Clinical Case Discussion

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CUSOM

October 30, 2025

CAMPBELL UNIVERSITY | Jerry M. Wallace School of Osteopathic Medicine

A 63-year-old woman is brought to the ER after her husband found her with new onset R- sided weakness and inability to speak. She was last seen feeling well and functioning normally about one hour before.

According to her family, she has had no recent illnesses. Her appetite has been good. Her weight has been stable. She has not complained of fevers, chills, chest discomfort, difficulty breathing, changes in bowel movements or urination. She has not complained of recent changes in vision, slurred speech, focal weakness, balance problems or headache. She has not fallen recently and has had no other trauma.

Medications:

- ASA 81 mg po daily
- Lisinopril 20 mg po daily
- HCTZ 25 mg po daily
- Metformin 1000 mg po bid
- Alendronate 70 mg po weekly

PMH:

- HTN
- Type 2 diabetes mellitus
- Osteoporosis
- Osteoarthritis of the knees

Social history: She lives with her husband. She works at a local store. She smoked 1ppd x 20 years but quit 10 years ago. There is no hx of alcohol or drug use. She walks for exercise for 20 minutes three times weekly.

Fam hx:

- Mother d. 89 stroke; hx HTN
- Father d. 90, COPD; hx type 2 diabetes
- 1 brother age 60, HTN
- 1 sister age 56, HTN
- Three children, ages 38, 35, and 30, all healthy

On exam, the patient is a mildly overweight woman who is sleepy and aphasic. She opens her eyes briefly with verbal and tactile stimuli and follows some commands.

Vitals: bp 170/100 p 84 RR 20 temp afebrile O2 sat 93% on RA

HEENT:	PERRL; eyes conjugate oropharynx: moist mucous membranes; no exudate
Neck:	supple; no anterior or posterior cervical adenopathy; trachea midline; no thyromegaly
Car:	r/r/r without r/m/g; no JVD; there is a soft left carotid bruit
Lungs:	CTA without w/r/r
Abd:	nondistended; soft, nontender; no organomegaly
Extr:	no edema; dp pulses 1+ symmetric
Skin:	no rashes or other skin lesions

Neuro:

Cranial nerves:

II,III:	PERRL; eyes conjugate; she has a right visual field cut to confrontational testing
III, IV, VI:	EOMI
V:	facial sensation to light touch symmetric
VII:	there is a R lower facial drop; strength is 5/5 over both sides of the forehead
VIII:	hearing adequate to finger rub
IX, X:	soft palate elevates symmetrically with gag
XI:	shoulder shrug 5/5 bilaterally
XII:	tongue deviates to the L

Motor:

- Bulk: normal in all four extremities
- Tone: R arm flaccid; R leg tone normal
- Strength: R arm 0/5
  R leg: hip flexion 4/5; leg extension 4/5; plantar flexion 4/5
  L arm: 5/5 throughout
  L leg: 5/5 throughout

Sensation: Withdraws to painful stimuli to L arm and L leg only

Cerebellar: L finger to nose and heel to shin testing intact; unable to complete R finger to nose or heel to shin testing.

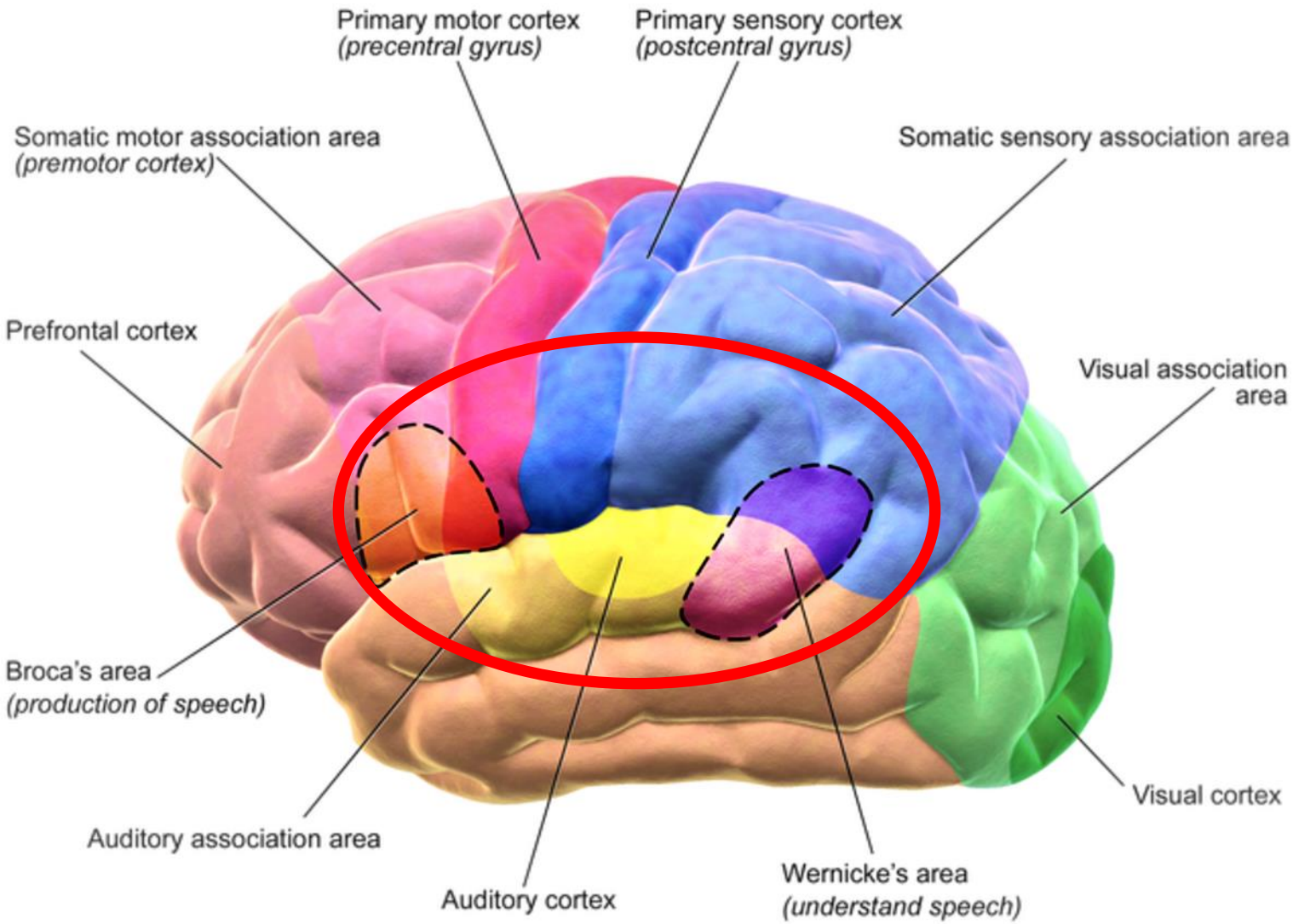
Reflexes:

	Right	Left
biceps	2+	2+
triceps	2+	2+
brachioradialis	2+	2+
patellar	1+	1+
ankle jerks	3+	1+
Babinski	present	absent

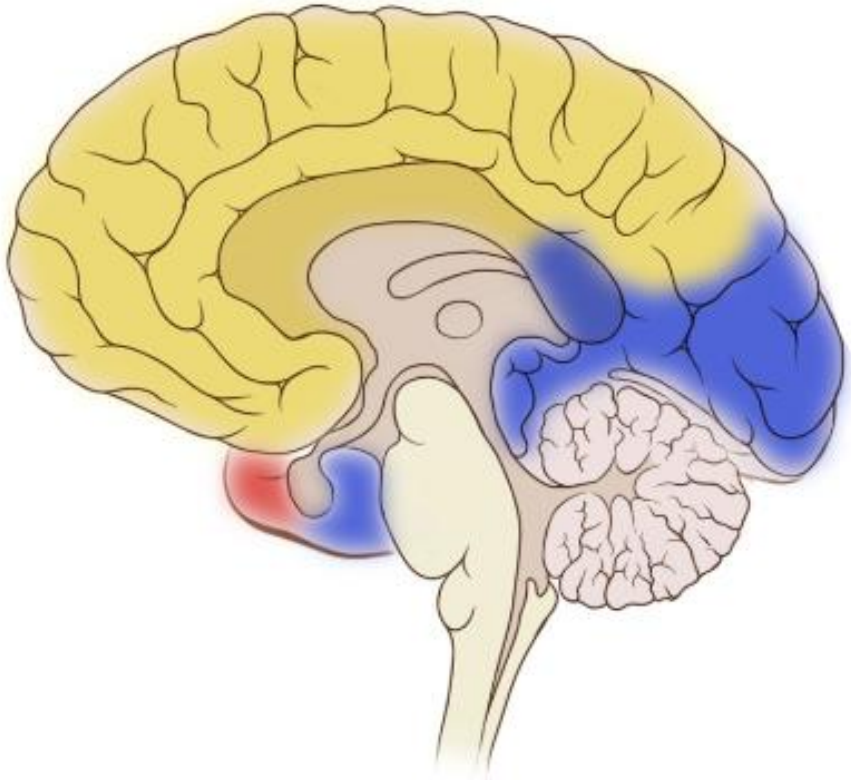
Where are we in the nervous system?

- Cerebral cortex
- Subcortical structures
- Brain stem
- Spinal Cord
- Peripheral nerves
- Neuromuscular junction
- Muscles

