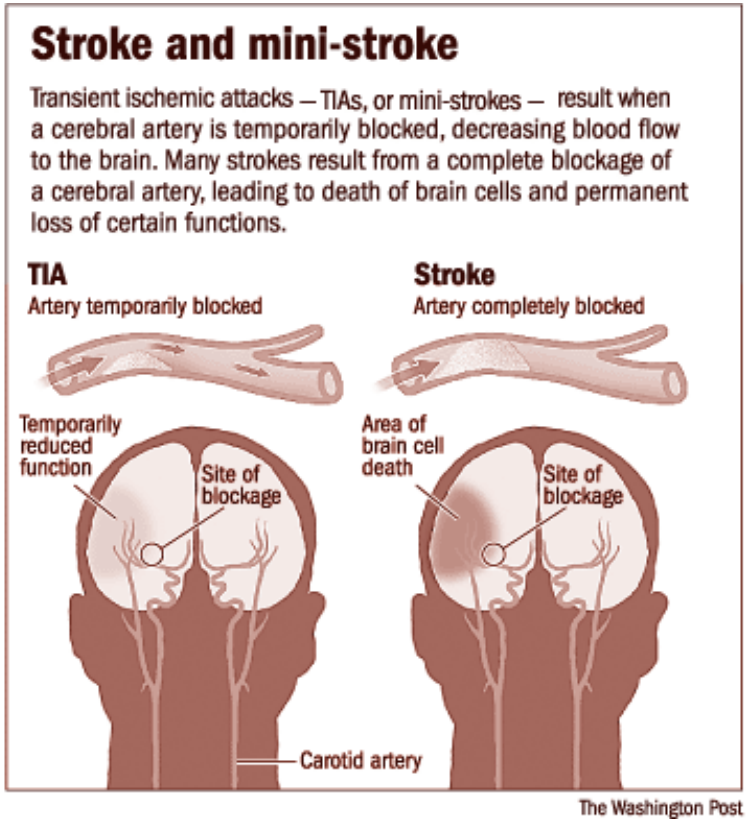
- **Kead → otak iskemia ok aliran darah otak ↓ / ok sumbatan trombus / embol**
- **Insiden ~ tabel**
- **WHO : 1999 → 5,54 juta**
  (9,5% dari seluruh kematian di dunia)

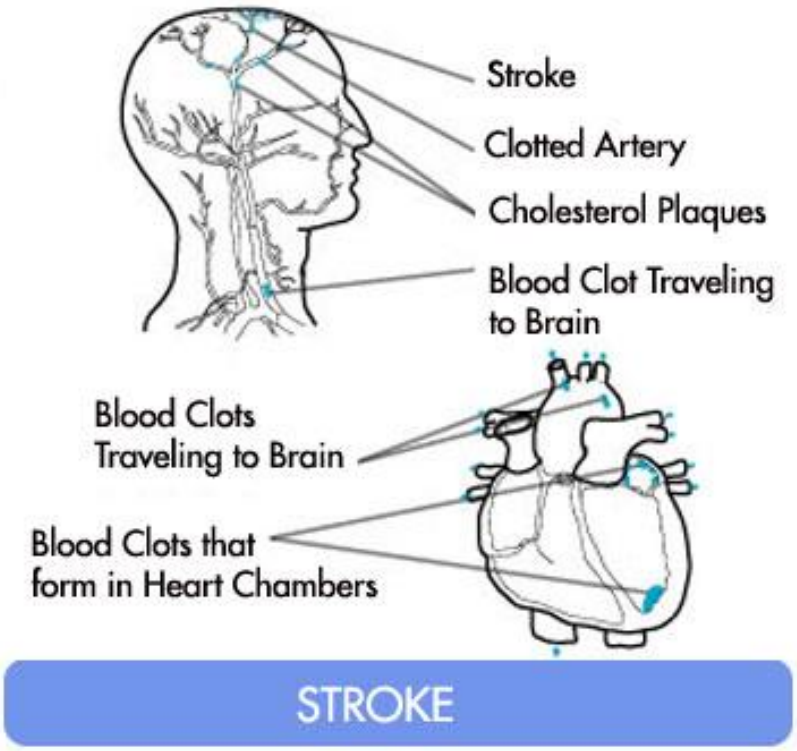
Usia	Insiden
35 – 44 tahun	0,2 0/00
45 – 54 tahun	0,7
55 – 64 tahun	1,8
65 – 74 tahun	2,7
75 – 84 tahun	10,4
> 84	13,9

Dikutip dari Delachaux A : Epidemiologie et prevention du ramollissement cerebral. Revmed Suisse rom 1970; 90 9 719-726


Coronal section of the brain showing middle cerebral artery
Atherosclerotic clot
Blood clot
A.D.A.M.


