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Interactions Between Anesthesia Regimens and Neurological Disorders in Brain-Computer Interface Procedures: Insights from Literature and A Simulation Study

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This manuscript reviews the differential effects of anesthesia on brain networks in neurological disorders and their implications for brain-computer interface (BCI) procedures. Drawing on empirical literature, it highlights disorder-specific anesthetic considerations for conditions such as Parkinson's disease, epilepsy, and amyotrophic lateral sclerosis (ALS). A simulated logistic regression model, based on synthetic data approximating real-world clinical scenarios (n=1,000), examines factors influencing BCI implantation success (defined as no major complications and signal quality $\geq 85\%$). Key findings include significant main effects of age, vascular proximity, electrode density, and disorder severity on success probability, with non-significant effects for anesthesia type. Implications for tailored anesthetic strategies are discussed, alongside limitations of the simulation approach. Future research should validate these insights with real clinical data.

Keywords: Brain-Computer Interfaces; Neurodegenerative Diseases; Deep Brain Stimulation; Logistic Models**Introduction**

Anesthesia disrupts brain networks in ways that vary according to underlying neuropathology. For instance, propofol reduces connectivity in networks associated with consciousness, with potential exacerbation in neurodegenerative conditions.¹ Electroencephalography (EEG)-based brain-computer interfaces (BCIs) in medical applications underscore the importance of disorder-specific anesthetic paradigms to mitigate artifacts induced by anesthesia.

In Parkinson's disease and related movement disorders, dexmedetomidine is often preferred for procedures like deep brain stimulation (DBS) or BCI implantation, as it facilitates intraoperative microelectrode recordings (MER) without suppressing critical thalamic signals, unlike volatile anesthetics.^{2,3} Patients with dyskinesia may require sedation to reduce movement artifacts, though this must be balanced against autonomic instabilities common in these disorders.

For epilepsy, propofol-based anesthesia is commonly used during electrode placement, although intraoperative seizures can still occur under general anesthesia.⁴ Low-dose propofol can activate cortical electroencephalograms (ECoGs), potentially aiding in the accurate identification of epileptic foci during glioma-related epilepsy surgery.⁵

In amyotrophic lateral sclerosis (ALS) or tetraplegia, endovascular BCIs (e.g., Synchron) have demonstrated feasibility under sedation.⁶ However, severe bulbar involvement heightens aspiration risks, particularly with volatile anesthetics.⁷ Total intravenous anesthesia (TIVA) may minimize these risks while preserving cortical signals suitable for communication decoding.⁸ Dexmedetomidine has also been explored as a primary induction agent, with evidence suggesting neuroprotective and anti-neuroinflammatory properties.^{9,10}

The interaction between anesthesia regimen (e.g., TIVA with propofol/remifentanyl, dexmedetomidine, or volatile agents) and neurological disorder severity (measured on scales such as the ALS Functional Rating Scale-Revised [ALSFRRS-R; 0-48] for ALS, or analogous metrics like the Unified Parkinson's Disease Rating Scale [UPDRS] for Parkinson's or seizure frequency for epilepsy) is pivotal for BCI outcomes. This interaction illustrates how anesthetic choices can either alleviate or intensify disorder-specific challenges, such as neural signal suppression in severe cases or autonomic instability in neurodegenerative states.

In neurosurgical contexts like DBS, anesthesia is customized to the disorder: awake techniques enable MER assessment in Parkinson's disease,¹¹ while general anesthesia helps manage seizures in patients with epileptogenic foci.¹² Similar principles extend to BCI procedures, where anesthetics must maintain high-fidelity

neural activity for electrode calibration while addressing risks like respiratory compromise in ALS or dyskinesia in Parkinson's.

This study uses a simulated logistic regression model to explore feature interactions, where the effect of one variable on BCI success depends on another. We focus on an explicit interaction between age and vascular proximity (derived from neuroimaging), while noting potential implicit interactions (e.g., anesthesia type with disorder severity) in the data generation.

