Value of SWI in Identifying Benign Incomplete Suppression of CSF on T2-FLAIR

Joseph Yetto, MD (jmyettojr@gmail.com)
Allen Mao, MD
Xavier Lopez-Garib, MD
Catalina Restrepo-Lopera, MD
Orrin Dayton, MD
Michael Cathey, MD
Ibrahim Tuna, MD

Department of Radiology, University of Florida College of Medicine, 1600 SW Archer Road, Gainesville, FL 32610

Northwest Imaging, 44 Second Ave W, Kalispell, MT 59901

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Educational Objectives

Show examples of T2-FLAIR leptomeningeal non-suppression

Detailed physics review of Susceptibility Weighted Imaging (SWI) with a focus on what the neuroradiologist needs to know

Provide an illustrative case-based approach showing how to use SWI to troubleshoot T2-FLAIR non-suppression into pathologic versus physiologic categories

Reinforce troubleshooting techniques with unknown case challenge

Introduction

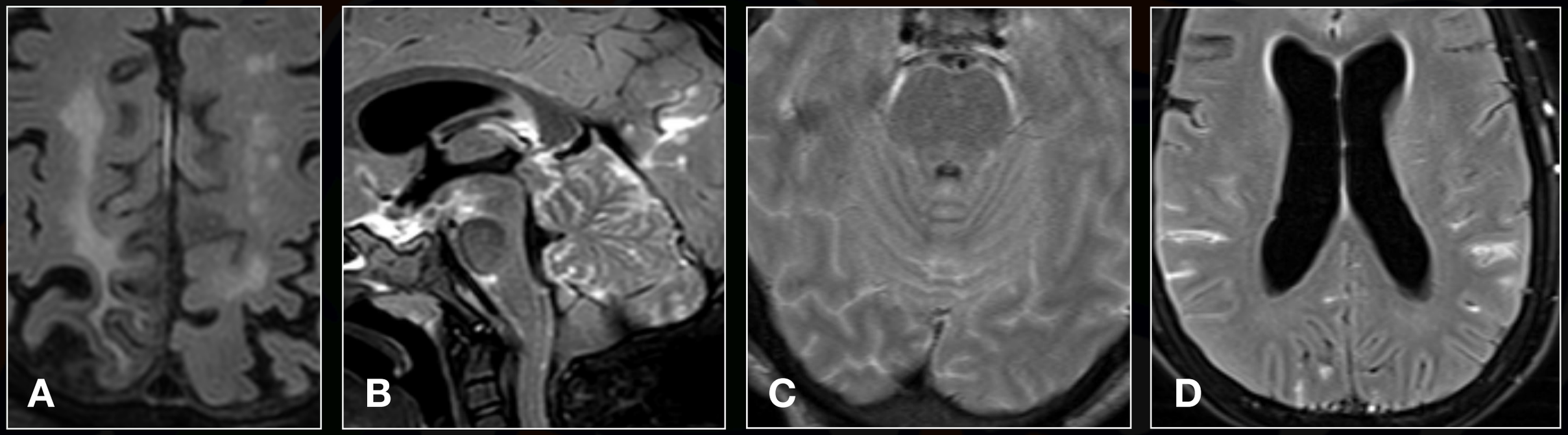
Neuroradiologists are frequently asked to distinguish between benign and pathologic processes that may present with identical or near identical imaging findings.

For example, many inpatient studies are completed on clinically complex patients some of whom may be intubated. Physical exam and minimally invasive laboratory parameters may be unclear or altogether conflicting.

In this setting, a commonly encountered problem is leptomeningeal non-suppression of T2-FLAIR imaging - a “positive” study that typically indicates the abnormal presence of blood, infection, or cells within CSF.

However, particularly in an intubated, there is a benign cause of leptomeningeal non-suppression that can be distinguished from a pathologic processes by understanding the physics of SWI. That is, an astute neuroradiologist can use SWI to troubleshoot a “positive” T2-FLAIR, which may prevent an unnecessary invasive examination such as a lumbar puncture, or vice versa.

Leptomeningeal T2-FLAIR Non-suppression

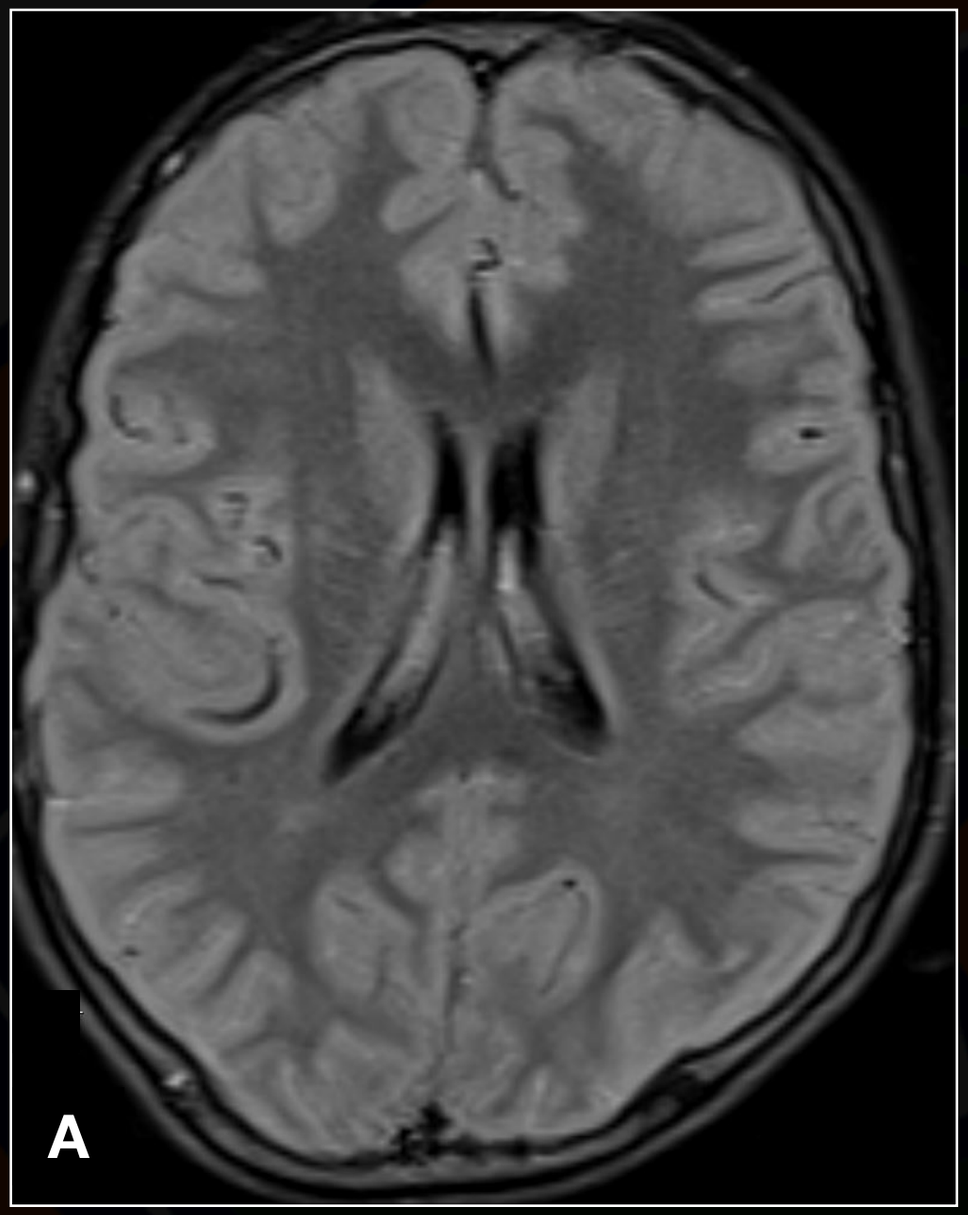


Examples of variable degrees and causes of T2-FLAIR CSF non-suppression. A, Hemosiderosis related to recurrent bleeds in the setting of cerebral amyloid angiopathy; no acute blood products. B, Leptomeningeal carcinomatosis from PNET. C, Bacterial meningitis. D, Subarachnoid blood products after trauma.

Ultimately, the reason for non-suppression is typically dictated by the clinical scenario, or CSF analysis after lumbar puncture if there is clinically uncertainty regarding the etiology.

Thus, the primary role of the radiologist is to determine the presence or absence of CSF non-suppression.

Hyper-oxygenation Effects on T2-FLAIR



This is an example of leptomeningeal T2-FLAIR non-suppression due to hyper-oxygenation. This particular exam was performed in a patient requiring intubation due to severe claustrophobia. There were no other clinical reasons for the FLAIR signal abnormality.

Why does hyper-oxygenation cause T2-FLAIR non-suppression?

Key concept: Increased arterial oxygenation leads to increased dissolved free oxygen in the CSF, which demonstrates slightly increased T1 shortening relative to normal CSF.

The following slides are arranged to show how to use SWI to identify or exclude hyper-oxygenation as the cause of FLAIR non-suppression in cases where the clinical evaluation was confusing and imaging was crucial in determining whether or not to proceed to lumbar puncture in these complex patients.

AJNR Am J Neuroradiol 25:274–279, February 2004

Paramagnetic Effect of Supplemental Oxygen on CSF Hyperintensity on Fluid-Attenuated Inversion Recovery MR Images

Yoshimi Anzai, Makiko Ishikawa, Dennis W. W. Shaw, Alan Artru, Vasily Yarnykh, and Kenneth R. Maravilla

Susceptibility Weighted Imaging Overview

Gradient Echo Sequence
+
Acquired in 3D
+
Flow compensation
+
Phase and Magnitude information

Susceptibility Weighted Imaging

Modern SWI sequences are far more sensitive and specific for the detection and identification of iron, blood products, and calcification when compared to the older, simple T2*-weighted gradient echo (GRE) sequences.