Affected portion of the brain
Embolus blocks blood flow to part of the brain
Internal carotid artery
Common carotid artery
Atrial fibrillation in the left atrium
Embolus (clot)
Aorta
Thrombus (clot)
Heart


Stroke and mini-stroke
Transient ischemic attacks – TIAs, or mini-strokes – result when a cerebral artery is temporarily blocked, decreasing blood flow to the brain. Many strokes result from a complete blockage of a cerebral artery, leading to death of brain cells and permanent loss of certain functions.
TIA
Artery temporarily blocked
Stroke
Artery completely blocked
Temporarily reduced function
Site of blockage
Area of brain cell death
Site of blockage
Carotid artery
The Washington Post


Stroke
Clotted Artery
Cholesterol Plaques
Blood Clot Traveling to Brain
Blood Clots Traveling to Brain
Blood Clots that form in Heart Chambers
STROKE

II. KLASIFIKASI

A. Perjalanan penyakit

1. TIA (Transient Ischemic Attact) < 24 j
2. RIND (Reversible Ischemic Neurological Deficit) > 24 j s/d
3. Stroke in Evolution (Stroke PRogresif)
4. Stroke Komplit

B. Berdasarkan : Patogenesis

1. Stroke √ trombotik viskemik
2. Stroke iskemik embolik
3. Stroke iskemik sebab lain (hematologik)
4. Stroke iskemik ?

III. FAKTOR RESIKO PADA STROKE

Non Modifiable	Modifiable
Age Gender Hereditary Race Ethnicity	Hypertension Atrial Fibrilation Diabetes Mellitus Hypercholesterolemia Asymptomatic carotid Disease Smoking Alcohol Consumption T I A

IV. PATOGENESA STROK ISKEMIK

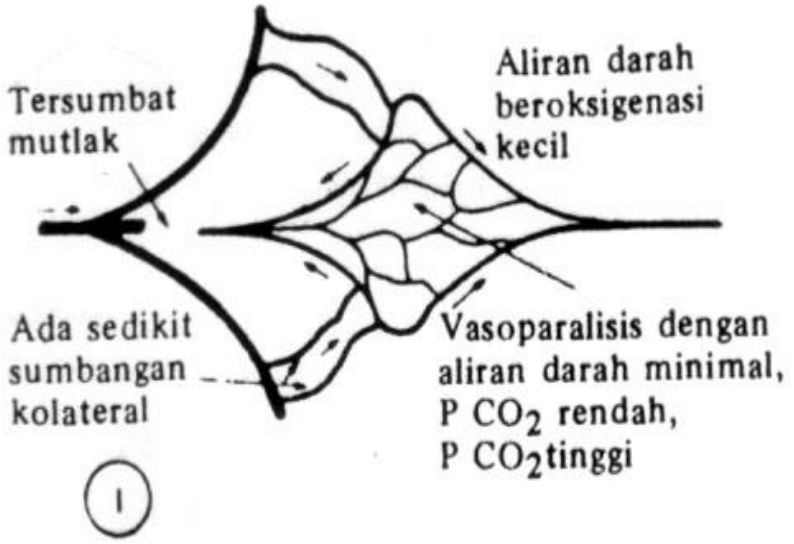
Sumbatan (trombus / embolus)