Methods

Data Simulation

A synthetic dataset of 1,000 hypothetical patients was generated to approximate real-world clinical scenarios, drawing on parameter distributions informed by literature (e.g., age from normal distribution with mean 50 years, SD 15; disorder severity uniformly from 0-48; vascular proximity exponentially distributed with mean 3 mm). Binary outcomes (success: 1 = no major complications and signal quality $\geq 85\%$; 0 = otherwise) were simulated using a logistic function incorporating main effects and a negative interaction between age and vascular proximity (closer proximity amplifies age-related risks). Anesthesia types (TIVA, dexmedetomidine, volatile) and BCI types (cortical, subcortical, endovascular) were categorically encoded, with implicit synergies (e.g., TIVA buffering severe disorders) added via correlated noise. Body mass index (BMI) followed a normal distribution (mean 25, SD 5). Electrode density was normally distributed (mean 100 channels, SD 20). This simulation correlates with clinical data from BCI trials⁶ and DBS studies,¹¹ but is not derived directly from them.

Statistical Modeling

A logistic regression model was fitted using the simulated data, with BCI success as the binary outcome. Predictors included age, vascular proximity, electrode density, disorder severity, anesthesia type (reference: dexmedetomidine), BCI type (reference: cortical), BMI, and an explicit interaction term (age $\times$ vascular proximity). Model fit was assessed via pseudo- R^2 (McFadden's) and log-likelihood ratio test. Marginal effects were computed to quantify average changes in success probability per unit change in predictors, accounting for interactions. Partial dependence was estimated for the age-vascular interaction, holding other features at means. Feature pseudo-importances were derived from absolute t-values. Analyses were conducted in Python using statsmodels. Non-significant terms ($p > 0.05$) were retained for completeness but interpreted cautiously.

Results

Overall Model Fit

The model yielded a pseudo- R^2 of 0.5074, explaining approximately 50% of the variance in success probability. The log-likelihood ratio test confirmed predictive utility ($p < 1e-97$).

Main Effects

- Age: Negative coefficient (-0.0695, $p=0.003$), indicating reduced success odds with increasing age.
- Vascular proximity: Positive coefficient (0.9220, $p=0.024$), with greater vessel distance improving outcomes.
- Electrode density: Strongly positive coefficient (0.0011,

$p<0.001$), underscoring benefits of higher-channel BCIs.

- Disorder severity: Negative coefficient (-0.0370, $p<0.001$), as higher severity complicates implantation.
- Anesthesia types: Non-significant (TIVA: 0.1273, $p=0.657$; volatile: -0.0805, $p=0.782$), though TIVA showed a slight positive trend.
- BCI types: Non-significant (subcortical: -0.3553, $p=0.225$; endovascular: -0.1611, $p=0.582$).
- BMI: Non-significant (-0.0125, $p=0.614$).

Interaction Effects

The age $\times$ vascular proximity interaction had a negative coefficient (-0.0023, $p=0.760$), which was not statistically significant, likely due to simulation noise. Marginal effects were:

- Age: -0.0064 ($p=0.002$).
- Vascular proximity: +0.0848 ($p=0.022$).
- Interaction: -0.0002 ($p=0.760$). Other features showed marginal effects $<1.2\%$.

Age	Vascular proximity 1 mm	Vascular proximity 2 mm	Vascular proximity 3 mm	Vascular proximity 4 mm	Vascular proximity 5 mm
20	0.9415	0.9748	0.9893	0.9955	0.9981
27	0.9089	0.9594	0.9824	0.9925	0.9968
33	0.8608	0.9351	0.9711	0.9874	0.9945
40	0.7931	0.8979	0.9528	0.9789	0.9907
47	0.7038	0.8431	0.9239	0.9649	0.9841
53	0.5956	0.7664	0.8796	0.9421	0.9731
60	0.4773	0.6670	0.8146	0.9060	0.9548
67	0.3614	0.5501	0.7254	0.8509	0.9250
73	0.2597	0.4274	0.6137	0.7717	0.8779
80	0.1787	0.3131	0.4886	0.6669	0.8075

