


An Integrated Approach to the Evaluation of Brain Dysfunction: Brain Imaging, Neuropsychological Assessment, and Medical Chart Review

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Financial Disclosure

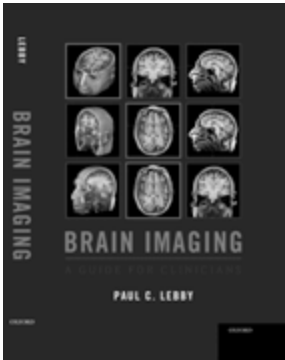
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Brain Imaging
A guide for Clinicians.
Leiby, P. C., 2013, Oxford University Press



- All patients, and if appropriate parents/legal guardians of patients, depicted in this presentation have given their written permission for me to disclose their personal health information (PHI) to allow for the teaching and education of others.
- Please respect their generous and selfless contribution to your continued education by maintaining their privacy, and extending appropriate confidentiality to these patients.

Ethics

- The use of collateral medical information is well within the scope of practice for neuropsychology.
- However **with increasing access to EMRs and DICOM data, there is an increased opportunity to review information outside of the scope of expertise** for any individual neuropsychologist.

Caution

This course will not make it ethical for you to interpret and discuss medical chart information or neuroimaging in your practice...

This does not mean that neuropsychologists are precluded from doing so with the appropriate:

- **Education**
- **Training**
- **Experience**

Only you know your specific level of expertise

My thoughts

- To consider it ethical for you to interpret medical and/or neuroradiology information/images, you need to consider your level of expertise, and communicate such to the listener, tempering your statements appropriately.

ALSO CONSIDER:

- Are you capable of passing voir dire in court
- Are you credentialed, if on medical staff
- Are you confident in your (education, training, exper.)
- Is it necessary for your role, or opens you up for liability

What goes into expertise... in addition to the big three (education, training, experience)...

- Have you written papers, chapters, books on the topic?
- Are you credentialed to act within that scope of practice?
- How often do you conduct such service and in what capacity?
- What do your colleagues think, do other's with greater experience and training sometimes rely on your opinion?
- Can you defend yourself in deposition, court, litigation?
- Do you know enough to know what you don't know?
- Do you know the limitations of the information you are using?

Utilizing neuroimaging or medical chart information

What is your medical literacy level?

- ROS, DTRs, GCS, MSE, A+Ox4
- PRN, QD, BID, TID
- ICP, CPP, ICA, PCA, MCA
- ROI, EDH, SDH, SAH, IPH, IVH
- DKA, GSW, HIE,
- ETT
- Etc.

Important questions

What is the reason for reviewing the chart, the imaging reports or actual images?

- Curiosity
- To direct assessment/intervention
- To determine if modifications with assessment or intervention may be required
- To assist patient and/or care providers in understanding the full extent of injury
- To assist with determination of prognosis
- Combination of these factors or others.

11

Advantages of Reviewing Imaging

It can provide clues regarding:

- **Type of injury (traumatic, hypoxic, infectious, etc.)**
- **Extent of injury (mild, moderate, severe, profound)**
- **Areas most involved (diffuse/global, focal, both)**
- **Expected functional outcome (based on above)**
- **Expected time course for recovery (exponential vs light-switch).**

Comfort to families (if looks good)

Break through denial (if looks bad)

Realistic expectations

Advantages of Reviewing Chart

It can provide clues regarding:

- Type of injury (traumatic, hypoxic, infectious, etc.)
- Extent of injury (mild, moderate, severe, profound)
- Medical issues of concern (isolation, vent status, ETT, stability to tolerate exam)
- Medication management
- Treatment plan especially surgical
- Discharge plan/disposition (can exam wait a week)
- Past medical history
- Past developmental history

Imaging Chart Review Neuropsychological Assessment

Which do you do first and why?

Assessment first

More likely to be comprehensive, but may not focus enough on critical areas of functioning prompted by chart/images.

Review first

More likely to be focused with assessment, but may miss issues not prompted by chart/images.

Images First then Reports

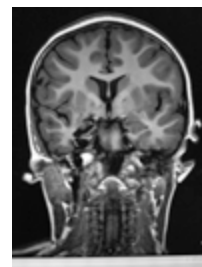
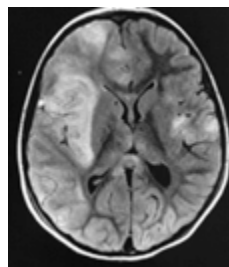
Look at images with broader view
My preference, but not recommended

Reports First then Images

Look at images with narrower view
Better for less experienced, however can miss clinically relevant findings

Example of diencephalic storming...

I may have missed the injury and explanation for symptoms if I only focus only on areas described in the radiology report



Critical Information To Consider

- Medications, especially those that result in:
 - Sedation
 - Cognitive disruption
 - “psychotic-like symptoms”

Don't forget, tapering of medications can result in unique neurocognitive and/or psychiatric symptoms unrelated to the brain pathology per se.

- Know the general dosing and side effects
- Know the general effects of increase/decrease
- Have an understanding of how your clinical impressions may or may not be influenced by medication issues

Consider conditions that result in damage to the brain, and review the medical chart for information relating to type, severity, extent, how long condition been present, etc.

- Seizures, especially status epilepticus
- Metabolic disorders
- DKA or other glucose related conditions
- Cancer of the body/brain, chemo, radiation
- Sepsis
- Respiratory distress/arrest
- Prolonged ICU stay

Be cautious about focusing too much on the medical chart or imaging data...

The medical and imaging findings can result in you failing to look for deficits not expected based on your review.

You may bias yourself to only assess areas of expected high yield, resulting in you having a greater chance of missing other clinically important findings.

Or, you over-interpret normal variation in performance due to pathological findings in the chart or imaging (confirmatory bias).

Do not undervalue your finding on examination just because they appear to conflict with information in the chart or radiologic findings

1. The same pathology can result in different deficits
2. The same deficits can be caused by different pathology.

A discrepancy between radiologic findings and functioning is frequently due to the specific level of analysis, or putting it another way, **"What is being measured."**

21

You are the experts on brain functioning...

- Information in the chart is helpful, but is usually based on a very short bedside MSE, or not even that.
- Each specialist evaluates the patient from a different point of view, different perspective, different level of analysis and for different reasons... **what is the reason for your examination?**

Normal neurological examination does not imply normal brain functioning...

- Intensivist ... Care about life sustaining functions
- Hospitalist ... Care about general health, DC issues
- Neurologist ... Care about seizures/motor/sensory
- Neurosurgeon ... Care about ICP, BP, HR
- Neuropsychologist ... Care about brain functioning including how the above issues impact that functioning.
- **Comment from ICU physician (Tara)...**

Surgeon Report:

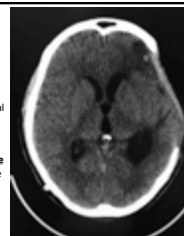
Pt. presents with some dysarthria but follows commands and responds accurately to demographic questions. Her expressive speech is somewhat diminished.

Speech Tx Report:

Pt appeared to be **globally aphasic** with very limited ability to respond to verbal commands. **Nodded 'yes' to all yes/no questions** irrespective of appropriateness with accuracy at chance. Was not able to match shapes to labels or match upper and lower case letters to labels. She could not point to object I named, field of two, with anything better than 'chance' accuracy. **Able to repeat some words after me; did not speak spontaneously.** Unable to write numbers well; did not complete number patterns, did not perform very simple addition and subtraction problems. **Pt. answered Y/N questions with 50% accuracy.** When answering a question when given a verbal choice of two, **always chose the last option presented** (i.e., repeated the last thing she heard). She did not answer "What's your name" correctly. She pointed to the correct object, upon verbal command, field of two with <50% accuracy. When asked basic conversational questions, she did not answer correctly.

My Report:

Severe global (mixed receptive and expressive) aphasia syndrome, with features of mixed transcortical aphasia (some repetition and echolalia is evident but not for complex information and without understanding of what she is saying), disrupting clear communication. She produces some **paraphasic and neologistic** errors and at times "word salad," further disrupting verbal communication. She is not only **dysfluent with dysnomia** and halting/telegraphic speech, but also has **disrupted language comprehension** for even basic commands in English and Spanish. She nods "yes" to all Yes/No questions and is at chance for identification of pictures when given a verbal cue, and at chance when responding to "yes" or "no" written on a board upon provided one-part questions. When she does verbalize, her responses are consistently inappropriate given the specific question or "no se." When responding to questions after being provided a choice of two options, she consistently chooses the last option provided to her, irrespective of the question provided.



Not a problem with neurosurgery assessment, as they are there to assess for critical neuromedical/surgical issues that are life threatening... It is not their role to assess cognition in detail, that is our job.

However, just a review of the chart notes would lead one to believe there was no significant aphasia... something that is clearly incorrect.

You need to evaluate where the information is coming from... then determine the degree to which it should be relied upon, or further investigation is warranted.

Physicians may label symptoms based on limited neuropsychological or neuropsychiatric training or experience... Reading between the lines can be very helpful (consider the source... ICU MD, etc).

Similar behaviors can be related to one or more of the following... And just called "psychotic" in the chart. It is your job to figure out which fits the best.

- Hallucinations
- Hypnagogic Perceptions
- Confabulations
- REM sleep behavior disorder
- Malingering
- Medications, post anesthesia, other

Similar issues relate to the use of imaging data (actual image review) and imaging reports when integrating information into your clinical formulation.

Take time to consider the relevance of any findings as they pertain to your functioning as a neuropsychologist.

Neuroimaging The Basics

- Terminology/Nomenclature
- Basics of Reading Brain Scans
- Common Imaging Techniques
- Interpretation of Brain Pathology

TERMINOLOGY NOMENCLATURE

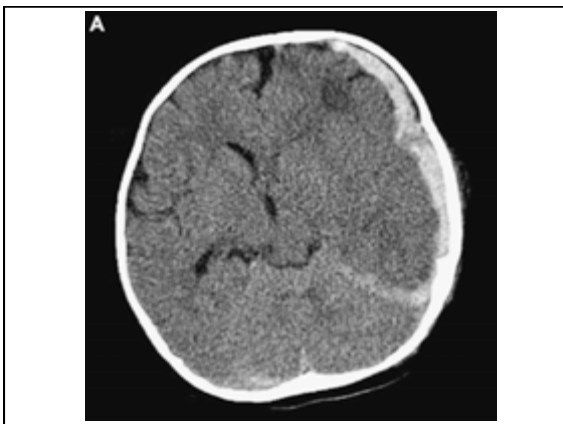
29

Extra-Axial

Any lesions/pathology external of the brain parenchyma (tissue)

Examples:

Epidural Hematoma – MOST COMMON USE
Subdural Hematoma
Subarachnoid Hemorrhage
Infections (Meningitis)
Some brain tumors
Some foreign objects (don't penetrate brain)



Intraparenchymal

Any lesions/pathology within the brain parenchyma (tissue)

Examples:

Strokes/Hemorrhages
Infections (Encephalitis)
Diffuse Axonal or Shear Injuries
White Matter Disorders (MS, GBS, ADEM)
Edema (swelling)

Midline Shift

On Axial or Coronal images, bowing of the falx is usually the best indicator of the extent of midline shift.

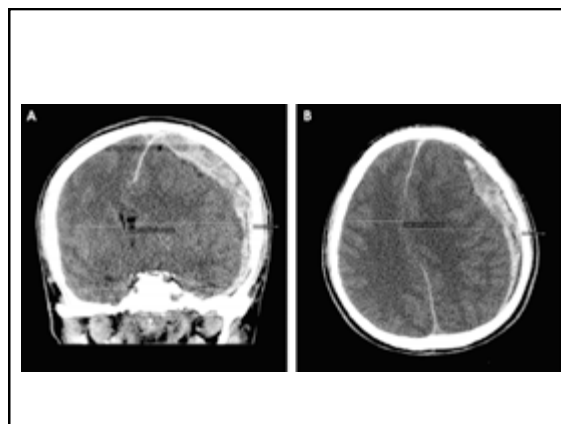
Also, assess the ventricles for asymmetry caused by disproportionate pressure

Midline Shift

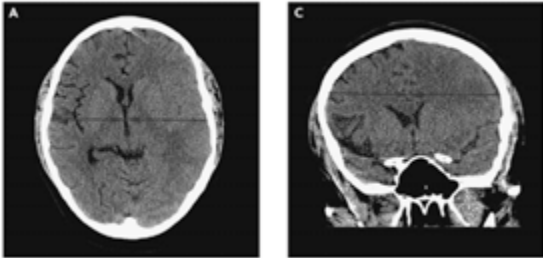
Consider what may be causing the shift?

Extra-axial process such as EDH/SDH
Intraparenchymal process (neoplasm)
Edematous Tissue
Combination of above

But also,
Head not orthogonal in the scanner
Steroid effects greater for healthy tissue

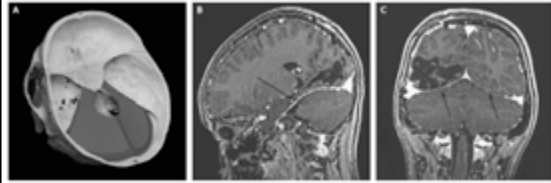


Is the tissue on the left atrophied and the tissue on the right normal?
 Is the tissue on the right edematous and the tissue on the left normal?
 Is the tissue on the right edematous and the tissue on the left atrophied?
 Is the tissue on left normal (with steroids), tissue on right edematous?