This is due to following:

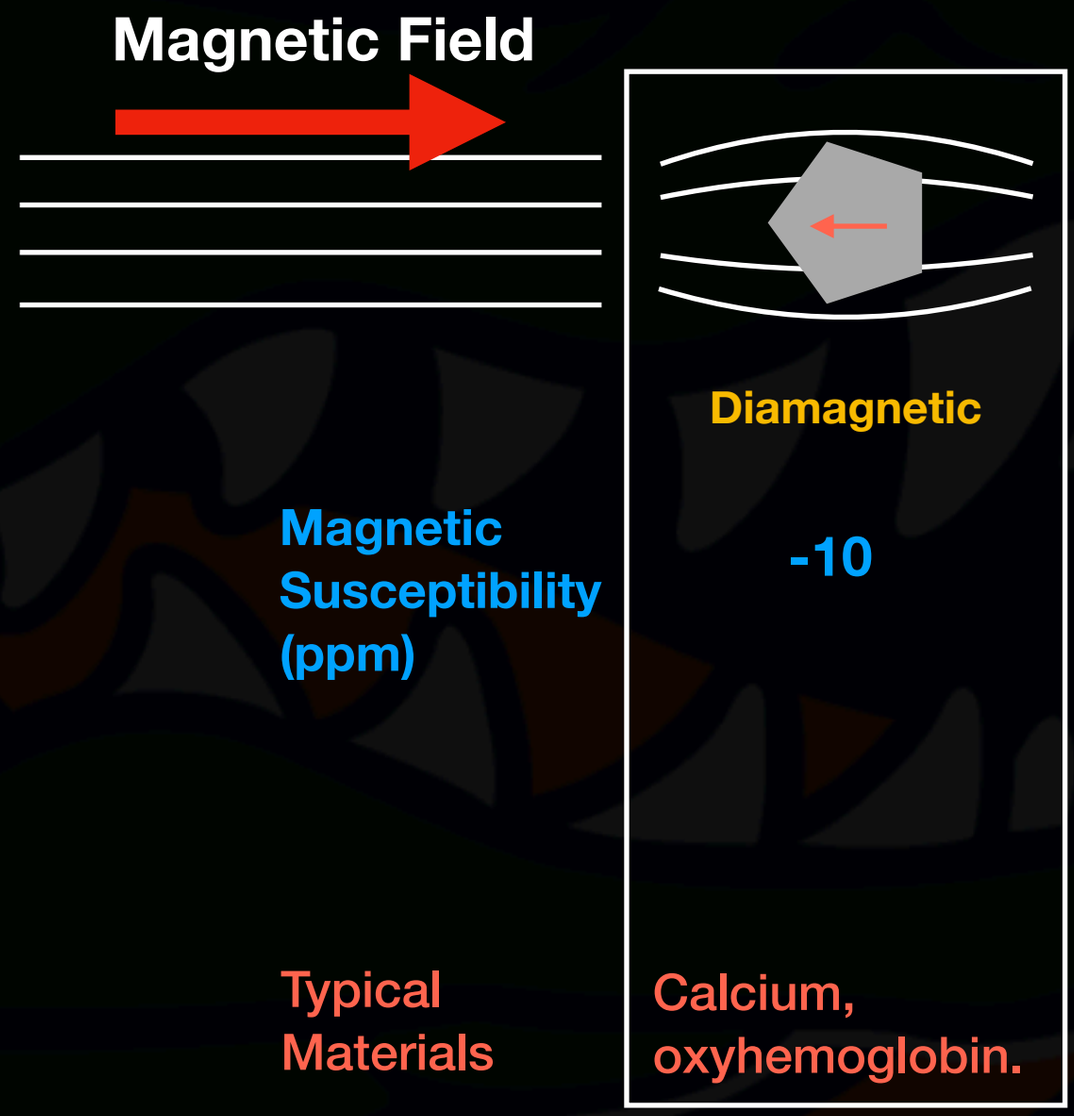
1. Modern SWI sequences are typically three-dimensional acquisitions (rather than 2D), allowing thinner slices (i.e. increased resolution), smaller voxel sizes (i.e. reduction of defacing across slices), and longer echo times (i.e. allows for flow compensation).
2. Flow compensation in all three directions is used to reduce flow induced phase changes (i.e. reduce artifacts).
3. A key feature of SWI is that phase and magnitude data are independently processed and displayed as well as combined for diagnostic purposes.

Physics of Susceptibility Weighted Imaging I

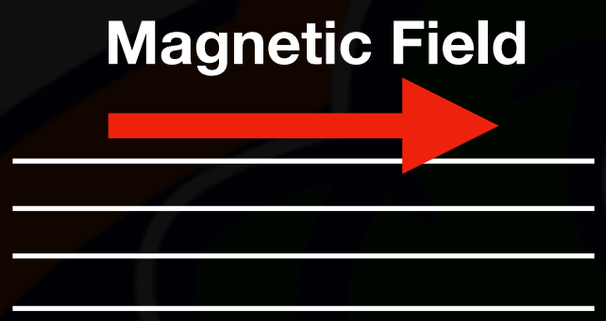
Susceptibility (or “magnetizability) is a measure of the extent to which a substance become magnetized when it is placed in an external magnetic field.

When matter interacts with the magnetic field, an internal magnetization (or polarization) is created that either opposes or augments the external field.

If the polarization opposes the applied field, the effective field within the object is reduced, the magnetic lines are dispersed, and the effect is known as diamagnetism.



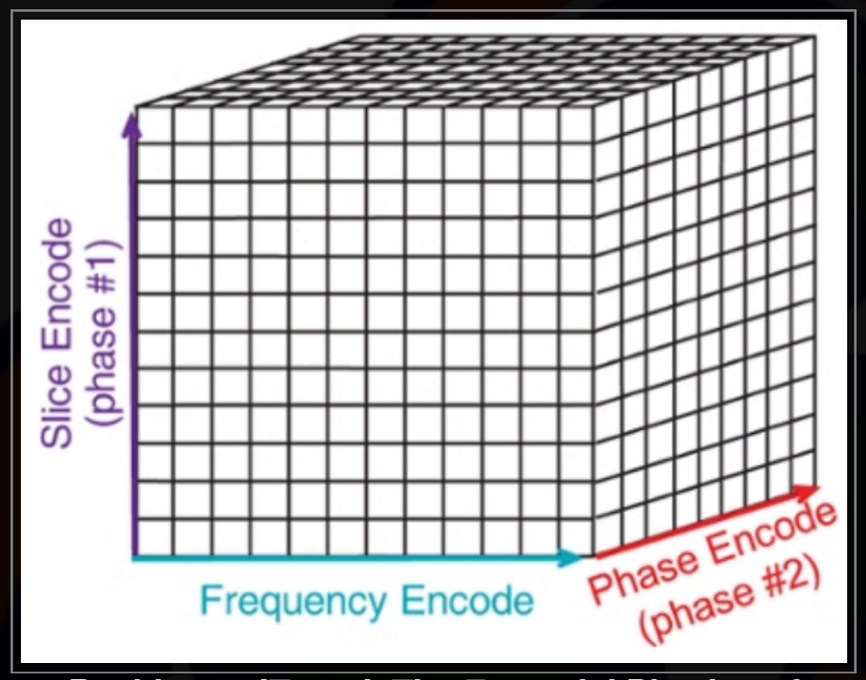
Physics of Susceptibility Weighted Imaging II



	Paramagnetic	Superparamagnetic	Ferromagnetic
Magnetic Susceptibility (ppm)	+1	+5000	>10,000
Typical Materials	Chelates of metals (Gd, Fe, Mn, Cu), deoxyhemoglobin, methemoglobin, scavengers of free metals & radicals (i.e. melanin).	Ferritin, hemosiderin.	Iron, steel

However, if the polarization is in the same direction as the external field, the magnetic lines are concentrated within the object, resulting in paramagnetism, superparamagnetism, or ferromagnetism, depending on the *degree* of augmentation.

Three-Dimensional Acquisition

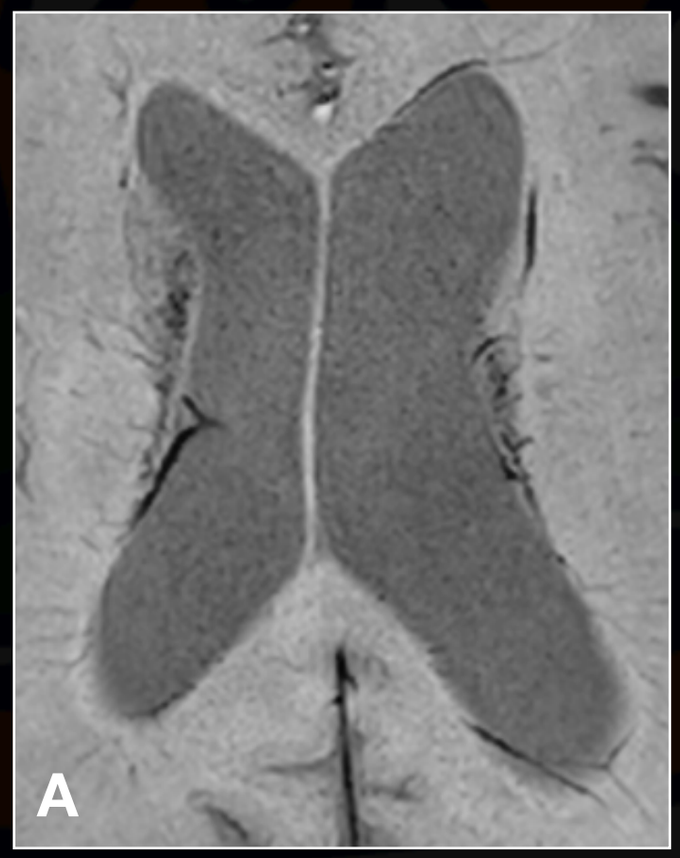


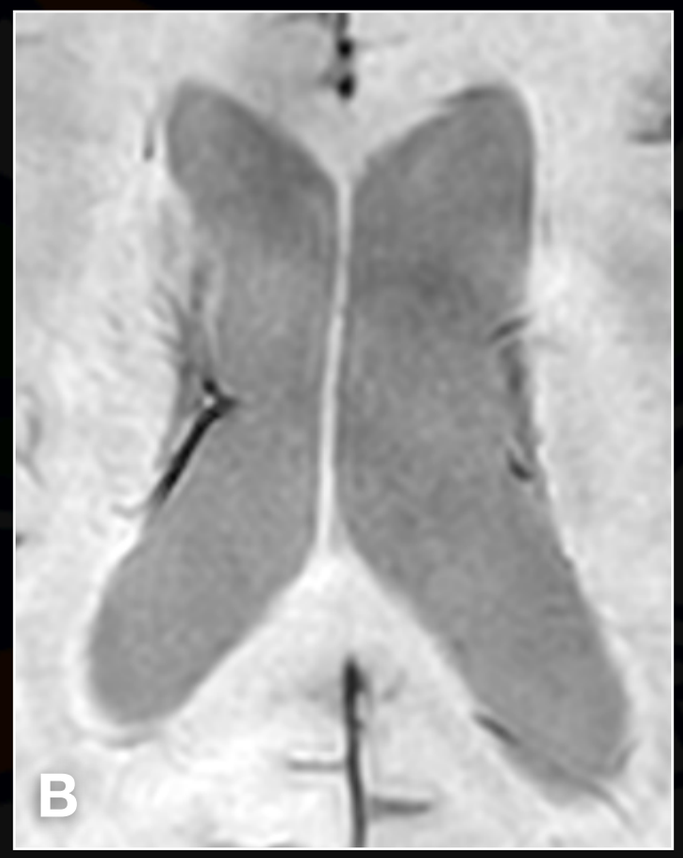
Bushberg, JT et al. The Essential Physics of Medical Imaging. Third Edition.

