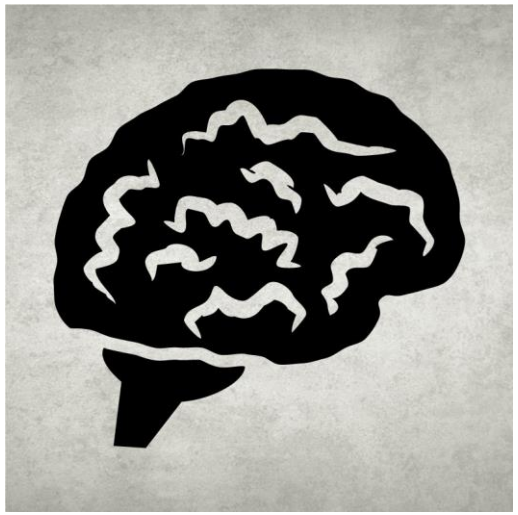
EMERGENCY MEDICINE

BOOT CAMP

INTRODUCTION TO NEUROLOGY



José A. Rubero, MD, FACEP, FAAEM

Professor in Emergency Medicine

GLASGOW COMA SCORE

Eye Opening

Spontaneous	- 4
To voice	- 3
To pain	- 2
Closed	- 1

Verbal Response

AAOx3	- 5
Confused	- 4
Inappropriate	- 3
Incompressible	- 2
None	- 1

Motor Response

Spontaneous	-6
Localize pain	-5
Pain withdrawal	-4
Flexion	-3
Extension	-2
No movement	-1

AMS/COMA: TIPS AEIOU

- Trauma, Temperature
- Infection
- Psychogenic
- Stroke, SAH, Shock, Seizures, Space occupying lesion
- Alcohol, other drugs
- Electrolytes, encephalopathy, endocrine, epilepsy
- Insulin (DM, DKA, hypoglycemia); Interssupception
- O2 (hypoxia), Opiates, Organ failure
- Uremia

ACUTE AGITATION/DELIRIUM (FIND ME)

- **F**unctional / Psych
- **I**nfection
- **N**euro deficits
- **D**rugs
- **M**etabolic
- **E**ndocrine

Foundations Challenge

Visual Diagnosis

31 YO M P/W INABILITY TO MOVE R FACE X 2 HOURS

Dx and Tx?

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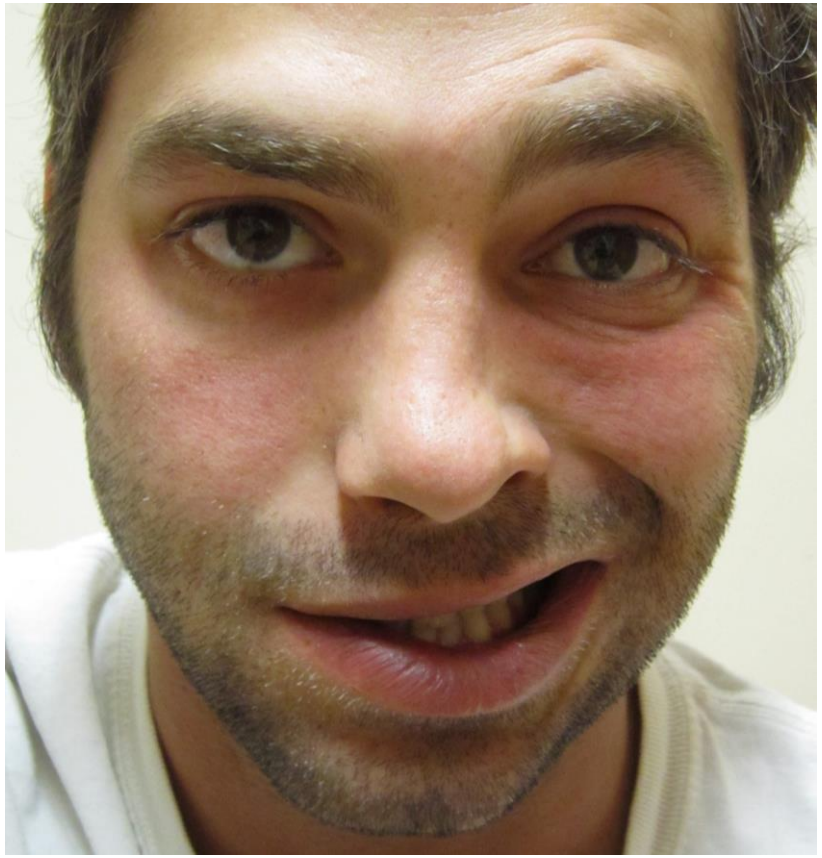
Foundations Challenge

Visual Diagnosis



31 YO M P/W INABILITY TO MOVE R FACE X 2 HOURS

Dx: Bell's palsy
Tx: steroids, artificial tears

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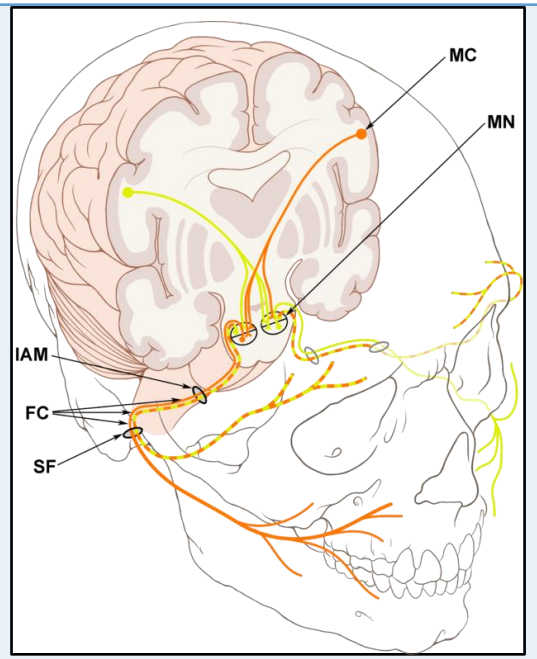
Foundations Challenge
Knowledge Bomb

BELL'S PALSY

Signs	Upper *and* lower facial involvement due to peripheral nerve lesion In contrast, CVA will have only lower facial involvement due to bilateral cortical innervation of the upper face.	
Causes	Usually idiopathic, some viral (HSV or VZV); if bilateral Bell's palsy is present, think Lyme disease	
Treatment	Steroids if within 72 hours +/- valacyclovir for severe symptoms (e.g. inability to close eye) x 1 week, dry eye prevention	

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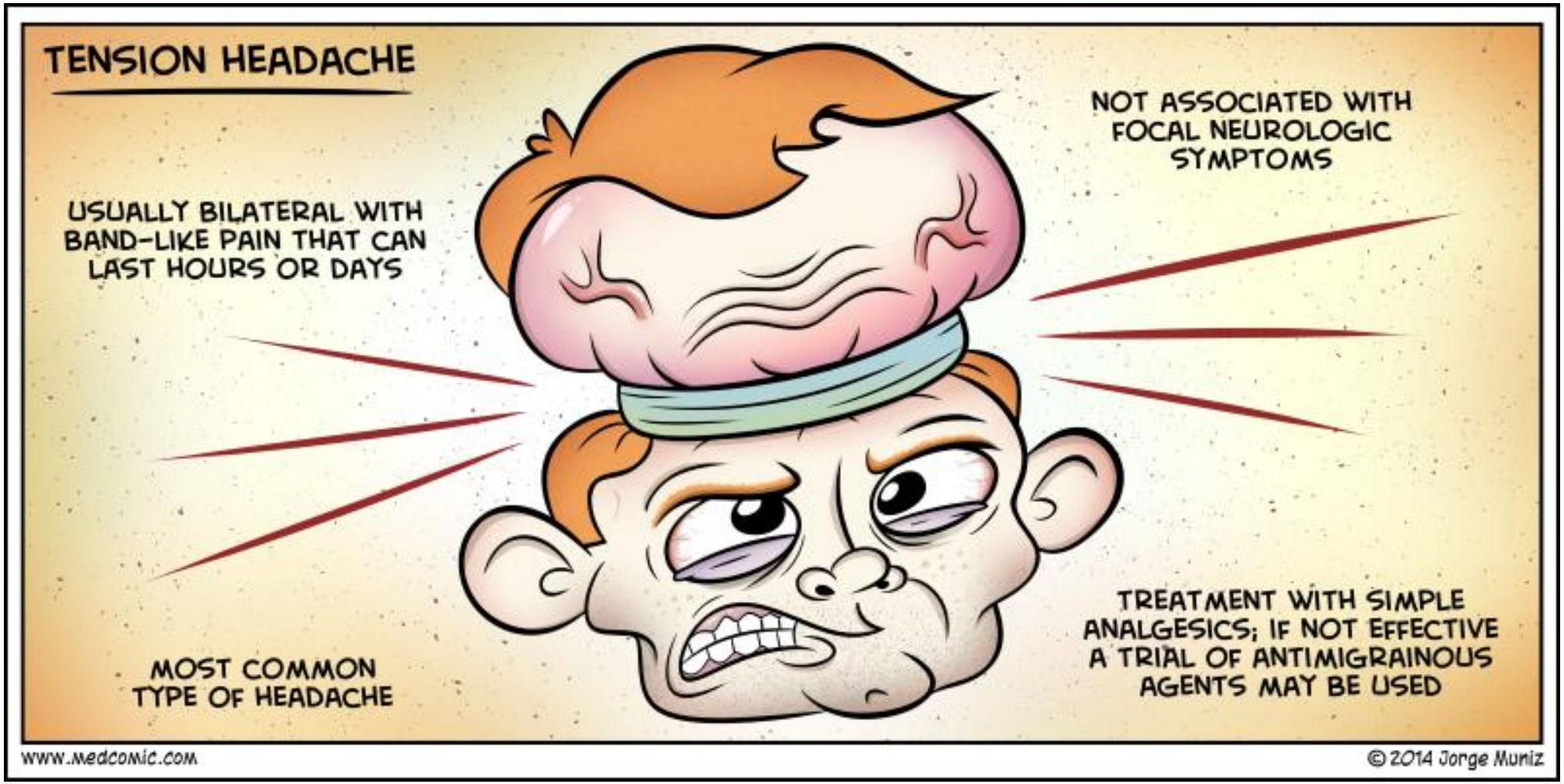
TENSION HEADACHE
USUALLY BILATERAL WITH BAND-LIKE PAIN THAT CAN LAST HOURS OR DAYS
NOT ASSOCIATED WITH FOCAL NEUROLOGIC SYMPTOMS
MOST COMMON TYPE OF HEADACHE
TREATMENT WITH SIMPLE ANALGESICS; IF NOT EFFECTIVE A TRIAL OF ANTIMIGRAINOUS AGENTS MAY BE USED
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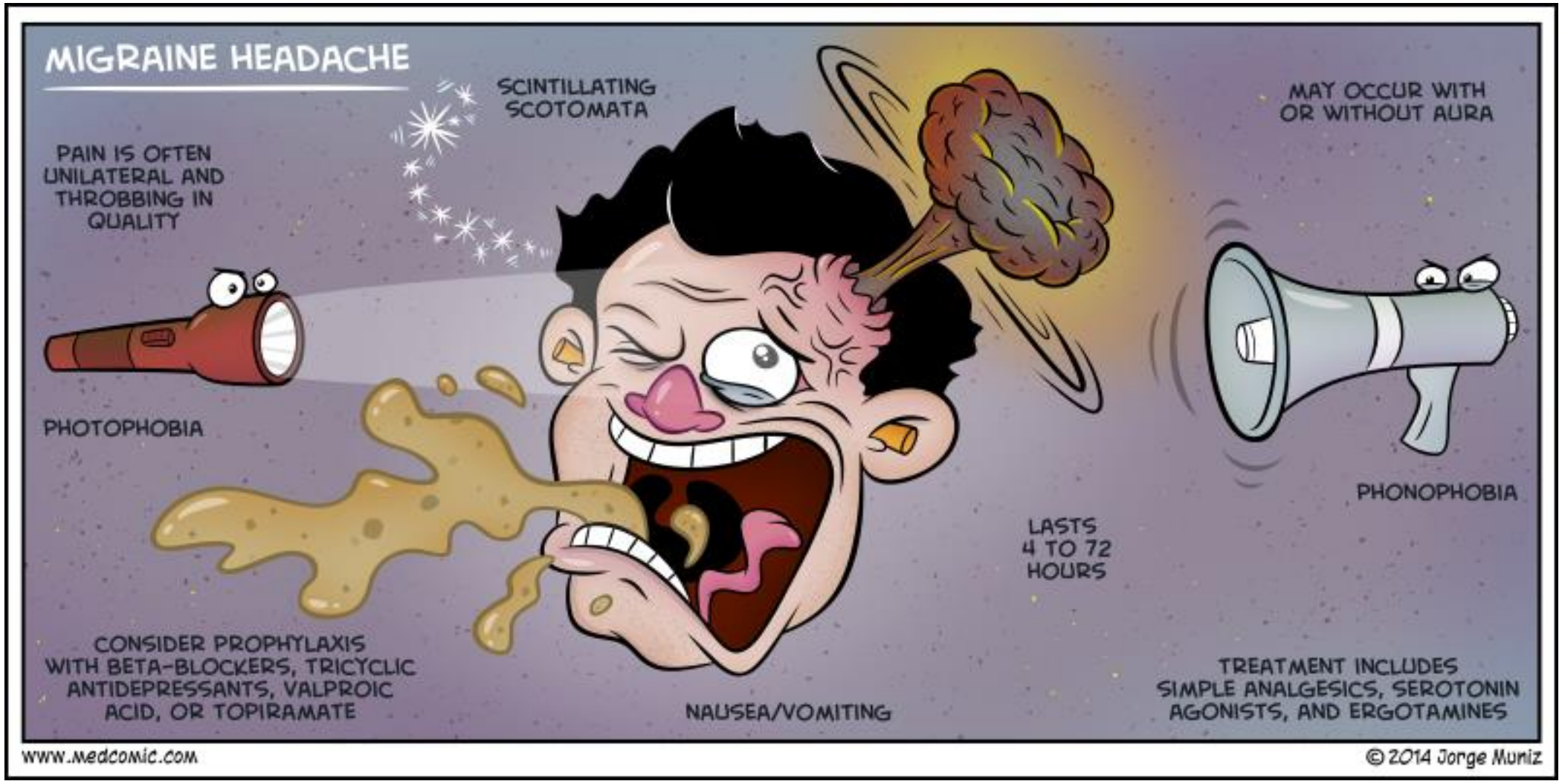
MIGRAINE HEADACHE
SCINTILLATING SCOTOMATA
MAY OCCUR WITH OR WITHOUT AURA
PAIN IS OFTEN UNILATERAL AND THROBBING IN QUALITY
PHOTOPHOBIA
PHONOPHOBIA
LASTS 4 TO 72 HOURS
CONSIDER PROPHYLAXIS WITH BETA-BLOCKERS, TRICYCLIC ANTIDEPRESSANTS, VALPROIC ACID, OR TOPIRAMATE
NAUSEA/VOMITING
TREATMENT INCLUDES SIMPLE ANALGESICS, SEROTONIN AGONISTS, AND ERGOTAMINES
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Table 7. Historical Buzzwords Describing Headache.

