



Magnetic Resonance Imaging Physics in Brain Tumor Imaging: A Primer for Neurosurgeons

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Key words

- Diffusion imaging
- DTI
- Functional mapping
- Glioblastoma
- Glioma
- Magnetic resonance imaging
- Perfusion imaging

Abbreviations and Acronyms

ADC: Apparent diffusion coefficient
AI: Artificial intelligence
BOLD: Blood-oxygen-level-dependent
B₀: Main static magnetic field
Cho: Choline
CSF: Cerebrospinal fluid
DSC: Dynamic susceptibility contrast
DTI: Diffusion tensor imaging
DWI: Diffusion-weighted imaging
FLAIR: Fluid-attenuated inversion recovery
fMRI: Functional magnetic resonance imaging
GBM: Glioblastoma
GRE: Gradient-recalled echo
IDH: Isocitrate dehydrogenase
MRI: Magnetic resonance imaging
MRS: Magnetic resonance spectroscopy
NAA: N-acetylaspartate
rCBV: Relative cerebral blood volume
RF: Radiofrequency
SWI: Susceptibility-weighted imaging
TR: Repetition time
TE: Echo time
T1: Longitudinal relaxation time
T2: Transverse relaxation time
T2*: Effective transverse relaxation

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Magnetic resonance imaging (MRI) is central to the management of brain tumors and is deeply integrated into the neurosurgical workflow. From initial diagnosis through surgical planning and postoperative assessment, MRI guides nearly every stage of care. Yet the images that inform these decisions are shaped by underlying physical principles that may not be fully appreciated in clinical practice. This review provides a comprehensive and accessible overview of MRI physics as it applies to brain tumor imaging, with a focus on clinical relevance for neurosurgeons. We begin with core concepts such as spin behavior, relaxation mechanisms, and image formation and explain how these principles translate into the contrast mechanisms used in common and advanced imaging sequences. Key modalities, including T1-weighted, T2-weighted, fluid-attenuated inversion recovery, diffusion, perfusion, and functional imaging, are discussed in terms of what they reveal, how they operate, and the limitations that must be considered. We examine how these tools support surgical decision-making, including functional mapping, tractography, and intraoperative navigation, while also addressing common pitfalls such as pseudoprogression and imaging artifacts. The review concludes by highlighting emerging technologies such as artificial intelligence-based segmentation, ultra-high-field MRI, quantitative imaging, and radiomics, all of which may shape the future of neurosurgical imaging. For the modern neurosurgeon, fluency in MRI physics is not merely academic; it is essential for accurate interpretation, effective collaboration with radiology, and safer, more personalized surgical care.

INTRODUCTION

Magnetic resonance imaging (MRI) is integral to the management of brain tumors, guiding neurosurgeons from diagnosis through preoperative planning, intraoperative navigation, and postoperative assessment. While often viewed as a radiologic tool, MRI has become inseparable from modern neurosurgical practice. Beyond anatomical visualization, it offers insights into tumor biology through diffusion, perfusion, spectroscopy, and functional imaging.¹ A fundamental understanding of MRI physics empowers neurosurgeons to interpret images more critically, recognize limitations, and collaborate effectively with radiology colleagues.²

While several reviews have addressed MRI physics for nonradiologist physicians and the applications of MRI in brain tumor

imaging,³⁻⁴ our work is distinct in being specifically tailored to neurosurgeons. Rather than focusing solely on the technical aspects of image acquisition or broad oncologic applications, this review emphasizes how the underlying physics of MRI sequences directly inform neurosurgical decision-making, from operative planning to intraoperative navigation and postoperative assessment. By bridging fundamental biophysical concepts with surgical strategies, this article aims to provide a practical, accessible resource uniquely suited for a neurosurgical audience.

FOUNDATIONAL MRI PHYSICS FOR NEUROSURGEONS

MRI is based on the magnetic properties of hydrogen protons, which are abundant in water and fat throughout the brain.⁵ When

a patient is placed inside the MRI scanner, a strong external magnetic field, known as B_0 , causes many of these protons to align either with or against the direction of the field.⁶ Slightly more protons adopt the lower energy orientation aligned with B_0 , producing a small net magnetization along the axis of the magnetic field.

In addition to aligning with the field, protons precess, or wobble, around the axis of B_0 . The speed of this precession is called the Larmor frequency, which is directly proportional to the strength of the magnetic field. At a magnetic field strength of 1.5 T, the Larmor frequency is approximately 64 MHz.⁷

To generate an MRI signal, the scanner applies a radiofrequency (RF) pulse at the Larmor frequency.⁸ This pulse tips the net magnetization vector away from its alignment with B_0 and into the transverse plane. After the RF pulse is turned off, the protons begin to relax and return to their original alignment.⁶ As they do so, they emit signals that are detected by receiver coils and used to form the MRI image. Relaxation occurs

through two processes that happen simultaneously: T1 and T2 (Figure 1).

T1 Relaxation

T1 relaxation, also known as longitudinal relaxation, refers to the recovery of the magnetization along the B_0 axis. This occurs as protons transfer energy to their surrounding environment, a process influenced by the tissue's molecular structure and composition.⁹ Tissues with short T1 times, such as fat, return to equilibrium quickly and appear bright on T1-weighted images. In contrast, tissues with long T1 times, such as cerebrospinal fluid (CSF), recover more slowly and appear dark.⁹

T2 Relaxation

T2 relaxation, also known as transverse relaxation, describes the gradual loss of phase coherence among protons in the transverse plane. Immediately after the RF pulse, the protons are synchronized and precessed together, creating a strong signal. Over time, local magnetic interactions and tissue microstructure cause

them to fall out of sync, resulting in signal decay.¹⁰ Tissues with more complex structures, such as cellular tumors, tend to have shorter T2 times due to faster dephasing.¹ Water-rich tissues, including edema and cystic components, retain coherence longer and appear bright on T2-weighted images. A related parameter, T2*, reflects additional dephasing caused by magnetic field inhomogeneities. Because it is sensitive to differences in magnetic susceptibility, T2*-weighted imaging is particularly helpful for detecting hemorrhage, calcification, and air.¹¹

k-Space

The signals produced during relaxation are measured as voltages in the receiver coils and stored in a data matrix known as k-space.¹² k-Space holds spatial frequency information, not direct image data. An inverse Fourier transform is used to convert these data into an image.¹ The center of k-space determines image contrast and overall signal intensity, while the outer edges define spatial resolution and sharpness. The more fully

