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Review Article

Electroencephalographic Signatures of Dexmedetomidine and Propofol Sedation: A Structured Narrative Review

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ABSTRACT

Background: Carpenters perform repetitive and forceful upper-limb activities that may increase the risk of lateral Background: Dexmedetomidine and propofol are frequently used intravenous sedatives with distinct molecular targets and effects on arousal networks. Although both agents can achieve clinically comparable sedation, their electroencephalographic manifestations differ in spectral composition, spatial organization, processed index behavior, functional connectivity, and emergence dynamics. These differences are clinically important because simplified depth-of-sedation indices may not represent equivalent neurophysiological states across drug classes. Objective: This structured narrative review aimed to critically synthesize contemporary evidence regarding the raw and processed electroencephalographic signatures associated with dexmedetomidine and propofol sedation in adults. Methods: PubMed/MEDLINE, Scopus, Web of Science, and CENTRAL were searched for English-language human studies and relevant reviews published principally between January 2014 and May 2024. Foundational earlier studies were considered when necessary to explain established propofol EEG mechanisms. Literature selection prioritized direct comparative studies, controlled volunteer investigations, prospective clinical studies, secondary EEG analyses, and mechanistically informative reports addressing spectral power, spectral edge frequency, bispectral index, entropy, coherence, functional connectivity, and emergence trajectories. Findings were organized through a structured thematic synthesis rather than statistical pooling. Results: Dexmedetomidine was commonly associated with increased slow-delta activity accompanied by frontal spindle- or alpha-range oscillations and relative preservation of selected coherence measures. Propofol produced prominent slow-wave activity with concentration- and state-dependent frontal alpha oscillations, reduced faster-frequency activity, and greater disruption of selected long-range cortical interactions. At comparable clinical sedation levels, bispectral index and spectral edge frequency values frequently differed between agents. Evidence concerning entropy, connectivity, and emergence trajectories was informative but less extensively replicated. Conclusion: Dexmedetomidine and propofol produce distinct, context-dependent EEG profiles that should not be interpreted through interchangeable processed-index thresholds. Drug-specific EEG interpretation may improve sedation assessment, but standardized prospective studies linking electrophysiological patterns with patient-centered outcomes remain necessary. Keywords: Dexmedetomidine; Propofol; Electroencephalography; Conscious Sedation; Bispectral Index; Spectral Analysis; Anesthesia Monitoring.

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INTRODUCTION

Procedural and monitored sedation are integral to contemporary diagnostic and therapeutic practice, allowing potentially uncomfortable or anxiety-provoking interventions to be completed while maintaining an appropriate balance between patient comfort, responsiveness, ventilation, and hemodynamic stability. Conventional clinical sedation scales remain useful for bedside assessment, but they provide intermittent and behavior-dependent estimates of sedation. They may be influenced by procedural stimulation, hearing impairment, neuromuscular limitations, baseline cognition, language, and the timing of assessment.

Electroencephalography offers a continuous and direct measure of cortical electrical activity and therefore provides an additional physiological perspective on drug-induced alterations in consciousness (1).

The clinical EEG contains information across multiple frequency bands and spatial relationships. Slow and delta activity generally occupy frequencies below approximately 4 Hz, theta activity occurs between approximately 4 and 8 Hz, alpha activity between 8 and 13 Hz, and beta activity above approximately 13 Hz, although exact boundaries vary among studies and analytic systems. Sedative and anesthetic drugs alter not only the absolute or relative power within these bands but also the regional distribution, temporal stability, phase relationships, coherence, and interactions among cortical networks. Processed EEG monitors simplify portions of this complex information into numerical indices, such as the bispectral index, spectral entropy, response entropy, state entropy, or spectral edge frequency. These measures can support clinical monitoring, but their accuracy and interpretation depend on the pharmacological state from which their algorithms were developed (1).

