PI Session: 2022.01.05_D and T_Radiology: Imaging of Neurology 1_Brain Imaging_ Preparatory_Marcus John Julius, M.D.

Objectives: At the end of this session, students will be able to:

1. Explain the simplified overview of MR imaging (using terms related to 'low-energy' and 'high-energy' protons)
2. Compare and contrast the concepts of T1 relaxation and T2 relaxation
3. Describe the imaging findings associated with normal aging of the brain
4. Formulate an imaging approach to the use of CT imaging in the workup of suspected acute cerebral ischemia
5. Contrast the CT-perfusion findings of 'infarct core' and 'ischemic penumbra'
6. Describe the MR imaging findings associated with cerebral ischemia/infarction
7. Differentiate acute, subacute, and chronic intracranial hemorrhage based on CT imaging appearance
8. Differentiate the various etiologies of extra-axial and intra-axial hemorrhage based on their CT imaging features
9. Contrast imaging characteristics of intra-axial and extra-axial neoplasia
10. Demonstrate a familiarity with the imaging features essential to the description of cerebral neoplasia

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Magnetic Resonance Imaging

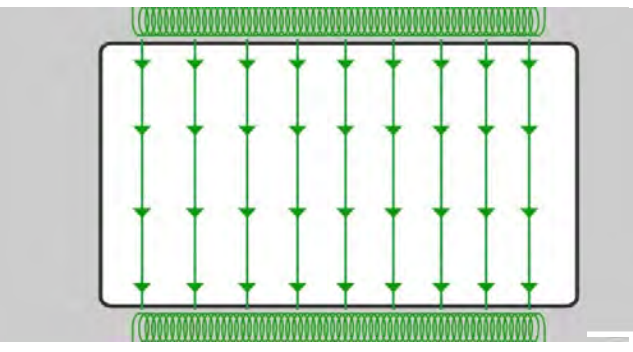
-**PHYSICS**:

-Protons and neutrons each have a magnetic field called a *magnetic dipole*.
- -When neutrons and protons are equal in number within a nucleus, their magnetic moments cancel out
- -Nuclei with an *uneven* number of protons and neutrons have a *net* magnetic moment, and act like tiny bar magnets
- **-Hydrogen nucleus** (1 proton; no neutron), therefore, has a large magnetic moment.

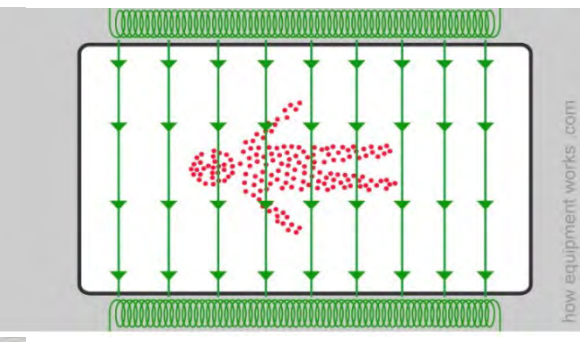
-Hydrogen is abundant in the human body and is optimal for MR imaging
- -Outside a magnetic field, nuclear spins of hydrogen protons are random (with no net magnetization vector)
- -Within an external magnetic field, nuclear spins of hydrogen protons align either *with* or *against* the external field
  - -A *slight excess* (3/1,000,000) of protons align *with* the magnetic field. (It is this vector that is used in MRI imaging to generate a signal)

-Simplified overview:

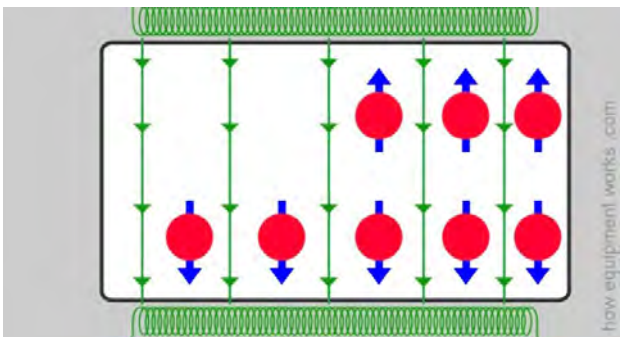
-Subject an individual to a magnetic field
-Align the *net* magnetization vector of the small excess of protons *with* the external field
-Introduce a radiofrequency pulse (RF)
-Perturbing the magnetization vector from its equilibrium position
-Observe the signal generated as the magnetization vector returns to its equilibrium position
- -Different tissues (i.e. proteins, water, fat, etc.) generate differing signals. Once analyzed and localized, these signals assist in creating an image



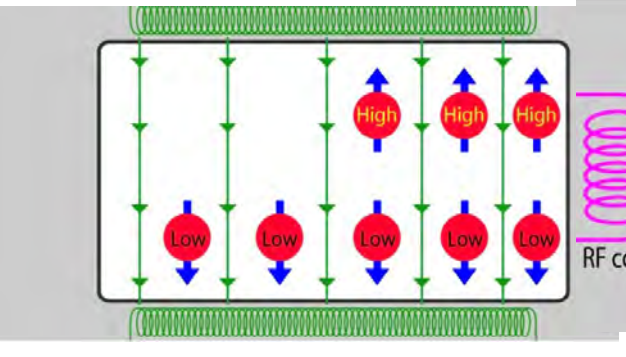
Magnetic field



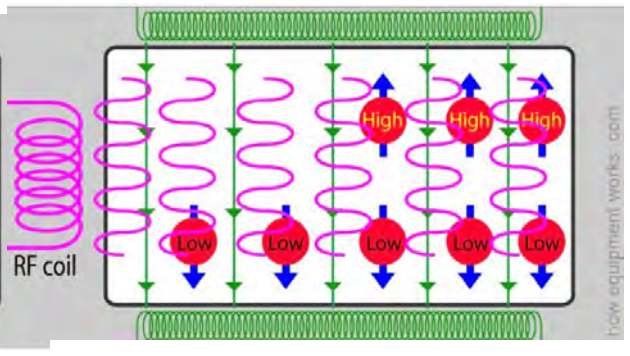
Our patient in magnetic field



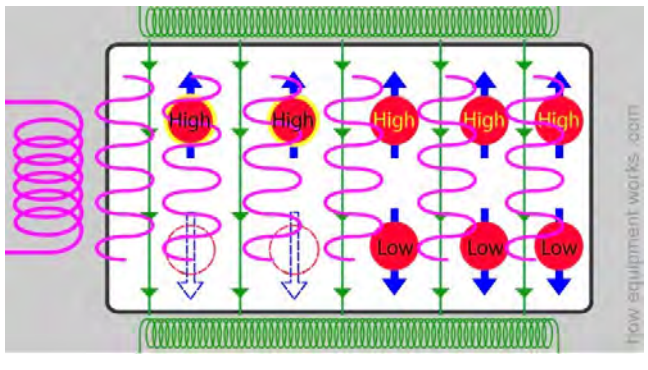
A sample of our patient's protons



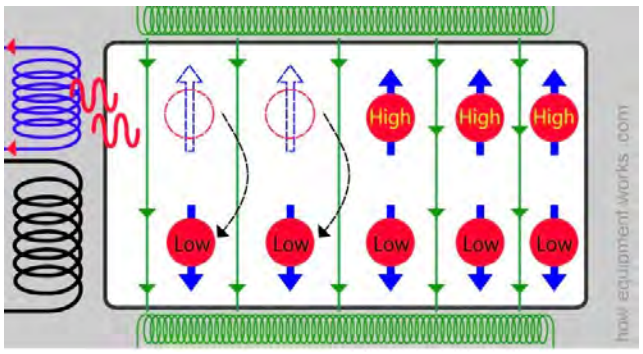
Protons aligned with the external field (Low) and against the external field (High)



RF coil emits energy absorbed by Low-energy protons



Some of the Low-energy protons assume High-energy state



The recently 'energized' high-energy protons prefer to return to their lower energy state. In doing so ('relaxing'), they release energy. This energy, received by 'receiver coils' (in blue), helps to create an MRI image

-Magnetic nuclei may also be called *magnetic dipoles, spins, or magnetic moments*

-Commonly, texts will illustrate the magnetic nuclei (i.e. protons) as a vector (i.e. magnitude and direction).

