CT HEAD

TECHNIQUE: Axial non-contrast CT head at 5mm collimation from vertex through skull base without contrast.

FINDINGS:

For patient’s stated age, ventricles, cerebral sulci, and basal cisterns are normal in size.
No intracranial bleed or acute transcortical infarct.
No focal mass, mass-effect, or midline shift.
Evaluation of brainstem and posterior fossa is limited due to beam-hardening artifact.
Within the limits of this exam, orbits, paranasal sinuses, and mastoids are normal.
No depressed skull fracture or destructive calvarial lesions.

IMPRESSION: No acute bleed or transcortical infarct. No acute intracranial findings.

POINTERS

• Age-commensurate cerebral atrophy or volume loss (prominent cerebral sulci) and compensatory ventricular prominence or dilatation (ventriculomegaly)
• Old infarct with encephalomalacia (approaching CSF density), ex-vacuo ventricular dilation and ipsilateral wallerian degeneration (cerebral peduncle CST atrophy)
• Scattered/confluent hypodensities within periventricular, subcortical, and deep white matter related to SVIC (microvascular ischemic changes) or post-inflammmatory/infectious process
• Centrum semiovale (above vents) or Corona radiata (around vents)
• Cavum septum pellucidum (CSP)=anterior superior triangular with apex pointing post; Cavum vergae (CV)=posterior superior rectangular; CSP+CV (cavum septum pellucidum et vergae) can occur together; Cavum velum interpositum (CVI)=posterior inferior triangular with apex pointing ant
• Lacunar infarct within lentiform nucleus + caudate = basal ganglia (deep gray matter) or thalami
• Prominent perivascular space (Virchow-robin spaces)
• Age-indeterminant lacune
• Petechial gyriform hemorrhage (TPA ok) vs hemorrhagic stroke (transformation)
• Luxury perfusion (seen on contrast CT)
• Vasogenic or cytotoxic edema
• Intracranial atherosclerotic vascular calcs (carotid siphons and vertebrals)
• Mucoperiosteal thickening, mucus retention cyst vs polyp
• Intra-ocular lens implant (thin lens), scleral band, cataract extraction (post-op changes of globe), orbital prosthesis (fake eye), glaucoma drainage devices
• Hyperostosis frontalis
• Inner or outer table of calvarium (diploic space)
• Dural calcification (meningioma)
• Falx fenestrations with interdigitating gyri
• Parafalcine; paramidline; vertex; cerebral convexities; high frontal/parietal lobe etc
• Arachnoid granulations; venous lake
• Dental amalgam; crown; root canal (endodontic filling material); edentulous; braces

BLEED
There is curvilinear/lentiform extra-axial fluid collection (along inner table) extending from left/right [frontoparietal] to [parieto-occipital] region measuring [cm] in max thickness.

The fluid collection is predominantly hypo/isodense (likely subacute/chronic hemorrhage) with interspersed hyperattenuating acute hemorrhage. The fluid collection is predominantly hyperdense (acute hemorrhage) with swirling hypodense hyperacute blood. There is mixed attenuation within this fluid collection indicating acute and subacute blood products. [Heterogenous hyperdense layering material (fluid-fluid level) seen dependently likely represents acute on subacute/chronic hemorrhage]. [There is a second focal fluid collection...]


Herniation:

**STROKE**

[medium/large] area of parenchymal hypoattenuation (cytotoxic edema) with loss of gray-white matter differentiation within left/right [MCA/ACA/PCA] territory (> or <1/3 the territory) which involves [caudate head, lentiform nucleus]. There is [mm] leftward/rightward midline shift. There is gyriform petechial hemorrhage. [mild/mod/sig] mass effect on [lobe] with [partial/complete] effacement of [lateral ventricle] and resultant dilatation of [temporal horns]. No frank obstructive hydrocephalus (trapped ventricle) or significant herniation. MRA demonstrates [focal occlusion/thrombosis/cutoff sign/dissection/paucity of arterial branches/hyperemia suggesting subacute infarct/aneurysm/vasc malformation]

- Other etiologies (stroke mimickers)?
  - Aneurysm
  - Dissection
  - Sinus thrombosis (venous infarct)
  - AV malformation
  - Tumor (hypercellular tumor has restricted diffusion)
  - Abscess
  - Infx/Inflammation (encephalitis, MS)

**CTA:** occlusion/thrombosis (filling defect/cutoff), stenosis, dissection, aneurysm, vascular malformation, hypervascular tumor. Recent clot (meniscus/flattened) vs old clot (reverse meniscus) vs recanalized/non-occlusive (tram-track). Collateral retrograde flow (luxury perfusion) vs delayed antegrade flow (decreased parenchymal perfusion). >70% stenosis of ICA is significant (also comment on post-stenotic dilation).

**F/U**

Evolving infarct (brain parenchyma more hypoattenuating).

- Extension of previous infarct?
- Increasing edema?
- Increasing mass effect or shift or herniation?
- New infarct?
- Hemorrhagic transformation?
- Hydrocephalus?
Evolving blood products. No new acute hemorrhage.

**TUMOR**

[size]
[focal/multifocal/infiltrative/invasive]
[intra/extra-axial] look for CSF cleft on T2
[location: medial/lateral aspect of lobe; crosses midline; extends into]
[signal characteristics—esp T2 dark; necrosis; cyst with nodule; mixed solid/cystic; etc]
[enhancement pattern—usually not specific (patchy, serpiginous, nodular, rim/peripheral, heterogenous)]
[Hemorrhage on T1; hemosiderin staining vs blood products vs calcification seen as susceptibility artifact on GRE—CT best for calcifications]
[Mass effect/midline shift/hydrocephalus/trapped ventricle/effacement of cistern/displacement or distortion of adjacent structures/herniation]
[Vasogenic edema look at FLAIR to decide btwn edema vs infiltration]
[Neovascularity/flow voids]
[Perfusion imaging demonstrate increased CBV and CBF suggestive of high grade tumor]
[Low ADC values or restricted diffusion suggest high cellularity or high grade tumor]
[Destructive calvarial lesion]

**Tumor characteristics:**
- Low grade= low atten on NCCT; well circ; don’t enhance; no calc/necrosis
- Hi grade=hyperdense on NCCT; infiltrative and ill-defined margins; focal or patchy enhancement; may have calc/necrosis; highly vascular; +DWI suggests hypercellularity
- Necrosis (ring-enhancement on post-gad) suggest high-grade
- Infiltrative margins (look at T2 and FLAIR for irregular margins; FLAIR helps determine margins vs vasogenic edema)
- Cellularity (hypercellular tumor is DWI bright but ADC dark at tumor core which distinguishes it from shine-thru due to vasogenic edema)
- Attenuation on non-conCT (look for calcs; hypercellular tumors are hyperdense; hemorrhage)
- Bright DWI (with low ADC)=can be seen with hypercellularity (no restricted diffusion in necrotic or cystic portion of tumors)
- TTP map= not bright
- rCBV= bright if high grade (not bright if low grade)

WHO grade I= pilocytic astrocytoma
II=diffuse/fibrillary (frontal/temporal), subependymal GCA, pleomorphic xanthrocytoma brainstem glioma
III=anaplastic
IV=GBM

- **SIGNS OF EXTRA-AXIAL TUMORS:** CSF cleft (on T2); displaced subarachnoid vessels; cortical gray btwn mass and white matter; broad dural base; bony reaction
- **Tumors positive on DWI:** restricted diffusion suggests hypercellularity; epidermoid, abscess, cellular tumors (lymphoma/meningioma/PNET etc), mucinous mets, oligodendrogioma
- **High T1 tumors:** blood products (pit apoplexy, hemorr mets, thrombosed aneur); fat (dermoid cyst, lipoma); cholesterol (colloid cyst); melanin (melanoma mets); slow flow; paramag (cu, mn)
• Low T2 tumors (hypercellularity, calcium, blood, protein, melanin, flow-voids): lymphoma, medulloblastoma, pineoblastoma, PNET, mucinous mets, melanoma mets, colloid cyst, old hemorrhage in tumor or vascular malformation
• Tumors containing calcium: meningioma, oligodendroglioma, craniopharyngioma, choroid plexus tumor, ependymoma
• Dural based tumors: mets (breast/lung/prostate/neroblastoma), chondrosarcoma, hemangiopericytoma
• Dural tail: meningioma, mets, lymphoma
• Cortical-based or tumor involving gray matter (peripherally located; may present with seizure): DNET (esp if bubbly), Oligodendroglioma (calcification; usually not <30yo); Ganglioglioma/gangliocytoma (cystic with enhancing nodule); PXA; ddx= infarct, cerebritis, post-ictal; herpes encephalitis
• Tumors attached to septum pellucidum: central neurocytoma (enhances), ependymoma (does not enhance)
• Intraventricular tumors: ependymoma, subependymoma, CPP, central neurocytoma, colloid cyst, meningioma, GCA
• Tumors w/ CSF dissemination (tumoral nodules on surface of brain and spinal cord): PNET, medulloblastoma, pineoblastoma, ependymoma, GBM, lymphoma, choroid plexus papillomas
• Peds tumors: JPA, medulloblastoma, PXA, craniopharyngioma, choroid plexus papilloma, PNET
• Supratentorial tumors in kids: astrocytoma, PXA, PNET, DNET, ganglioglioma
• Infratentorial tumors in kids: JPA (1/3), medulloblastoma (1/3), brainstem glioma, ependymoma (least common)
• Cystic tumor w/ mural nodule: JPA, PXA (temporal lobe), ganglioglioma (temporal lobe), hemangioblastoma (post fossa), DNET
• Fat-containing lesions: lipomas, dermoid cyst, teratoma
• Calc-containing tumors: astrocytoma (20%), oligo (90%), mets; ependymoma (50%), CPP (25%), ganglioglioma (40%), meningioma, craniopharyngioma (90%), chordoma, chondrosarcoma
• Hemorrhagic tumor: mets (lung, RCC, melanoma, chroioCA, GBM, oligo
• Brain mets (can be solitary within hemisphere>cerebellum>BG or brain-stem): lung, breast, renal, GI, melanoma
• Skull base tumors: chordoma, chondrosarcoma, mets/myeloma, lymphoma, esthesioneuroblastoma, parangangioma, sinonasal CA
• Sellar/Parasellar tumor: pituitary adenoma, rathke’s cleft cyst, craniopharyngioma, meningioma, dermoid, epidermoid, germinoma, schwannoma, chiasmatic glioma, meningioma, mets
• CPA tumor: schwannoma (8th>5th), arachnoid cyst, meningioma, epidermoid, mets, parangangioma
• Pineal tumor: pineal cyst, pineocytoma, germ cell tumor (teratoma/germinoma), PNET, tectal glioma, meningioma, dermoid, arachnoid cyst
• Enhancing pineal mass: pineocytoma, pineoblastoma (kids), germinoma (engulfs calcs; females), teratoma, meningioma, VOG aneurysm