Motor and Sensory Regions of the Cerebral Cortex



Medial view



Lateral view

Posterior cerebral artery

Middle cerebral artery

Anterior cerebral artery

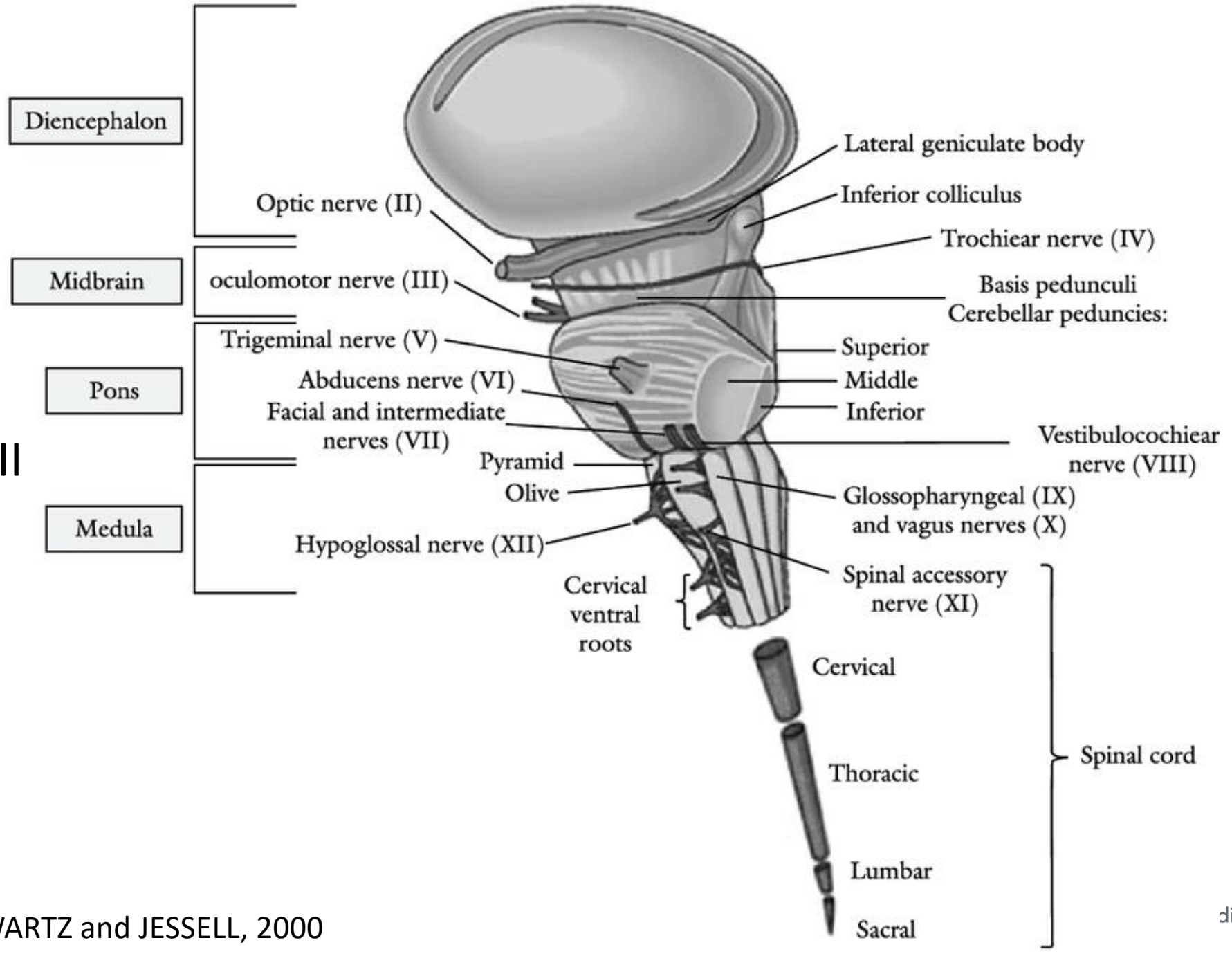
GEEKYMEDICS.COM

Clinical Neuroanatomy: Cranial nerves

Midbrain:
CNs III, IV

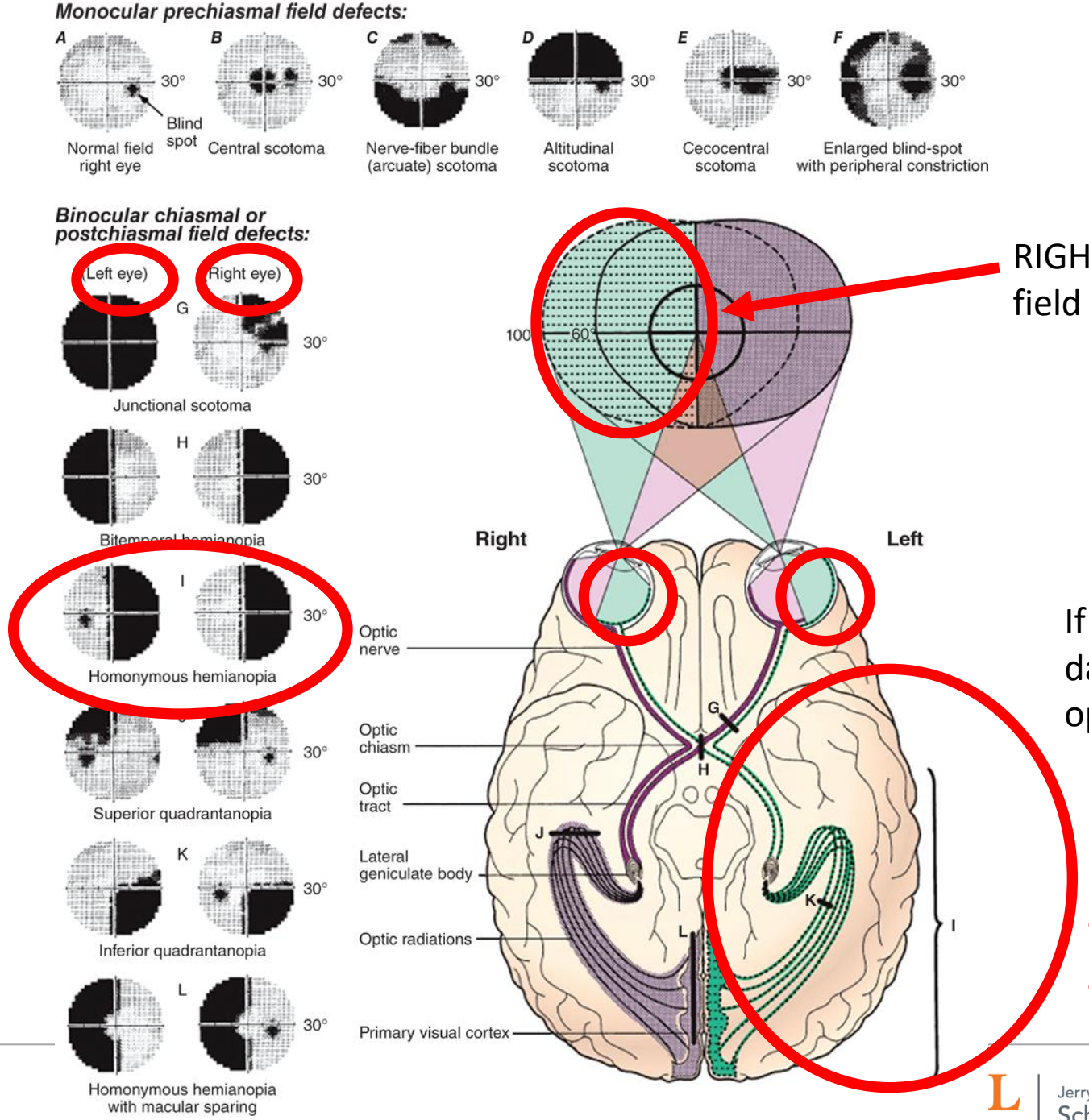
Pons:
CNs V, VI, VII, VIII

Medulla
CNs IX, X, XI, XII



Adapted from KANDEL, SCHWARTZ and JESSELL, 2000

Results in a RIGHT-sided visual field cut (ie. RIGHT homonymous hemianopia)



RIGHT-sided field cut

If LEFT MCA infarct damages the LEFT optic radiations

We are looking up to the brain from the feet of the patient so R is L and L is R!

Source: J.L. Jameson, A.S. Fauci, D.L. Kasper, S.L. Hauser, D.L. Longo, J. Loscalzo: Harrison's Principles of Internal Medicine, 20th Edition

Citation: Chapter 28 Disorders of the Eye, Jameson J, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J. *Harrison's Principles of Internal Medicine, 20e;* 2018. Available at: https://accessmedicine.mhmedical.com/content.aspx?sectionid=192011900&bookid=2129 Accessed: May 18, 2021

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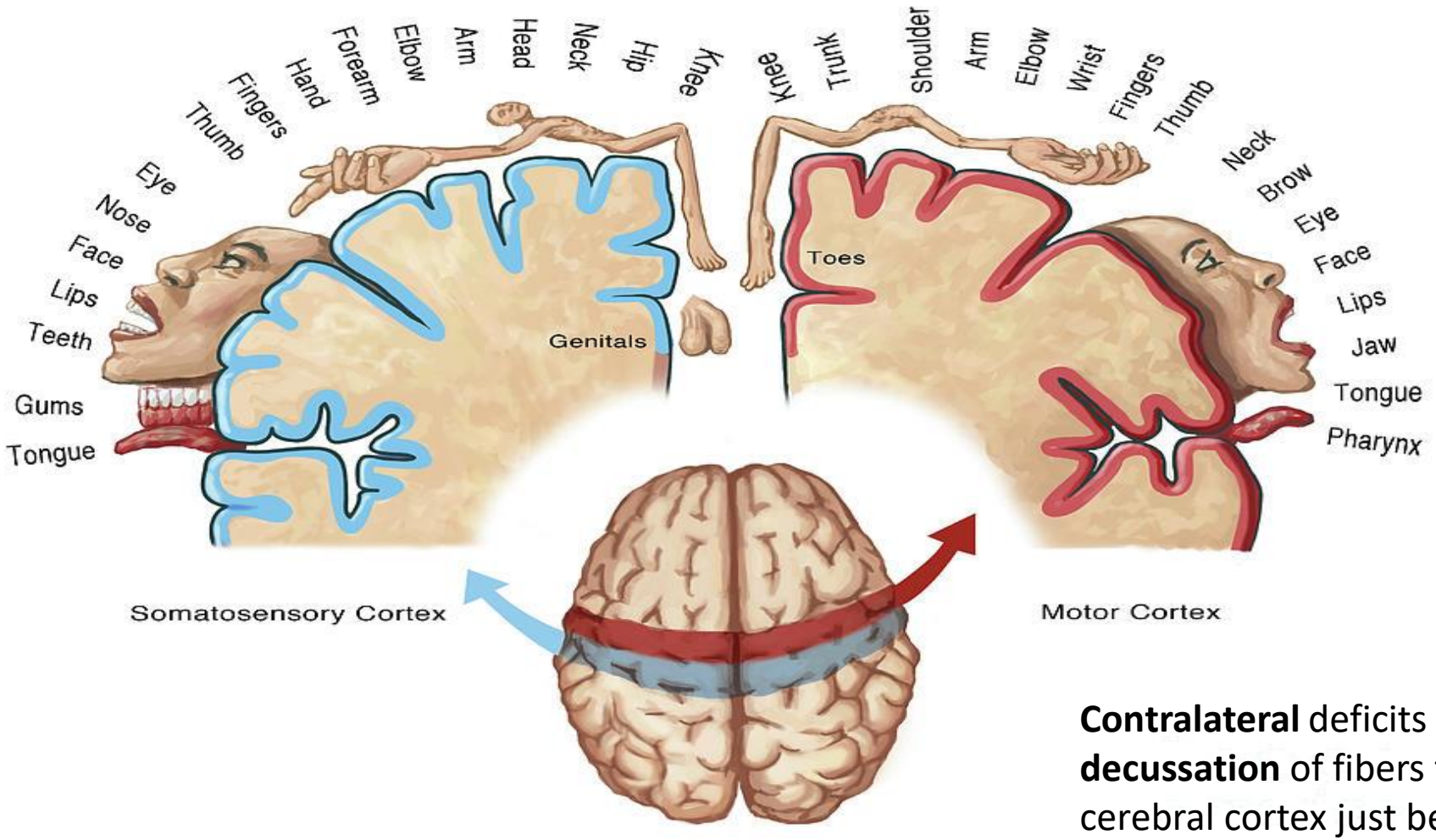
Motor Exam

Pronator Drift

- Detects subtle contralateral upper motor neuron deficits not detected by other components of the motor exam

Clinical Neuroanatomy: Motor exam

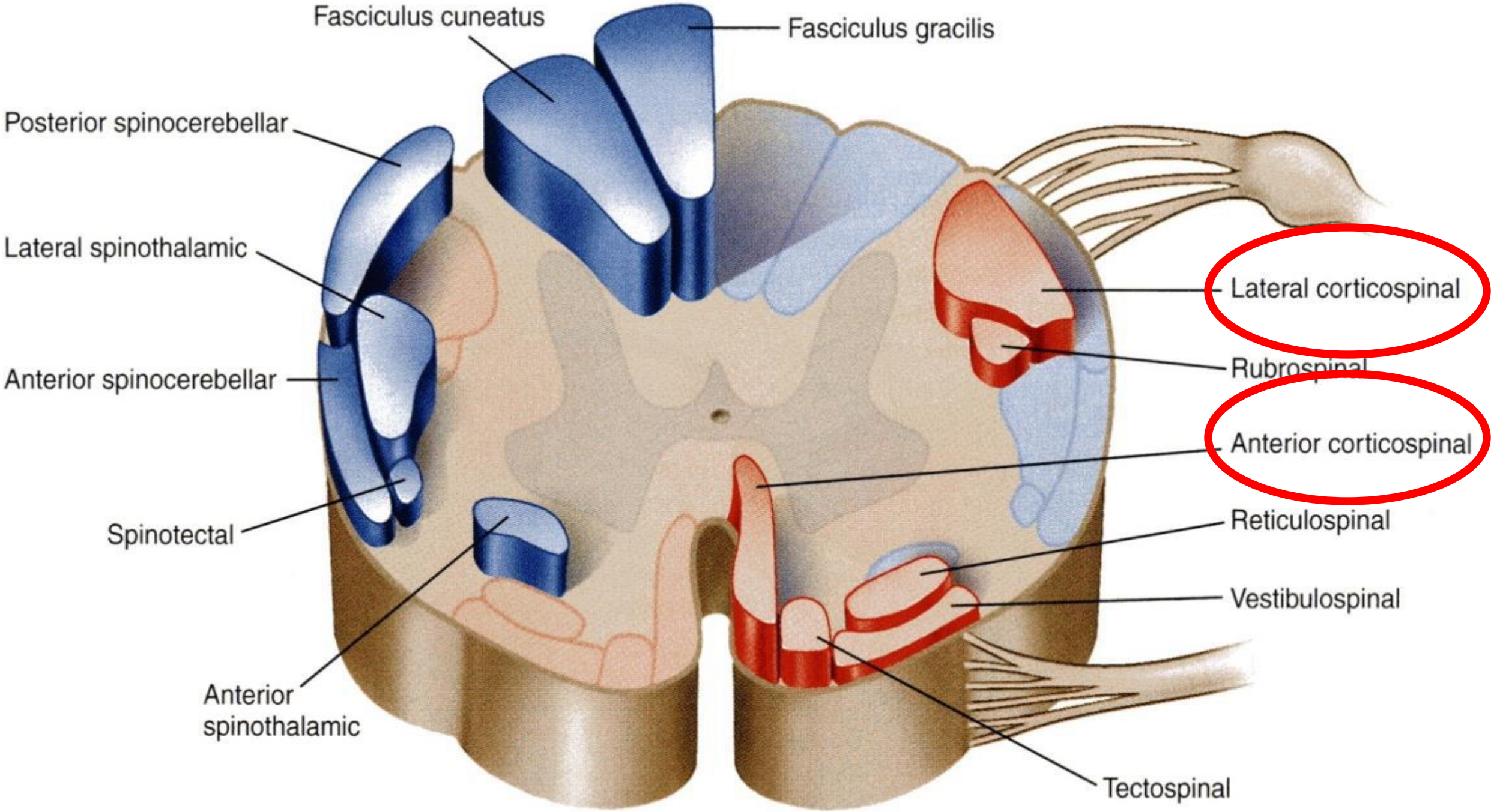


Contralateral deficits occur due to **decussation** of fibers from the cerebral cortex just before the **junction between the medulla oblongata and the spinal cord.**