Dexmedetomidine and propofol are widely used intravenous sedatives, yet they act through substantially different neuropharmacological pathways. Dexmedetomidine is a highly selective α_2-adrenergic receptor agonist that reduces central noradrenergic transmission, particularly through actions involving the locus coeruleus and interconnected sleep–wake networks. Its clinical profile is characterized by sedation, anxiolysis, sympatholysis, and a comparatively limited direct effect on respiratory drive. Patients may remain arousable to verbal or tactile stimulation, particularly at light-to-moderate infusion levels. Propofol primarily enhances γ-aminobutyric acid type A receptor-mediated inhibitory neurotransmission and produces dose-dependent sedation, hypnosis, amnesia, immobility, and cardiorespiratory depression. Increasing propofol concentration progressively alters thalamocortical and corticocortical communication and may produce EEG states extending from light sedation to unconsciousness and burst suppression (2).

The term “sedation depth” can therefore conceal important neurophysiological differences. Two patients may demonstrate similar scores on the Modified Observer’s Assessment of Alertness/Sedation scale while exhibiting different spectral patterns, levels of cortical integration, or responsiveness to stimulation. Similarly, identical values from a commercial processed EEG monitor may not represent equivalent states when one patient receives dexmedetomidine and another receives propofol. Recognition of these drug-specific patterns is important because uncritical reliance on a single numerical threshold may lead to unnecessary dose escalation, inadequate sedation, delayed recovery, or failure to distinguish expected drug effects from pathological cerebral slowing.

Dexmedetomidine-associated EEG activity has frequently been described as sharing selected characteristics with non-rapid eye movement sleep. This comparison is based primarily on the presence of slow-delta activity and spindle- or alpha-range oscillations, particularly over frontal regions. However, pharmacological sedation should not be considered identical to physiological sleep. Similarity in selected oscillatory components does not establish equivalence in sleep staging, sensory processing, autonomic regulation, restorative function, network integration, or arousability. The expression of dexmedetomidine-associated oscillations also depends on dose, participant age, behavioral state, electrode location, and analytic method (3,4).

Propofol produces similarly complex and state-dependent EEG changes. At lighter levels, faster-frequency activity may remain evident, while deeper sedation and unconsciousness are associated with increased slow-wave activity and prominent frontal alpha oscillations. At still higher concentrations, alpha activity may diminish as the EEG progresses toward burst suppression. Propofol should therefore not be described simply as suppressing alpha activity. Its EEG signature is better understood as a dose- and state-dependent reorganization of slow, alpha, and higher-frequency activity, accompanied by alterations in cortical communication and responsiveness (1,12).

Direct comparisons of dexmedetomidine and propofol have identified differences in spectral power, density spectral array appearance, bispectral index values, spectral edge frequency, entropy, coherence, connectivity, and emergence trajectories (3–9). However, the literature remains heterogeneous. Studies

have involved healthy volunteers, endoscopic procedures, surgery under spinal anesthesia, intracranial interventions, major surgery, and postoperative emergence. Sedation targets, loading doses, infusion rates, co-administered medications, clinical stimulation, EEG montage, frequency-band definitions, and analysis windows have varied considerably.

Existing clinical reviews of dexmedetomidine and propofol have predominantly focused on hemodynamic stability, respiratory effects, recovery time, postoperative complications, or delirium (10,11). These outcomes are clinically important, but they do not fully explain the cortical states generated by the two drugs. A focused synthesis of EEG evidence can provide a mechanistic framework for understanding why patients receiving clinically comparable sedation may display different processed monitor values, arousal characteristics, and recovery profiles.

A structured narrative approach is particularly appropriate for this topic because the relevant evidence encompasses diverse study designs and EEG methods that cannot be meaningfully reduced to a single pooled estimate. The purpose of the present review was therefore to critically examine and integrate contemporary evidence concerning the EEG signatures of dexmedetomidine and propofol sedation. The review addresses spectral power, alpha–delta organization, spectral edge frequency, bispectral index behavior, entropy, density spectral array patterns, functional connectivity, and emergence trajectories. It also considers the limitations of applying standardized EEG thresholds across pharmacologically distinct sedative states and identifies priorities for future EEG-guided precision sedation research.