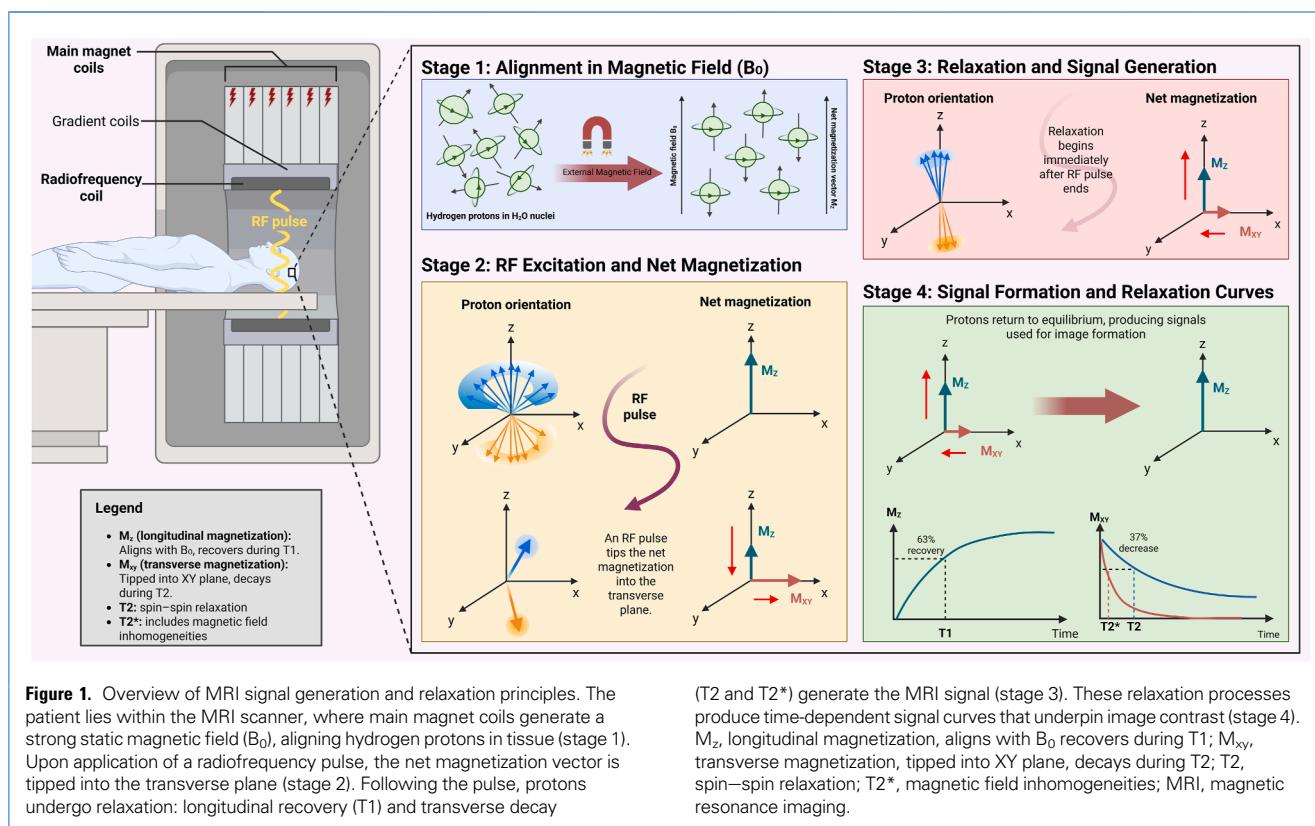
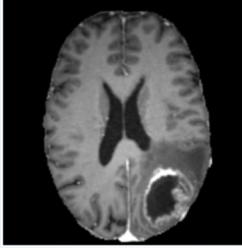
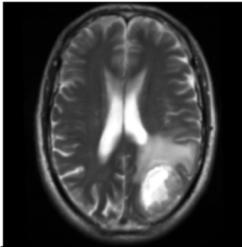
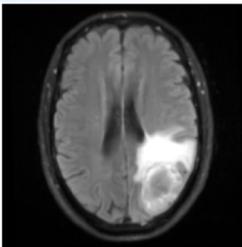
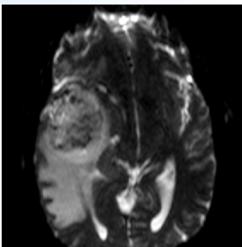


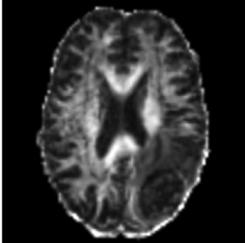
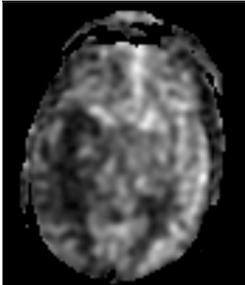
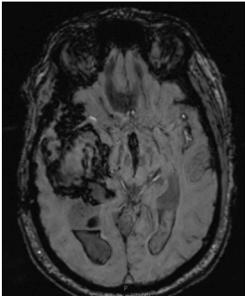
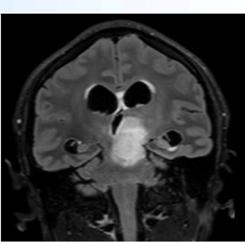
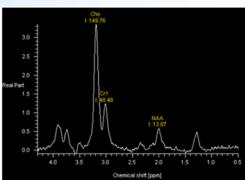
Table 1. Key MRI Modalities in Brain Tumor Imaging

Modality	What It Measures	Typical Tumor Findings	Clinical Utility for Neurosurgeons	Example MRI
T1-weighted imaging	T1 relaxation; anatomy and contrast enhancement with gadolinium	Enhancing tumor core; nonenhancing in low-grade gliomas	Identifies tumor margins; guides biopsy and resection planning	
T2-weighted imaging	T2 relaxation; water content	Hyperintense signal in edema, tumor, and cystic areas	Maps edema and nonenhancing tumor; informs surgical field	
FLAIR	T2 with CSF suppression	Highlights peritumoral edema and nonenhancing tumor	Enhances lesion visibility; distinguishes infiltrative margins	
DWI	Random water motion; ADC quantifies diffusivity	Restricted diffusion in high-grade tumors; high ADC in edema/necrosis	Assesses cellularity; refines biopsy targeting	

MRI, Magnetic resonance imaging; T1, Longitudinal relaxation time; T2, Transverse relaxation time; FLAIR, Fluid-attenuated inversion recovery; CSF, Cerebrospinal fluid; DWI, Diffusion-weighted imaging; ADC, Apparent diffusion coefficient; DTI, Diffusion tensor imaging; FA, Fractional anisotropy; DSC, Dynamic susceptibility contrast; DCE, Dynamic contrast enhanced; rCBV, Relative cerebral blood volume; SWI, Susceptibility-weighted imaging; T2*, Effective transverse relaxation; GBM, Glioblastoma; MRS, Magnetic resonance spectroscopy; NAA, N-acetylaspartate; fMRI, Functional magnetic resonance imaging; BOLD: Blood-oxygen-level-dependent.