TUMOR MIMICS:
-Abscess (restrictive diffusion centrally; dark T2 and int high T1 rim which enhances; rim is smoother on outside than inside margin; rim is thicker laterally than medially)
-Encephalitis
-Tumerfactive demyelination (mixed T2 signal; incomplete ring enhancement; other WM lesions; low attenuation on non-con CT)
-Subacute infarct (do follow-up)
-Radiation necrosis (rim-enhancement; usually around 12wks post-XRT; do follow-up)

EXAMPLES:
GBM: butterfly (may cross CC); frontal>temporal>parietal; multifocal-multicentric; thick irreg enhancing rim with nodular inner wall and central necrotic core (increased rCBV at rim); moderate vasogenic edema; tendency for CSF seeding

Gliomatosis cerebri: diffuse infiltrative (involves ≥2 lobes), enlarges brain but preserves architecture; no enhancement

Lymphoma: hyperdense on NCCT; t2 dark; periventricular; solid enhancement (not rim); necrosis rare (unless treated)

Mets: multiple; at gray-white jct; solid or rim-enhancement

Oligodendrogioma: 40-50yo; cortical/subcortical (expands gyrus); most common frontal; 80% calcs; +/-enhancement

Ganglioglioma: kids/young adult; temporal lobe; mixed solid/cystic or cystic with mural nodule; 30% calcs; rim-enhancement with mural nodular enhancement

Central neurocytoma: 20-40yo; intra-ventricular; attached to septum pellucidum; bubbly lesion; variable enhancement

Meningioma locations: Parasagittal, sphenoid wing, tentorial, cerebral convexity, suprasellar/parasellar, cavernous sinus, clival, optic nerve sheath, paranasal/olfactory/planum sphenoidale, posterior fossa, cerebellar/infratentorial, CPA, optic sheath, foramen magnum, intraventricular

Meningioma: dural-based; low T2 (isointense to grey matter); restricted diffusion; intense enhancement; T2 CSF cleft; dural tail; hyperostosis

**Tumor Follow-up:**
Pseudoresponse (decreasing edema and enhancement but FLAIR signal still present): with steroids and Avastin

Pseudoprogression (worsening enhancement): with XRT and Temozolamide (for astrocytoma)

**Post-op**

Patient is s/p [mass resection]. Fluid and blood products are seen at the post-operative bed or resection cavity [without] any nodular enhancement or residual mass. Mild to moderate volume post-operative pneumocephalus is seen along convexities with blood products layering posteriorly. Interval decrease in resection cavity. Thin-rim of enhancement along periphery of resection cavity likely secondary to breakdown of BBB from post-surgical changes. Blood products within resection site preclude evaluation of residual underlying mass. [Persistent] mass effect on ventricle [with effacement/obscuration of basal cistern]. [CSF-shunt at right frontal horn of lateral ventricle]. [No] obstructive hydrocephalus (trapped ventricle with transependymal edema), midline shift, or herniation. Mild extra-axial [subdural and subarchanoid] hemorrhage and small-mod volume pneumocephalus deep to craniotomy site. Diffuse parenchymal enhancement related to post-op state. [Occipital] craniotomy [with flap fixation] and overlying scalp soft tissue swelling.

Impression: expected post-op findings s/p [surgery]

**Tips:**
Restrictive diffusion around the surgical bed can due to post-op injury (direct surgical trauma; retraction; vascular injury; devascularization of tumor)

**Infx [Intracranial]**

**Patterns:**

**Extra-axial**
1. Epidural empyema
   -usually paranasal sinus dz esp frontal sinus
   -lentiform
   -DWI and +rim enhancement
   -look for complications: dural sinus thrombosis including cavernous sinus thrombosis, meningitis, osteomyelitis
2. Subdural empyema
   -cresenteric
DWI and rim enhancement
-seen as sequela of meningitis in young kids
-DDX: subdural effusion (not DWI pos; no enhancement); chronic SDH (+susceptibility on GRE); subdural hygroma (hx of trauma)

3. Leptomeningeal enhancement (pia matter and arachnoid enhancement involving sulci and basal cisterns)
-classically seen in meningitis (ddx: carcinomatous meningitis and sarcoidosis)
-complications: hydrocephalus, ependymitis/ventriculitis, effusion/empyema, cerebritis/abcess, sinus thrombosis, infarct

Look for complications: hydrocephalus, subdural effusion/empyema; infarct, abscess; dural sinus thrombosis
-Viral meningitis and sarcoidosis may also give enhancement along CN at skull base
-More nodular/lumpy enhancement with fungal meningitis
-Carcinomatous meningitis from primary or secondary tumors (medulloblastoma, ependymoma, GBM, oligo, lymphoma, breast CA)

4. Pachymeningeal enhancement (dura mater and arachnoid enhancement)
-Post-op
-SIH
-dural mets (breast/prostate)
-TB/fungal and Sarcoid (also gives leptomeningeal enhancement along CN at skull base)

5. Iso-density/iso-intensity within basilar cisterns with enhancement
-TB/fungal
-Sarcoidosis
-Basilar meningitis (may have associated BG infarcts)

6. Bright signal within subarachnoid space around sulci on FLAIR
-Meningitis
-Subarachnoid hemorrhage
-Slow flow
-Carcinomatosis
-Oxygen therapy (diffuse)

Gyral enhancement
1. Vasodilation after reperfusion of infarct (luxury perfusion subacute phase)
2. Vasodilation phase of migraine
3. Vasodilation with seizure
4. PRES (occipital)
5. Meningitis (see above)
6. Encephalitis
-Parenchymal involvement (usually diffuse; related to viral infx)
-Signs/symptoms similar to meningitis plus added features (examples like seizure, delirium, altered consciousness, aphasia, hemiparesis, ataxia, weakness, eye symptoms etc)
-Etiologies: Herpes, Lymes, ADEM (s/p vaccination)
-Herpes encephalitis=medial temporal lobe→limbic (insula, cingulate gyrus of medial frontal lobe, and inferior frontal lobe); bilateral asymmetric; early patchy→late classic gyriform enhancement; +DWI; can be hemorrhagic; ddx=glioma and MCA infarct

7. Glioma
8. Cerebellitis (usually kids <6yo; bilateral symmetric; self-limiting; look for complications like obstructive hydrocephalus, tonsillar herniation; ddx=dysplastic cerebellar gangliocytoma or Lhermitte Duclos disease which is hyperintense on T2 with preserved striations)

**Periventricular enhancement**

1. **Ventriculitis/Ependymitis**
   - Etiologies: meningitis; shunting; ruptured abscess; CMV
   - Subependymal enhancement and intra-ventricular debris (which is +DWI)
   - Thin <2mm linear enhancement
   - Ventriculitis has high mortality

2. **Tumors (lymphoma, GBM)**
   - Primary CNS lymphoma has thick-rind of periventricular enhancement (“lamb wool”)
   - Ring enhancement is more commonly seen in AIDS or immunocompromised pts

**Ring enhancing**

1. **Mets**
   - Multiple
   - Within G-W jct

2. **Pyogenic abscess**
   - Early involvement=cerebritis (edema and patchy enhancement)
   - Thin regular rim of enhancement (inner margin may be smoother than slightly irregular outer margin during early stages)
   - Enhancing rim is thinner towards ventricle side and thicker towards gray-matter side
   - Light bulb bright on DWI centrally (dark ADC)—due to high viscosity pus
   - Intermediate low intensity T2 rim with bright centrally and surrounding vasogenic edema
   - T1 dark centrally
   - May have adjacent daughter cyst (multiloculated on post-Gad)
   - Look for complications: empyema; ventriculitis (subependymal enhancement and ventricular debris); dural vein thrombosis
   - Caseating Tuberculoma=hyperdense on non-con CT; T2 hypointense centrally
   - Fungal infx=neutropenic; little mass effect; some hemorrhage; may not enhance much based on immune status; may have associated septic infarcts

3a. **GBM**
   - Thick wall enhancement (shaggy inner margins unlike abscess)
   - Solid and necrotic components (may have internal hemorrhage)
   - Involves CC and deep gray
   - Neovascularity

3b. **Fluid-secreting cystic tumors**
   - Eg Pilocystic astrocytoma etc
   - Cystic component may have incomplete or minimal rim-enhancement b/c margins surrounding the fluid is composed of neoplastic tissue and also compressed normal parenchyma

4. **Hematoma**
5. XRT necrosis
- Thin feathery rim enhancement
- Nodular enhancement is suspicious for residual tumor

6. Tumeractive demyelination
- Incomplete or open ring-enhancement (acute phase)
- Little mass effect or vasogenic edema despite its size

7. Infarct
- Rim-enhancement may be seen esp with BG or cerebellar infarcts
- More gyriform enhancement than rim-enhancement

8. Atypical infections
- TB/fungal
  Caseating Tuberculoma=hyperdense on non-con CT; T2 hypointense centrally unlike pyogenic abscess
  Fungal infx=neutropenic with little mass effect; some hemorrhage; may not enhance much based on immune status; may have associated septic infarcts

