Emergency in neurology

COMA

Smell?

respiratory rate & patterns (

Look for abnormal posturing.

- Decorticate (Flexion of UE with Extension of LE)
- Decerebrate (Extension of all Ext.)

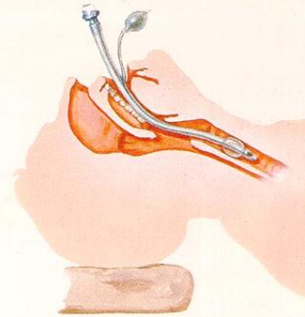
Look for needle marks, cyanosis, signs of trauma

Slide 3383

Initial Management of Severe Head Injuries

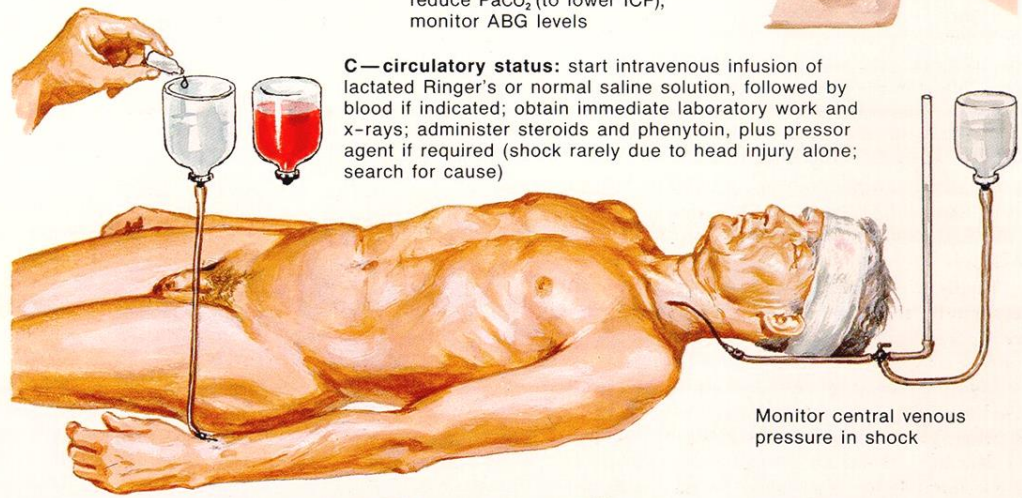
"ABC" assessment

A—airway: suction to free pharynx from blood and other material; intubate after cervical spine evaluation



B—breathing: evaluate rate, rhythm and breath sounds; ventilate to raise PaO_2 and reduce $PaCO_2$ (to lower ICP); monitor ABG levels

C—circulatory status: start intravenous infusion of lactated Ringer's or normal saline solution, followed by blood if indicated; obtain immediate laboratory work and x-rays; administer steroids and phenytoin, plus pressor agent if required (shock rarely due to head injury alone; search for cause)



Monitor central venous pressure in shock

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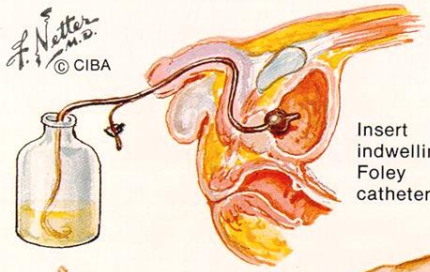
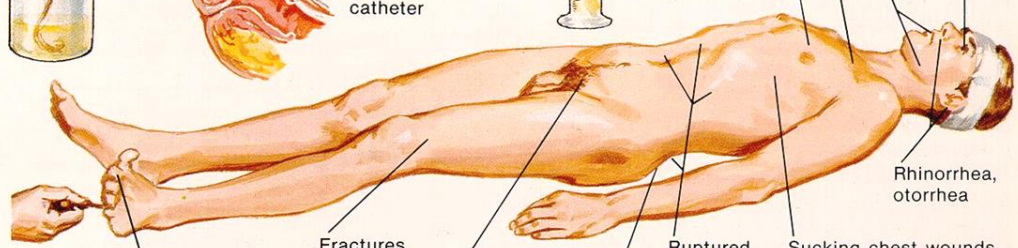


Insert indwelling Foley catheter

Measure urine flow hourly



Maxillary or mandibular fractures

Pupillary dilatation; ocular palsies

Ruptured aorta

Hemopericardium

Rhinorrhea, otorrhea

Babinski sign (neurologic examination)

Fractures, paralysis

Ruptured bladder

Back injuries

Ruptured spleen, liver, intestine, kidney

Sucking chest wounds, fractured ribs, flail chest, hemothorax, pneumothorax

Conduct complete physical examination and repeat periodically

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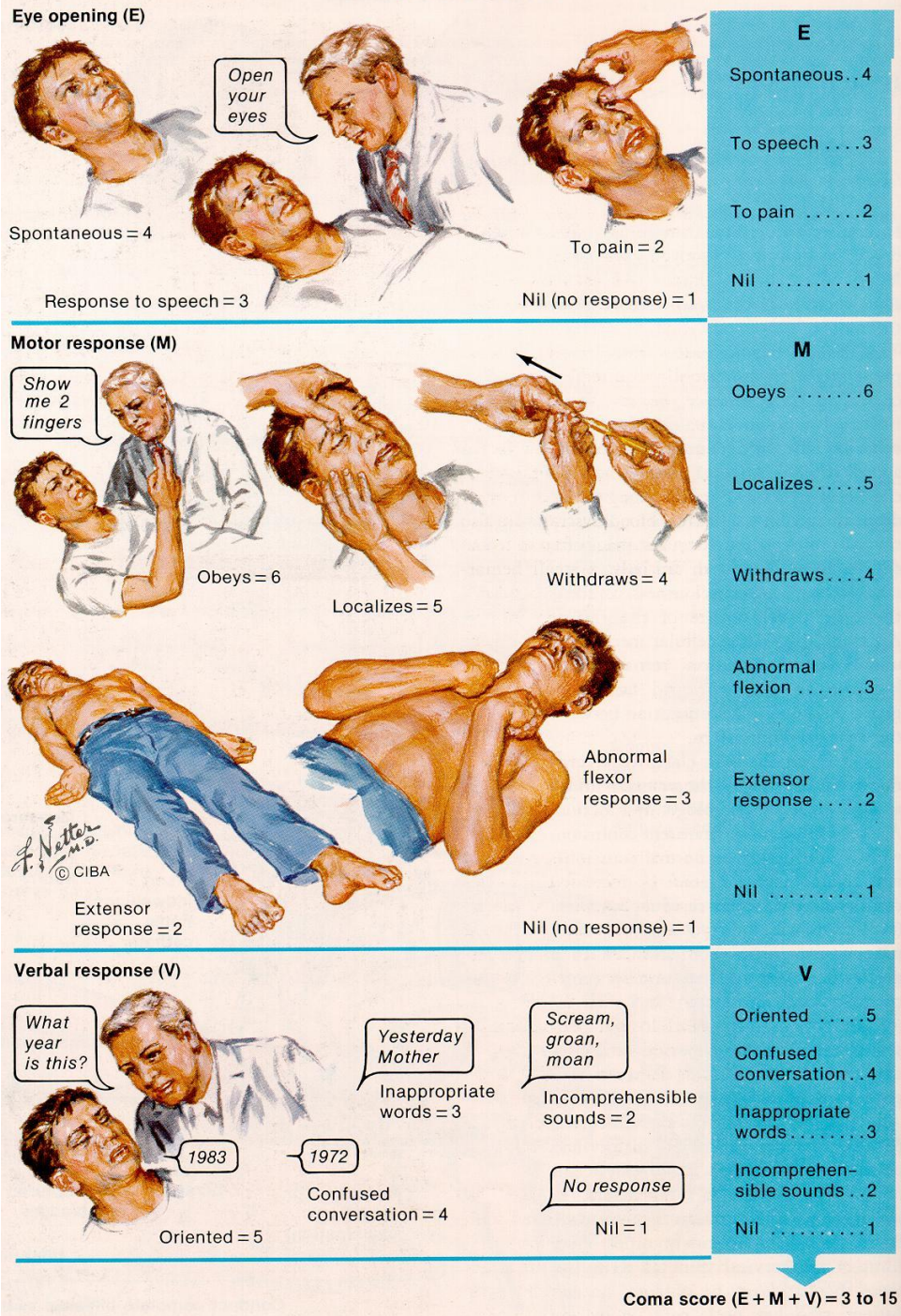
Glasgow Coma Scale



AMS / COMA

Always assess & stabilize ABC’s first

- airway with C-Spine immobilization / protection.
- Oxygenate!
- IV line , fluids, Thiamine 100mg IV,

Complete history and physical exam

C-Spine?

Labs / CT

Headaches

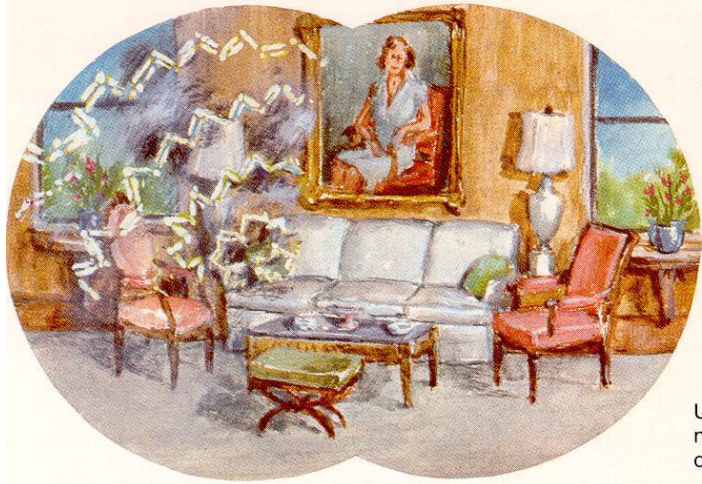
- History!!
- New or changing the characteristics
- Autonomous or symptom of some other dis?
- Neurol. symptoms?
  - Start with the simplest ther.
  - Go up till the max dosage
  - At the beginning of the HA

Headache

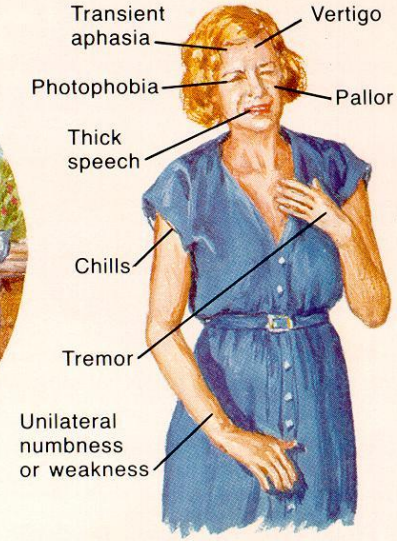
- Migraine
  - Severe headache either preceeded by a visual “aura”(scintillating scotoma or VF cut) or motor disturbance.
  - Nausea, vomiting, light sensitivity, sound sensitivity
- Factors that may provoke an attack include:
  - Menstruation, Sleep/food deprivation
  - Physical activity or certain foods (chocolate)
  - Contraceptives