Three-Dimensional Acquisition. 3D image acquisition (i.e. volume imaging) requires the use of broadband, nonselective RF pulse to excite a large volume of protons simultaneously. Two phase gradients are applied, one in the slice encode and another in the phase encode directions, prior to the frequency encode (readout) gradient.

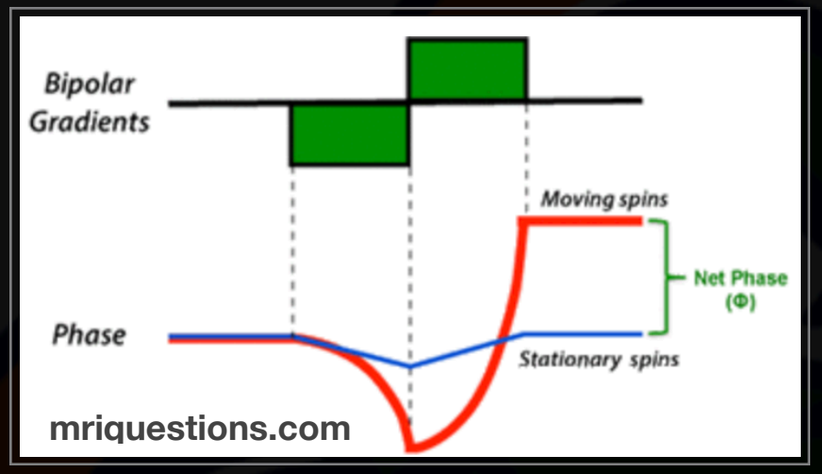
The advantages of volume imaging include 1) high spatial resolution, and 2) high signal-to-noise. Also, the addition of a second phase gradient increases the echo time (TE), which allows for the application of flow compensation.

Comparison of 2D and 3D sequences. *A*, 3D susceptibility weighted image exquisitely demonstrates the medullary and septal veins. *B*, 2D gradient echo sequence is provided for comparison.

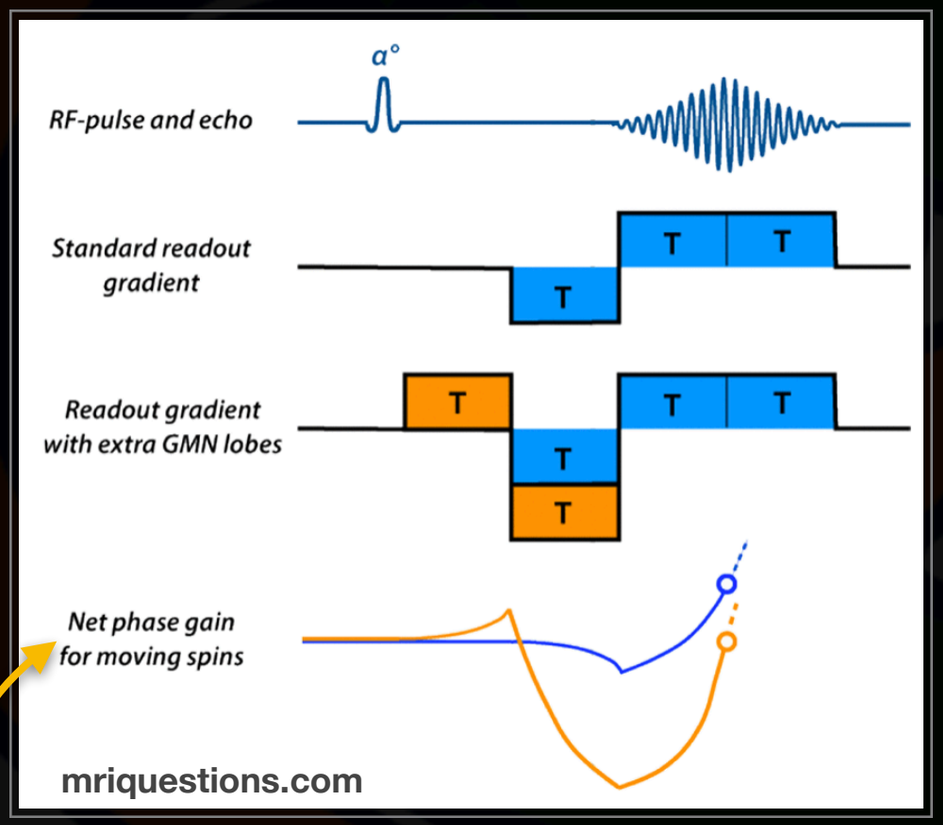




Flow Compensation



A stationary spin subjected to a gradient pair (green lobes) will experience no net phase shift, but a moving spin will have a net phase shift (ϕ) proportional to its velocity. This is the basis for phase contrast MR angiography.

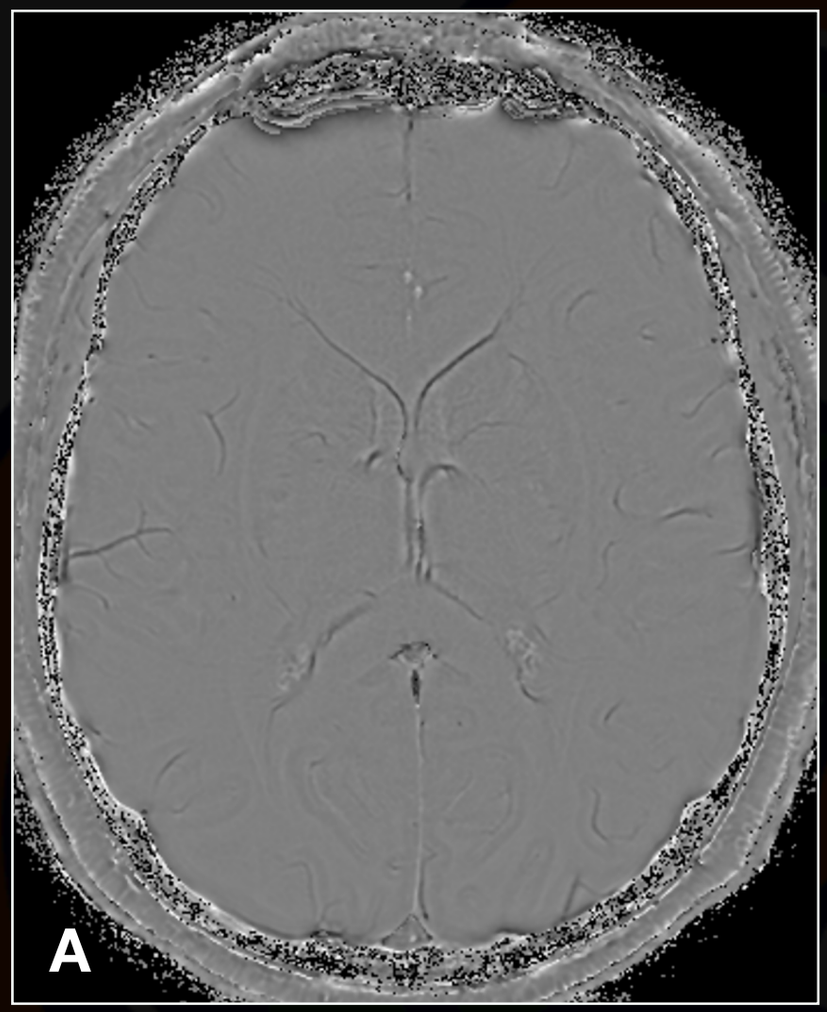


Flow Compensation. Additional gradient lobes are added (orange lobes) prior to signal readout to compensate in advance for motion induced dephasing at the time of the echo. These additional gradient lobes reduce motion induced phase shifts caused by moving spins (and therefore provide clearer delineation of vessels).

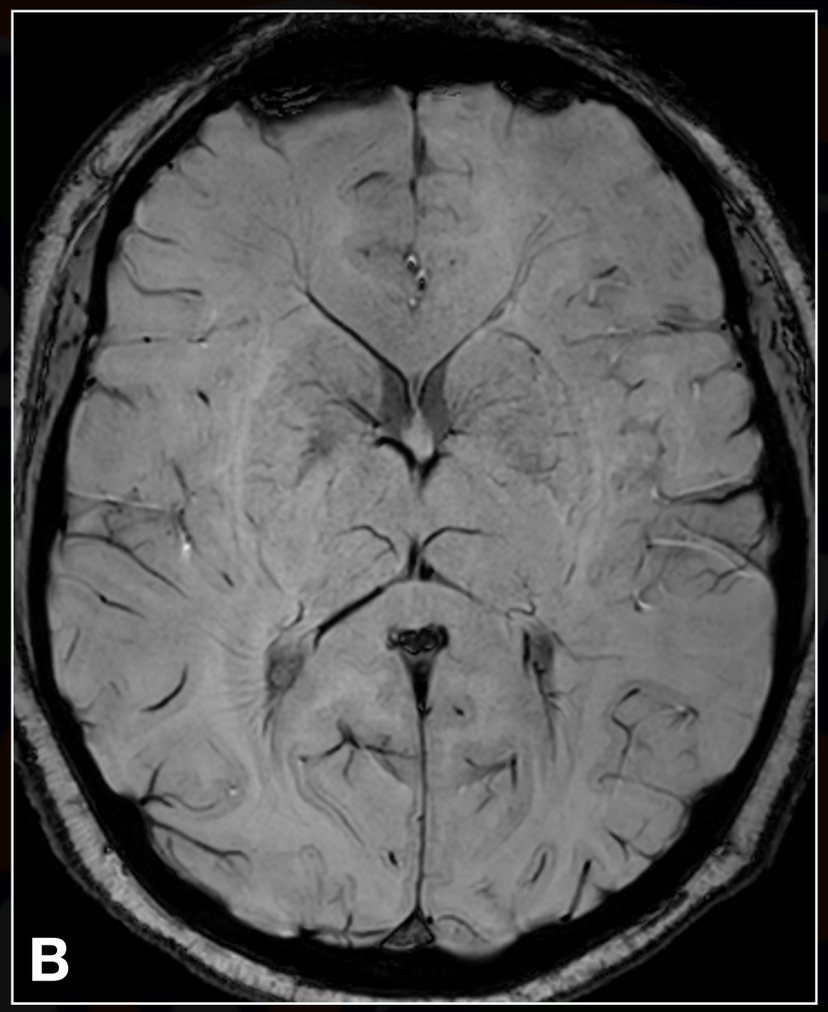
Among the limitations of this technique, the minimum TE is lengthened because time is needed to fit in the extra gradient lobes.