Finding	Etiology Considerations
Fever	Infection (CNS or others)
Thunderclap	Subarachnoid hemorrhage
Pain with eye movement	Optic neuritis
Temporary indoor heating	Carbon monoxide poisoning
Pregnancy	(Pre)eclampsia
Ataxia, diploplia, nystagmus	Intracranial pathology

HISTORY

- Sudden vs. gradual
  - With or without neuro changes
  - Thunderclap
    - Need to r/o SAH
  - Gradual onset that is constant for weeks/months
    - Tension HA
  - New onset that worsens in intensity over weeks
    - CA
  - Episodic HA with episodic symptoms free intervals
    - Migraine or cluster HA
- Worst HA or different from before

HISTORY

- Immunocompromised patient
- New onset of HA > 50 y/o
- Begins with exertion
- When started or what you were doing prior?
  - Coming out from a movie theater
  - Using cocaine
  - Working outside on the roof, yard
  - Inside of the house is worst
  - Drinking EtOH
  - Having sex!

HISTORY

- Wake up with HA
  - HTN
  - Cluster HA
  - CA
- Wake up with no HA but develop one as the day progress
  - Tension HA

HA

VICTIM

- Vascular (bleeding, vasculitis, thrombosis, or embolism)
- Infection (abscess, sinusitis, meningitis, toxo)
- CO poison, Caffeine withdrawal, Cluster HA
- Trauma, Tumors, Temporal arteritis, toxins
- i
- Migraine, Muscle contraction, or tension

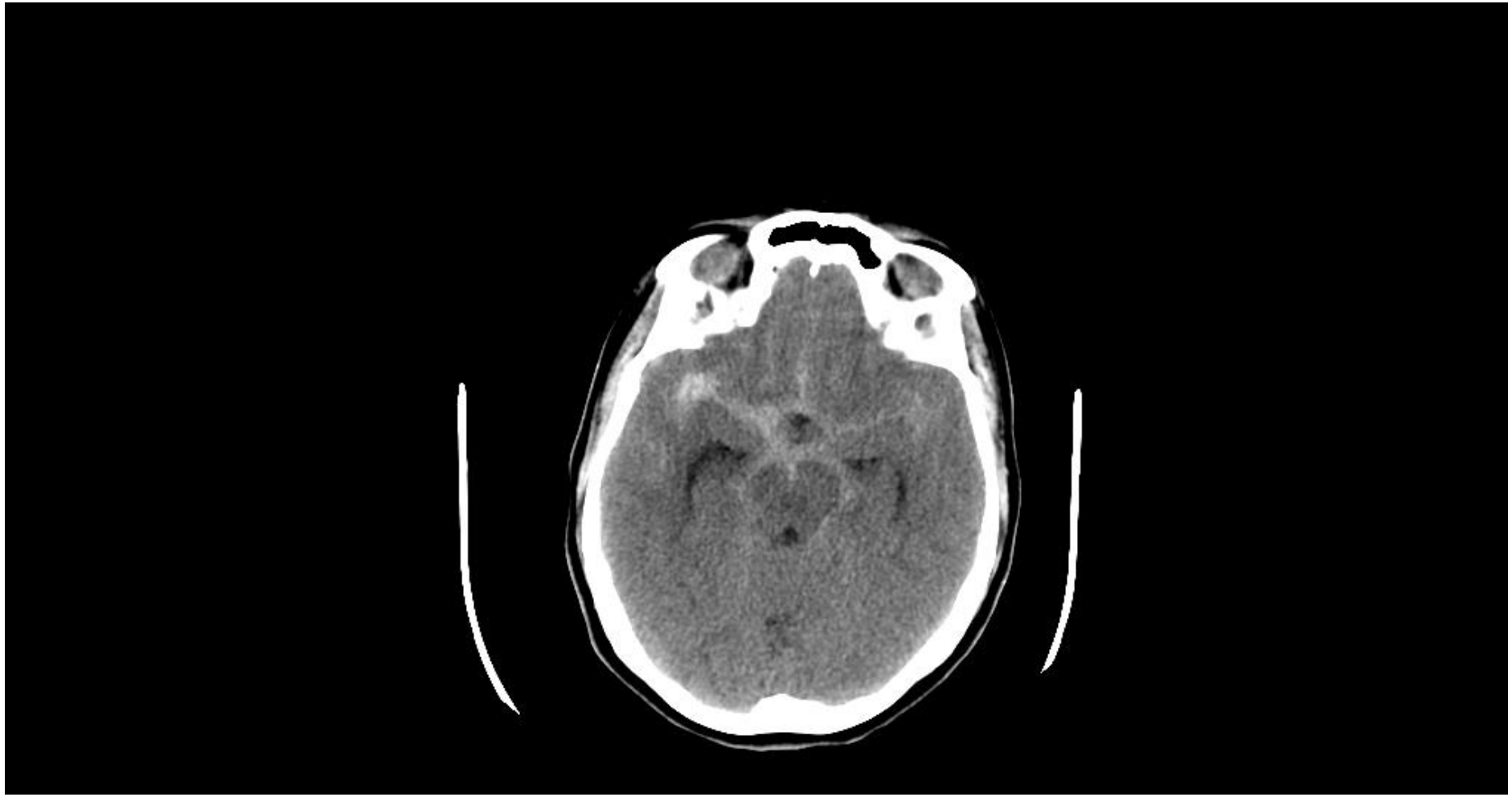
HA

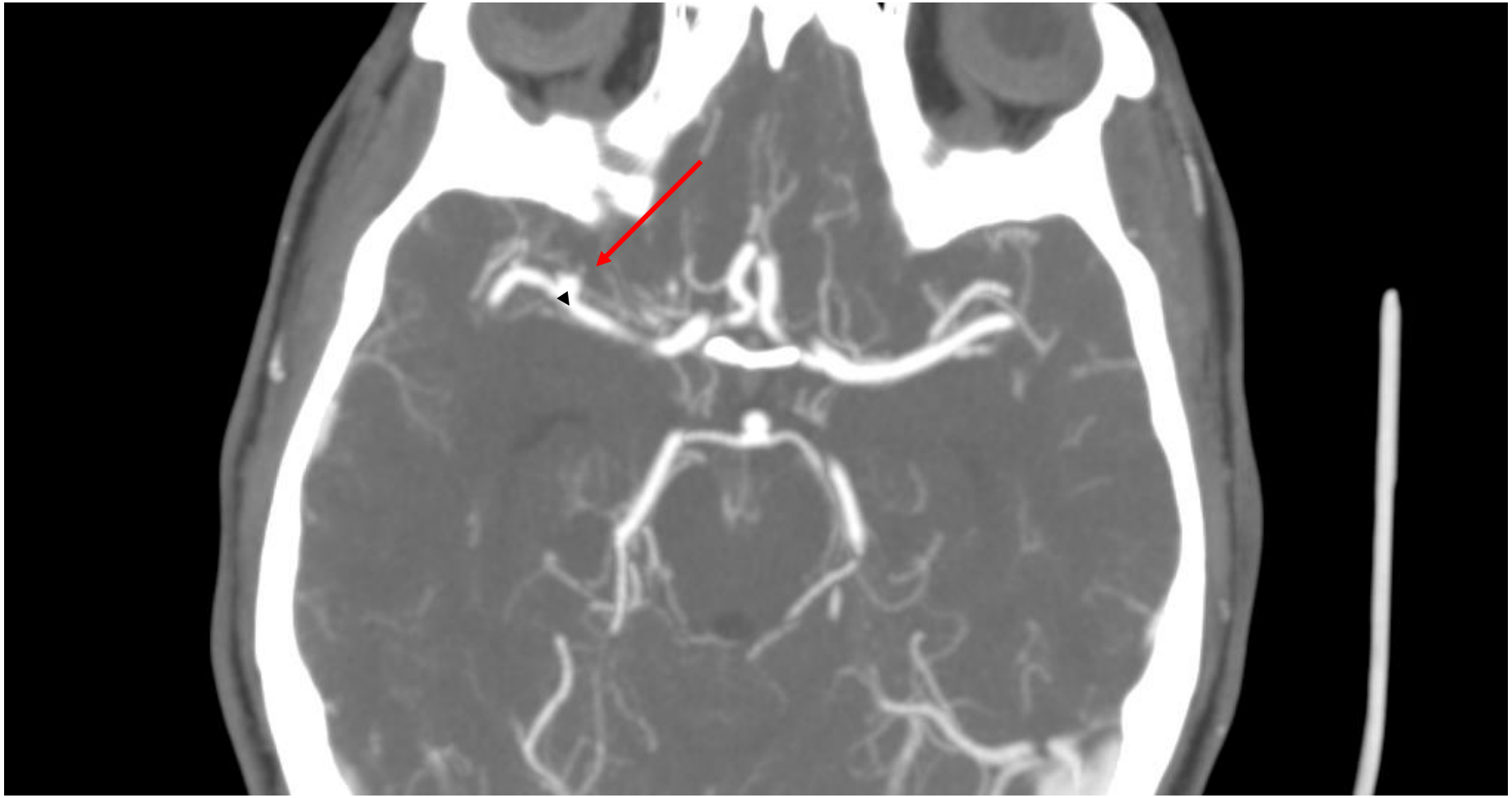
KILLERS (TOBIC)

- Temporal arteritis, Tumor, or other causes of ICP such as HTN crisis
- Ophthalmic disease such as glaucoma or optic neuritis
- Bleeding
- Infection
- CO poisoning

- 35yo woman with a history of migraine headaches was awakened by the worst headache of her life and severe nausea. A few minutes later, she vomited.