METHODS

This article was designed as a structured narrative review. The narrative approach was selected because the relevant literature includes randomized trials, controlled volunteer studies, prospective clinical comparisons, mechanistic EEG investigations, secondary analyses, and foundational neurophysiological studies. Considerable heterogeneity exists in participant populations, procedure type, sedation target, drug administration, EEG acquisition, frequency-band definitions, behavioral endpoints, and statistical reporting. These characteristics limited the suitability of formal quantitative pooling but supported a transparent thematic synthesis of the available evidence. The review was prepared with attention to the quality principles described in the Scale for the Assessment of Narrative Review Articles, including justification of the review's importance, statement of aims, description of the literature search, appropriate referencing, scientific reasoning, and presentation of clinically relevant conclusions (13).

A structured literature search was conducted using PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Central Register of Controlled Trials. The principal search period extended from January 2014 to May 2024 to capture contemporary EEG acquisition systems, processed monitoring techniques, and analytic approaches. Earlier foundational studies were retained when they provided essential mechanistic context, particularly regarding propofol-associated loss and recovery of consciousness or the established interpretation of anesthetic EEG patterns.

Search terms were organized around four concepts: dexmedetomidine, propofol, sedation, and electroencephalography. Terms included "dexmedetomidine," "propofol," "procedural sedation," "conscious sedation," "monitored anesthesia care," "electroencephalography," "EEG," "spectral power," "spectral edge frequency," "bispectral index," "entropy," "coherence," "functional connectivity," "density spectral array," "loss of consciousness," and "emergence." Boolean operators were used to combine drug, clinical, and EEG concepts. Reference lists of relevant primary studies and review articles were also examined to identify additional mechanistically important publications.

The literature selection process prioritized adult human studies that examined dexmedetomidine- or propofol-associated EEG changes during sedation, reduced responsiveness, unconsciousness, or emergence. Direct head-to-head comparisons were given the greatest interpretive weight because they reduced differences arising from study setting and analytic methodology. Controlled volunteer

investigations and prospective clinical studies were included when they provided detailed information concerning drug-related and state-related EEG effects. Secondary analyses were considered when they addressed emergence trajectories or neurocognitive associations not available in the primary reports.

Studies were considered especially relevant when they reported quantitative or visually interpretable EEG outcomes, including absolute or relative spectral power, power spectral density, spectral edge frequency, bispectral index, state entropy, response entropy, density spectral array patterns, coherence, phase relationships, functional connectivity, or changes during emergence. Reports focused exclusively on hemodynamic outcomes without EEG analysis were not used as primary evidence for electrophysiological conclusions. Animal studies, isolated case reports, non-clinical signal-processing reports without human drug comparisons, and publications without sufficient description of the EEG findings were not emphasized.

Information was extracted narratively from the selected literature. The principal elements considered were study design, participant population, procedure or experimental setting, sedation regimen, targeted level of responsiveness, EEG equipment and montage, timing of recording, reported spectral or processed outcomes, and the principal between-drug or state-related findings. Because the included literature used non-uniform EEG definitions and summary measures, no pooled effect estimates were calculated. Numerical findings were interpreted at the level of the individual study and were not combined when sedation depth, recording conditions, or measurement methods were not sufficiently comparable.

The synthesis was organized into predefined clinical and neurophysiological domains: overall spectral organization, slow-delta activity, alpha- and spindle-range oscillations, theta and beta activity, spectral edge frequency, bispectral index, entropy, density spectral array appearance, coherence and functional connectivity, emergence trajectories, and implications for neurocognitive outcomes. Findings observed in multiple independent studies were distinguished from observations primarily supported by a single study. Mechanistic interpretations were considered alongside methodological limitations, and causal conclusions were avoided when the literature demonstrated only associations.

CHARACTERISTICS OF THE EVIDENCE BASE

The most informative literature included direct comparative and mechanistic studies conducted in volunteer and clinical populations. The settings ranged from controlled sedation experiments to endoscopy, surgery under spinal anesthesia, major spinal procedures, intracranial aneurysm embolization, and postoperative emergence. This diversity increased the clinical breadth of the evidence but also introduced substantial variation in stimulation, co-administered medication, regional anesthesia, patient age, physiological condition, and targeted sedation depth.