Using the standard GRE readout gradient, moving spins accumulate phase that is nonzero at the center of the echo (blue circle). This non-zero phase at mid-echo results in signal loss. However, with the addition of the additional orange gradients, the net phase of the moving spins has returned to zero (orange circle). GMN=gradient moment nulling.

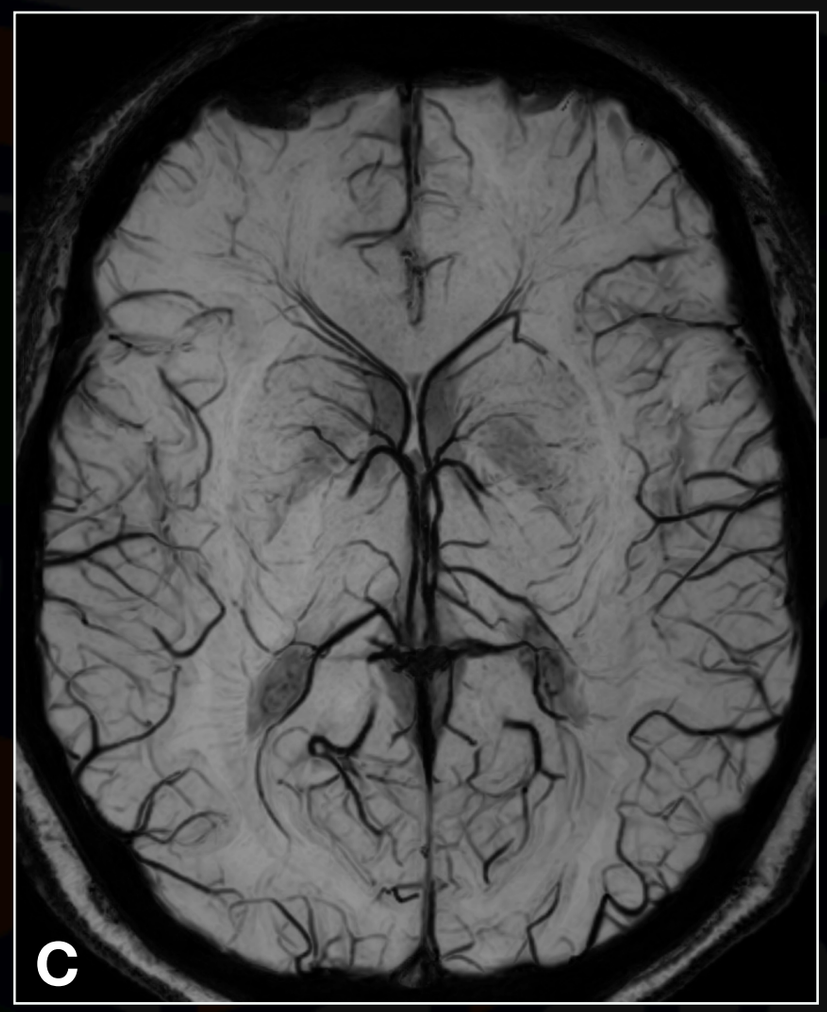
Phase and Magnitude Data



Filtered Phase Image



SW Image



SW Image (minIP)

A, The filtered phase image is created from the raw phase image that is dominated by large susceptibility gradients. Then a phase mask is created that scales the data from the filtered phase image to accentuate tissues with different susceptibilities. The magnitude image (not shown) is digitally multiplied by the phase mask multiple times until the desired mix of phase information is imparted. The end results is a susceptibility weighted image, (*B*) and its minimum intensity projection (minIP) (*C*), that simultaneously contain both phase and magnitude information.

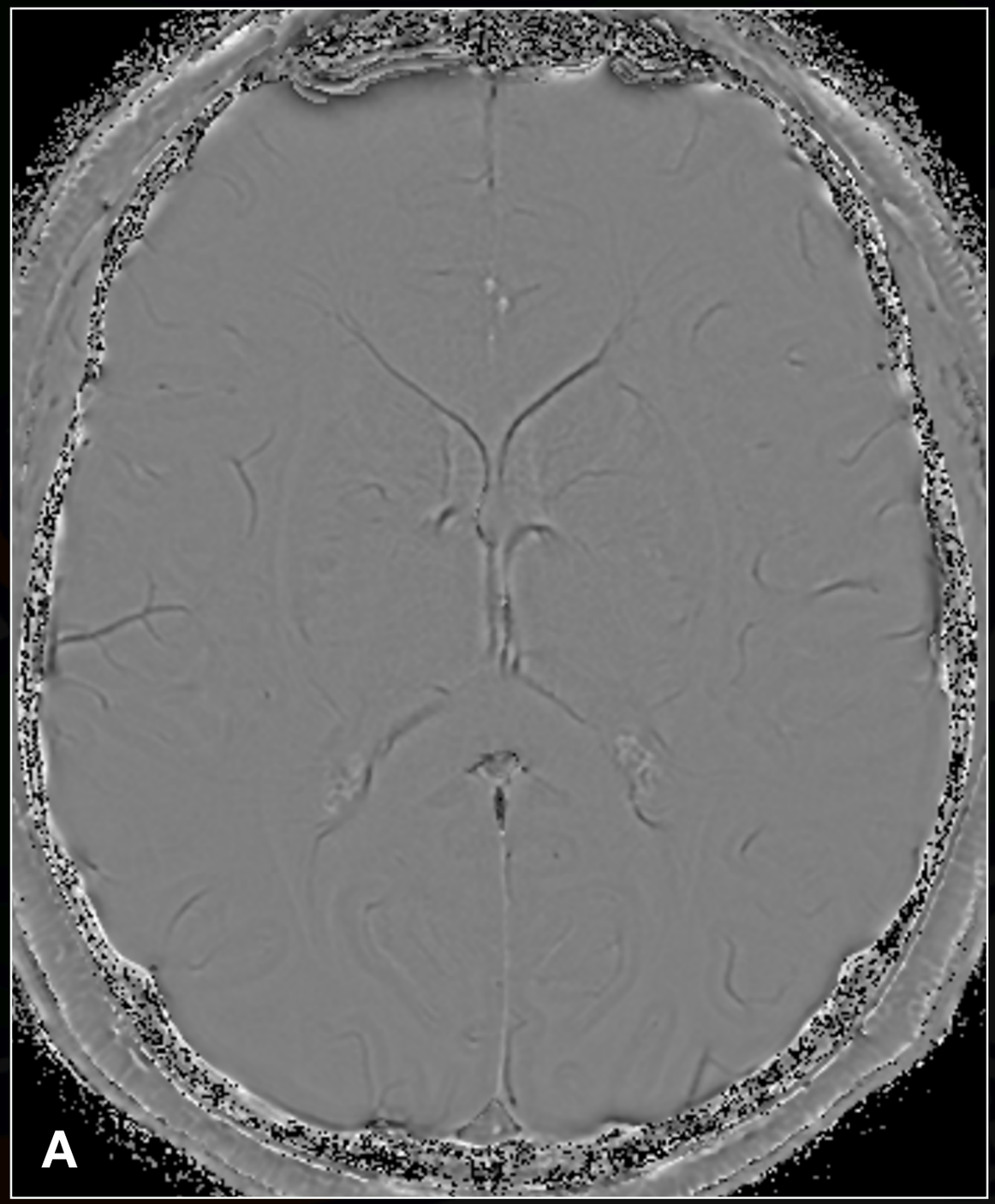
Filtered Phase Imaging

Raw Phase Image → (filter) → Filtered Phase Image

Phase images contain information about *all magnetic fields*; microscopic (e.g. small amounts of iron deposits and their ferromagnetic effects) and macroscopic (e.g. field changes caused by the geometry of the object, such as air/tissue interface effects and by inhomogeneities in the magnetic field).

That is, both microscopic and macroscopic magnetic fields effect the degree of phase shift.

However, macroscopic magnetic fields create artifacts and for this reason, are removed with a filter to generate a filtered phase image, *A*.