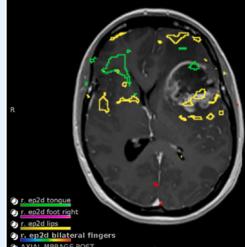
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Modality	What It Measures	Typical Tumor Findings	Clinical Utility for Neurosurgeons	Example MRI
DTI	Directional water motion; white matter tract integrity	Reduced FA in infiltrated tracts; displaced or disrupted fibers	Tractography maps eloquent tracts for surgical avoidance	
Perfusion (DSC/DCE)	Blood flow, volume (rCBV), and permeability (Ktrans)	High perfusion in aggressive tumors; low perfusion in necrosis or treatment effect	Grades tumors, assesses recurrence, and selects biopsy targets	
SWI/T2*	Magnetic susceptibility differences (blood, calcium)	Microhemorrhage in GBM; calcifications in oligodendrogloma	Detects hemorrhage, venous structures; helps anticipate surgical risks	
MRS	Metabolic profile: NAA, choline, lactate, lipids	High choline:NAA ratio in tumors; lactate in necrotic tissue	Identifies tumor metabolism; supports characterization and targeting	 

Continues

Table 1. Continued

Modality	What It Measures	Typical Tumor Findings	Clinical Utility for Neurosurgeons	Example MRI
fMRI	BOLD signal during tasks; maps functional cortex	Activations in motor/language areas near tumor	Localizes eloquent cortex; informs approach, especially without awake mapping	

MRI, Magnetic resonance imaging; T1, Longitudinal relaxation time; T2, Transverse relaxation time; FLAIR, Fluid-attenuated inversion recovery; CSF, Cerebrospinal fluid; DWI, Diffusion-weighted imaging; ADC, Apparent diffusion coefficient; DTI, Diffusion tensor imaging; FA, Fractional anisotropy; DSC, Dynamic susceptibility contrast; DCE, Dynamic contrast enhanced; rCBV, Relative cerebral blood volume; SWI, Susceptibility-weighted imaging; T2*, Effective transverse relaxation; GBM, Glioblastoma; MRS, Magnetic resonance spectroscopy; NAA, N-acetylaspartate; fMRI, Functional magnetic resonance imaging; BOLD: Blood-oxygen-level-dependent.

the high-frequency components in k -space are sampled, the higher the image resolution.¹²

Repetition Time and Echo Time

Two timing settings largely determine MRI contrast. Repetition time (TR) is how often the scanner excites the tissue, while echo time (TE) is how long it waits before collecting the signal.¹³ Short TR and TE emphasize T1 contrast, highlighting fat and gadolinium-enhancing tumor. Longer TR and TE emphasize T2 contrast, making edema, cysts, and infiltrative tumor margins appear bright.

Spin Echo versus Gradient Echo Sequences

The choice of sequence also shapes the image. Spin echo uses a refocusing pulse, producing cleaner T1 and T2 images with fewer distortions. Gradient echo (GRE) is faster but more sensitive to magnetic field imperfections,¹⁴ making it ideal for detecting blood, calcification, or microvascular changes. For neurosurgeons, this explains why GRE and T2* images are critical for identifying intratumoral hemorrhage and planning surgery near vascular structures.

To determine the precise origin of each signal within the brain, MRI relies on gradient magnetic fields. These gradients vary the magnetic field slightly across space, causing protons in different locations to precess at slightly different frequencies.¹ This allows the scanner to localize signals and generate a spatially

accurate image. The timing of RF pulses and gradient applications is controlled by the pulse sequence, which defines how the image will be weighted, whether it highlights T1, T2, or other properties. Parameters such as TR and TE are adjusted to emphasize specific types of tissue contrast.

KEY MRI MODALITIES IN BRAIN TUMOR IMAGING

MRI's versatility arises from multiple contrast mechanisms that can be targeted to reveal different aspects of brain tumors. Key modalities include conventional T1- and T2-weighted imaging and advanced techniques that probe water diffusion, tissue perfusion, magnetic susceptibility, metabolic content, and functional activity (Table 1).

T1, T2, and Fluid-Attenuated Inversion Recovery Imaging

T1-, T2-, and fluid-attenuated inversion recovery (FLAIR)-weighted imaging form the foundation of MRI in brain tumor evaluation. Together, these sequences provide complementary information on tumor extent, tissue composition, edema, and biological behavior, which is essential for diagnosis, surgical planning, and follow-up.

T1-weighted imaging highlights tissues based on differences in T1 relaxation. Fat- and protein-rich tissues appear bright, while fluid like CSF appears dark. After

gadolinium administration, regions with disrupted blood-brain barrier, such as enhancing tumor, neovascularity, or inflammation, show increased signal.¹⁵ Postcontrast T1-weighted images are critical for delineating tumor borders, selecting biopsy sites, and planning resection. Importantly, nonenhancing tumors, such as low-grade gliomas, may still be infiltrative, necessitating correlation with other sequences.

T2-weighted imaging emphasizes water content and highlights areas of edema or infiltrative tumor as hyperintense. It is especially useful in identifying tumor-associated changes beyond the enhancing core.¹⁵ FLAIR imaging, a T2-weighted variant, suppresses CSF signal to improve lesion visibility near ventricles and cortical surfaces. It enhances the detection of nonenhancing tumor, peritumoral edema, gliosis, and other subtle abnormalities.