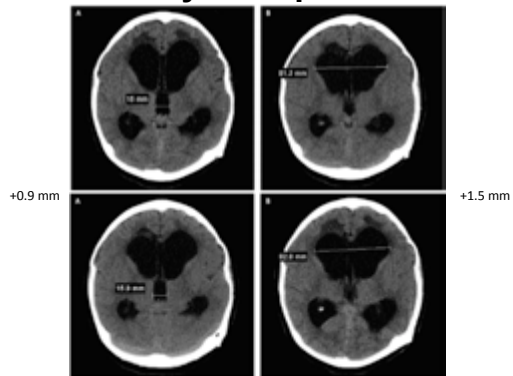
Infra/Supra Tentorial

Above or below the tentorium cerebelli

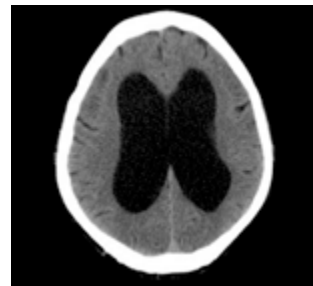


Figures 3-11A-C. Tentorium cerebelli shown in the appropriate location on a skull model (red), and on sagittal (B) and coronal (C) brain images.

Hydrocephalus



Normal Pressure Hydrocephalus

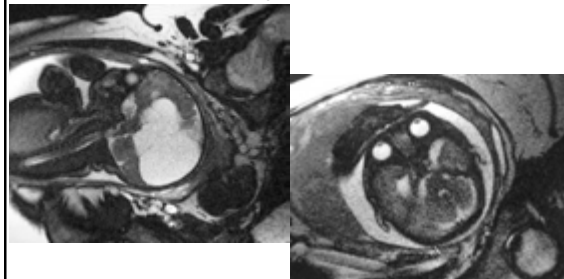


Hydrocephalus Ex-Vacuo

Resulting from loss of brain tissue



Prenatal Hydrocephalus



MRI – Intensity

Hyper – relatively bright

Hypo – relatively dark

Iso – Neutral or same as reference

CT – Density

Hyper – Relatively bright

Hypo – Relatively dark

Equi – Neutral or same as reference

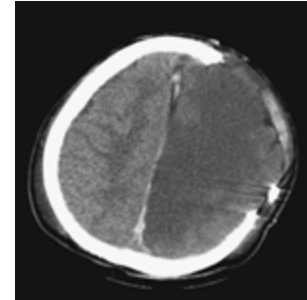
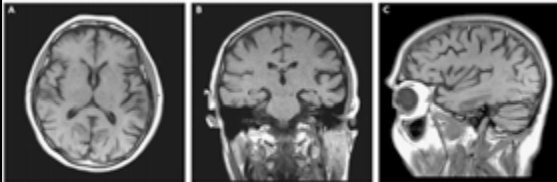
Gray-White Differentiation**Atrophy - Normal Aging**

Figure 2-38A-C Brain scan appearing atrophied, although within normal limits for the 80-year-old patient.

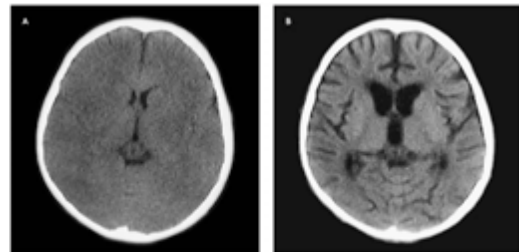
Atrophy - Drowning

Figure 6-42A Acute CT imaging of patient after a near-drowning incident showing diffuse low-density and loss of gray-white differentiation.

Figure 6-42B CT imaging of same patient, taken several weeks later, showing a clearly atrophic appearance due to ongoing diffuse hypoxic ischemic encephalopathy.

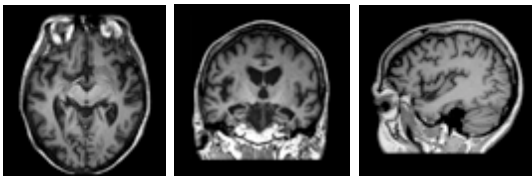
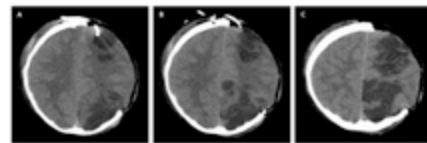
Atrophy – Dementia**Watershed Infarction**

Figure 6-42B-C Coronal CT scans of patient with watershed infarction. A shows normal brain. B shows small area of infarction. C shows larger area of infarction.

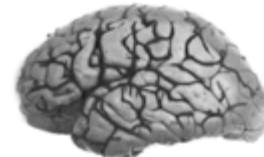


Figure 3-119 Illustration of the watershed area (purple), or the region between the trunks of the middle cerebral artery (red), anterior cerebral artery (orange) and posterior cerebral artery (yellow).

Encephalomalacia

CT

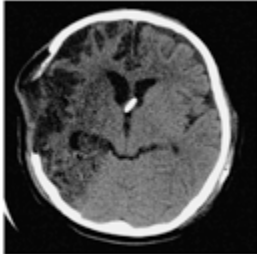


Figure 5-46 Clinical example: encephalomalacia. CT imaging 3 weeks post-injury.

T2 FLAIR

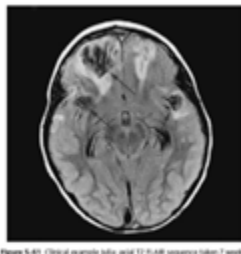
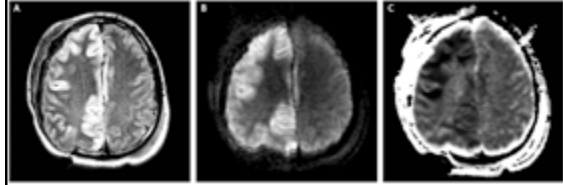


Figure 5-47 Clinical example: encephalomalacia. T2 FLAIR sequence taken 7 weeks post-injury.

Lamina Necrosis



Hypoxic Ischemic Encephalopathy (HIE)

Damage to brain cells due to loss of oxygen.

View the diffusion (DWI and ADC) images for reduced diffusion in the basal ganglia structures and also to a lesser degree the hippocampi, white matter pathways and cortical tissue.

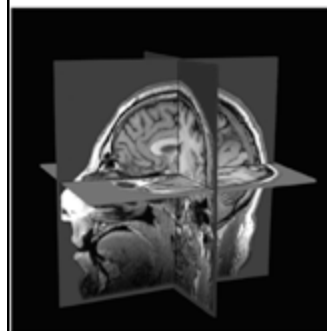
Herniation

Tissue forced across a barrier, usually dura or skull after craniectomy.

Downward herniation of the brain stem into the foramen magnum is life threatening, and a primary risk of increased intracranial pressure or volume.

Basics of Reading Brain Images

Slice orientations
Generally orthogonal, but not always.

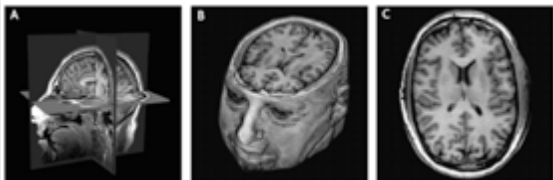


Green: Axial
Red: Coronal
Blue: Sagittal

Axial

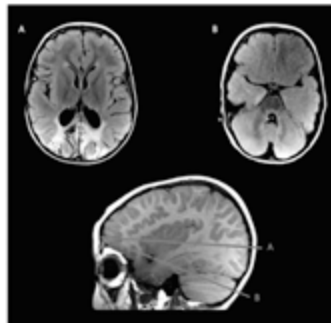
Generally considered the standard slice orientation. Up to only a few of years ago, CTs were only produced in the axial plane.

Remains the most useful for most pathologies.

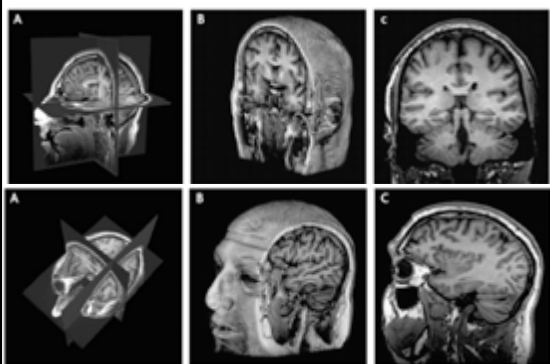


Slice acquisition angle varies...

Anatomical appearance varies with slice angle.

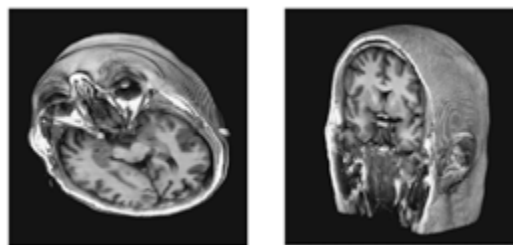


Coronal and Sagittal Slices



??? LEFT IS RIGHT and RIGHT IS LEFT ???

Images are produced from the perspective of the clinician interacting in an inpatient hospital setting.



Neuropsychologists use specific tests to assess specific functions...

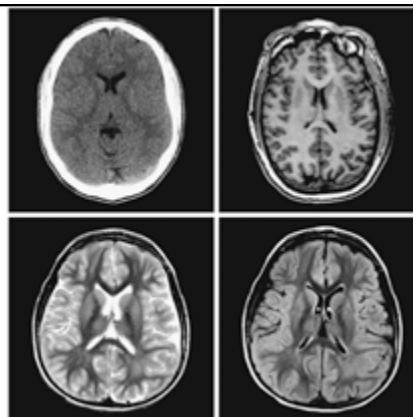
In a similar way...

Use the correct image sequence to answer a specific question.

Anatomy: T1, T1 FLAIR, FSPGR, MPRAGE, BRAVO
Pathology: T2, T2 FLAIR, DWI, SWI, GRE, FIESTA

Small structures are difficult to appreciate on a "pathology" scan, but can be clear on an "anatomy" scan.

Mild pathology may not be visible on an "anatomic" scan, but readily evident on a "pathology" type scan.



How to deal with different scans/ sequences without knowing anything about the different scans/sequences

- Check the shade of the eyes (black/white/gray)
 - Check the shade of the ventricles (black/white/gray)
 - Check the shade of the grey matter
 - Check the shade of the white matter
- Use these as references when assessing for pathology.

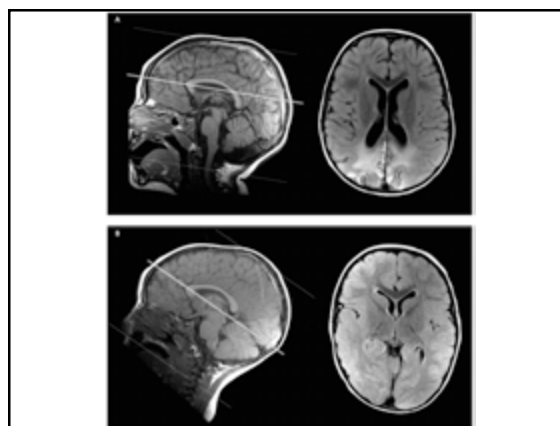
67

Know your anatomy!!!

If you only study one primary landmark,
learn the structure of the ventricles...

They provide wonderful landmarks and are
easily visible on all imaging sequences

**Be sure to study the 3D structure
of the ventricles**



Common imaging techniques

Many can be used with or without
CONTRAST AGENTS

Introduction of a radiopaque material to a CT or MRI can be used
to enhance areas where the blood brain barrier is impaired.

BBB can be impaired by various processes such as blood vessel
damage, neoplasms (tumors), infections, etc.

BBB – Because there are no perforations in the capillaries, certain
substances are prevented from entering the brain. Damage to the
capillaries can disrupt this barrier.

71

CT – Measure of Radiopacity Absorption spectrum of x-rays

Choice for ED/Trauma
Good for blood, bone, edema
No ferromagnetic concerns

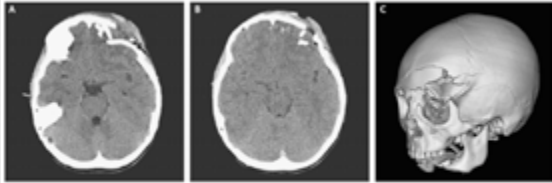
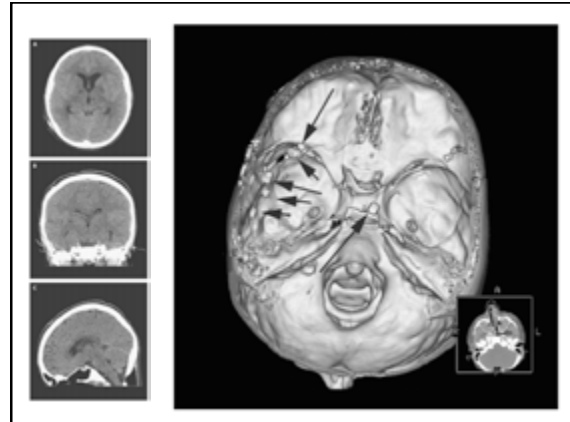
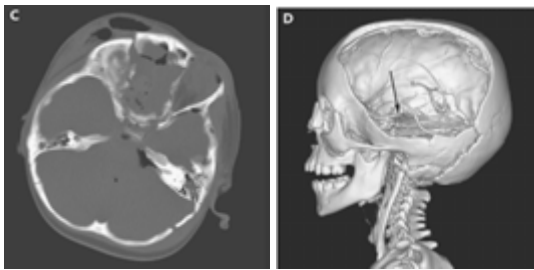


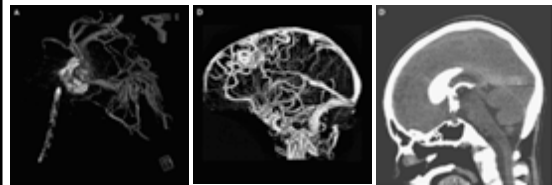
Figure 5-69A-C Clinical example free penetration by bone due to orbital-facial fractures on standard 2D CT imaging (A and B) and 3D CT volume rendering.



