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TITLE PAGE

REVIEW ARTICLE:

Title: Validation conditions for the use of diffusion tensor imaging for neuroprognosis: a clinical and technical review

Authors: Pierre Simeone, MD, PhD^{a,b,c*}; Vincent Perlberg, PhD^d; Guillaume Auzias, PhD^b; Sylvain Takerkart, PhD^b; Olivier Coulon, PhD^b; Vincent Doat-Sarfati, MD^e; Vincent Degos, MD, PhD^e; Valentine Battisti, PhD^e; Lionel Velly MD, PhD^{a,b}; Damien Galanaud, MD, PhD^{f,c}; Jean-François Hak, MD, PhD^g; Nadine Girard, MD, PhD^{b,g}; Mélanie Péligrini-Issac, PhD^c; Louis Puybasset, MD, PhD^{c,e}

^a Department of Anesthesiology and Critical Care Medicine, Aix Marseille University, AP-HM, University Hospital Timone, Marseille, France

^b Timone Neuroscience Institute, CNRS, Aix Marseille University, UMR7289, Marseille, France

^c Biomedical Imaging Laboratory, Sorbonne University, CNRS, INSERM, Paris, France

^d BrainTale SAS, Strasbourg, France

^e Department of Anesthesiology and Critical Care Medicine, Sorbonne University, AP-HP, Pitié-Salpêtrière Hospital, Paris, France

^f Department of Neuroradiology, Sorbonne University, AP-HP, Pitié-Salpêtrière Hospital, Paris, France

^g Neuroimaging Department, Aix Marseille University, AP-HM, University Hospital Timone, Marseille, France

Corresponding author: Pierre Simeone, Department of Anesthesiology and Critical Care Medicine, Aix Marseille University, AP-HM, University Hospital Timone, Marseille, France. Phone: 00 33 (0)6 59 24 03 13; fax 00 33 (0)4 91 38 58 50;
e-mail: pierre.simeone@ap-hm.fr

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ABSTRACT

In the growing field of neuroprognosis in intensive care patients, the use of diffusion tensor imaging (DTI) MRI to quantify white matter integrity is particularly promising, with solid demonstrations for predicting outcomes in cardiac arrest and severe traumatic brain injury. However, as with any new technique available to practitioners in routine practice, it is essential to provide clinicians and future users with the validity criteria they need to master for an appropriate interpretation of their results. This pragmatic review aims to provide clinicians and future users with a comprehensive understanding of the medical and technical conditions necessary for the accurate interpretation of DTI-derived metrics. While DTI offers significant potential, its reliability can be influenced by various factors, including previous neurological disorders, acquisition artifacts, and data preprocessing failures. This review highlights the importance of considering classical MRI contraindications and the impact of physiological conditions such as age and the timing of MRI scans. Additionally, it discusses the influence of pre-existing neurological disorders and associated lesions on DTI metrics, which can lead to classification biases and prognostication errors. Technical considerations, including the necessity for rigorous quality control and preprocessing steps, are also emphasized to ensure the precision and accuracy of DTI metrics. By addressing these factors, this review aims to enhance the interpretability and clinical utility of DTI in neuroprognostication, ultimately facilitating more informed decision-making for intensive care unit patients. Furthermore, ongoing research and data sharing are needed to refine DTI techniques and improve their predictive accuracy in various clinical contexts.

Keywords: diffusion tensor imaging; diffusion metrics; outcome; neuroprognosis; artifacts; intensive care.

Abbreviations: **AD** = axial diffusivity; **ADC** = apparent diffusion coefficient; **ALS** = amyotrophic lateral sclerosis; **AUC** = area under the curve; **BSD** = B-matrix spatial distribution; **CFES** = cerebral fat embolism syndrome; **CNS** = central nervous system; **CSF** = cerebrospinal fluid; **CSVD** = cerebral small vessel disease; **DTI** = diffusion tensor imaging; **DW** = diffusion-weighted; **ECMO** = extracorporeal membrane oxygenation; **FA** = fractional anisotropy; **FLAIR** = fluid-attenuated inversion recovery; **GOSE** = Glasgow outcome scale extended; **ICU** = intensive care unit; **JHU** = Johns Hopkins University; **MCI** = mild cognitive impairment; **MD** = mean diffusivity; **MNI** = Montreal Neurological Institute; **MRI** = magnetic resonance imaging; **MS** = multiple sclerosis; **RD** = radial diffusivity;; **SCD** = subjective cognitive decline; **SVM** = support vector machine; **SW** = susceptibility-weighted;