In high-grade gliomas, T1 postcontrast enhancement is often surrounded by a broader T2/FLAIR hyperintense region, reflecting both edema and infiltrative tumor. This mismatch is crucial in planning resections beyond just the enhancing component. However, T2/FLAIR hyperintensity is nonspecific and may also arise from radiation effects, postoperative change, or demyelination.¹⁶

One notable imaging sign is the T2/FLAIR mismatch, where a lesion appears uniformly bright on T2 but shows a relatively dark core on FLAIR with only a

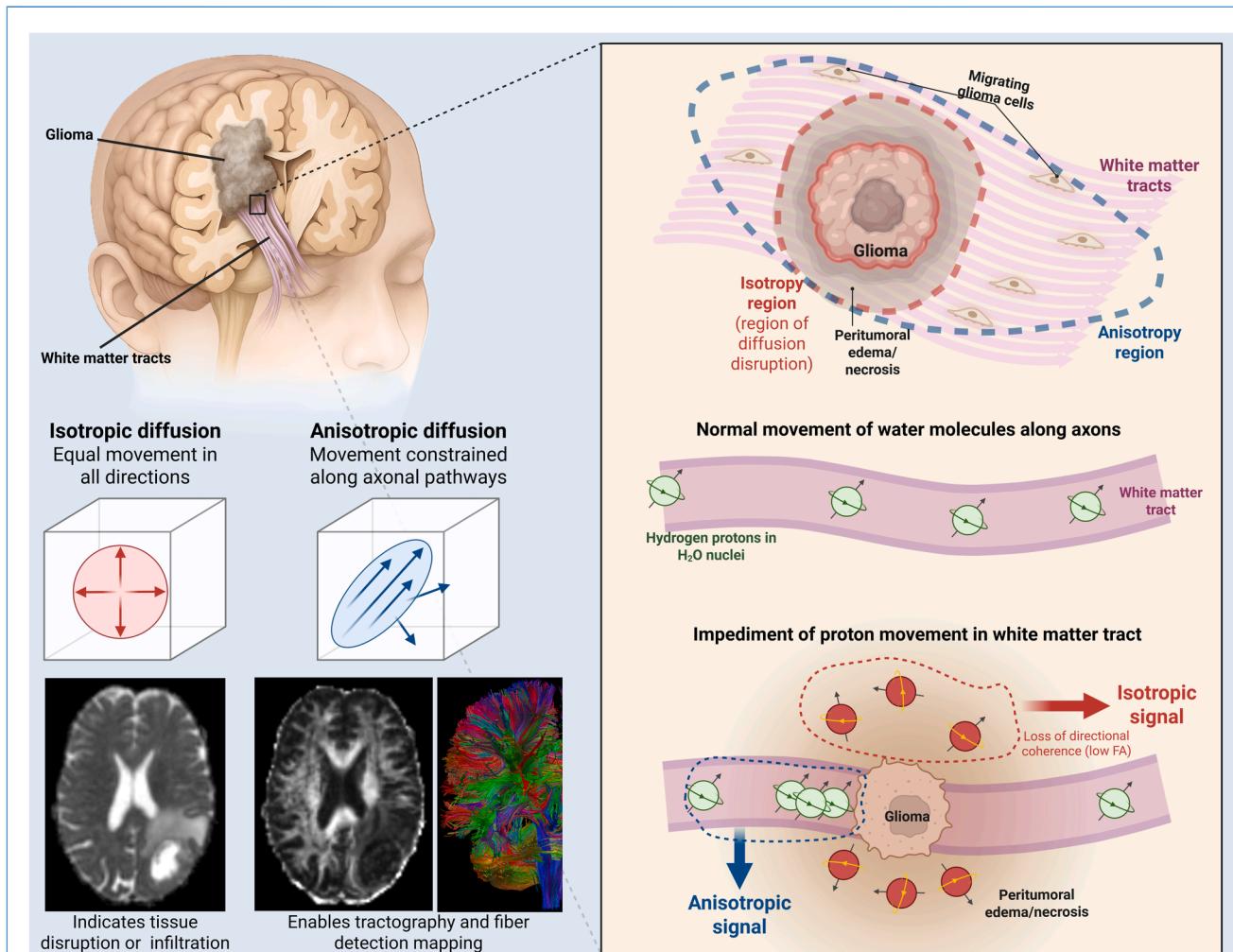


Figure 2. Illustration of isotropic and anisotropic diffusion patterns in relation to glioma infiltration. *Left:* In healthy brain tissue, water diffusion is anisotropic, preferentially constrained along white matter tracts, supporting tractography and fiber mapping. In contrast, in unstructured or damaged tissue (e.g. tumor or necrosis), diffusion becomes isotropic, occurring equally in all directions. *Right:* In gliomas, infiltrative tumor cells

disrupt white matter architecture, reducing directional coherence (fractional anisotropy). This results in a transition from anisotropic diffusion within intact tracts to isotropic diffusion in regions of peritumoral edema, necrosis, or infiltration. These diffusion characteristics are key for interpreting diffusion tensor imaging metrics and planning tumor resections. FA, fractional anisotropy.

hyperintense rim. This pattern suggests an isocitrate dehydrogenase (IDH)-mutant, *tp19q*-intact astrocytoma with 92% accuracy,¹⁷ and serves as a useful noninvasive biomarker in preoperative planning.¹⁵

These imaging observations have direct surgical implications. The concept of "FLAIREctomy," the resection of peritumoral FLAIR signal abnormality, has emerged in response to histopathological evidence that these areas often contain infiltrative tumor.¹⁸⁻²⁰ Recent studies demonstrate that the extent of FLAIR abnormality resection correlates

with improved survival, even when the contrast-enhancing tumor is already maximally removed.²¹⁻²³ While complete resection of the enhancing tumor remains a standard goal in glioblastoma (GBM), the survival advantage increases with greater volumetric resection of nonenhancing tumor as well.²⁴⁻²⁶ In light of these findings, the Response Assessment in Neuro-Oncology resect criteria offer a standardized framework to quantify and report resection extent in both enhancing and nonenhancing tumor compartments.²⁷ Future clinical trials are expected to incorporate these

criteria to better evaluate the impact of resection on survival and response to therapy.²⁸

Research has shown that even modest resection of FLAIR hyperintensity—removing as little as 20–45% of this region—can improve outcomes in GBM.^{22,23} Moreover, residual nonenhancing tumor volume has emerged as a significant predictor of survival, reinforcing the value of supratotal resection strategies that target both the contrast-enhancing and nonenhancing components.^{29,30} This has prompted the incorporation of molecular data into resection decisions. For

instance, younger patients with IDH-wildtype GBM derive survival benefit from aggressive resection of both enhancing and nonenhancing tumor volumes.³¹

In parallel, advances in imaging and intraoperative techniques are helping translate these insights into clinical practice. Preoperative and intraoperative molecular profiling technologies are under development to assist neurosurgeons in tailoring resection to tumor biology in real time.³²⁻³⁵ Aggressive strategies, such as anterior temporal lobectomy or tailored lobectomies in noneloquent regions, have demonstrated significantly improved progression-free and overall survival compared to conventional resection of the enhancing lesion alone.³⁶ A critical consideration is whether infiltrative tumor, which is often visualized as nonenhancing FLAIR abnormality, will lead to functional deficits if left behind, either at baseline or upon progression. Emerging evidence suggests that resecting these regions may delay tumor progression and the onset of neurological decline, supporting a proactive surgical approach when safe. Nonetheless, these potential gains must be balanced against the immediate risk of postoperative neurological deficits, which are themselves associated with worse survival.^{37,38} The use of advanced intraoperative navigation, cortical and subcortical mapping, and diffusion imaging can guide maximal safe resection while preserving function.³⁹ Achieving the balance of aggressively reducing tumor burden without inducing new deficits remains central to optimizing GBM outcomes.^{40,41}

Susceptibility Imaging: SWI and T2*

Susceptibility imaging detects local magnetic field distortions caused by substances like blood products, calcium, or air. These distortions are not always visible on standard T1 or T2 images but can be critical in understanding tumor composition and surgical risk.