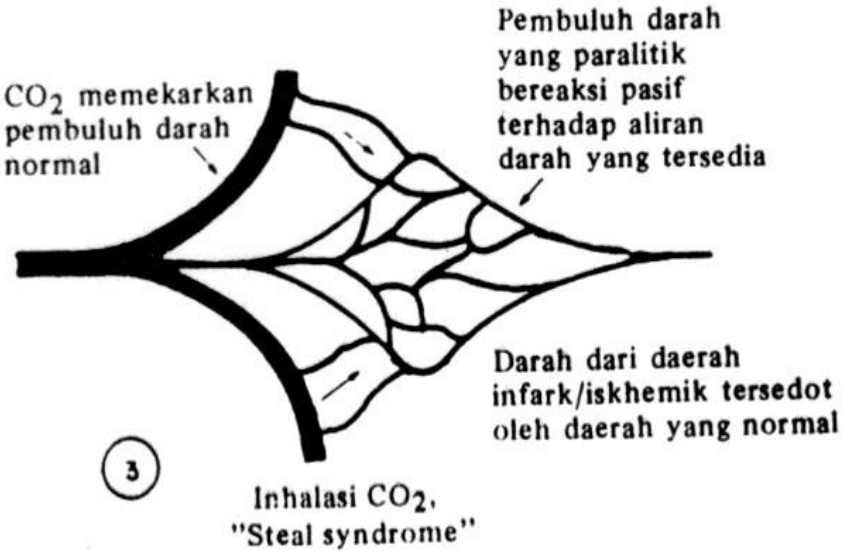
↓

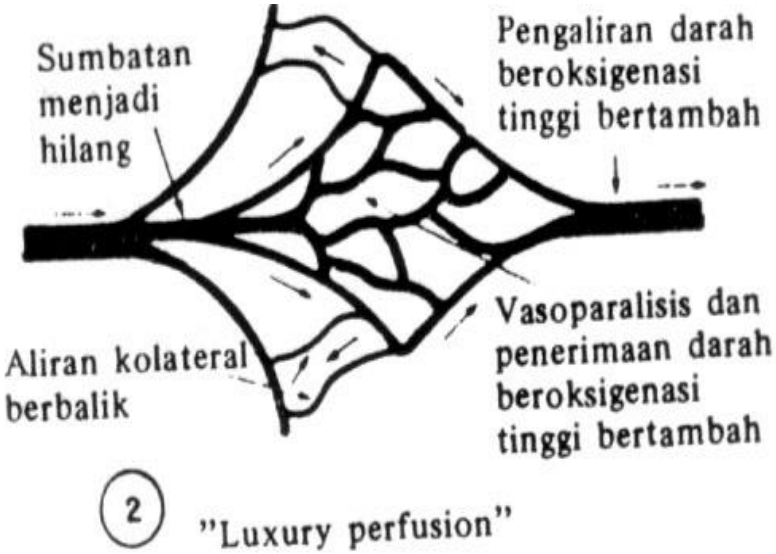
Hipoksia jar. Otak

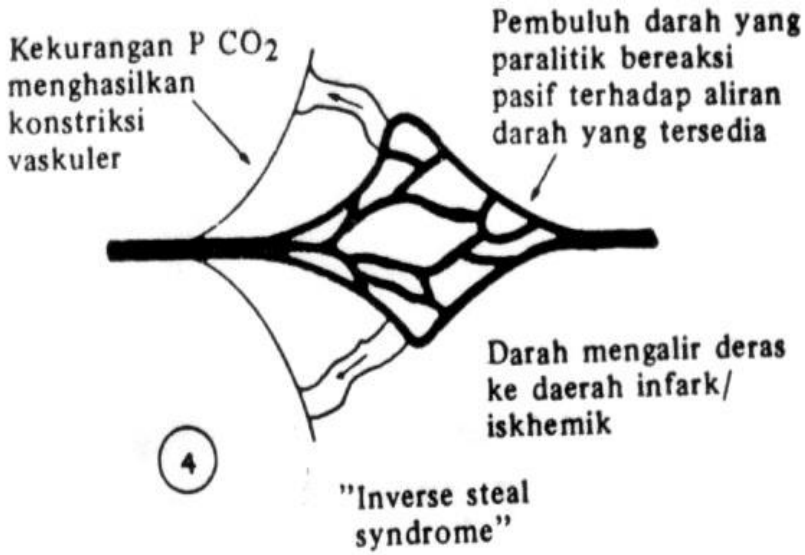
↓

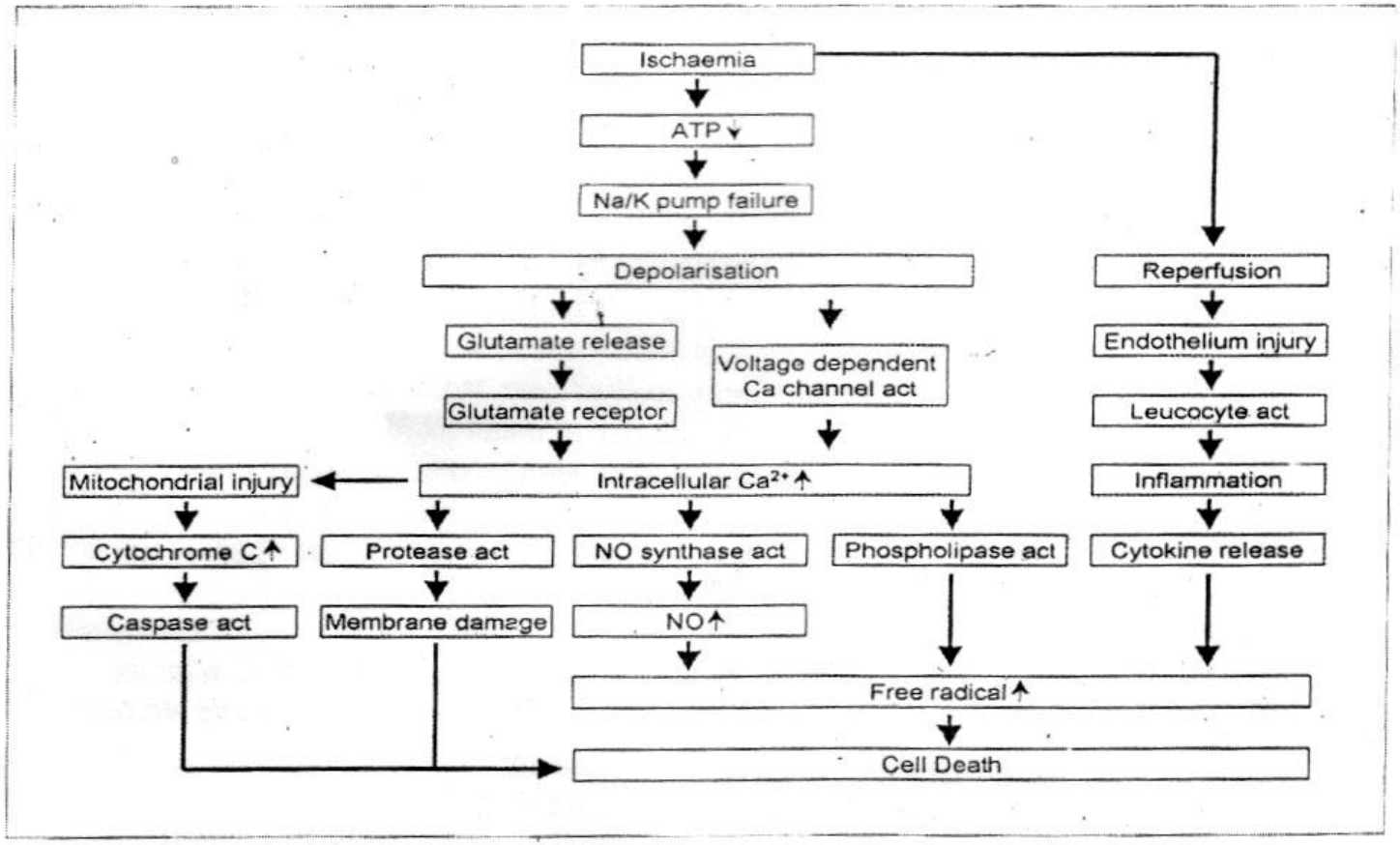
Infark kritis $\leq$ 20 ml / 100 gr / menit
(dikelilingi penumbra)
ADO 10 – 20 ml / 100 gr / menit


Tersumbat mutlak
Aliran darah beroksigenasi kecil
Ada sedikit sumbangan kolateral
Vasoparalisis dengan aliran darah minimal, P CO_2 rendah, P CO_2 tinggi
1


CO_2 memekarkan pembuluh darah normal
Pembuluh darah yang paralitik bereaksi pasif terhadap aliran darah yang tersedia
Darah dari daerah infark/iskhemik tersedot oleh daerah yang normal
3
Inhalasi CO_2, "Steal syndrome"


Sumbatan menjadi hilang
Pengaliran darah beroksigenasi tinggi bertambah
Vasoparalisis dan penerimaan darah beroksigenasi tinggi bertambah
Aliran kolateral berbalik
2
"Luxury perfusion"


Kekurangan P CO_2 menghasilkan konstriksi vaskuler
Pembuluh darah yang paralitik bereaksi pasif terhadap aliran darah yang tersedia
Darah mengalir deras ke daerah infark/ iskhemik
4
"Inverse steal syndrome"



The ischaemic cascade leading cell death

Proses Kematian Sel

Emboli dapat terbentuk dari :

- Gumpalan darah
- Fibrin trombosit
- Kholesterol
- Lemak
- Udara
- Tumor, meta
- Bakteri
- Benda asing