-When aligned with the external magnetic field (parallel), the magnetization vector is labelled M_Z

-When the vector is displaced into the orthogonal (XY) plane, it is called $M_{XY.}$

-A certain time after M_Z is displaced, it always eventually returns to the stable (parallel) orientation (through ***T1, 'spin lattice', or longitudinal relaxation)***

-T1 relaxation times are tissue specific (but also vary with strength of external magnetic field)

-The magnetization vector, M_Z, is displaced from its alignment with the external field by the application of radiofrequency (RF)

-Applied radiofrequency (RF) causes the M_Z to rotate through an angle ('flip angle'), proportional to the RF intensity

-A *displaced* vector is often best represented by two vectors (one *perpendicular* to the external field and one *parallel* to the external field)

-The parallel vector is called **longitudinal magnetization (M_Z)**

-The perpendicular vector is called **transverse magnetization (M_{XY})**

-For illustration, let's assume a 90-degree RF pulse which displaces ***all of the net magnetization into the transverse plane (M_{XY}).*** Therefore, ***M_Z is zero.***

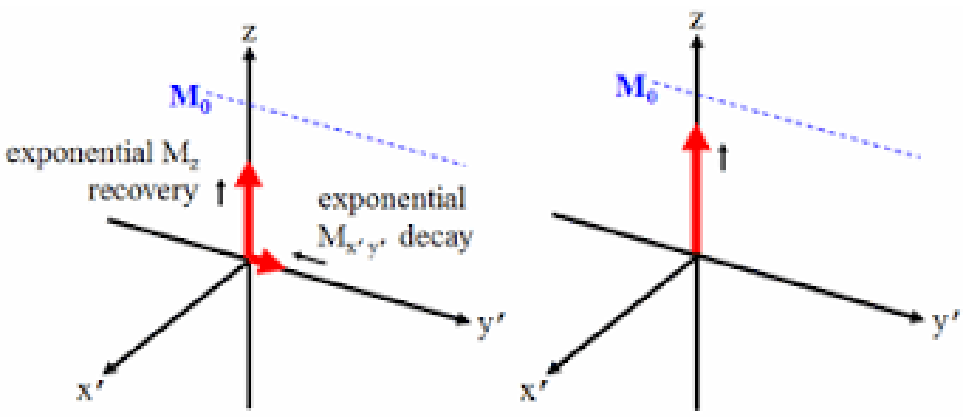
-The transverse magnetization vector (M_{XY}) rotates/precesses about the external field.

***-**Any given transverse magnetization vector, M_{XY}, **decays** as it interacts with other spins **(T2, 'spin-spin', or transverse relaxation).** Once spins interact (and dephase), the M_{XY} is irretrievably lost.*

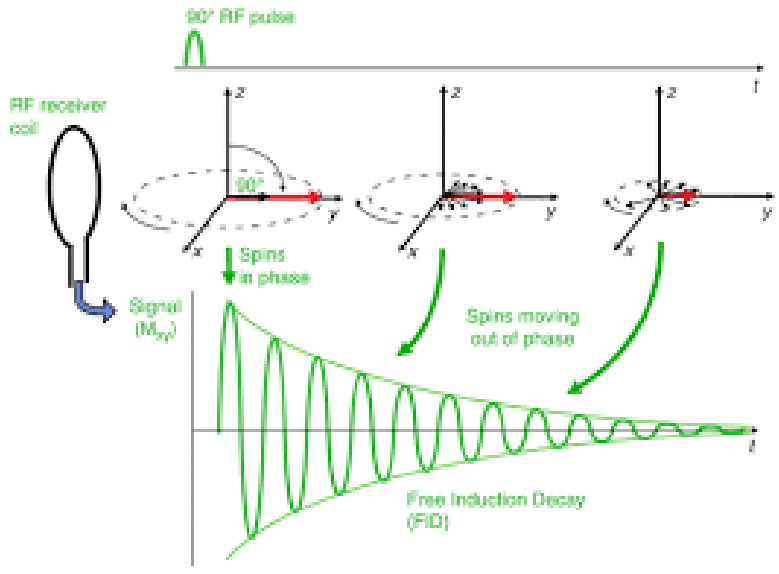
- T2 relaxation times are tissue specific (but independent of the strength of the external magnetic field)

-<u>Note</u>: In addition to spin-spin interaction, M_{XY} may be lost due to external magnet field inhomogeneity, adjacent magnetic tissues, and tissue boundaries. These factors create what is called T2* relaxation (which is faster than T2 relaxation).

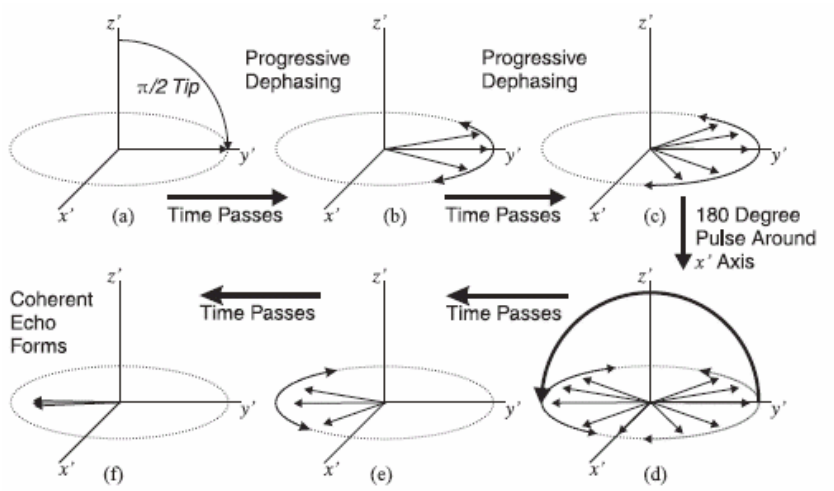
-These T2* factors can be offset with the rephasing of spins in order to generate an *echo* for RF receiver detection. *Very simply stated*, this rotating transverse magnetization (after *rephasing* with 180-degree RF pulse) generates an *echo* that can be detected as an induced voltage in a receiver coil, (RF coil placed close to a given body region). With a great deal of processing (beyond the scope of this lecture), an image is created. **Only spins in the XY plane assist in image creation.**



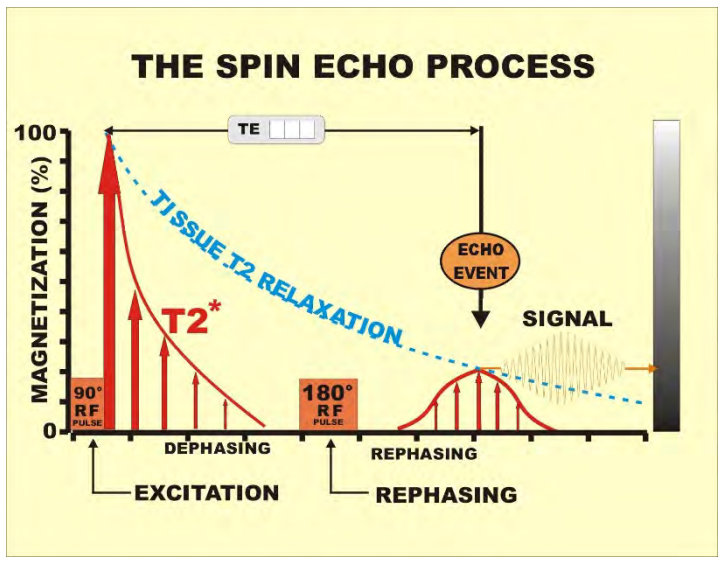
T1 recovery (in M_Z) and T2 decay (in M_{XY})



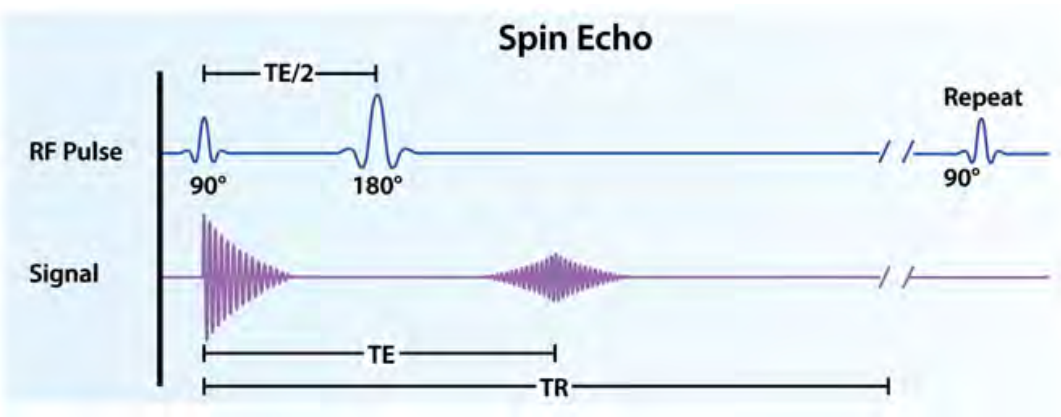
Dephasing in the transverse plane (with decay over time)