- ED: BP 170/90. Ill and uncomfortable. Holding an emesis basin, preferred to keep her eyes closed. Slightly drowsy. Resisted passive neck flexion.





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SUDDEN, SEVERE HEADACHE

- Differential diagnosis
  - Aneurysmal subarachnoid hemorrhage
  - Aneurysmal subarachnoid hemorrhage
  - Aneurysmal subarachnoid hemorrhage
  - Aneurysmal subarachnoid hemorrhage
  - **Aneurysmal subarachnoid hemorrhage**

SUDDEN, SEVERE HEADACHE

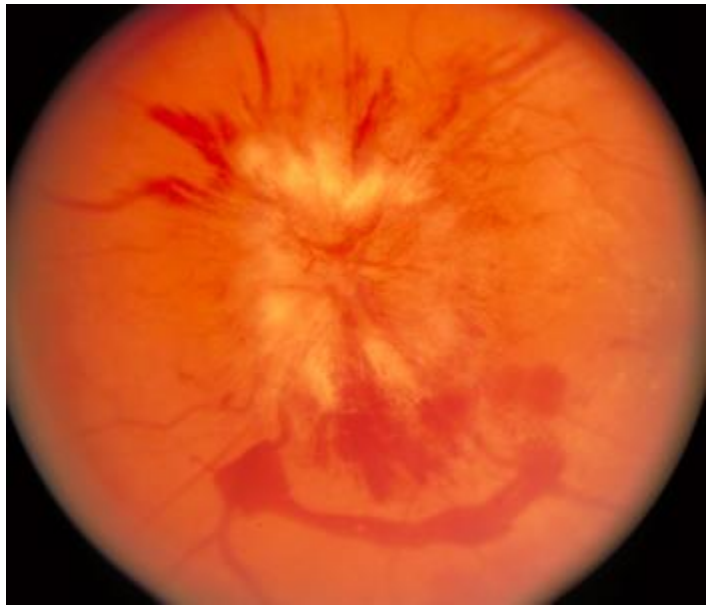
- Differential diagnosis
  - Cervical artery dissection
  - Cerebral venous sinus thrombosis
  - Intracranial mass
  - Pituitary apoplexy
  - Meningitis
  - Encephalitis
  - Spontaneous intracranial hypotension

SUDDEN, SEVERE HEADACHE

- Rapid diagnosis
  - History (features of aneurysmal SAH)
    - Instantaneous onset of headache
    - Decrease in arousal/loss of consciousness at onset
    - Nausea, vomiting
    - Family history of aneurysm, SAH
    - Neck stiffness

SUDDEN, SEVERE HEADACHE

- Exam
  - Meningismus
  - Retinal subhyaloid hemorrhages (Terson syndrome)
  - CN III palsy (ptosis; deviation "down and out"; pupil fixed and dilated)



SUDDEN, SEVERE HEADACHE

- Rapid diagnosis
  - Imaging
    - CT sensitivity declines with time after ictus
      - Nearly 100% sensitive within 6h
      - >95% sensitive for SAH within 12h
    - CT angiogram: identifies aneurysm
      - Treatment planning
      - 20% will have multiple aneurysms
  - CSF
    - LP ***required*** if SAH diagnosis is considered and CT negative
    - 90-95% sensitive for SAH when CT negative
    - Findings
      - Gross blood
      - Xanthochromia

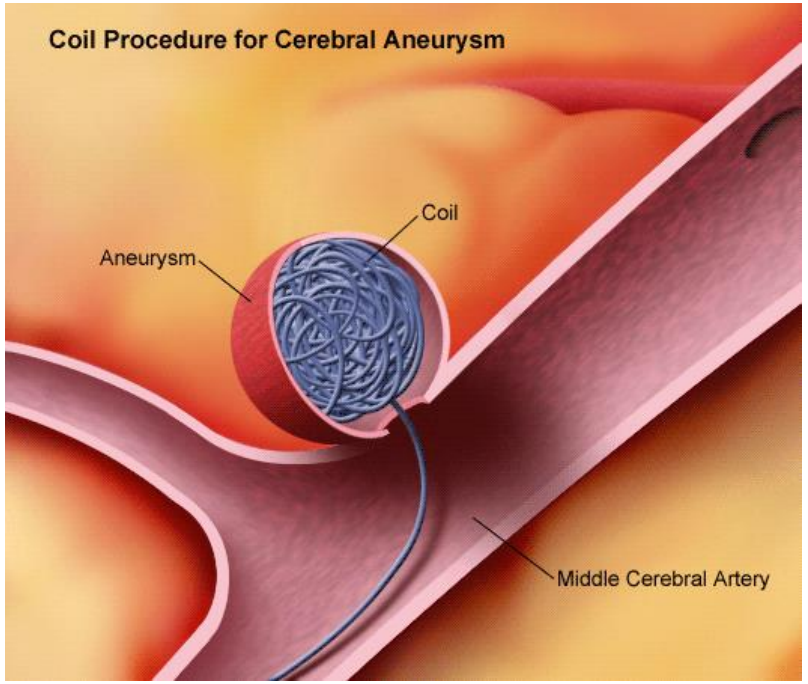
EMERGENCY TREATMENT OF ANEURYSMAL SAH

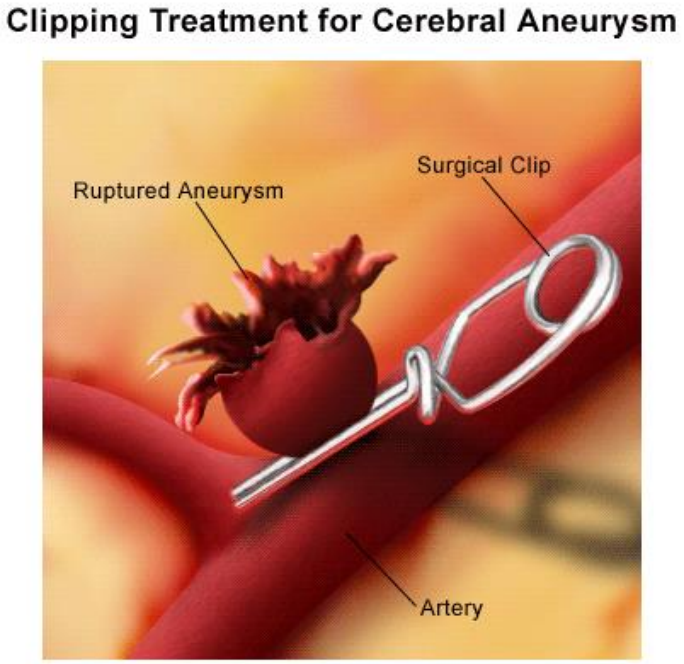
- Notify neurosurgery and neurointerventional team immediately

- Prevent rebleeding
  - Risk = 5-15% in 1st 24h; mortality 70-80%
  - Treat hypertension: Keep SBP 110-150 mmHg
    - IV Antihypertensives
      - Prns: labetalol, hydralazine
      - Nicardipine gtt
    - Judicious analgesia
      - Tylenol → Ultram → very low-dose IV fentanyl or hydromorphone
  - Antifibrinolytics (tranexamic acid) if securing is expected to be delayed > 6h after arrival

EMERGENCY TREATMENT OF ANEURYSMAL SAH

- Secure aneurysm
  - Goal: ASAP; within 18h of presentation
  - Conventional angiogram from ED
    - Operative planning
    - Endovascular coils if possible
  - Otherwise, surgical clipping





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- 45yo man with a history of IV heroin abuse presented to the ED with 3 days of worsening headache, confusion, and lethargy

- Exam: Temp 102, BP 100/50, HR 110. Opened eyes to pain only. Uncomfortable, groaning unintelligibly. Meningismus. Systolic murmur

- CSF: RBC 6, WBC 1090 (85% PMNs), glucose 32 (serum 81), protein 234 (nl <70)

- Cultures of blood, urine and CSF all grew MRSA

FEVER AND CONFUSION

- Differential diagnosis
  - Meningitis (bacterial, viral)
  - Encephalitis (viral)
  - Cerebral abscess (bacterial, toxoplasma, fungal)
  - Subdural empyema
  - Endocarditis with septic embolic brain infarcts
  - Non-CNS infection with secondary encephalopathy

FEVER AND CONFUSION

- Rapid diagnosis
  - History
    - Headache, neck stiffness
    - Oral/nasal infection
    - Immunosuppression (HIV, chemotherapy, transplant, diabetes, sickle cell disease, poor nutrition)
    - Alcohol abuse
    - IV drug use
    - Sick contacts
    - Travel

FEVER AND CONFUSION

- Rapid diagnosis
  - Exam: meningismus, skin rash, embolic skin lesions, heart murmur
  - Imaging: CT to look for a mass lesion
  - CSF (bacterial meningitis)
    - 10-10,000 WBC/mm3; ≥ 80% neutrophils
    - Glucose – CSF:serum ratio ≤ 0.5
    - Elevated protein (> 45 mg/dL)
    - Check Gram stain and bacterial culture
  - Labs: Blood cultures (3 sets), urine culture

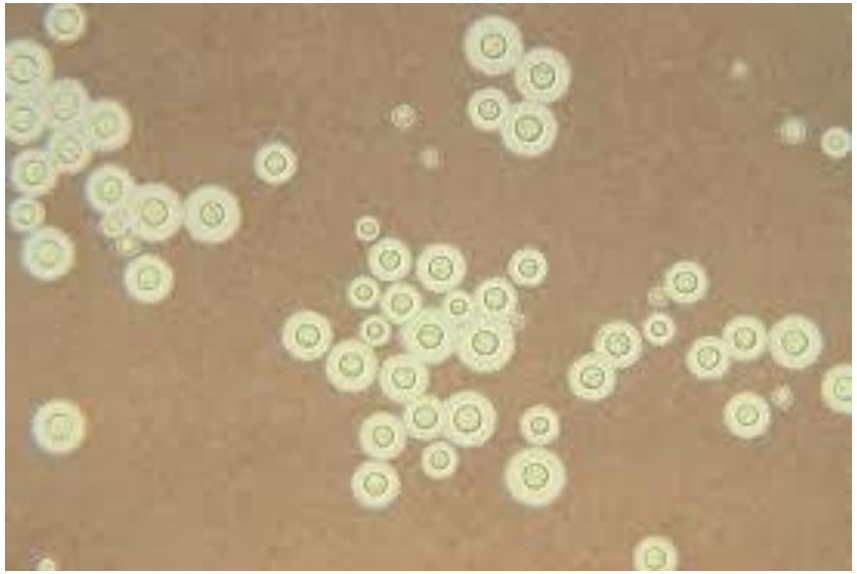
EMERGENT TREATMENT OF ACUTE BACTERIAL MENINGITIS

- Rapid administration of corticosteroids and antibiotics is the key. Within ***two hours***:

1. Blood culture
2. Dexamethasone 10mg IV (20min before ABx)
3. Antibiotics (all IV)
   - Vancomycin 1.5mg/kg, ceftriaxone 2g
   - If >50yo or immunosuppressed: add ampicillin 2g
4. CT
5. LP

- LP
  - Projectile CSF
  - What test to ask for?