T2-weighted gradient echo (T2*-GRE) imaging is sensitive to these susceptibility effects and highlights signal loss in areas with hemosiderin, deoxyhemoglobin, or calcification.⁴² It helps detect internal hemorrhage or mineralization but has limited resolution and sensitivity.

Susceptibility-weighted imaging (SWI) builds on T2* by incorporating both magnitude and phase information, significantly enhancing sensitivity.⁴³ SWI can reveal small veins, microbleeds, calcifications, and hemorrhagic components within tumors. This is particularly relevant in high-grade gliomas, which often show intratumoral hemorrhage due to fragile neovasculature.

Clinically, SWI can help differentiate blood from calcification, an important distinction in lesions such as oligodendroglomas (calcification) versus GBMs or metastases (hemorrhage).⁴⁴ It also offers detailed venous anatomy, which may influence surgical planning, especially when cortical draining veins are at risk. Postoperatively, SWI is useful for detecting residual blood products and complications like venous infarction.

Diffusion Imaging (DWI, DTI)

Diffusion-weighted imaging (DWI) measures the random motion of water molecules within tissue and is a cornerstone of brain tumor evaluation. In tumors with high cellularity, such as GBMs and lymphomas, densely packed cells restrict water diffusion, leading to high signal intensity on DWI and low values on apparent diffusion coefficient (ADC) maps.⁴⁵ In contrast, areas with free water movement, such as edema or necrosis, show low DWI signal and high ADC values.^{45,46}

The clinical use of DWI evolved from earlier work in the 1960s exploring water mobility in tissue.⁴⁷ By the 1980s, diffusion imaging was introduced into MRI practice, producing maps where each voxel displayed a single intensity value that reflected overall water motion.⁴⁸ While this allowed detection of restricted diffusion, it could not reveal the direction of water movement, an important limitation when evaluating white matter architecture.

This limitation was addressed in the 1990s with the development of diffusion tensor imaging (DTI), which extended the capabilities of DWI by capturing diffusion in multiple directions.⁴⁹ DTI enabled clinicians to characterize not just the magnitude of water motion but also its preferred pathways (Figure 2). This is crucial in the brain, where water moves directionally along axonal

fibers. This directional data laid the foundation for modern tractography, now widely used to visualize and preserve white matter networks in surgical planning.

DWI remains valuable for identifying high-cellularity tumor components, guiding biopsies, and assessing tumor infiltration. However, artifacts such as T2 shine-through and T2 blackout can confound interpretation, necessitating correlation with ADC maps and conventional sequences.⁴⁵ Diffusion properties also offer clues to tumor grade and genotype. For example, IDH-mutant gliomas typically show higher ADC values than IDH-wildtype counterparts, reflecting lower cell density and less restricted diffusion.^{46,50}

DTI further augments neuro-oncologic imaging by quantifying white matter integrity through metrics such as fractional anisotropy and mean diffusivity.¹⁵ However, traditional reliance on FA alone can be misleading due to inconsistent responses across tumor types.⁵¹ More advanced analysis decomposes the diffusion tensor into isotropic and anisotropic components. The isotropic component represents increased water diffusion often linked to edema, while the anisotropic component captures directionally constrained diffusion along axons, reflecting intact or infiltrated white matter.⁵² This decomposition enhances the characterization of glioma invasion patterns, reveals intratumoral heterogeneity, and correlates with progression-free survival and recurrence risk.⁵³⁻⁵⁶

Clinically, DTI has become integral to preoperative and intraoperative neurosurgical workflows. Tractography reveals how tumors displace, infiltrate, or disrupt eloquent white matter pathways, such as the corticospinal tract or language networks, guiding safer and more effective resections.^{51,52} Incorporating DTI into preoperative planning reduces the likelihood of postoperative neurologic deficits, especially for lesions near critical structures.

Multiparametric MRI strategies incorporating DTI have revolutionized surgical precision, allowing neurosurgeons to map and target infiltrative tumor margins that extend beyond contrast-enhancing

borders. Unlike conventional imaging, which may underestimate tumor spread, DTI highlights regions of white matter invasion across the entire brain. This enables more extensive but tailored resections, particularly in GBM, where residual infiltrative tumor predicts poorer outcomes.⁵³⁻⁵⁵ DTI can be integrated into most MRI scanners, enhancing accessibility.

Postoperatively, DTI continues to add value by monitoring changes in white matter integrity and tumor progression, informing adjuvant therapy and long-term planning. As GBM is a dynamic and invasive disease, this ongoing evaluation supports adaptive strategies that can improve functional outcomes and overall survival.

Perfusion Imaging (Dynamic Susceptibility Contrast/Dynamic Contrast Enhanced/Arterial Spin Labeling)

Perfusion-weighted imaging assesses how blood flows through brain tissue, offering functional insights into tumor vascularity, microvascular density, and capillary permeability,⁵⁷ which are often altered in gliomas and metastases. Unlike standard MRI, which shows anatomy, perfusion techniques capture the hemodynamic behavior of tumors.

The foundation of perfusion-weighted imaging dates back to early efforts in the 1960s and 1980s that used radioactive tracers to measure cerebral blood flow.⁵⁸⁻⁶⁰ These approaches were later replaced by safer and more practical MRI-based methods. In the 1990s, two major contrast-enhanced MRI techniques were developed: dynamic susceptibility contrast (DSC) and dynamic contrast-enhanced imaging. These innovations allowed clinicians to track how gadolinium-based contrast moved through the brain in real time, without radiation, making perfusion imaging more widely applicable and clinically useful.

In DSC-MRI, a gadolinium bolus is injected, and rapid T₂*-weighted images are acquired.⁶¹ As contrast passes through the brain, susceptibility effects cause a temporary signal drop, from which parameters like relative cerebral blood volume (rCBV) can be calculated. The measure is considered “relative” because it reflects blood volume in the tumor

compared to normal-appearing white matter, rather than providing an absolute value. High rCBV indicates increased vascularity, typical of high-grade gliomas, and can help distinguish them from lower grade lesions.⁶² Another metric, percent signal recovery, reflects capillary integrity and vascular compliance.