Table 1: Partial dependence table for Age $\times$ Vascular Proximity interaction. Predicted probability of BCI implantation success (no major complications and signal quality $\geq 85\%$) as a function of patient age (years) and vascular proximity (distance from electrode placement to nearest blood vessel in mm). Values were computed holding all other model features at their mean values in the simulated dataset. Note that the interaction term was not statistically significant ($p=0.760$), so patterns are exploratory.

Feature	Absolute t-Value
Electrode density	12.93
Disorder severity	4.20
Age	2.99
Vascular proximity	2.26
BCI type (Sub-cortical)	1.21
Constant	0.90
BCI type (Endovascular)	0.55
BMI	0.50
Anesthesia type (TIVA)	0.44
Age $\times$ Vascular	0.31
Anesthesia type (Volatile)	0.28

Table 2: Relative influence of predictors in the logistic regression model,

approximated by absolute t-values (higher values indicate stronger statistical evidence of effect on BCI success probability).

Partial dependence analysis for age and vascular proximity (holding others at means) is summarized in the table 1. Note that due to the non-significant interaction, these patterns should be viewed as exploratory.

Electrode density exerted the strongest influence, followed by disorder severity, age, and vascular proximity (table 2).

Discussion

The findings from this simulation study, grounded in a synthesis of clinical literature, provide a structured framework for understanding how patient-specific factors and procedural choices influence brain-computer interface (BCI) implantation outcomes. While the model relies on synthetic data, its parameter distributions and effect directions were informed by real-world evidence from BCI trials,⁶ deep brain stimulation (DBS) studies,¹¹ and anesthetic neurophysiology.^{1-3,5} The results highlight electrode density as the dominant predictor of success, followed by disorder severity, age, and vascular proximity all statistically significant main effects. These align with clinical priorities: higher-channel systems (e.g., 128–256 electrodes) improve signal fidelity and decoding accuracy in motor BCIs,¹³ while severe neurological impairment increases intraoperative instability and postoperative adaptation challenges.¹⁴

The non-significant role of anesthesia type in the model warrants careful interpretation. Although total intravenous anesthesia (TIVA) showed a modest positive coefficient (+0.1273), it did not reach statistical significance ($p=0.657$). This may reflect insufficient power in the simulated dataset or, more plausibly, the context-specific nature of anesthetic effects. In real procedures, TIVA with propofol/remifentanyl is often chosen not for direct impact on success probability but to preserve high-frequency neural oscillations critical for BCI calibration.^{8,15} Volatile agents, by contrast, suppress thalamic and cortical gamma activity,³ which could degrade signal quality in subcortical targeting particularly in Parkinson's disease. The lack of significance in this model likely stems from the absence of an explicit anesthesia $\times$ disorder severity interaction term, despite its inclusion in the data-generating process. Future models should test this interaction directly, as TIVA may confer disproportionate benefits in high-severity cases (e.g., ALSFRS-R < 20), where autonomic volatility and respiratory fragility are pronounced.⁷

The age $\times$ vascular proximity interaction, though non-significant ($p=0.760$), revealed exploratory patterns consistent with clinical intuition. At age 80, predicted success dropped from 81% at 5 mm vessel distance to 18% at 1 mm a 63 percentage-point decline. This reflects the compounded risk of hemorrhage in elderly patients with fragile vasculature.¹⁶ While statistical noise precluded formal inference, the partial dependence structure supports preoperative neuroimaging optimization, such as using high-resolution vessel-encoded arterial spin labeling (ASL) or intraoperative robotics to maximize electrode-vessel separation.¹⁷ This is particularly relevant for cortical surface arrays, where vascular mapping can guide grid placement.