- India ink



HERPES ENCEPHALOPATHY

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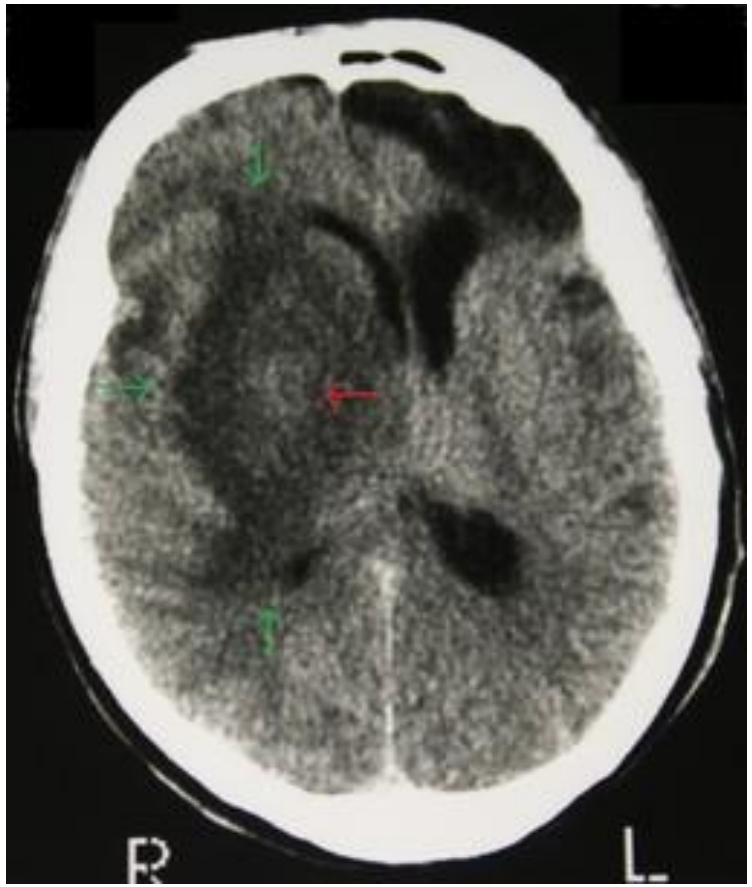
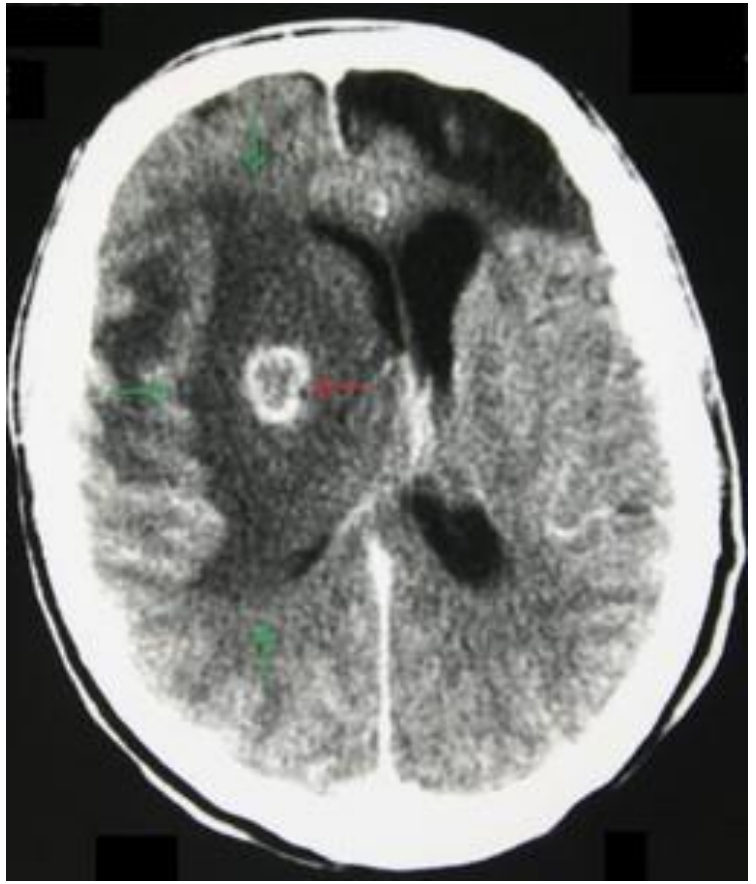
TOXOPLASMOSIS





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- 75 y/o female c/o unilateral HA that gets worst at night or going out to the street
- The pain irradiates to the jaw
- ROS: chronic shoulders and hips pain for month
- PE?

TEMPORAL ARTERITIS

- Patients >50 y/o; female>male
- Caused by: arteritis
  - Giant cell arteritis
  - PMR
- Frontotemporal throbbing HA
- Jaw claudication
- Non-pulsatile or tender temporal artery
- Visual loss
- Biopsy
- Steroids

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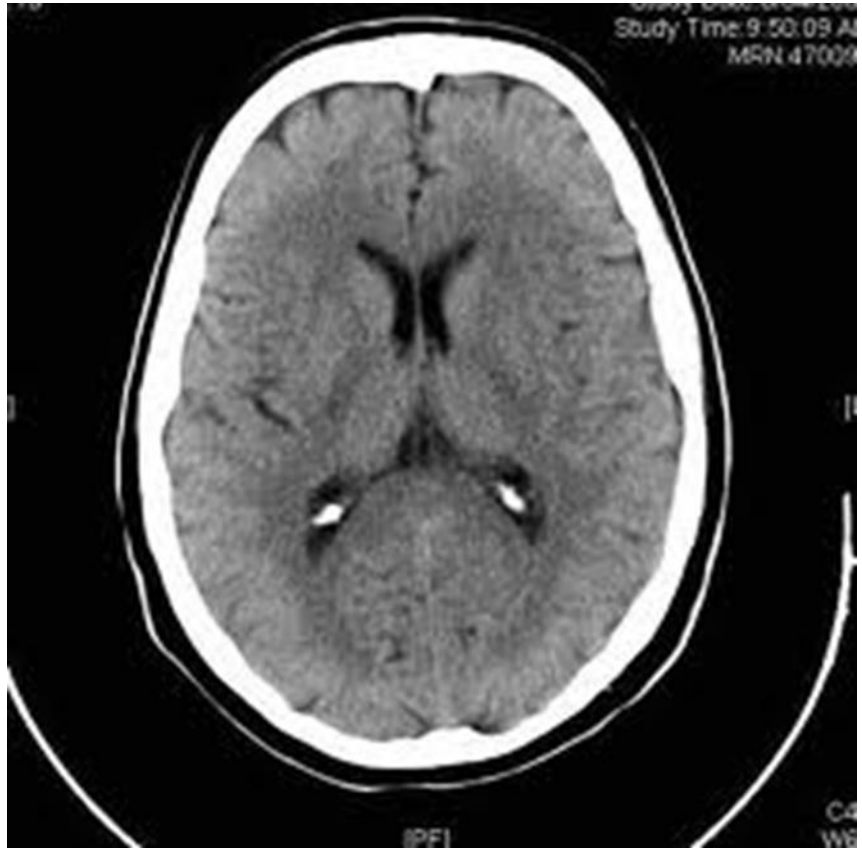
- 35 y/o male c/o unilateral HA (abrupt onset), retro-orbital pain, weakness on the other side of the HA
- PE:

Horner's syndrome

WHAT TO DO?

- CT?



Now, what?

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CAROTID AND VERTEBRAL ARTERY DISSECTION

- Acute unilateral HA, facial and neck pain
- Age ~40 y/o
- Contralateral hemiparesis
- Visual disturbance
- Aphasia
- N/V and vertigo if vertebral

- 56 y/o female that c/o acute onset of HA after coming out from Disney's "the haunted mansion"
- Some blurred vision in one eye
- PE

ACUTE CLOSED ANGLE GLAUCOMA

- IOP
- Treatment
  - Ophtha
  - B-blocker, carbonic anhydrase inhibitors, miotics, alpha adrenergic
  - Sur[illegible]

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Foundations Challenge

Visual Diagnosis

35 YO F P/W HEADACHE X1 WEEK AND BLURRY VISION

Dx and Tx?

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Foundations Challenge

Visual Diagnosis

35 YOF P/W HEADACHE X1 WEEK AND BLURRY VISION

Dx: Idiopathic Intracranial HTN
Tx: Lumbar puncture

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Foundations Challenge
Knowledge Bomb

IDIOPATHIC INTRACRANIAL HYPERTENSION
(PSEUDOTUMOR CEREBRI)

Classic SSx	Obese young woman with a new headache and visual disturbances; exam may show papilledema, visual field loss, and/or a CN6 palsy	
Diagnosis	CT brain is essential to r/o intracranial mass; LP is diagnostic (opening pressure >20 cmH2O is abnormal, *but this should always be done in lateral decubitus position*)	
Treatment	**Procedural:** Serial LPs, VP shunt	**Medical:** weight loss, acetazolamide (first line)
Prognosis	The goal of treatment is to reduce headaches and prevent vision loss; ophtho and neuro followup is essential	

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26 YO ASIAN MAN WITH ATRAUMATIC BLE PARALYSIS

Labs to check?

Treatment?

26 YO ASIAN MAN WITH ATRAUMATIC BLE PARALYSIS

Labs to check?

TSH, free T4, K

Treatment?

Gentle oral K repletion, beta blockers

Foundations Challenge
Knowledge Bomb

PERIODIC PARALYSIS

Thyrotoxic PP	Hypokalemic PP
Poorly understood mechanism; likely due to increased shuttling of K into cells under the influence of increased T4 combined with insulin or epinephrine	Familial channelopathy resulting in K shuttling into cells and resultant hyperpolarization leading to muscle weakness
Precipitants: exercise (epinephrine), heavy meal (insulin)	
Labs: low K, high T4	**Labs:** low K, normal T4
Treatment: gentle K repletion (avoid rebound hyperK), propranolol (treats hyperthyroid)	**Treatment:** gentle K repletion (avoid rebound hyperK)
Be sure to rule out other causes of weakness or paralysis: GBS, tick paralysis, myopathy, myelopathy, botulism, etc.	