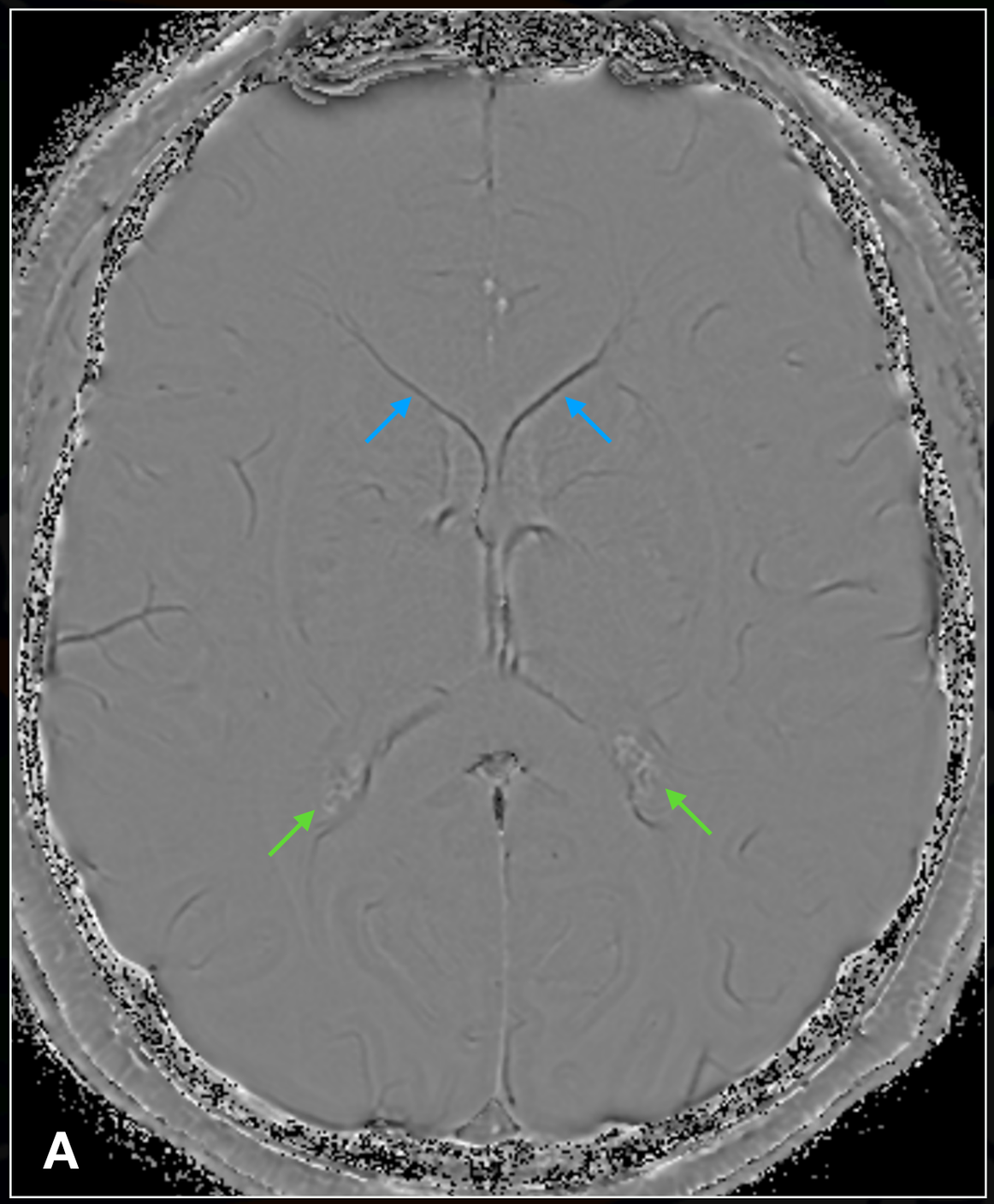


Filtered Phase Imaging *Interpretation*

A, Again, macroscopic magnetic field effects are removed by applying a filter to the raw phase image, which leaves behind the filtered phase image - this image is the basis for differentiating one type of materials from another, depending on their susceptibilities.

For a right-handed system, the phase is positive when the spins precess counterclockwise and will appear dark. The reverse is true for a left-handed system.

Thus, for a right-handed system: Paramagnetic substances, such as deoxyhemoglobin within veins, increase the magnetic field, resulting in a positive phase shift relative to the surrounding parenchyma and appear dark (→). Diamagnetic substances, such as calcium within the choroid plexus, cause a negative phase shift and appear bright (→).



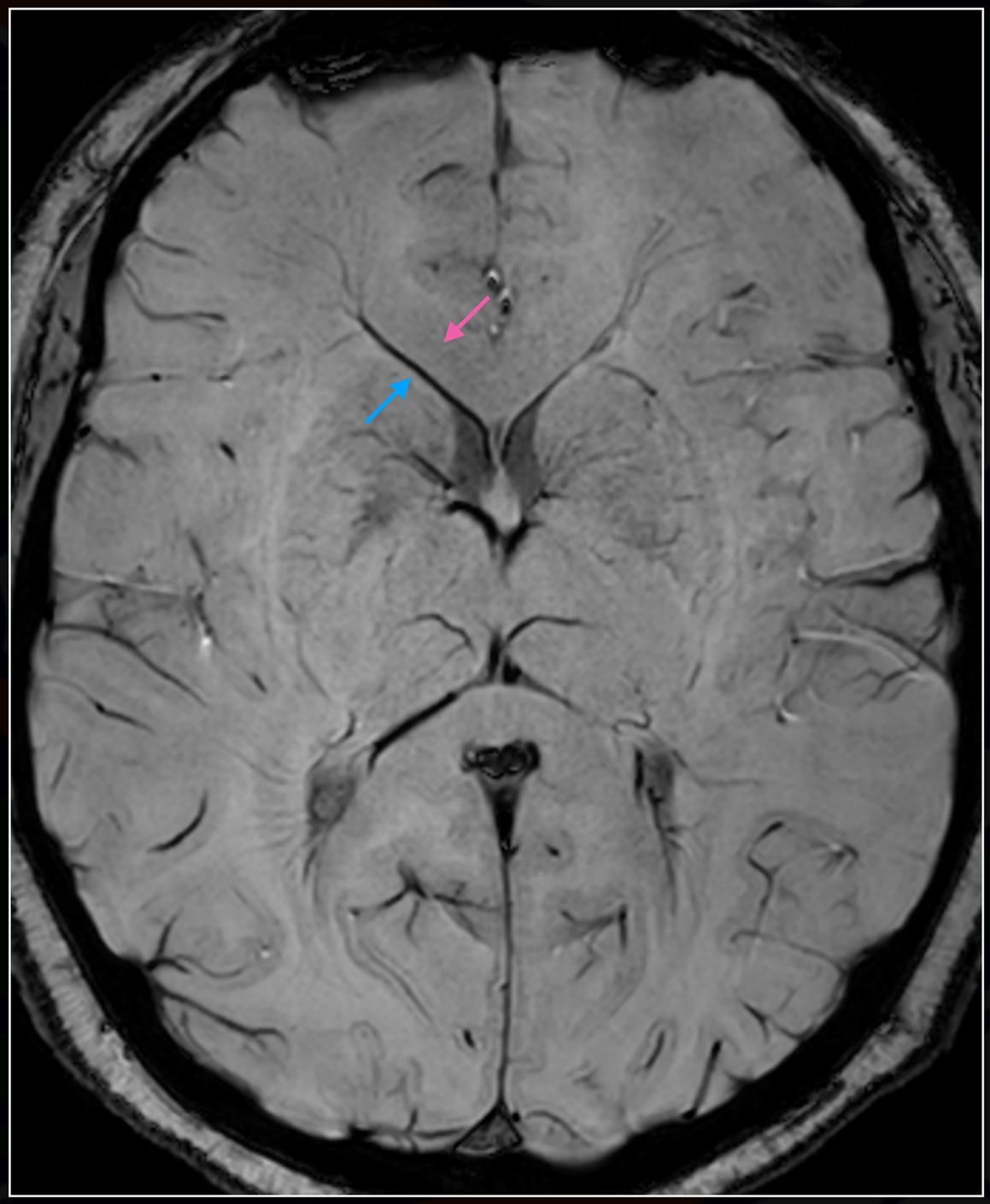
SW Image

SW Imaging. The addition of magnitude data to filtered phase imaging imparts high resolution (typically 0.5 mm (read) x 1.0 mm (phase) x 2mm (section)) to the final susceptibility weighted image.

Parameters are chosen so that the contrast is somewhat flat between normal grey matter, white matter, and CSF.

Custom post-processing is then applied (i.e. the magnitude image is digitally multiplied by the phase mask multiple times) until the desired level of contrast is achieved between tissues with different susceptibilities, for example between veins (→) and brain parenchyma (→).

The combination of parameters and post-processing allow for the SW image to highlight areas with short T2*.



SW Image

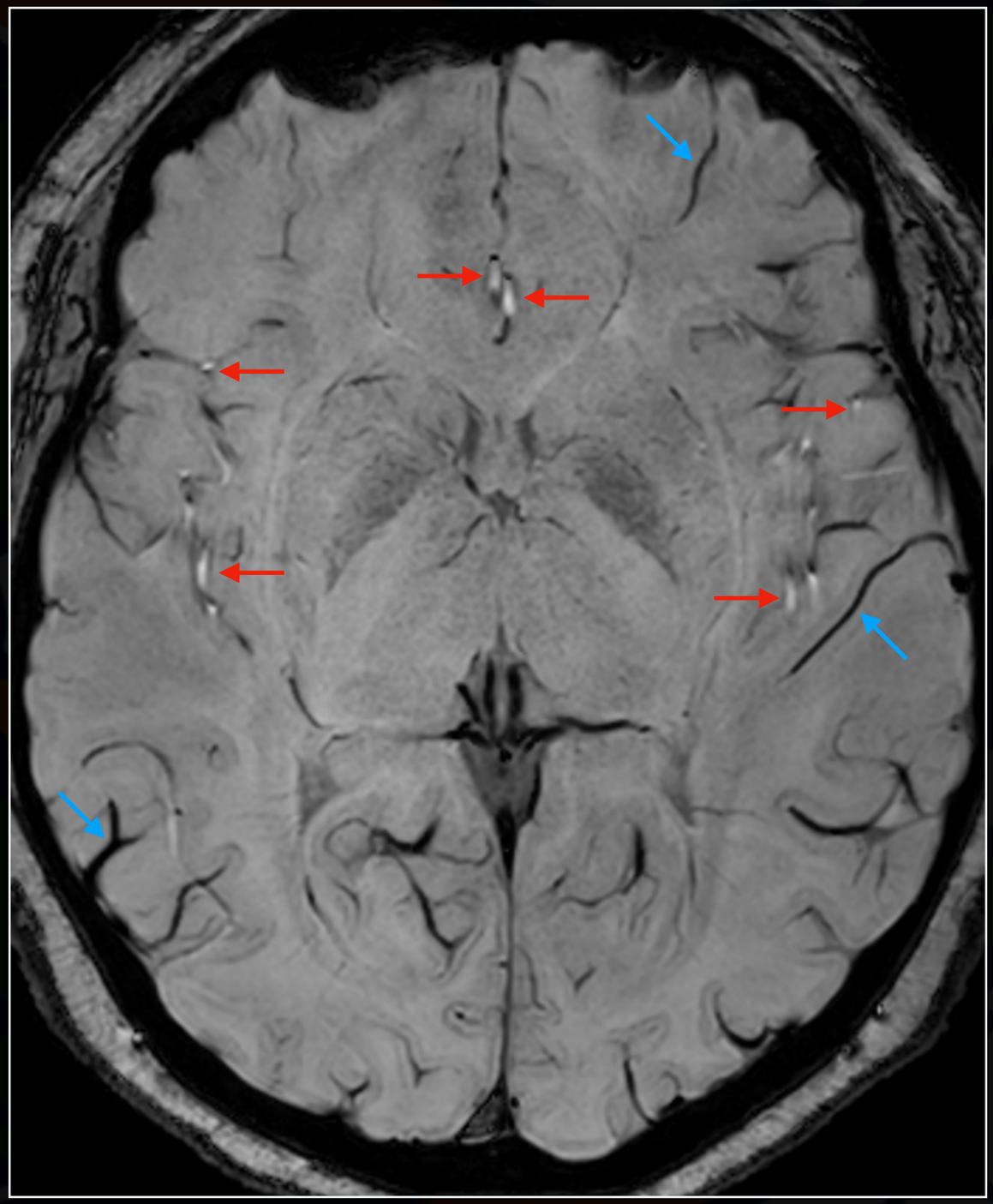
SWI Detects Susceptibility and T2* Effects

The deoxyhemoglobin iron in venous blood acts as an intrinsic contrast agent by causing:

1) a shift in phase relative to the surrounding tissues due to susceptibility differences
2) T2*-related losses in the magnitude image.