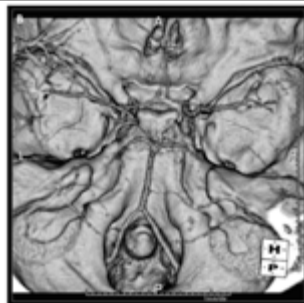
Bone Window
3D MiP



CTA, CTV, Cisternogram



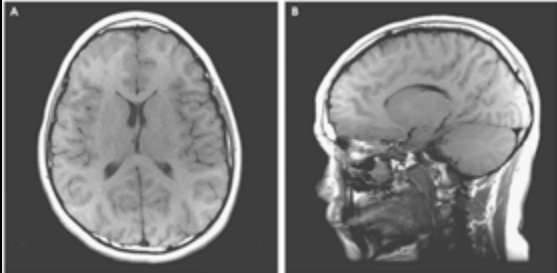
3D CTA with
MiP Bone
Rendering



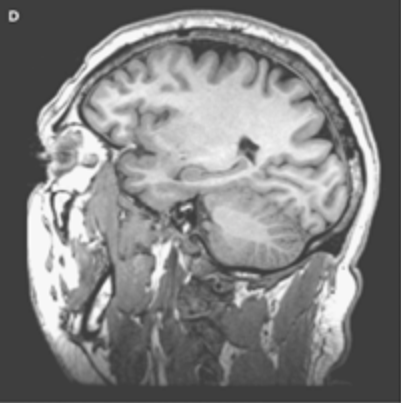
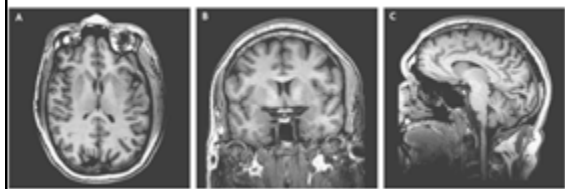
Figures 3-115A and B Circle of Willis shown using time-of-flight sequence (ECOW TCF) in Figure 3-115A and three dimensional CT vessel reconstruction in Figure 3-115B. A = Internal Carotid Artery; B = Middle Cerebral Artery; C = Anterior Cerebral Artery; D = Anterior Communicating Artery; E = Vertebral Artery; F = Basilar Artery; G = Posterior Cerebral Artery; H = Posterior Communicating Artery.

MRI

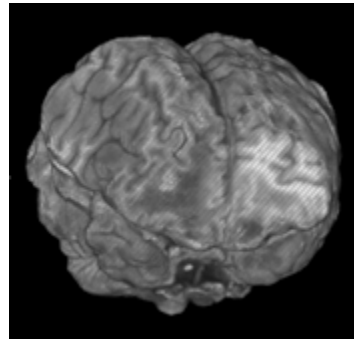
2D T1 acquired in axial and sagittal orientations



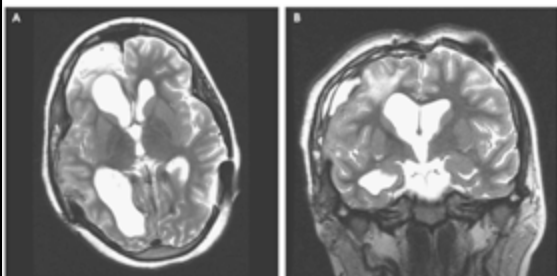
3D acquired T1 FSPGR - BRAVO



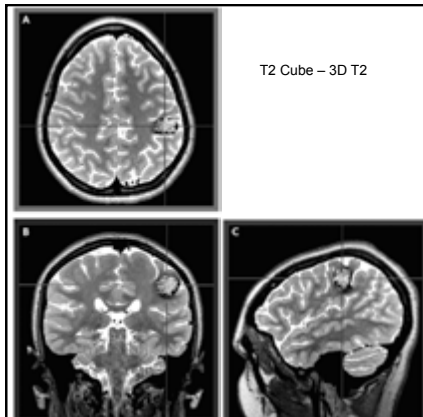
3D MIP reconstruction



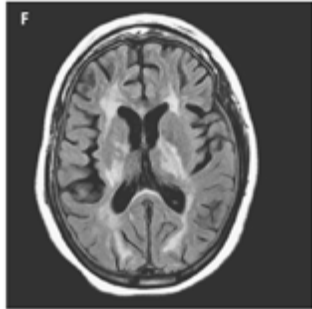
2D T2 acquired in axial and coronal orientations



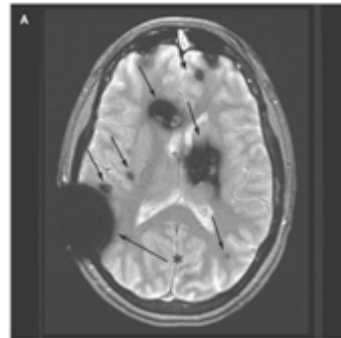
T2 Cube - 3D T2



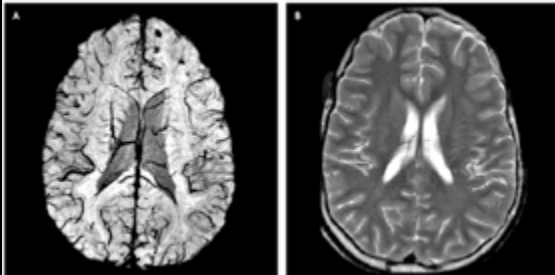
T2 Fluid Attenuated Inversion Recovery (FLAIR)



Gradient Echo (GRE) or T2*

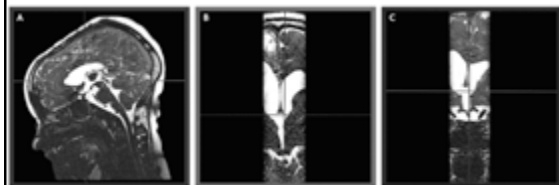
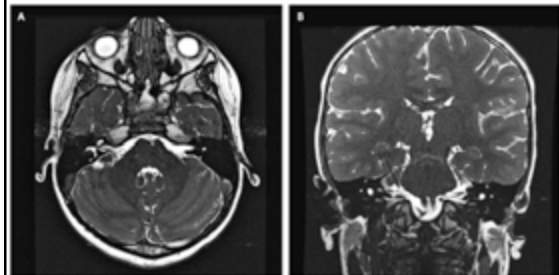


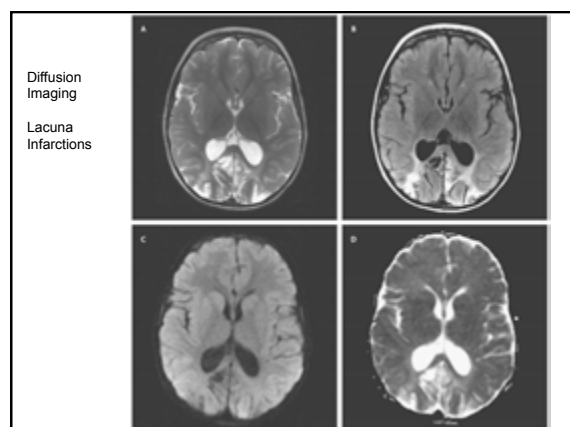
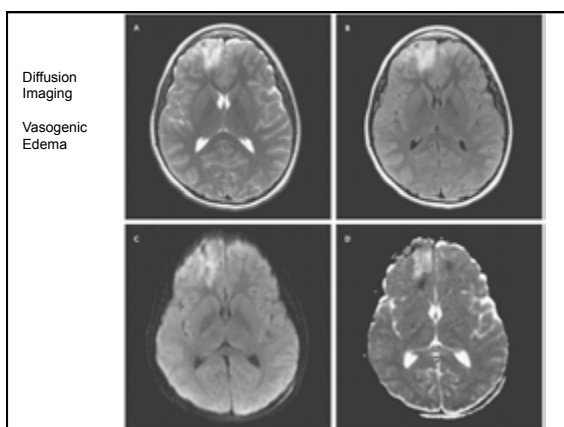
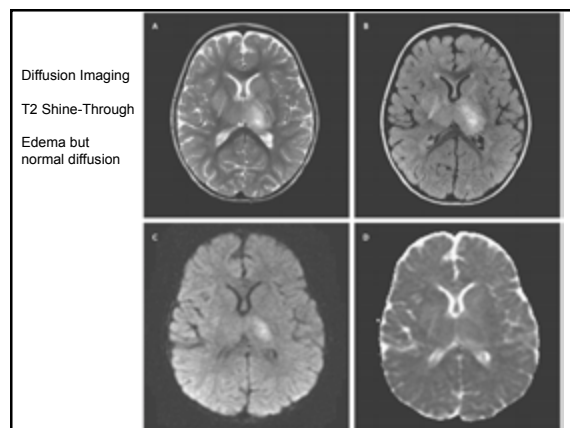
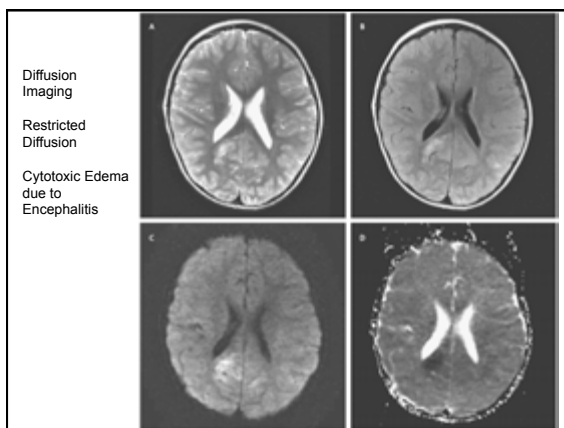
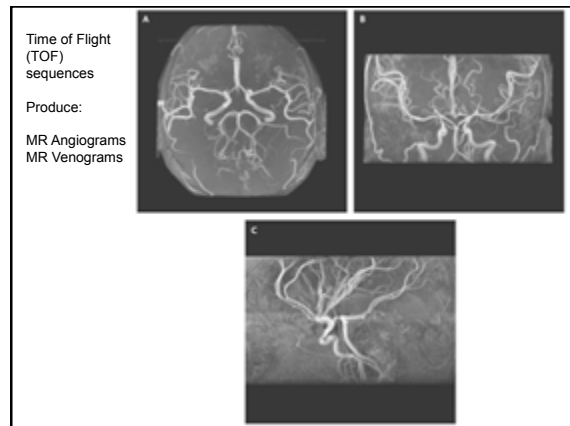
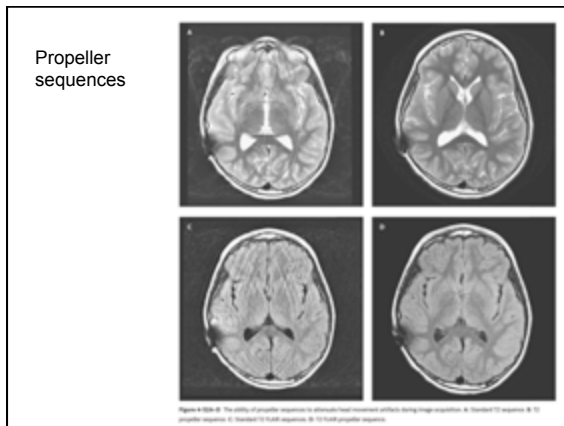
Susceptibility Weighted Images (SWI)



Courtesy of E. Mark Haacke, Ph.D.

Fast Imaging Employing Steady State Acquisition (FIESTA)



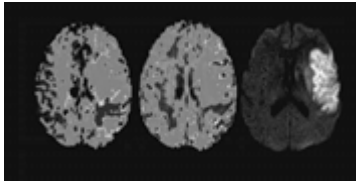


Diffusion-perfusion penumbra

RED: Regions of decreased perfusion on MTT map

BLUE: Regions of increased cerebral blood volume

GREY: DWI – poor diffusion.



The reduced perfusion area is larger than the area of infarction (diffusion-weighted).

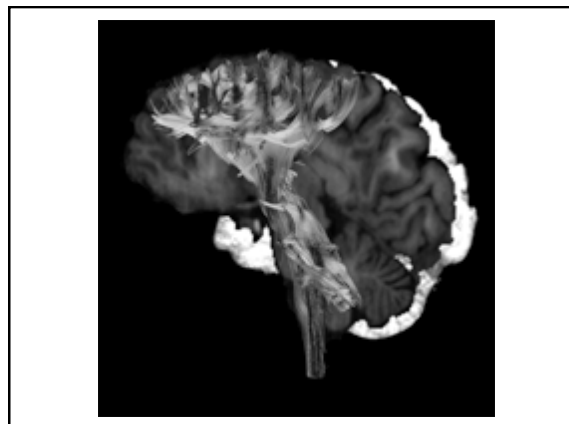
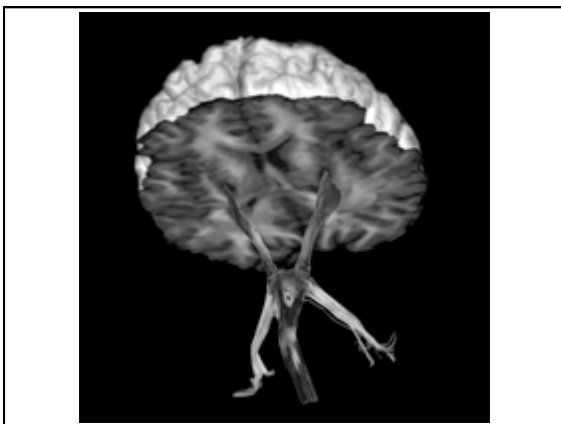
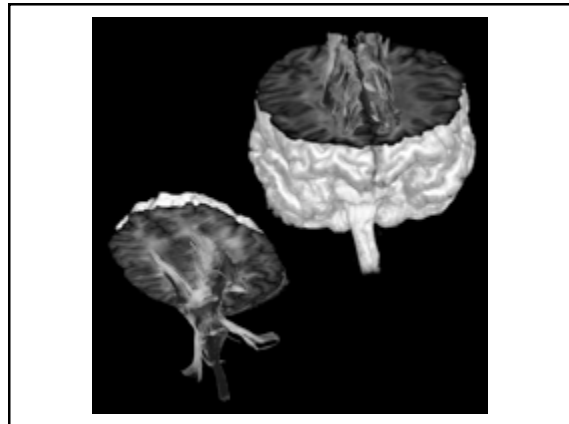
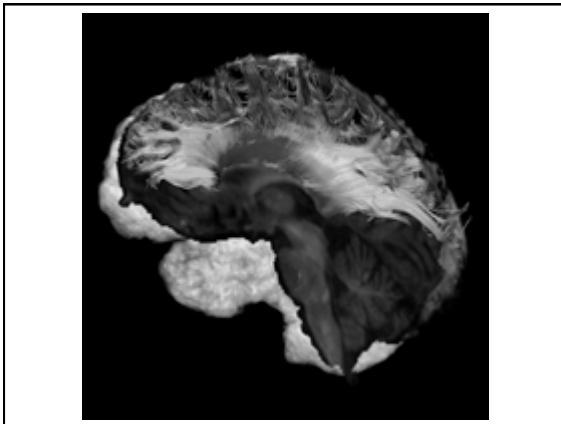
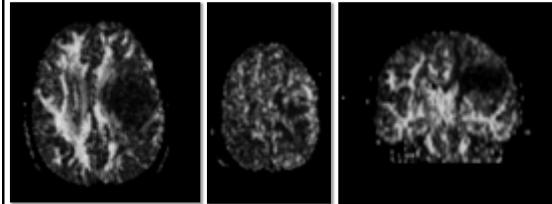
Areas of low perfusion *without* low diffusion, suggests regions that may be rescued if blood perfusion is restored (potentially salvageable brain tissue).