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- The most common cause of ischemic stroke is:
- a. Atherosclerotic thrombus
- b. Complicated migraine
- c. Vasculitis
- d. IV drug use
- e. Embolic

ISCHEMIC STROKES

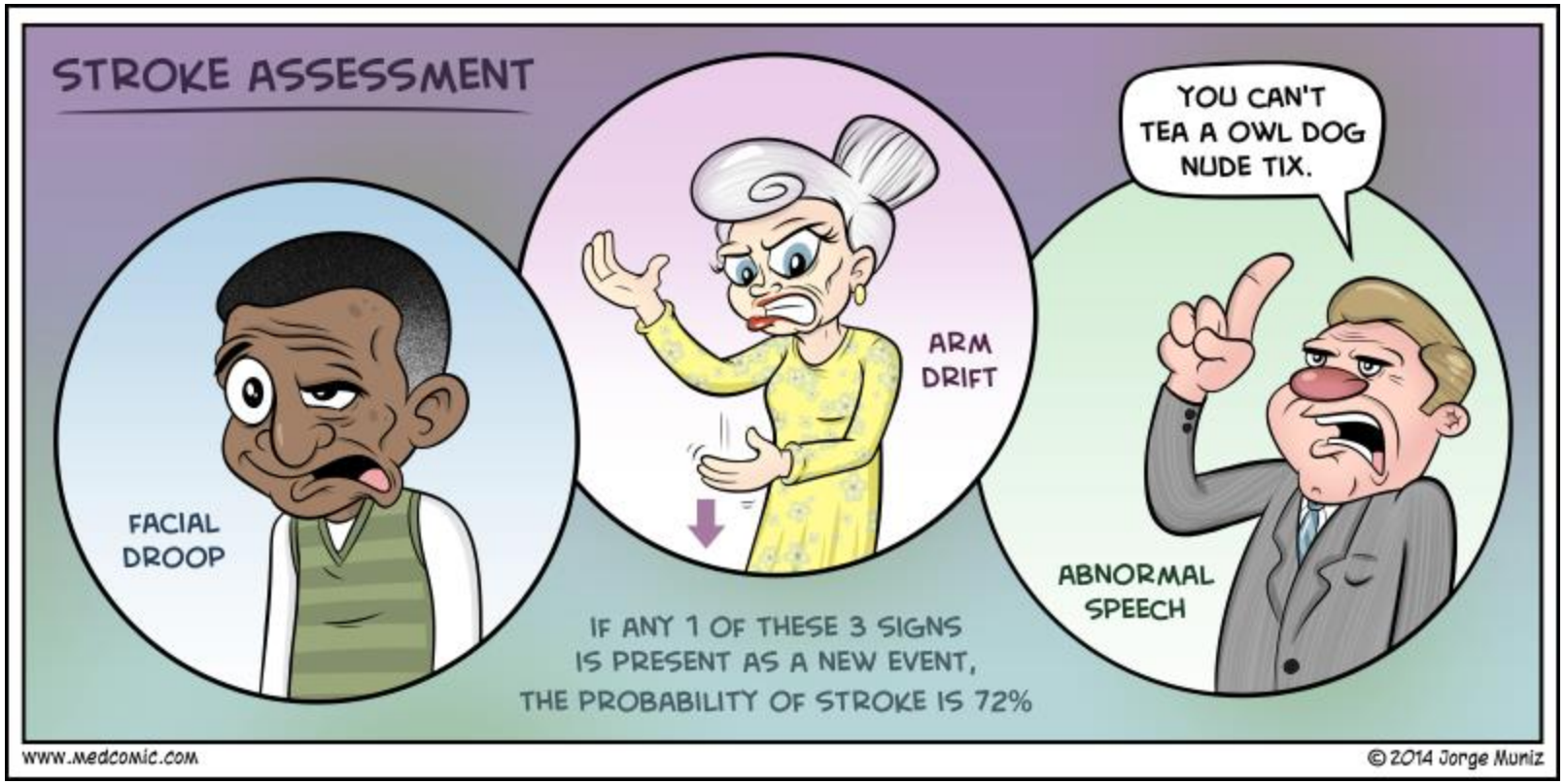
- Thrombotic
  - most common
  - result from: artherosclerosis, infective arteritis, vasculitis, dissection, hypercoagulable states
- Hypoperfusion
  - reduced Oxygen

ISCHEMIC STROKES

- Embolitic
  - 1/5 of strokes
  - result from: plaques from large neck vessels, mural thrombi from heart (Afib, MI), endocarditis, valvular ds, long bones FX

THROMBOTIC VS EMBOLIC

- Thrombotic
  - Slow, progressive onset of symptoms; wakes in the early morning w/ deficits
- Embolic
  - Abrupt onset w/ maximum deficit which improves slowly as the embolus breaks up and moves peripherally



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HEMORRHAGIC STROKE

- ICH
  - HTN, amyloidosis, anticoagulation, cocaine, vascular malformations
- SAH
  - ruptured artery (spontaneous rupture of saccular aneurysm or AV malformation)
- Trauma

- Eyes look toward the lesion where is it
  - hemispheric abnormality (tumor, CVA)
- Eyes look away the lesion when is
  - brainstem

PINPOINT PUPILS

- Opiates OD
- Cholinergic syndrome
- Pontine hemorrhage

- Romberg sign?
- Cerebellar dysfunction

WHERE IS THE LESION?

83 y/o m p/w two hours of …

1. RLE weakness/numbness

2. Aphasia + R facial and RUE weakness/numbness

3. Binocular vision changes

WHERE IS THE LESION?

83 Y/O M P/W TWO HOURS OF …

- RLE weakness/numbness (>RUE); Loss of frontal lobe control
  - ACA
- Aphasia + R facial and RUE (>RLE) weakness/numbness; Conjugate gaze impaired; MCC
  - LMCA
- Binocular vision changes; Contralateral hemiplegia, homonymous hemianopsia, and hemisensory loss; memory loss; ipsilateral CN III nerve palsy
  - PCA

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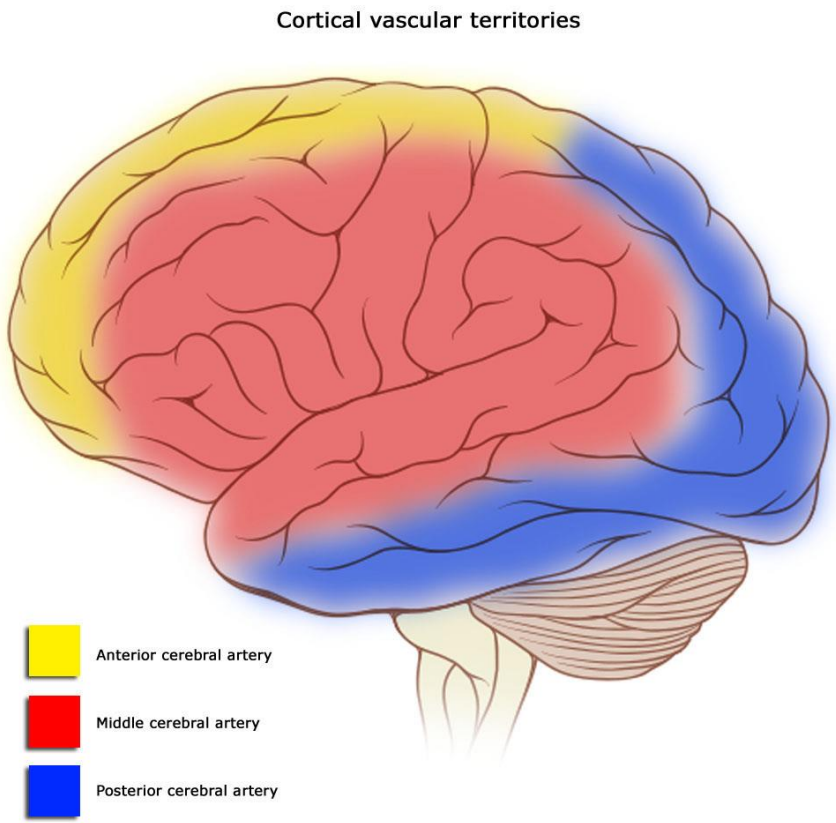
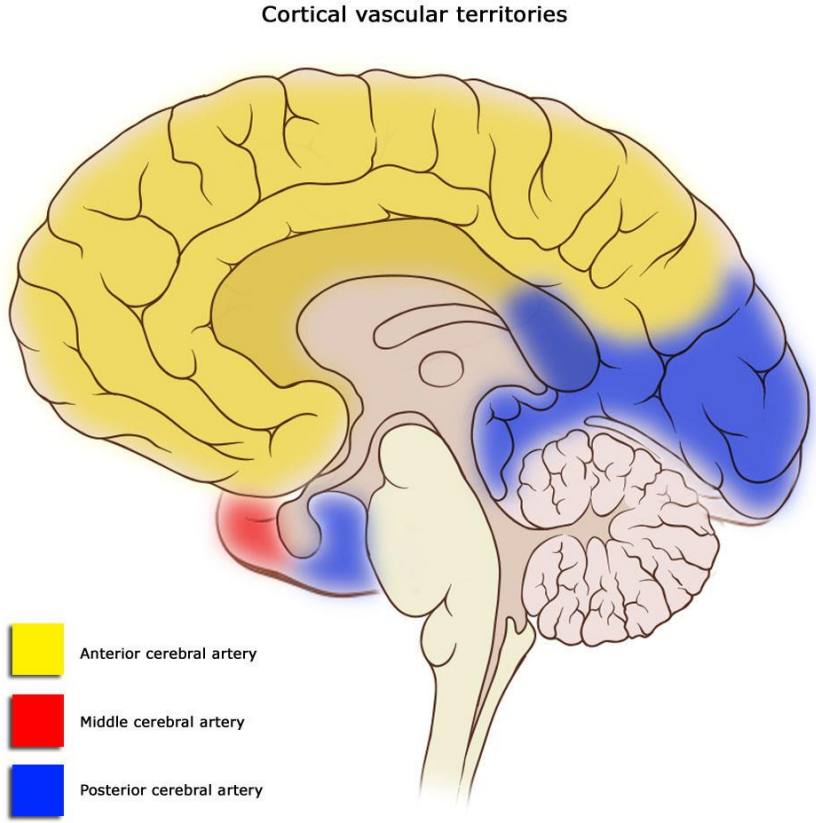
LARGE VESSEL OCCLUSION SYNDROMES