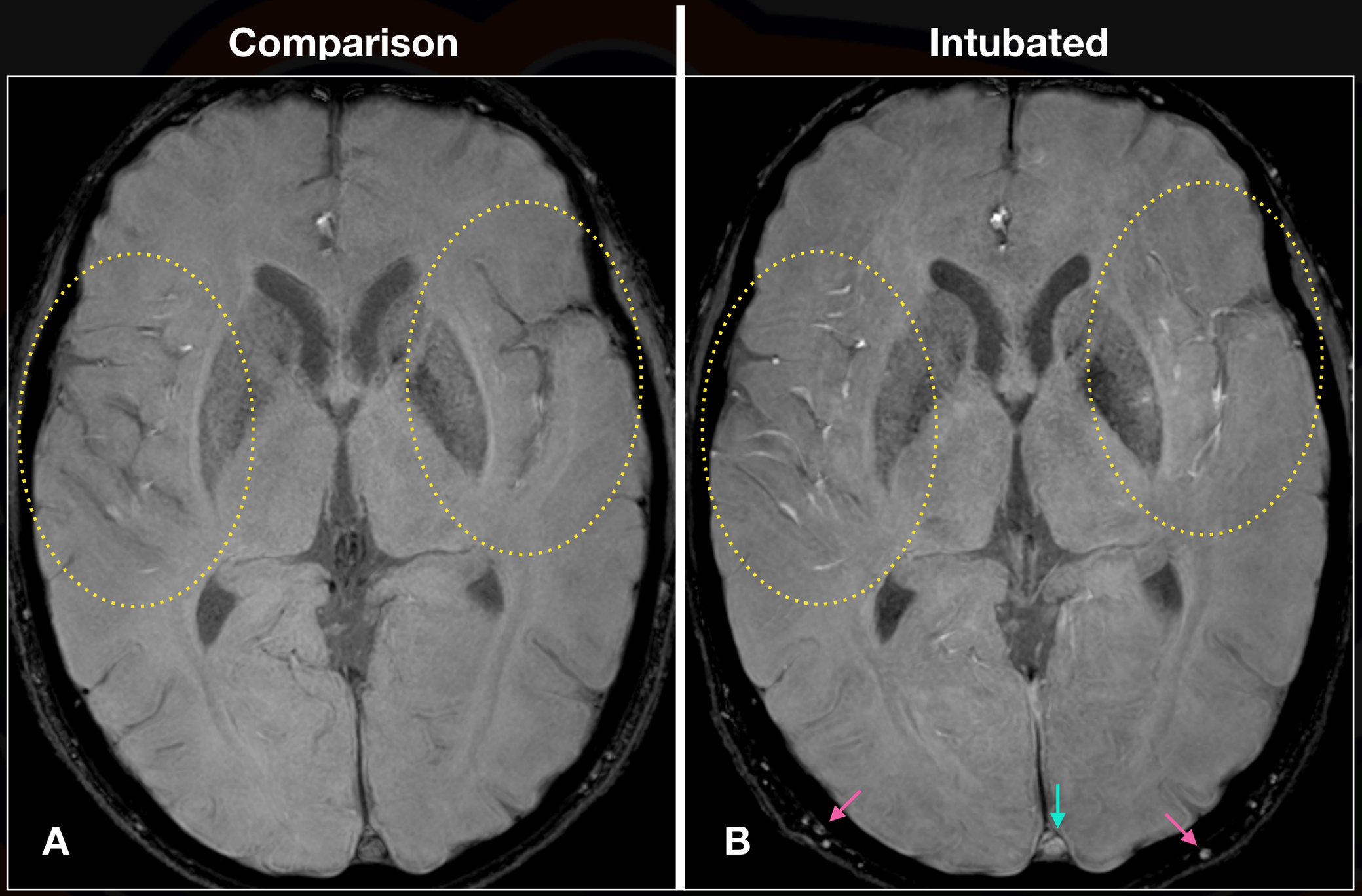
SW imaging thus ensures detectability of signal-intensity changes coming from both susceptibility and T2* between tissues.

Key concept: The oxygen in the oxyhemoglobin shields the iron so the susceptibility and T2* effects are only seen in venous blood. This provides a natural separation of venous (→) and arterial (→) blood. Note the hypointense signal in the cortical veins versus hyperintense signal in the branches of the ACA and MCA.



SW Image

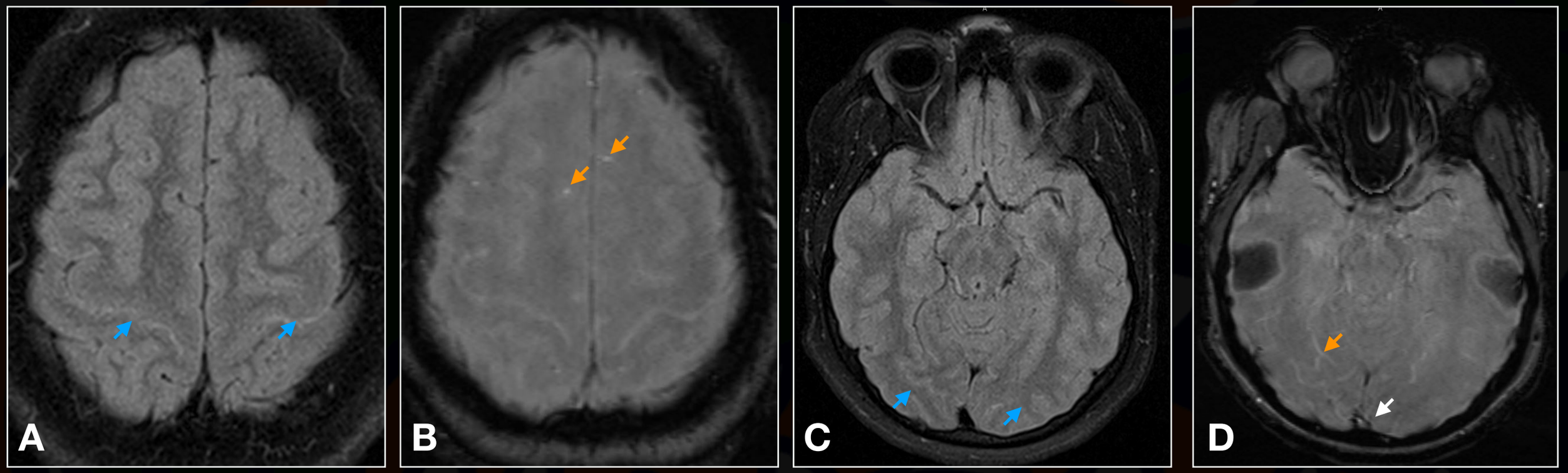
Hyper-oxygenation Oxygenation Effects on SWI



A, miP (5 mm) of the SW image from 2 years prior shows a normal appearance of the hyperintense arteries within the Sylvian fissure. *B*, miP (5 mm) of the SW image at the time the patient was intubated and oxygenated demonstrates increased hyperintensity of the vessels in the sylvan fissures. Also note the increased signal intensity in the dural venous sinuses (→) and the vessels of the scalp (→). This is likely due to an increased shielding effect of oxygen (due to hyper-oxygenation) on iron in hemoglobin.

Key concept: That is, increased oxygen levels lead to oxygen shielding effects on hemoglobin iron throughout the vessels. Normal oxygen levels result in oxygen shielding effects only within arteries, by time this blood makes it into the venous system enough oxygen has been removed to negate the shielding effects.