Migraine

Aura

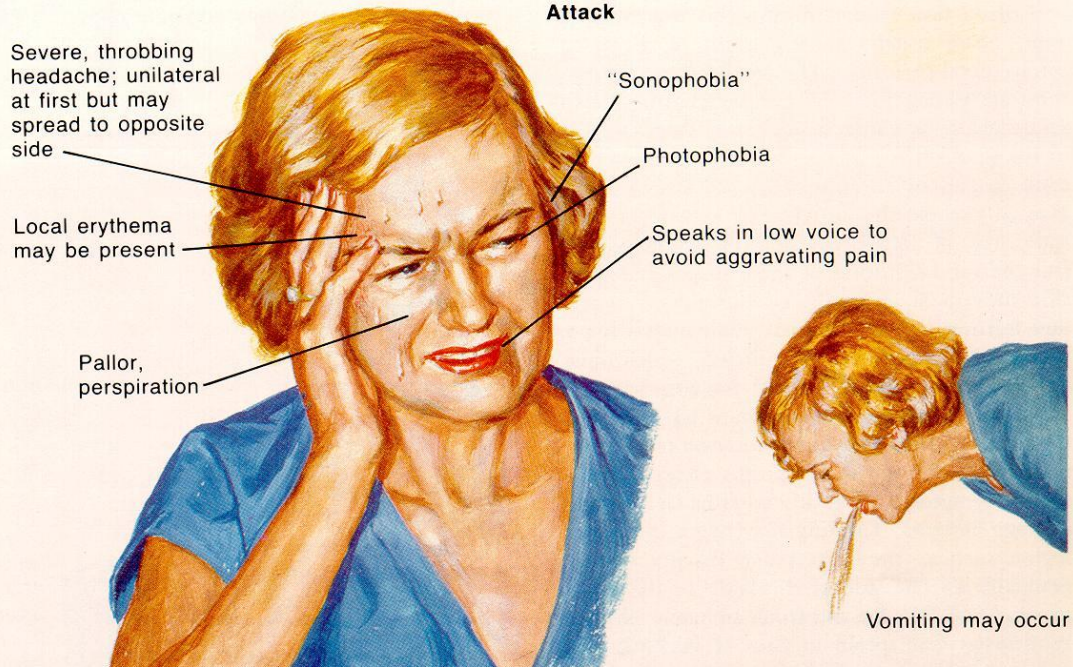


Visual disturbances, most common element of migraine aura: blurred cloudy vision, scotomas, scintillating zigzag lines (fortification spectrum), flashes of light, etc



Some other manifestations of aura, which may occur individually or in combination

Attack



Vomiting may occur

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Migraines

- History & PE
  - CRUCIAL to obtain HA history from patient
    - Is this HA similar to others or is it “worst HA of life”
    - Medications
    - Foods
    - Menses
  - FULL PE including Neuro exam

Migraine

Management

cool, quiet, dark environment

IV fluids if dehydrated

Abortive therapy:

- Phenothiazines (antimigraine and antiemetic)
- DHE (vaso/venoconstrictor) + antiemetic
- triptan (5-HT agonist)
- Opiods as LAST RESORT!!

Headaches

Cluster Headaches

boring headache on one side behind the eye.

facial flushing, tearing, nasal stuffiness

TX: 100% O2 by N/C at 6-8 l/min

- If no relief, Sumatriptan

Subarachnoid hemorrhage SAH

Abrupt onset of severe thunderclap “worst HA of life”.

Usually associated nausea and vomiting

Nonfocal neurologic exam (usually)

Etiology: aneurysm

DX: CT +LP A MUST

If CT (-), MUST LP

Temporal Arteritis

granulomatous inflammation of the external carotid artery

Clinically presents as:

- Severe unilateral HA over Temporal area
- Usually in middle aged females.
- PE reveals: a tender, warm, frequently pulseless temporal artery, with decreased visual acuity on the affected side.

Temporal Arteritis

DX:clinically + ESR elevation, usually >50 mm/h

Confirm with biopsy of artery

TX: HIGH dose steroids are VISION SAVING!

- Start on prednisone IMMEDIATELY
  - Prednisone 60 – 80 mg Q day

Vertigo

- **Central**

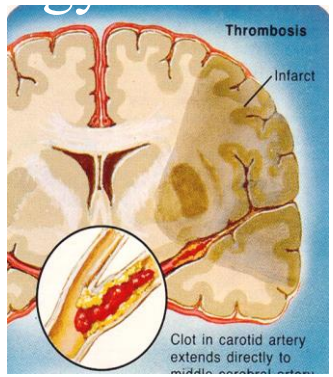
- mild
- foc. neurol. sympt.
- Deviation and nystagmus direction:the same

- **Peripheral**

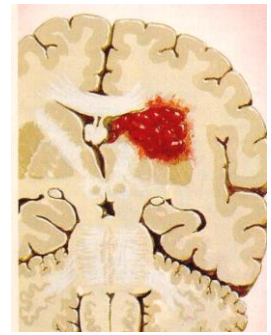
- intensive
- NO foc. neurol. Sympt.
- Deviation and nystagmus direction:different

Stroke
differential diagnosis---CT/MRI!!!

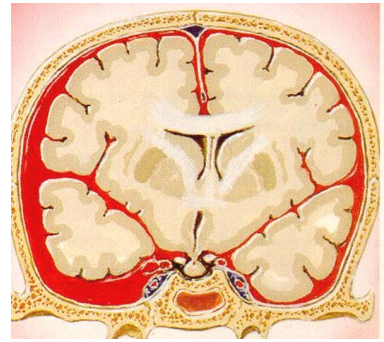
ischemia 80%



sp. Hemorrhage 10-15%



subarachnoidal bleeding



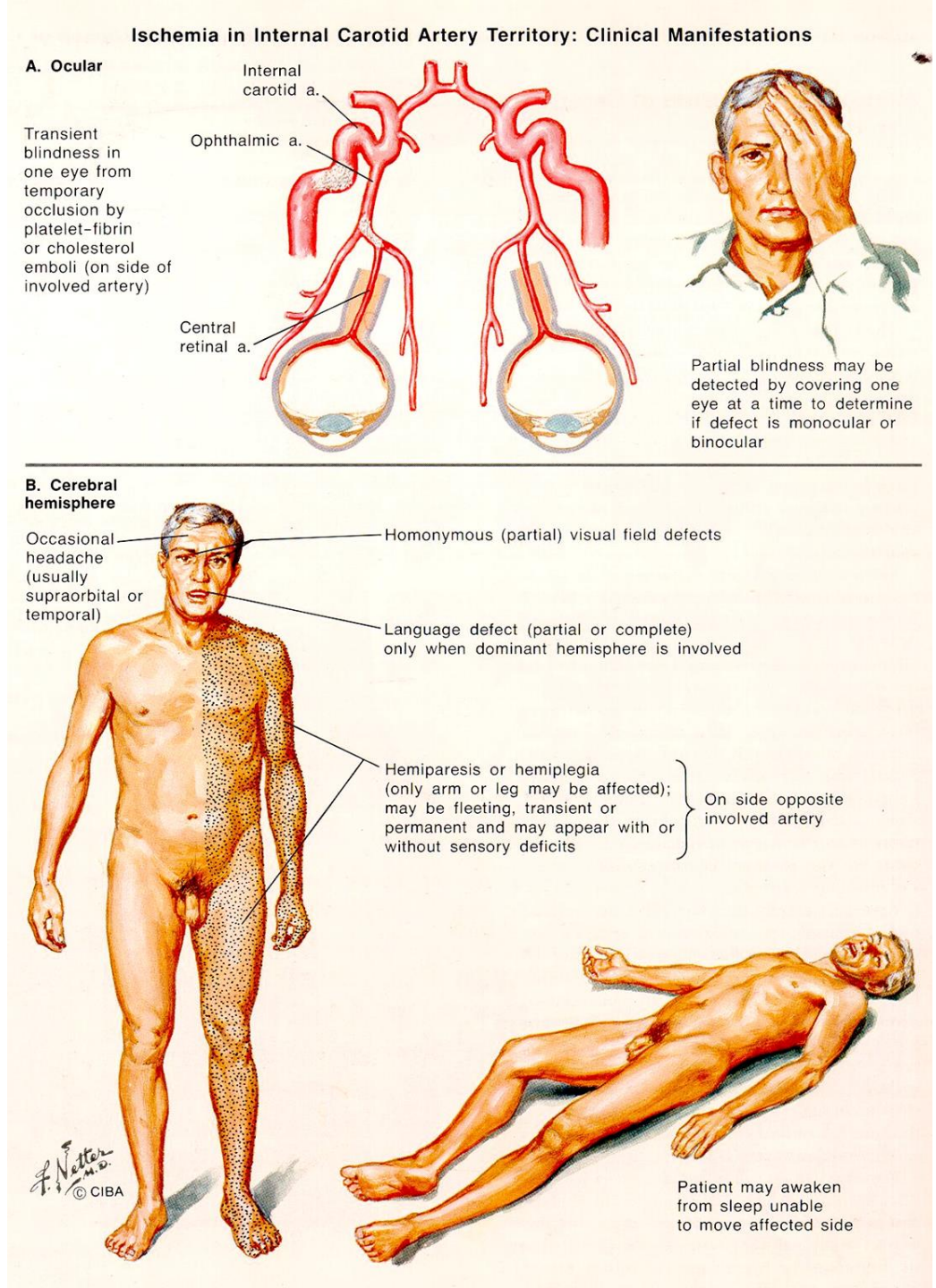
Ischemic Stroke Syndromes: thrombotic vs. embolic

Thrombotic Syndromes

- usually slow, progressive onset
- Sx develop shortly after awakening and are progressive