V. DIAGNOSIS STROKE ISKEMIK

- Anamnesa
- Pemeriksaan Fisik / Neurologis
- Pemeriksaan Penunjang

ANAMNESA

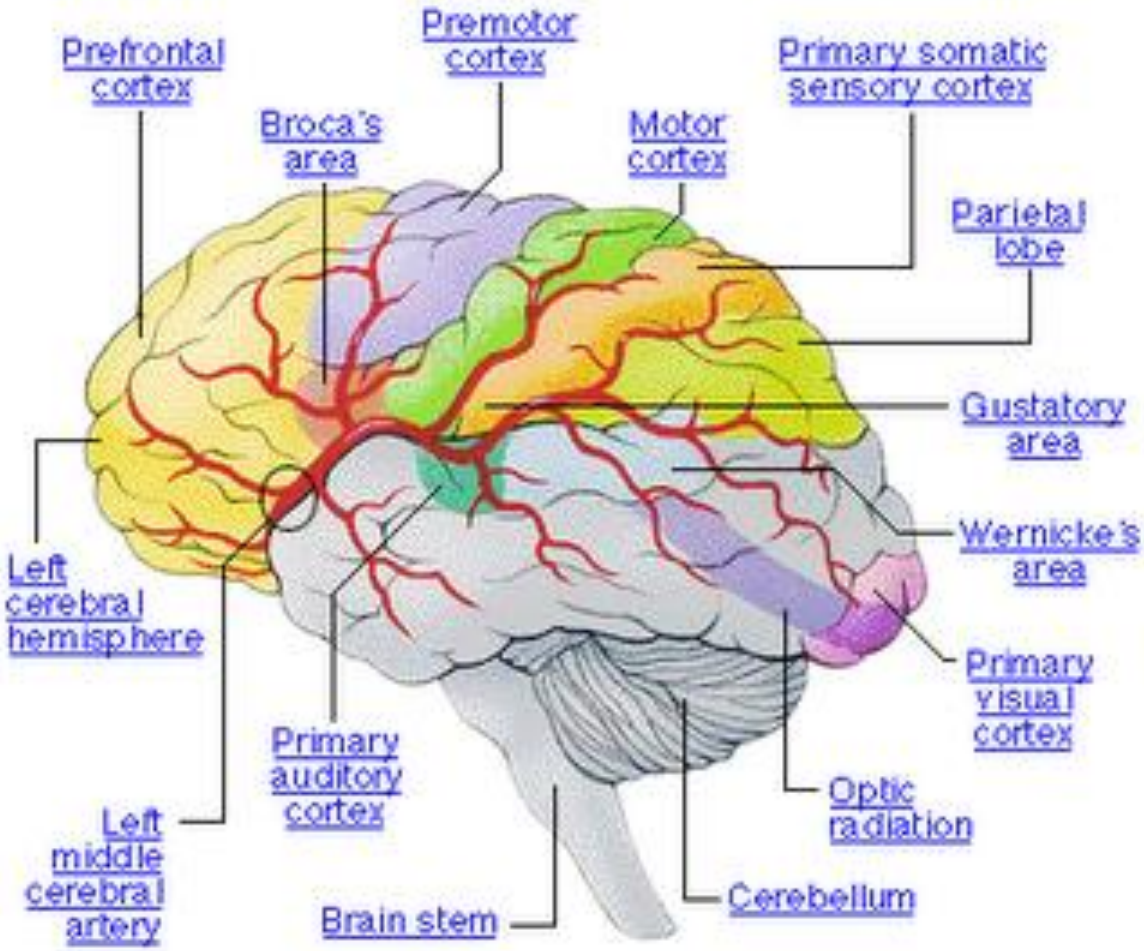
- Mendadak
- Saat istirahat / bangun tidur
- Kesadaran baik
- Tidak sakit kepala
- Tidak muntah
- Faktor resiko

PEMERIKSAAN NEUROLOGIS

- GCS $E_4M_6V_5$, E_4 M…V…
- Defisit Neurologis
  - Gangguan FKL
  - M Granialis
  - Koordinasi
  - Motorik
  - Sensorik
  - S S O

Gangguan FKL (Fungsi Kortikal Luhur)

- Gangguan berbahasa (afasia)
- Gangguan memori
- Gangguan praktis (apraksia)
- Gangguan atensi & konsentrasi
- Gangguan orientasi


Prefrontal cortex
Premotor cortex
Primary somatic sensory cortex
Broca's area
Motor cortex
Parietal lobe
Gustatory area
Wernicke's area
Left cerebral hemisphere
Primary visual cortex
Primary auditory cortex
Optic radiation
Left middle cerebral artery
Cerebellum
Brain stem

Gangguan Berbahasa (Afasia)

- Afasia motorik (Broea's ekspresif)
- Afasia sensorik (wernieke aphasia) / reseptif
- Afasia global
- Afasia anomik (amnestik)
- Afasia transkortikal