Diffusion Tensor Imaging FA Maps

RED = Lateral Fibers (across the brain)

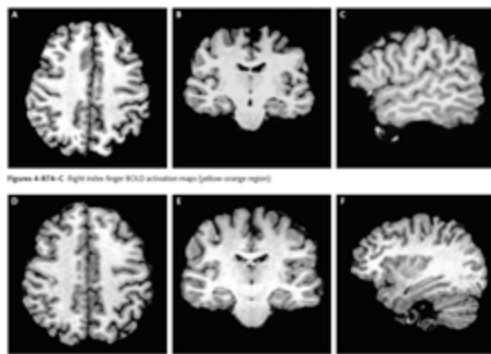
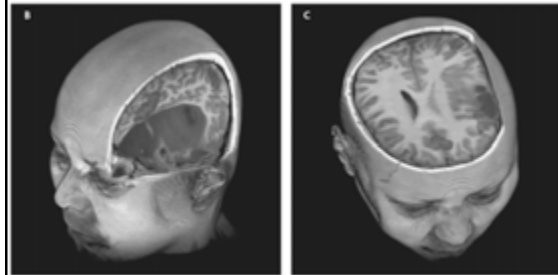
BLUE = Superior-Inferior or Rostral-Caudal

Green = Anterior-Posterior



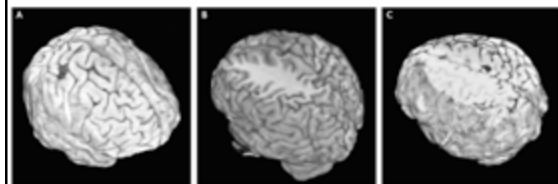
fMRI

fMRI showing displacement of eloquent cortex over the many years the cyst has been forming.



Figures 4-87B-C: Right index finger BOLD activation maps (yellow-orange regions).

Figures 4-87D-F: Left index finger BOLD activation maps (yellow-orange regions).



Figures 4-88A-C: A: Left hand sensory BOLD activation map (red area). B: Right index finger motor BOLD activation map (red area). C: Left foot motor BOLD activation map (red area).

MR Spectroscopy

At specific frequencies, resonant peaks are identified from the presence of specific metabolites in the brain.

Choline - Membrane synthesis and turnover

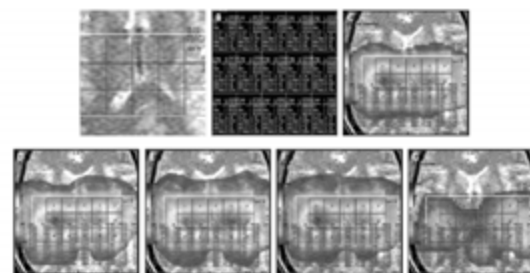
Creatine - Energy requirements of the cell

N-Acetylaspartate (NAA) - Marker of healthy neurons

Lactate - Anaerobic metabolite not found in healthy brain, but in ischemic tissue.

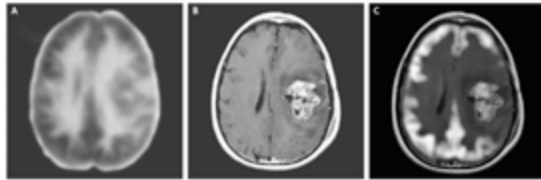
Lipids - Not seen in healthy brain, but in necrotic tissue.

NAA MRS may assist in dx of concussion and mTBI as levels are linearly related to electrochemical changes after injury.



Figures 4-89A-C: Clinical example: Coronal MRI of a high-grade astrocytoma (glioblastoma multiforme) and normal tissue around the tumor. A: Spectrum map. B: Spectrum with data. C: Composite map. D: N-acetyl map. E: Choline map. F: Creatine map. G: Lactate map.

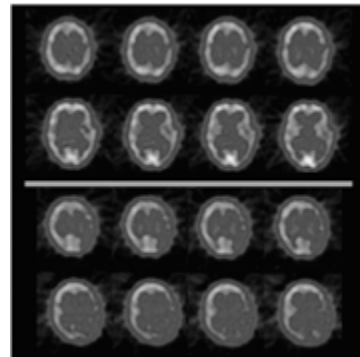
PET – MRI – PET+MRI



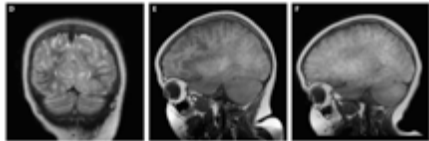
Figures 4-82A-C: PET, MRI, and PET+MRI acquired from a patient with a left parietal tumor. A: PET. B: (T1) MRI. C: PET+MRI (brighter orange/yellow signifies greater metabolic activity).

Brian Jefferson, MD

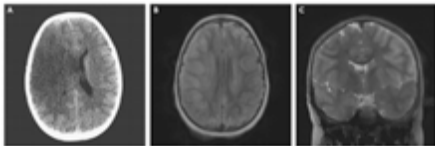
Single Photon Emission Computed Tomography (SPECT)



SPECT+MRI



Figures 4-82B-F: SPECT+MRI fusion images within the right internal carotid, middle cerebral and anterior cerebral distributions. The image A (SPECT) shows the degree of SPECT activity for areas colored purple, greater for areas colored orange, and highest in areas that are yellow. B: Posterior coronal SPECT+MRI. C: Sagittal SPECT+MRI right hemisphere. D: Sagittal SPECT+MRI left hemisphere.

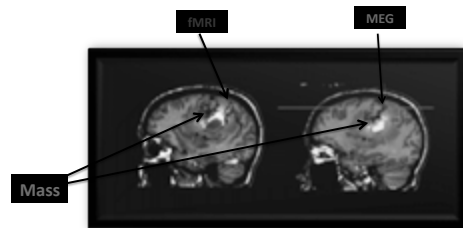


Figures 4-82G-I: CT scan showing hyperdensity of blood within the right internal carotid and middle cerebral artery distributions. SPECT+MRI fusion images within the right internal carotid, middle cerebral and anterior cerebral distributions. For images A-B and C, the brightness or saturation of the purple corresponds to the extent of SPECT, with a brighter appearance corresponding to more flow. B: Axial SPECT+MRI. C: Coronal SPECT+MRI.

fMRI co-registered with MEG-MSI

fMRI study showing activation secondary to finger tapping in a patient with a large mass.

MEG study showing the location of the magnetic dipole secondary to finger movement in the same patient.



Brian Jefferson, MD

Interpretation of brain pathology

Radiology is nothing like neuropsychology

Radiology is exactly like neuropsychology

Bad radiologists makes the same mistakes involving interpretation as do bad neuropsychologists...

Great professionals of all clinical fields consistently follow the same golden rules.

***"We see what we look for,
and we recognize what we know."***

Merrill C. Sosman, M.D. (1890–1959)

"Absence of evidence is not evidence of absence!"

Carl Sagan, Astronomer

"Explanation does not confirm etiology"

Lebby

Anatomy is NOT function

**A brain scan is just a picture of an engine,
it is not the test drive...**

Your examination is the test drive!

Abnormal anatomical appearance with normal functioning
Normal anatomical appearance with abnormal functioning.

116

JD

Highly abnormal presentation upon admit

- Functional Basic Language (receptive and expressive)
- Impaired level of consciousness
- Impaired orientation, insight, awareness of deficits
- Impaired Judgment
- Impaired social-Interpersonal functioning
- Impaired Attention (passive, complex, divided)
- Impaired Verbal and Visual Reasoning
- Impaired Verbal and Visual Memory
- Impaired ADL's

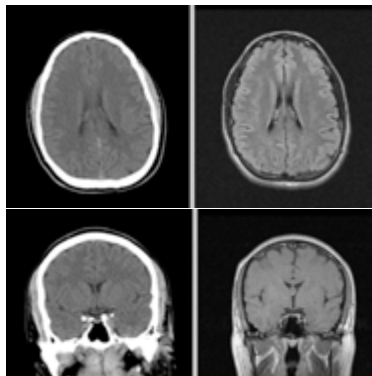
Normal CT and MRI

Normal presentation on 6-month follow-up

- Intact Basic Language (receptive and expressive)
- A+Ox4 with intact judgment
- Intact attentional abilities
- Intact Executive Functioning (ss 09-14)
- Intact Verbal and Visual Reasoning (VIQ = 108, PIQ = 115)
- Intact Verbal and Visual Memory (ss 11 – 15)
- Intact ADL's

Anti-N-methyl-D-aspartate receptor antibody encephalitis
A-NMDAR

NORMAL NEUROIMAGING



I've never been the same since that last hit... Something is not right!

- History of multiple concussions playing HS and college football
- Consistently flat affect, apathy and abulia
- Dropped out of college, never returned.

Intellect (VCI, PRI, FSIQ) superior range (> 90th %ile)

Academics in the superior range (> 90th %ile)

Executive Functioning < 1st to 2nd %ile

Complex Attention < 1st to 5th %ile

Anterograde memory < 1st to 5th %ile

Brain scans image “trees” NOT “leaves.”

They are more useful for larger anatomical structures, pathways or systems than for microscopic structures

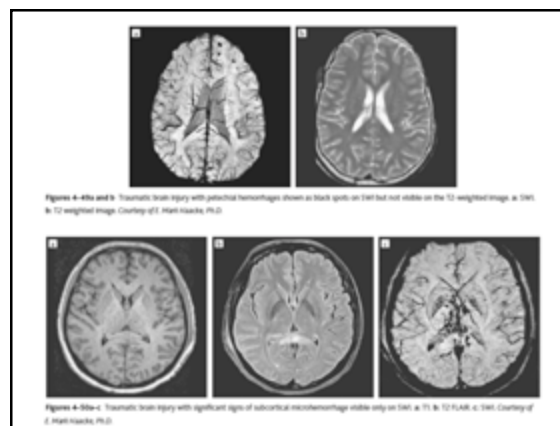
Just because you don't see damage on a brain image does not mean there was no damage...

WERE THERE NEURONS BEFORE Santiago Ramón y Cajal and Camillo Golgi?



CT vs MRI T1 vs T2 vs T2 FLAIR vs GRE-EPI vs SWI

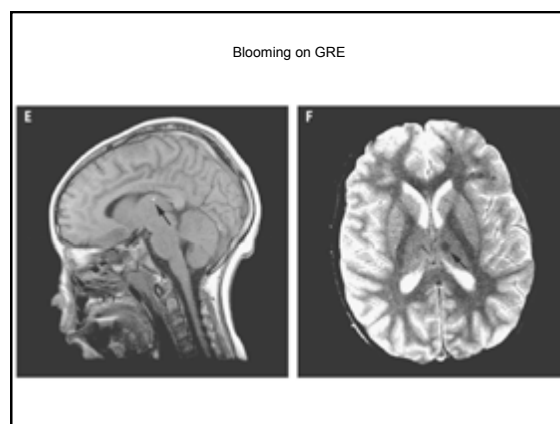
The method of analysis can determine the clinician's ability to detect neuropathology following injury to the brain.



Even with pathology evident on a
brain scan...

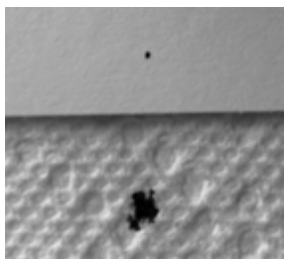
What you see is NOT what you get!

For example, markers of microscopic damage such as microbleeds are often clear, but can be misleading.



For example, micro-hemorrhages are smaller than they appear on an image due to a variety of factors, including the resolving power of the scan, or the way the the scan acquires the image.

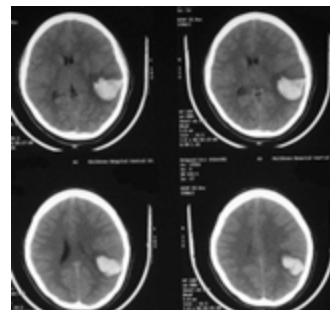
For example, the blooming effect of GRE or SWI sequences is caused by magnetic field susceptibility interference (artifact) and is like writing a period with a fountain pen on copy paper versus a paper towel.



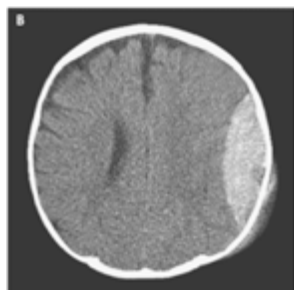
Larger hemorrhages with extensive edema can “Mask” underlying tissue integrity and can be misleading.

Left parietal stroke from MCA hemorrhage

You would expect Wernicke's type aphasia, although there was no evidence of aphasia in this patient or other cognitive or functional compromise.