Dynamic contrast-enhanced MRI complements DSC using T₁-weighted imaging to track gadolinium leakage over time.⁶⁰ It provides estimates of vascular permeability (K-trans-) and extracellular volume fraction (ve),⁶⁰ which are especially relevant in tumors that disrupt the blood–brain barrier.

A third method, arterial spin labeling, emerged as a noncontrast alternative that magnetically labels the patient’s own arterial blood to estimate cerebral blood flow.^{63,64} Arterial spin labeling is particularly useful for patients who cannot receive contrast, such as those with renal impairment. However, it is less commonly used in standard neuro-oncology protocols because of its lower signal-to-noise ratio (the perfusion signal is relatively weak compared to background noise) and longer acquisition times.^{65,66}

Clinically, perfusion imaging plays a vital role in tumor grading. High rCBV suggests more aggressive tumor biology, even in nonenhancing lesions, and may influence surgical or treatment decisions.⁶⁷ Perfusion imaging is also helpful in distinguishing tumor recurrence from treatment-related effects such as pseudoprogression or radiation necrosis.¹⁶ High perfusion favors recurrent tumor, whereas low perfusion supports a treatment effect.

From a surgical perspective, regions with elevated rCBV or abnormal permeability can guide biopsy targeting and improve the diagnostic yield,^{68,69} particularly in heterogeneous or partially treated tumors.

Magnetic Resonance Spectroscopy

Magnetic resonance spectroscopy (MRS) and functional MRI (fMRI) offer complementary insights beyond structural imaging, informing tumor biology and functional anatomy. While not used routinely in all cases, both techniques can meaningfully guide surgical decision-

making when applied in the appropriate clinical context.

MRS emerged in the 1980s as a clinical extension of nuclear MRS, which had previously been used only in chemistry laboratories.⁷⁰ By the early 1990s, single-voxel and multivoxel MRS techniques became available on clinical scanners, allowing *in vivo* measurement of brain metabolites.⁷¹ This development opened a new dimension in neuro-oncology: biochemical profiling of tumors. Tumors typically exhibit elevated choline (Cho) (reflecting increased membrane turnover) and reduced N-acetylaspartate (NAA) (a marker of neuronal integrity), with a high Cho:NAA ratio suggesting neoplasm.⁷² Additional peaks such as lactate and lipid can suggest necrosis and tumor aggression.⁷² These spectral patterns help characterize ambiguous lesions, identify aggressive tumor components, and refine biopsy targeting in radiographically heterogeneous masses. However, MRS has practical limitations including low spatial resolution, motion sensitivity, and interpretation challenges in the posttreatment setting.

Over the past decade, MRS and fMRI have undergone significant refinement, enhancing their clinical relevance in brain tumor management. One major advance in MRS has been the ability to noninvasively detect the oncometabolite 2-hydroxyglutarate, which serves as a surrogate marker for IDH mutations in gliomas.⁷³ This capability allows for molecular subtyping without the need for immediate tissue sampling, improving diagnostic precision. Additionally, high-resolution MRS and whole-brain spectroscopic imaging are now being used to monitor treatment response, with changes in Cho:NAA ratios and lactate peaks helping differentiate recurrent tumor from posttreatment effects.⁷⁴

Functional MRI

fMRI was developed in the early 1990s following the discovery of the blood-oxygen-level-dependent (BOLD) effect by Ogawa et al.⁷⁵ It revolutionized functional brain mapping by allowing noninvasive localization of eloquent cortex based on task-induced hemodynamic changes.⁷⁶ In neuro-oncology, task-based fMRI can identify motor, language, and sensory

areas that are at risk during tumor resection. This is especially valuable for tumors adjacent to presumed eloquent cortex, allowing neurosurgeons to plan safer resections. For patients unable to undergo awake mapping, fMRI serves as a noninvasive alternative to localize functionally critical regions.⁷⁷ It also complements DTI tractography by connecting cortical activation zones with subcortical fiber pathways.⁷⁸

Despite its strengths, fMRI has known limitations. It depends on patient cooperation and reliable task performance. Accuracy is reduced near air–tissue interfaces and in tumor-infiltrated cortex, where neurovascular uncoupling may blunt the BOLD signal. Moreover, BOLD fMRI reflects vascular responses rather than direct neuronal activity, and its spatial resolution may not resolve adjacent functional areas with precision.

fMRI has likewise evolved with the broader adoption of resting-state fMRI, which maps functional networks without the need for patient task performance, especially valuable for uncooperative or neurologically impaired patients.⁷⁹

Moreover, studies integrating task-based fMRI with intraoperative mapping have confirmed its accuracy in localizing motor and language cortices, reinforcing its role in surgical planning.⁸⁰ However, attention has shifted toward the challenge of neurovascular uncoupling in tumor-infiltrated cortex, which can blunt BOLD signal responses and yield false negatives.⁸¹ Multimodal approaches that combine fMRI with perfusion or positron emission tomography imaging are increasingly used to mitigate this limitation.^{82,83} Recently, artificial intelligence (AI) has begun to assist in analyzing fMRI data, reducing interobserver variability and enhancing prediction of postoperative functional outcomes^{84,85} (Table 2).

SURGICAL INTEGRATION AND DECISION-MAKING

MRI has become a cornerstone of neurosurgical practice, not just for visualizing tumor anatomy but also for refining surgical strategy, anticipating complications, and navigating intraoperative challenges. A working knowledge of MRI physics,

especially the principles underlying contrast generation and spatial localization, empowers neurosurgeons to interpret scans beyond their surface appearance and apply imaging insights more effectively in the operating room.⁸⁶

Surgical planning traditionally centers on postcontrast T1-weighted sequences to delineate the enhancing tumor core. However, enhancement merely reflects the disruption of the blood–brain barrier, not the true extent of neoplastic infiltration. T2-weighted and FLAIR sequences, sensitive to tissue water content, frequently reveal a broader zone of peritumoral abnormality, often representing nonenhancing infiltrative tumor.⁸⁷ Recognizing this distinction is crucial, particularly in diffuse gliomas, where resection guided solely by enhancement may underestimate tumor burden. Incorporating these nonenhancing but biologically active regions into surgical goals can extend resection margins while preserving function.