Table 1. Principal Studies Informing the Structured Narrative Synthesis

Study	Design and setting	EEG domains	Principal contribution
Akeju et al. (2014) (3)	Comparative human sedation study	Spectral power, coherence, spatial organization	Demonstrated distinct spectral and coherence patterns during dexmedetomidine and propofol sedation.
Scheinin et al. (2018) (4)	Controlled comparative volunteer study	Drug-related versus state-related EEG effects	Distinguished effects attributable to the sedative agent from effects associated with behavioral responsiveness.
Maksimow et al. (2019) (5)	Controlled comparative infusion study	Spectral entropy, state entropy, response entropy, autonomic measures	Showed that entropy measures and their relationship with sedation depth differed between agents.
Lee et al. (2021) (6)	Comparative clinical study during major spinal surgery	BIS, SEF95, spectral power	Demonstrated differences in processed indices and spectral patterns under clinically targeted sedation.
Kim et al. (2021) (7)	Comparative sedation study during spinal anesthesia	BIS, frequency-band power, recovery	Reported drug-dependent EEG and recovery characteristics at comparable clinical sedation levels.
Ni et al. (2022) (8)	Comparative study during intracranial aneurysm embolization	BIS, SEF95, density spectral array	Illustrated clinically visible differences in density spectral array and processed monitoring.
Hesse et al. (2022) (9)	Secondary analysis of a randomized study	Emergence EEG trajectories, delirium association	Examined differences in EEG recovery patterns and their relationship with postoperative outcomes.
Purdon et al. (2013) (12)	Foundational propofol consciousness study	Spectral signatures of loss and recovery of consciousness	Provided mechanistic context for propofol-associated slow and frontal alpha oscillations.

The evidence base was not uniform in methodological strength. Some studies offered controlled comparisons at predefined sedation states, whereas others were conducted in clinically complex environments in which procedure-related stimulation, analgesia, regional anesthesia, age, and

comorbidities could modify EEG activity. Several studies used frontal commercial monitors, while others used broader electrode montages and advanced coherence or connectivity analyses. These differences are important because an apparently inconsistent finding may reflect variation in electrode location, behavioral state, or signal-processing method rather than a true biological contradiction.

SPECTRAL ORGANIZATION DURING DEXMEDETOMIDINE SEDATION

Dexmedetomidine sedation is commonly characterized by increased slow-delta activity accompanied by frontal spindle- or alpha-range oscillations. This organization is consistent with reduced activation of central noradrenergic arousal systems and altered interactions among thalamic, cortical, and sleep–wake networks. The presence of oscillatory activity in spindle-related frequency ranges has contributed to the frequent description of dexmedetomidine as producing a sleep-like state (3,4).

The term “sleep-like,” however, requires careful qualification. Dexmedetomidine may reproduce selected electrophysiological features associated with non-rapid eye movement sleep, but it does not necessarily reproduce natural sleep architecture, cyclical progression through sleep stages, sensory gating, restorative physiology, or normal autonomic patterns. Furthermore, the observed alpha- or spindle-range activity varies with dose, recording site, participant characteristics, and level of responsiveness. A patient who remains responsive to verbal stimulation may demonstrate a different EEG organization from a patient who is unresponsive despite receiving the same agent. Dexmedetomidine-associated slow oscillations are frequently most visible over frontal regions. Increased power in lower frequencies may coexist with organized alpha- or spindle-range activity rather than replacing it. This combination distinguishes dexmedetomidine from a simple model of generalized cortical slowing. The coexistence of slow and organized faster oscillations may reflect a state in which arousal systems are suppressed but selected thalamocortical interactions remain structured.