The Homunculus

Fineartamerica.com

Corticospinal Tract


Fasciculus cuneatus
Fasciculus gracilis
Posterior spinocerebellar
Lateral spinothalamic
Anterior spinocerebellar
Spinotectal
Anterior spinothalamic
Lateral corticospinal
Rubrospinal
Anterior corticospinal
Reticulospinal
Vestibulospinal
Tectospinal

Corticospinal Tract:
Main descending (motor) tract

Motor Cortex

Internal Capsule

Midbrain

Pons

Most Fibers cross (decussate) at medulla

Lateral corticospinal tract

Anterior horn cells

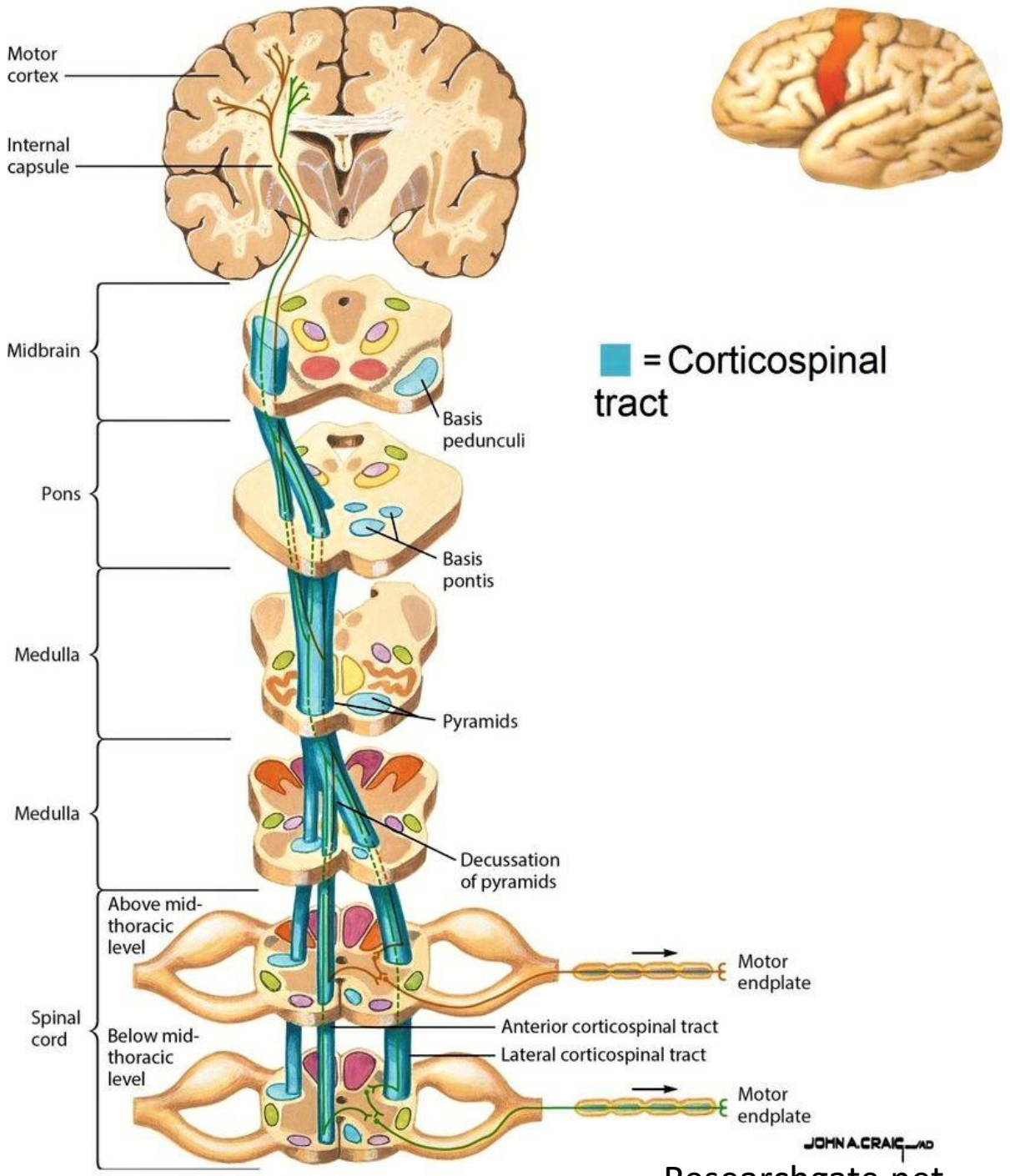
Motor end plates to muscles

Upper motor neurons

Lower motor neurons

This is why a small lacunar infarct in the internal capsule is a BIG problem

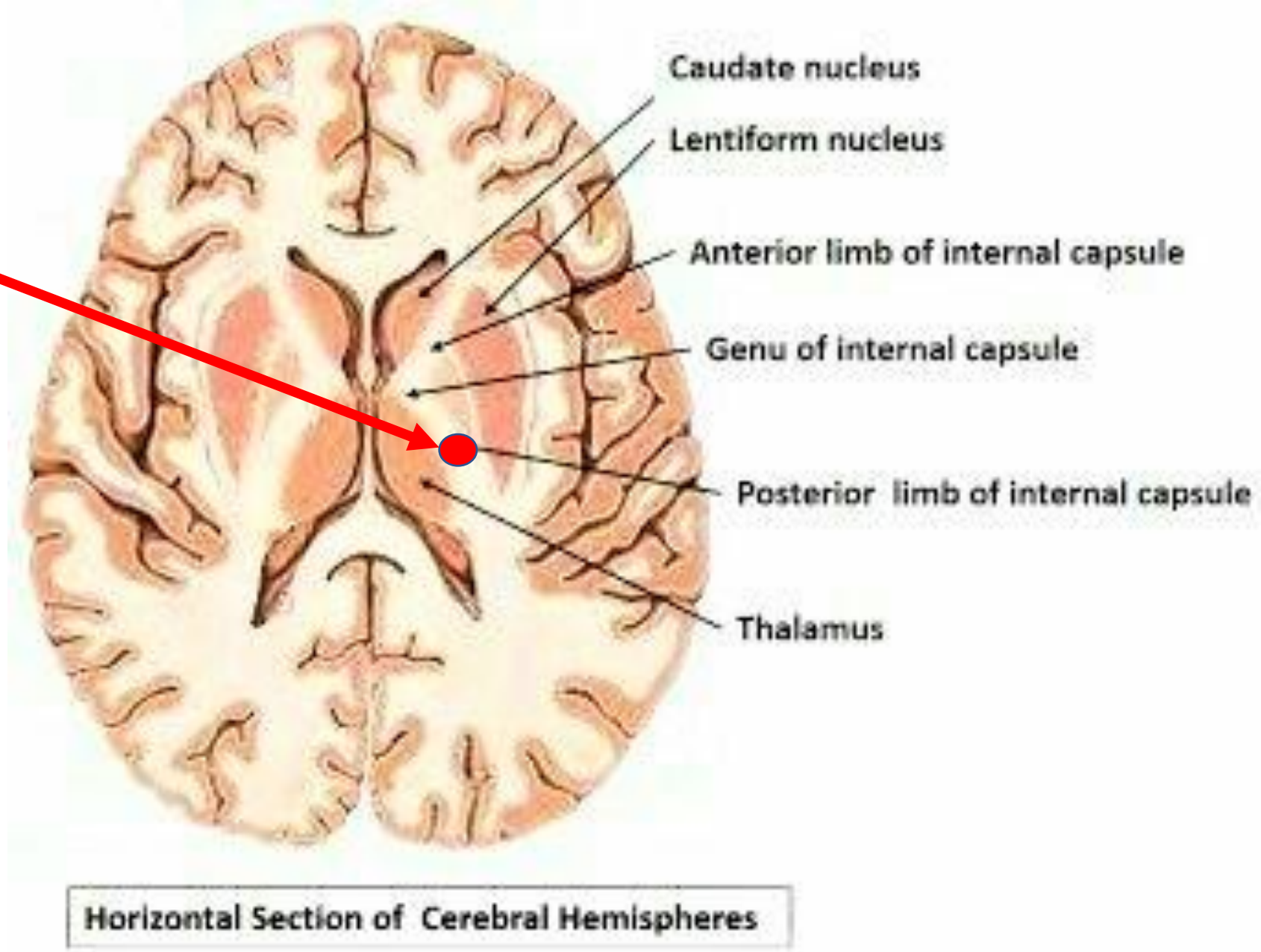
On exam, motor deficits are contralateral to a lesion in the motor cortex



Researchgate.net

Clinical example:
Patient with a lacunar infarct in the posterior limb of the LEFT internal capsule:

- Normal mental status exam
- 4/5 strength in the RIGHT ARM
- No other deficits



Coordination/Cerebellar Function

Clinical Neuroanatomy: Cerebellar function

Anterior (ventral) spinocerebellar tract

Anterior (ventral) Spinocerebellar Tract

Most fibers then decussate AGAIN in the pons to the ipsilateral cerebellar peduncle to the cerebellum

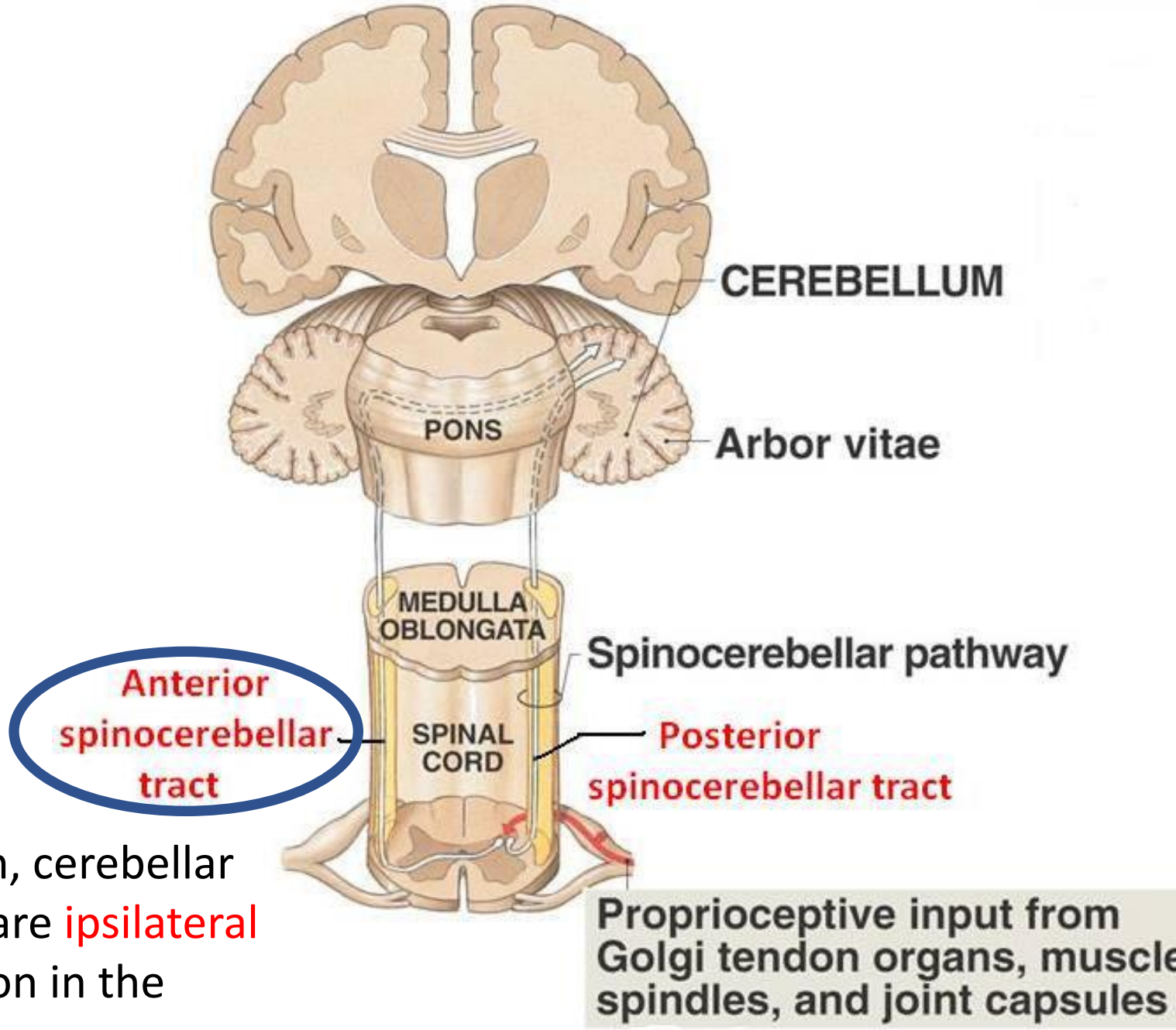
Fibers ascend via the contralateral anterior (ventral) spinocerebellar tract