Pemeriksaan Penunjang

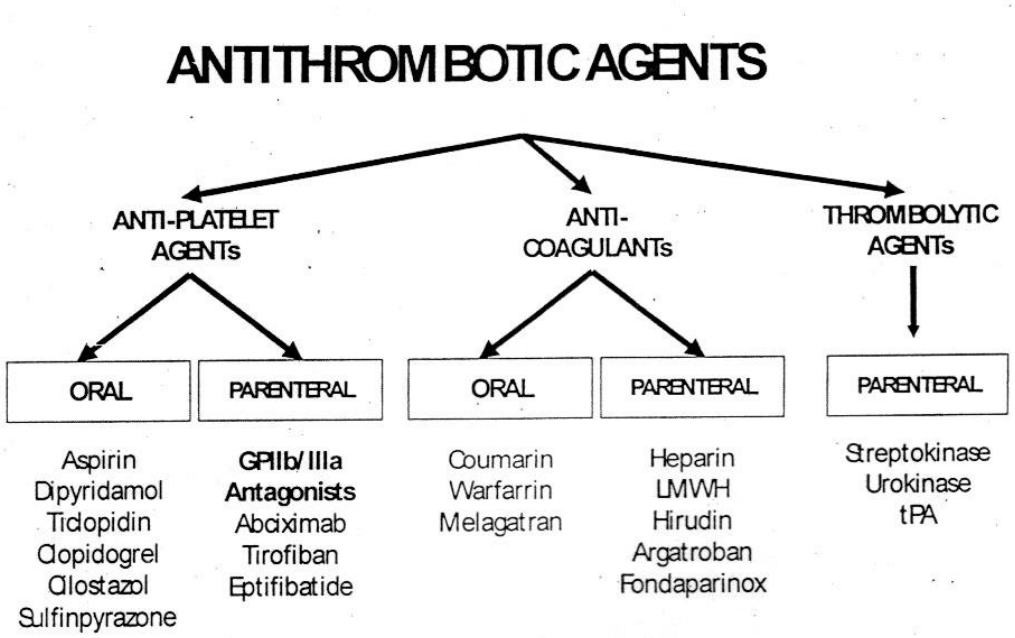
- CT Scan (Gold standard)
- Foto Thorax
- EKG
- Darah lengkap
- Kimia darah

Penatalaksanaan Strok Iskemik

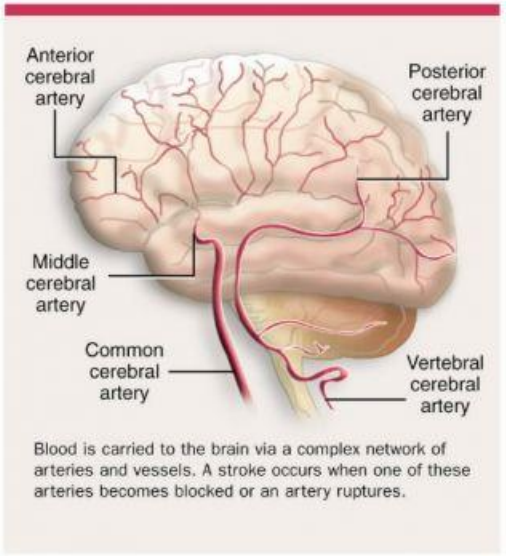
I. Terapi Umum (5 B)

II. Terapi Khusus

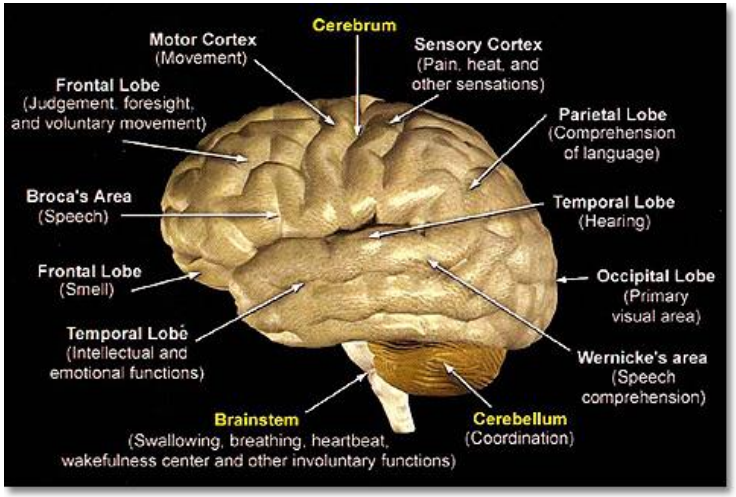
- **Anti Trombotik (Reperfusi)**
- **Neuroprotektor**