Case 1

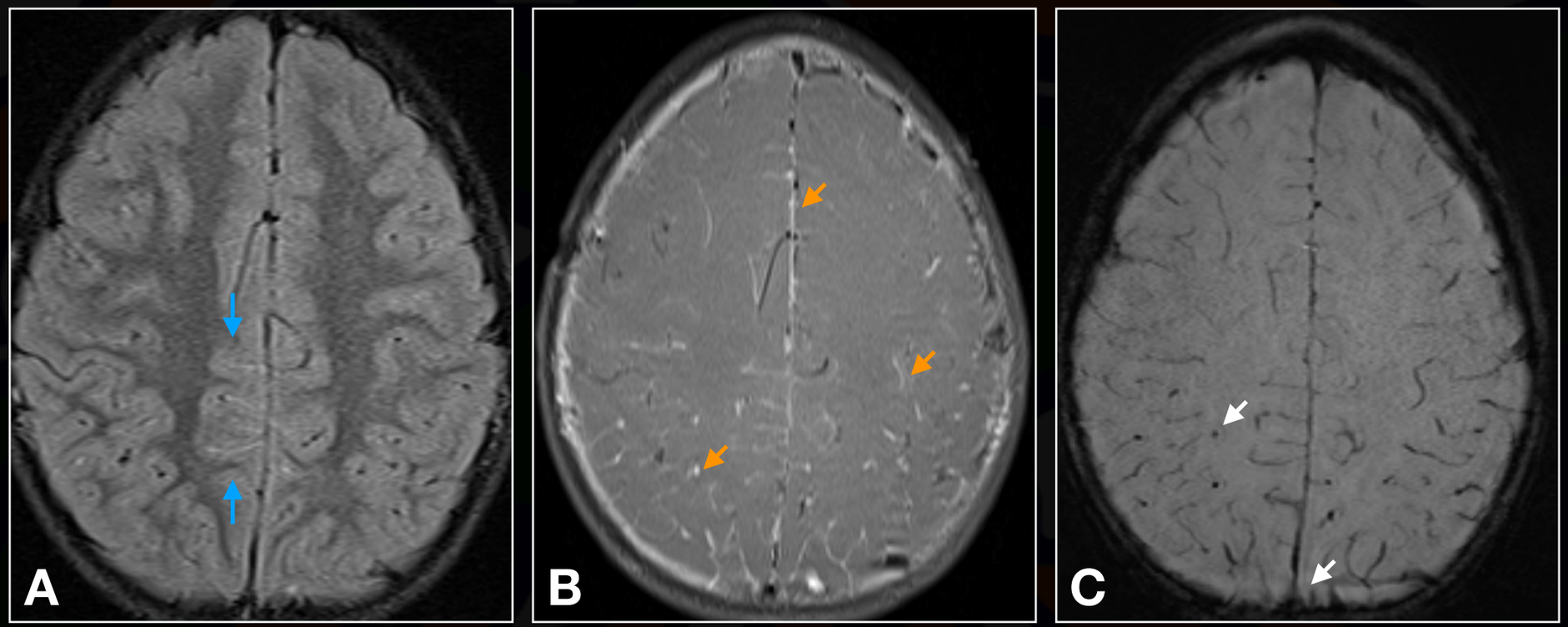


A, and C, T2-FLAIR imaging with non-suppression within the leptomeninges (→). *B*, and *D*, SWI demonstrates a near complete "absence" of the normally present hypointense cortical veins; recall that a normal SWI has hypointense venous structures due to deoxyhemoglobin iron (a paramagnetic substance) acting as an intrinsic contrast agent.

In this case, this patient was intubated and the FLAIR and SWI findings were due to hyper-oxygenation. The cortical veins are obviously not absent, but are less noticeable because they are hyperintense (→) compared to normal SWI imaging and they blend in with the neutral parenchyma. This hyperintensity is secondary to hyper-oxygenation which leads to an increased amount oxyhemoglobin in cortical veins and venous structures. Again, recall that oxyhemoglobin is diamagnetic and it shields the iron in blood from the magnetic field, which minimizes the susceptibility and T2* effects. Notice the hyperintense signal in the confluence of sinuses (→).

Diagnosis: Hyper-oxygenation

Case 2

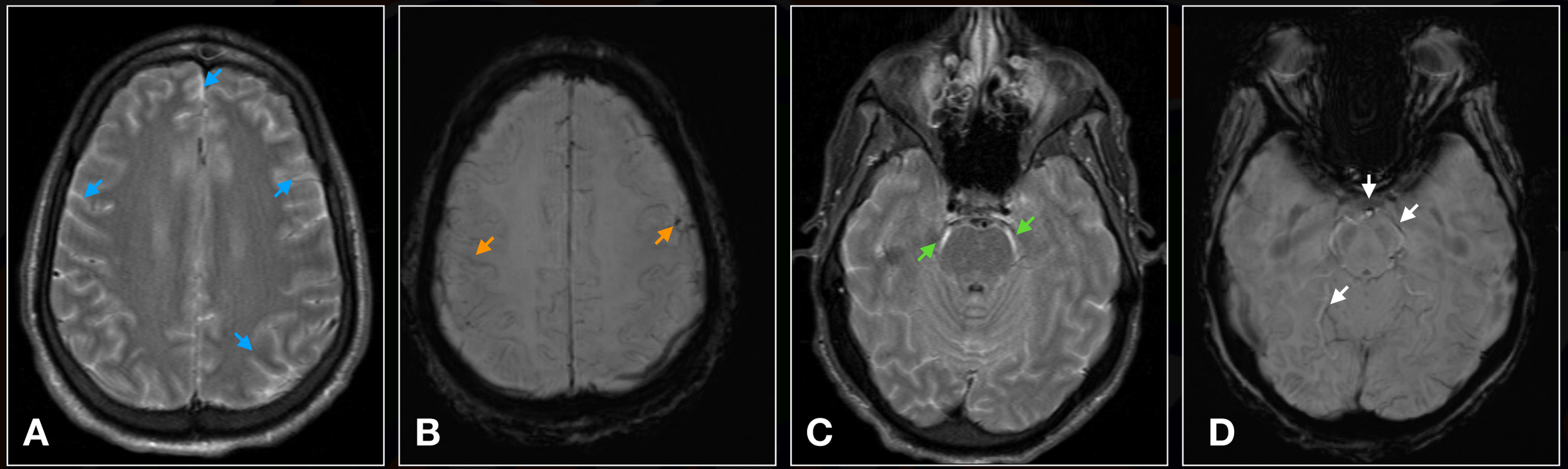


A, T2-FLAIR imaging with non-suppression within the leptomeninges (→). *B*, Post-contrast T1 imaging demonstrates diffuse enhancement of leptomeninges (→). This patient was extremely sick and intubated, with clinical concern for infection. Given the clinical scenario along with FLAIR and T1 post-contrast imaging, it is clear that this patient has an infectious meningitis.

However, this case can be used to illustrate that the cortical veins and dural venous sinuses are hypointense (→) on SWI (*C*), which excludes hyper-oxygenation as the cause for FLAIR non-suppression. Again, hyper-oxygenation leads to diamagnetic oxyhemoglobin in veins and venous structures, which shields the iron in blood from the magnetic field, which minimizes the susceptibility and T2* effects. So, the absence of hyper intense cortical veins indicates the absence of hyper-oxygenation.

Diagnosis: Meningitis

Case 3



A, and C, T2-FLAIR imaging with extensive non-suppression throughout the supratentorial sulci (→) and perimesencephalic cisterns (→). *B*, and *D*, SWI demonstrates venous hypointensity (→) indicative of deoxyhemoglobin iron within cortical veins and dural venous sinuses. The hyperintense vessels are the basilar artery and likely branches of the PCAs (→).

This patient was obtunded with a fever and elevated WBC.

Diagnosis: Bacterial meningitis

Unknown Case Challenge

As discussed on the prior cases, utilize the following technique to provide a confident diagnosis on the unknown cases:

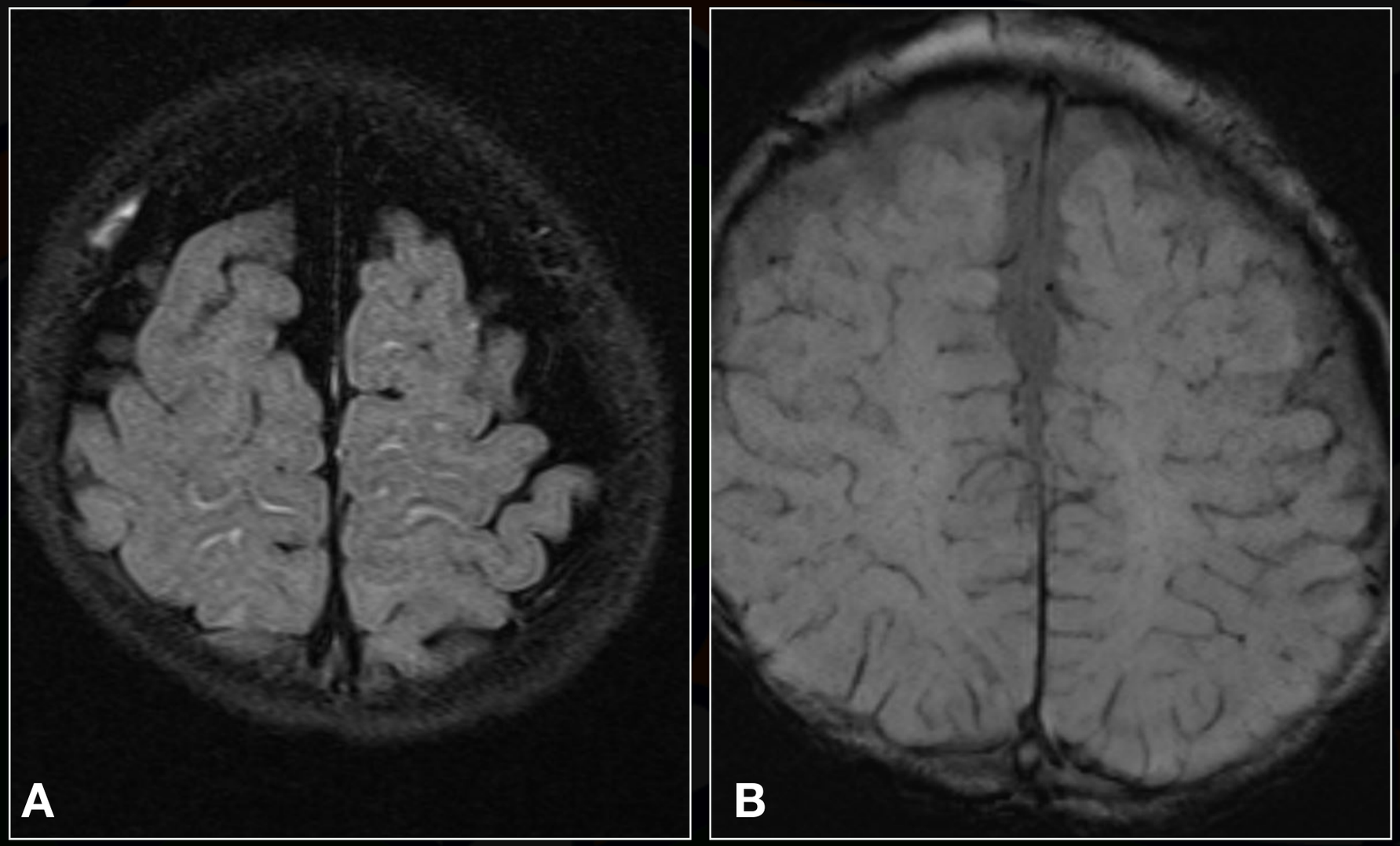
1. Is there CSF non-suppression on T2-FLAIR? If yes, then assess the cortical veins and dural venous sinuses on SWI.

2. Are the cortical veins and dural venous sinuses hypointense or hyperintense on SWI?

- If hypointense, then think normal arterial oxygen level, which means normal amount of dissolved free oxygen within the CSF.