Advanced MRI sequences offer additional physiologic context. DWI and ADC maps highlight areas of restricted water diffusion,⁸⁸ often correlating with

Table 2. MRI Features by Brain Tumor Type

Tumor Type	T1-Weighted	T2/FLAIR	DWI/ADC	Perfusion (rCBV)	SWI	MRS
Glioblastoma	Hypointense; enhances post-contrast	Hyperintense core with peritumoral edema	Restricted diffusion in solid components; low ADC	Elevated rCBV in enhancing areas	Microhemorrhages visible	↑↑ Cho, ↓ NAA, lipid/lactate +
Low-grade glioma (IDH-mutant)	Isointense to hypointense; often nonenhancing	Homogeneous hyperintensity	No restricted diffusion; higher ADC	Low-to-normal rCBV	Typically negative	↑ Cho, ↓ NAA
Primary CNS lymphoma	Homogeneously enhancing mass	Mild hyperintensity	Markedly restricted diffusion	Low-to-moderate rCBV	May show hemorrhage	↑ Cho, ↓ NAA
Metastasis	Well-defined; ring-enhancing lesions	Surrounding vasogenic edema	Restricted diffusion in viable tumor	Variable; often high in solid portions	Can show hemorrhage/calcification	↑ Cho, ↓ NAA
Meningioma	Isointense to cortex; strong enhancement	Variable	No significant restriction	Very high rCBV	May show calcifications	↑↑ Cho, ↓ NAA
Oligodendroglioma	Mixed signal; may calcify	Hyperintense with possible T2-FLAIR mismatch	No restriction; high ADC	Moderately elevated rCBV	Calcifications common	↑ Cho, ↓ NAA, myo-inositol +
Medulloblastoma	Isointense to hypointense; may enhance	Hyperintense	Restricted diffusion (dense cellularity)	High rCBV	May show hemorrhage	↑ Cho, ↓ NAA
Ependymoma	Isointense to hypointense; variable enhancement	Hyperintense	Mild restriction	Variable	Calcifications may be seen	↑ Cho, ↓ NAA, lipid +

T1, Longitudinal relaxation time; T2, Transverse relaxation time; FLAIR, Fluid-attenuated inversion recovery; DWI, Diffusion-weighted imaging; ADC, Apparent diffusion coefficient; rCBV, Relative cerebral blood volume; SWI, Susceptibility-weighted imaging; MRS, Magnetic resonance spectroscopy; Cho, Choline; NAA: N-acetylaspartate; IDH, Isocitrate dehydrogenase; CNS, Central nervous system.

hypercellularity, while perfusion imaging, especially DSC-derived rCBV, identifies zones of neovascular proliferation.⁶⁹ These signal properties are rooted in the physics of water motion, susceptibility effects, and contrast kinetics. Used together, they provide a biological map that complements structural imaging, guiding both extent-of-resection decisions and biopsy targeting in heterogeneous tumors.

When tumors encroach upon eloquent cortex, fMRI and DTI provide indispensable tools. fMRI relies on the BOLD signal, which measures changes in local blood oxygenation as an indirect marker of neuronal activity. While valuable, fMRI is limited by both spatial resolution (the smallest brain area it can localize) and temporal resolution (its ability to capture rapid changes in brain activity).⁸⁰ The BOLD response reflects a delayed hemodynamic change occurring several seconds after neuronal firing, making it too slow to capture fast neural events in real time.^{90,91} DTI-based tractography, on the other hand, maps the trajectories of critical white matter pathways,⁹² complementing fMRI in preoperative planning. Together, these modalities enable more informed selection of surgical corridors and help determine when intraoperative mapping or patient-specific adjustments are warranted.

Understanding the physical basis of these methods helps neurosurgeons better anticipate limitations, such as neurovascular uncoupling, edema-related signal degradation, and spatial distortions.

Geometric distortion is a critical artifact in echo planar imaging sequences such as DWI and fMRI, with direct implications for neurosurgical planning. These sequences are highly sensitive to magnetic field inhomogeneities, especially near air–tissue interfaces like the skull base and paranasal sinuses, leading to spatial warping of the image.^{93,94} In practice, this means that structures may appear displaced by several millimeters from their true anatomical location, which has direct consequences for surgical navigation.^{95–97} For neurosurgeons, this distortion is particularly relevant when planning resections near eloquent cortex or deep white matter tracts, as even small misregistrations can increase the risk of functional injury. Correction techniques, including field mapping and distortion correction algorithms, can mitigate these effects and improve spatial fidelity, enhancing the reliability of neuronavigation.⁹⁸

Intraoperative MRI addresses the fundamental problem of brain shift, which renders preoperative navigation less reliable as surgery progresses.⁹⁸ Real-

time imaging updates allow revisionalization of tumor boundaries, but even intraoperative MRI is susceptible to limitations rooted in image acquisition physics, such as gradient nonlinearity, field inhomogeneities, and distortions near resection cavities. Recognizing these constraints enables a more realistic interpretation of intraoperative images.

Finally, interpretive pitfalls remind us that MRI is not infallible. Susceptibility artifacts on SWI can mimic or obscure findings near blood, calcification, or hardware. DWI interpretation is complicated by T2 shine-through, and perfusion metrics may be confounded by motion artifacts or miscalculated input functions (Table 3). Moreover, differentiating true tumor progression from pseudoprogression remains a critical imaging challenge with direct surgical implications. Pseudoprogression, the transient radiographic changes that mimic recurrence, particularly following chemoradiotherapy, is most common in MGMT-methylated GBM and typically manifests within 12 weeks of treatment.⁹⁹ On conventional postcontrast T1 imaging, pseudoprogression can appear indistinguishable from true tumor growth. Advanced imaging techniques such as perfusion MRI (e.g. rCBV analysis), diffusion imaging, and MRS may help differentiate these entities,^{100–102} but no single modality offers definitive diagnostic

Table 3. Pitfalls and Limitations Across MRI Modalities

Modality	Common Pitfalls	Example Clinical Impact	How to Mitigate
DWI	T2 shine-through	False interpretation of necrosis as solid tumor	Correlate with ADC map and T2-weighted imaging
fMRI	Neurovascular uncoupling	Underestimation of functional cortex near tumors	Supplement with intraoperative mapping or MEG
DWI/fMRI	Geometric distortion (EPI-related susceptibility artifact)	Misregistration with structural MRI, inaccurate neuronavigation, or mislocalization of eloquent cortex	Apply distortion correction algorithms, coregister with high-resolution T1/T2 imaging, or validate with intraoperative mapping
SWI	Susceptibility artifact	Distorted signal near air–tissue or bone interface	Cross-reference with T2*/CT or avoid overreliance in those areas
Perfusion	Motion artifacts, inaccurate arterial input function	Misestimation of rCBV and perfusion metrics	Repeat acquisition or use standardized acquisition protocols
DTI	Edema/infiltration affects anisotropy	Loss of tract coherence, misplacement of eloquent pathways	Interpret in context of T2/FLAIR and structural anatomy