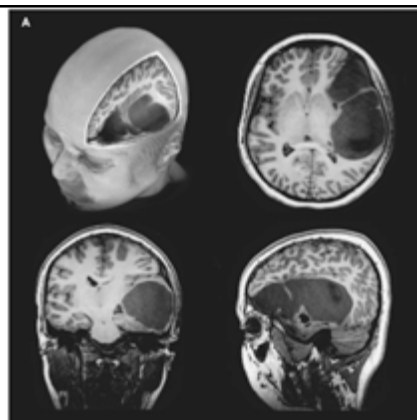
Epidural bleeds can look bad but there may be no significant damage to brain tissue



Slow growing benign neuroglial cyst.

Some fluid was removed due to minimal midline shift and bowing of the falx likely resulting from her concussive/mTBI event precipitating examination.

NPSY testing Ave to High Ave in all domains assessed

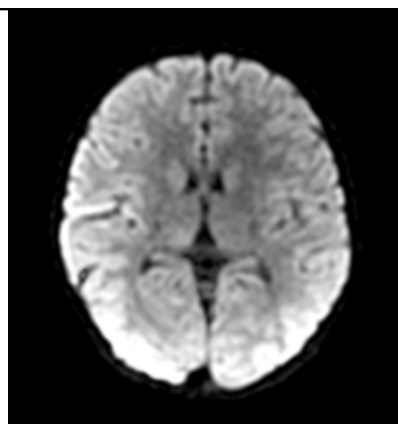


Artifacts can result in a pathological appearance, without any damage or dysfunction to the brain.

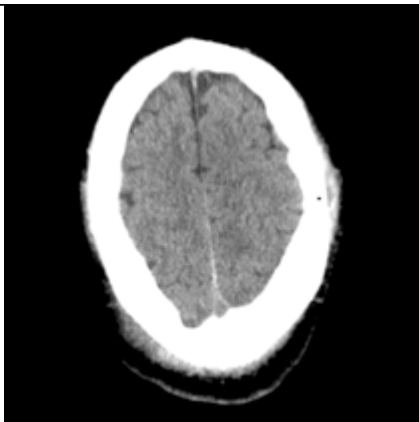
On diffusion imaging (DWI) restricted diffusion is shown as bright regions on the brain.

BUT

Bright regions are also produced by T2 prolongation, and artifacts caused by the bone/air and posterior coils closer to the brain parenchyma than anterior coils



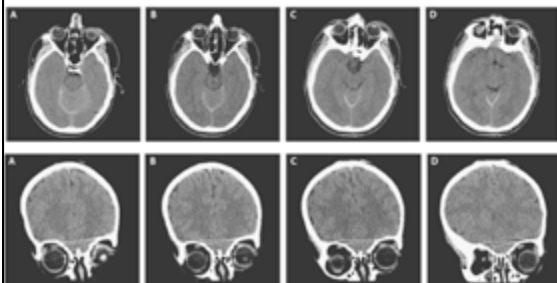
As the patient lays in the scanner, the brain rests against the back of the skull producing a false appearance of atrophy in the superior frontal region.



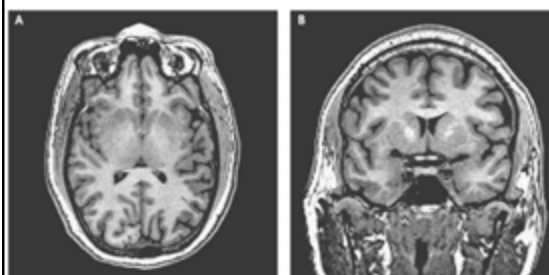
Use symmetry as a guide...

... be cautious

Check asymmetry by viewing the eyes/symmetric structures.

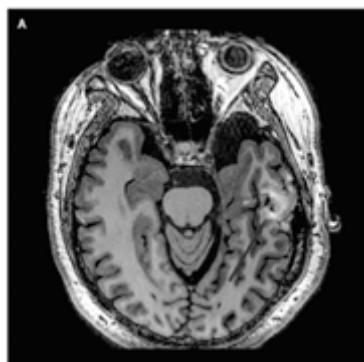


Symmetrical structures



Asymmetry:

But caused by head position and artifact resulting from tissue transection



Compare areas of concern to other regions of known appearance to determine if the region of interest (ROI) is similar or different in appearance.

BUT YOU MUST ALSO determine why different:

Could be artifact

Head position in the scanner

Differences are relative:

An area appearing more wrinkled (atrophic) may be normal if compared to a slightly edematous other region.

An area appearing edematous may be normal if compared to an area of atrophy.

Don't become distracted from critical but subtle pathological signs by salient features of little or no clinical relevance.

Asymmetry from benign arachnoid cyst distracts from the real damage caused by a TBI.



Normal anatomical variability

There is an enormous variance in the size, shape and general appearance of brain structures across individuals.

Do not use your confirmatory bias to over-interpret normal variance in a particular brain structure just because it fits with your clinical impressions.

There is a trend for brain injury clinics to over-interpret "thinning" of the corpus callosum as evidence of diffuse axonal injury.

But, there are NO STANDARDS regarding the size, volume and shape of the structure.

Unless you have premorbid scans, this should only be performed with extreme caution.



Figure 3-27 Schematic of normal corpus callosum structures imaged from a sagittal orientation.

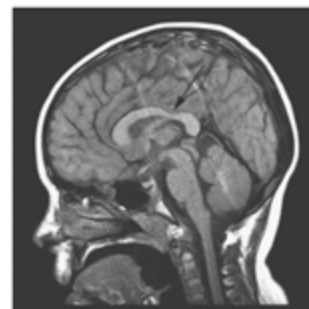
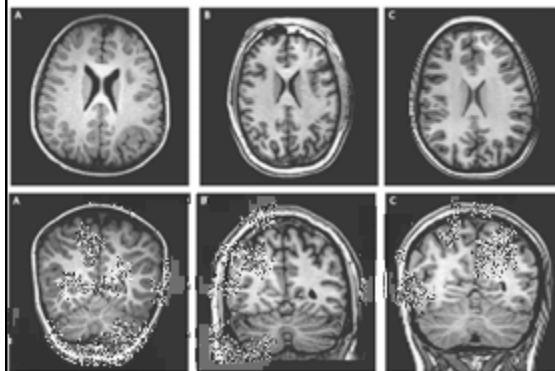


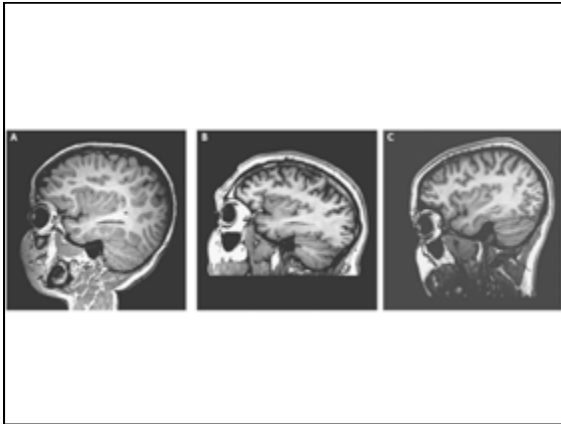
Figure 3-28 Schematic of the corpus callosum.

There is also a trend to use DTI FA maps and tractography to assess white matter damage.

However, these are diffusion techniques and edema or inflammatory processes can appear as lost tissue (atrophy). Again, be cautious, there are no standards for this.

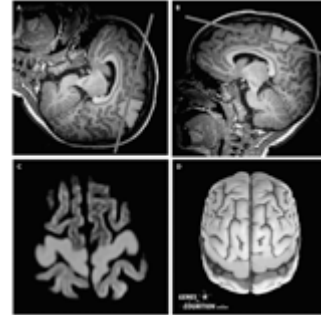
Cortical variation on imaging





Know where structures will appear on a brain scan...

This is different from how structures appear in a text book



You have to assess collateral information over time

In medicine, things change over time, and that is considered normal in many cases.

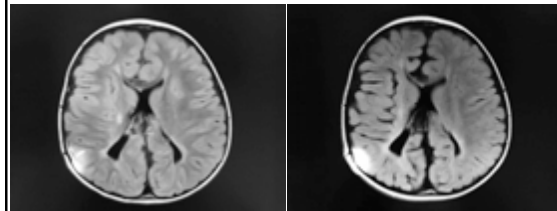
Not unlike the fluctuations in performance from morning to afternoon, from day to day, etc. in recovery from TBI

The same is true of medical data, values, and even brain images.

Pediatrician reported to mother that child appears to have a neurodegenerative condition based on brain atrophy over time. However, her functioning was improving over time, just not at the expected rate (she was falling further behind her peers).

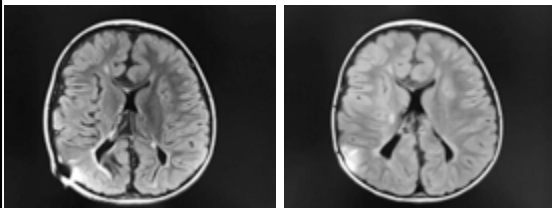
A

B



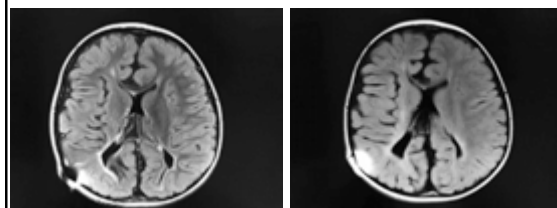
< A

A



< A

B



Understand that brain pathology is dynamic, and imaging findings change over time

It is critical to know how long has passed since a particular pathological process has taken place.

Concussion? or Not Concussion?

Confusion
Memory loss
Dizziness
Headache

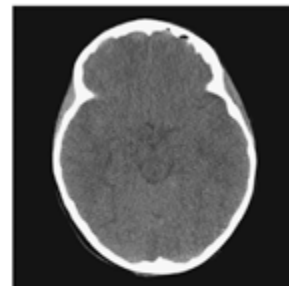


Figure 5-58 Clinical example Julia initial CT scan of traumatic brain injury.
250 | Brain Imaging

Patient diagnosed with a concussion

I was asked to see the patient for protracted Sx. Her presentation suggested significant bi-frontal and temporal lobe injury and so I requested follow-up imaging.

Two days post “Concussion.”

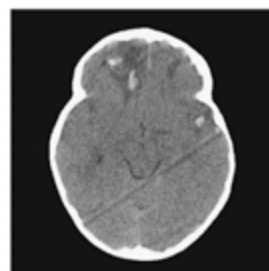


Figure 5-59 Clinical example Julia CT scan of traumatic brain injury taken 2 days post-injury.

**MRI T2 FLAIR
2 Weeks (left) and 7 Weeks Post
“Concussion”**

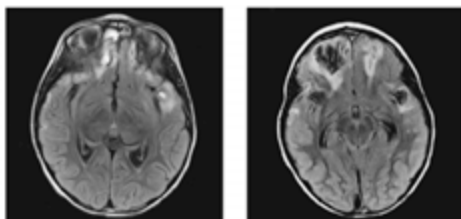


Figure 5-60 Clinical example Julia axial T2 FLAIR sequence taken 2 weeks post-injury. Figure 5-61 Clinical example Julia axial T2 FLAIR sequence taken 7 weeks post-injury.

Pearl

Brain Injury Is Dynamic – Waiting to formulate prognosis can be beneficial

Peril

The sooner you attempt prognosis, the less accurate you will be.

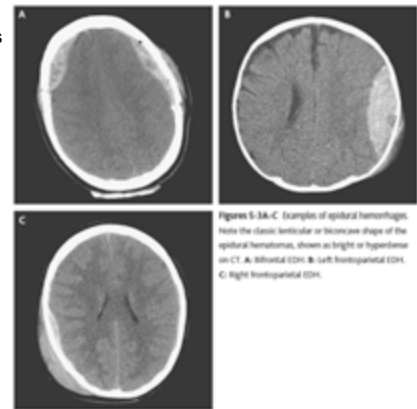
What you see in the ICU or on initial imaging may be different from what you see later

**Know how different types of pathology appear
on the primary types of sequences**

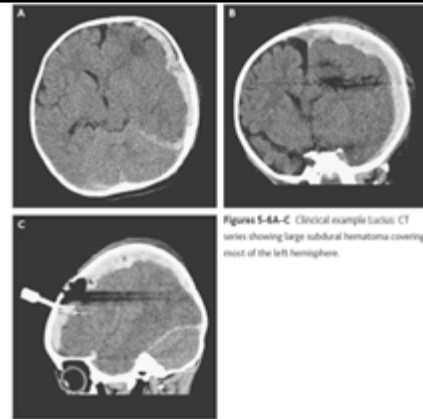
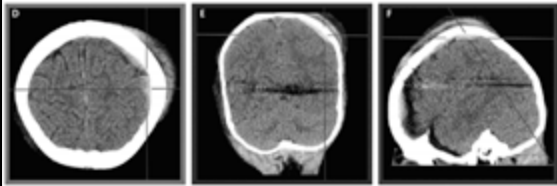
A PARTIAL SUMMARY

Epidural bleeds

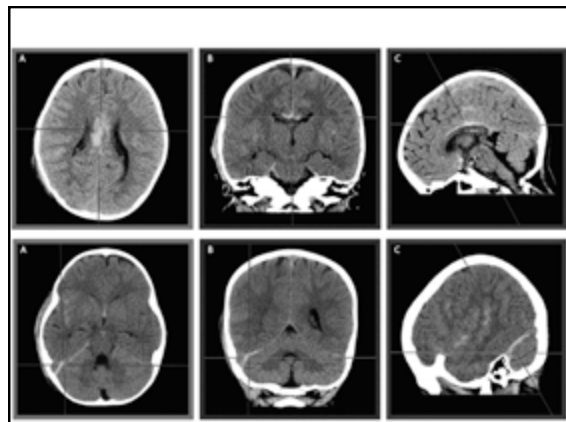
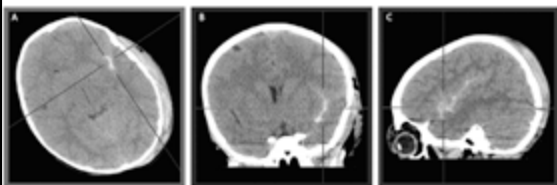
Lenticular
shape