Embolic Syndromes

- Usually abrupt onset with maximal deficit that tends to improve


Ischemia in Internal Carotid Artery Territory: Clinical Manifestations
A. Ocular
Transient blindness in one eye from temporary occlusion by platelet-fibrin or cholesterol emboli (on side of involved artery)
Internal carotid a.
Ophthalmic a.
Central retinal a.
Partial blindness may be detected by covering one eye at a time to determine if defect is monocular or binocular
B. Cerebral hemisphere
Occasional headache (usually supraorbital or temporal)
Homonymous (partial) visual field defects
Language defect (partial or complete) only when dominant hemisphere is involved
Hemiparesis or hemiplegia (only arm or leg may be affected); may be fleeting, transient or permanent and may appear with or without sensory deficits
On side opposite involved artery
Patient may awaken from sleep unable to move affected side
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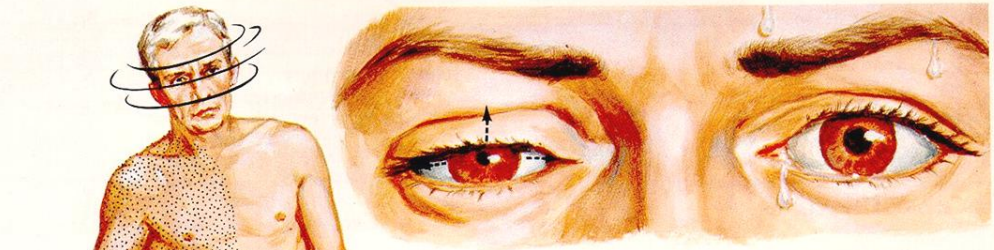
Ischemic Stroke Syndromes

Middle Cerebral Artery Occlusion (MCA)

- # 1 type
- Contralateral hemiplegia, hemianesthesia and homonymous hemianopsia
- Upper extremity deficit >> Lower extremity
- Aphasia (if dominant hemisphere involved)
- Conjugate gaze impaired

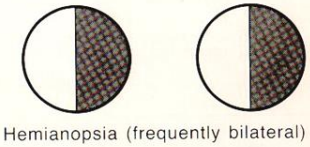
Ischemia in Vertebrobasilar Territory: Clinical Manifestations



Abnormal eye movements (cranial nerves III, IV and/or VI). Horner's syndrome may be present

Vertigo, ataxia; motor and sensory deficits, which may be unilateral, bilateral or alternating

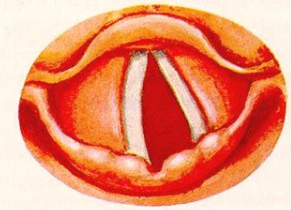
Motor and sensory deficits in face; unilateral, bilateral or alternating (cranial nerves V and VII)



Hemianopsia (frequently bilateral)

Headache, vomiting

Dysphagia (cranial nerve X)



Dysphonia (cranial nerve X)

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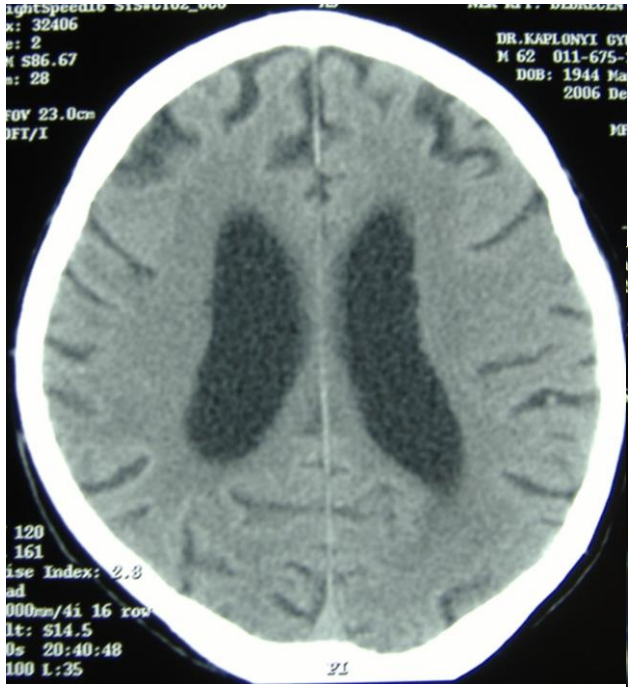
Altered consciousness (partial or complete) may be fleeting, transient or of long duration

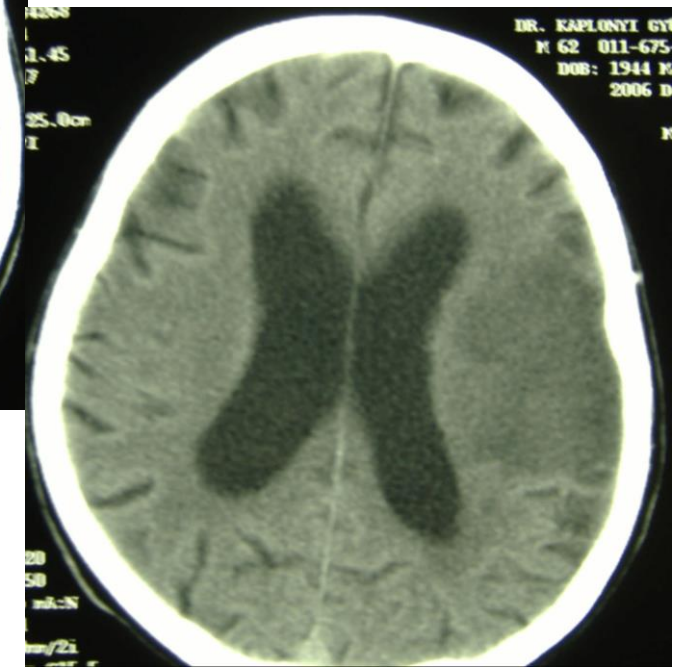
CT

62 yrs stroke at admission

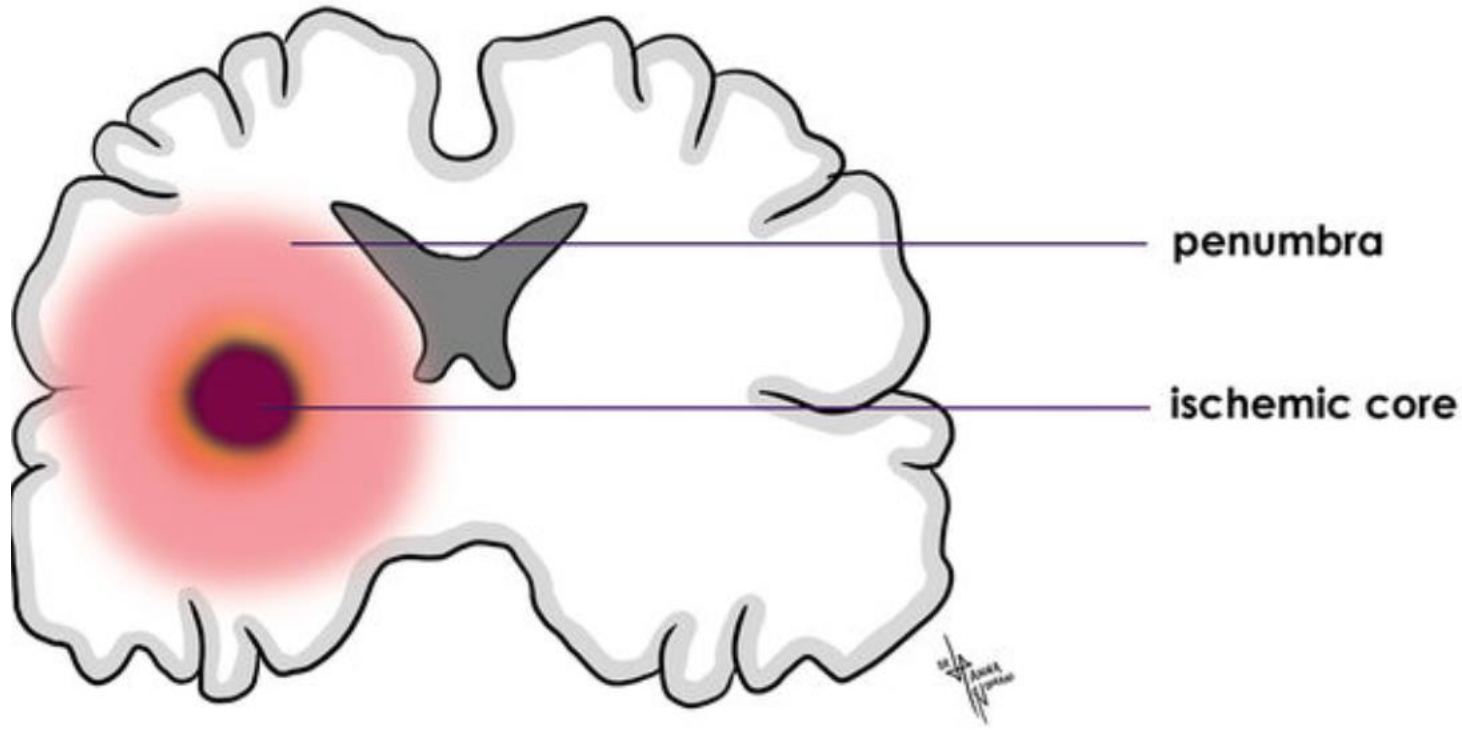


One day later

2 days later



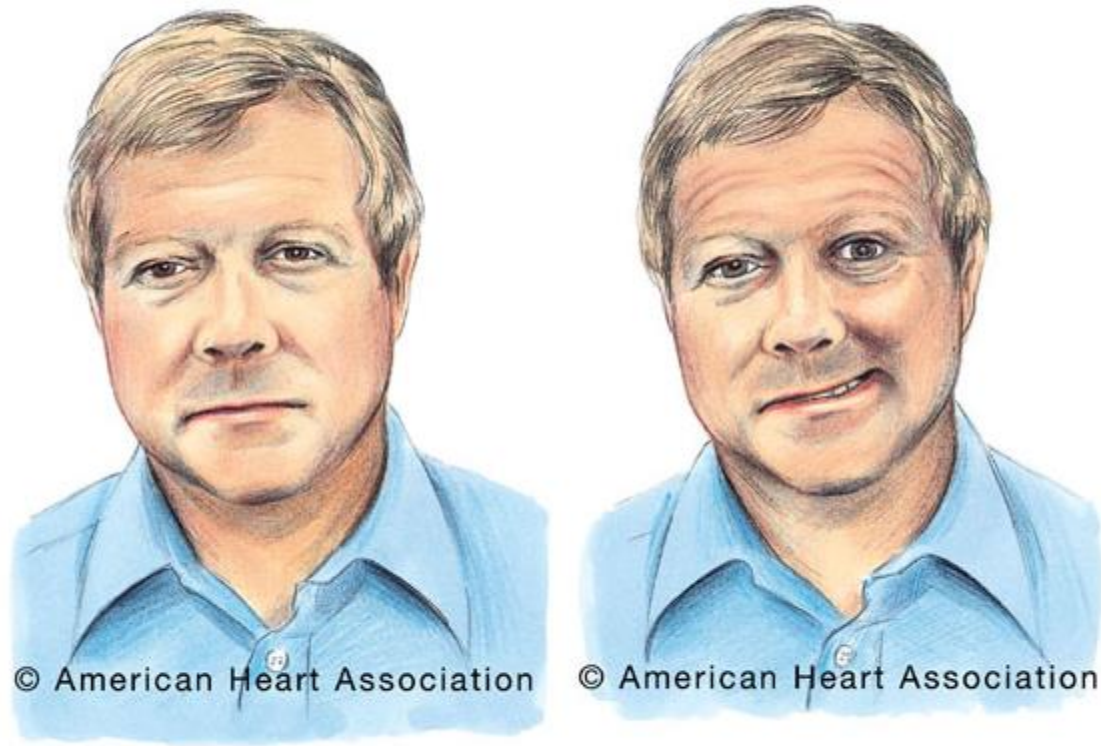
Ischemic Stroke



Cincinatti scale 1.