The electrophysiological pattern also helps explain the clinically observed arousability associated with dexmedetomidine. Patients may appear deeply sedated during the absence of stimulation but respond relatively promptly to verbal or tactile input. This characteristic does not imply that the EEG remains normal; rather, it suggests that the network configuration produced by dexmedetomidine may preserve selected pathways that support stimulus-induced transition toward responsiveness.

SPECTRAL ORGANIZATION DURING PROPOFOL SEDATION

Propofol produces dose-dependent alterations across slow, delta, alpha, beta, and higher-frequency activity. At lighter levels of sedation, the EEG may retain faster activity, although redistribution of power begins as responsiveness decreases. With deeper sedation and loss of responsiveness, prominent slow-wave and frontal alpha oscillations commonly emerge. At very high concentrations, the EEG may progress toward discontinuity and burst suppression (1,12).

A simplified description of propofol as producing only generalized delta activity or suppressing alpha oscillations is therefore inaccurate. Frontal alpha activity is one of the most recognizable features of propofol-associated unconsciousness. Its expression depends on dose, age, cortical region, and behavioral state. Alpha power may be prominent in frontal channels while posterior alpha rhythms associated with wakefulness diminish. This anterior redistribution is part of the broader reorganization of thalamocortical dynamics produced by propofol.

Propofol also increases slow oscillations that may reflect alternating periods of cortical neuronal activity and reduced excitability. These slow rhythms can modulate the amplitude and timing of alpha activity, producing characteristic patterns visible on density spectral array displays. As sedation deepens, beta and other faster-frequency activities generally decrease, although transient faster oscillations may occur during transitions or stimulation.

The propofol EEG should therefore be interpreted as a dynamic trajectory rather than a fixed pattern. The distinction among light sedation, deep sedation, loss of responsiveness, unconsciousness, and burst

suppression is clinically significant. A single processed value cannot fully represent these transitions, especially when the algorithm compresses multiple spectral and spatial features into one number.

DIRECT COMPARISON OF SPECTRAL POWER

Direct comparative studies demonstrate that dexmedetomidine and propofol can achieve similar behavioral sedation while producing different distributions of spectral power (3–8). Both agents increase slow-frequency activity, but differences arise in the relative magnitude, spatial location, and interaction of slow and alpha-range oscillations.

Dexmedetomidine is more frequently associated with spindle-like or alpha-range activity superimposed on slow-delta oscillations. Propofol may generate stronger global slow-wave activity and prominent frontal alpha oscillations at reduced responsiveness. The distinction is therefore not that one drug produces alpha activity while the other does not. Rather, the drugs differ in the frequency, spatial distribution, coherence, and behavioral context of their alpha-range activity. Theta activity has been reported under both agents, but its clinical interpretation is less consistent. In some studies, theta power increases as part of a broad shift toward lower frequencies. In others, differences between agents are influenced by the chosen frequency boundaries and analysis period. Beta and higher-frequency power generally decrease with deepening sedation, although the degree of reduction may depend more strongly on sedation depth and stimulation than on drug identity alone. These observations caution against using isolated frequency-band power as a complete measure of sedation. Interpretation should consider the full spectral profile, regional distribution, temporal stability, clinical responsiveness, and administered drug.

Table 2. Comparative Interpretation of Major EEG Domains

EEG domain	Dexmedetomidine	Propofol	Clinical interpretation
Slow-delta activity	Increased, often with organized spindle- or alpha-range activity	Increased, frequently prominent with deeper sedation	Both agents produce cortical slowing; relative organization differs.
Alpha-range activity	Frontal spindle- or alpha-like oscillations commonly described	Frontal alpha oscillations may become prominent during reduced responsiveness	Alpha activity is drug- and state-dependent and should not be interpreted in isolation.
Theta activity	Variable and influenced by sedation depth	Often increases as part of broader low-frequency dominance	Evidence is heterogeneous and montage-dependent.
Beta and faster activity	Generally decreases with deeper sedation but may persist during arousable states	Generally decreases with deeper sedation and returns during emergence	Changes reflect sedation depth as well as drug effects.
SEF95	Often comparatively higher at similar clinical sedation levels	Frequently lower at comparable clinical scores	Thresholds are not necessarily interchangeable.
BIS	May remain comparatively higher despite clinically evident sedation	Frequently lower at comparable behavioral sedation	BIS should be interpreted in a drug-specific context.
Entropy	Changes with depth but follows a drug-specific relationship with responsiveness	Changes with depth and may differ from dexmedetomidine at similar clinical states	Evidence remains limited and algorithm-dependent.
Coherence/connectivity	Selected frontal coherence and network relationships may be relatively preserved	Greater disruption of selected long-range interactions has been reported	Findings are mechanistically important but require replication.
Emergence	May show gradual redistribution of slow and alpha activity	May show a more marked transition toward faster activity	Evidence is mainly derived from secondary analysis.