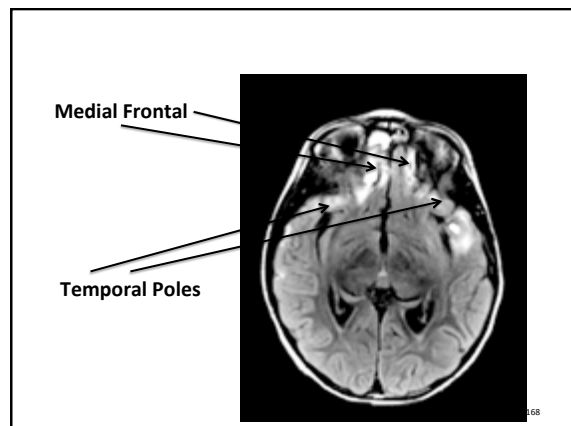
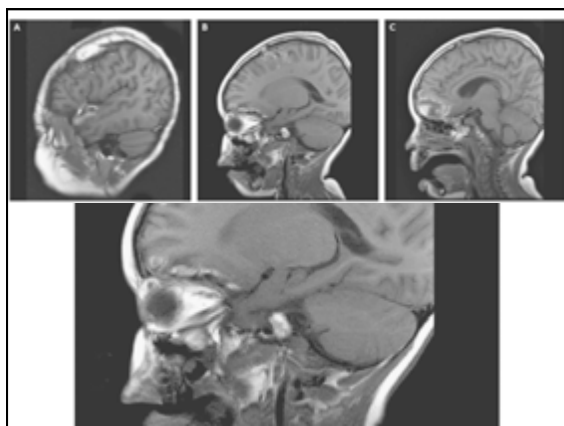
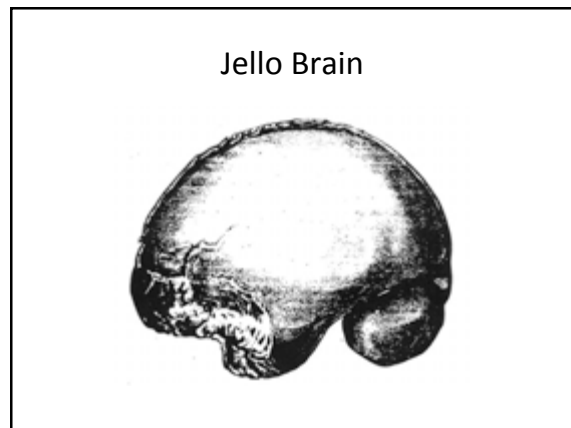
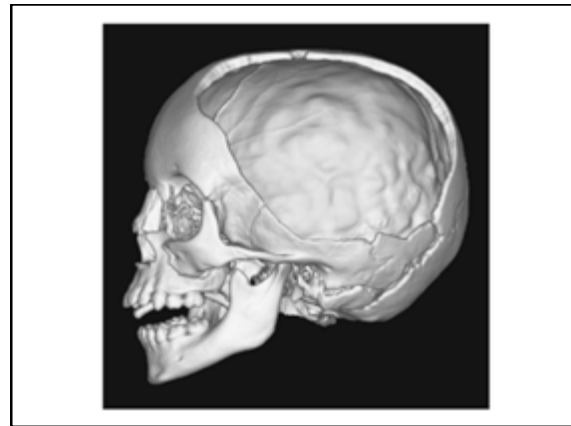
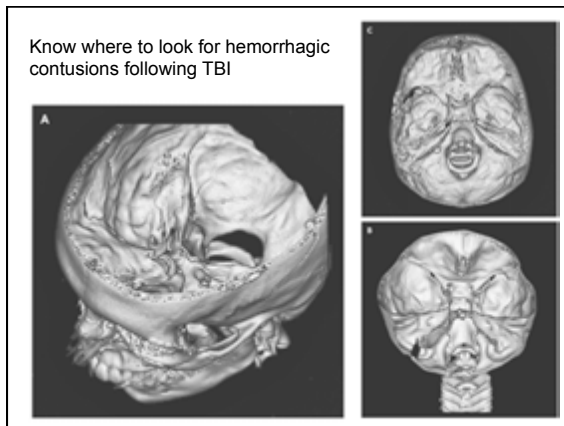


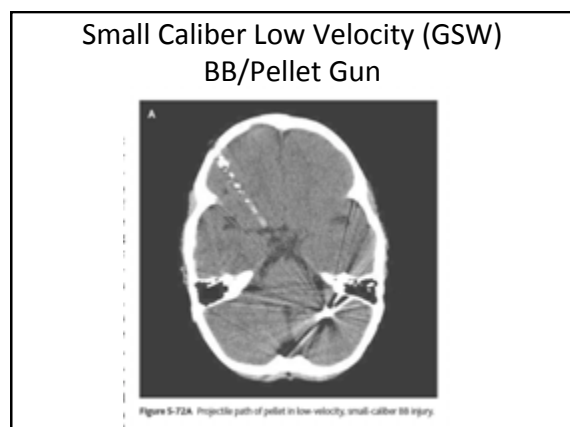
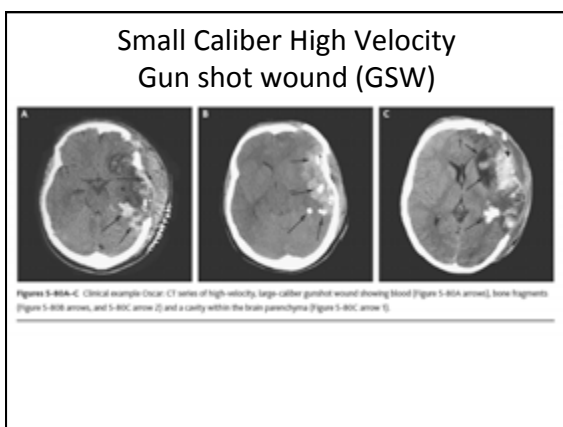
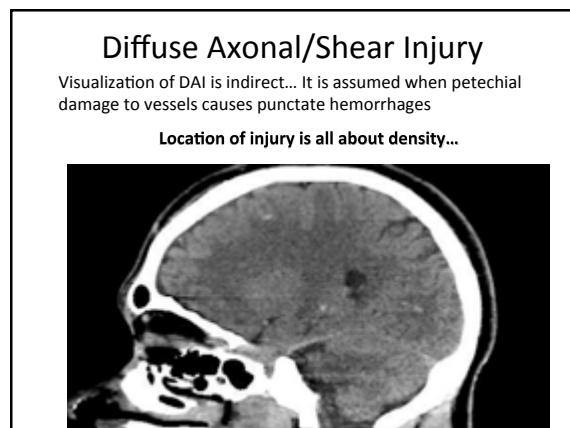
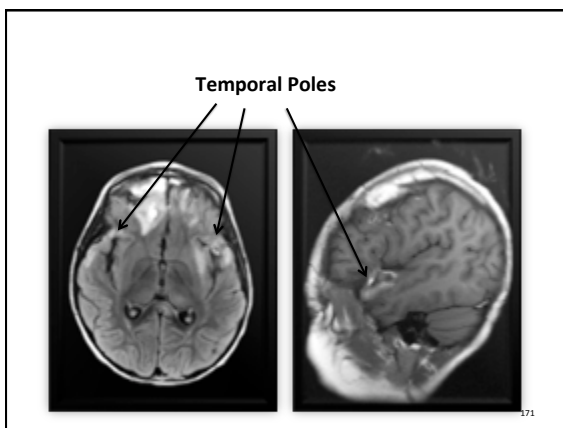
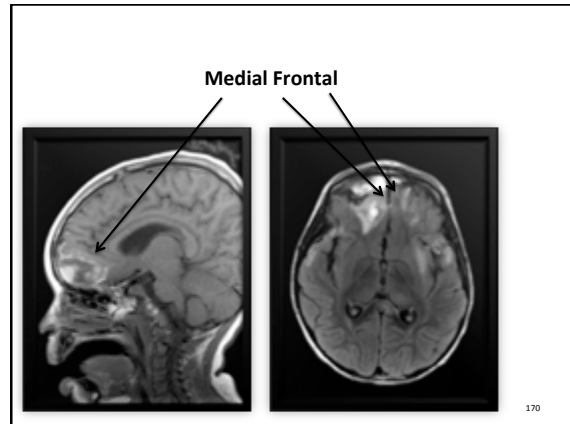
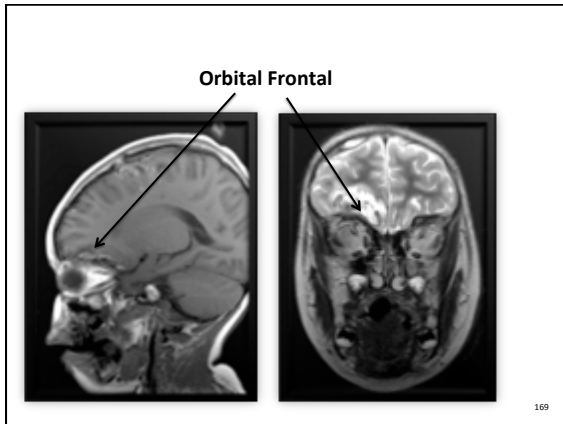
Subdural bleeds – follow the contours of the cortex



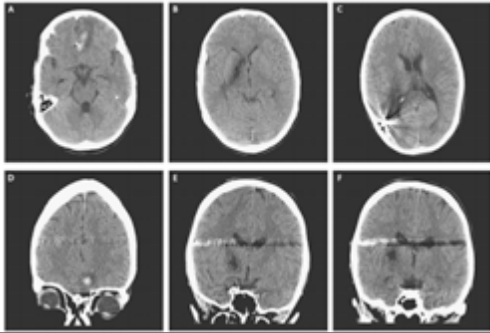
Subarachnoid bleeds



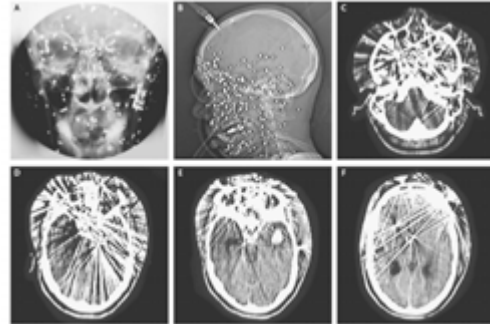




Large Caliber Low Velocity GSW



Shotgun Wound



Figures 5-416-F Clinical example (Steven): shotgun injury to the face and brain on X-ray (A), CT bone window (B) and CT axial series (C-F).

Skull fractures do not predict the degree of brain injury, and for some patients may provide a protective factor for the brain.

Think Crumple Zone on a Vehicle.

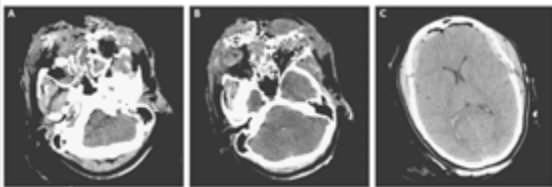


Figure 5-37A-C Clinical example Susan: CT series illustrating the crushing injury to Susan's face.



Figures 5-38A-C Clinical example Susan: Post-facial reconstruction 2D CT and 3D CT volume rendering.

Contrecoup

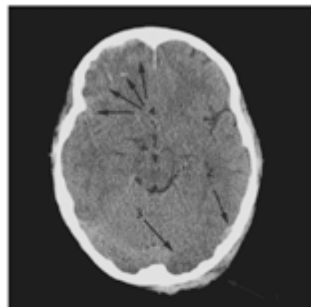


Figure 5-22 Clinical example Kathryn: contrecoup injury. Imaging taken on the day of the injury. Arrow 1 shows the area of direct impact. Arrow 2 shows a small extra-axial left temporal-parietal hemorrhage. Arrow 3 shows some right occipital low density from the direct impact (coup), and arrows 4 shows the right frontal and temporal injuries caused by the contrecoup.

Laminar necrosis due to HIE (Top) and Compression (Bottom)

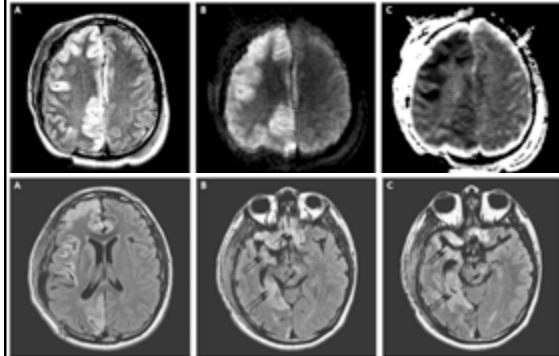
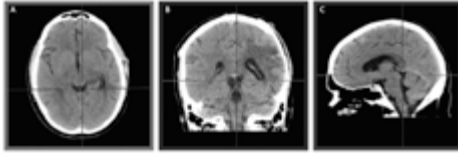
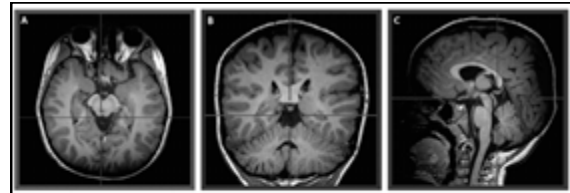
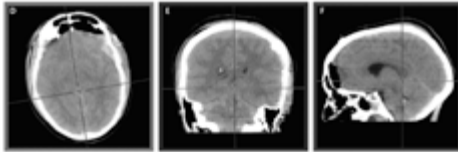


Figure 5-45A-C MRI T2 FLAIR axial series showing extensive cortical laminar necrosis (arrows), resulting from compression of the right hemisphere cortex and medial left hemisphere subcortical and cortical structures due to swelling of the right hemisphere.