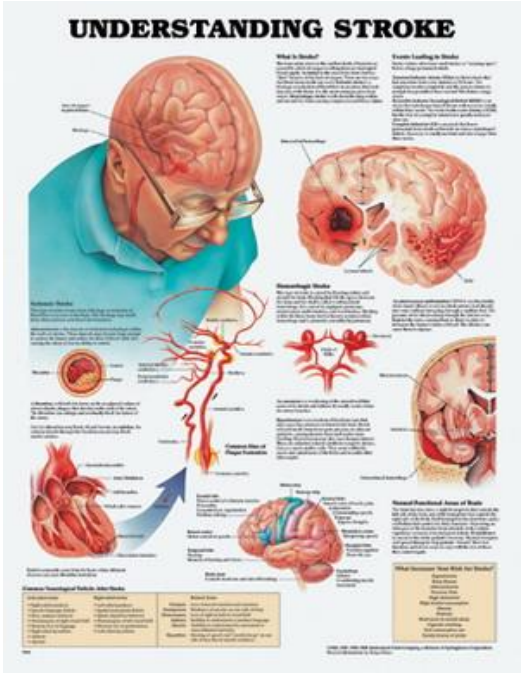


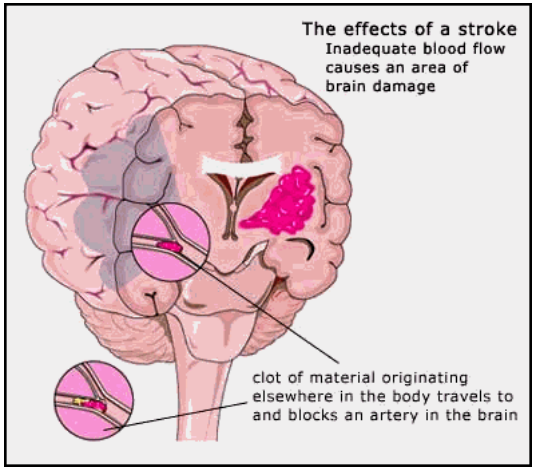
III. Rehab Medik

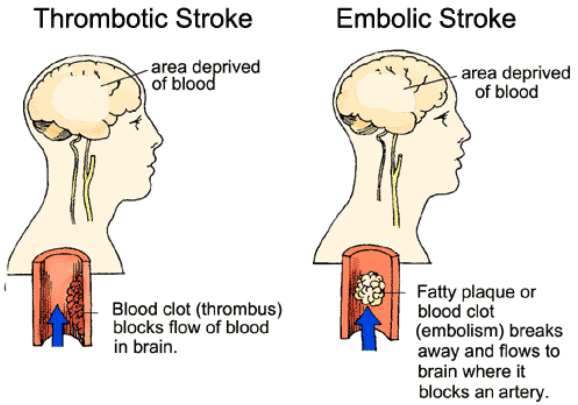


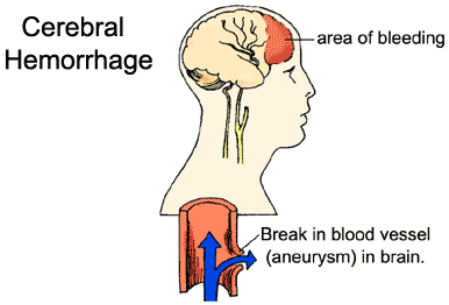
Blood is carried to the brain via a complex network of arteries and vessels. A stroke occurs when one of these arteries becomes blocked or an artery ruptures.