Cincinnati Stroke Scale

printable version PDF



Facial Droop

- *Normal*: Both sides of face move equally
- *Abnormal*: One side of face does not move at all

Cincinatti scale 2.

Arm Drift

- *Normal*: Both arms move equally or not at all
- *Abnormal*: One arm drifts compared to the other

Cincinatti scale 3.

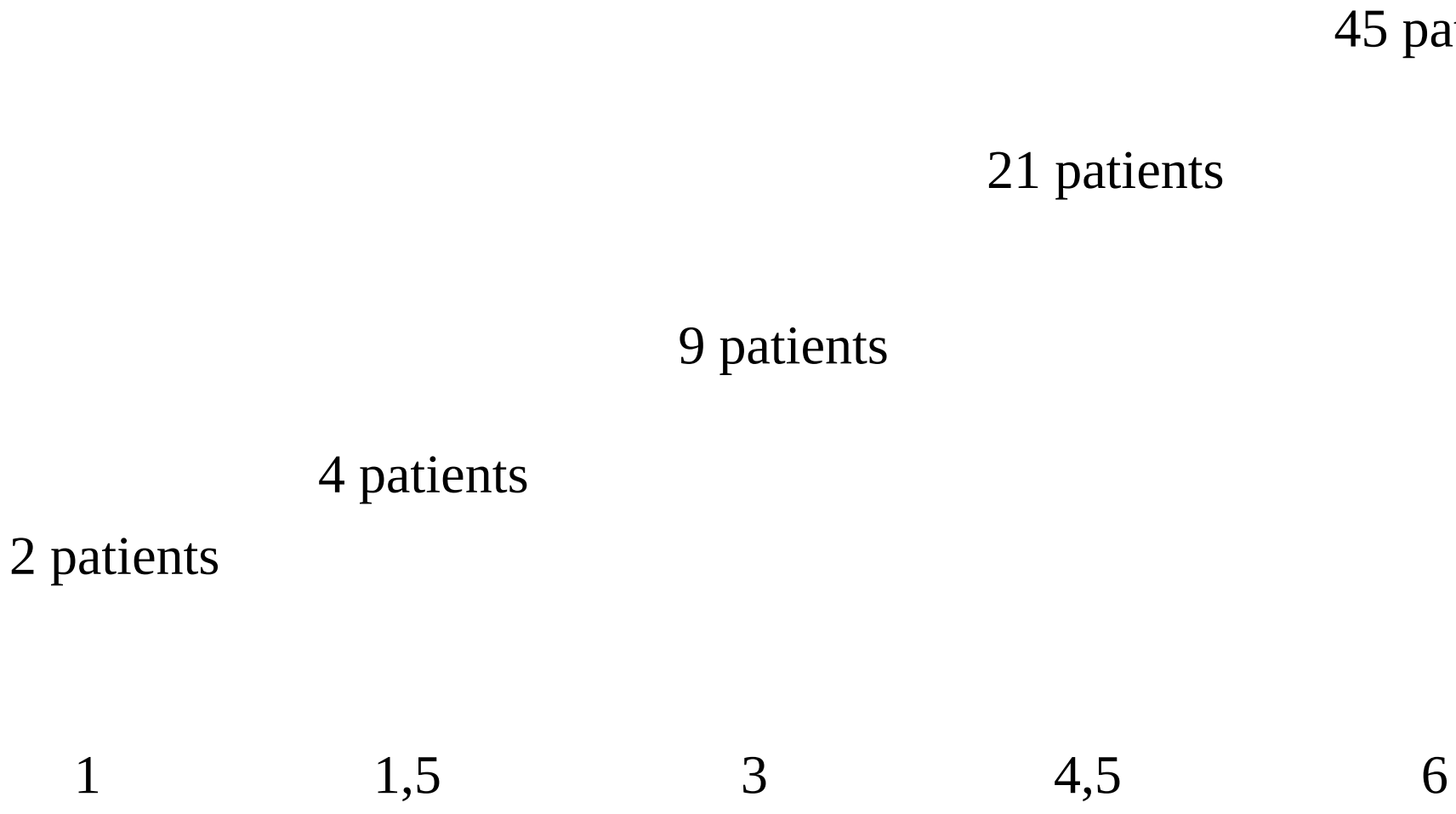
- Slurred speech, aphasia

TIA
(TRANSIENT ISCHEMIC ATT.)

- Transient symptoms
- Minutes
- No residual tissue deficit (diff. MRI?)

TIA is emergency!!! High risk of devastating stroke

Open the artery as soon as possible



Time window: depends on the occluded vessel and elapsed time eafter stroke?

Within 4,5 hours (some subgroups 3 hours) **iv.** lysis if small vessel occlusion

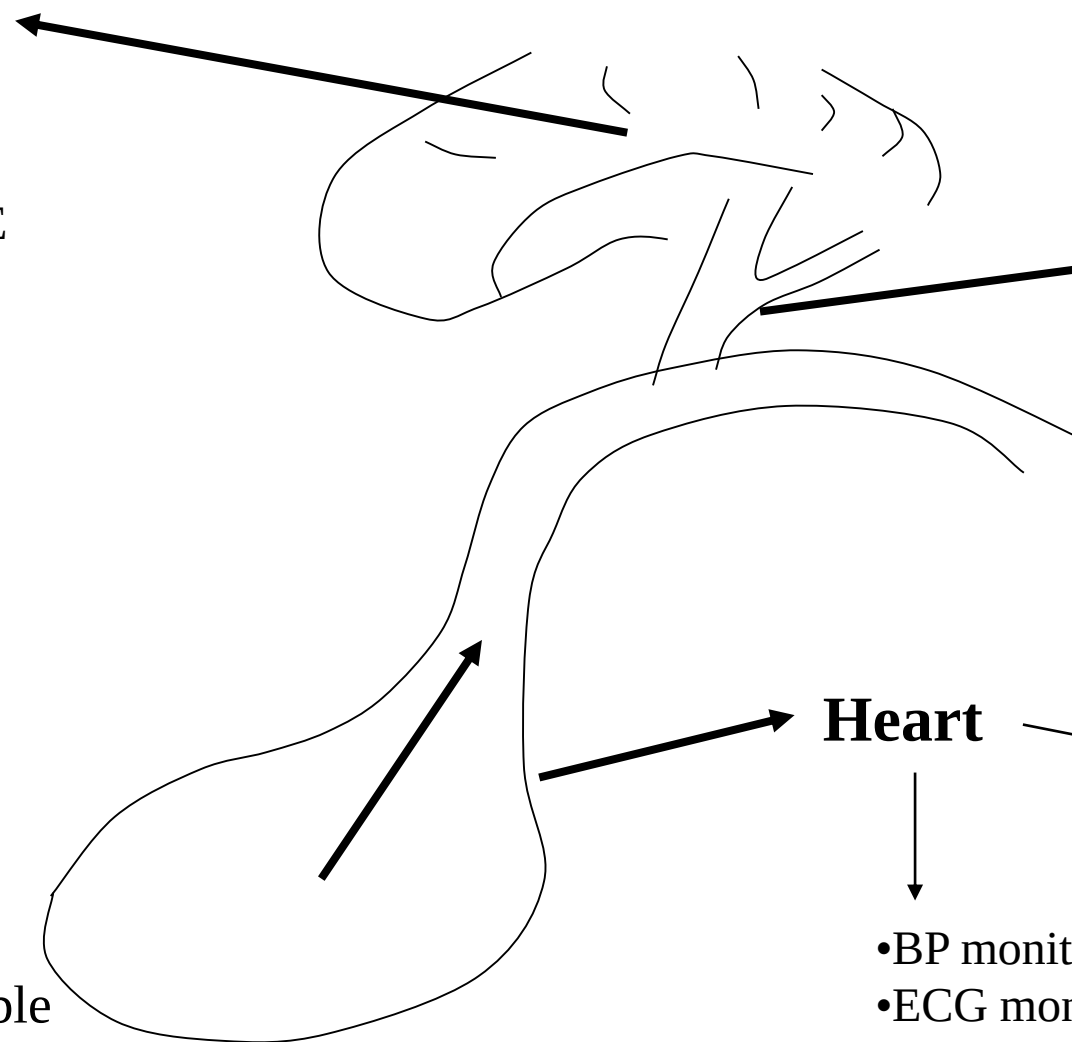
6-8 hours IF ICA or MCA occlusion:intraarterial or mechanical thrombectomy (MET) BUT start with iv. If specific constellation of MRI and sympt. up till 24 h!!!

12 h if basilar artery occluded either **iv or ia. lysis**

If out of time window?

- 100-300 mg aspirin
- Monitoring of BP and ECG
- Do NOT decrease BP till 220/110 Hgmm!
- Pulsoximetry, 2-4 lit oxigen, if less 94%
- Normoglycemia
- LMWH or heparin to prevent DVT deep venous thromb.
- Nasogastric tube if dysphagia
- Antipyretic ther.
- If seizure antiepilept.
- antibiotics

Diagnosis acute stroke



•CT+CT angio!!