TEXT

Background

The prognosis of patients resuscitated from acute neurological causes is a major concern, particularly during the first few weeks while patients are still in the intensive care unit (ICU). Conducting a comprehensive neurological examination can be particularly challenging due to the lingering effects of sedatives, neuromuscular blocking agents, hypothermia, and an unstable physiological condition [1]. During the initial phase, patients require a high level of care, while their neurological prognosis remains uncertain [2]. There exists a significant risk of prematurely discontinuing care, since there might be a potential for recovery. In the second phase, while the confidence in the patient's prognosis increases, the need for care is significantly reduced. Indecision regarding outcome may result in patients remaining in a vegetative or minimally conscious state, which may go against the patient and family's wishes or patient's advance directives. The ability to predict a patient's outcome while they are still in the ICU could facilitate a more thoughtful approach to withdrawing life-sustaining support. Regarding coma prognostication, a recent literature review of the biological, electrophysiological, and imaging tools available to clinicians, as well as their level of evidence for the most common etiologies has been published underlying brain injury [3]. This review highlights magnetic resonance diffusion tensor imaging (DTI) as an emerging structural marker for outcome prediction. DTI appears promising for etiologies such as cardiac arrest or severe traumatic brain injury, but requires further investigation for other causes, including toxic, metabolic, or intracerebral hematoma, stroke, as well as subarachnoid hemorrhage. To promote the use of this technique in real-life conditions, it is essential to provide clinicians

and future users with its validity conditions and limitations. This pragmatic review aims to take a comprehensive look at the medical and technical framework required to use DTI metrics for neuroprognostication in intensive care patients.

Theoretical ground: DTI and conventional metrics

Magnetic resonance imaging (MRI) is one of the most flexible imaging tools to explore the central nervous system (CNS). Among the MRI modalities available, diffusion-weighted MRI (DW-MRI) has emerged as a powerful technique to measure signal attenuation resulting from water diffusion [4,5]. Water diffusion is assumed to be anisotropic in white matter regions (i.e., water diffusion is highly restricted and hindered in directions perpendicular to the axons) and isotropic in both gray matter and cerebrospinal fluid (CSF). More generally, water diffusion properties are related to the local microstructure of tissues.

DW-MRI consists in adding diffusion sensitization gradients in multiple directions (minimum 6, but typically 12 or more) to conventional MR sequences (e.g., a spin-echo sequence) to enhance anisotropic diffusion effects [6], with the movement of water molecules in tissues resulting in signal loss. These gradients are tuned using a diffusion-weighting factor b (typically $b = 1,000 \text{ s/mm}^2$).

DTI is a method to model water diffusion from DW-MRI data as an ellipsoid, with three principal axes (or directions, e_1, e_2, e_3) associated with three ellipsoidal radii ($\lambda_1, \lambda_2, \lambda_3$) [5,7]. The major direction e_1 of the ellipsoid, associated with λ_1 , is generally assumed to be parallel to the main direction of white matter tracts. This information is further simplified as scalar maps, the most common metrics computed being:

- mean diffusivity (MD) (also called the apparent diffusion coefficient, or ADC), which is the average of the 3 radii and considered as a global measure of the magnitude of diffusion;
- fractional anisotropy (FA)[8], computed as follows:

$$FA = \sqrt{\frac{(\lambda_1 - MD)^2 + (\lambda_2 - MD)^2 + (\lambda_3 - MD)^2}{2(\lambda_1^2 + \lambda_2^2 + \lambda_3^2)}},$$

which reflects water diffusion anisotropy;

- radial diffusivity (RD= $(\lambda_2 + \lambda_3)/2$), which appears to be modulated by myelin in white matter [9];
- axial diffusivity (AD= λ_1), more specific to axonal degeneration [10];

As water diffusion in tissues is highly sensitive to differences in the microstructural architecture of cellular membranes, DTI was primarily developed to assess the structural integrity of large axonal bundles in the brain and, more recently, in the cervical spinal cord, nerves, and peripheral nerves.