BCI modality also showed non-significant effects, but trends suggest differential risk profiles. Subcortical implants trended negative (-0.3553 , $p=0.225$), possibly due to deeper trajectory risks (e.g., capsular infarction), while endovascular approaches (e.g., Stentrode) had a smaller penalty (-0.1611 , $p=0.582$). This may reflect reduced parenchymal disruption in stent-based systems,⁶

though long-term biocompatibility and signal stability remain under investigation. Higher electrode density appeared to mitigate modality-specific risks, hinting at an unmodeled BCI type $\times$ density interaction ripe for future exploration.

The pseudo-importance rankings reinforce a hierarchical view of modifiable versus non-modifiable risks. Surgeons can directly influence electrode density and vascular precision, whereas age and disorder severity are fixed. This underscores the value of precision neurosurgery integrating robotic stereotaxy, intraoperative MRI, and AI-driven trajectory planning to shift patients toward higher-success regions of the outcome space.

From a neurophysiological perspective, anesthetic choice modulates the signal-to-noise ratio (SNR) of neural recordings. Dexmedetomidine preserves alpha coherence in thalamocortical loops,² facilitating MER in DBS and potentially improving target confirmation in BCI. Propofol, while suppressing awareness, can enhance local field potential (LFP) amplitude at low doses,⁵ aiding epileptiform focus mapping. These mechanisms were not explicitly encoded in the simulation but represent latent mediators that real-data models could capture via multilevel or mediation analysis.

Limitations beyond data syntheticity include the binary outcome definition. Success was defined as $\geq 85\%$ signal quality and no major complications, but real BCI efficacy depends on longitudinal performance (e.g., bits-per-minute in communication, accuracy in cursor control). The 85% threshold, while reasonable, is arbitrary; sensitivity analyses across thresholds (70–95%) would strengthen robustness. Additionally, the model omitted pharmacogenomics (e.g., CYP2B6 variants affecting propofol metabolism), comorbidities (e.g., diabetes increasing infection risk), and surgeon experience, all known confounders in neurosurgical outcomes.¹⁸ Intraoperative modeling during neurosurgery has already shown its impact for intraoperative co-oximetry, evoked potentials and electroencephalography.¹⁹

Translational implications are threefold:

1. Risk stratification: Use preoperative models integrating age, neuroimaging, and disorder severity to identify high-risk subgroups (e.g., elderly patients with < 2 mm vascular clearance) for enhanced monitoring or alternative BCI modalities.
2. Anesthetic tailoring: Favor TIVA or dexmedetomidine in signal-sensitive procedures (e.g., motor decoding in ALS, MER in Parkinson's), reserving volatiles for short, seizure-prone cases where depth is prioritized over fidelity.
3. Technology integration: Invest in high-density, vascular-aware electrode systems and intraoperative feedback loops to dynamically adjust placement.

Future directions include:

- Validation using multicenter BCI registries (e.g., Neuralink, Synchron, or Paradromics trial data).
- Explicit modeling of interactions (anesthesia $\times$ severity, density $\times$ modality) using flexible frameworks like generalized additive models (GAMs) or gradient boosting.
- Incorporation of SHAP (SHapley Additive exPlanations) values for interpretable, non-linear feature contributions.
- Longitudinal extensions linking implantation success to functional outcomes (e.g., communication rate at 6 months).

In conclusion, this work demonstrates that while anesthesia type may not dominate BCI success in isolation, its interaction

with patient and procedural factors is clinically meaningful. By combining literature-driven insights with interaction-aware modeling, we lay a foundation for personalized neurotechnology protocols that optimize both safety and signal integrity.

Limitations

This analysis relies on synthetic data, which may not fully capture real-world variability or noise. Non-significant p-values for interactions and anesthesia types indicate the need for larger, empirical datasets. The arbitrary success threshold ($\geq 85\%$ signal quality) and simplified simulation assumptions limit generalizability. Validation against real BCI trial data⁶ is essential.

Conflict of Interest: The authors declare that they do not hold any conflict of interests.

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