•MRI
- •Diff. WI+GRE

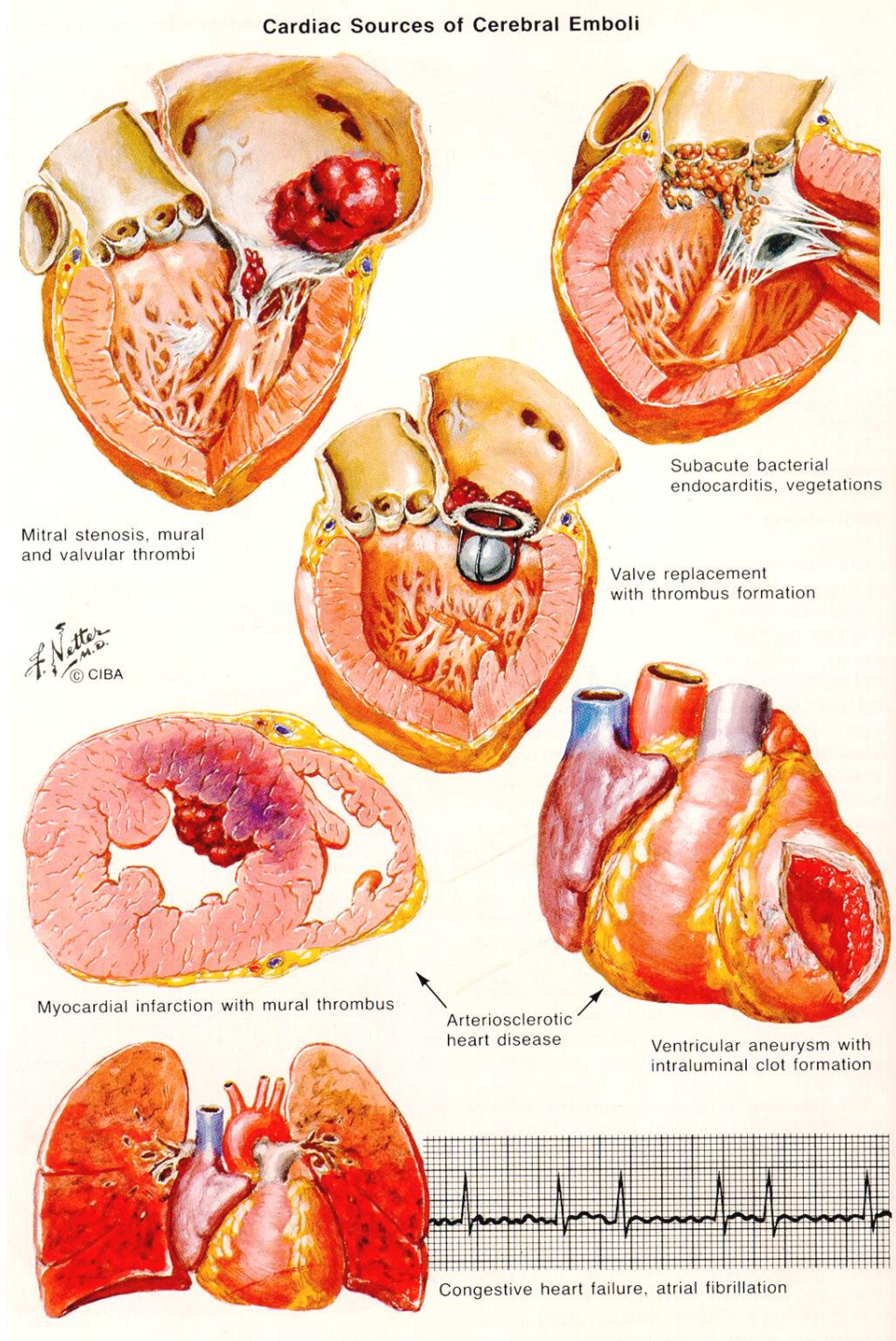
Carotid, vertebral
- •US
- •CTA
- •MRA
- •DSA

Blood
- •platelets
- •Hgb
- •glucose
- •INR!
- •O2 sat
- •hypercoagulable

Heart

TTE
TEE

- •BP monitoring
- •ECG monitoring



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Subarachnoid Hemorrhage SAH

Highest incidence in 35-65 year old.

Usually from the rupture of a berry aneurysm

Clinically:

- abrupt onset of "worst headache of life"
- Nuchal rigidity, photophobia, vomiting, retinal hemorrhages.

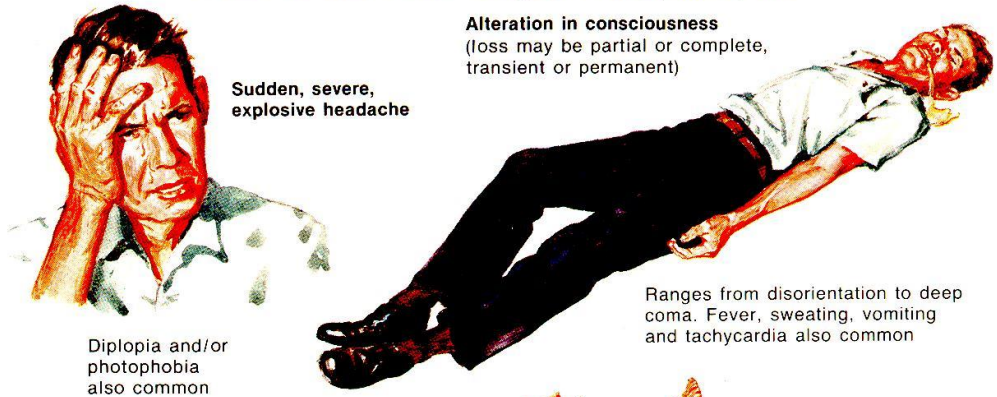
Diagnosis: CT + LP!!!!

- CT only 92% sens. within 24 hours of event, loses sensitivity >24 hours out from headache.
- 72 hours out CANNOT r/o without LP!

Management:

- Consider adding Nimodipine 60 mg Q6 to reduce vasospasm

Clinical Manifestations of Congenital Aneurysm Rupture



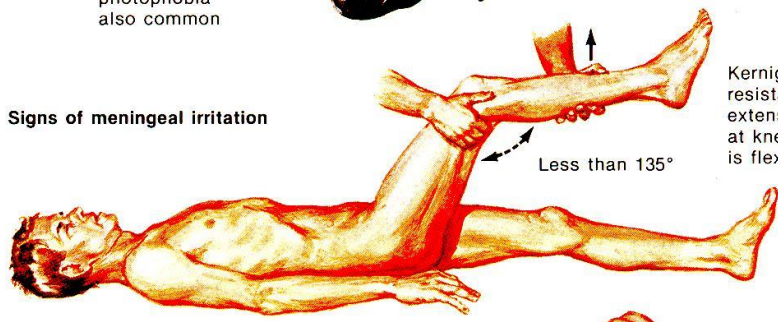
Sudden, severe, explosive headache

Alteration in consciousness (loss may be partial or complete, transient or permanent)

Ranges from disorientation to deep coma. Fever, sweating, vomiting and tachycardia also common

Diplopia and/or photophobia also common

Signs of meningeal irritation



Kernig's sign: resistance to full extension of leg at knee when hip is flexed

Less than 135°

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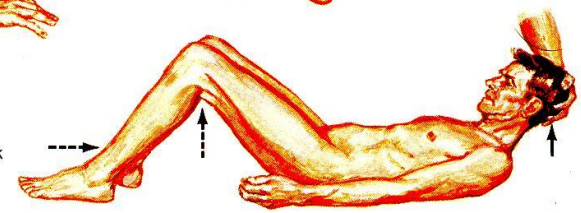
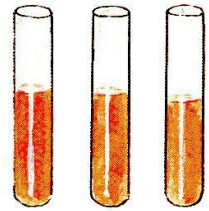
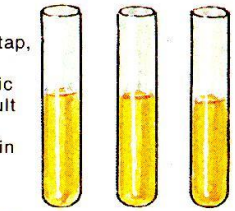


Brudzinski's sign: flexion of both hips and knees when neck is passively flexed

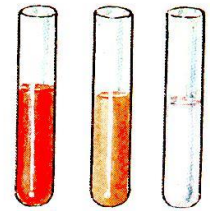
Cerebrospinal fluid



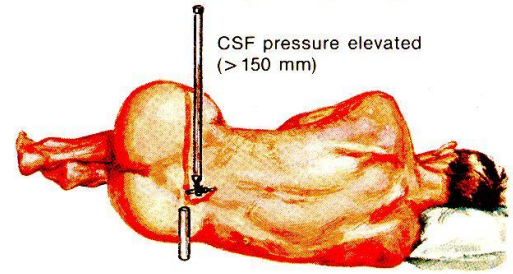
Three successive fluid samples collected. Shortly after or during bleeding, all 3 samples frankly bloody or orange



Later, on repeat tap, all 3 samples are xanthochromic (yellow) as a result of hemoglobin release or bilirubin formation

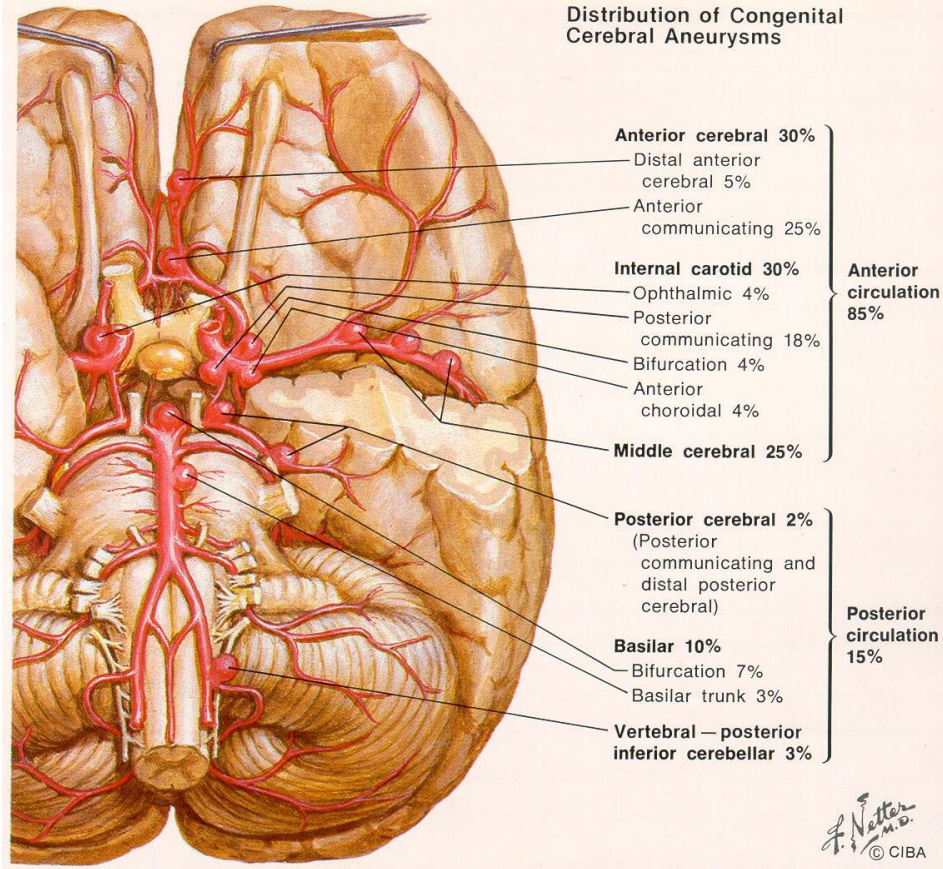


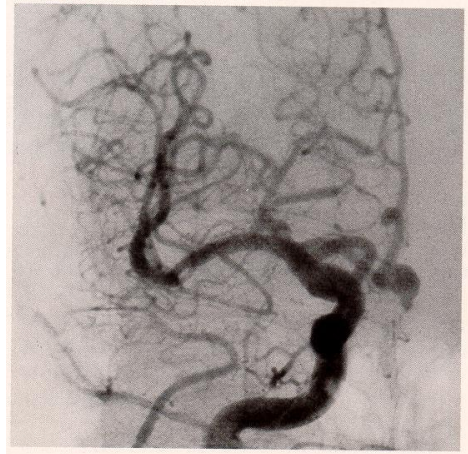
If blood is due to traumatic tap, fluid clears progressively in successive samples