State of the art of the use of DTI for outcome prognostication

To better circumscribe the context in which DTI can be used for ICU patients, it is important to remind practitioners of the state of the art and current applications of this tool. As noted in Fischer and Edlow's recent literature review [3], applications of DTI metrics for outcome prognostication in patient cohorts of more than 150 individuals have related to two etiologies only: cardiac arrest and severe traumatic brain injury [11,12]. **In these two examples, values of DTI metrics that fell outside the range of ± 2 standard deviations (SD) from the mean measured in age-matched healthy controls were considered abnormal, indicating the**

presence of white matter lesions. The first study focused on survivors after a cardiac arrest who were still unresponsive to simple orders after 5 days [11]. FA was extracted in the whole white matter. A support vector machine (SVM) classifier predicted a poor outcome based on the Cerebral Performance Categories Scale scale, grades 1 and 2, with a high specificity of 100% and a sensitivity of 89%. A similar methodology was endorsed for traumatic brain injury patients, except that DTI metrics were computed in the John Hopkins University (JHU) atlas of deep white matter tracts [13] and outcome definition relied on GOSE scale. DTI showed once again good prognostic performance in predicting unfavorable outcome (GOSE 1-3) with 96.8% specificity and 49.0% sensitivity, as well as 97.1% specificity and 58.5% sensitivity in predicting favorable outcome (GOSE 4-8) [12].

Notwithstanding the promising performance of these two studies, it is important to identify situations in which erroneous DTI measures could lead to prognostication failure with serious clinical consequences.

Classical MRI contraindications

The same contraindications as to conventional MRI apply to DTI. It is necessary to verify MRI compatibility of pacemakers [14] and other medical implants. In case of head injury of ballistic origin, the high risk of movement of residual ferromagnetic shrapnel in the scanner magnetic field poses a significant safety problem [15,16]. Due to the risks associated with transportation to the MRI settings and the need for patients to remain motionless during lengthy acquisitions, it is crucial to remain vigilant in the event of respiratory or hemodynamic failure. Access to the patient for medical intervention and monitoring conditions can be often compromised during MR imaging, potentially jeopardizing patient stability: most secondary aggressions observed in neuro-injured patients in intensive care are caused by episodes of deep or prolonged

hypoxemia or hypotension occurring during the imaging session, which may not be managed appropriately in the MR room and subsequently damage the brain in a second phase after the initial injury [17–19]. Similarly, MRI scan should not be considered for patients with life-threatening extra-cerebral lesions [20]. Besides, in patients with persistent or at risk of intracranial hypertension, prolonged supine position is not compatible with scanning in optimal conditions since it may cause major decompensation and require emergency repositioning. Additionally, severe decompensation may require medical therapies that are not accessible during the scan.

Physiological condition: influence of age

Large data sharing initiatives such as ENIGMA [21] and open datasets such as Lifespan HCP [22] have enabled the characterization of the spatiotemporal dynamics of DTI measures in normal subjects across the entire lifespan. Although a comprehensive study aggregating as much data as possible, such as Brainchart [23] for morphometry measures, is not available to date for DTI features, the major trends of the evolution of DTI measures with age in healthy controls can be recapitulated from the literature.

A series of works on childhood and adolescence [24–26] has reported highly dynamic nonlinear changes in DTI measures with a sharp increase in FA with age associated with a decrease in MD. Finer-grained analyses have revealed a complex regional pattern, with the primary sensory regions maturing first, followed by the fiber tracts connecting the association cortices. Later in life, studies covering 20 to 85 years of age with longitudinal MRI measurements have reported a nonlinear accelerated increase in MD, RD, and AD with age, and an accelerated FA decreasing trend after 50 years (e.g., [26]).

It is thus critical to take the potential influence of age on DTI measures into account in the analyses and interpretations. Note that in the context of clinical evaluation, possible interactions between the age of the patient and biophysical mechanisms acting at the cellular level in pathological brain regions can also affect DTI measures. As these complex interactions remain to be characterized quantitatively, interpretations of DTI measures in abnormal tissue remain mostly speculative.

Pathological condition: influence of MRI timing

One of the main factors influencing DTI results is the timing of the scanning session with respect to the detrimental event. Indeed, if the objective is neuroprognostication, performing an examination too early could imply not being able to detect belated lesions leading to white matter degradation. As a result, early DTI measures may not reflect the final state of white matter integrity. Consequently, a minimum delay of one to two weeks between the event and the MRI session has been recommended to increase the sensitivity of the technique [3,11,12]. Additionally, if the initial scan is inconclusive, it is advisable to perform a follow-up scan at a later time and benefit from these longitudinal measures to better evaluate the evolution of the underlying pathophysiological mechanisms. (Figure 1)