Fibers decussate at level of spinal cord through anterior white commissure

Dorsal Root Ganglia

Proprioceptive input from Golgi tendon organ, muscle spindles and joint capsules

On exam, cerebellar deficits are ipsilateral to a lesion in the cerebellum

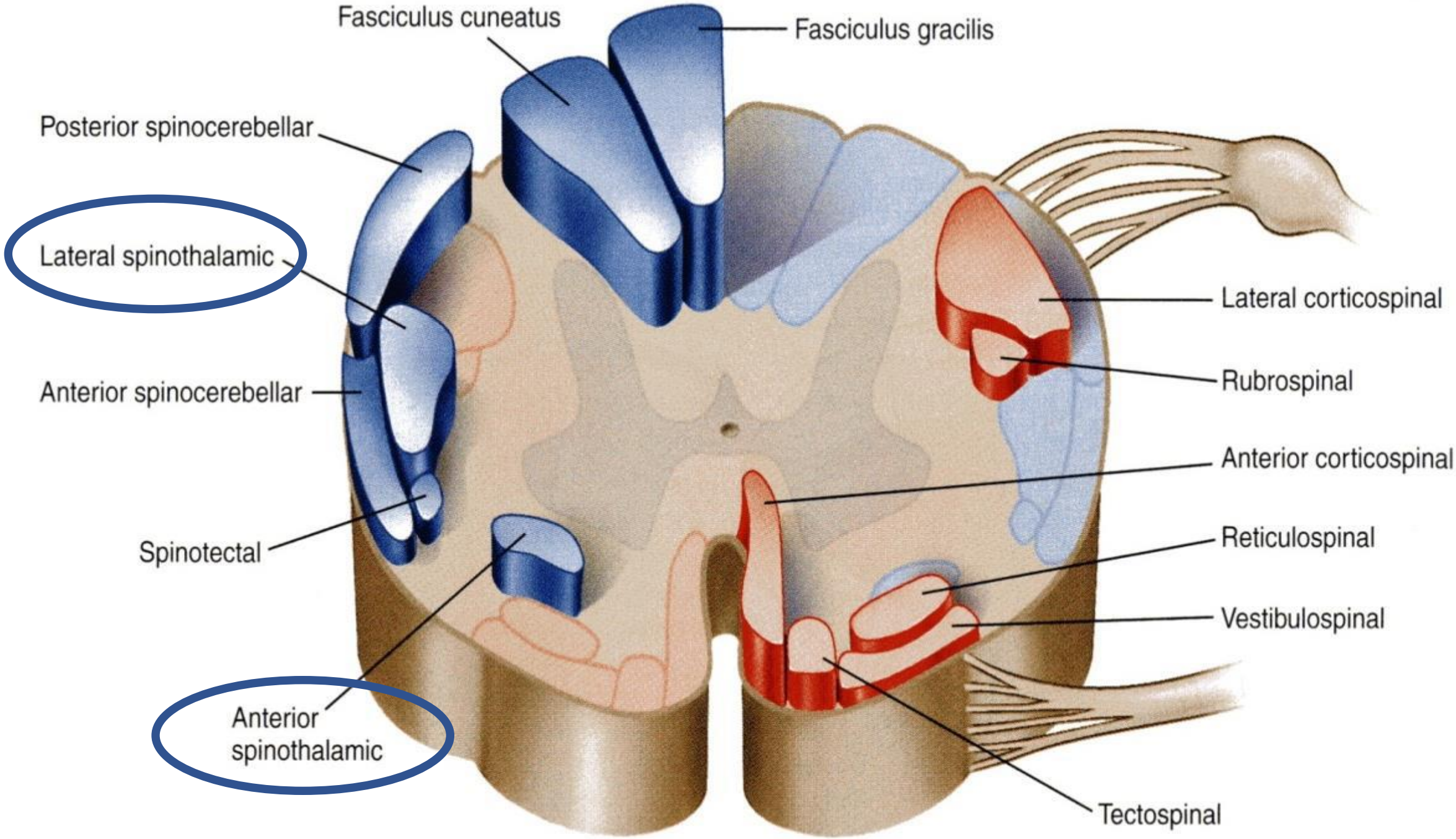


Dr. Jamila El-Medany Dr. Essam Eldin Salama, "Sensory (Ascending) Spinal Tracts

Sensory Exam

Clinical Neuroanatomy: Sensory exam

Spinothalamic tract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Rubrospinal
Anterior corticospinal
Reticulospinal
Vestibulospinal
Tectospinal

Spinothalamic tract

To corona radiata, sensory cortex

Fibers ascend to internal capsule

Fibers ascend to the thalamus

Fibers ascend in the contralateral spinothalamic tract

Fibers decussate in the spinal cord at the anterior white commissure

Spinal Dorsal Root Ganglia

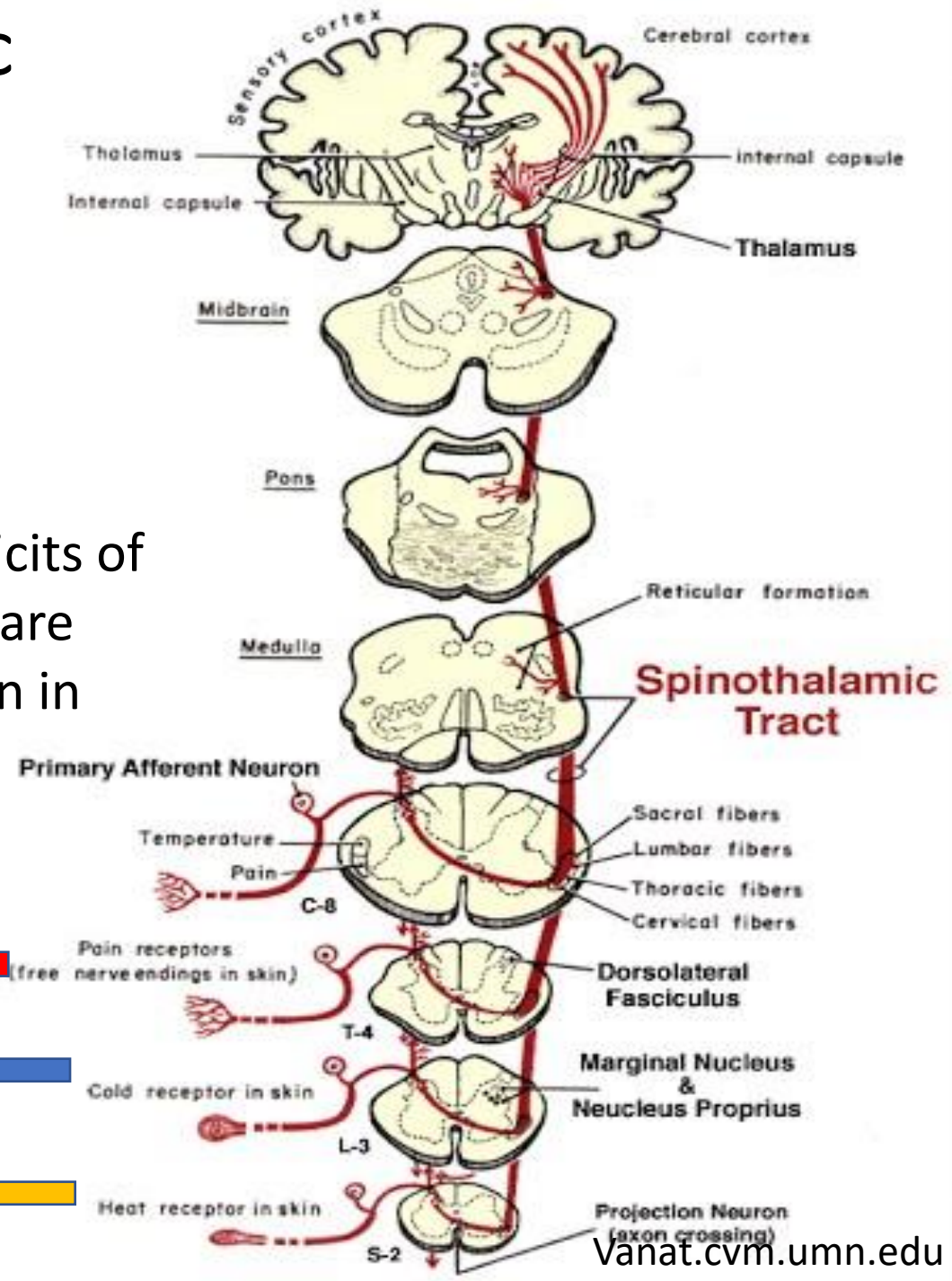
This is why a small lacunar infarct in the internal capsule is a BIG problem

On exam, sensory deficits of pain and temperature are contralateral to a lesion in the sensory cortex

Pain receptors

Cold receptors

Heat receptors



Dorsal Columns

Dorsal (Posterior) Columns

Somatosensory cortex

Thalamus

Midbrain

Pons

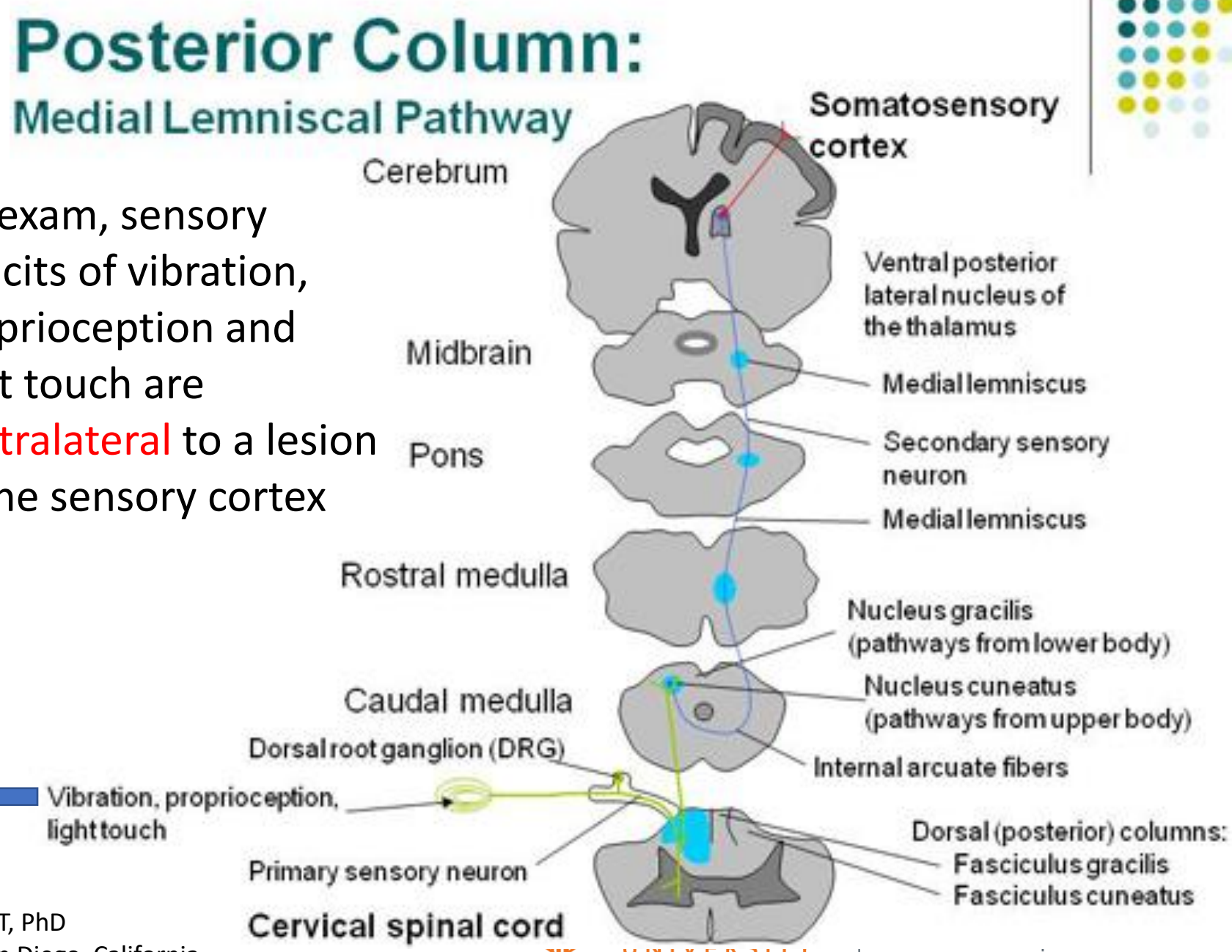
Fibers decussate at the medulla

Fibers ascend via the dorsal columns

Dorsal Root Ganglia

Vibration
Proprioception
Light touch

On exam, sensory deficits of vibration, proprioception and light touch are contralateral to a lesion in the sensory cortex



Reflexes

Clinical Neuroanatomy: Reflexes

Remember where you are in the spinal cord when testing reflexes (“1,2,3,4”)

Reflex	**Spinal nerve root level**
Ankle jerks	S1, S2
Patellar reflexes	L3, L4
Biceps reflexes	C5, C6
Triceps reflexes	C7, C8

Reflexes

- Biceps, triceps, brachioradialis, patellar, ankle jerks, Babinski sign

- Reflex grading:
  - 0 absent
  - 1 hypoactive
  - 2 normal
  - 3 hyperactive
  - 4 hyperactive with clonus

Gait

Gait Testing: integrates motor, sensory and coordination

- Observe patient walking to and from the exam room or down the hall
- Tandem walking
- Toe walking
- Heel walking

Back to our patient . . .