CSF pressure elevated (>150 mm)

Distribution of Congenital Cerebral Aneurysms





Frontal carotid arteriogram disclosing bilobate aneurysm of anterior communicating artery

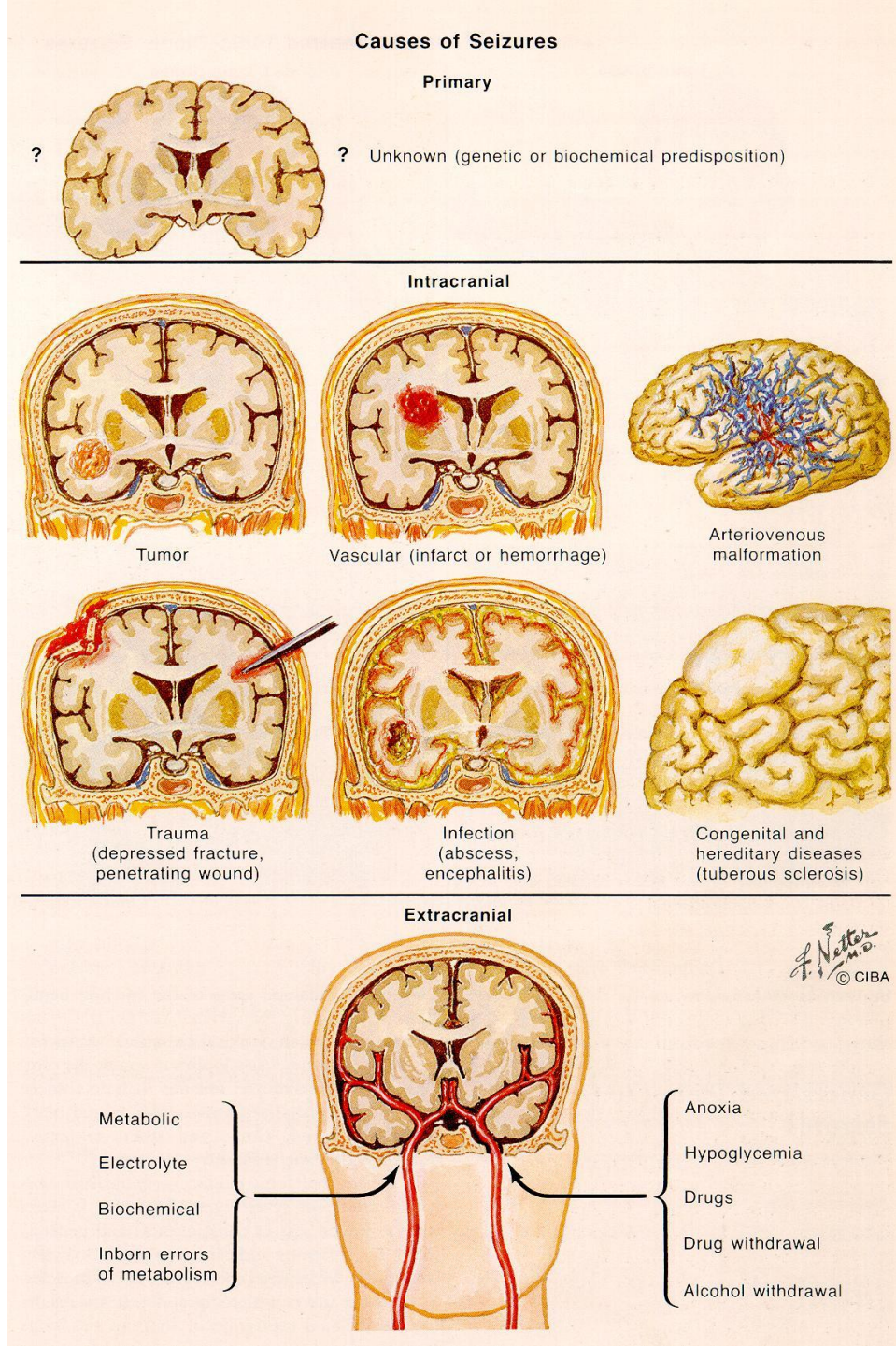
Different patient: lateral view showing large aneurysm of internal carotid artery at origin of posterior communicating artery

Seizure

Seizures & Status Epilepticus

Background:

- 1 – 2% of the general population has seizures
- Primary
  - Idiopathic epilepsy:onset ages 10-20
- Secondary
  - Intracranial pathology
    - Trauma, Abscess, Infarct, tumor
  - Extracranial Pathology
    - Toxic, metabolic, hypertensive, eclampsia



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Seizure Types

Generalized Convulsive Seizures (Grand Mal):

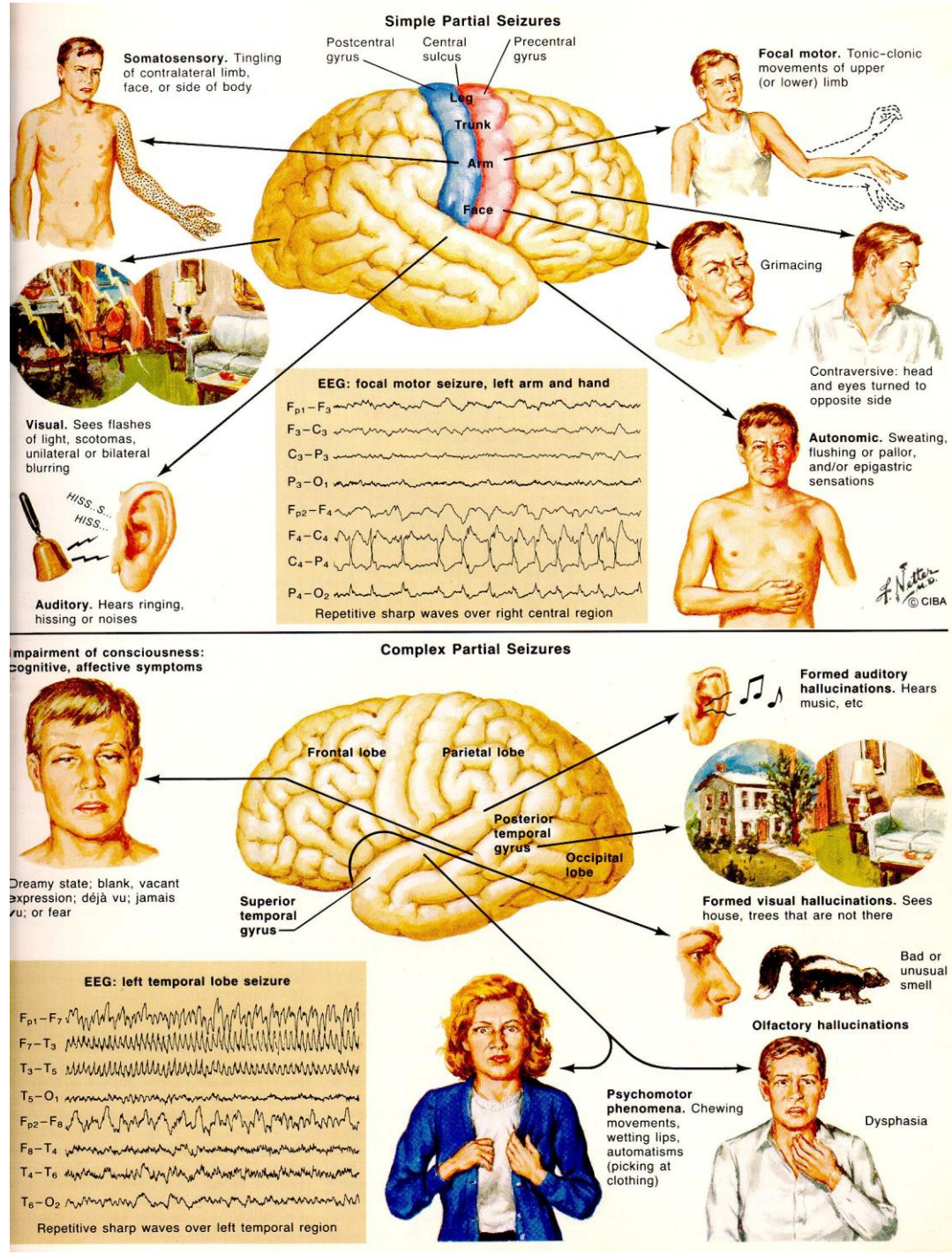
- Tonic , clonic movements, (+) LOC, apnea, incontinence and a post-ictal state

Non Convulsive Seizures (Petit Mal)

- Absence seizures – “blank staring spells”
- Myoclonic – brief contractions of selected muscle groups

Partial Seizures

- Characterized by presence of auditory or visual hallucinations
- Simple = somatic complaints + no LOC
- Complex = somatic complaints + Altered Mental Status or LOC


Simple Partial Seizures
Postcentral gyrus
Central sulcus
Precentral gyrus
Leg
Trunk
Arm
Face
Somatosensory. Tingling of contralateral limb, face, or side of body
Focal motor. Tonic-clonic movements of upper (or lower) limb
Grimacing
Contraversive: head and eyes turned to opposite side
Visual. Sees flashes of light, scotomas, unilateral or bilateral blurring
HISS...S...
HISS...
Auditory. Hears ringing, hissing or noises
EEG: focal motor seizure, left arm and hand
Fp1–F3
F3–C3
C3–P3
P3–O1
Fp2–F4
F4–C4
C4–P4
P4–O2
Repetitive sharp waves over right central region
Autonomic. Sweating, flushing or pallor, and/or epigastric sensations
F. Netter M.D.
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Complex Partial Seizures
Impairment of consciousness: cognitive, affective symptoms
Dreamy state; blank, vacant expression; déjà vu; jamais vu; or fear
Frontal lobe
Parietal lobe
Posterior temporal gyrus
Occipital lobe
Superior temporal gyrus
Formed auditory hallucinations. Hears music, etc
Formed visual hallucinations. Sees house, trees that are not there
Bad or unusual smell
Olfactory hallucinations
EEG: left temporal lobe seizure
Fp1–F7
F7–T3
T3–T5
T5–O1
Fp2–F8
F8–T4
T4–T6
T6–O2
Repetitive sharp waves over left temporal region
Psychomotor phenomena. Chewing movements, wetting lips, automatisms (picking at clothing)
Dysphasia

Approach for 1st Seizure, New Seizure, or Substance/ Trauma Induced Seizure

- As always ABC's First
- IV, O2, Monitor.
  - Send blood for CBC, Chem 20, Tox screen as appropriate
  - Anticonvulsant levels
  - Prolactin levels / Lactate level
- CXR / UA/ Head CT
- Is patient actively seizing? Post ictal? Pseudoseizure?
  - Consider treatment options
- Complete History and Physical Exam
  - Including detailed Neuro Exam
  - Repeat Neuro evaluations a must!