Impact of known previous neurological disorders on neuroprognostication

Existing neurological pathologies can impact DTI metrics and lead to classification biases. Here is a list of common pathologies and their effect on DTI metrics, listed in descending order of incidence (Table 1):

- Leukoaraiosis: leukoaraiosis is associated with the effects of cardiovascular risk factors on white matter. Leukoaraiosis may be asymptomatic but is correlated with stroke occurrence

[27] Several studies have reported a decrease in FA and an increase in MD correlated to the severity of leukoaraiosis.[28] Therefore, in neuroprognostication studies, patients with a Fazekas score greater than 4 were systematically excluded to avoid this confounding factor. [3,11,12]

- Epilepsy: Primary epilepsy per se is associated with disturbances in DTI metrics. A recent meta-analysis of temporal epilepsies has reported a decrease in FA and an increase in MD in both temporal lobes, as well as in the white matter of all brain areas preferentially connected to the temporal lobes.[29]

- Parkinsonian Syndrome: Cochrane and Ebmeier [30] found a notable effect size for reduced FA in the substantia nigra and basal ganglia in patients compared to controls. Parkinsonian syndromes can also be distinguished by the unique anatomical distributions of their microstructural abnormalities detected by reduced FA and increased MD.[31] For example, abnormal values of DTI metrics in the cerebellum and middle cerebellar peduncle may be primarily involved in multiple system atrophy, the superior cerebellar peduncle is primarily vulnerable in progressive supranuclear palsy and supratentorial white matter regions are primarily involved in corticobasal syndrome. DTI can correlate with severity and motor dysfunction, assessment of disease progression and treatment response.[31]

- Alzheimer's disease, mild cognitive impairment (MCI), subjective cognitive decline (SCD): patients with MCI or Alzheimer's disease show extensive FA decrease and MD increase in the brain compared with the normal cognitive ageing population, and this situation worsens with disease progression. [31] Lo Buono et al. [32] reported a decrease in FA in MCI patients in most studies, indicating the association between white matter diffusivity changes and Alzheimer's disease progression. Sexton et al. [34] reported a decrease in FA and an increase

in MD in patients with Alzheimer's disease in all brain regions except in the parietal white matter and the internal capsule. Similar structural abnormalities can be detected in the corpus callosum, hippocampus, cingulum and other medial temporal lobe regions in patients presenting with an SCD.

- Severe psychiatric condition: Severe psychiatric disorders such as schizophrenia, anxiety and mood disorders, personality disorders, developmental disorders in childhood, addiction problems, can have a significant impact on DTI metrics.[35] Damage to the brain's large associative bundles and areas dedicated to emotional control is often the cause of the symptomatology and typically include a decrease in FA and an increase in MD.[36] Besides, it has been shown that FA can return to normal levels in depressed patients responding to electroconvulsive therapy.[37] However, it is important to note that the literature reveals a considerable heterogeneity of methods, making it difficult to draw definitive conclusions.[38] Therefore, in most neuroprognostication studies, the criterion of severity of psychiatric illness that led to exclusion was guardianship.[3,11,12]

- Cerebral small vessel disease (CSVD): CSVD is a common age-related condition that affects the small blood vessels in the brain with two primary types: arteriolosclerosis and cerebral amyloid angiopathy characterized by the accumulation of amyloid-beta protein in the walls of small blood vessels in the brain. A review by Safri et al. [39] has summarized the recent advances and prospects of DTI measures for the clinical detection and assessment of cerebral white matter integrity. However, no definitive conclusions regarding the impact of this disease on DTI metrics have yet been drawn. It therefore seems reasonable to exclude patients with proven CSVD.

- Multiple sclerosis (MS): Increased MD and decreased FA have been reported in patients with MS due to microstructural changes such as axonal loss, myelin damage or edema.[40] However, most studies have reported similar changes in RD and AD, suggesting an overall loss of myelin integrity.[40] Gupta et al. [41] found that DTI-based FA values were significantly lower in contrast-enhancing multiple sclerosis lesions compared to non-enhancing lesions, while other MRI biomarkers such as MD were not significantly modified.