Rephasing with 180-degree pulse (in order to generate an echo for receiver coil detection)



-90 degree RF: initial pulse moves M_Z into M_{XY};
-T2 is dephasing from field inhomogeneity*
-180-degree pulse: 'rephases' the spins in M_{XY} and yields an echo detected by receiver RF coil at TE (echo time)



TE: Echo time
TR: Repetition time (time until next 90-degree RF pulse, beginning the process again)
TR and TE can be altered to vary the appearance of tissues

CLINICAL CONDITIONS (Brain)

Normal aging: a multitude of findings

-Atrophy: widened sulci, diminished volume of gyri, ventriculomegaly (**red arrows)**

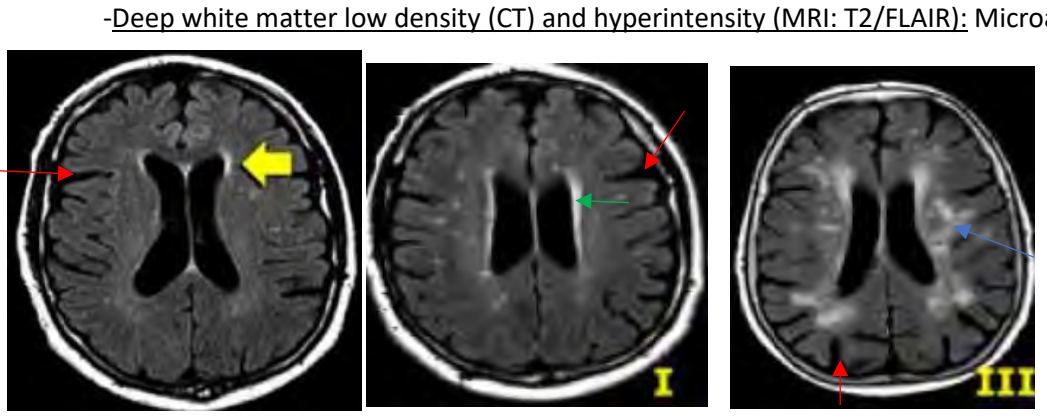
-Periventricular caps (anterior and posterior poles of lateral ventricles): myelin pallor and prominent perivascular spaces (**yellow arrows**)

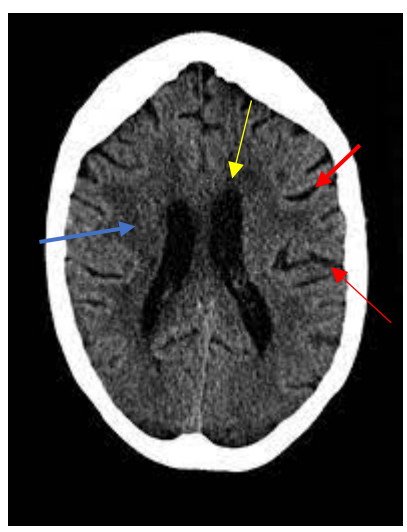
-T2/FLAIR hyperintensity (and CT hypodensity)

-Periventricular bands/rims (thin linear lesions along the bodies of the lateral ventricles): subependymal gliosis (**green arrow**)

-T2/FLAIR hyperintensity (and CT hypodensity)

-Deep white matter low density (CT) and hyperintensity (MRI: T2/FLAIR): Microangiopathy (**blue arrows**)





CT of aging brain (see findings in text)
www.esnr.org

MRI of aging brain (see findings in text)
Radiology assistant

Cerebral ischemia/infarction: Sudden interruption of adequate volume of cerebral blood flow necessary to maintain normal cerebral function

-Clinical symptomatology will depend on the affected vascular territory

-Ischemia: Diminished perfusion within a vascular territory

-Infarction: Tissue death (as a result of ischemia)

-Mechanisms of vascular obstruction:

-*Embolic*:

-cardiac embolism (i.e. atrial fibrillation, endocarditis, LV aneurysm)

-paradoxical aneurysm (i.e. DVT in the presence of R>L cardiac shunt)

-atheromatous embolism

-fat/air embolism

-*Thrombotic*:

-plaque rupture (with associated overlying thrombosis)

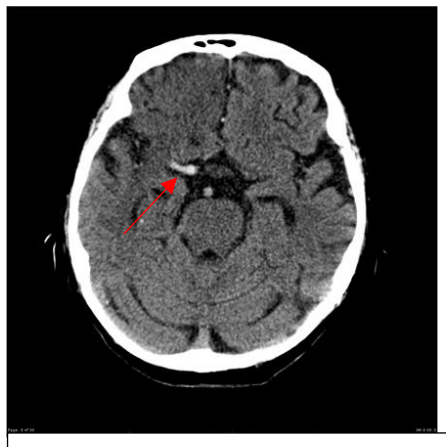
-thrombosis of perforator vessel (i.e. lacunar infarction)

-Arterial dissection:

-internal carotid artery (ICA)

-vertebral artery

-Global hypoperfusion (i.e. cardiac arrest)



Hyperdense MCA sign (red arrow)
Radiopaedia.org

-Imaging findings depend on the timeframe from initial vascular insult relative to the time of imaging:

-Different terminology and timeframes exist (one set of which is annotated below):

- 'Hyperacute': 0-24 hours
- 'Acute': 1 day <X< 7 days
- 'Subacute': 1 week <X< 3 weeks
- 'Chronic': > 3 weeks

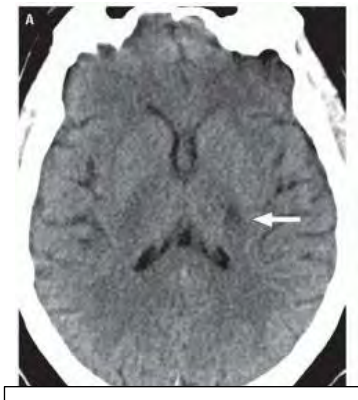
-Imaging workup of acute vascular insult (i.e. 'ischemic stroke'):

-***Non-contrast (unenhanced) head CT*: initial imaging examination of choice**

-fast, readily available, relatively inexpensive (advantages)

-excellent at excluding acute intracranial hemorrhage, which would preclude thrombolysis

-can help to exclude other intracranial pathology (i.e. neoplasia)



Left thalamic lacunar infarct (arrow) *ResearchGate*

-may have limited sensitivity to ischemia/infarction in the hyperacute setting (relatively disadvantage)

-vessels with collateral circulation (i.e. MCA): infarction is visualized 60-70% of the time (within ***6 hours***)

-end arteries (i.e. lenticulostriate arteries): infarction is visualized in 60% of patient (within ***1 hour***)

-Potential CT findings (based on imaging timeframe, above)

-*Hyperacute*:

- 'hyperdense artery' (most common in MCA)

-visualization of acute thrombus/embolus within a vessel (an immediate effect)

-hypodense deep nuclei (i.e. edema)

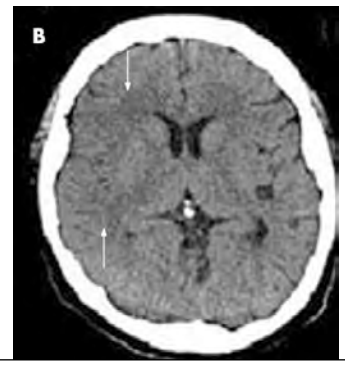
-loss of gray matter-white matter differentiation (edema)

- cortical hypodensity and gyral effacement (edema)

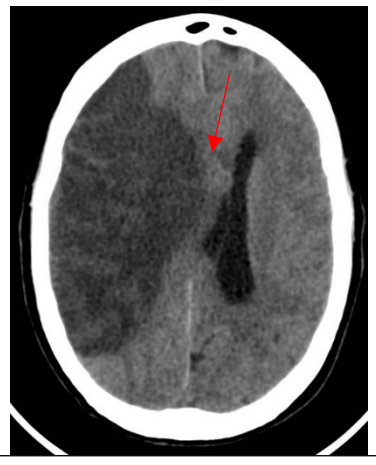
-*Acute*:

-cerebral hypodensity and mass effect increase

-midline shift and potential intracranial herniation occur most commonly at this phase



Hyperacute CVA (arrows) loss of gray matter-white matter differentiation
Research Gate