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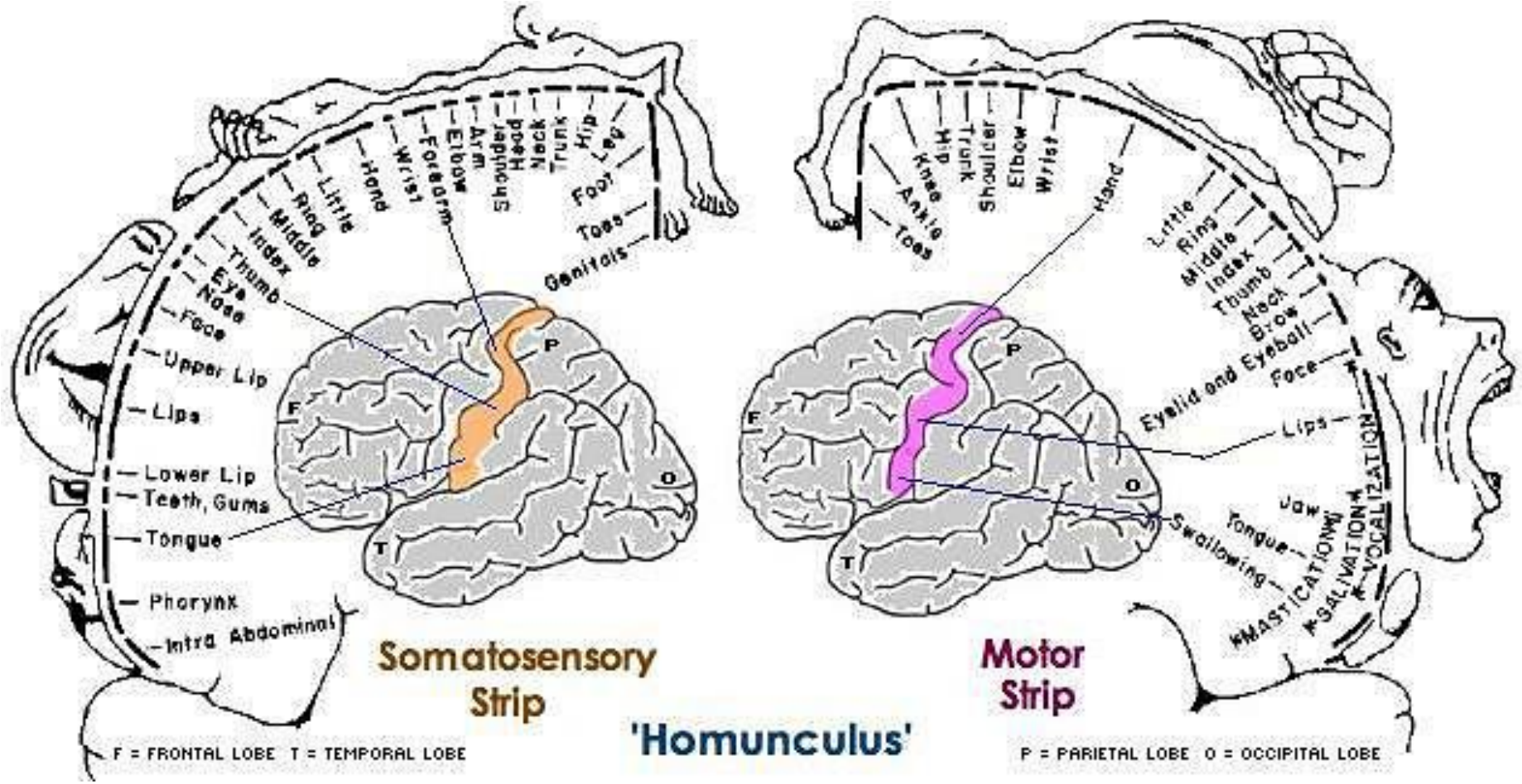
LARGE VESSEL OCCLUSION SYNDROMES



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OTHER IMPORTANT CVA SYNDROMES

Brainstem lesions	
Wallenberg syndrome	Lateral medullary infarct ("wall" of the medulla) from PICA lesion; causes ipsilateral facial sensory and contralateral body sensory deficits to pain and temperature, ipsilateral Horner syndrome (takes out sympathetic fibers), and ipsilateral CNIX-XII deficits
Weber syndrome	Anterior midbrain infarct from PCA branch lesion; causes ipsilateral CNIII palsy and contralateral hemiparesis/-plegia
Locked-in syndrome	Ventral pontine infarct usually caused by basilar artery lesion; preserves cognitive and often sensory function but results in complete inability to move anything except for the eyes (due to preserved CNIII and IV function)

INDICATIONS FOR TPA? (NEED ALL 4)

1.

2.

3.

4.

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INDICATIONS FOR TPA? (NEED ALL 4)

1. Adult patient (>18 yo)
2. No ICH on CT
3. Symptom onset <4.5 hours
4. No other clear reversible cause

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ABSOLUTE CONTRAINDICATIONS FOR TPA?

Name 3

ABSOLUTE CONTRAINDICATIONS FOR TPA? (NAME 3)

- Within 3 mos: ischemic stroke, neurosurgery, or head trauma
- Current or previous ICH
- Major surgery within 2 weeks
- BP >185/110 after attempted reduction
- Possible SAH
- Known intracranial tumor, aneurysm, or AVM
- Possible reversible cause
- Recent bleeding or coagulopathy (PT >15s, INR > 1.7, platelets <100k

ISCHEMIC STROKE EMERGENT TREATMENT

- Goal: maximize perfusion to limit infarction
  - *Earlier reperfusion = more salvaged brain = better functional outcome*
- Allow hypertension, give IV normal saline, lay head of bed < 30 degrees
  - Do NOT treat hypertension unless >220/110 mmHg or end-organ dysfunction
- IV tissue plasminogen activator (tPA)
  - Within 4.5h of symptom onset
  - Exclusion criteria extensive (bleeding)
- Endovascular therapy
  - Contraindications to or failure of IV tPA
  - Mechanical thrombectomy
  - Intra-arterial tPA
  - Within 6h in anterior circulation (ACA, MCA)
  - Within 12h in posterior circulation (vertebral, basilar)

ICH EMERGENT TREATMENT

- Goal
  - Prevent hematoma expansion
    - Occurs in 70% of patients, mostly in 1st 6h
    - 10% volume increase =
      - 5% mortality increase
      - 16% increase in chance of worsening by 1 point on the modified Rankin scale
  - Treat hypertension
    - Goal SBP 130-150 mmHg
    - IV Drugs!!!
      - Prns: labetalol, hydralazine
      - Nicardipine gtt
  - Correct coagulopathy FAST!
    - Goal INR < 1.4, platelets > 100k
    - PCC, Vitamin K, fresh frozen plasma

- 45yo man presented to the ED complaining of back pain, generalized weakness, and shortness of breath

- Illness began 5d ago when he awoke with tingling in the feet. Later that day, his walking became clumsy. Cold 3 weeks ago

- Exam: Afebrile. RR 35, diaphoretic, anxious. Bifacial weakness and mild dysarthria. Symmetric weakness of proximal limbs. Muscle stretch reflexes diffusely absent.

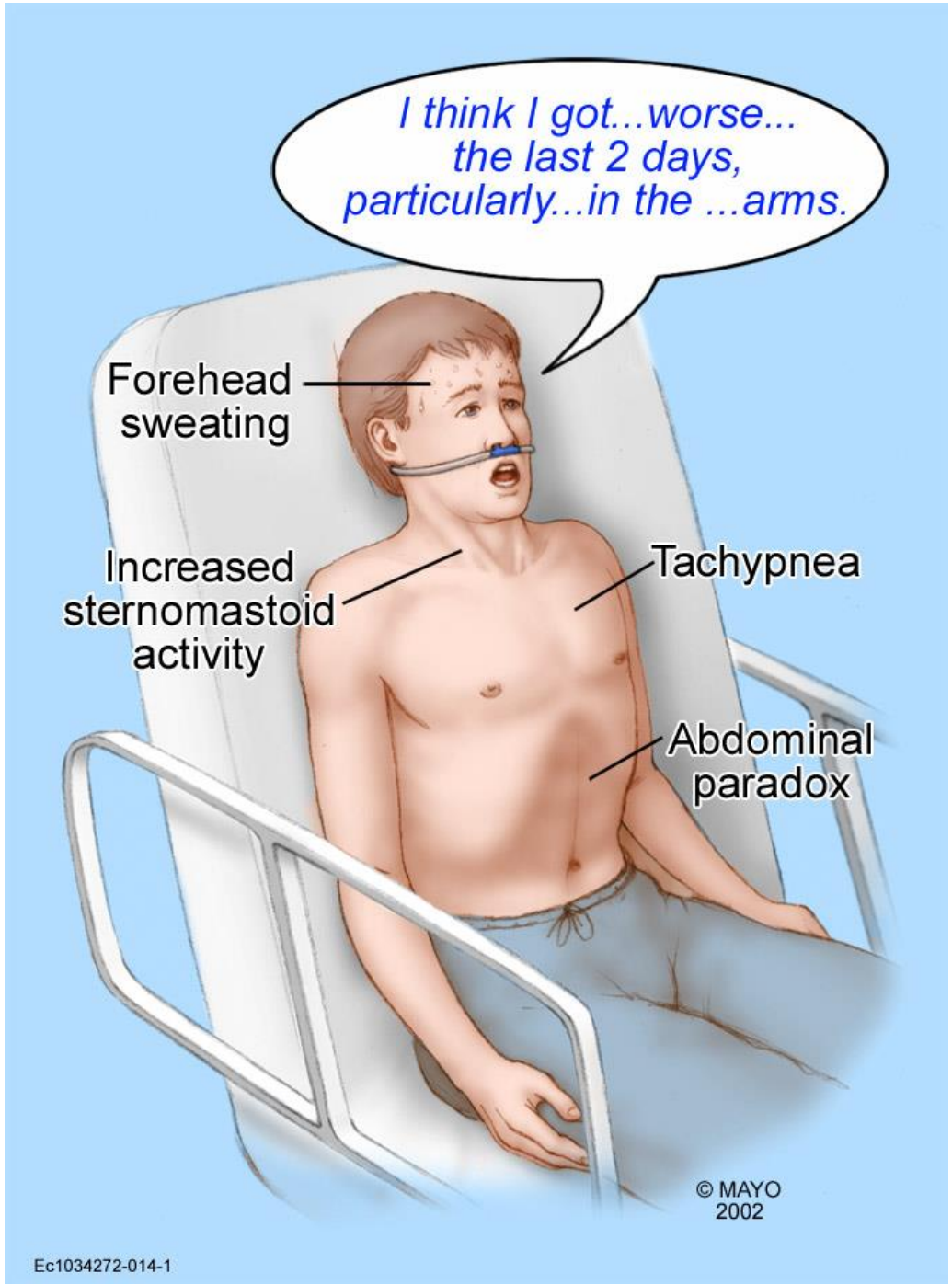
- Intubated in ED
- LP: Protein 100mg/dL (normal < 70); RBC, WBC, glucose normal.

WEAKNESS AND DIFFICULTY BREATHING

- Differential diagnosis
  - Guillain-Barre syndrome
  - Myasthenic crisis
  - Cervical cord lesion
  - Severe myopathy
  - Sepsis

WEAKNESS AND DIFFICULTY BREATHING

- Rapid diagnosis
  - History
    - GBS: gait unsteadiness, distal limb paresthesias, proximal weakness, cramping, back pain
    - Myasthenic crisis: history of MG, prominent CN involvement (diplopia, "nasal" voice, dysphagia, nasal regurgitation) *fatigability*
  - Exam
    - GBS: diffuse hyporeflexia or areflexia
    - MG: prominent CN symptoms; fluctuation of symptoms



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WEAKNESS AND DIFFICULTY BREATHING

- Rapid diagnosis
  - Imaging: consider MRI of cervical spine if CNs are spared
  - CSF
    - GBS: elevated protein with up to 10 WBC ("albuminocytologic dissociation")
  - Labs: arterial blood gas

EMERGENCY TREATMENT OF GBS AND MYASTHENIC CRISIS

- *Goal: Control breathing before catastrophe*
- Intubation and mechanical ventilation
  - Airway compromise from CN dysfunction
  - Even if O2 and CO2 are OK
    - Vital capacity < 15mL/kg
    - Negative inspiratory force worse than -30 cm H2O
    - Rapidly worsening respiratory function
  - Do not wait for ABG results

EMERGENCY TREATMENT OF GBS AND MYASTHENIC CRISIS

- GBS
  - Intravenous pooled human immunoglobulin (IVIg)
  - Plasma exchange
- Myasthenic Crisis
  - Plasma exchange → more rapid improvement
  - IVIg only if plasma exchange contraindicated

NAME THE DIFFERENCE BETWEEN:

Myasthenia Gravis

AND

Lambert-Eaton Myasthenic Syndrome

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NAME THE DIFFERENCE BETWEEN:

Myasthenia Gravis

Antibody vs. post-synaptic acetylcholine receptors

Worsens with repetitive movement

Lambert-Eaton Myasthenic Syndrome

Antibody vs. pre-synaptic voltage-gated calcium channels

Improves with repetitive movement

Usually paraneoplastic (esp. small cell lung cancer)

MYASTHENIA GRAVIS VS. LAMBERT-EATON SYNDROME

Myasthenia Gravis	Lambert-Eaton
Cause: antibodies against post-synaptic acetylcholine receptors (competes against ACh)	**Cause:** antibodies against pre-synaptic Ca channels (inhibits ACh release)
SSx: weakness worse with repetitive activity (uses up ACh), causing e.g. diplopia late in the day	**SSx:** weakness better with exertion or repetitive activity (forces more ACh release), causing mainly leg weakness and rarely diplopia
Treatment: pyridostigmine (decreases ACh degradation, increasing competition vs. receptor antibodies), intubate EARLY for decreased respiratory capacity, plasmapheresis/IVIG	**Treatment:** LEMS is usually 2/2 underlying cancer, so ca treatment is usually the most helpful; pyridostigmine can work here too, as can IVIG

NAME A DISEASE THAT TYPICALLY CAUSES...