- Brain tumors: Many patterns may exist depending on the type of tumor, its aggressiveness, and the status of the peripheral parenchyma. [42] Overall, FA is high due to the high fiber density, unless there exist areas of necrosis or a fluid component leading to a decrease in FA. MD is also increased to varying degrees depending on the type of tumor. In general, edema in the periphery of the lesion leads to a decrease in FA and an increase in MD in patients compared to controls.[43]

- Encephalitis: DTI results can vary greatly depending on the causative agent, with changes in topography affecting either gray or white matter in different ways. It is important to note that FA and MD may vary depending on the severity of the pathology and the timing of the scan.[44,45] The following examples illustrate the complex nature of these phenomena, which are still poorly understood: In the subacute phase of herpes encephalitis, an early scan revealed a decrease in MD and an increase in FA. Conversely, at a distance, the results showed an increase in MD and a decrease in FA, which the authors attributed to vasogenic edema.[44] High MD values associated with vasogenic edema together with low FA values have been shown to be predictive of a poor outcome. However, low MD may also lead to poor prognosis in some cases.[46]

- Neurolupus: Despite a lack of data, it is generally agreed upon that neurolupus results in an increase in MD and a decrease in FA. The most topographically accurate studies have shown an overall increase in MD, which suggests a loss of white matter integrity due to inflammation.[47] Another study has reported a global reduction in FA, including areas such as the right superior corona radiata, the right superior longitudinal fasciculus, the body of the corpus callosum, the right inferior fronto-occipital fasciculus, the right thalamic radiation, and the right uncinate fasciculus. [48] FA and MD variations may be attributed to the timing of inflammatory outbreaks, as well as the possible presence of neuropsychological sequelae.[49]

- Tuberos sclerosis complex: The tubercles that cause the disease also have a neurological tropism. They appear in DTI images as structures with decreased FA and increased MD, compared by some authors to tissues from poorly organized collections of large, dysplastic cells. [50]

- Amyotrophic lateral sclerosis (ALS): A decrease in FA and an increase in MD, RD, and AD along the entire corticospinal tract have been reported in ALS patients.[51]. Another meta-analysis showed symmetrical regions of FA reduction in the bilateral frontal white matter/cingulate gyrus and the posterior limb of the bilateral internal capsule.[52] Zhang et al. [53] published a meta-analysis showing a significant reduction in FA in the whole white matter in ALS patients compared to healthy controls, supporting the view that ALS is a multisystem degenerative disease. These findings on DTI indicate that results from this technique should be interpreted with caution in ALS patients, especially given the broader implications of the disease, which often raise chronic ethical questions related to end-of-life management. Additionally, the concurrent presence of various biological and immunological disorders can further complicate the interpretation of DTI results. [54]

- Huntington's disease: A systematic review has reported an increase in FA in both pre-symptomatic and symptomatic patients, whereas an increase in MD was only observed in the symptomatic group. [55] In both groups, FA was significantly increased in the putamen and globus pallidus and decreased in the corpus callosum. MD was also significantly increased in the putamen and thalamus in both groups.

In this overview, we have demonstrated that pre-existing diseases exert a significant influence on DTI metrics. Ideally, it would be prudent to exclude patients with these conditions from analyses regarding coma outcome prognostication. We acknowledge that many ICU patients present with comorbidities listed above, which could theoretically limit the applicability of DTI-based neuroprognostication. However, in clinical practice, the most challenging prognostic decisions arise for patients in the "gray zone"—those who neither die early nor recover rapidly after sedation withdrawal, and whose pre-existing conditions are not severe enough to preclude meaningful interpretation of DTI metrics. In the two studies we reviewed, only 3% of patients were deemed ineligible due to medical histories that would have confounded DTI interpretation. [11,12] When uncertainty persists, we strongly advocate for a multimodal approach, integrating clinical, electrophysiological, and other imaging data rather than relying solely on diffusion tensor metrics. This strategy aligns with current ethical and clinical guidelines for neuroprognostication in the ICU. [3,17]

Associated lesions modifying DTI metrics in comatose patients

Stroke or patient with secondary massive ischemic lesion

It is widely recognized that a variety of neurological injuries can be linked to ischemic lesions. In fact, patients who present with traumatic brain injury, infectious or immune etiologies, thromboembolic events, subarachnoid or intracerebral hemorrhage, brain tumors, seizures, or vascular issues beyond stroke, may develop secondary ischemic areas as a complication.[56] In the acute phase of cerebral ischemia, MD experiences a significant decrease within the lesion, typically returning to normal levels within 5 to 7 days. Conversely, chronic ischemic lesions, i.e. those occurring after a two-week period, generally exhibit a notable increase in MD. Likewise, recent research indicates that FA tends to rise in acute lesions, followed by a decrease below normal levels in the chronic phase.[4] (Figure 2)