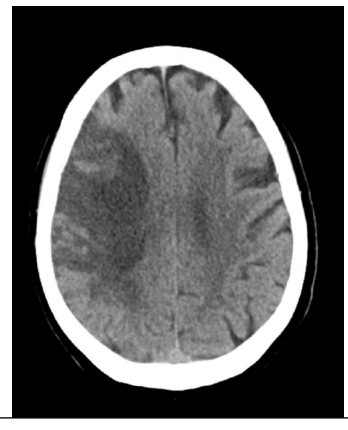
Acute Right MCA infarction (with subfalcine herniation, arrow)
Radiopaedia.org

-*Subacute*:

-cerebral edema begins to subside (see image at right)

-petechial hemorrhages may occur (increasing lesion density)

-cortical contrast enhancement is most prominent at this phase

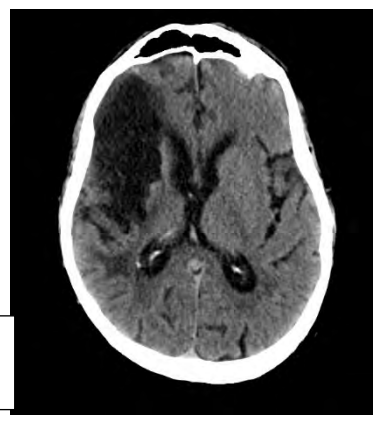


Subacute Right MCA infarction
Radiopaedia.org

-*Chronic*:

-encephalomalacia and gliosis develop

-negative mass effect occurs (i.e. potential midline shift to the side of volume loss)



Chronic Right MCA infarction
Radiopaedia.org

-**CT-perfusion:** contrast-enhanced imaging of brain parenchyma

-imaging tool critical in selection of patients for potential reperfusion therapies

-parameters:

-Tmax: Time-to-maximum (i.e. time delay in arrival of contrast bolus in brain *parenchyma*, compared with proximal large *arteries*)

-CBF: Cerebral blood flow (i.e. blood volume passing through a given amount of tissue per unit time)

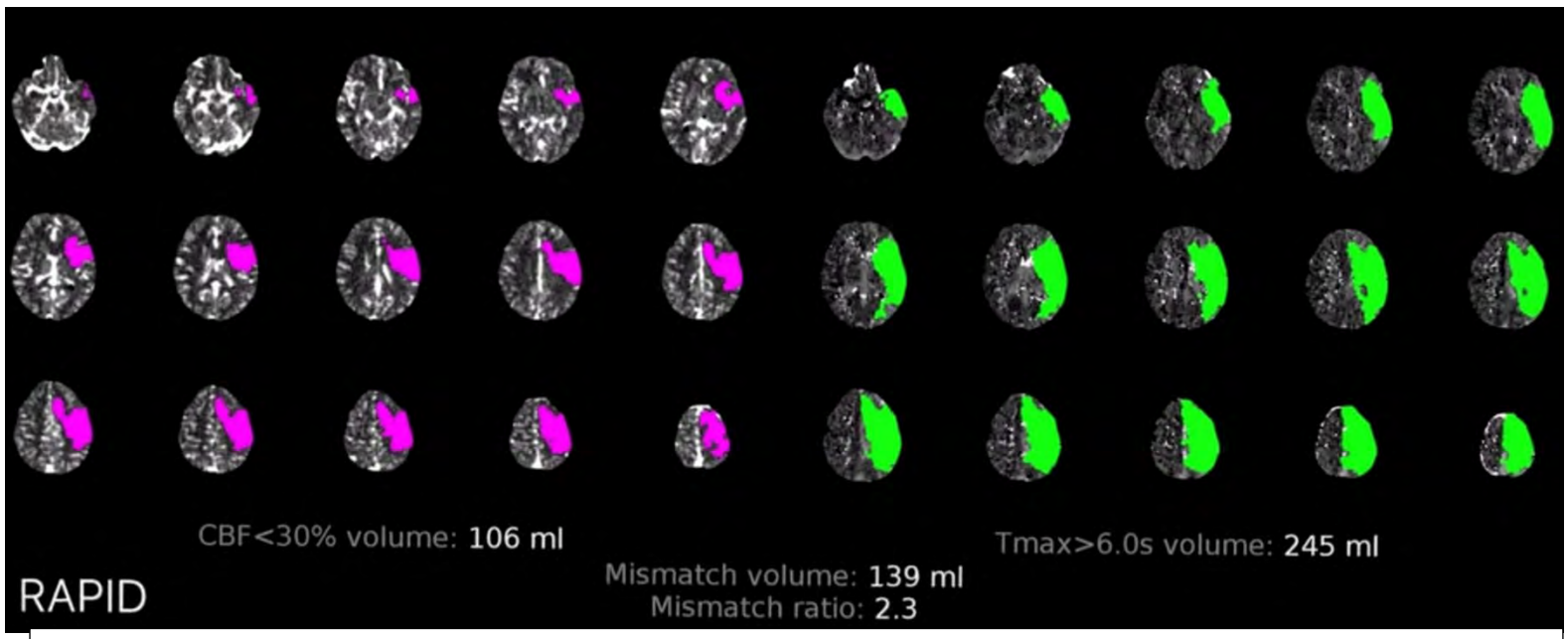
-CBV: Cerebral blood volume (i.e. volume of blood in a given amount of brain tissue)

- '*infarct core'*: brain destined to infarct (regardless of therapy)

-Tmax (prolonged). CBF (markedly reduced). CBV (markedly reduced)

-'*ischemic penumbra'*: brain surrounding the infarct core (and potentially salvageable)

-Tmax (prolonged). CBF (moderately reduced). CBV (normal/increased, secondary to vasodilatation)



CBF (Cerebral Blood Flow) <30%: Infarct core - severe impaired perfusion that indicates likely infarcted tissue
Tmax > 6 sec: Penumbra - delayed arrival of contrast indicates tissue that will likely progress to infarction if not reperfused
***Mismatch volume*:** Potentially salvageable tissue (difference between ischemic penumbra and infarct core)
***Mismatch ratio*:** Ratio of Tmax > 6 sec / CBF < 30%

Note: One type of calculation (RAPID automated CT perfusion) delineates *infarct core* as tissue volume (with CBF <30% of expected) -AND- *ischemic penumbra* as tissue volume with Tmax (>6sec).

Note: '***Mismatch volume'*** is the difference between the *ischemic penumbra* and *infarct core*
Note: *Mismatch ratio'* is the ratio of *ischemic penumbra* to *infarct core*

Note: The greater the mismatch volumes (or the higher the mismatch ratios), the greater the amount of *salvageable brain tissue* (in the presence of reperfusion therapies). The discussion of decision-making based on CT-perfusion is beyond the scope of this document

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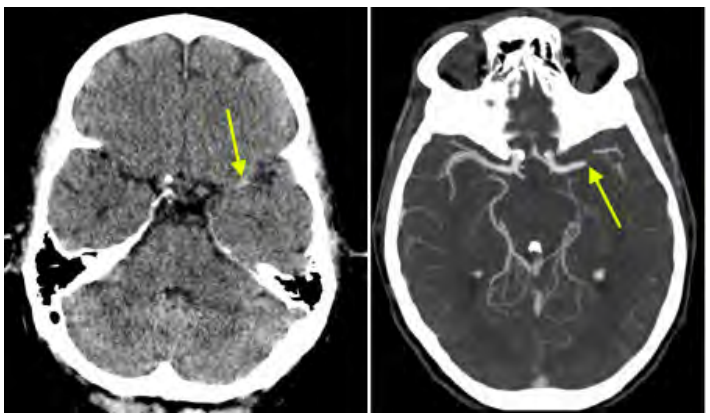
-**CT-angiography**: contrast-enhanced imaging of extracranial/intracranial vasculature (as an adjunct to CT-perfusion)

-assessment of carotid artery and vertebrobasilar systems (i.e. atheromatous disease/stenosis/dissection)

-visualization of intracranial vasculature (i.e. stenosis, thrombus, vasculitis, aneurysm)

-guidance for intra-arterial therapy (i.e. thrombolysis, clot retrieval) as well as post-therapy assessment



Left image: Hyperdense left MCA (arrow on unenhanced CT of the brain)
Right image: Focal occlusion of MCA (arrow on CT-angiogram of the brain)
svuhradiology

-**MR imaging**: increased sensitivity and specificity for diagnosis of acute ischemia/infarction (compared with CT imaging)

-Potential MR findings (based on imaging timeframe, above)

-*Hyperacute*:

-abnormally restricted water movement (hyperintensity) on diffusion-weighted images (DWI)

-within minutes of the arterial occlusion

-correlates with *infarct core* (see above)