Approach to Breakthrough Seizure

As Before, But History, History, History!!

Main causes of Breakthrough Seizure:

- Noncompliance with anticonvulsant regimen
- Start of new medication (level alteration)
  - Antibiotics, OCP’s
- Infection
  - Fever
- Changes in body habitus, eating patterns

Status Epilepticus

Definition:operationally defined as seizure lasting greater than 5 minutes OR two seizures between which there is incomplete recovery of consciousness.

Treatment algorhythm:

- As before ABC's
- IV, O2, Monitor
- Consider ALL potential causes
  - INH (pyridoxime/B-6 deficiency)
  - Eclampsia
  - Alcoholic (thiamine/B-1 deficiency)
  - Other Tox ingestion (TCA's, sulfonylurea)
  - Trauma

Status Epilepticus Treatment

FIRST LINE TREATMENT

Lorazepam 2mg/min IV up to 10 mg max.

OR Diazepam 5mg/min IV or PR up to 20mg

SECOND LINE TREATMENT

Phenytoin or Fosphenytoin

20mg/kg IV at rate of 50mg/min

THIRD LINE TREATMENT

Get Ready to intubate at this point!!

Phenobarbial 10-20mg/kg @ 60 mg/min

Status Epilepticus Treatment

- FINAL TREATMENT
  - Barbiturate Coma
    - Pentobarbitol 5mg/kg @ 25 mg/min
    - Stat Neurology consult for evaluation and EEG
    - Pentobarbitol titrated to EEG response.
- Always get a through HISTORY
  - Possible trauma
  - Medications in house
  - Other diseases?
  - Overall appearance of patient

Status Epilepticus Adjunctive Treatment by History

- Thiamine 100mg IV, 1-2 amps D 50
  - If suspect alcoholic, malnourished, hypoglycemia
- Magnesium Sulfate 20cc of 10% solution
  - As above of if eclampsia (BP does NOT have to be 200/120!!)
- Pyridoxine 5 gms IV
  - INH or B-6 deficiency

Why emergency?

Guillain-Barré sy **respir. Insuffic.**


Acute Spinal Cord Syndromes: Evolution of Symptoms
Back pain: onset acute or gradual
Numbness of limbs
Weakness
Progression over minutes, hours or few days
EMERGENCY ROOM
LADIES
Urinary urgency
F. Netter M.D.
© CIBA
Paralysis (may occur without premonitory symptoms)

Emergent Peripheral Neuropathies

- Guillain-Barre Syndrome
  - Most common acute polyneuropathy.
  - 2/3's of patients will have preceeding URI or gastroenteritis 1-3 weeks prior to onset.
  - Presents as:paresthesias followed by ascending paralysis starting in legs and moving upwards.
    - Remember Miller-Fischer variant: has minimal weakness and presents with ataxia, areflexia, and ophthalmoplegia.
  - DX:LP will show cytochemical dissociation (only days after the onset!).
    - Normal cells with HIGH protein.
  - TX: Self limiting, early and aggressive airway stabilization.

Emergent Peripheral Neuropathies

Myasthenia Gravis

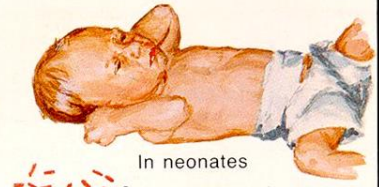
- Most common disorder of neuromuscular transmission.
- An autoimmune disease that destroys acetylcholine receptors (AchR) which leads to poor neurotransmission and weakness.
- Proximal >> Distal muscle weakness
- Commonly will present as:
  - Muscle weakness exacerbated by activity and is relieved by rest
- Clinically:ptosis, diplopia and blurred vision are the most common complaints. Pupil is spared!

Emergent Peripheral Neuropathies

- Myasthenia Gravis
  - Myasthenic crisis = a true emergency!!
  - Occurs in undiagnosed or untreated patients
    - Due to relative Ach (acetylcholine) deficiency
    - Patients present with profound weakness and impending respiratory failure
  - TX:Stabilize and manage airway
    - Consider edrophonium 1 -2 mg IV
      (AchE inhibitor)
    - Plasmapheresis and/or immunglobulin therapy

Bacterial Meningitis

Most common causative organisms

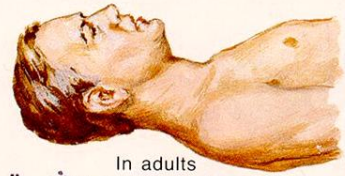


In neonates

- Gram-negative bacilli, 50% (*E coli*, *H influenzae*, etc)
- Streptococci, 20%
- Other (*S aureus*, etc)

In children

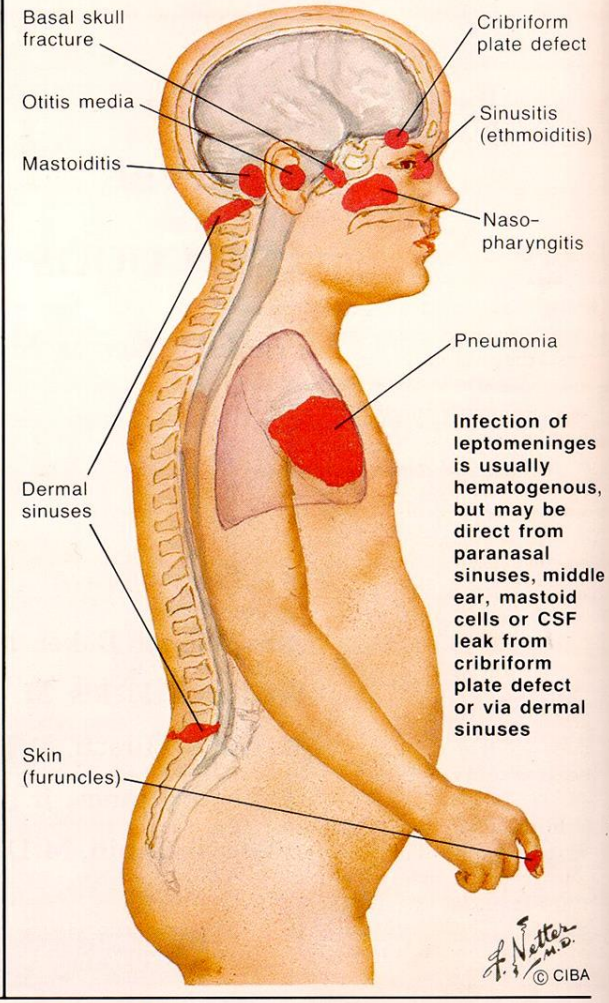
- *H influenzae*, 50%
- *N meningitidis*, 25%
- Other (*Listeria*, etc)



In adults

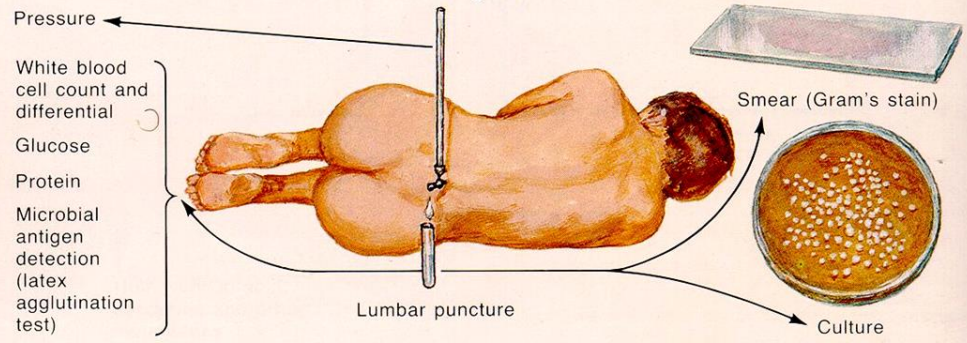
- *S pneumoniae*, 30%
- *N meningitidis*, 15%
- Gram-negative bacilli
- Other (*Listeria*, etc)

Sources of infection



Infection of leptomeninges is usually hematogenous, but may be direct from paranasal sinuses, middle ear, mastoid cells or CSF leak from cribriform plate defect or via dermal sinuses

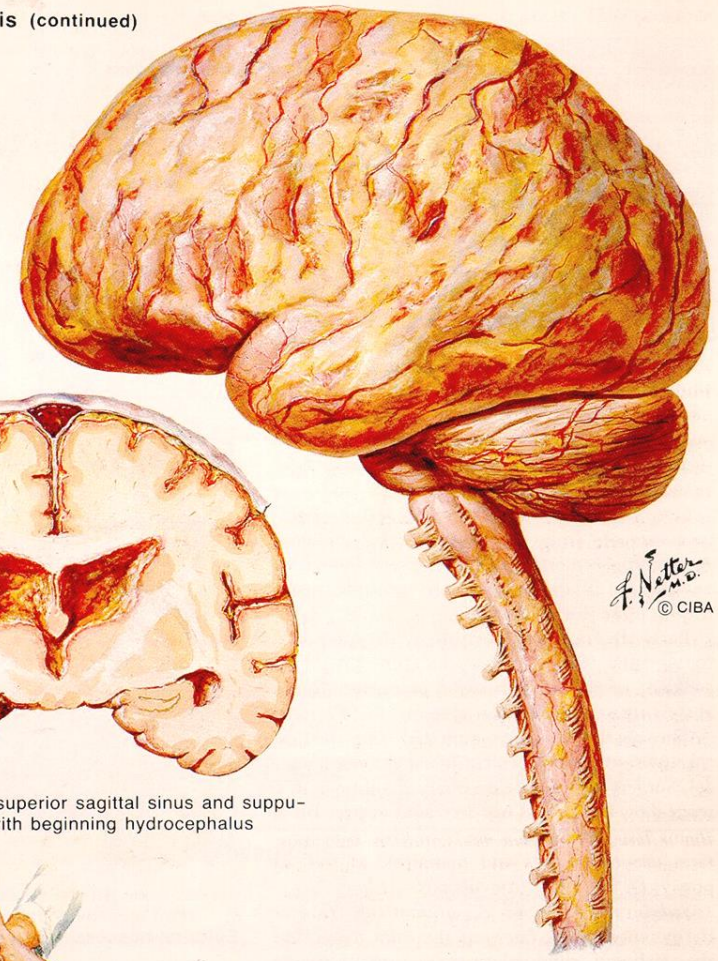
Diagnosis



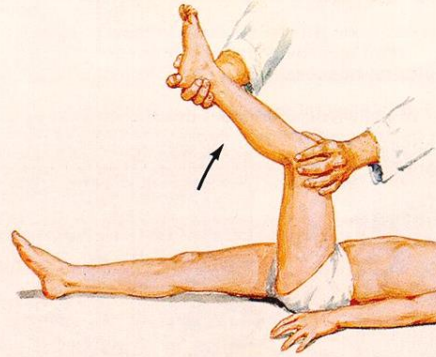
Netter Atlas

Bacterial Meningitis (continued)