Cerebral fat embolism syndrome

Another injury associated with trauma patients is the cerebral fat embolism syndrome (CFES), which occurs in the context of major trauma, particularly in the case of a contemporary diaphyseal fracture of a long bone.[57] Despite the low incidence of approximately 2%, diagnosis of CFES in these comatose patients can be challenging since they may present with specific MRI findings that need to be considered.[58] Lesions may appear in specific areas in conventional MR images, such as the basal ganglia, corpus callosum, cerebellar hemispheres, and deep white matter [11]. These lesions may be caused by the breakdown of the blood-brain barrier, which can be induced by free fatty acids. Typically, these lesions involve areas of vasogenic edema on T₂-weighted images and/or cytotoxic edema on diffusion-weighted images, consistent with microembolic infarcts. Multiple petechial hemorrhages can be seen

on susceptibility-weighted (SW) images. These lesions also impact DTI metrics. Early-onset myelin damage may increase RD and decrease FA while being transient. The absence of a significant decrease in MD and AD could be due to the presence of both parenchymal vasogenic edema and cytotoxic edema, [58] and lead to the incorrect conclusion that this type of patient could have a poor outcome (Figure 3).

Active hydrocephalus

Patients with neurological damage, especially those with severe traumatic brain injury, intraparenchymal hematoma, or subarachnoid hemorrhage, are at a higher risk of developing hydrocephalus.[59] This is because there is a high risk of intraventricular hemorrhage, which can cause CSF resorption and hydrocephalus, resulting in intracranial hypertension.[60] Hydrocephalus can be diagnosed based on clinical criteria such as Glasgow score deterioration, impaired awareness, headache, nausea, or vomiting. Imaging criteria can also be used when there is dilatation of the ventricular system caused by impaired cerebrospinal fluid circulation or clearance. Another diagnostic option is to perform a therapeutic test with CSF depletion or osmotherapy.[59,60] According to a recent meta-analysis, there may be significant changes in DTI metrics due to the mechanical modification of brain structures during CSF accumulation and the risk of periventricular CSF resorption. However, the results are not consistent across studies. Some authors report increased FA in certain areas, such as the caudate nucleus [61,62] or the corona radiata,[63] due to greater fiber packing caused by mechanical pressure. Meanwhile, others show a decrease in FA in certain periventricular areas due to edema accumulation in the context of ventricular resorption. Changes in FA may also occur after drainage.[64]

Lesions consecutive to extracorporeal membrane oxygenation

Extracorporeal membrane oxygenation (ECMO) provides cardiac and/or pulmonary support to patients who are refractory to conventional therapies.[65,66] This technique may also be used in cases of neurological causes where there is associated cardiac or pulmonary damage, or traumatic causes, particularly complex thoracic trauma.[67] However, the use of extracorporeal circuits carries a high risk of thrombo-embolic events, fibrinocruoric or gaseous embolisms.[68] Several studies suggest that patients equipped with ECMO show substantial modification on their MR images inducing significant variability in neurological outcomes. The observed lesions may take several forms, including hematomas and widespread microbleeds in the subcortical and deep white matter on T_2^* -weighted or SW images, while other studies also report punctuate ischemic infarct. [69] Although there is limited evidence regarding changes in DTI metrics in patients with ECMO, some studies have revealed a decrease in MD in some of these lesions.[70] Preliminary data in children also suggest a decrease in FA and an increase in RD in patients who have undergone ECMO.[71] (Figure 4)

Devices known to artifact DTI metrics

As previously stated, patients admitted to the ICU for neurological causes are at a higher risk of developing hydrocephalus, which may be associated with intraventricular hemorrhage. In some cases, external ventricular drainage may be necessary. When it is difficult to wean off external ventricular drainage, an internal ventriculoperitoneal or ventriculoatrial shunt may be needed. However, the use of shunt valves can also result in significant imaging artifacts and involuntary settings change when exposed to heterogeneous magnetic fields.[72] In addition,

artifacts can vary drastically and are sometimes unpredictable depending on the manufacturer and the type of sequence.[73] Some authors report the use of valves that are more compatible with MRI, or a particular surgical technique that avoids fixing the material close to the skull, but to date no work has validated these techniques with DW sequence.[74] Similarly, intracranial hypertension may require decompressive craniectomy.[75–77] In this situation, some teams use titanium bone flap replacement to prevent the patient from remaining without protection at the skull level after surgery.[78] However, this type of material can not only cause significant artifacts, but also distort all subsequent DTI measurements. (Figure 5) These two examples highlight the need for vigilance when performing DW imaging in such patients, since this could lead to false conclusions and outcome misclassification due to lack of validation of DTI metrics.