-T2/FLAIR hyperintensity: begins after 6 hours (and increasing in the coming days)

-T1 hypointensity: begins after 16 hours (less apparent than T2/FLAIR signal abnormality)

-*Acute*:

-midline shift and potential intracranial herniation occur most commonly at this phase

-DWI hyperintensity persists

-T2/FLAIR hyperintensity persists (becoming more apparent over time)

-T1 hypointensity persists (with areas of T1 hyperintensity indicating *cortical laminar necrosis*)

-*Subacute*:

-cerebral edema begins to subside

-T2/FLAIR hyperintensity persist; T1 hypointensity persists (with T1 hyperintense foci possible, see above)

-DWI hyperintensity diminishes (in complex pattern, based on interplay between T2 signal and hemorrhage)

-petechial hemorrhages may occur (with sites of hemorrhage also possible in the acute phase)

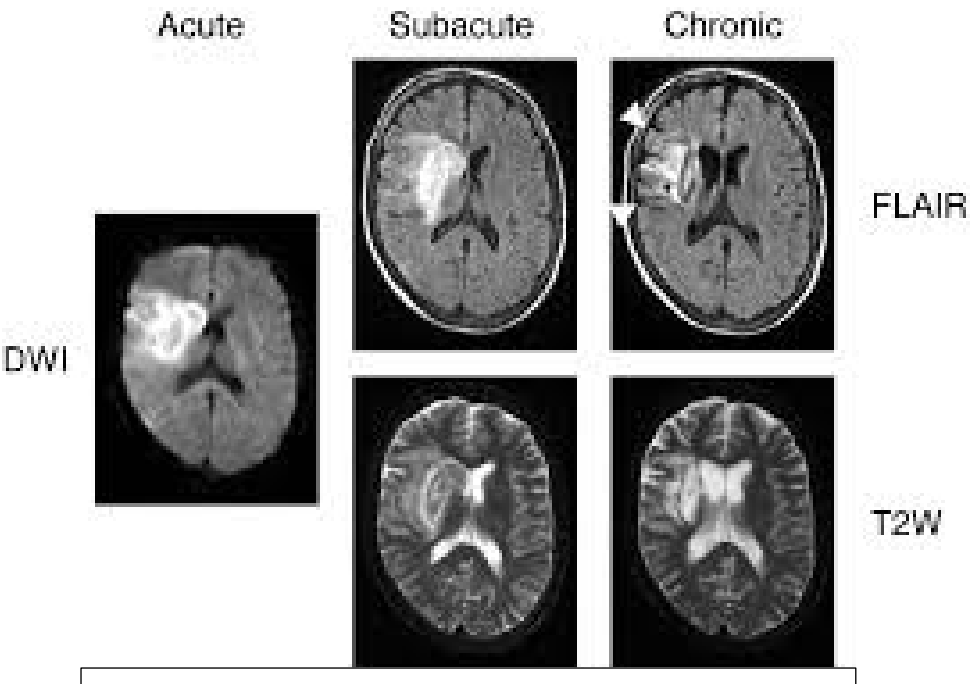
-cortical contrast enhancement is most prominent at this phase (i.e. T1 post-contrast hyperintensity)

-*Chronic*:

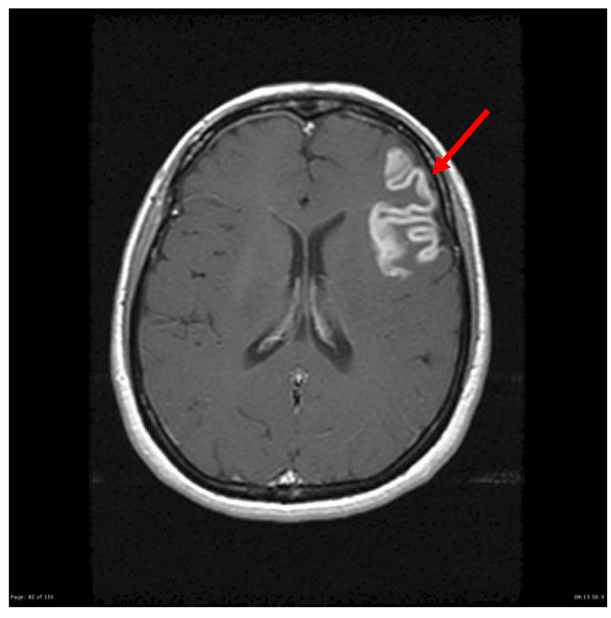
-encephalomalacia and gliosis develop

-negative mass effect occurs (i.e. potential midline shift to the side of volume loss)

-FLAIR/T2 hyperintensity; T1 hypointensity (i.e. gliosis)



Acute image: obtained within 1 day of right MCA infarction
Subacute images: obtained beyond 1st week after initial insult
Chronic images: obtained approximately 4 months after initial insult
JMRI



Gyral enhancement (arrow) in left frontoparietal region (on T1-weighted post-contrast enhanced image)
Medicalrojak.wordpress.com

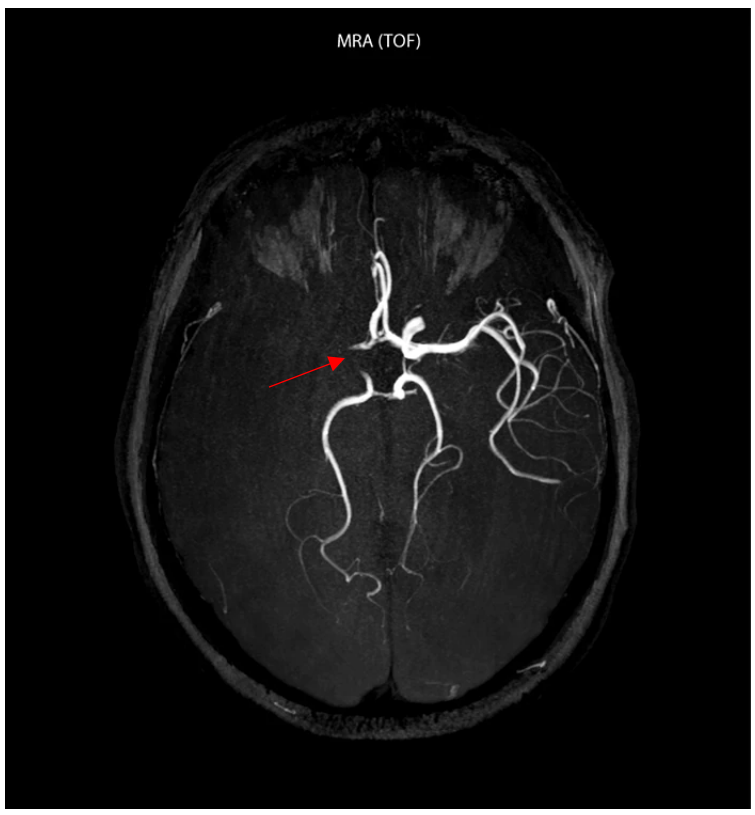
Note: Sites of contrast enhancement (other than cortical) are less common (i.e. meningeal, intra-arterial)

-contrast enhancement is potentially also noted in the acute phase

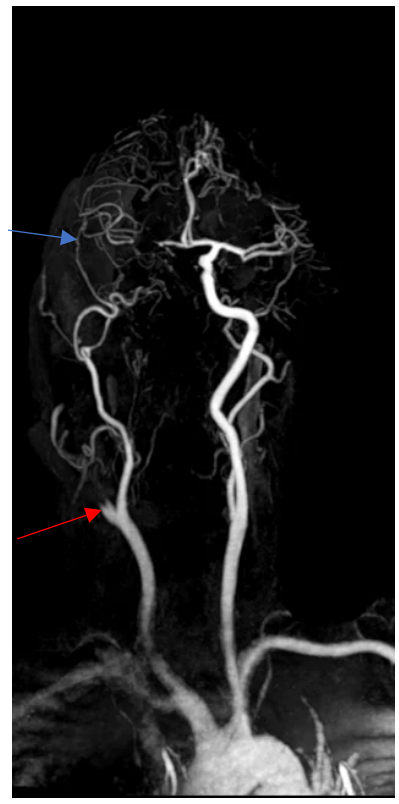
-if contrast enhancement persists (> 3months): underlying neoplasia is a possibility

Note: Contrast enhancement also occurs in other disease processes (i.e. neoplasia, inflammatory/infectious, post-operative)

Note: MR-angiography (i.e. head and neck) may be performed for vascular assessment
-MRA head (unenhanced imaging)
-MRA neck (unenhanced/enhanced imaging, with gadolinium)