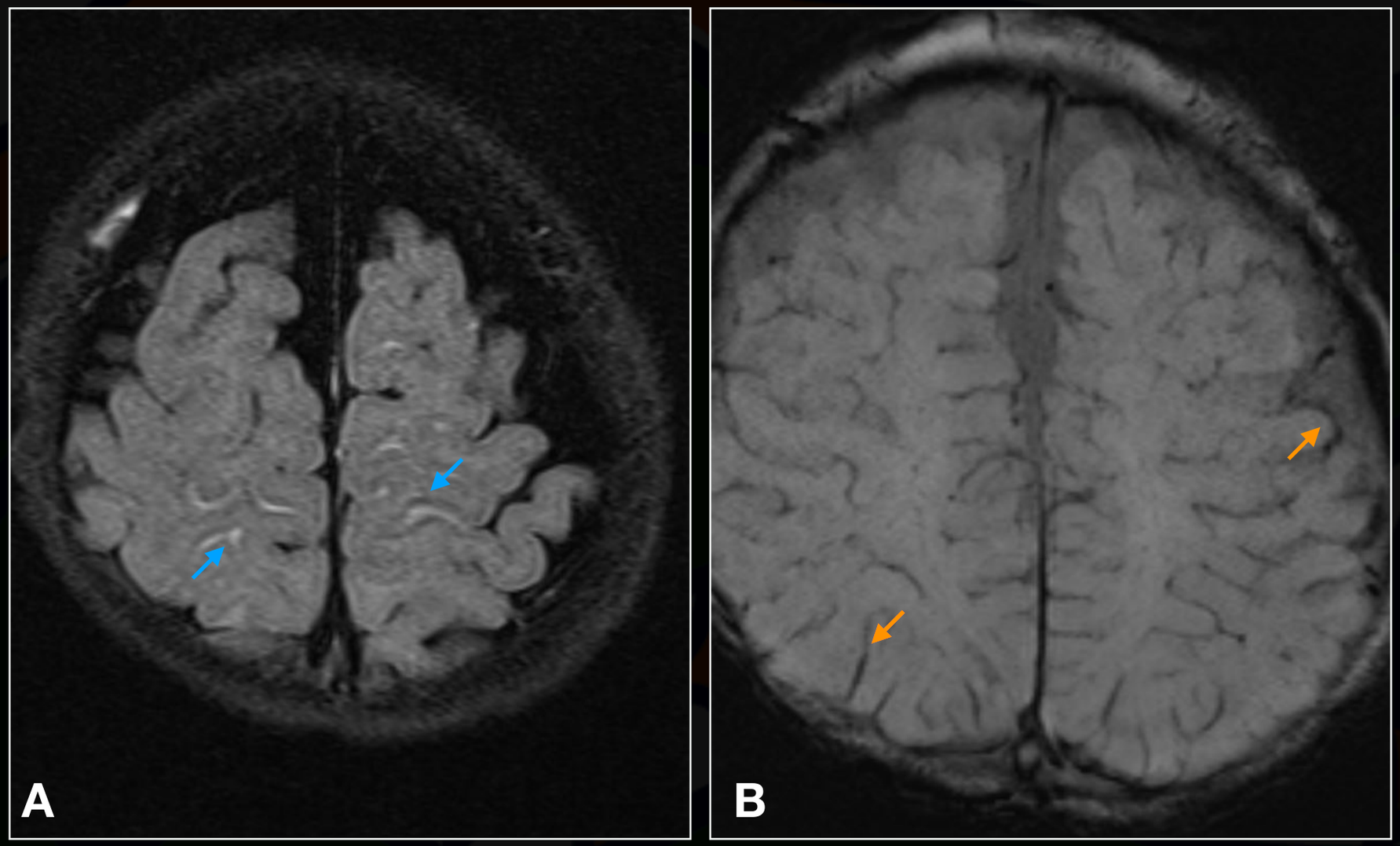
- If hyperintense, then think hyper oxygenation leading to increased dissolved free oxygen in the CSF, which results in an increased T1 shortening effect in the CSF.

1st Unknown Case



Intubated patient. Rule out meningitis.

Meningitis

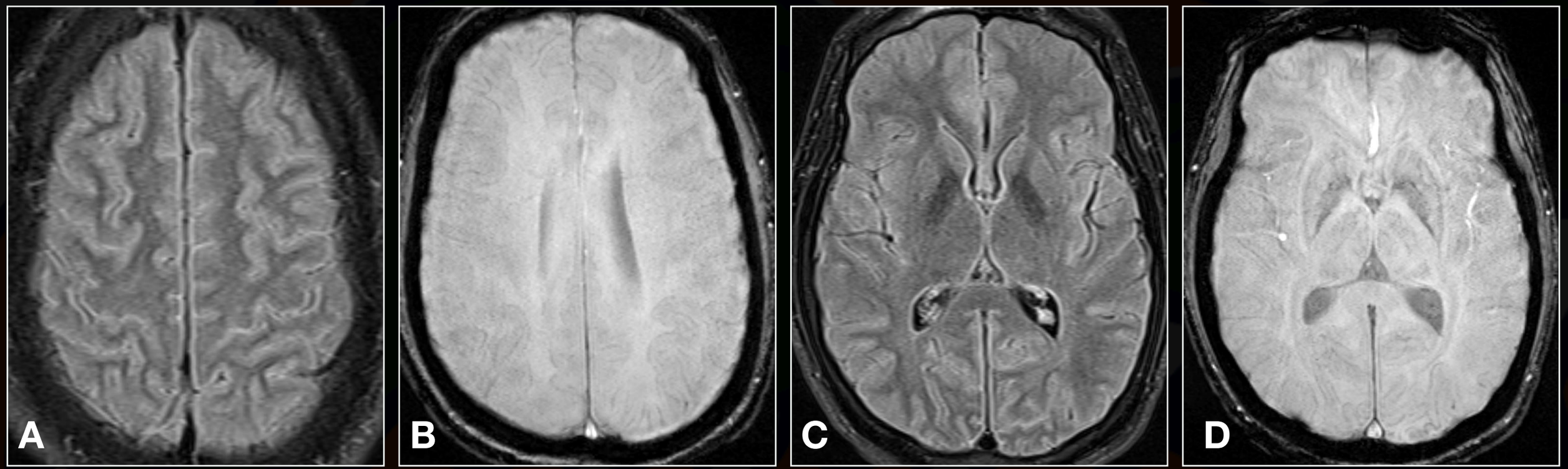


A, T2-FLAIR imaging with non-suppression throughout the convexal sulci (→).

B, SWI demonstrates venous hypointensity (→) indicative of deoxyhemoglobin iron within cortical veins and dural venous sinuses.

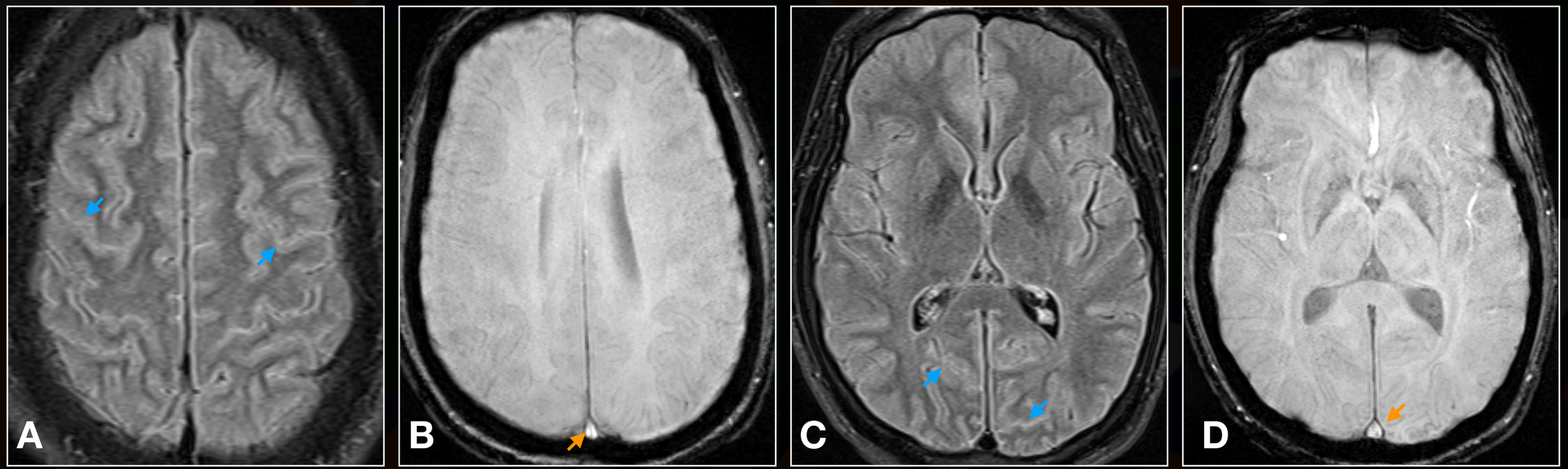
Diagnosis: Meningitis

2nd Unknown Case



Intubated patient. Rule out meningitis.

Hyper-oxygenation



A, and C, T2-FLAIR imaging with non-suppression throughout the supratentorial sulci (→).

B, and *D*, SWI demonstrates dural venous sinus hyperintensity (→) indicative of oxyhemoglobin shielding effect; cortical veins are also not well visualized due to oxyhemoglobin shielding effect.

Diagnosis: Hyper-oxygenation

Conclusion

Medicine is hard. Patients often present with confusing or conflicting signs/ symptoms.

Radiology is very hard. Imaging appearance of benign and pathologic processes can be *near* identical.

Clinical history is key; particularly when the imaging differential is broad.

Understanding physics concepts for certain imaging sequences will provide the astute neuroradiologist clues in establishing a confident diagnosis amongst a disparate differential.

Key SWI concepts:

- Arterial oxygen in the oxyhemoglobin shields (diamagnetic effect) the iron in arterial blood such that the susceptibility and T2* effects are only seen in deoxyhemoglobin-predominant venous blood.
- Hyper-oxygenation leads to oxyhemoglobin within both the arterial and venous system, which produces the diamagnetic effect throughout the vessels.

Key T2-FLAIR concept:

- Increased arterial oxygenation leads to increased dissolved free oxygen in the CSF, which is demonstrates slightly increased T1 shortening relative to normal CSF.

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