Technical exclusion criteria after MRI completion

To effectively use DTI metrics in real-life clinical settings for evaluating individual patients, it is essential to first acquire MRI scans with at least 12 gradient directions on 1.5T scanners and 29 directions on 3T scanners. Additionally, the variability introduced by MRI protocols, scanner models, and scanner environments must be addressed, as it can be equivalent to intersubject variability [79]. Establishing reliable reference control values is crucial for accurately detecting abnormalities. Harmonization methods have been proposed to reduce intercenter variability in retrospective group analysis [80]. But as of today, the best analysis method to provide individual prospective DTI values while controlling for intercenter variability is based on the acquisition of reference data in healthy controls (at least 5 subjects) used to calibrate scanner- and MRI protocol specific values [81]. Detection of individual abnormalities can then be achieved by comparison with the distribution (mean and standard deviations) of DTI values measured in a cohort of healthy controls as a reference [82]. Finally, the practical implementation of DTI metrics in clinical routine must be accompanied by rigorous preprocessing steps and sanity/quality check procedures [83]. Sanity check consists in verifying that the MRI protocol perfectly corresponds to that used for control acquisitions conducted for calibration purpose as the sequence parameters strongly influence the DTI metrics results. The main preprocessing steps of DW data include the correction of motion and eddy-current effects and nonlinear coregistration procedure to align standardized white matter regions of interest onto the subject brain's space [84]. Quality checks thereby include:

- Brain coverage acquisition above 90%,
- Number of signal dropouts (intensity artifact) in less than 5% of the diffusion-weighted volumes,
- No vibration artifacts detected [85],

- Motion estimation with allowed maximum displacement below 5 mm between two successive directions of the diffusion gradient,
- No failure in the coregistration procedure between the subject's space and the 3D white matter atlas (Dice score above 60%).

All the steps of the sanity and quality check are summarized in Figure 6.

Technical pitfalls to consider for external validity

To further enhance patient databases and neuroprognostic assessments, it is crucial to consider external validity, particularly the technical specifics of DTI. It is essential to account for the influence of sequence parameters on DTI results and strive to standardize practices as much as possible. Moreover, there are sometimes difficulties in reporting essential parameters such as diffusion time, pulse duration or b-value to replicate studies. These parameters significantly affect the diffusion regime of specific tissue volumes and can lead to variations in DTI metrics, thereby impacting the interpretation of results. The lack of robust quality control procedures for DWI/DTI measurements — which are more prone to artefacts than conventional T1 or T2 images due to the use of larger gradients — highlights the importance of researchers' raising awareness of and disclosing these limitations. Establishing standardized procedures, along with explicit reporting of all relevant parameters, is essential for improving the reliability and comparability of DTI studies – see sanity and quality check procedures (Figure 6). [83]

Sources of Systematics Errors

Beyond the intrinsic difficulties associated with the clinical application of DTI in clinical settings, several critical concerns warrant further examination to ensure the accuracy and reliability of DTI findings. These errors are primarily caused by various imperfections inherent in the imaging process, including: [86,87]

- Magnetic field inhomogeneities: variations in the static magnetic field (B_0) can cause geometric distortions and signal dropouts, especially in areas such as the frontal lobes and the brainstem. These distortions are exacerbated at higher field strengths (e.g., 3T vs. 1.5T) and

can systematically bias DTI metrics such as fractional anisotropy (FA) and mean diffusivity (MD),

- Eddy currents, induced by time-varying magnetic fields within the MRI scanner's conductive components, generate additional magnetic fields that distort imaging gradients,
- Gradient nonlinearities: spatial distortions due to gradient nonlinearities are particularly problematic for tractography, where even small errors in fiber orientation can propagate and lead to incorrect reconstruction of white matter pathways [88],
- Chemical shift artifacts, arising from differences in resonance frequencies between fat and water protons in a magnetic field, cause image misregistration,
- Partial volume effects, occurring when a voxel contains multiple tissue types with varying diffusion properties, lead to systematic errors in estimating diffusion parameters,
- Thermal noise and instrumentation errors, which introduce inaccuracies, affect the signal-to-noise ratio and lead to diffusion measurement mistakes.