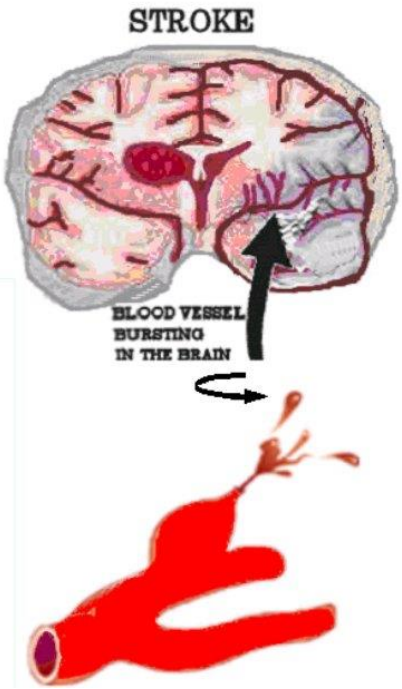


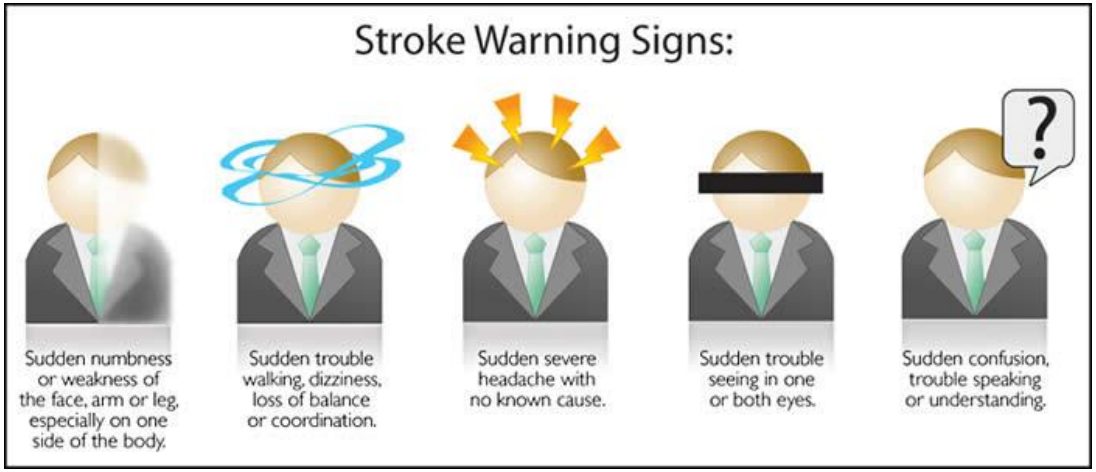

UNDERSTANDING STROKE


The effects of a stroke
Inadequate blood flow causes an area of brain damage
clot of material originating elsewhere in the body travels to and blocks an artery in the brain


Thrombotic Stroke
area deprived of blood
Blood clot (thrombus) blocks flow of blood in brain.
Embolic Stroke
area deprived of blood
Fatty plaque or blood clot (embolism) breaks away and flows to brain where it blocks an artery.


Cerebral Hemorrhage
area of bleeding
Break in blood vessel (aneurysm) in brain.


STROKE
BLOOD VESSEL BURSTING IN THE BRAIN


Stroke Warning Signs:
Sudden numbness or weakness of the face, arm or leg, especially on one side of the body.
Sudden trouble walking, dizziness, loss of balance or coordination.
Sudden severe headache with no known cause.
Sudden trouble seeing in one or both eyes.
Sudden confusion, trouble speaking or understanding.
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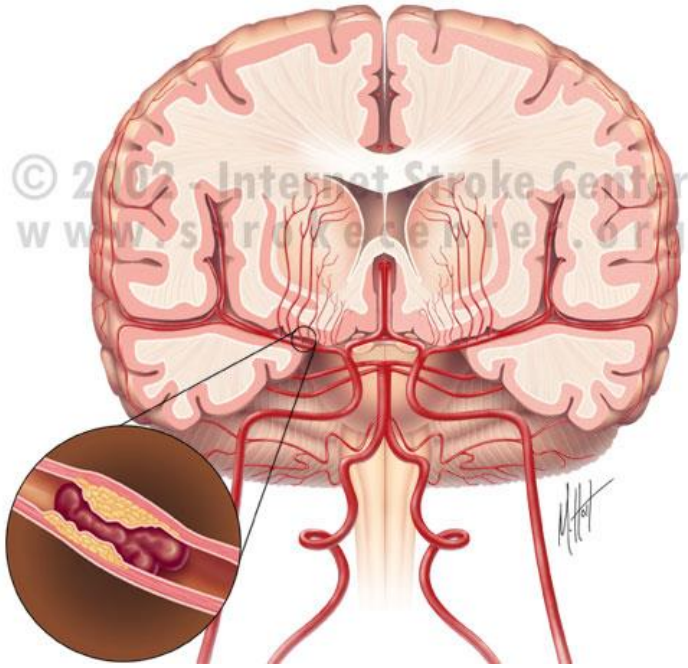
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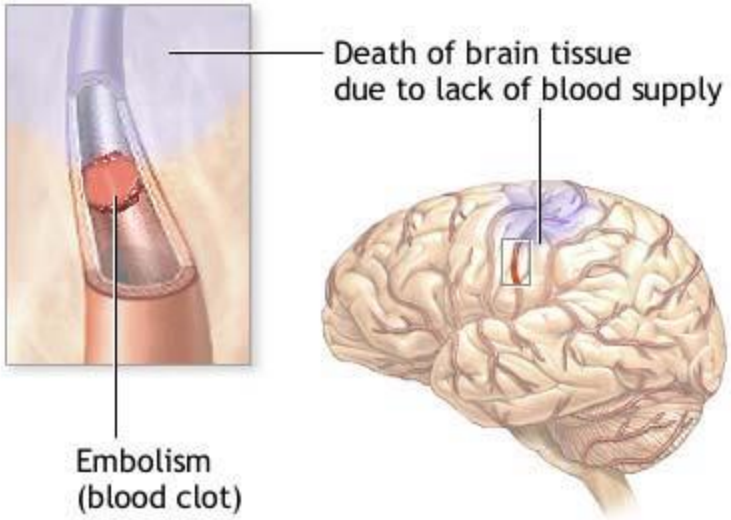
Death of brain tissue due to lack of blood supply
Embolism (blood clot)
A.D.A.M.