EKG: shows a normal sinus rhythm, rate 80, normal axis, PR interval 156 msec, QRS duration: 110 msec, QTc interval 450 msec. There are nonspecific ST and T wave changes

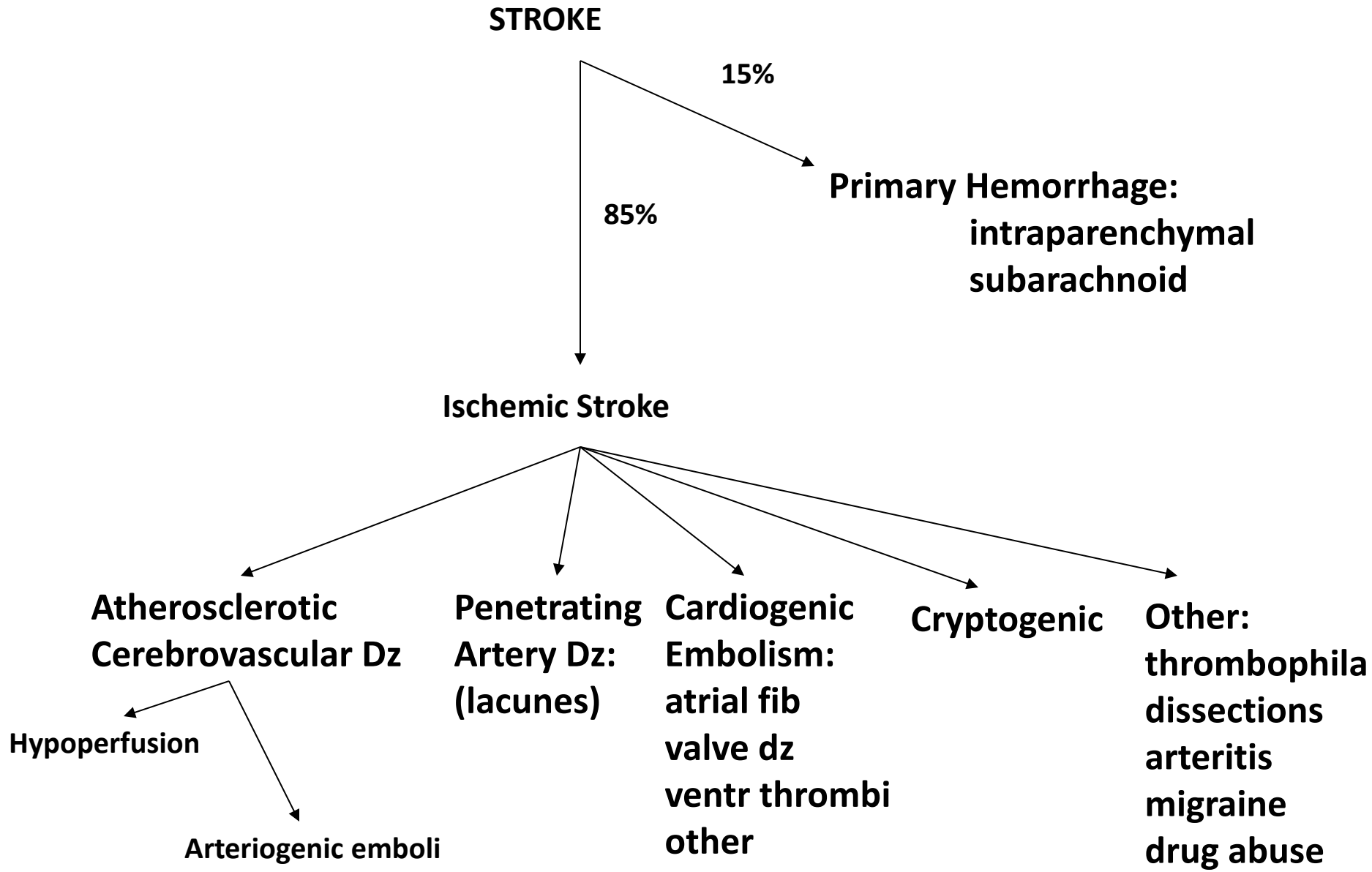
A Head CT is negative for hemorrhage or other acute changes . . .

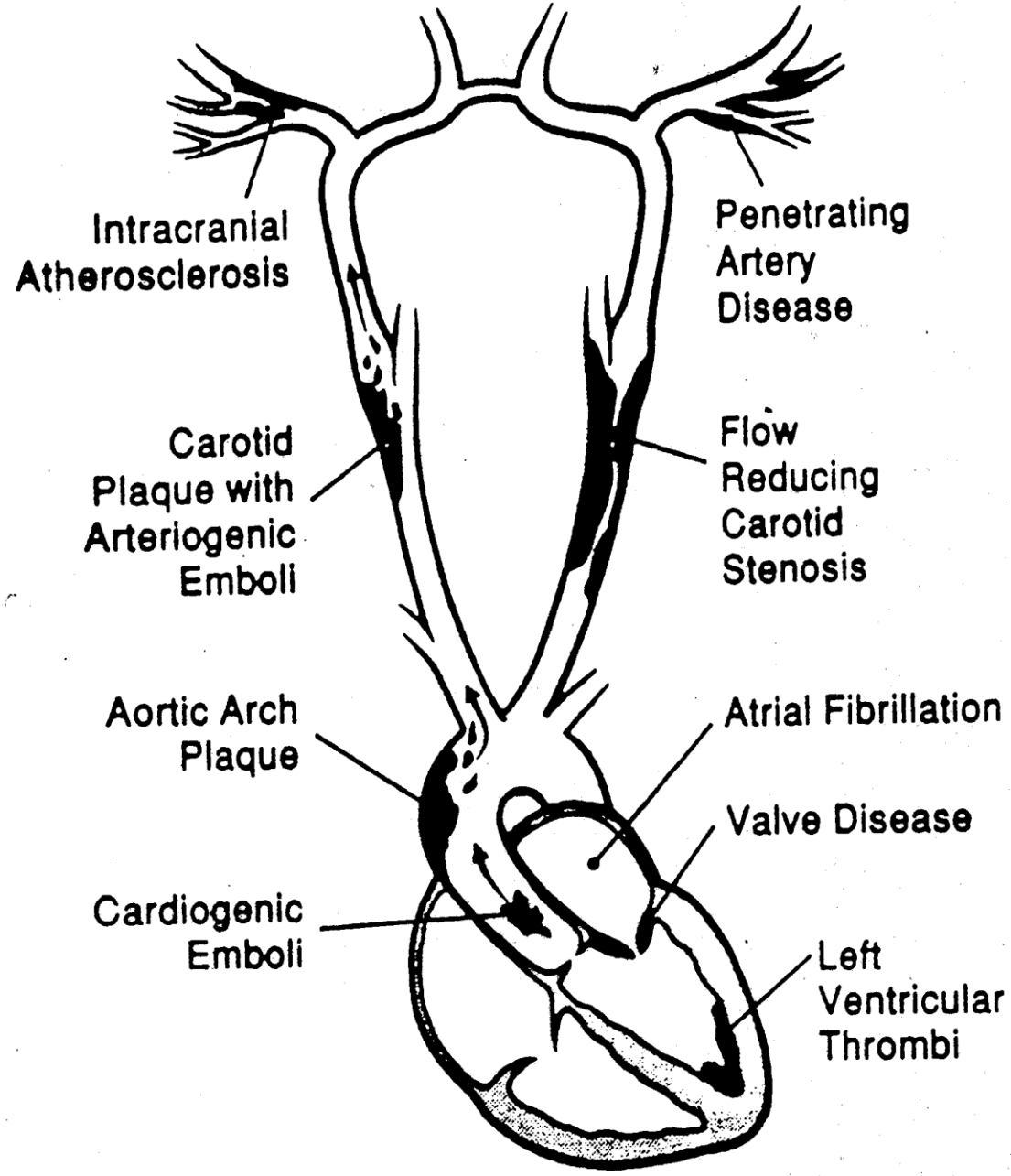
Differential Diagnosis of Acute Stroke

- Migraine
- Intracerebral hemorrhage
- Head trauma
- Brain tumor
- Todd’s palsy
- Functional (conversion reaction)
- Systemic infection
- Toxic-metabolic disturbance

The patient is diagnosed with an acute left middle cerebral artery distribution ischemic stroke.

Per the report of the patient's family, the patient has been compliant with her daily ASA.




Intracranial Atherosclerosis
Penetrating Artery Disease
Carotid Plaque with Arteriogenic Emboli
Flow Reducing Carotid Stenosis
Aortic Arch Plaque
Atrial Fibrillation
Valve Disease
Cardiogenic Emboli
Left Ventricular Thrombi

Hemorrhagic Stroke: Clinical Features

Stroke Type	Clinical Course	Risk Factors	Clues
Intracerebral hemorrhage	Gradual progression over minutes to hours	HTN Trauma Drug abuse Bleeding diatheses	Precipitated by Sex or other physical exertion; Decreased level of consciousness
Subarachnoid hemorrhage	Abrupt onset of sudden severe headache	Drug abuse Bleeding diatheses	See above

Thrombotic or Embolic?

Stroke Type	Clinical Course	Risk Factors	Clues
thrombotic	Stuttering progression with periods of improvement	Atherosclerotic risk factors	May have neck bruit
embolic	Sudden onset with deficit maximal at time of onset	See above	Can be precipitated by getting Up at night to urinate, sudden Coughing or sneezing

Initial Management of the Stroke Patient

Initial management of the stroke patient

- Medically stabilize (ABCs)
- Focused history, exam and labs
- Search for and treat reversible causes
- Imaging studies
  - CT
  - MRI/MRa
  - Carotid u/s
  - Echocardiography
- Aggressive Rehabilitation: PT/OT/Speech and Swallowing\
- DVT prophylaxis

Blood pressure management in acute ischemic stroke

- Most consensus statements recommend that bp not be treated in acute ischemic stroke unless systolic bp > 220 and/or diastolic bp > 120 mm Hg

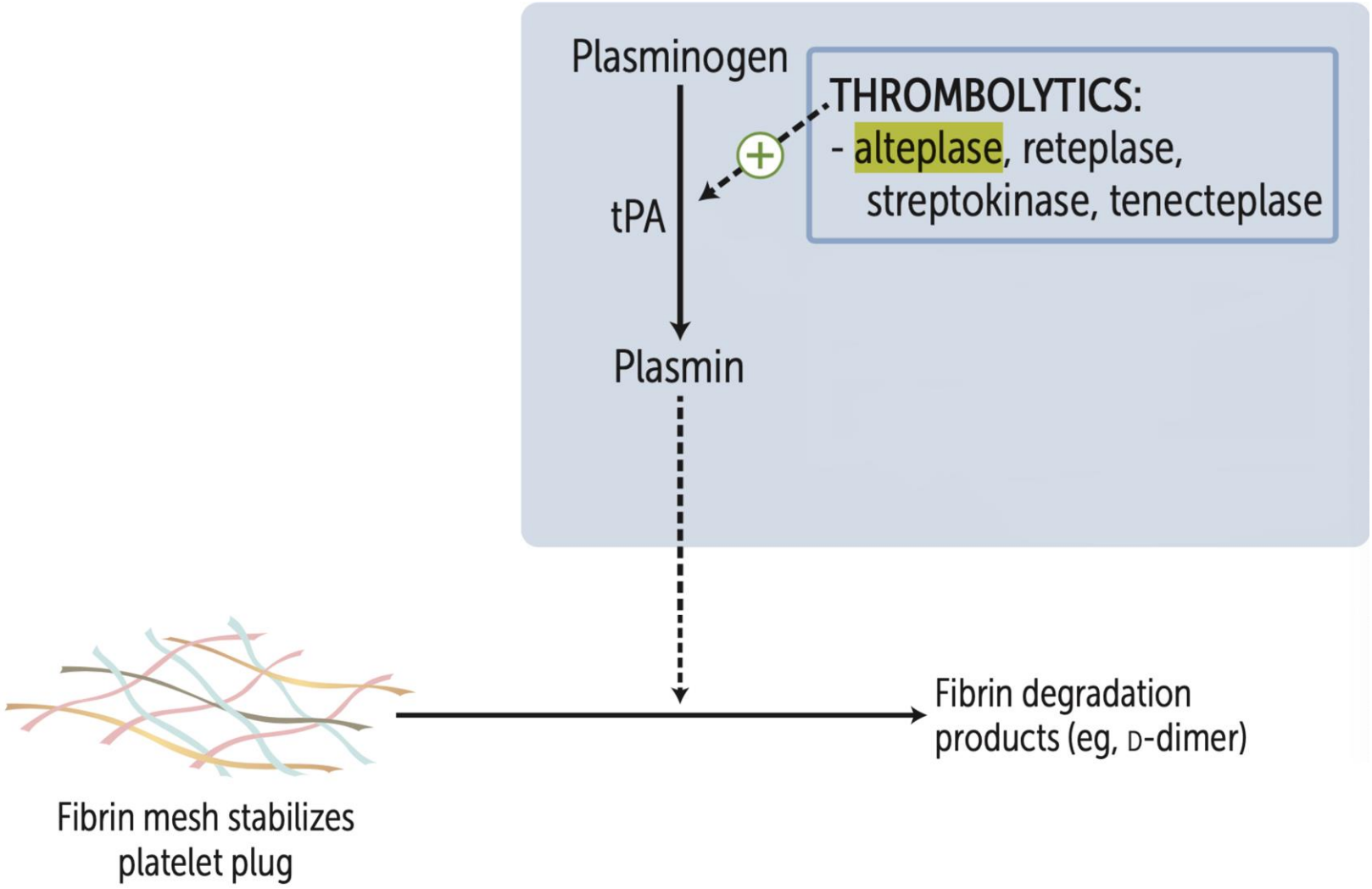
- If thrombolytic therapy is being considered, bp must be below 180/105 mm Hg during thrombolytic infusion and 24 hrs thereafter

Blood pressure management in acute ischemic stroke

- Severe hypertension in acute stroke is common and transient and is likely protective to ischemic brain tissue

- Studies using animal models indicate that that bp returns to baseline in five to ten days

What about Tissue Plasminogen Activator (tPA)???