- Cysticercosis
  3phases: vesicular (cyst with dot sign; dot or scolex may be T1bright but no enhancement); colloidal (rim-enhancing with turbid fluid hyperintense than CSF; with surrounding edema; scolex may be seen as eccentric dark focus on T2); granular (edema decreases; cyst retracts; decreasing enhancement); nodular (calcified; no edema or enhancement; signal drop out on T2 and T2*)

- Toxo vs Lymphoma (immunocompromised)
  Toxo=multiple; eccentric target sign is specific; usually empirically treat and repeat MRI in 7days to see any improvement
  Lymphoma=t2 dark; periventricular with subependymal enhancement; hyperdense on NCCT; +Thallium SPECT or FDG PET

**BG (deep gray)**

1. Infx
- Toxo (multiple; eccentric target sign is specific; usually empirically treat and repeat MRI in 7days to see improvement)
- Cryptococcus (bilateral BG and thalami; no enhancement; no DWI)
- CJD (+FLAIR/T2, +DWI but no enhancement; diffuse/extensive cortical ribboning and caudate/putamen involvement)

2. Tumor (esp if solitary lesion)
- Lymphoma (periventricular with subependymal enhancement; t2 dark; hyperdense on NCCT; ddx=toxo in immunocompromised)
  - Glioma like GBM

3. Ischemic (internal cerebral veins; lenticulostriate; HIE if diffuse bilateral along with cortical involvement)

4. Toxic/metabolic (CO, Leigh’s, Wilson’s, etc)

**White matter lesions**
1. SVIC
2. XRT/chemo
3. Transependymal CSF flow with hydrocephalus
4. MS/ADEM
5. IMMUNOCOMPROMISED
   - HIV encephalopathy (bilateral diffuse symmetric; no enhancement; +volume loss; CD4<100; dementia)
   - PML (focal neuro symptoms unlike HIV encephalopathy; multifocal subcortical lesions along U fiber; usually involves parieto-occipital; clue is dark on T1; also no enhancement)
   - CMV (thin periventricular and subependymal enhancement)

**CT TEMPORAL BONES (MASTOIDS)**

Checklist:

- External ear (pinna)
- External auditory canal (cerumen; ST or osseous hypertrophy; osteoma)
- Middle ear
  - TM (thickened/retracted)
  - Ossicular chain (Malleus/Incus/Stapes; oval window)
  - Scutum/Prussak’s space (coronal)
  - Attic (attic ad antrum=communication to mastoid) and Tegmen tympani (thin roof of middle ear cavity on coronal view)
  - Sinus tympani/pyramidal eminence/facial nerve recess (important to look here for any residual cholesteatoma)
  - Cochlear promontory (site of origin of glomus tympanicum)
  - Eustascian tube
- Inner ear
  - Stapes®Round window®vestibule
  - Semicircular canals (sup/post/lat or horiz—think “SLaP”)
  - Cochlea (2 ½ turns) and modiolus
  - IAC (look at size of geniculate ganglion)
  - Vestibular aqueduct (contains endolymphatic duct)—≤1.5mm
- Mastoid (anterior/lateral wall or external cortex; sigmoid plate; antrum=roof)
  - Bony CN7 canal (IAC/labyrinthine/geniculate ganglion/tympanic/mastoid/ stylomastoid foramen)—tympanic seg and labyrinthine seg seen as eyes of snail (cochlea) on coronal
- Vessels (aberrant ICA/high riding or dehiscent Jug bulb)
- Petrous apex
- TMJ
- Skull base (foramens including jug foramen and hypoglossal canal etc) and Nasopharynx
- Paranasal sinuses

Complete/partial soft tissue opacification of middle ear cavity without blunting or scutum or erosion/disruption of ossicular chain. There is soft tissue mass within prussak’s space in the lateral attic of middle ear cavity with associated blunting of scutum and erosion/disruption of ossicular chain. No lesion at cochlear promontory. No fluid level within middle ear.

Thickened/retracted/irregular tympanic membrane. External auditory canal is patent. No otitis externa.
There is soft tissue within middle ear attic, extending across into mastoid antrum with coalescent complete/partial opacification of mastoid cavity with loss of intervening air cells/septae. Undeveloped and opacified mastoid air cells. Associated chronic erosion of anterior/lateral mastoid wall aka external cortex. Sigmoid plate is intact. Inner ear or bony labyrinth (semicircular canals and cochlea) appears unremarkable.

Bony canal of CN 7 appears intact.

No sigmoid sinus thrombosis, epidural empyema (posterior fossa), meningitis, subperiosteal abscess, or osteomyelitis.

Paranasal sinuses are unopacified. No nasopharyngeal mass (fossa of rosenmuller or torus tubarius).

TMJs are normal.

No aberrant ICA or high riding/dehiscent jugular bulb.

**IMPRESSION:**
1. acute/chronic otitis media/otomastoiditis/coalescent mastoiditis.
2. acquired cholesteatoma.
3. otitis externa
4. paranasal sinus disease.
5. no nasopharyngeal mass.

**PULSATILE TINNITUS DDX**
1. Vascular tumor
   - Glomus (enhancing; salt and pepper)
     a) Glomus tympanicum (along promontory of middle ear)
     b) Glomus jugulare (look for erosion of lat and ant wall of jugular fossa on CT; may extend up into middle ear i.e. jugulotympanicum)
2. Vascular malformation
   - AVM
     - Dural AVM/AVF (Look for dilated transverse or sigmoid sinuses; unusually enlarged or numerous cortical veins; ECA supplies it; Venous drainage can be intracranial or extracranial or both)
3. Vascular abnl (congenital or acquired)
   - aberrant ICA (lateral course herniating into middle ear)
   - dehiscent ICA
   - hi-riding Jugular bulb (above level of floor of IAC)
   - dehiscent Jugular bulb (bulges into middle ear from below)
   - Jugular diverticulum (protrusion of jug bulb sup and medial to jugular fossa)
   - Persistent stapedial artery (originates from normal or aberrant ICA; courses middle ear; can mimic mass; look for absent ipsi foramen spinosum)
   - Aberrant sigmoid sinus (lateral course into mastoid air cells or medial course close to posterior semicircular canal)
   - Redundant arterial loops compression of coclear nerve near IAC or 8th CN at brainstem root entry zone)
   - Other rare causes: ICA dissection; Otosclerosis of inner ear; Idiopathic intracranial hypertension

**NON-PULSATILE TINNITUS DDX**
- CPA mass (acoustic neuroma; meningioma)
- TMJ issues
- Endolymphatic sac tumor
- Other rare causes: MS; chiari malformation

CT SINUS

-Frontal sinus absent at birth (fully dev by puberty); max sinus first to develop; ethmoid dev ant to post; sphenoid by 10yo

-Sinus CT evaluation CHECKLIST
  • Septum (coronal and axial)
    – Deviation (may be S-shaped)
    – Pneumatized
    – Septal spur which may approach/contact lateral wall → may need septoplasty
  • Middle turbinate (coronal)
    – Concha bullosa (which may narrow surrounding middle meatus)
    – Lateral attachment of basal lamella to lamina papyracea (imp for FESS)
  • OMC (osteomeatal complex)
    – Uncinate process (coronal)
      – Pneumatized
      – Medial vs lateral attachment (determines where frontal sinus drains)
      – Lateralized contacting orbital wall (results in hypoplastic maxillary sinus)
    – Ethmoidal bulla (normal) vs Haller cell (infraorbital)
    – Infra-orbital nerve dehiscence
  • Frontal recess (sagittal)
    – Prominent frontal beak
    – Prominent Ager nasi (anterior ethmoidal cell)
  • Lamina papyracea (axial)
    – Deformity/dehiscence (with prolapse of orbital fat into ethmoid sinus; EOM entrapment)
  • Olfactory recess (coronal)
    – Look for any abnormal ST on coronal which is btwn septum and vertical lamella of middle turbinate—differential diagnosis of ST in this region is tumor (esthesioneuroblastoma) and encephalocele/cephalocele
    – Look for asymmetric depth of cribiform plate and olfactory fossa (Keros classification 1-3mm vs 4-7mm vs 8-16mm)—imp for FESS
    – Fovea ethmoidalis is lateral to cribiform plate
  • Sphenoid anatomy (coronal)
    – Look for vertical sphenoid septum insertion on carotid canal
    – Pneumatized anterior clinoid or pterygoid recess
    – Dehiscent ICA and optic canal
    – Onodi cell (horizontal septa within sphenoid sinus on coronal view is a clue to this posterior ethmoid cell variant that is located superior and lateral to sphenoid sinus and can be close to optic nerve or ICA)—imp for FESS

-Sinus disease
  - mucoperiosteal thickening; partial/complete opacification
- mucus retention cyst vs polyp
- acute sinusitis (AFL)
- chronic rhinosinusitis (dense secretions)
- dense secretions (T2 dark to point where it may mimic air) ddx: dessicated secretions or inspissated mucus vs hemorrhage (rare) vs fungal dz (less common) vs calcifications (fungal calcifications are central and fine while non-fungal are egg-shell)
- sinolith or antrolith
- effect on bone: expansion/remodeling/reactive osteitis or sclerosis/thinning/erosion

- Sinonasal polyposis (polyps enhance w/ contrast while retention cyst or mucoceles dont)
- Mucocele (2/3 frontal and 1/3 ethmoid; variable MR signal; smooth osseous expansion; does not enhance)
  - supra-orbital mucocele: can be very large; pushes eye down
- Inverted papilloma: classic striated appearance (does enhance)
- Potts puffy tumor (frontal sinusitis, outer table OM, subgaleal abscess)