Inflammation and suppurative process on surface of leptomeninges of brain and spinal cord



Thrombophlebitis of superior sagittal sinus and suppurative ependymitis, with beginning hydrocephalus



Kernig's sign. Patient supine, with hip flexed 90°. Knee cannot be fully extended

Neck rigidity (Brudzinski's neck sign). Passive flexion of neck causes flexion of both legs and thighs

Infectious Neurologic Emergencies

Meningitis: inflammation of the meninges

History:

- Acute Bacterial Meningitis:
  - Rapid onset of symptoms <24 hours
    - Fever, Headache, Photophobia
    - Stiff neck, Confusion

Meningitis

- Lymphocytic Meningitis (Aseptic/Viral)
  - Gradual onset of symptoms as previously listed over 1-7 days.
  - Etiology:
    - Viral
- Atypical Meningitis
  - History (medical/social/environmental) crucial
  - Insidious onset of symptoms over 1-2 weeks
  - Etiology:
    - TB(#1)
    - Coccidiomycosis, cryptococcus

Meningo-encephalitis

Physical Exam Pearls

- Infants and the elderly lack the usual signs and symptoms, only clue may be headache
- Look for papilledema, focal neurologic signs, ophthalmoplegia and rashes
- As always full exam
  - Checking for above
  - Brudzinski's sign
  - Kernig's sign
- KEY POINT:If you suspect meningococcemia do NOT delay antibiotic therapy, MUST start within 20 minutes of arrival!!!!!

Meningitis

Emergent CT Prior to LP

- Those with profoundly depressed MS
- Seizure
- Head Injury
- Focal Neurologic signs
- Immunocompromised with CD4 count <500

DO NOT DELAY ANTIBIOTIC THERAPY!!

Meningitis

Lumbar Puncture Results

TEST	NORMAL	BACTERIAL	VIRAL
Pressure	<170	>300	200
Protein	<50	>200	<200
Glucose	>40	<40	>40
WBC's	<5	>1000	<1000
Cell type	Monos	>50% PMN's	Monos
Gram Stain	Neg	Pos	Neg

Meningitis Management

Antibiotics By Age Group

Neonates(<1month) = Ampicillin + Gent. Or Cefotaxime + Gent

Infants (1-3mos) = Cefotaxime or Ceftriaxone+ Ampicillin

Children (3mos-18yrs) = Ceftriaxone

Adults (18yr-up) = Ceftriaxone + Vancomycin

Elderly/Immunocomp = Ceftriaxone +Ampicillin +Vancomycin

Meningitis Management

Steroids

In children, dexamethasone has been shown to be of benefit in reducing sensiorneural hearing loss, when given before the first dose of antibiotic.

Indications:

- Children> 6 weeks with meningitis due to H. flu or S. pneumo.
- Adults with positive CSF gram stain

Dose: 0.15mg/kg IV

Encephalitis

Always think of in the young/elderly or immunocompromised with FEVER + AMS

Common Etiologies:

Viral

- West Nile
- Herpes Simplex Virus (HSV)
- Varicella Zoster Virus (VZV)
- Arboviruses
  - Eastern Equine viruses
  - St. Louis Encephalitis

Encephalitis

Defined as: inflammation of the brain itself

Most cases are self limited, and unless virulent strain, or immunocompromised, will resolve.

The ONLY treatable forms of encephalitis are:

- HSV
- Zoster

Encephalitis

Management:

- Emergent MR
- ABC's with supportive care.
- Lumbar puncture:
  - Send for ELISA and PCR
- Acyclovir 10 mg/kg Q 8 hours IV for HSV and Zoster
- Steroids not shown to be of benefit.

Headache

Complications	Head trauma
Fluid-elektrolyte	>50% aldosterone and ADH: sodium and fluid retention
Respiratory system	aspiration, infection and atelectasia, adult-respiratory-distress syndrome (ARDS)
Gastrointestinal	stomach erosion
Cardiovascular	cardiac arhythmias, arrest
Hemostasis	DIC, coagulopathy, 5% - 10%

Closed head Injury Facts

The single most important factor in the neurologic assessment of the head injured patient is level of consciousness (LOC)

Always assume multiple injuries with serious mechanism.

ESPECIALLY C - SPINE!!!!

Unless hypotensive WITH bradycardia and WARM extremities (spinal cord injury); hypotension is ALWAYS secondary to hypovolemia from blood loss in the trauma patient!

The most common intracranial bleed in CHI is subarachnoid hemorrhage.

Closed Head Injuries with Hemorrhage

Cerebral Contusion

- Focal hemorrhage and edema under the site of impact.
- Susceptible areas are those in which the gyri are in close contact with the skull
  - Frontal lobe
  - Temporal lobes
- Diagnostic Test of Choice:NC Head CT
- Treatment: Supportive with measures to keep ICP normal.
- Repeat Neuro checks.
- Repeat Head Ct in 24 hours.

Contusion

Subdural Hematoma

Occurs secondary to acceleration/decelleration injury with resultant tearing of the bridging veins that extend from the subarachnoid space to the dural sinuses.

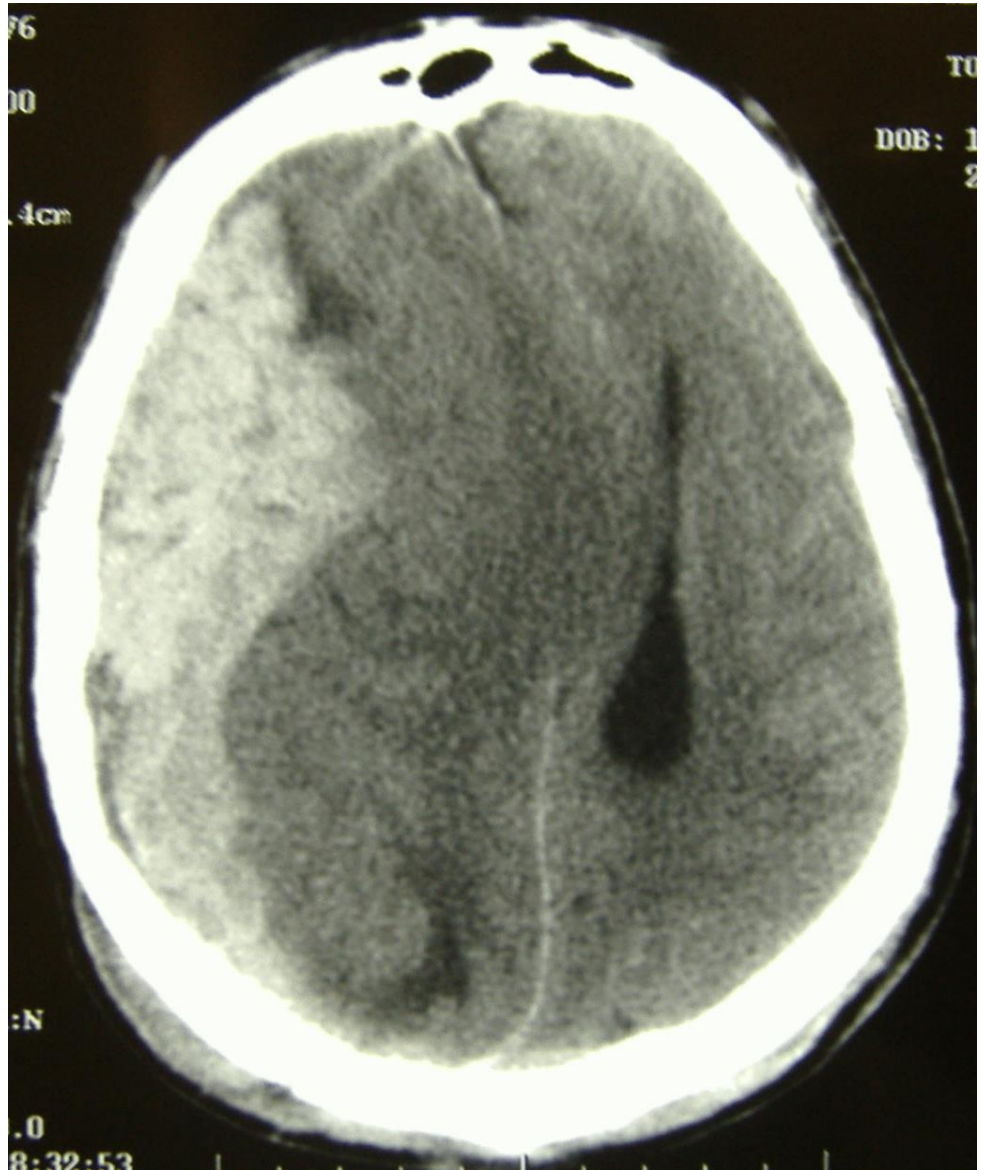
Blood dissects over the cerebral cortex and collects under the dura overlying the brain.

Patients at risk:

- alcoholics
- elderly
- anticoagulant users

Appears as “sickle shape” and does not extend across the midline

Subdural hematoma



Epidural Hematoma

Occurs from blunt trauma to head especially over the parietal/temporal area.

Presents as LOC which then patient has lucid interval then progressive deterioration, coma , death. (Patient talks to you & dies!)

Commonly associated with linear skull fracture

Mechanism of bleed is due to tear of artery, usually middle meningeal.
Sometimes ipsilateral pupillary dilatation with contralateral hemiparesis.

CT Scan :a BICONVEX (lens) density which can extend across the midline

Epidural hematom

Management of Closed Head Injuries

As always ABC's with C-Spine precautions

IV, O2, Monitor.

Stabilize and resuscitate

- Sao2>95%
- SBP>90
- Treat Fever

Head of Bed 30% (once C-Spine cleared)

Stat head CT with Stat Neurosurgical evaluation for surgical lesions.

Repeat Exams, looking for signs of herniation.

Signs of Herniation / Increased ICP

Headache, nausea, vomiting

Decreasing LOC

Decreased respiratory rate

Cushing reflex (hypertension/bradycardia/bradynpea)

Papilledema

Development of signs of herniation

- Fixed and dilated pupil
- Contralateral hemiparesis
- Posturing