MR-angiography (brain): right ICA and right MCA occlusion, red arrow
Note: Cross-filling of right ACA (via patient ACOM)
Phillips imaging

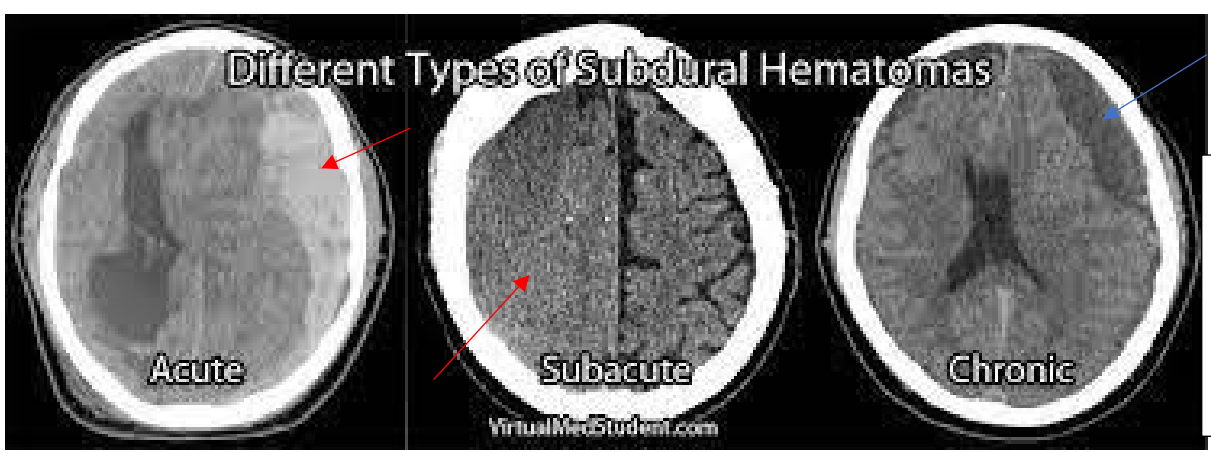


MR-angiography (neck): occlusion of origin (bulb) of right ICA, red arrow
Note: Distal right MCA branches (blue arrow) are visible via cortical collateral circulation
Phillips imaging

Intracranial hemorrhage: presence of extravascular accumulation of blood within a variety of intracranial locations
-Our discussion will focus on CT imaging findings

-CT appearance of hemorrhage: CT density/attenuation corresponds to hematocrit, hemoglobin concentration, protein (globin) content
-appearance can vary depending on clot volume, location, surrounding structures, technical factors

-hallmark imaging characteristics are as follows (with timeframes somewhat 'generalized'):
-Acute hemorrhage: 1-3 days
-hemorrhage is hyperdense ('brighter') compared with brain parenchyma
-Subacute hemorrhage: 3 days to 3 weeks
-hemorrhage is isodense ('similar in density') compared with brain parenchyma
-Chronic hemorrhage: greater than 3 weeks
-hemorrhage is hypodense ('darker') compared to brain parenchyma



Different CT densities of intracranial hemorrhage
-Hyperdense to brain: *acute* (left image)
-Isodense to brain: *subacute* (middle image)
-Hypodense to brain: *chronic* (right image)
Simply radiology

Note: Subdural collections are used as an example

-Caveats:
-In *anemia*, clot density is relatively decreased (compared to expected findings, above)
-In *coagulopathies*, failure of clot retraction may lead to decreased density of clot in acute setting

-Hemorrhage description

-size (ideally in three dimensions)

-surrounding (vasogenic) edema (if present)

-CT: hypodensity

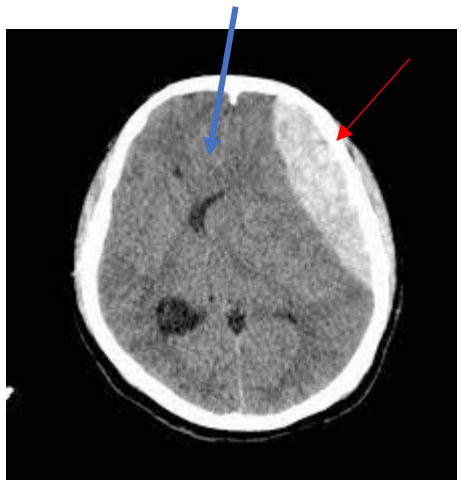
-density (i.e. age) of hemorrhage

-mass effect (on ventricles)

-midline shift (quantified, if present)

-signs of herniation

-intraventricular extension and associated hydrocephalus (if present)



Acute left frontoparietal epidural hematoma (red arrow)

Note: Rightward midline shift and subfalcine herniation (blue arrow)
Radiopaedia.org

-location: extra-axial vs intra-axial

-extra-axial

-epidural: located between inner surface of skull and outer dura

-*shape*: usually biconvex

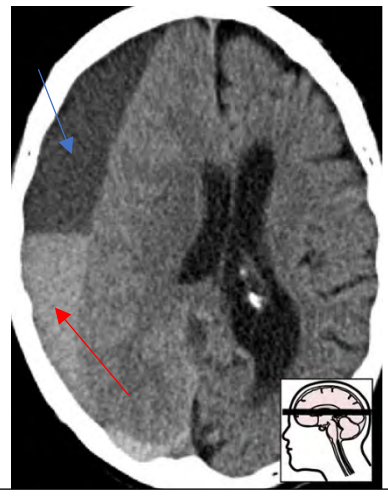
-*location*: supratentorial (95%) (most commonly temporoparietal)

-usually does not cross suture lines

-may cross midline (as well as dural reflections: falx, tentorium)

-associated with skull fractures (95%)

-arterial source (90%); venous source (10%, dural sinuses)



Acute (red arrow) -ON- Chronic (blue arrow) right supratentorial subdural hematoma.
Note; Leftward midline shift exists
Start radiology

-subdural: located between dura and arachnoid mater

- *shape*: usually crescentic (i.e. inner concave; outer convex)

-*location*: supratentorial (95%)

-frontoparietal, middle cranial fossae; parafalcine

-can cross suture lines

-does not cross midline

-does not cross dural reflections (falx, tentorium)

-associated with tearing of bridging veins (i.e. venous source)

-'shaken baby syndrome': bilateral in 75-85%

-MVC (motor vehicle crash) in adults

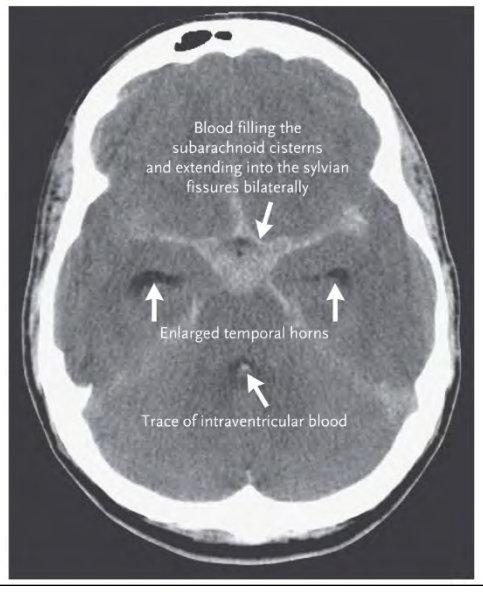
-fall in the elderly

-subarachnoid: located within subarachnoid space

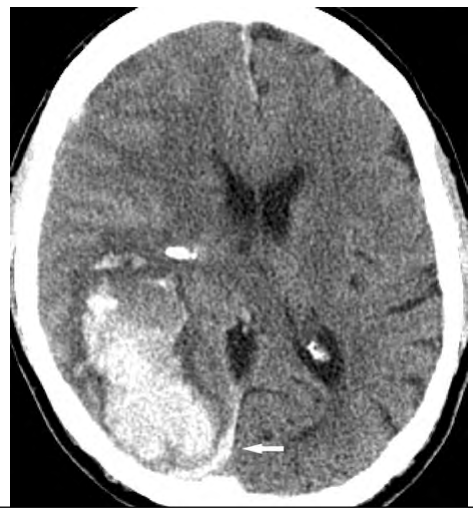
-hyperdense material (acute hemorrhage) overlying brain parenchyma (within fissures)

-extension into cisterns (i.e. suprasellar, interpeduncular, ambient)