- AGGRESSIVE INFECTION
  - INVASIVE FUNGAL SINUSITIS (immunocompromised or poorly controlled diabetic)
    - look for unilateral nasal cavity ST (this is usually where it starts)
    - nasal septal ulceration
    - bone erosion
    - abnormal ST or stranding along peri-antral (premaxillary), medial canthus, nasolacrimal duct, PPF fossa, orbit or orbital apex
    - Cavernous sinus (thrombosis)—results in proptosis
    - Invasion of orbit or intracranial extension with complications
      - Epidural abscess
      - Subdural empyema
      - Cerebritis and abscess
      - CS thrombosis
      - Osteomyelitis (potts puffy tumor)
      - Pre/post-septal orbital cellulitis (including subperiosteal abscess)
      - Optic neuritis and myositis of EOM
  - BACTERIAL SINUSITIS
    - similar findings as above (but don’t start in nasal cavity unlike IFS)
    - complications are common

- SINONASAL TUMOR
  - b9 sinus disease may expand the sinus and thin the bone but does not erode or destroy the bone
  - b9 sinus disease does not have central enhancement or necrosis
  - tumor is intermediate on T2 (secretions are bright on T2; dessicated secretions are T2 dark)
  - tumor has heterogeneous/homogenous enhancement +/- central necrosis (while infx has peripheral enhancement)
  - tumor can involve orbit, ant/middle cranial fossa, central skull base, perineural extension

CT ORBIT

Terminology:
- intra/extra/per-orbital
- intra/extra-conal
-pre/post-septal
-Globe (anterior vs posterior chamber; lens)
-Intra-conal fat
-EOM (extra-ocular muscles)
-Optic nerve
-Ophthalmic vein
-Orbital rim or wall and orbital apex
-Lamina papyracea (medial wall)

Pathology

• Pre- vs Post-septal cellulitis
  – Pre-septal=periorbital cellulitis (PO abx)
  – Post-septal=orbital cellulitis (IV abx)
  – Complications
    • Subperiosteal abscess (along lamina papyracea)
    • SOV (sup orbital vein) thrombosis
    • Cavernous sinus thrombosis
    • Intracranial empyema
    • Meningitis
• Dacryocystitis (inflamed nasolacrimal duct)
• Pseudotumor (stranding of intraconal fat; respond to steroids; may involve lacrimal; isolated involvement of lateral rectus muscle; if extends into cav sinus, called tolosa hunt)
• Thyroid ophthalmopathy (spares muscle-tendinous insertion; lateral rectus involved the last; IMSOL)
• Coloboma (incomplete closure of choroidal fissure; microophthalmia; invagination into optic nerve; can be bilat)
• Hemangioma=most common B9 intraconal tumor(T2 bright, enhances, may have phleboliths)

Enlarged optic nerve ddx:
  – Optic neuritis (painful)=MS, Lyme, ischemia (adults)
  – Optic glioma=assoc with NF-1 in kids (may be bilat)
  – Pseudotumor=may also be painful
  – Meningioma=tram-track calcs
  – Dural ectasia=NF-1

Enlarged EOM ddx:
  – Graves ophthalmopathy (spares musculo-tendinous jct, IMSOL)
  – Pseudotumor
  – Myositis
  – Lymphoma/leukemia/sarcoid/mets
• Retinoblastoma (<5yo; calcified posterolateral globe; may be bilat/trilat/quadrilat)
• Retinopathy of Prematurity (premies w/ bilateral small globes, retinal detachment and dense vitreous due to neovascularity)—ddx is microphthalmia of Rubella
• PHPV (term babies w/ unilateral small globe, retinal detachment and dense vitreous due to persistent hyloid artery; lens also abnl)
• Coats (unilateral retinal detachment and dense vitreous due to retinal telangiectasias; no calc or mass; normal size globe)
• Toxocariasis (<12yo playing in soil w/ dog tapeworm; retinal detachment; may have calc; normal size globe)
• Drusen (macular degeneration w/calc at optic nerve and globe jct)
• Phthisis bulbi (scarred/calcified shrunken globe)
POST-OP CHANGES

- Scleral bands/buckles for retinal detachment
- Hydrojel scleral buckle hydration and expansion
- Cataract surgery w/ prosthetic lens implant
- Lensectomy (absent lens)
- Enucleation/Evisceration w/ globe prosthesis
- Orbital exenteration (for CA)
- Orbital tissue expanders
- Glaucoma valve/shunts
- Strabismus surgery (rectus muscle transposition)
- Dacryocystorhinostomy w/ stent (nasolacrimal duct stent)
- Medial canthus stabilization device
- Orbital wall reconstruction w/ plates/mesh
  - Infection; extrusion; subside; impinges/compresses EOM; obstructs lacrimal sac (dacryocystitis)

MR IAC

- size of CPA mass (cisternal vs intracanalicular component)
- extent into IAC (also look of lateral extent within IAC +/- extension upto or into cochlear aperture)
- cystic changes; heterogenous enhancement
- mass effect on CN V
- mass effect on 4th vent (+/- hydrocephalus)
- r/o mimickers of vestibular schwannomas
  - meningioma (dural tail; iso to cortex on T1 and T2; hyperostosis; calcifications; arise from dorsal aspect of petrous bone; may extend into IAC)
  - AICA aneurysm
  - Epidermoid (slightly hyperintense to CSF of FLAIR and restrictive diffusion on DWI)
  - Arachnoid cyst (iso to CSF on all seq)
  - Lipoma
  - Other schwannomas
    - CN VII lesion extends into labyrinthine segment towards geniculate ganglion → get CT to look for facial nerve canal enlargement
    - CN V lesion has more AP orientation and extends into meckels cave) or leptomeningeal processes (like TB/sarcoid/mets)
- post-op eval
  - subtemporal middle cranial fossa approach (for smaller lesion within IAC) vs retrosigmoid approach (for lesions with large CPA component) vs translabyrinthine approach (reserved for lesions extending far laterally within IAC—this approach results in permanent hearing loss)
  - residual enhancing mass
  - cerebellar contusion
  - AICA ischemia
  - Dural venous sinus thrombosis
- Note: linear dural enhancement is expected in IAC and along CPA margins (nodular enhancement is abnl)

MR TMJ
- **anatomy:** articular fossa; articular eminence; condylar head, biconcave meniscus (2mm ant band; thin int zone; 2-8mm thick post band), bilaminar zone (attaches post band to temporal bone)

- **closed mouth TMJ:** normally the thick posterior band of meniscus above condyle head

- **open mouth TMJ:** normally the condyle translates forward w/ thinner intermediate zone of meniscus now above condyle head; at fully open mouth, condylar head may even lie beneath anterior band of meniscus

- **Abnormal**
  - abnormal morphology: dessicated (int T1 signal); torn/perforated/attenuated (biconvex or rounded/thickened/thinned or flattened/folded); T2 hyperintensity at bilaminar zone
  - abnormal (anterior or anterolateral) displacement of meniscus (in front of condylar head) in closed position +/- reduces w/ open mouth
  - stuck adherent abnormal meniscus (does not change position)
  - limited translation of condylar head (w/ open mouth)
  - anterior dislocation of TMJ (condylar head ant and sup to articular eminence; cant close mouth)
  - OA (condylar head hypertrophy/flattening; osteophytes; subcondylar edema/cyst; effusion; erosion or pannus in RA)

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**MR SELLA**

Technique: cor T1 MEMP, cor T2, sag T1 FLAIR, and post-Gad sag/cor T1 FS. FOV=16mm and ST=3mm.

Findings: Pituitary enhances homogenously. Infundibulum is midline. Suprasellar cistern and optic chiasm are unremarkable. Cavernous sinuses enhances symmetrically. Cavernous carotid flow voids are intact.

- **Pituitary lesion**
  - normal pit upto 10mm in height; stalk/infundibulum extends from median eminence of hypothalamus to pit gland (2.8mm at midpoint and minimally widened superiorly measuring 3.5mm)
  - posterior pit is T1 bright
  - absent post pit is abnl only if it is ectopic (usually suprasellar; associated with absent infundibulum and hypoplastic ant pit) or assoc with endocrine abnl like diabetes insipidus
  - microadenoma: <1cm (benign, may be symptomatic like prolactinoma>150ng/ml); iso to hypo on T1; hypoenhancing (early phase) and delayed retension of contrast (after 1min);
  - macroadenoma: >1cm; iso to gray matter on all seq (unless hemorrhage=T1 bright/T2 dark or cystic degeneration=T2 bright); normal pit indistinguishable from macroadenoma; bilobed with waist if suprasellar extension; can encroach on optic chiasm; may extend into cavernous sinus (prolactin levels>1000) and incase cavernous ICA (but unlike meningioma does not narrow it) with min 30% encasement correlates with cavernous sinus invasion; can invade sphenoid sinus or dorsum sella; >4cm in diameter or <6mm from foramen monro are gaint macroadenoma; DWI+ (low ADC 0.66+/-0.1) macroadenomas are generally “soft” and can be easily resected with minimally invasive trans-sphenoidal technique while DWI- (high ADC value 1.36 +/-0.3) are “hard” and may require more extensive surgery
  - pars intermedia or colloid cyst: mimic microadenoma; subcm; non-enhancing btwn ant and post pit; often hypo T1 and hyper T2
  - (partial) empty sella: pit flat against sella floor (can be nl variant or assoc w/ pseudotumor cerebri where it resolves after surgery); pit enhances normally in patients with partial empty sella
  - rathke cleft cyst: midline sella or suprasellar cyst; non-enhancing; cyst with T1 bright and T2 dark mural nodule is classic (but imaging can be variable depending upon cyst contents)
  - pit tumor apoplexy/infarction: acute hemorrhage or infarction within pre-existing adenoma; sudden/acute expansion of adenoma w/ hemorrhage or fluid-fluid level
Sheehan syndrome: unlike pit tumor apoplexy, this occurs in setting of normal pit; post-partum infarction due to hypotension; may see peripheral but no central enhancement; hyperemia of surrounding sphenoid sinus mucosa; chronically get volume loss or atrophy of pit gland.