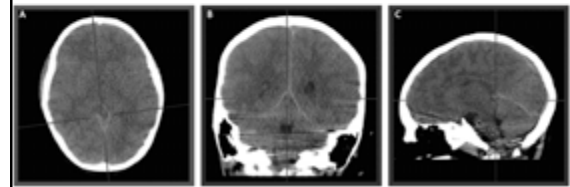
Diffuse edema (swelling) evident by closure of the dural cisterns



Figures 5-54A-C: Open ventricles and cisterns. Note open perimesencephalic or quadrigeminal cistern identified by coronals.

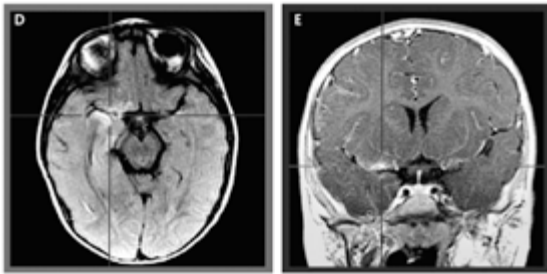


Figures 5-15A-C: Axial, coronal, and sagittal MRI at the level of the superior cistern.

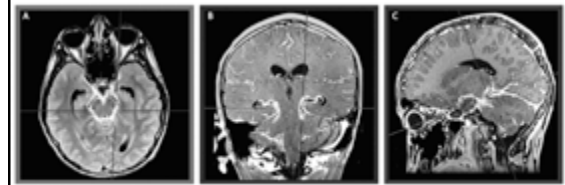


Figures 5-15D-F: Compressed superior perimesencephalic/quadrigeminal cisterns on CT.

Meningitis

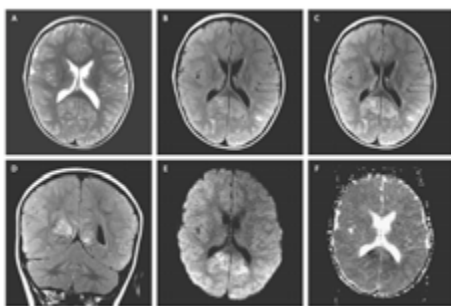


Severe meningeal inflammation



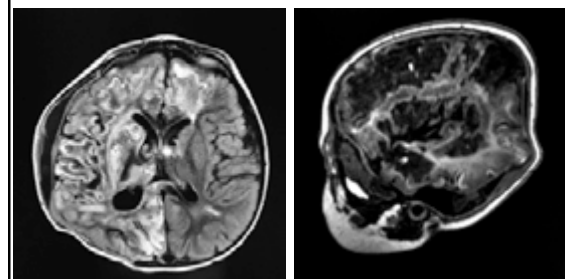
Figures 6-12A-C: Clinical example Tyler: contrast-enhanced T2 and T1 images acquired in mid-November, showing prominent meningeal inflammation. A: CE T2 FLAIR. B: CE T1. C: CE T1-FSPGR.

Encephalitis



Figures 6-16A-F: Clinical example Tyler: MRI scans acquired shortly after Tyler was hospitalized with encephalitis. A: T2. B: T2 FLAIR. C: Contrast-enhanced T2 FLAIR. D: Contrast-enhanced T2 FLAIR. E: DWI. F: ADC.

Fulminating Necrotizing Encephalitis from acute seasonal influenza



Abscess

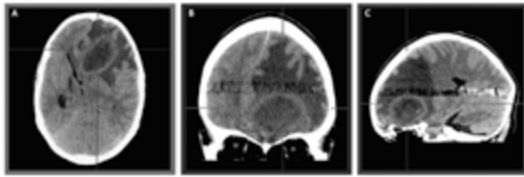


Figure 5-75A-C Clinical example Argemiro CT series showing Argemiro's frontal abscess.

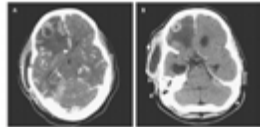


Figure 5-75B-E Clinical example Argemiro CT series showing Argemiro's frontal abscess.

Empyema

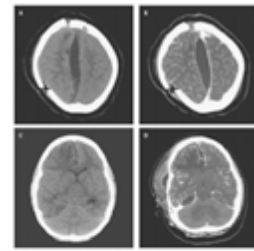


Figure 5-75A-D Clinical example Argemiro CT series showing Argemiro's frontal abscess.

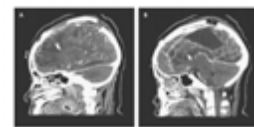
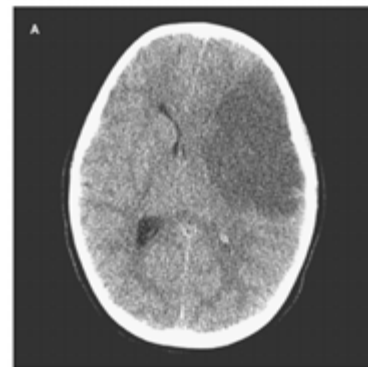
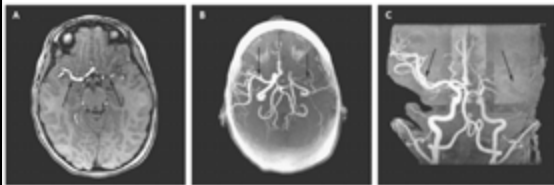


Figure 5-75E-F Clinical example Argemiro CT series showing Argemiro's frontal abscess.

Stroke - embolic



Stroke – Embolic (top) and hemorrhagic (bottom)

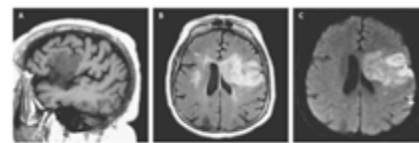


Figure 5-75A-C Clinical example Argemiro CT series showing Argemiro's frontal abscess.

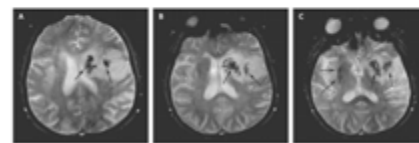
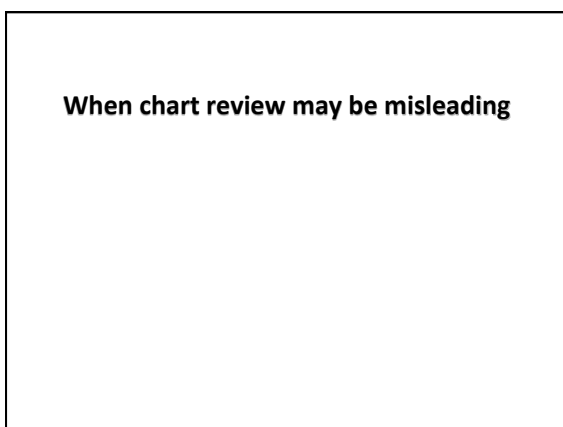
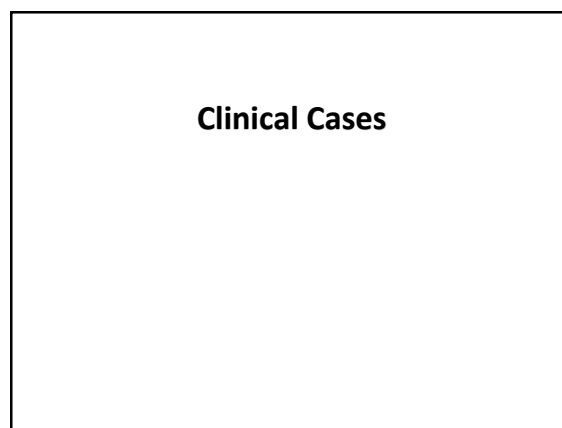
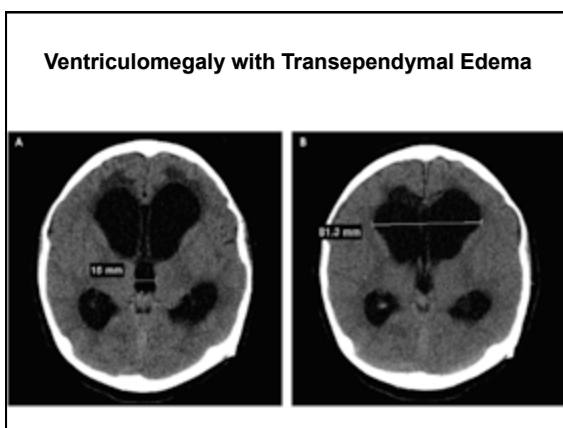
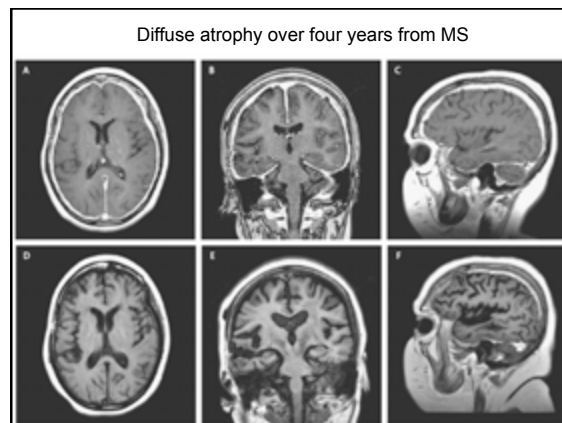
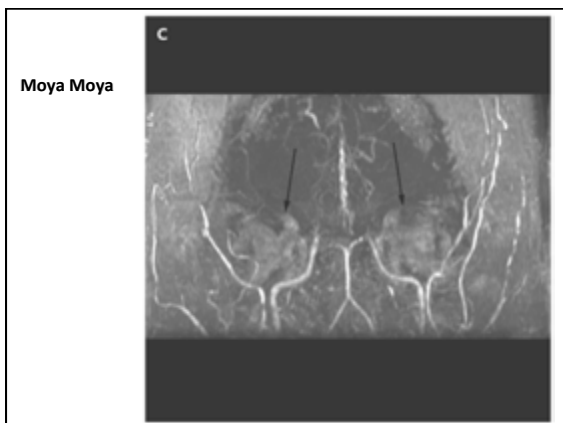


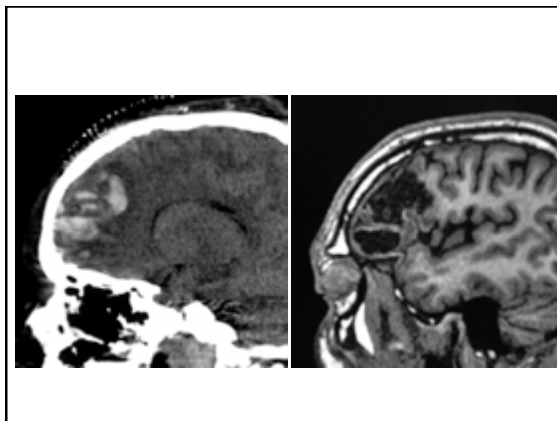
Figure 5-75D-F Clinical example Argemiro CT series showing Argemiro's frontal abscess.



- Chart review suggested bi-frontal contusions, with prognosis for recovery described as “good.”
 - Infant was functioning normally after only a few days of hospitalization, and being discharged.
 - Normal neurological examination.
 - Medically unremarkable.
 - Referred for follow-up with PCP.
 - No medications required post discharge.

As an adult, 22 years later

- Because injured as an infant, parents told she would fully recover. Based on chart review, one may expect relatively normal functioning and not do extensive assessment of higher-level abilities.
- However...
 - Low average verbal-linguistic reasoning (deficient verbal abstraction – concepts)
 - Average visual-spatial reasoning
 - **Severely impaired executive functioning (2-min for first line of CW-LS – on DKEFS)**
 - **Moderately impaired complex and divided attention**
 - Pleasant, just functions at a concrete (stimulus/category/feature) level.



- Chart review suggested:
 - Minimal developmental issues, with *“A small amount of fluid outside of the brain that does not require any medical treatment.”*
 - *Referred for behavioral and emotional difficulties.*
 - *Full cognitive assessment did not appear indicated, although revealed significant cognitive deficits.*

After pulling the older charts and imaging from storage, CT findings were quite marked. Follow-up MRI revealed severe cortical dysplasia and abnormal development of much of the brain.

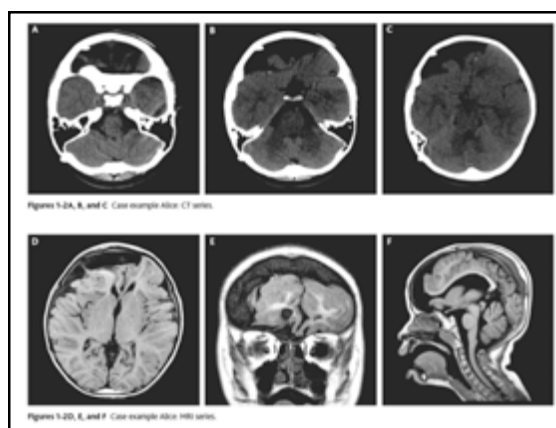
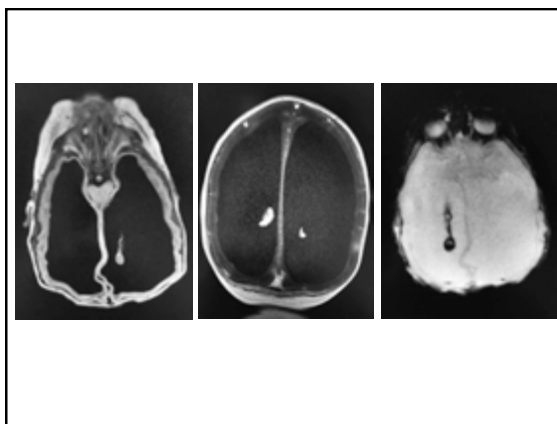


Chart review suggested relatively normal prognosis with the possibility of developmental delay and motor problems in the legs.