Thrombolytics: Alteplase (tPA)

Mechanism of action	• Binds fibrin in a clot • Converts plasminogen in the clot to plasmin
Pharmacology	Time of onset: within 5 minutes after infusion stopped Half-life: 5 minutes
Clinical use	• Acute ischemic stroke • Acute ST elevation MI • Acute treatment of massive pulmonary embolism with shock

Thrombolytics: Alteplase (tPA)

Side effects and toxicity	• Bleeding, including intracranial hemorrhage (5% risk)

Means of reversal	• Antifibrinolytic agents:
	  • Aminocaproic acid or
	  • Tranexamic acid
	• Cryoprecipitate
	• Platelet transfusion if platelet count < 100,000

Clinical Case Discussion

- Who should be considered for tPA?
- What are the benefits of tPa?
- What are the risks of tPA?
- What are the contraindications to tPA?
- What other interventions are available to reduce the risk of ischemic stroke?

tPA in Acute Ischemic Stroke

- National Institute of Neurologic Disorders and Stroke Study Group
- Randomized, double blind placebo controlled multicenter trial
  - 8 US centers

(N Engl J Med 1995; 333: 1581-7.)

tPA in Acute Ischemic Stroke

- Patients
  - Acute ischemic stroke
  - No evidence of intracranial hemorrhage on head CT
  - No contraindications to tPA (next slide)
- Intervention
  - tPA infusion given within three hours of onset of stroke symptoms
  - placebo infusion

Contraindications to tPA

- Ischemic stroke within last 3 months
- Hx of intracranial hemorrhage (ever)
- Major surgery within 14 days
- Syst bp > 185 mm Hg or Diast bp > 110 mm Hg
- Rapidly improving or mild symptoms

Contraindications to tPA

- Suspected SAH
- GI or GU hemorrhage within 21 days
- Non compressible arterial puncture
- Seizure at stroke onset
- Coagulopathy

tPA in Acute Ischemic Stroke

Medication	Minimal or no disability three months	Symptomatic intracranial hemorrhage	Mortality at three months
T PA	50% ($p = 0.03$) ARR 12% NNT 8	6.4% ($p < 0.001$) AHI 5.8% NNH 18	17% (NS)
placebo	38%	0.6%	21%

These data are for patients treated with tPA **within 3 hours** of onset of deficits

Follow up data showed that patients may also benefit from tPA given **within 3 to 4.5 hours** of onset of deficits but there are **additional exclusions**:

- Age > 80 years
- Oral Anticoagulant use
- Severe stroke (NIHSS) > 25
- Previous ischemic stroke and diabetes

(N Engl J Med 1995; 333: 1581-7.)

Stroke Risk Reduction

Risk Factor Reduction for Stroke (Primary and Secondary Prevention): “The Big 10”

- Control *chronic* HTN: bp at least < 140/90
- Antiplatelet therapy
- Antithrombotic therapy in atrial fibrillation
- Diabetic glycemic control: Hgb A1C < 7.0%
- Lipid lowering therapy: LDL < 70; HDL > 40

Risk Factor Reduction for Stroke (Primary and Secondary Prevention): “The Big 10”

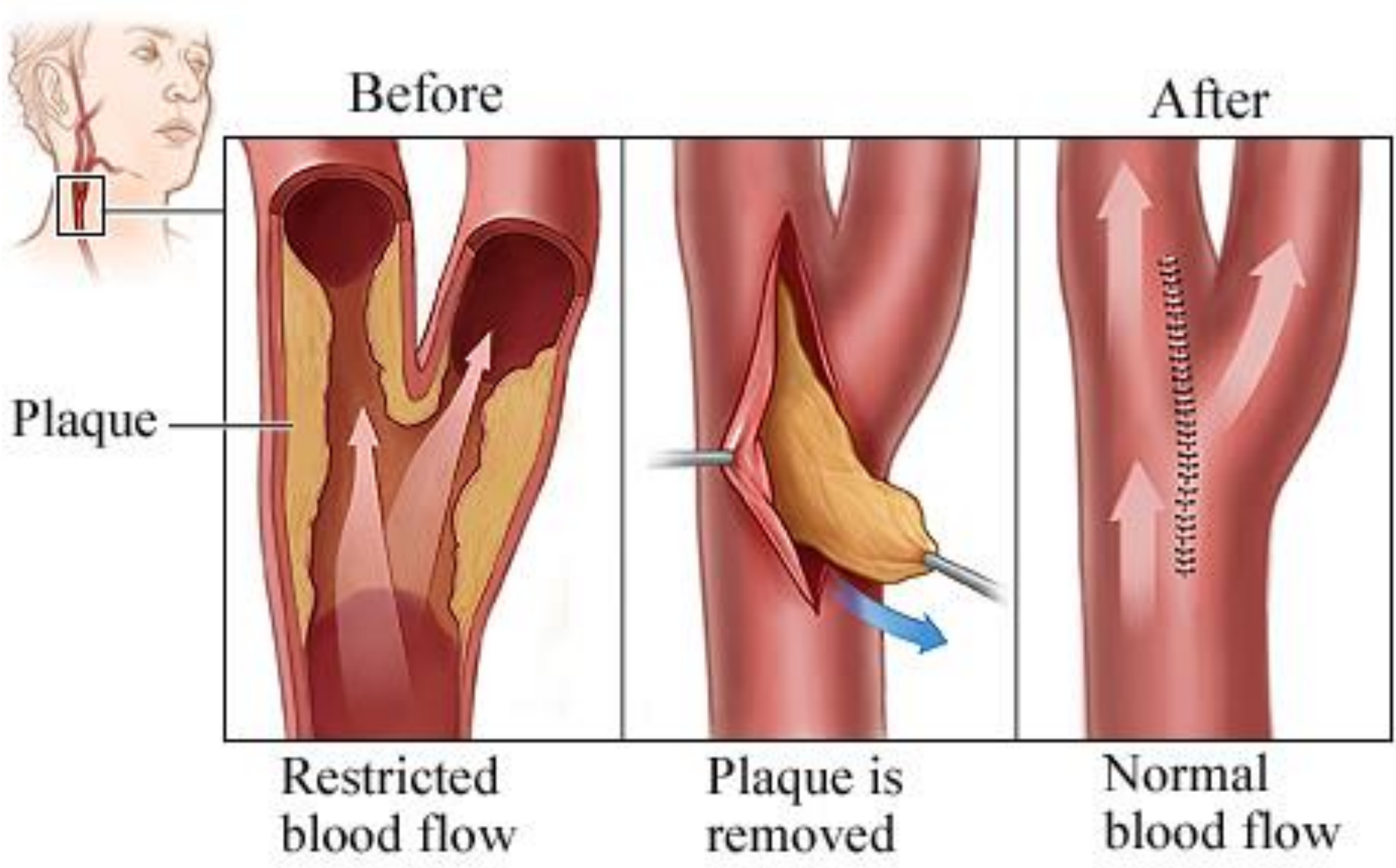
- Consider CEA
- Smoking cessation
- Avoid excess alcohol
- Diet
- Exercise

Carotid Endarterectomy

Carotid Endarterectomy in Stroke Prevention

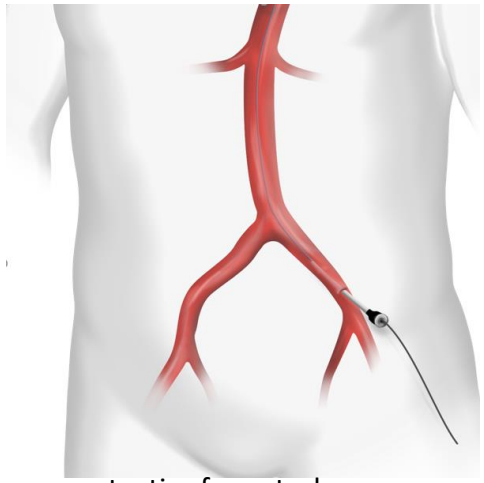
- Multiple randomized trials done in the last 15 yrs: carotid endarterectomy (CEA) vs. medical therapy
- Carotid artery stenosis generally measured by carotid ultrasound
- Both asymptomatic patients and patients with ipsilateral neurologic symptoms have been studied

Carotid Endarterectomy Surgery: The “Gold Standard”