Suprasellar tumors

-Cystic lesions (none of these enhance): arachnoid cyst; epidermoid cyst; dermoid cyst (contain fat); rathke cleft cyst (see above)

-Tuber cinereum hamartoma: kids; sessile encase hypothalamus and deform 3rd vent (gelastic seizure) vs pedunculated attached to hypothalamus via stalk and extend inf into suprasellar region (precocious puberty); iso to gray matter; don’t enhance

-Lipoma: more homogenous T1 bright than dermoid

-Craniopharyngioma: bimodal; uni or multi-locular cystic lesion w/ enhancing cyst walls +/- solid enhancing component; T1 bright cyst proteinaceous contents can help with diagnosis; vessel encasement is common; calcifications; adult form called papillary-craniopharyngioma in adults more solid and rare calcs

-Meningioma: along greater wing of sphenoid vs planum sphenoidale (top of sphenoid sinus); iso to gray on all seq; enhance avidly; dural tail; may invade cavernous sinus and tend to narrow cavernous ICA; hyperostosis

-Optic chiasm glioma: assoc w/ NF-1

-Suprasellar stalk lesion

-Germinoma: adolescent males/females (males more common is pineal germinoma); homogenously enhancing mass centered on pit stalk; post pit often absent; similar in imaging to EG or lymphocytic hypophysitis; bright on DWI; may extend to involve hypothalamus

-EG (LCH): imaging features similar to germinoma (enhancing mass in region of pit stalk w/ absent post pit); may extend to involve hypothalamus

-Lymphocytic hypophysitis: women in late pregnancy or post-partum; inflammatory condition that mimics a mass; usually see thickened enhancing infundibulum; enhancing sella/suprasella mass which may encase cavernous sinus; hypo T2 rim surrounding gland and cavernous sinus can help with diagnosis; may have enhancing basisphenoid osteitis

-Sarcoidosis: can also involve the stalk

-Cavernous sinus lesion

-Schwannoma: trigeminal nerve schwannoma is most common; well-defined lobulated mass

-Meningioma: narrows cavernous ICA

-Tolosa-Hunt syndrome: intracanal pseudotumor extension into cavernous sinus via sup orbital fissure

-Cavernous sinus thrombophlebitis: secondary to facial/odontogenic/sinus infection; extension from valveless sup/inf ophthalmalic veins; filling defect w/in cavernous sinus (indirect sign are dilated/thrombosed sup ophthalmalic vein and dilated cavernous sinus

-CCF: post-traumatic or sec to ruptured ICA aneurysm; arterialized flow in sup ophthalmalic vein; engorged cavernous sinus

-Perineural spread of CA: along V2 (foramen rotundum) or V3 (foramen ovale) divisions

-Chordoma: aggressive tumor; 30-50yo

-Chondrosarcoma

What to report

-Mass

-Size in three dimensions

-Presence of necrotic / cystic areas
- size of diaphragmatic opening and size of suprasellar component
  - a narrow waist of the tumor where it passes through the diaphragma sella may limit the amount of tumor which can be removed via a transphenoidal approach and whether the tumor will come down at surgery with Valsalva or air into a lumbar drain
- presence of prolapse of the suprasellar membrane (arachnoid)
  - this occurs in front of the tumor (visible as a little cleft of CSF in front of the mass)
  - presence of this space increases the likelihood of an intra-operative CSF leak
- presence of invasion into the cavernous sinus / clivus / sphenoid sinus / orbit
- location of normal pituitary tissue and infundibulum in relation to the mass
- vessels
  - medially located or aberrant carotid arteries
  - aneurysms or other visible vascular anomalies especially in the cavernous sinuses
    - entering an artery transphenoidally will not bode well for the patient, the surgeon or the radiologist
- bone
  - size of the bony sella, expanded or not, is useful, gives you an idea of the size of the surgical corridor
  - degree of pneumatization of the sphenoid, location of the sphenoid septum, and any anomalous sinus anatomy
    - this is best assessed on CT but is visible also on post contrast coronal T1 images
    - the location of sphenoid septum helps guide transphenoidal approach and ensures that the pituitary fossa is entered rather than adjacent cavernous sinus or orbit
  - bony dehiscence over the carotid arteries in the sphenoid (better seen on CT)
  - presence of florid sinus disease, nasal polyps, septal spurs etc.

**MR BRACHIAL PLEXUS**

- C5/C6/C7/C8/T1 Root → Trunk (sup/middle/inf) → Division (ant=flexors/post=extensors) → Cords (lat/med/post) → peripheral nerves (musculocutaneous/axillary/radial/median/unlar)
- Axials = C5 to T1 Nerve Roots ventral rami (btwn ant and middle scalene muscles=interscalene triangle)
- Cor obliques = supraclavicular Trunks (supraclavicular → C5/C6=superior, C7=middle, C8/T1=inferior) and infraclavicular Cords (named according to their anatomic relationship to axillary artery aka “medial” post to aa; “posterior” sup to aa; “lateral” ant to aa)
- Sag obliques (obtained from spinal cord to medial border of humerus on symptomatic side only)
  - ventral rami of nerve roots are seen as dots sup and post to subclavian artery in interscalene triangle where ant scalene is ant and middle scalene is post (both muscles insert on 1st rib); for reference subclavian vein is btwn clavicle and ant scalene
  - 3 trunks form at the lateral border of middle scalene muscle (sup to subclavian artery)
  - 2 divisions form when brachial plexus crosses clavicle
  - 3 cords form lateral to 1st rib (behind pectoralis minor muscle, subclavian becomes axillary artery; position of cord relative to axillary artery determines its name)
    - better to assess change in caliber and attention to signal intensity

- Pathology: primary tumor (schwannoma/neurofibroma and neuroma along proximal stump of injured nerve), adjacent tumors (pancoast, lymphoma, other neck masses), trauma (nerve root avulsion +/-pseudomeningocele and hemorrhage; clavicular fracture with callus), others (cervical rib)
MR BRAIN

FINDINGS:
For patient’s stated age, ventricles, cerebral sulci, and basal cisterns are normal.
Mild/mod/sig cerebral volume loss or atrophy with prominence of cerebral sulci and ventricles (ventricular prominence or enlargement → ventriculomegaly).
[Confluent/scattered/patchy] [subcortical/periventricular/deep] white matter [and basal ganglia] T2/FLAIR hyperintense [non-enhancing] foci which are non-specific and may represent age-indeterminant microvascular ischemic (SVIC) or post-inflammatory/infectious changes (migraine).
No restricted diffusion to suggest acute infarct.
No extra-axial blood or fluid collection.
[No abnormal post-Gad parenchymal enhancement.]
No mass, mass effect or midline shift.
Major intracranial flow voids are patent.
[Satisfactory flow voids demonstrated within distal vertebral, basilar, and internal carotids arteries into COW and proximal cerebral arteries. Satisfactory flow voids also seen within dural venous sinuses.]
The orbits, paranasal sinuses and mastoid air-cells are normal.
No destructive calvarial lesion.

IMPRESSION:
1. No acute intracranial findings.

SEARCH PATTERN:
-Sag T1: assess BM and then all midline structures (as below)
-DWI (link with ADC map): r/o acute infarct (also useful for abscess or hypercellular tumor)
-FLAIR: r/o bleed; less sensitive than T2 for posterior fossa and brainstem lesions
-GRE: susceptibility artifact for old hemorrhage (hemosiderin staining; microbleed) and calcium
-T2: flow voids (arterial and venous sinuses); orbits; paranasal sinuses; temporal bones/mastoids; calvarium
-Axial T1: r/o bleed; dissection (FS helps); thrombus and slow flow are bright

Midline structures (sagittal): bone marrow, CC, Optic chiasm, Pituitary/Infundibulum, Mammillary bodies, Tectal plate, Pineal, Clivus, Posterior fossa (tonsils), Clivus, C-spine
Gray matter: Sulci/Gyri (volume loss), Ventricles/choroid, Pineal
Deep gray matter (basal ganglia): caudate, GP/putamen (lentiform nuclei), thalami
White matter: gliosis, lacunar infarct, perivasc spaces (vichow-robin)
Brainstem: Midbrain, Pons, Medulla, cervical-medullary jct, CPA
Cerebellum: Hemispheres, Vermis, Tonsils
Basal Cisterns (subarachnoid spaces): suprasellar[star], quadrigeminal[smiley], ambient, paramesencephalic
Vessels: Basilar/Vertebral, Carotids, COW, Sinuses
(sagittal/cavernous/sphenoparietal/straight/transverse/petrosal/sigmoid)
Others: Orbits, Paransal sinuses, Mastoids/Temporal, Middle ear
Dura/Calvarium(inner/outer table/diploic)/Marrow: skull base

MRA
-TOF=flow-related enhancement
-2D TOF for carotids (less spatial res but has great stationary tissue saturation therefore good vascular signal; very sensitive for slow flow so detects string-sign; dephasing artifact due to turbulent flow overestimates stenosis; in-plane saturation; can miss collateral flow due to sat bands; T1 bright thrombus can be overlooked)
- 3D TOF MOTSA for COW (higher spatial res; less dephasing artifact; best for measuring stenosis; poor sensitivity to detect slow flow—so hard to differentiate high grade stenosis from string sign; T1 bright thrombus can be overlooked)
- CE MRA (MRA better for string sign and ulcerated plaque; timing is important to avoid venous contamination) + 3D TOF (use TOF for measuring stenosis which does not overestimates like CE MRA)
- Phase-contrast (long acquisition; need to select correct VENC; not affected by T1 bright thrombus)
- T1 bright vascular lesion = slow flow vs thrombus

Stenosis (narrowing) vs Embolus (cutoff)
NASCET Stenosis = (1 – Dz/Nl) x 100%
<50% = mild
50-69% = mod
≥70% = sig
Near-occlusion with string-sign
Occluded

MRA head: normal flow-related enhancement within anterior/posterior circulation. Conventional anatomy. No hemodynamically significant stenosis or occlusion. No aneurysms.

MRA neck: Bilateral CC, IC, and EC arteries are patent. Cervical segment of bilateral vertebral arteries are normal. No hemodynamically significant stenosis or occlusion.