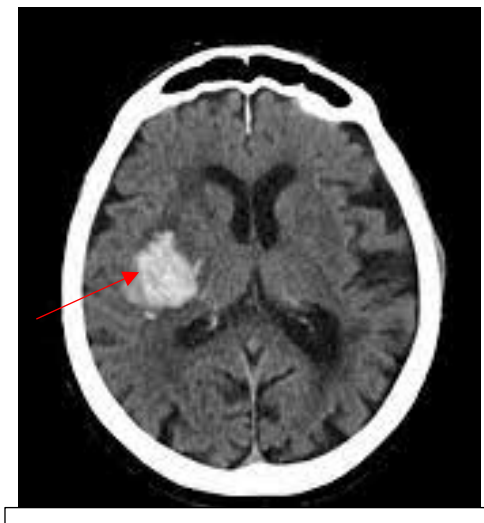
-possible intraventricular extension (with potential for obstructive hydrocephalus)



Acute subarachnoid hemorrhage (with intraventricular extension and obstructive hydrocephalus) *NEJM*



Acute intraparenchymal hemorrhage (cerebral amyloid angiopathy) *RSNA*



Acute right basal ganglia hemorrhage (arrow)
Radiopaedia.org

-intra-axial

-intraparenchymal: hemorrhage within brain parenchyma (from a multitude of causes, some of which are noted)

-lobar location:

-*primary*: cerebral amyloid angiopathy (elderly)

-*secondary* (to underlying lesion): AVM, neoplasia

-basal ganglia/pons/cerebellum:

-commonly poorly controlled, chronic hypertension

-intraventricular: hemorrhage within the ventricular system (with potential for complicating hydrocephalus)

-may be associated with subarachnoid hemorrhage

-other potential etiologies include trauma, neoplasia, anticoagulation, bleeding tendency

Brain neoplasia: heterogeneous group of tumor entities

-Detailed delineation of individual tumors is beyond the scope of this discussion

-please see clinical and pathology lectures for more details

-General features:

-pathophysiology:

-primary neoplasia (60%)

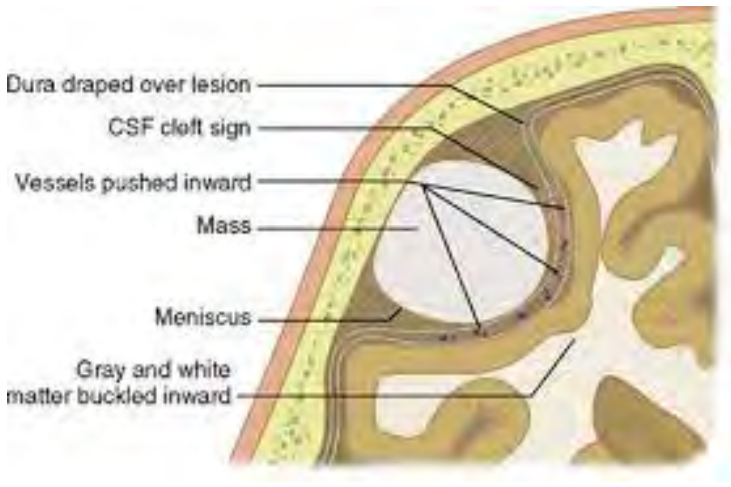
-metastases (40%)

-location:

-supratentorial (80%)

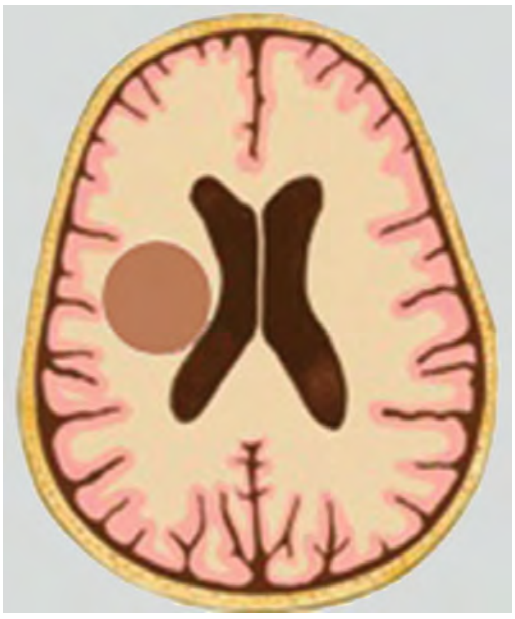
-infratentorial (20%)

-intra-axial vs extra-axial

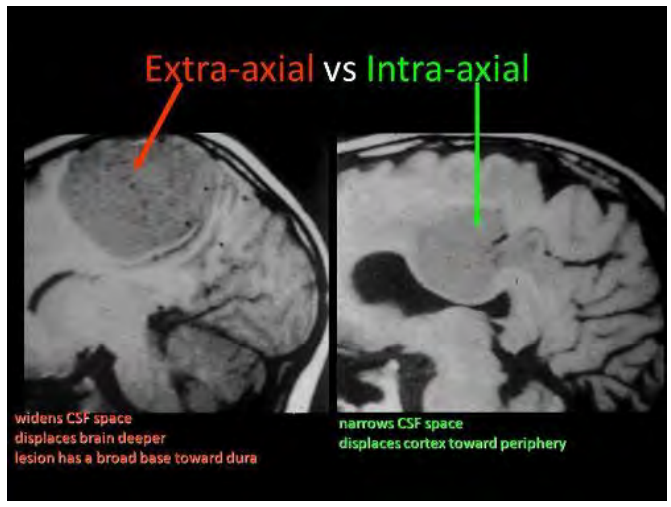


Extra-axial features: displaced pial vessels; widened subarachnoid space; CSF 'cleft' between mass and brain; wide dural/calvarial base (i.e. see MR imaging meningioma, below)

Radiology Key



Intra-axial features: narrows CSF spaces; displacement of cortex toward the periphery *link.springer.com*



Meningioma (left extra-axial mass)-VS-
Glioma (right intra-axial mass) on unenhanced T1-weighted sagittal MRI
The Trauma Pro

-CT imaging: often initial imaging examination of intracranial neoplasia (performed to assess clinical symptomatology)

-wide variety of appearances (depending on tumor type)

-useful to exclude hemorrhage

-location: Extra-axial vs intra-axial; Supratentorial vs infratentorial

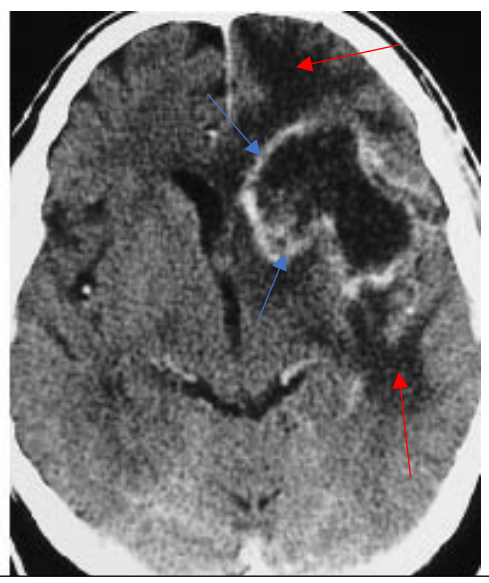
-allows for assessment of edema (vasogenic), mass effect, midline shift, and potential cerebral herniation

-vasogenic edema is hypodense on CT

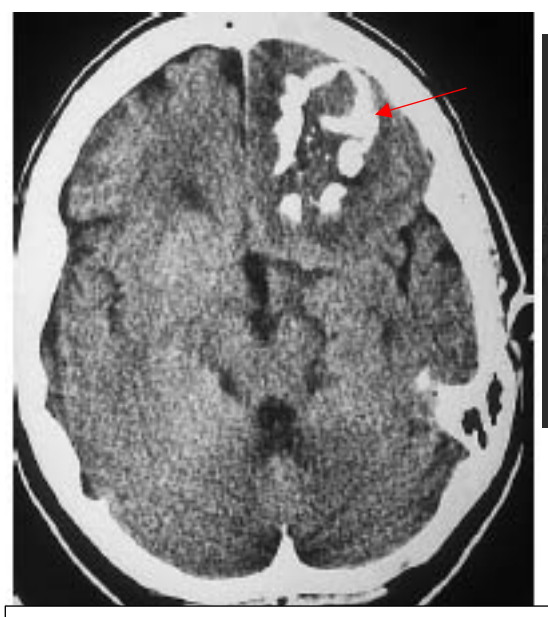
-contrast enhancement (with iodinated contrast media) may increase lesion conspicuity