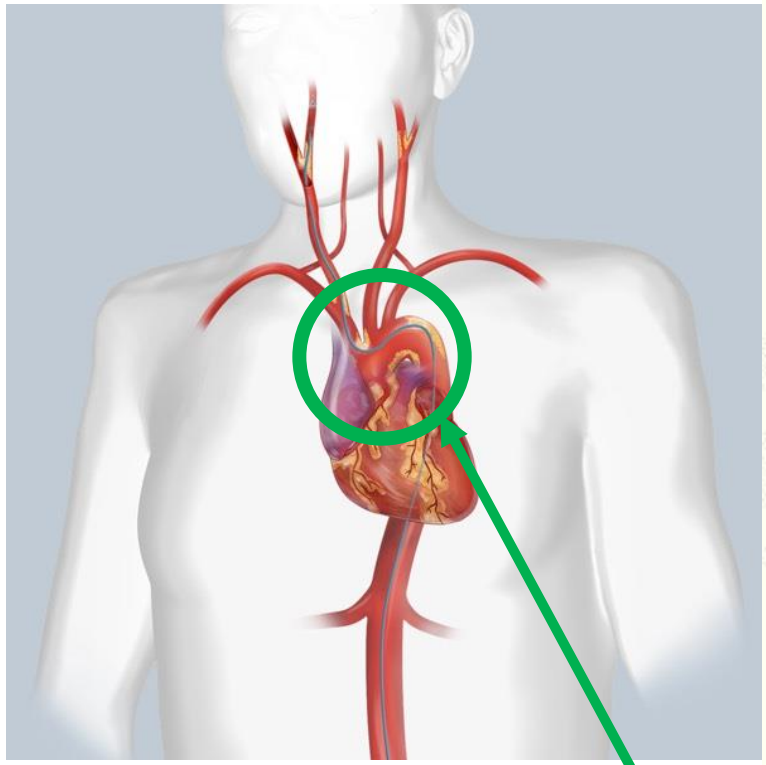


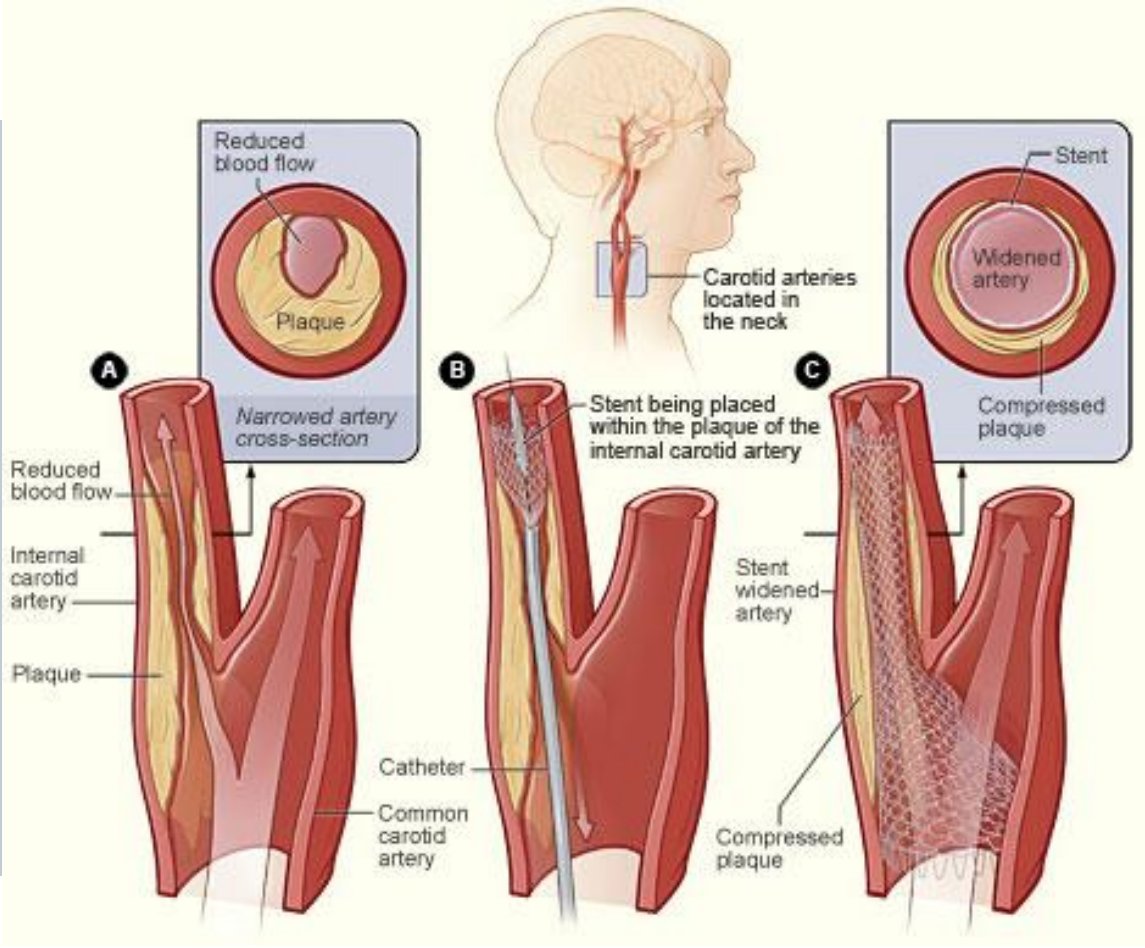
nyp.org

Transfemoral Carotid Stenting



protectionfromstroke.com





wikipedia.org

Higher risk of perioperative stroke and death with transfemoral carotid stenting (TCAS) compared to carotid endarterectomy (CEA)

Outcome (%)	CEA	TCAS
stroke or death at 30 days	5	8.2

Jim J, UpToDate 2022

Why is the risk of a transfemoral procedure higher than the risk of a carotid endarterectomy?

Manipulation of the aortic arch can dislodge atherosclerotic plaques

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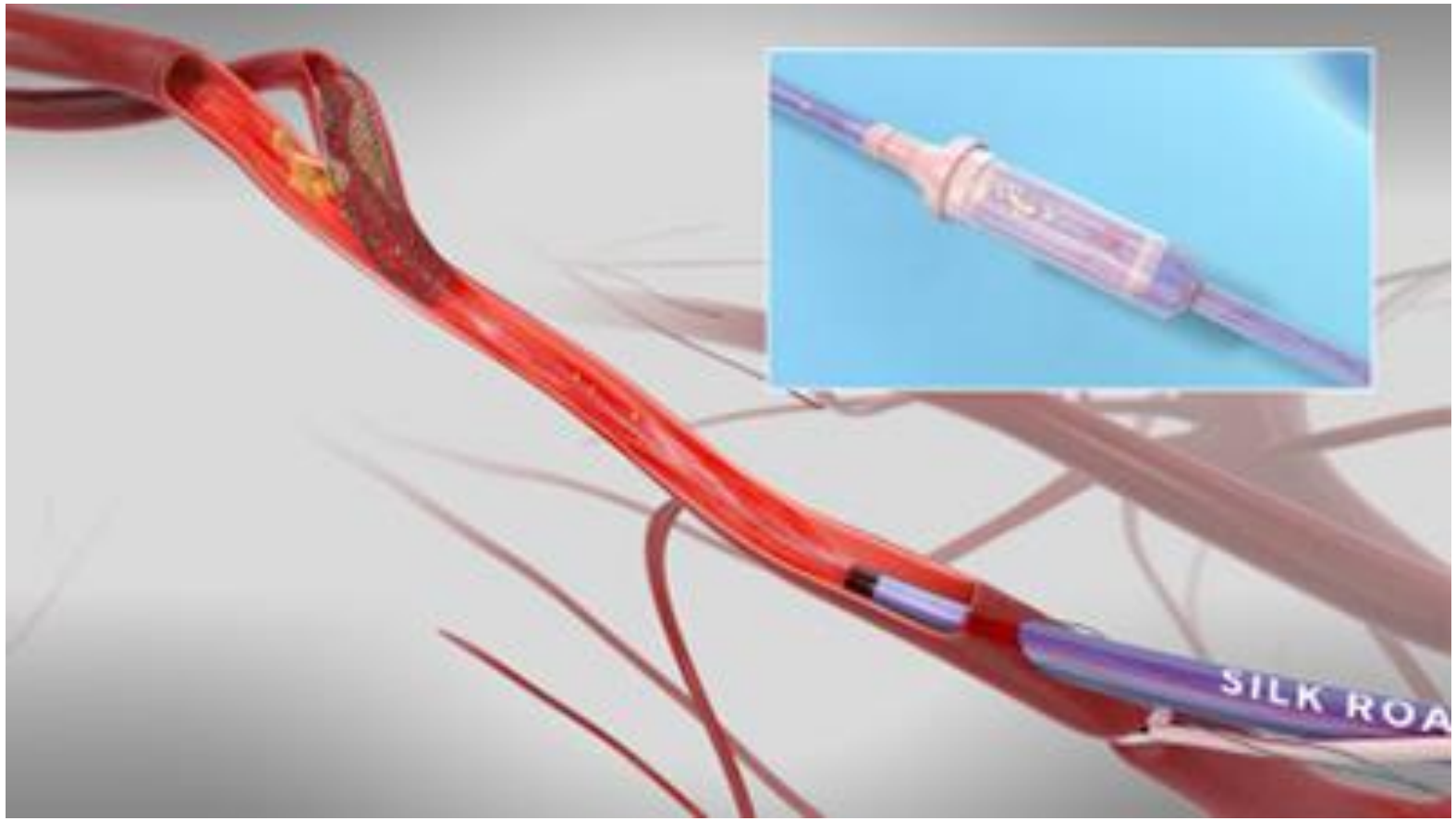
Transcarotid Artery Revascularization (TCAR)



ENROUTE Transcarotid Neuroprotection System

- Direct accesses carotid artery
- Avoids the aortic arch
- Uses a flow reversal mechanism to prevent embolization of plaque during stenting

interventionalnews.com

Outcomes with Transcarotid artery revascularization (TCAR) vs Carotid endarterectomy (CEA): Adjusted Results for ALL PATIENTS

Outcome	Odds ratio (OR)	95% CI	P value
Stroke or death	1.3	0.8 -2.2	0.28
Stroke or death or MI	1.4	0.9-2.1	0.18
Stroke	1.4	0.8-2.5	0.26

J Vasc Surg 2020; 71:87-95.

Outcomes with Transcarotid artery revascularization (TCAR) vs Carotid endarterectomy (CEA): Adjusted Results for ALL PATIENTS

Outcome	Odds ratio (OR)	95% CI	P value
In-hospital death	0.7	0.3-2.1	0.58
30-day death	1.5	0.7-3.2	0.34
MI	1.5	0.7-2.9	0.29

J Vasc Surg 2020; 71:87-95

Outcomes with Transcarotid artery revascularization (TCAR) vs Carotid endarterectomy (CEA): Unadjusted Results

Outcome (%)	**TCAR (n=1182)**	**CEA (n=10,797)**	**P value**
Perioperative hypertension	10	21	<0.001
Perioperative hypotension	13	9.8	0.01
Cranial nerve injury	0.6	1.8	<0.001

J Vasc Surg 2020; 71:87-95

Outcomes with Transcarotid artery revascularization (TCAR) vs Carotid endarterectomy (CEA): Unadjusted Results

Outcome	TCAR (n=1182)	CEA (n=10,797)	P value
Operative time (minutes)	78	111	<0.001
Length of stay > 1 day (%)	27	30	0.046

J Vasc Surg 2020; 71:87-95

Carotid Endarterectomy in Stroke Prevention: AHA Guidelines

- Ipsilateral symptomatic carotid stenosis of 70 to 99% is a proven indication for CEA if surgical risk does not exceed 6%.

- CEA is acceptable, but not of proven benefit for symptomatic patients with 30 to 69% stenosis

- CEA is not beneficial for symptomatic patients with 0 to 29% stenosis

Carotid Endarterectomy in Stroke Prevention: AHA Guidelines

- Asymptomatic patients with stenoses of 60 to 99% have a proven indication for CEA if surgical risk is less than 3% and life expectancy is at least 5 yrs.

Carotid Endarterectomy in Stroke Prevention: AHA Guidelines

- Exclusion criteria for CEA
  - Complete occlusion (100% stenosis)of carotid artery
  - severe comorbidity
  - previous stroke with persistent dense deficits
  - symptomatic patients with hemorrhagic component to stroke

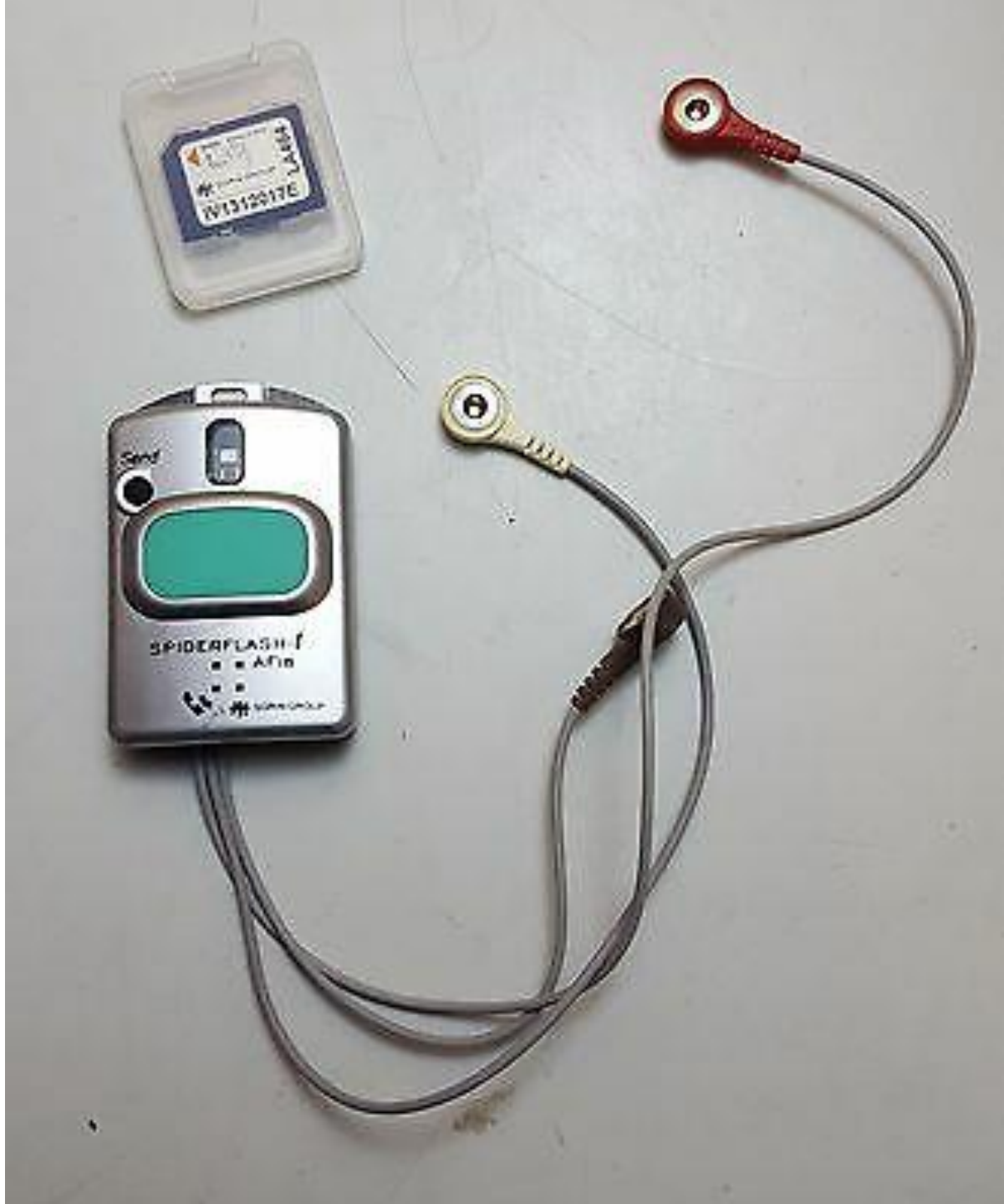
Risk Factor Reduction for Stroke (Primary and Secondary Prevention): "The Big 10"