Aneurysm:
- 90% ant circ (ACOM and ICA/ACOM jct; PCOM; MCA or M1 seg near bifurcation); 10% post circ (Basilar tip; PICA; SCA)
- report location, size (3 measurements; <7mm unlikely to rupture), largest measurement of neck, shape and lobulation, orientation or direction it points

Dissection: starts ICA at skull base and extends down to level of C2.
FMD: involves mid ICA (not seen within prox ICA and does not extend to skull base); can image renal arteries to confirm

SLOW FLOW ARTIFACT:
- With slow flow, the intravascular signal often changes when different imaging planes have been used; thrombus will have the same intensity regardless of the plane.
- Flow enhancement usually fades as TE is increased; thrombus is often of intermediate or high signal on all sequences. (If gradient-moment nulling was used on the long TE image, however, this trick will not work.)
- Generally, acute or subacute thrombus will be bright on DWI and dark on T2*/SWI images.
- T1- or T2-weighted images using a different plane of section than in the original. Slow flow may change its appearance, but thrombus will not.
- Although gadolinium may diffuse into a clot, seeing a significantly brighter signal post-contrast supports the diagnosis of vascular patency.

MRV
Superficial and deep draining dural venous sinuses are patent. Congenitally hypoplastic left/right transverse sinus. Prominent arachnoid granulation or venous lakes seen.

**MR brain for MS**

-20-50yo (DDX=lyme dz, sarcoidosis, HTN, migraine)
-Ovoid T2 and FLAIR bright lesions in perivenular distribution along callososeptal interface (dawson fingers)—perpendicular to lateral ventricles
-Lesions (unlike SVIC) shows T2 shine-thru on DWI
-Supra/infra-tentorial (CC, corona radiata, centrum semiovale, subcortical u-fibers, internal capsule, visual pathways, ventrolateral pons, and rarely deep gray 5%)—30% have brainstem and cerebellar lesion more common in adolescent
-Focal or diffuse atrophy (thinning) of CC in 40% (longstanding dz)
-5-10% plaques can occur in gray matter (cortex and BG)
-Plaques can coalesce and periventricular WM dz has lump-bumpy outer margin and is asymmetric (helps distinguish from other WM disorders like SVIC)
-T2 Sag FLAIR (3mm slices w/o gap) and post-Gad T1 (5min delay)
-Acute/active plaque—tend to be >1cm with indistinct margins; enhances for <2mos (solid vs concentric/incomplete rim; rim-enhancing lesion usually suggests reactivation of an old lesion), usually T2 bright, 20% iso or 80% hypo on T1, may have restricted diffusion
-Acute/active (enhances; enhancement only lasts a few days or weeks and usually <2mos) → subacute (no enhancement; decreasing T2 size = decreasing edema; 40% become iso on T1 = repair or remyelination) → chronic (T2 bright = gliosis; 40% remain T1 dark or black hole = axonal loss or permanent demyelination which is rare in post fossa or cord; volume loss and CC atrophy)
-Tumefactive=larger, dark leading edge, +edema, mass effect
-Look for optic nerve enhancement on Cor T1 post Gad (optic neuritis)
-Look for cervical lesion 60% (peripheral, elongated, well-circ lesion)
-Peripheral located (does not involve entire cross-section)
-Usually spans <2 vert body segments in CC
-Cord atrophy or myelomalacia in 40%
-Mostly enhances
-Reporting and followup
-Location (periventricular/subcortical, supratentorial/infratentorial)
-# of new or enlarging T2 lesions (also report decreasing size or intensity and if the lesions are coalescing)
-# of enhancing lesions (solid vs concentric/incomplete rim enhancement)
-T2 lesion burden (mild=few lesions; mod=multiple lesions with early or near-confluence; severe=many confluent lesions)
-Absent/mild/mod/severe Chronic T1 black holes (atleast 6mos old as confirmed on serial MRI)
-Mild/mod/severe vol loss (also report if CC atrophy)

**WHITE MATTER DISEASE DDX:**
-Autoimmune (MS; ADEM=monophasic demyelination after recent viral URI with no significant enhancement and no new lesions after 6mos; vasculitis like sarcoid and SLE)
-Metabolic (central pontine myelinolysis, marchiafava-bignami, B-12 def=dorsal column of spinal cord)
-Infection viral/bacterial (PML=asymmetric subcortical parietoccipital and fatal; HIV; Lyme dz; SSP=subacute sclerosing panencephalitis)
- Vascular (PRES=post reversible leukoencephalopathy with symmetric reversible parietocippital WM involvement; biswangers=HTN with periventricular lacunar strokes; anoxic/hypoxic encephalopathy=T2 dark putamen/thalamus and diffuse involved white matter with T2 bright CC/ufibers/int-ext capsules and positive DWI; CADASL)
  - Toxin (XRT, drugs, etc)
  - Trauma (DAI)

**MR Brain for Seizure**

- medial temporal sclerosis (hippocampal volume loss and increased T2/FLAIR signal with asymmetric prominence of temporal horn)
- focal cortical dysplasia (focal cortical/subcortical hyperintensity; cortical thickening; blurred gray-white interface), heterotopia, schizencephaly, polymicrogyria, hemimegalencephaly
  - cavernoma
  - tumor (ganglioglioma, PXA, DNET, hypothalamic hamartoma)
  - TS, Sturge-Weber

**SPINAL CORD LESION**

- Cord signal ddx: edema; compressive myelopathy; myelomalacia; demyelination; tumor (astrocytoma vs ependymoma); infarct
  - Dictation:
    - Cord edema/signal spans x length (if >2 vert seg and not well-delineated on T2, consider transverse myelitis)
    - Cord expansion (concerning for mass)
    - Well-delineated vs ill-defined T2
    - Central vs Peripheral
    - Ventral vs Dorsal
    - Enhancement
  - DDX:
    - Syrinx or Syringomyelia (aka cord cyst; give gad to exclude assoc mass; no enhancement; sharp cord-syrinx interface; assoc with chiari I/II, tumor, trauma, CSF obstruction etc)
    - Acute Transverse Myelitis (long >3-4 vert seg; >2/3 crossectional area of cord; poorly-delineated or subtle on T2; variable enhancement like patchy/peripheral/diffuse; key is that it gets better on f/u)
    - MS or Neuromyelitis Optica (15-50yo females; <2 vert seg; <1/2 crossectional area of cord; peripheral involving dorsal or lateral columns; variable enhancement; consider correlation with MR brain; devs dz=cord+optic neuritis)
    - ADEM (similar to MS but in younger pts after viral infx or vaccination)
    - Spinal Cord Infarct (paralysis s/p surgery; +DWI; involves multiple vert seg; enhances in subacute phase only; “snake eyes” on T2)
    - AVM (dural fistula seen as flow voids post epidural space on sag with cord atrophy; cavernoma has blooming artifact on GRE and may have cord edema; do angiography)
    - Sarcoid (dense irregular posterior enhancement)
    - Tumor (cord expansion if intramedullary; well-delineated on T2; usually some enhancement; cyst or syringohydromyelia; may have hemorrhage; slowly progressive clinical course)
      - Intramedullary (expand the cord; usually central cord; may enhance and enhancement is always bad; usually not very long segment <2 vert seg; may have associated syrinx)
        - astrocytoma vs ependymoma (may be hard to tell btw the two; if located at conus or cauda equine=myxopapillary ependymoma)
        - hemangioblastoma (less common; 1/3 assoc with VHL; also image the brain)
        - mets (usually breast and lung CA)
Extradural
- Disk dz (disk extrusion or sequestered disk fragment) or synovial cyst
- Diskitis with epidural abscess
- Vertebral mets with retropulsion (if assoc retroperitoneal mass, consider neuroblastoma in kids and lymphoma in adults)

Extramedullary Intradural (eccentric; displaces cord; may extend along nerve root)
- meningioma (usually T-spine; may be calcified; homogenous enhancement; dural tail)
- nerve sheath tumor like schwannoma or NF (dumbbell shaped; enlarged neuroforamen; multiple in neurofibromatosis; calc is rare; may enhance heterogeneously)
- drop mets (usually breast/lung/lymphoma; also medulloblastoma/pinealblastoma/retinoblastoma/germ-cell tumors etc)
- arachnoid cyst (CSF signal on all seq; loculated; no enhacement; fills with contrast on delayed myelogram)
- lipoma (assoc with teethered cord)

CT C-SPINE

Findings: Vertebral height and alignment is normal. No acute fracture or spondylolisthesis. No prevertebral soft tissue swelling. Atlantodental distance is normal. Multilevel mild/mod degenerative changes with disc space narrowing, endplate changes, uncovertebral hypertrophy and facet arthropathy.

Cx-Cy: Mild disc space narrowing with endplate changes. Diffuse disc-osteophyte complex at X along with uncovertebral hypertrophy and facet arthropathy results in mild central canal and bilateral neuroforaminal stenosis.

POST-OP: Post-surgical fusion (ACDF or posterior spinal instrumented) hardware from x through y is intact. Metal streak artifact limits central canal evaluation. Pedicle screws are well seated. Interbody graft is appropriately incorporated with adequate/partial intervertebral osseous fusion. No perihardware lucency or complication. No surrounding drainable fluid collection. Central canal and neuroforamina are grossly intact.

MR C-SPINE

Findings: Normal cervical spine alignment. No abnormal marrow signal. Prevertebral soft tissue are unremarkable. Cervical cord is normal in morphology and signal intensity. Captured portions of posterior fossa are unremarkable.

C1-C2: Atlanto-occipital and atlanto-odontoid articulations are nl. Atlantoaxial DJD with thickened and post bulging PLL.
C2-C3: Normal intervertebral disk height. No disk bulge. Central canal and neuroforamina are patent.