DWI, Diffusion-weighted imaging; T2, Transverse relaxation time; ADC, Apparent diffusion coefficient; fMRI, Functional magnetic resonance imaging; MEG, magnetoencephalography; EPI, Echo planar imaging; MRI, Magnetic resonance imaging; T1, Longitudinal relaxation time; SWI, Susceptibility-weighted imaging; T2*, Effective transverse relaxation; CT, Computed tomography; rCBV: Relative cerebral blood volume; DTI, Diffusion tensor imaging; FLAIR, Fluid-attenuated inversion recovery.

accuracy. Integration of imaging with clinical timing, symptom trajectory, and molecular features remains essential. For neurosurgeons, recognizing this limitation is crucial in avoiding premature or unnecessary reoperation and in guiding biopsy or re-resection decisions within the appropriate therapeutic window.¹⁰³

EMERGING DIRECTIONS

As MRI technology continues to evolve, several emerging trends are poised to reshape neuro-oncologic imaging and surgical planning. While not yet routine in daily neurosurgical practice, these innovations signal the next generation of tools neurosurgeons should be aware of.

AI is rapidly transforming image analysis. Deep learning models now enable automated tumor segmentation with accuracy approaching expert-level performance, offering faster and more consistent volumetric assessments.¹⁰⁴ AI-based algorithms can assist in tracking tumor progression, estimating the extent of resection, and even predicting survival based on imaging features. As these tools mature and integrate into clinical platforms, they may streamline preoperative planning and enhance intraoperative decision-making.

Recent advances have introduced diffusion-based generative models, which may surpass traditional deep learning approaches in segmentation accuracy, boundary delineation, and domain generalization.¹⁰⁵ These models

reconstruct medical images through iterative denoising steps, preserving subtle structural features crucial for glioma margin detection. Their robustness to scanner and protocol variability and improved performance with limited training data make them particularly suited for neuro-oncologic imaging. Additionally, transfer learning strategies, where pretrained networks are fine-tuned on brain tumor datasets, have shown promise in improving model performance and reducing the need for large annotated cohorts.¹⁰⁶

Ultra-high-field MRI (7 T and beyond) is expanding the boundaries of neuro-imaging resolution. These systems offer improved signal-to-noise ratios, enabling finer visualization of cortical microstructure, tumor margins, and small vasculature.¹⁰⁷ While currently limited to research settings or specialized centers, 7T MRI holds promise for better delineation of low-grade gliomas, tractography near eloquent areas, and early detection of subtle recurrence.

Quantitative MRI aims to move beyond subjective image interpretation by providing standardized, numerical maps of tissue properties, such as T₁, T₂, and diffusion metrics.¹⁰⁸ These values can improve reproducibility across centers and may serve as imaging biomarkers for treatment response or tumor biology. Unlike conventional sequences that rely on relative contrast, quantitative imaging offers objective, longitudinal assessment of disease.

Radiomics and MR fingerprinting represent novel paradigms in extracting high-dimensional data from MRI. Radiomics analyzes texture, shape, and intensity patterns that are imperceptible to the human eye, potentially linking them to histology or molecular markers. MR fingerprinting encodes multiple tissue properties simultaneously into a single scan, producing unique signal evolutions ("fingerprints") that can identify tissue types and pathologies with high specificity¹⁰⁹ (Table 4).

In contrast to prior reviews that have predominantly targeted radiologists or general oncologic audiences,^{3,4} this article was designed to translate MRI physics into the language of neurosurgery. By linking biophysical processes such as diffusion, perfusion, and relaxation to cellularity, vascularity, and tissue infiltration, we highlight how imaging insights can be operationalized in surgical planning and intraoperative decision-making. This deliberate focus on the neurosurgical perspective distinguishes our work from existing literature and positions it as a complementary resource for neurosurgeons and trainees seeking to integrate imaging physics into their clinical practice.

CONCLUSION

MRI is more than a diagnostic tool; it is an essential partner in every stage of neurosurgical care. From initial tumor characterization and operative planning to

Table 4. Emerging MRI Techniques and Clinical Applications

Technique	Principle	Clinical Promise	Current Limitation
AI-based segmentation	Deep learning models trained on labeled imaging data	Fast, consistent tumor delineation and progression monitoring	Requires clinical validation and regulatory approval
MR fingerprinting	Simultaneous acquisition of multiple tissue parameters with pattern matching	Quantitative tissue characterization in a single scan	Limited availability and implementation across centers
Ultra-high-field MRI (7T)	Higher magnetic field strength leads to improved SNR and spatial resolution	Finer tumor margin detection, microvascular imaging	Susceptibility artifacts, accessibility, SAR limits
Quantitative imaging	Numeric mapping of intrinsic tissue properties (T ₁ , T ₂ , etc.)	Objective, reproducible imaging biomarkers	Lack of standardization between scanners and protocols
Radiomics	High-dimensional extraction of image features for pattern analysis	Predictive modeling of tumor behavior and molecular subtype	Needs large annotated datasets and cross-validation

AI, Artificial intelligence; MR, Magnetic resonance; MRI, Magnetic resonance imaging; SNR, Signal-to-noise ratio; SAR, Specific absorption rate; T₁, Longitudinal relaxation time; T₂, Transverse relaxation time.

intraoperative navigation and post-operative evaluation, MRI provides not only anatomical detail but also critical insights into tumor physiology, metabolism, and microstructure. For neurosurgeons, a working knowledge of MRI physics is indispensable. Understanding how each sequence is generated, what it reflects, and where its limitations lie empowers sharper interpretation, more effective collaboration with radiologists, and safer, more personalized surgical strategies. As advanced techniques such as perfusion imaging, tractography, functional mapping, and quantitative imaging become more integrated into neurosurgical workflows, the neurosurgeon's role as an informed imaging interpreter grows increasingly important. Looking ahead, innovations in AI, ultra-high-field MRI, and radiomics are transforming imaging into a predictive, real-time decision support tool. Staying current with these advances will be key to translating imaging data into meaningful, patient-centered outcomes.

CRediT AUTHORSHIP CONTRIBUTION STATEMENT

Jawad Fares: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing — original draft, Writing — review & editing. **Yizhou Wan:** Investigation, Writing — review & editing. **Yonghao Li:** Investigation. **Tomasz Matys:** Investigation, Writing — review & editing. **T. Adrian Carpenter:** Investigation, Writing — review & editing. **Stephen J. Price:** Investigation, Supervision, Writing — review & editing.

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