Ascending Paralysis?

Descending Paralysis?

NAME A DISEASE THAT TYPICALLY CAUSES…

Ascending Paralysis?

Guillain-Barré Syndrome
Tick Paralysis

Descending Paralysis?

Botulism
Miller Fisher variant GBS
Myasthenia Gravis
Lambert-Eaton Myasthenic Syndrome

Diplopia with lateral gaze

Dx?

Bilateral Bell's palsy

Dx?

Diplopia with lateral gaze

Internuclear ophthalmoplegia (associated with MS)

Bilateral Bell's palsy

Lyme disease

- 20yo male college student is found confused and drowsy by his friends on a Sunday morning
- He has a history of epilepsy, is known to be poorly compliant with medications, and was drinking the night before
- 5 minutes after arriving at the ED, he begins to convulse. 3 min into the convulsion, he is not slowing down

GENERALIZED CONVULSIVE STATUS EPILEPTICUS

- Status epilepticus
  - Any single seizure lasting > 5min
  - ≥ 2 seizures without clearing of mental status between them

- Differential diagnosis
  - Underlying epilepsy with or without AED withdrawal
  - Drug intoxication (many types) or withdrawal (esp. EtOH and benzodiazepines)
  - Hypoglycemia
  - Vascular disease (infarct, ICH, SAH, AVM)
  - Electrolyte abnormalities (↓Na, Mg, Ca; ↑Na)
  - CNS infection
  - Tumor
  - Psychogenic, non-epileptic seizure (conversion disorder)

GENERALIZED CONVULSIVE STATUS EPILEPTICUS

- Rapid diagnosis
  - History: epilepsy, other neurologic disease, diabetes, drug ingestion/withdrawal, infectious symptoms, pre-seizure neurologic symptoms
  - Exam:
    - subtle signs of ongoing seizure (periorbital/perioral clonus, forced horizontal conjugate eye deviation, hippus)

GENERALIZED CONVULSIVE STATUS EPILEPTICUS

- Rapid diagnosis
  - Imaging: CT for associated mass lesion
  - Labs: glucose, electrolytes, urine and serum toxicology screens
  - CSF
    - Evidence of infection OR
    - No other clear cause from history, exam, CT, and labs

GENERALIZED CONVULSIVE STATUS EPILEPTICUS

- Seizures beget seizures
  - Early treatment = higher chance of success
  - Balance this with side effects of treatment (need for intubation, hypotension)
- Excitotoxic neuronal death

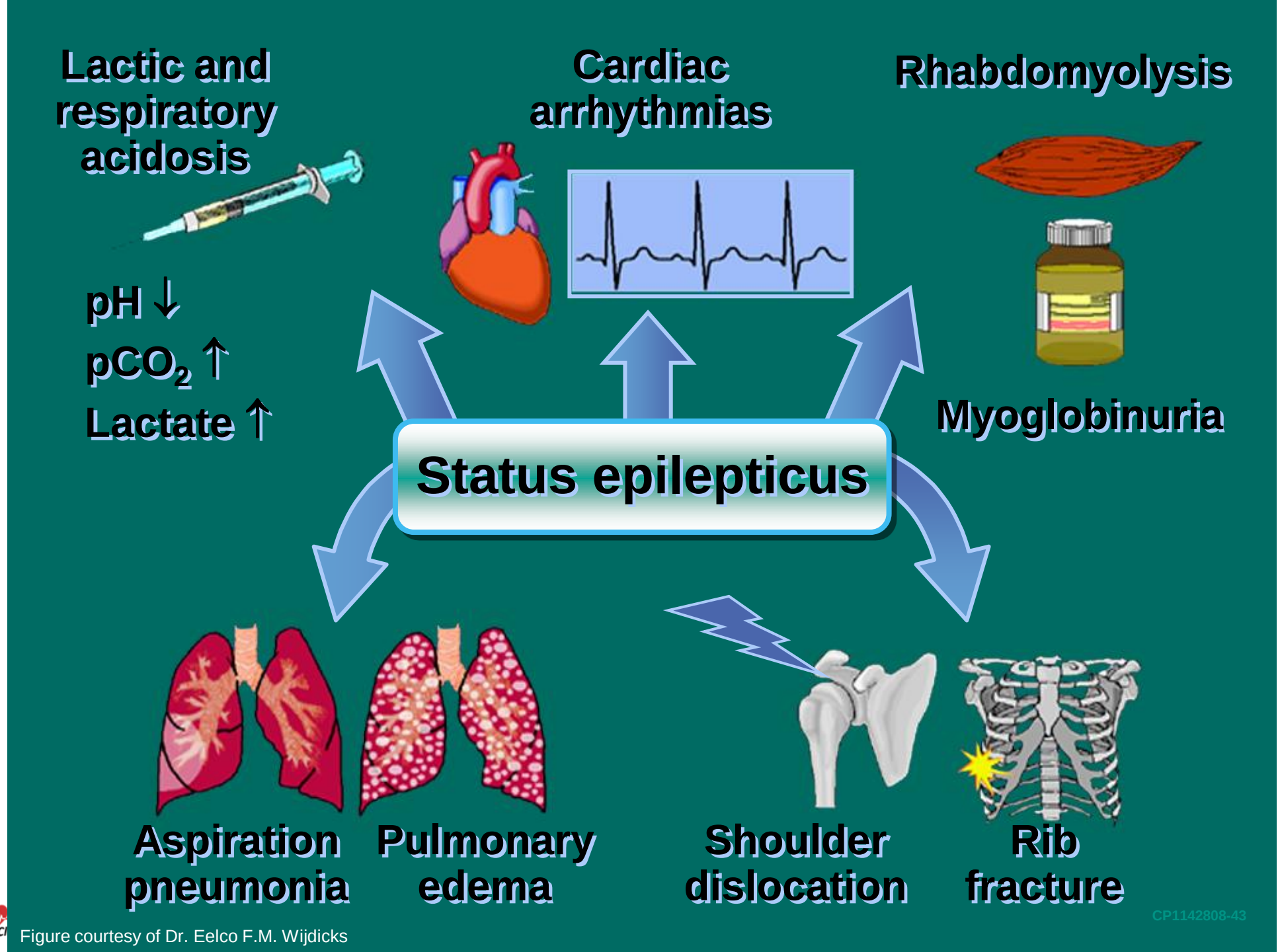
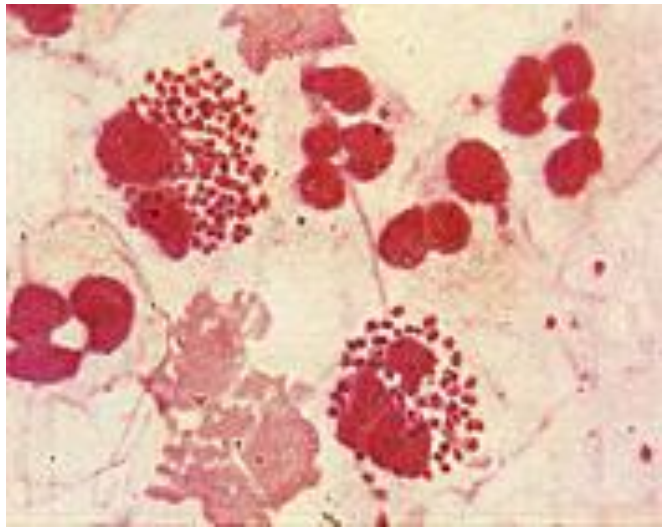


Figure courtesy of Dr. Eelco F.M. Wijdicks

EMERGENCY TREATMENT OF GENERALIZED CONVULSIVE STATUS EPILEPTICUS

- Abort the seizure
  - Lorazepam 4-6mg IV push
  - Repeat 5min later if seizure continues or returns

- Prevent future seizures
  - Phenytoin load: 20mg/kg IV infusion
  - DO NOT just give 1g → only enough for a small, 50kg person
  - Alternatives:
    - IV valproic acid 20-30mg/kg
    - IV levetiracetam 25-30mg/kg



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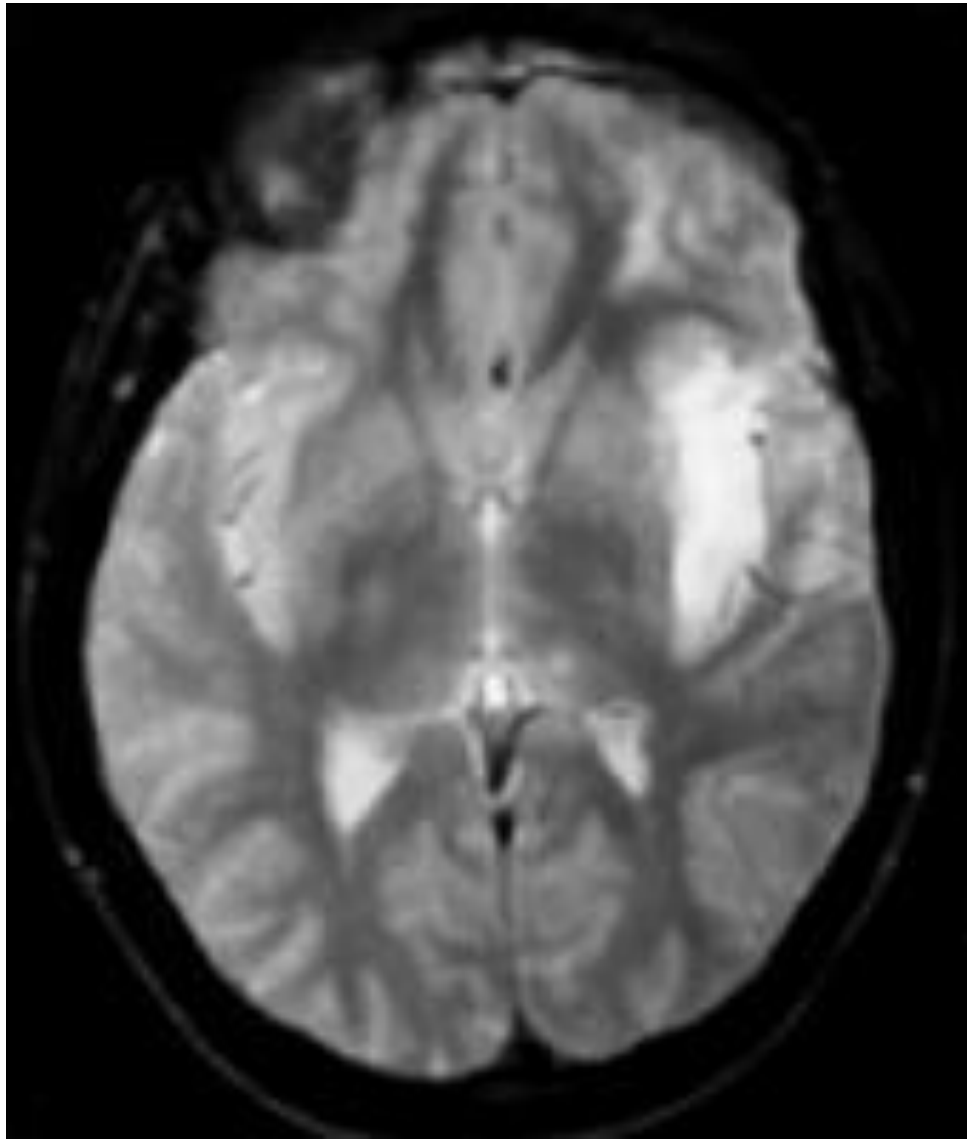