-contrast enhancement is hyperdense on CT

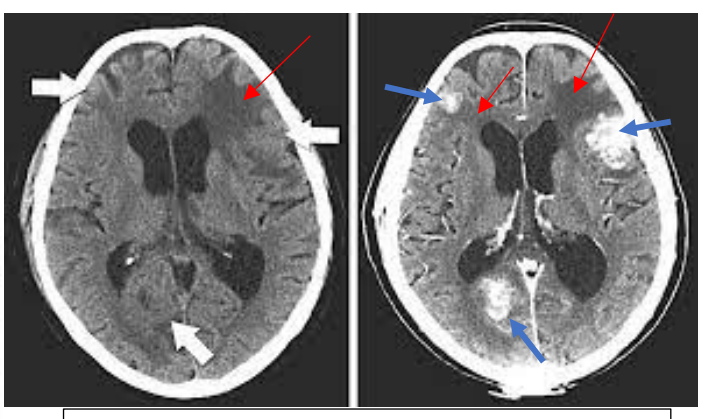
-Note: In patients who cannot undergo MR imaging, follow-up (unenhanced/enhanced) CT images allow for serial assessment



Glioblastoma multiforme (GBM): peripheral enhancement (blue arrows), vasogenic edema (red arrows), and mass effect (on this ***enhanced*** CT) *Radiology Key*

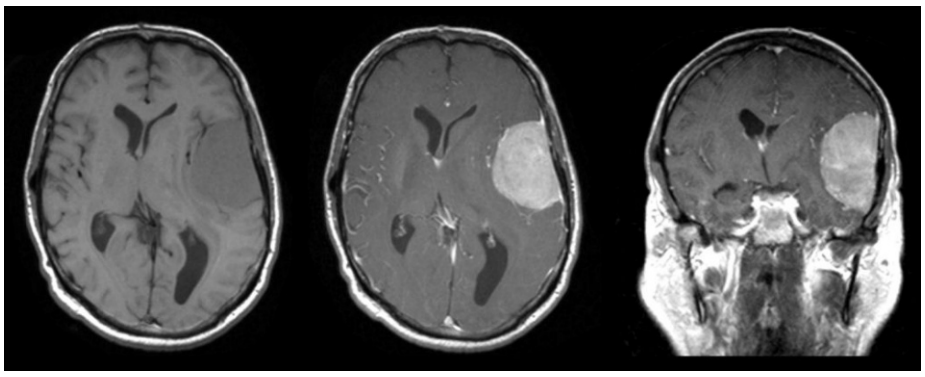


Oligodendroglioma: Coarse intraparenchymal calcifications, arrow (on this ***unenhanced*** CT image) *Radiology Key*

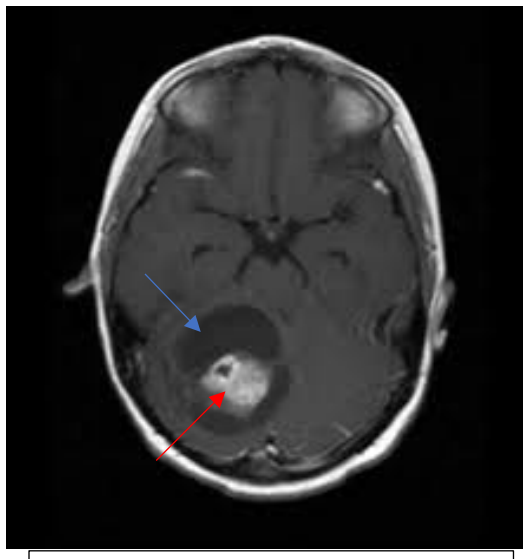


Cerebral metastases:
Left Image (pre-contrast CT): vasogenic edema (red arrow); metastases (white arrows
Right image (post contrast): vasogenic edema (red arrow); metastases (blue arrows
www.karger.com

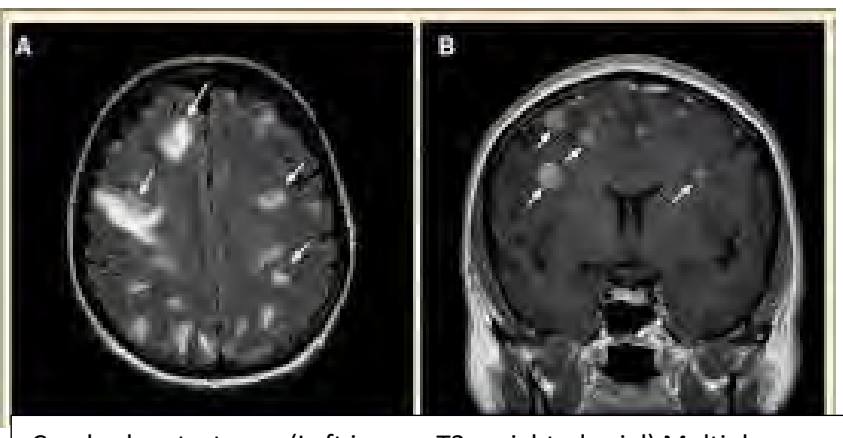
-MR imaging: imaging examination of choice for assessment of intracranial neoplasia
-unenhanced and enhanced imaging (utilizing gadolinium, Gd, contrast media) is optimal
-spatial and contrast resolution allows for optimal lesion assessment/characterization
-assists with decision-making (i.e. potential biopsy)
-allows for serial assessment/follow-up



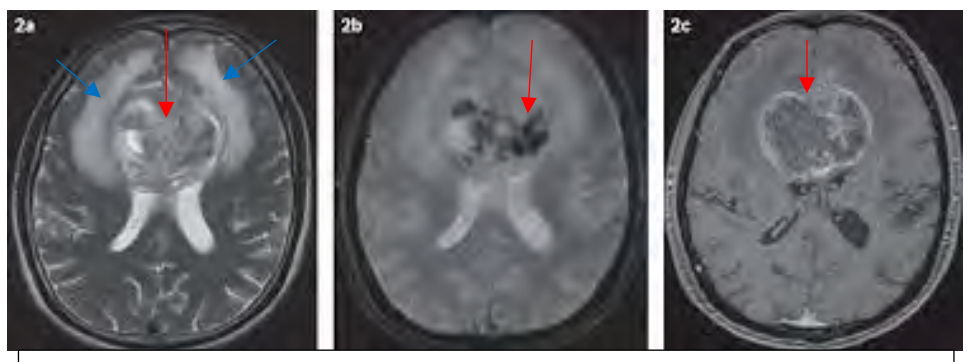
Supratentorial meningioma: Left image (pre-contrast T1 axial), Middle image (post-contrast T1 axial), Right image (post-contrast T1 coronal)
www. case.edu



Juvenile pilocytic astrocytoma:
Enhancing mural nodule (red arrow) and cystic component (blue arrow) on T1-post-contrast axial image
Radiopaedia.org



Cerebral metastases: (Left image, T2-weighted axial) Multiple hyperintense masses (with surrounding vasogenic edema)
(Right image, T1 post contrast coronal) Multiple T1 hyperintense enhancing masses



GBM ('butterfly glioma': (Left image, T2-weighted) Mass (red arrow) crossing the corpus callosum (with surrounding edema, blue arrows).
(Middle image, gradient echo) Mass demonstrates hypointense intra-tumoral hemorrhage (arrow)
(Right image, T1-post-contrast) Heterogeneously-enhancing mass (arrow)
smj.org

-Tumor description
-size (ideally in three dimensions)
-location: extra-axial vs intra-axial; supratentorial vs infratentorial
-density (CT) or intensity (MRI) of the tumor: includes potential enhancement and potential calcifications
-enhancement: *hyperdense* (CT); T1 post-contrast *hyperintense* (MRI)
-calcification: hyperdense (CT); T1/T2 hypointensity (MRI)
-surrounding (vasogenic) edema (if present)
-hypodense (CT)
-T1 hypointense; T2/FLAIR hyperintense; DWI hyperintense (but less than that seen with cytotoxic edema of CVA)
-mass effect (on ventricles), midline shift (quantified, if present), midline extension (via corpus callosum): GMB/metastases, signs of herniation

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Note: Imaging of demyelinating diseases will be provided in clinical sessions

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References:

-Clinical Radiology: The Essentials. Daffner et al. 4th ed. (Chapter 12).

-Primer of Diagnostic Imaging. Weissleder et al. 4th ed. (Chapter 6).

-Diagnostic Neuroradiology. Osborn, Anne. (Chapters 7, 11, and 12)

-**Note**: Medical images are from anonymized patient data and online archives (as detailed)

OPTIONAL: Want to know more?

https://www.med-ed.virginia.edu/courses/rad/

www.auntminnie.com www.acr.org www.rsna.com