These systematic errors can substantially alter statistically significant differences and are characterized by large effect sizes, necessitating their inclusion in analyses or at least an assessment of their impact on study results. Addressing these systematic errors is essential for enhancing the accuracy and reliability of DTI metrics. **The magnitude of the impact of these systematic errors on DTI metrics is illustrated in Table 2.**

Mitigation and Corrections of Systematics Errors

Moreover, systematic errors could persist and present an ongoing obstacle, especially in tractography or DTI where local effects are crucial. In larger regions of interest, these systematic errors might be averaged out across the volume, potentially obscuring genuine

biological effects. Recent studies have begun to address these challenges using methods like phantom-based diffusion tensor patterns, the BSD-DTI (B-matrix spatial distribution in DTI) method, and field camera techniques to effectively map and rectify field inhomogeneities [89–93]. In clinical practice, in addition to using tools to partially manage these issues, the creation of control groups by center and the normalization of DTI metrics greatly help to control these limitations [3,11,12]. All these steps are summarized in a practical approach, as shown in **Table 3**. This approach is particularly useful for comparing patients and healthy controls within the same protocol.

In recent studies, harmonization of diffusion MRI protocols is increasingly recognized as essential for reliable multi-site neuroimaging studies. Some algorithms have demonstrated efficacy in reducing scanner-specific variability in diffusion metrics preserving biological signal integrity while mitigating technical confounds [94]. Automated pipelines integrating harmonization with advanced preprocessing further enhance data comparability, addressing challenges like gradient nonlinearities and field inhomogeneities [95]. As well, some authors developed approaches to improve DTI accuracy using deep learning to correct B-matrices with the reference method BSD-DTI for both phantom and in vivo data [96]. These advances collectively improve the robustness of DTI biomarkers, facilitating more accurate clinical and research applications. Harmonization thus emerges as a critical step for large-scale, reproducible neuroimaging studies.

CONCLUSION

In the growing field of neuroprognosis, use of DTI metrics from DW MR imaging seems particularly promising, with validated work in cardiac arrest and severe head trauma. However, when using such a sensitive technique, it is important to remember the validation conditions to perform this examination under the best conditions of interpretability for the patient. It is therefore essential to keep in mind the classical MRI contraindications and factors influencing DTI results, such as known previous neurological disorders, associated lesions, factors known to artifact DTI metrics. Similarly, there are technical reasons with exclusion criteria that need to be known after the MRI is completed requiring the realization of a strong quality control after acquisition. All these conditions are summarized in [Table 4](#). While this technique holds significant promise for neuroprognostication, its clinical utility depends critically on addressing systematic errors and standardizing acquisition and analysis protocols. Recent literature has highlighted the need for rigorous quality control, including field mapping, gradient nonlinearity corrections, and phantom-based calibration. By adopting these advances, clinicians can improve the accuracy and reliability of DTI metrics, ultimately enhancing their value in guiding patient management in the ICU. Future work should focus on integrating these technical refinements into routine clinical workflows and expanding multi-center collaborations to validate DTI prognostic performance across diverse patient populations. In practice, the presence of an underlying disease must be considered when weighing the benefits and risks of using the technique. It is essential to highlight that, similar to any diagnostic or prognostic tool, the accuracy and performance of this technique will enhance as more data is acquired. This includes data from healthy controls, new patients with various pathological conditions but also the need to establish standards for the MRI protocols

used for these patients and to develop clear guidelines to enhance and share safety and quality check procedures. By expanding and sharing datasets, future applications could better account for variability and nuances in patient outcomes, potentially leading to more robust and reliable predictions. Furthermore, continuous refinement and validation of these techniques are essential as medicine practices are constantly evolving. Nevertheless, the use of DTI for neuroprognostication is fully in line with current medical and ethical issues, with the possibility of using personalized decision-making in the ICU for cardiac arrest and traumatic brain injury patients

DECLARATIONS

Standard protocol approvals, registrations, and patients consents

Not applicable.

Consent for publication

Not applicable.

Availability of data and materials

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

Competing interests

VP is the President and Chief Scientific Officer of BrainTale SAS. DG, LV, and LP are co-founders and members of the advisory board of Braintale SAS in terms approved by Sorbonne Université (Paris, France) in accordance with its policy on objectivity in research. PS received two academic grants ("SFAR Mobility Grant - 2024" and "Phoceo Mobility Grant - 2024"). The other authors declare that they have no competing interests.

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Running Head: DTI metrics validity for neuroprognosis.

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