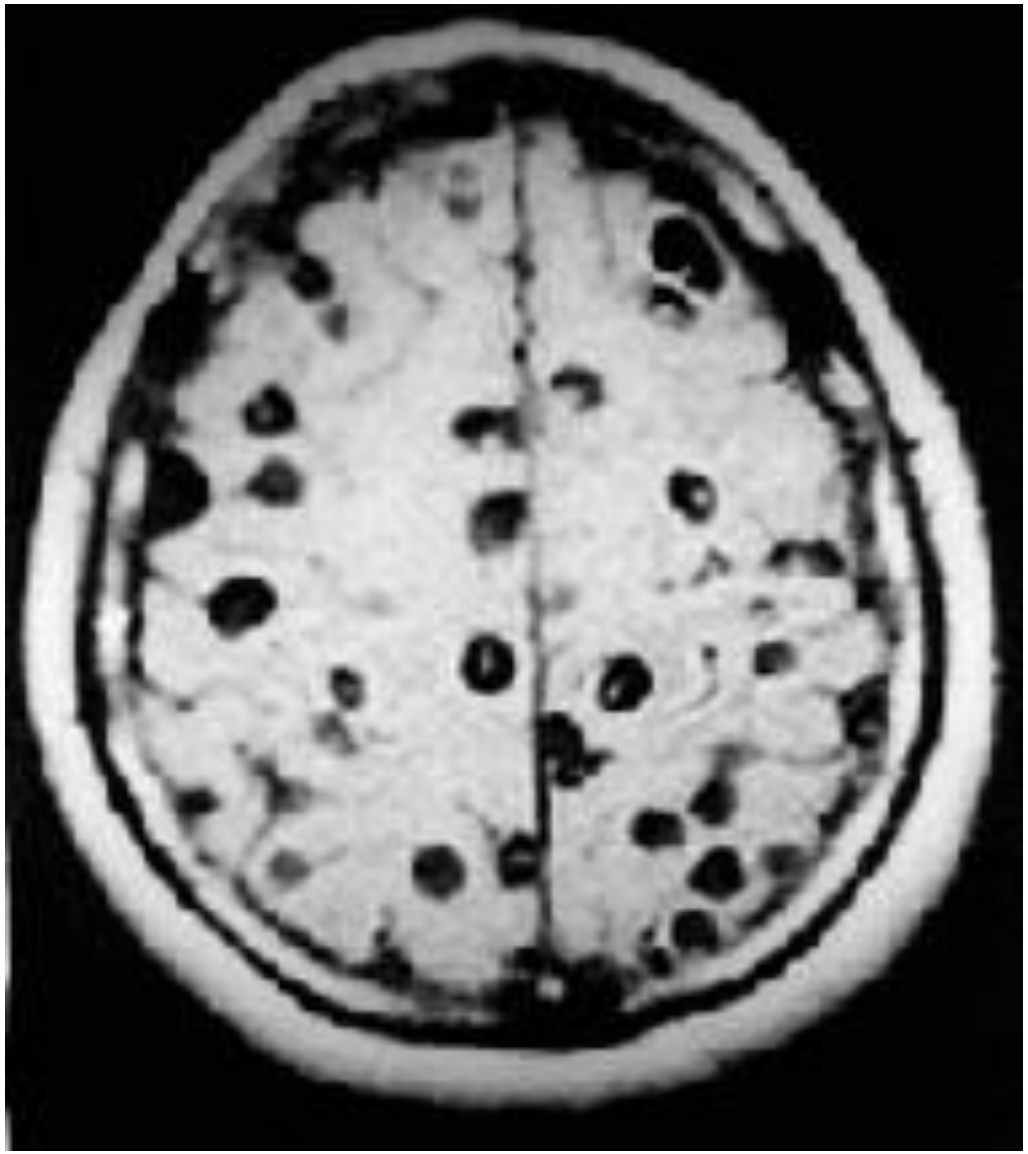

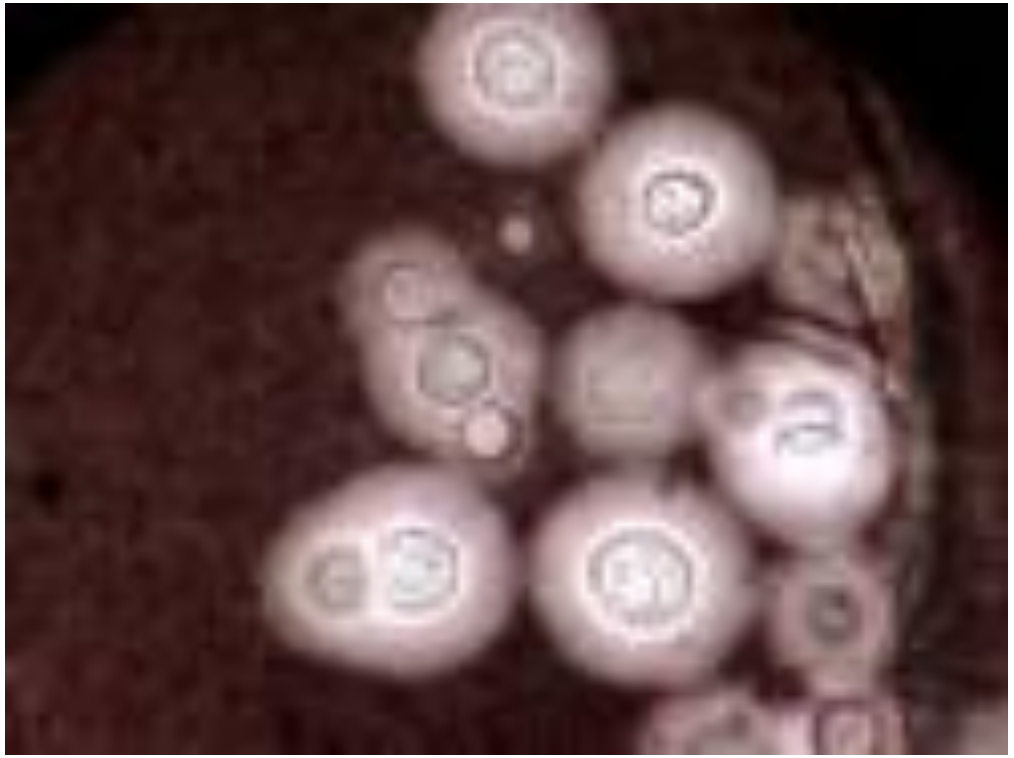

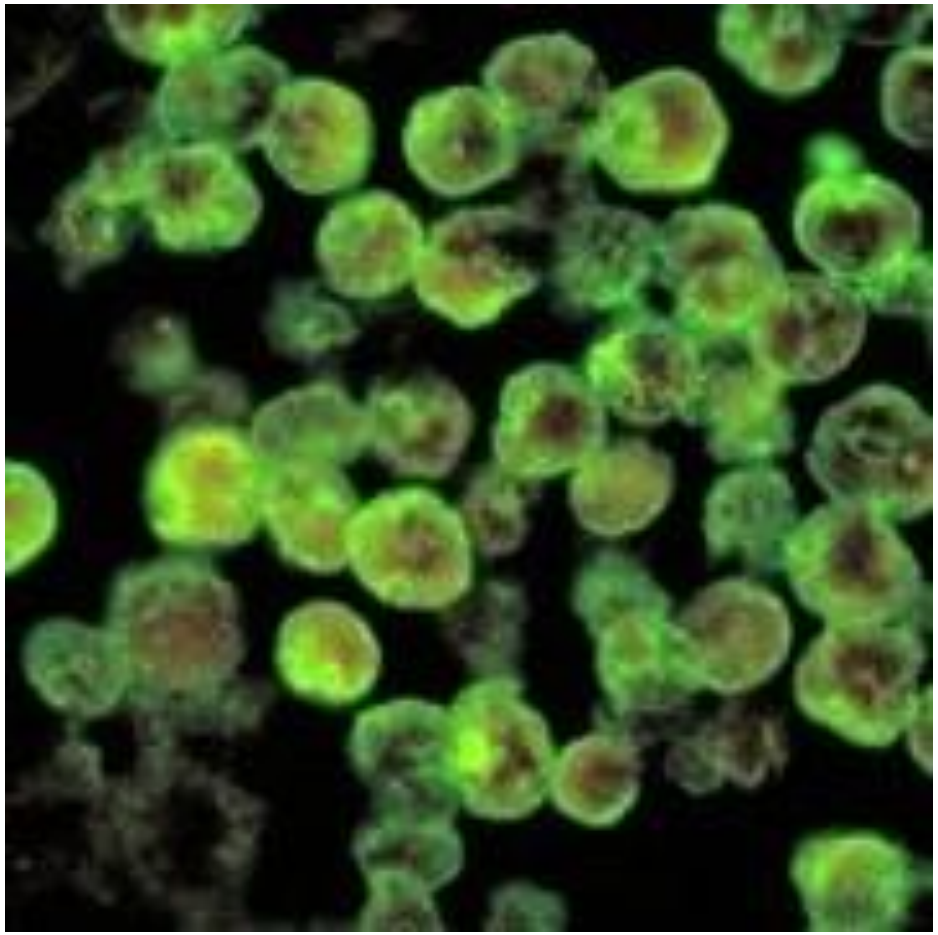

INFECTION

- Meningococcemia
- Herpetic encephalopathy
- Cysticercosis
  - *Taenia solium* (pork tapeworm)
  - PZA
- Toxoplasmosis
- Cryptococcus
- *Naegleria fowler*
  - Ampho B

HOW DO YOU TREAT SEIZURES RELATED TO…

Eclampsia?

Isoniazid overdose?

Status epilepticus?

HOW DO YOU TREAT SEIZURES RELATED TO…

Eclampsia?	Magnesium sulfate
Isoniazid overdose?	Pyridoxine (Vit B6)
Status epilepticus?	1. Benzos 2. Phenytoin/Fosphenytoin 3. Phenobarbital

WHAT IS THE TREATMENT FOR …

CVA in a sickle cell patient?

Tick paralysis?

Healthcare worker exposed to *Neisseria* meningitis patient?

WHAT IS THE TREATMENT FOR …

CVA in a sickle cell patient?

Exchange transfusion

Tick paralysis?

Remove the tick

Healthcare worker exposed to *Neisseria* meningitis patient?

Rifampin 600mg BID x2d OR
Cipro 500mg PO x1 OR
Ceftriaxone 250mg IM x1