SPECTRAL EDGE FREQUENCY

Spectral edge frequency represents the frequency below which a specified proportion of total EEG power is contained, commonly 95%. It provides a compact summary of spectral distribution but does not convey spatial organization, phase relationships, or the presence of specific oscillatory peaks. SEF95 generally decreases as EEG power shifts toward lower frequencies.

Comparative clinical studies have frequently reported lower SEF95 values during propofol than during dexmedetomidine at similar clinical sedation levels (6–8). This observation is consistent with a comparatively greater concentration of propofol-associated power within lower frequencies under the studied conditions. However, the magnitude of the difference varies among studies because SEF95 is influenced by electrode placement, filtering, artifact handling, sedation depth, procedure-related stimulation, and the analytic device.

SEF95 should not therefore be treated as a universal threshold defining equivalent sedation under both drugs. A lower value during propofol may reflect the expected pharmacological spectral distribution rather than excessive sedation. Conversely, a comparatively higher value during dexmedetomidine does not necessarily indicate inadequate sedation. Clinical interpretation requires integration with responsiveness, respiratory status, hemodynamics, drug dose, and the raw or displayed spectral pattern.

BISPECTRAL INDEX

The bispectral index is one of the most widely used processed EEG measures in anesthesia and sedation. It combines information derived from several EEG characteristics to generate a dimensionless value, generally ranging from 0 to 100. Lower values are typically associated with deeper hypnotic states, but the relationship is influenced by drug class, electromyographic activity, age, neurological condition, and signal quality.

Several comparative studies have reported that dexmedetomidine may produce a higher BIS than propofol at clinically similar levels of sedation (6–8). This discrepancy is important because it indicates that identical clinical responsiveness does not necessarily produce identical BIS values across the two drugs. The difference may arise because the BIS algorithm weights spectral and bispectral features that are expressed differently during α_2-adrenergic and GABAergic sedation.

A higher BIS during dexmedetomidine should not automatically be interpreted as inadequate sedation. Escalating dexmedetomidine solely to reach a propofol-derived numerical target could increase bradycardia, hypotension, delayed recovery, or other dose-related effects without achieving a clinically meaningful benefit. Similarly, a lower BIS during propofol may represent its expected spectral signature rather than an unnecessarily deep state.

BIS remains potentially useful when interpreted as part of a multimodal assessment. Trends within the same patient may be more informative than direct comparison with a universal threshold. Raw EEG, density spectral array, clinical sedation scores, respiratory function, and procedural stimulation should be considered alongside the processed number.

ENTROPY-BASED MEASURES

State entropy and response entropy are processed measures derived from the irregularity and frequency content of EEG and frontal electromyographic signals. They are intended to reflect cortical state and responsiveness, but their relationship with clinical sedation varies by drug and signal composition.

Maksimow et al. demonstrated that entropy measures changed during both dexmedetomidine and propofol infusions but did not necessarily represent equivalent behavioral states in an identical manner (5). Differences in autonomic activity and spectral composition further complicated direct comparison. These findings support the broader conclusion that processed algorithms are sensitive to the pharmacological pathway producing sedation.

The entropy literature remains less extensive than the evidence concerning BIS and spectral power. Consequently, no single drug-specific entropy threshold can be recommended. Future studies should report state entropy, response entropy, raw spectral measures, sedation scores, and electromyographic contamination simultaneously to improve interpretability.