Dx: Feeding Difficulties in Newborn, Neurogenic Bladder, Hyperbilirubinemia, Chiari Malformation

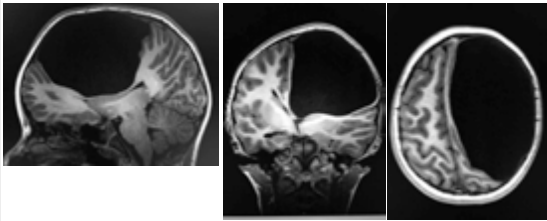
ROS, Neurologic:

Hydrocephalus – bilateral
 Poorly seen cerebellum, suspected Chiari II malformation
 Cisterna Magna difficult to appreciate
 Poor function of legs anticipated (PT/OT to follow as needed)
 Poor prognosis for ambulation, discussed with mother
 High possibility of developmental delay
 Mother optimistic he will not need a wheelchair



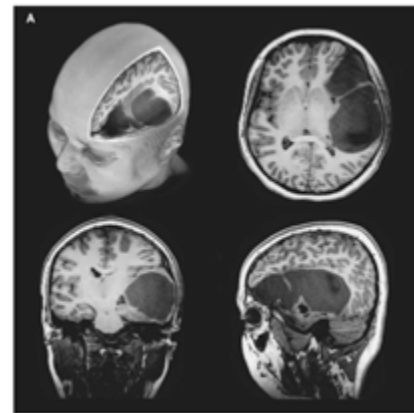
Sometimes, the chart information seems bad, although the prognosis is good.

Benign cysts, such as arachnoid or neuroglial can appear bad, be described in a manner to suggest pathology, but result in no deficits on examination or normal functioning...
Be very cautious when examining such patients... there is a tendency to over interpret normal variability as related to the cyst, or even bias your findings in a manner to suggest pathology, just because of the images on MRI, or notation in the chart.



Abnormal
Brain

Normal
Functioning



Acute Necrotizing Encephalopathy of Childhood

In one year I had three patients with this condition

One died within a few weeks of dx

One was severely disabled with life long disabilities

One recovered to full independent functioning

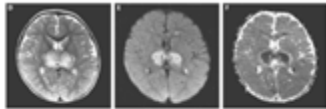


Figure 10.10.4 Clinical Example: Acute necrotizing encephalopathy of childhood. Diffuse white matter lesions with ring-like appearance on T2-weighted, and a dot appearance on T1, are seen in this patient.

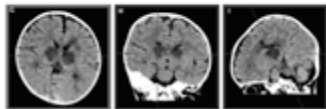


Figure 10.10.5 Clinical Example: Acute necrotizing encephalopathy of childhood. T2-weighted images show bilateral, symmetric, hyperintense lesions in the white matter, suggesting demyelination.

Radiology report indicated, **“white matter lesions throughout her parenchyma, within both hemispheres. Findings suggest ADEM or other demyelinating inflammatory process.”**

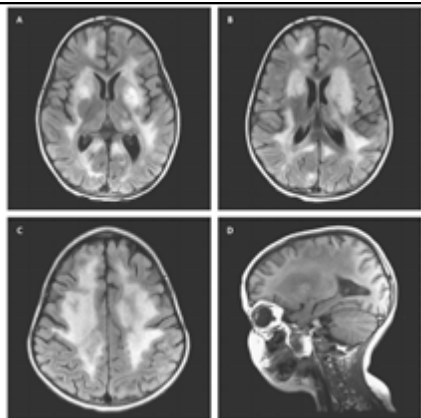
The parents of this patient were told (paraphrased):
“There are lesions all over her brain.”

When asked how much of her brain was involved, they were told, **“Every part of her brain had lesions.”**

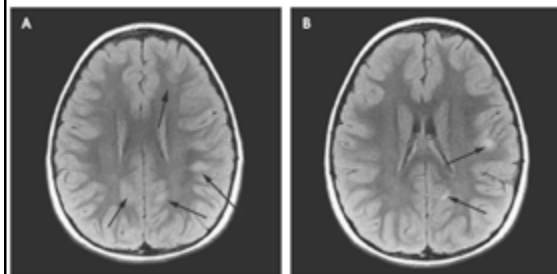
The parents were devastated by this news and emotional distraught when I first met them...

This is a picture of severe ADEM

This is not the patient being discussed



T2 FLAIR imaging of patient's ADEM



JL

Stroke s/p AVM rupture
Post neurosurgical embolization
Post neurosurgical resection

SK

Neoplasm – left frontal (low grade glioma)
fMRI and DTI
s/p resection

VCI = 109
PRI = 120
FSIQ = 118
Executive Function (ss range 11-15)
Attentional Function (ss range 12-14)
Memory/Learning (ss range 11 - 14)
Reading (ss range 13 - 15)
Math/Arithmetic (ss range 12-15)

Hypoxic Ischemic Encephalopathy (HIE)

How to interpret the degree of acidosis and lab values

I am more comfortable using extreme values (>7.2 or <6.8)

General Rule (pH values... Lower = Acidotic from loss of oxygen)

7.4 Normal
7.2 Mild Damage but will do ok
7.0 Mod to Severe Damage and will have life long impairments
6.8 Severe to Profound Damage, life long disability, dependent
6.6 Dead

- MB
 - **pH 6.7** status post drowning incident
 - One year post drowning, at 3 years, she walked up to me and gave me a big hug... Functioning was close to normal at 3 years.

Myself and her hospitalist had discussed end of life options with her mother... We still talk about her as being our miracle patient.

- JB
 - **pH 6.9** status post drowning
 - Was never able to be extubated, remained ventilator dependent until he passed a few months following his drowning incident

- KW
 - **pH 7.1** status post drowning incident (20 yr old)
 - Two weeks post hospitalization, he was discharged home with clean NPSY findings
 - Six months, and one year later, no limitations found on NPSY or in real life functioning

- TE
 - **pH 7.0** status post cardiac arrest x 45 minutes (CPR)
 - Follow up examination six months later, she was able to walk, run, talk and play.

Mother reminded me, **“Remember when she was in the ICU, you were the only doctor who gave us hope and a realistic range of possibilities.”**

She was told, TE was in a vegetative coma and was imminently expected to die.

I noticed she would change her affect in response to interactions, in a consistent and non-reflexive manner, suggesting a degree of being locked-in.

Neuropsych. Data 6 months post

- Orientation: WNL
- Awareness/Insight: WNL
- Social/Interpersonal/Behavioral: WNL
- Passive/Sustained Attention: Mildly limited
- Expressive Language: Superior (ss = 15, 95th %ile)
- Receptive Language: High Average (ss = 14, 91st %ile)
- Verbal/Linguistic Reasoning: High Average (VCI = 117, 87th %ile)
- Visual/Spatial Reasoning: High Average (PRI = 112, 79th %ile)
- Follow-up is critical as she may grow into her injury.

- **MS pH 7.0** status post cardiac arrest
 - Follow up examination six months later, he was vegetative with decerebrate posturing and ventilator dependent. He remains in that state several years after his cardiac arrest.
 - Same age, similar circumstances, almost identical lab values and medical histories...

Two very different outcomes.

**Never assume you know the future
Provide probabilities, expectations,
best/worst/expected outcomes...**

**Chart documentation can miss critical
neuropsychological issues important for the
patient's care.**

**You have an opportunity to fill in the
missing information... For the medical
team, patient and/or family**

I have an advantage of having a standing order to review all trauma, ICU, and neurosurgery cases (they are sent each morning to my computer), to determine if NPSY services are appropriate in the care of the patient.

I also have the advantage of being asked by our physicians to review imaging with patient/family for many cases, allowing me to become involved and provide assessment if indicated.



NR

- 17 Y/O Male presents with personality change, odd speech, visual hallucinations, violent behavior, increased sexuality.
- Previous hospitalization in psychiatric facility.
- Psychiatric meds didn't help, he continued to deteriorate in all functional abilities.
- Admit Dx
 - Altered Mental Status
 - Psychiatric Symptoms
- **What is going on with this patient?**

Mother not satisfied with treatment, so presented to our hospital and I was called to assess.

- Based on past medical history and extensive chart review, one must consider psychosis
- **NPSY:**
 - Intact orientation, insight, awareness of sx
 - Intact rec/exp lang and verbal reasoning
 - Mildly impaired visual/spatial reasoning
 - Severe deficits
 - Executive Function, mental flexibility, memory, complex/divided attention.

- **NPSY cont.**
 - Disrupted self-control, highly impulsive
 - Behaviorally disinhibited
 - Emotionally labile
 - Disrupted HPA-Amygdala Axis (fight or flight)

**Would masturbate in front of his mother while saying...
"I'm gonna kill you bitch."**

Through history and discussion with mother, it appears that he was experiencing ongoing loss of motor ability and had a mild but progressive ataxia (BUE), and had become incontinent.

Didn't sound like classic psychosis to me, especially with the progressive loss of ADL's described by mother over the last six months.

Sounded more "frontal" to me...

Ordered Imaging...

Dx...

Long term outcome...

Two months post discharge,
and 8 months following
onset of symptoms:

Mute
Dystonia
Severe Dementia
Non-ambulatory
Perpetually Happy

CA

- Assessment
 - Critically ill female with multiple complex problems
 - Right Intraventricular tumor, status post resection
 - Obstructive hydrocephalus, s/p EVD DC'd, S/P VPS
 - Aphasia – resolved
 - Left Hemiplegia – resolved
 - Neurologically intact without focal deficits or findings
 - Functioning back to pre-morbid baseline

- Post operative imaging was viewed by PT who then asked ICU attending to request NPSY, even though patient was apparently fully intact and back to pre-morbid baseline functioning.

- Imaging Impressions:
 - Postoperative changes of a transcallosal approach to the intraventricular tumor. Some soft tissue density in the right lateral ventricle is currently noted and the tumor probably has been resected. Alternatively, hemorrhage within the tumor could be present but this seems less likely. Ventricles are not markedly dilated with the ventricular catheter in place.

- On NPSY:
 - Alert, oriented (x4), insightful, intact safety awareness
 - Intact receptive and expressive language
 - Intact basic reasoning
 - Deficits involving:
 - Processing speed and efficiency
 - Integration of complex information
 - Executive functioning, mental flexibility, divided attention, multi-tasking, etc. (D-KEFS CWI, CWIS, Trails SW = ss 1s)

**"There is no physician who would be aware of, or even attuned to these difficulties in their patient."
Comment from a colleague regarding these findings.**

- Imaging Findings:

LZ – 17 yr old male

- Excellent recovery, appeared to be functioning normally by discharge from the acute care unit.
- ROS unremarkable, even for neurological
- I was asked by resident to discuss expected long-term issues relating to his TBI (as if it was just like any other TBI).

- Imaging:
- Concern:
 - Higher-level attention, especially to environment
 - Social-integrative functions
 - Social-interpersonal, inhibition, impulsivity
 - Inappropriate behaviors

- Father returned for emergency visit due to concerns...
 - Inappropriate socially, especially sexually
 - Delusional
 - Has a GOD/DEVIL complex/addiction
 - Reports he has been “selected to sacrifice someone to appease God and keep the Devil away”

MS

Assessment and Plan

- General Appearance: Uncomfortable, no distress
- Neurologic: Strength 5/5 all Ext, GCS 15, Follows commands, moves extremities well
- Left subdural hematoma
- Concussion with post concussive symptoms

“Subdural hematoma trumps concussion...”

“What we call something is important.”

Symptoms were protracted, and more consistent with TBI, as was NPSY evaluation and difficulties upon RTL

JR

Prolonged coma, persistent vegetative state
Prognosis in the chart was consistently bad

But...

Imaging revealed significant midbrain, brainstem, reticular injury in addition to the tearing of white matter, and left occipital.

Suggested possibility of “Light Switch Injury.”

In an individual child, it is not possible to predict outcome reliably from the length or severity of coma.

You need to consider the caused of the coma.

Remember, coma can mask many symptoms of brain injury, AND NORMAL COGNITIONS, making determination of expectations more difficult.

The absence of a response is less informative than the presence of a response.

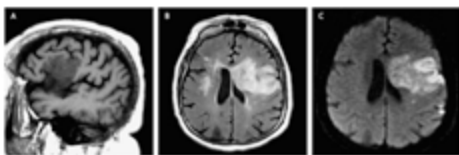
Example JS (end of life/DNAR issues)

Thrombosis in leg required surgery...
Resulted in embolic stroke

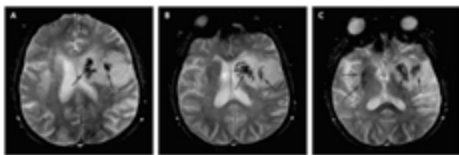
Family informed: “she would recover from the stroke over the next year or two.”

**However... Advanced Directive
Didn't want to live if couldn't talk
Husband died of ALS and couldn't talk**

Stroke – Embolic (top) and hemorrhagic (bottom)

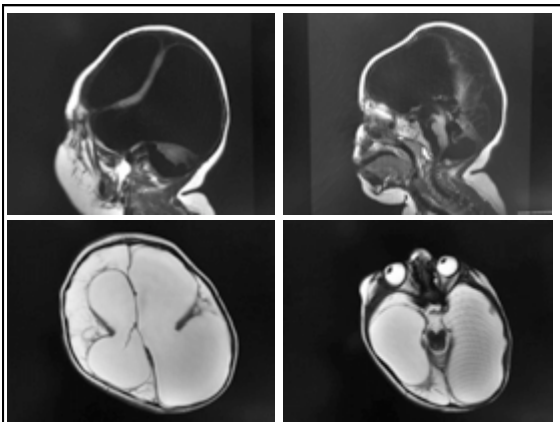


Figures 8-34A-C Clinical examples per MRI evidence of significant hyperintense ischemic infarction within the left anterior MCA distribution. A, T1-weighted; B, T2-weighted; C, DWI.



Figures 8-35A-C Clinical examples per follow-up MRI series in large vessel stroke showing areas of hemorrhage on DWI, decreasing regions of susceptibility artifact (arrows).

End of life issues for NAT



Questions