- Control *chronic* HTN: bp at least < 140/90
- Antiplatelet therapy
- Antithrombotic therapy in atrial fibrillation
- Diabetic glycemic control: Hgb A1C < 7.0%
- Lipid lowering therapy: LDL < 70; HDL > 40

Sometimes paroxysmal afib is hard to find

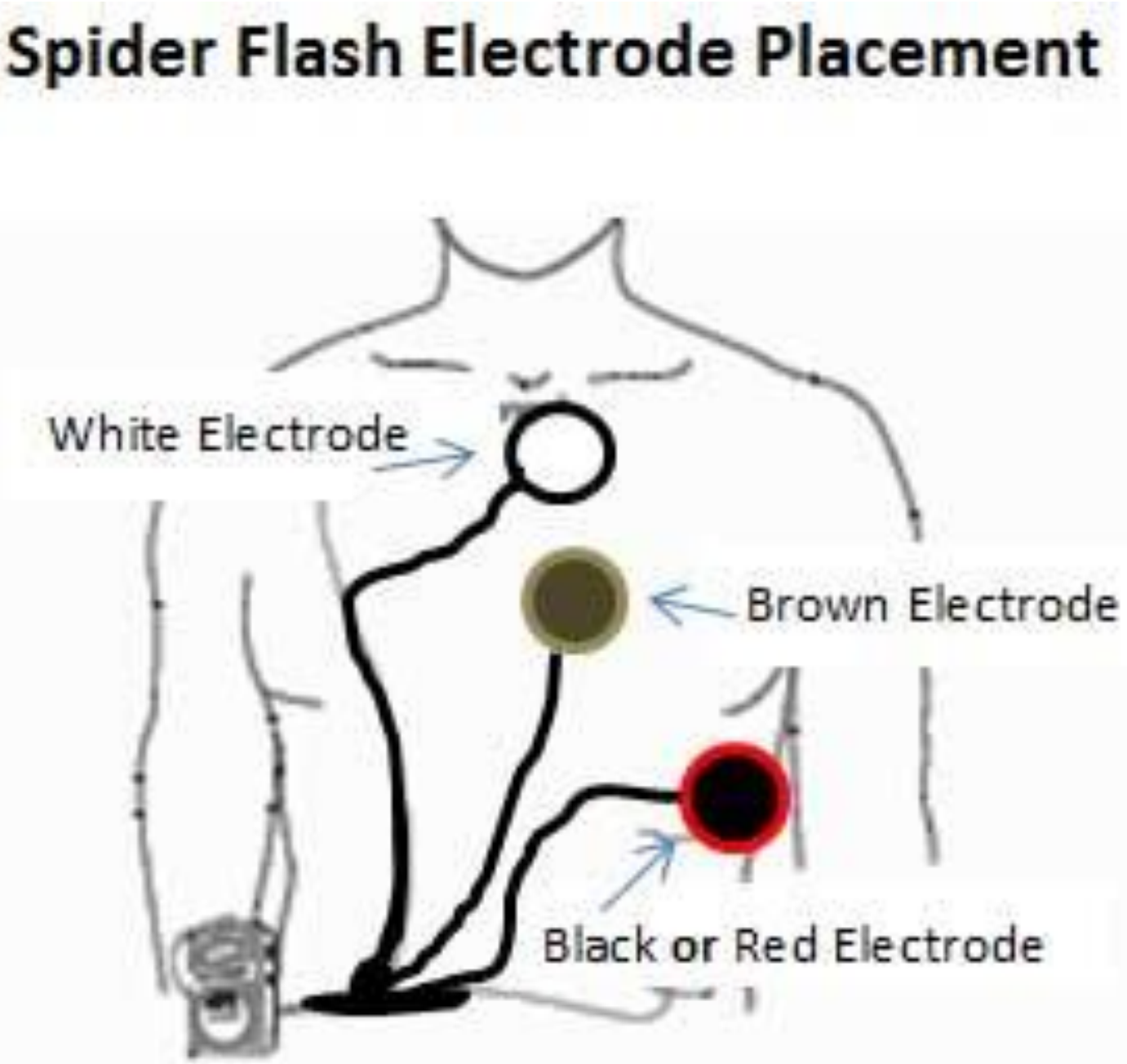
External Loop Recorder

Prolonged outpatient monitoring for atrial fibrillation



External Loop Recorder Placement

Prolonged outpatient monitoring for atrial fibrillation

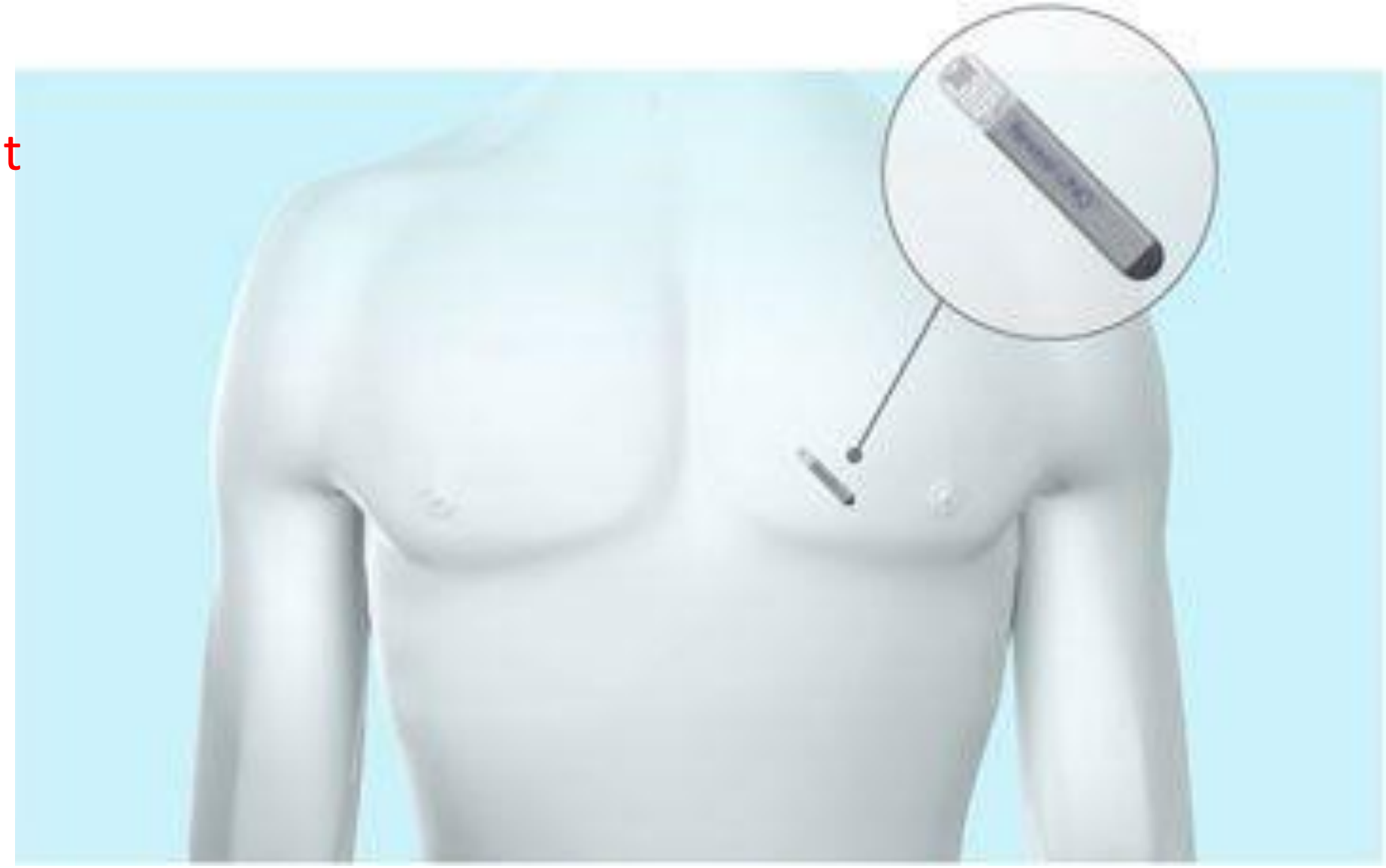


Implantable Loop Recorder/Cardiac Monitor

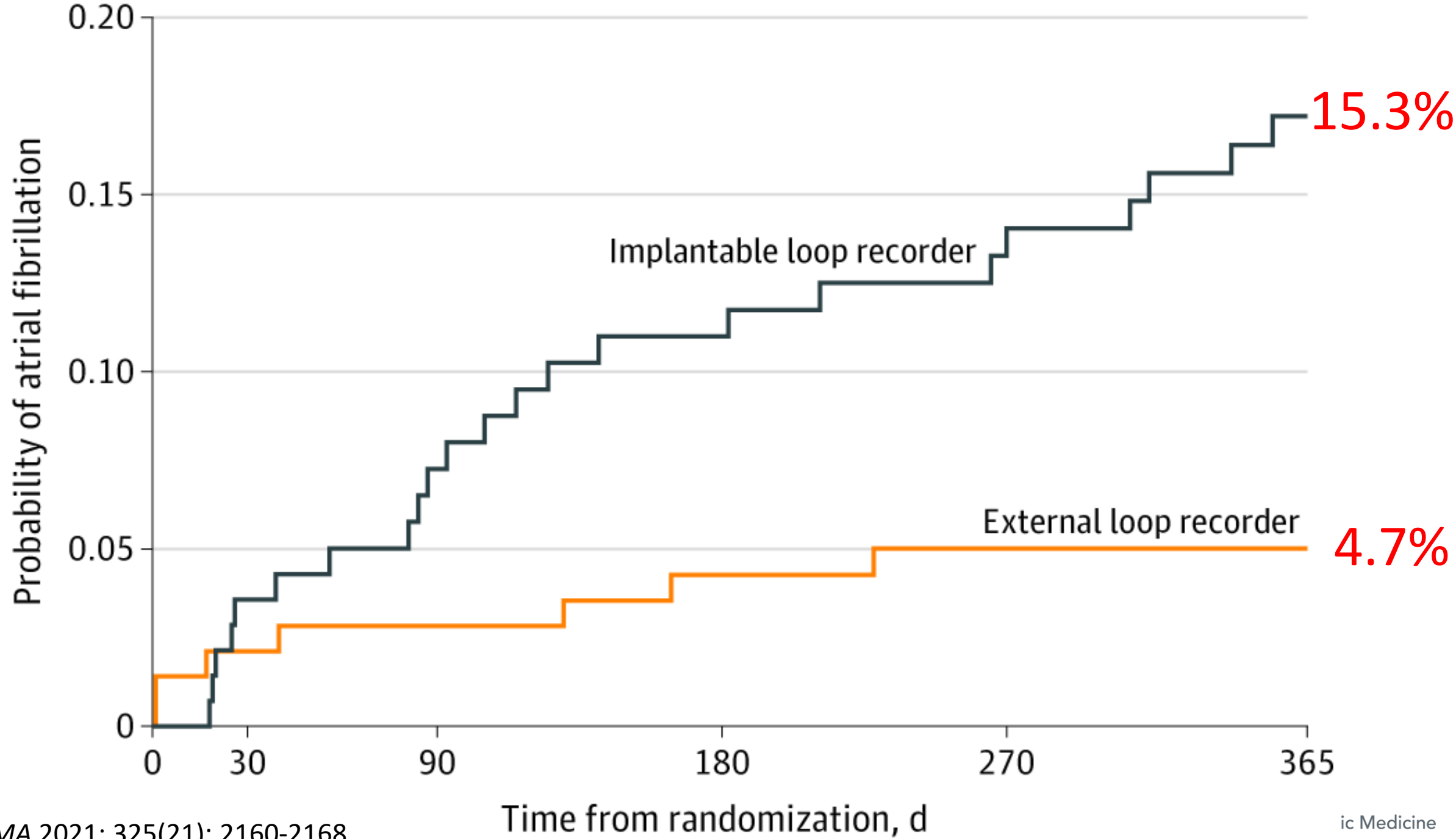
Prolonged outpatient monitoring for atrial fibrillation

mpo-mag.com

Prolonged outpatient monitoring for atrial fibrillation



Implantable Loop Recorder/Cardiac Monitor Placement



JAMA 2021; 325(21): 2160-2168.

ic Medicine

Risk Factor Reduction for Stroke (Primary and Secondary Prevention): “The Big 10”

- Control *chronic* HTN: bp at least < 140/90
- Antiplatelet therapy
- Antithrombotic therapy in atrial fibrillation
- Diabetic glycemic control: Hgb A1C < 7.0%
- Lipid lowering therapy: LDL < 70; HDL > 40

Risk Factor Reduction for Stroke (Primary and Secondary Prevention): “The Big 10”

- Consider CEA
- Smoking cessation
- Avoid excess alcohol
- Diet
- Exercise

Osteopathic Considerations in the Patient with Stroke

- Autonomic
- Biomechanical
- Lymphatic

Questions?

Thank you!