-Alignment: spondylolysis
-Marrow signal:
-Disc height:
-Cord signal alteration (confirm on axial T2):
-Prevertebral ST:
-Paraspinal ST:
-Misc: Posterior fossa (tonsils), Pituitary, Thyroid, Airways, Lung apex
Disk space narrowing +/- endplate spondylosis (reactive endplate changes)
Endplate spondylosis
Uncovertebral hypertrophy/arthrosis
[severity] diffuse/central/paracentral disk-osteoophyte complex (discogenic ridging) or disk protrusion
Facet arthropathy
[abuts/effaces] ventral thecal sac
and results in [severity] CC stenosis (w/ residual AP diameter of x mm)
and [left/right/bilateral] [severity] NF narrowing
No cord compression or abnormal cord signal or enhancement
Effaces ventral thecal sac ➔ Abuts ventral cord ➔ Displaces cord ➔ Flattens ventral cord ➔ Compresses/Deforms cord

**Don't talk about nerve root impingement**

Cord signal= compressive myelopathy, edema, myelomalacia, demyelination, transverse myelitis, tumor (asytocytoma vs ependymoma), infarct

**MR L-SPINE**

Findings: Alignment, disc height, and marrow signal is maintained. Conus medullaris terminates at approp level. Thecal sac and contents are normal. No significant disc bulge/herniation or neuroformainal or central canal stenosis or compromise.

For purpose of this dictation, the inferior most level of fully formed intervertebral disk will be referred to as L5/S1.

- Transitional anatomy (look for ilio-lumbar lig on axial images to identify L5-S1)
- Alignment (listhesis)
- Pars defect
- Marrow signal (modic changes/hemangioma/schmorl’s node)
- Disc space narrowing
- Disc dessication
- Annular tear/fissure
- Conus medullaris
- Arachanoditis
- Misc: Aorta/iliacs, Kidneys, SI joints, Pelvis organs

Disc dessication.
[severity] disk space narrowing +/- endplate spondylosis
[Central/paracentral/lateral or foraminal/far-lateral or extraforaminal] diffuse disk bulge] vs [broad-based herniation/protusion/extrusion (neck) +/-migration/sequestered or free disk fragment located cranio/caudal ant epidural space]
Lig flavum hypertrophy
Facet arthropathy
[abuts/flattens, effaces/displaces ventral thecal sac]
[abuts/effaces lateral recess resulting in originating NR impingement/compression]
[results in neuroformainal narrowing with exiting NR impingement/compression; but NR exits freely]
[central canal stenosis]

**CT post TACE or RF/cryo-ablation**

Findings:

Liver: Shrunken liver w/ nodular contour consistent with chronic cirrhosis. Nodular contour of liver with [hypertrophy of caudate and left lobe] [prominence of periportal spaces].
Homogenously distributed/segmentally distributed/localized/scattered/faint accumulation/no significant accumulation] high-density iodized oil within treatment bed of segment [] s/p TACE. Non-target embolic material is seen within seg []

Tumor has been replaced by larger area of non-enhancing hypodensity with sharp-margin at interphase with normal hepatic parenchyma representing treatment response. Hyperattenuating homogenous (non-focal/nodular) halo surrounding the lesion likely represents granulation tissue after recent treatment. There is ill-defined [hyperemia/heterogeneity] [of surrounding hepatic parenchyma] [along the periphery of tumor bed] related to treatment. Non-enhancing hyperdense focus within the treatment bed on non-contrast imaging represent post-treatment localized hemorrhage. Minimal post-treatment air within the lesion represents necrosis. Overlying capsular retraction is noted.

No focal arterial enhancing focus is seen at the treatment bed. There is nodular arterial phase enhancement at the periphery of treatment bed which demonstrates [variable] washout on delayed/PV phase concerning for residual/recurrent disease. Separate from the treatment bed there is a new arterially enhancing focus which [becomes hyodense as contrast washes out on delayed] [becomes isodense to liver parenchyma on delayed] [remains hyperdense to liver parenchyma on delayed] may represent dysplastic nodule vs vascular malformation vs small HCC—short-term follow up is recommended if no treatment is planned for this small lesion. Wedge-shaped area of arterial enhancement which does not persist on portovenous phase consistent with THAD.

No evidence for treatment complication. No intra-hepatic ductal dilation. Portal vein is patent but dilated measuring []cm c/w portal hypertension. Attenuated [] portal/HV. Proximal left and right HA are patent. There are []varices and recanalized umbilical vein. [Moderate] ascites. Splenomegaly. Portahepatis and portocaval nodes are seen.

**Kidneys:** Mild non-nodular homogenous peripheral enhancing halo likely represents granulation tissue after recent ablation. Tumor has been replaced by larger area of non-enhancing hypodensity with sharp-margin at interphase with normal renal parenchyma representing treatment response. The margins of the treatment bed with normal renal parenchyma remain somewhat ill-defined however there is no enhancement to suggest residual/recurrent disease. There is surrounding perinephric stranding related to inflammatory response to treatment. There has been interval contraction of treatment bed related to post-treatment scarring/fibrosis. Treatment bed demonstrates central fat without solid-component and mild peripheral perinephric stranding. No evidence for treatment complication. No pseudoaneurysm, perinephric hemorrhage, or clot with the collecting system. Mild thickening of ant/post perirenal fascia and inflammatory changes within peri/pararenal space.

Impression: No evidence of recurrent/residual disease at treatment bed of seg [] after chemoembo/ablation. No new arterial-enhancing lesion within the liver.

**CT RUNOFF**

Technique: CT angiography of abdomen/pelvis with lower extremity runoff to bilateral feet with Xcc IV contrast. Contrast opacification of distal lower extremities is suboptimal/adequate/good/excellent.

Common femoral → prox/mid/distal superficial femoral and deep femoral (profunda) → hunter’s canal to popliteal → anterior tibial and tibioperoneal trunk → posterior tibial and peroneal → AT continues as dorsal pedis (dorsal) and PT continues as common plantar artery (medial)
-[Mild/mod/severe] focal/diffuse atherosclerotic disease with calcified and non-calcified plaques without significant stenosis
-[focal/multifocal/diffuse] eccentric/concentric/circumferential [calcified/non-calcified/mixed calcified and non-calcified] atherosclerotic plaque resulting in mild/mod/sig stenosis
-diffuse luminal irregularities
-[mild/mod/severe] focal/multifocal/diffuse stenosis/luminal narrowing
-[long/short] segment stenosis up to [<50/≥50]%
-[[]% high-grade stenosis due to [eccentric/circumferential] calc/non-calc/mixed plaque
-[Patent][High-grade stenosis][near-occlusion][occluded +/- distal reconstitution via collateral flow]
-3 vessel runoff
   -Poor distal opacification and runoff
   -Dominant runoff vessel is X
   -AT continues at DP
   -PT extends below ankle mortise to supple plantar arch
-[Diminutive][small caliber]
-Reconstitution of flow within [] likely from collateral flow
-Meniscus sign with abrupt cutoff suggestive of embolic disease

BYPASS
-Material (PTFE/Dacron synthetic graft vs Vein graft; Endovascular repair with stent graft; Bypass vs Extra-anatomic bypass; Limbs/conduits; Stent)
-Bypass type
   -Femoropopliteal (CFA vs SFA; below vs above knee)
   -Femorodistal (AT, PT, and less commonly Peroneal; Dorsal Pedis, Plantar arch)
   -Popliteal-distal
-Bypass evaluation
   -Bypass patent?
   -Prox/distal anastamosis stenosis?
   -Inflow/outflow vessels patent and adequate caliber?
   -Any graft infx (perigraft fluid, pseudoaneurysm, air collection, fistula)?
   -Caliber of bypass adequate? Any intra-graft stenosis or kinks?
   -Any non-ligated branches of venous graft? Any AV fistula?

VASCULAR TRAUMA
-Active arterial hemorrhage (irregular collection of contrast c/w extravasation)
-Intimal injury (focal eccentric contour defect; ddx: vasospasm)
-Dissection (intimomedial flap seen as curvilinear luminal filling defect)
-Occlusion (abrupt luminal termination; may have distal reconstitution via collaterals)
-Pseudoaneurysm (extraluminal outpouching)
-AV fistula (early venous filling c/w direct communication btwn artery and adj vein)
-Venous injury (seen on delayed imaging)
-Complication: hematoma, compartment syndrome, compression neuropathy, DVT, distal emboli

CXR DICTATION
• Technique
Portable
Upright, semi-upright, supine, decubitus, x-table lateral
Slightly/moderately rotated towards [left/right] (spinous process should be midway between clavicular heads)
Low lung volumes (should see 6 ant and 10 post ribs) accentuate cardiome diastinal silhouette and vascular markings/crowding

Lines and tubes
ETT tip at X cm (should be 2-5 cm) above the carina (should be below inferior aspects of clavicles aka thoracic inlet)
Tracheostomy tube tip at or just below thoracic inlet
Enteric tube (NGT/OGT) courses beyond the expected region of GEJ with tip not visualized on this exam (tip and side port should be in stomach); looped/coiled within gastric fundus with tip directed towards GEJ; tip appears to be post-pylorus
Feeding tube (DHT) tip at/near Ligament of Treitz
Chest tube/thoracostomy tube/pleural drain is directed superiorly/apically and medially with tip near the apex or directed inferiorly and medially with tip near the cardiophrenic angle; side port projects outside thoracic cavity with subq emphysema; stable effusion/pneumothorax — correlate with drain output or tube patency
Mediastinal drain and retained epicardial leads are noted
Subclavian/IJ CVC tip at or near superior cavo-atrial jct (CVC sheath in place)
Tunneled dialysis (hickman, Broviac) dual/triple-lumen catheter
Implanted chest port (port-a-cath) with a Huber access needle at the port site
Stable dual/triple-lead cardiac device (ICD or pacer)
IJ/subclav/femoral Swan Ganz catheter tip approx [ ] cm from left/right hilum (correlate with expected pressure measurements)
IABP tip located [1-2 cm] below aortic arch with balloon inflated during diastole
Correlate for desired location

Interval change
Accounting for differences in technique and patient positioning...
Overall no significant change compared to most recent prior exam from [date][time]
Interval improved lung volumes and aeration within upper lungs
Decreased pulm vascular congestion and effusion suggesting response to diuresis
Resolved bibasilar opacities suggesting atelec or edema

Lung volumes
Low lung volumes (should see 6 ant and 10 post ribs) accentuate cardiome diastinal silhouette and vascular markings/crowding
Low lung volumes likely related to poor inspiratory effort or expiratory phase
Stable underlying emphysematous lung changes; symmetrically increased lung volumes (hyperinflation) suggestive of obstructive lung physiology like emphysema or asthma

Parenchyma
Biapical pleural thickening/scarring vs pleuroparenchymal scarring
Chronically elevated hemidia; eventration
[Diffuse/patchy/multifocal/confluent] [peripheral/perihilar/central/bibasilar] consolidations/opacities [obscuring heart border or hemidiaphragm]
Asymmetric left basilar airspace disease
Bibasilar airspace opacities (ddx: PNA vs atelec vs aspiration)
Partial hemidia obscuration may be due to atelec vs infiltrate
Reticulonodular interstitial changes
Peripheral/basilar reticular changes related to emphysema
Nipple shadow simulates nodule within bilateral lower lungs; possible nodule vs overlapping shadows
Mild diffuse airways thickening “cheerios” and dirty lung (ddx: bronchitis vs RAD vs sequela of smoking)
Interface seen along lateral chest most likely represent skin fold
Small apical PTX; large tension PTX with mediastinal shift
Vessel-on-end vs pulmonary nodule
Calcified pulm granuloma
Calcified pleural plaques

- Vasculature
  - Cephalization or pulm vasc prominence (volume overload or perioperative hydration/fluid resuscitation)
  - Indistinct pulm vasculature \(\rightarrow\) pulm vascular congestion
  - Vascular pedicle enlargement
  - Increased interstitial marking (kerley B lines) suggesting interstitial edema
  - Airspace opacities suggesting alveolar edema

- Effusion
  - Blunting of CPA may represent small effusion vs atelec/scarring
  - Obscuration of hemidia with hazy (veil-like) opacification of lower lung likely represents layering pleural effusion with associated compressive atelec (infiltrate not excluded)
  - Fluid within fissure
  - Convex margins suggest possibility of loculated pleural effusion

- Heart
  - Cardiomegaly
  - Stable enlarged cardiomedastinal silhouette
  - Tortuous asc/desc aorta

- Misc
  - Penia (osseous demineralization)
  - Old compression fx
  - Mild/moderate degen (spondolotic) changes
  - Multilevel flowing vertebral osteophytes (DISH)
  - Old healed rib fx deformities
  - Post-surgical partial resection of posterior rib

- Recommendations
  - Attention to this region on follow up exam
  - Follow up CXR in 4-6wks post tx to ensure resolution
  - Consider CT chest for further assessment/confirmation

**CT CHEST**

-PE: Contrast opacification of pulmonary arteries is poor/adequate/good/excellent. Subsegmental pulmonary arteries are not well opacified and therefore not well evaluated. Otherwise, no filling defect within central pulmonary arteries to suggest acute PE. Motion artifact at lung bases. There is extensive near-occlusive thrombus within main, lobar and proximal segmental arteries bilaterally with no focal pulmonary infarct. Triangular juxtrapleural opacities are seen within [] most likely representing pulmonary infarction. There is flattening of interventricular septum along with reflux of
contrast into hepatic veins suggestive of right heart strain. Main pulmonary artery is enlarged (measuring over 3cm) suggestive of pulmonary arterial hypertension.

- **Nodule**: []mm [pleural-based/juxtapleural/subpleural/peripheral/central/perihilar/periﬁssural] [solid/part-solid/GG] nodule within [location] with [smooth/lobulated/speculated] margins (see image# or series) with pleural tag and satellite nodules. [No benign calcification like central, popcorn, or ring/concentric.] Scattered calcified lung granulomas along with mediastinal/hilar calcified nodes consistent with sequela of old-healed granulomatous disease. Low dose and non-contrast technique limits mediastinal/hilar evaluation.


- **CHF**: Pulmonary vascular prominence, cephalization, and vascular congestion. Bilateral pleural effusions along with groundglass opacities and septal thickening. In setting of cardiomegaly, ﬁndings are suggestive of pulmonary edema. Cardiomegaly with biatrial enlargement. Reﬂux of contrast into hepatic veins is suggestive of right heart strain.


- **ILD**: upper/lower lobe predominant; central/peripheral/along bronchovascular bundles; interlobular septal thickening; intralobular reticulations (ﬁbrosis); groundglass opacities; mosaic attenuation (with/without air-trapping); micronodules (random/centrilobular/perilymphatic); traction bronchiectasis and architectural distorion; tree-in-bud opacities; airways thickening; honeycombing/ﬁbrosis; centrilobular/paraseptal emphysema (with/without bullous changes); dendritic calcifications; parenchymal bands; pleuroparenchymal scarring with cystic changes, dystrophic calcifications, and hilar retraction

**CT ENTEROGRAPHY**

- No intestinal mucosal hyperenhancement or signiﬁcant wall thickening
- Terminal ileum is unremarkable
- No ﬁbrofatty proliferation (creeping fat) or mesenteric fat stranding
- No distension/hyperemia of vasa recta (comb sign)
- No reactive mesenteric adenopathy
- No intestinal stricture, phelmon/abscess formation, or obvious fistula
- Anorectum is unremarkable
- No gallstone, renal calculi, biliary dilatation, or sacroiliitis
- No extravasation of IV contrast within bowel lumen to suggest active GI bleed
Figure 3-10. Arterial vascular territories, in the axial (A-E) and coronal (F-J) planes. ACA = anterior cerebral artery; ACh = anterior choroidal artery; AICA = anteroinferior cerebellar artery; BA = perforating branches of the basilar artery; H = recurrent artery of Heubner; LSA = lenticulostriate artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; PICA = posteroinferior cerebellar artery; SCA = superior cerebellar artery; WSCA = watershed region supplied predominantly by the SCA.
Figure 7  The venous territories of the brain as described by Rhoton.25 Red indicates the internal cerebral veins; green, the superior sagittal sinus; blue, the sylvian vein; yellow, the vein of Labbe; pink, the galenic system; light blue, the petrosal veins; and orange, tentorial group. (Color version of figure is available online.)
Axial NCCT images showing the MCA territory regions as defined by ASPECTS. C- Caudate, I- Insular ribbon, IC- Internal Capsule, L- Lentiform nucleus, M1- Anterior MCA cortex, M2- MCA cortex lateral to the insular ribbon, M3- Posterior MCA cortex, M4, M5, M6 are the anterior, lateral and posterior MCA territories immediately superior to M1, M2 and M3, rostral to basal ganglia. Subcortical structures are allotted 3 points (C, L, and IC). MCA cortex is allotted 7 points (insula cortex, M1, M2, M3 M4, M5 and M6).
What is ASPECTS

- Alberta Stroke Program Early CT score (ASPECTS) is a 10-point quantitative topographic CT scan score
- ASPECTS was developed to offer the reliability and utility of a standard CT examination with a reproducible grading system to assess early ischemic changes on pretreatment CT studies in patients with acute ischemic stroke of the anterior circulation

How to compute ASPECTS

- ASPECTS is determined from evaluation of two standardized regions of the MCA territory: the basal ganglia level, where the thalamus, basal ganglia, and caudate are visible, and the supraganglionic level, which includes the corona radiata and centrum semiovale
- All cuts with basal ganglionic or supraganglionic structures visible are required to determine if an area is involved. The abnormality should be visible on at least two consecutive cuts to ensure that it is truly abnormal rather than a volume averaging effect
- To compute the ASPECTS, 1 point is subtracted from 10 for any evidence of early ischemic change for each of the defined regions.
- A normal CT scan receives ASPECTS of 10 points.
- A score of 0 indicates diffuse involvement throughout the MCA territory

- Scores of 7 or less, indicating more extensive cerebral hypoattenuation in the MCA territory, are correlated with both poor functional outcome and symptomatic intracerebral hemorrhage example
- ASPECTS applied to NCCT scans from various clinical trials suggest that higher ASPECTS value (ASPECT 8-10) were associated with a greater extent of benefit from i.v. thrombolysis example
<table>
<thead>
<tr>
<th>Segment</th>
<th>Course</th>
</tr>
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<tbody>
<tr>
<td>C1 (cervical)</td>
<td>Extends from the origin of the internal carotid artery to its entry into the skull base</td>
</tr>
<tr>
<td>C2 (petrous)</td>
<td>Portion of the artery within the carotid canal of the petrous temporal bone initially, ascends vertically within the canal (vertical portion) and then turns anteriorly, medially, and superiorly within the canal (g扣s) and continues horizontally (horizontal portion) toward the petrous apex, where it exits the temporal bone</td>
</tr>
<tr>
<td>C3 (iacemum)</td>
<td>Begins where the internal carotid artery exits from the carotid canal and extends up to the level of the petroclinoid ligament</td>
</tr>
<tr>
<td>C4 (cavernous)</td>
<td>Begins at the superior aspect of the petroclinoid ligament and includes the portion of the internal carotid artery that courses through the cavernous sinus</td>
</tr>
<tr>
<td>C5 (clainoid)</td>
<td>Short segment: courses through the dural reflections related to the anterior clinoid process</td>
</tr>
<tr>
<td>C6 (ophthalmic)</td>
<td>Begins at the distal dural reflection around the anterior clinoid process and extends to the level of the posterior communicating artery</td>
</tr>
<tr>
<td>C7 (communicating)</td>
<td>Begins just proximal to the origin of the posterior communicating artery and terminates where the internal carotid artery bifurcates into the anterior and middle cerebral arteries</td>
</tr>
</tbody>
</table>
Figure 5 Schematic illustration showing persistent carotid-basilar anastomoses at various levels. a, artery; PcomA, posterior communicating artery. (©2010 Lydia Gregg.) (Color version of figure is available online.)