DENSITY SPECTRAL ARRAY PATTERNS

Density spectral array displays present EEG power across frequency and time, allowing clinicians to recognize evolving patterns that may be obscured by a single numerical index. These displays can demonstrate the emergence, persistence, or disappearance of slow, theta, alpha, and beta activity during drug administration and recovery. Clinical studies have illustrated differences in the density spectral array appearance of dexmedetomidine and propofol (8). Dexmedetomidine may show persistent slow activity accompanied by organized spindle- or alpha-range bands, whereas propofol may demonstrate stronger low-frequency dominance with a distinct frontal alpha component. The exact appearance depends on

montage and display settings. Density spectral array interpretation may be particularly valuable when the BIS or another processed index appears inconsistent with the clinical examination. A clinician may identify expected drug-specific oscillations, artifact, electromyographic activity, or progression toward excessive slowing. However, visual interpretation requires training, and standardized educational frameworks remain underdeveloped.

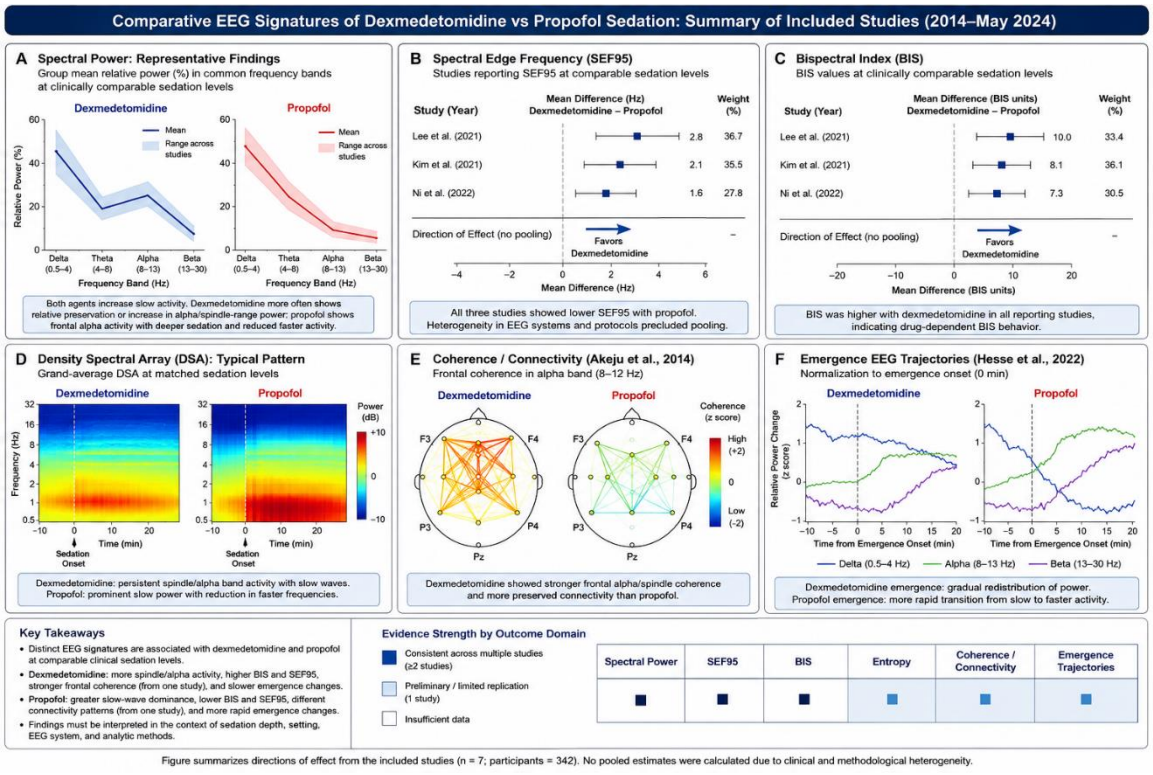

Figure 1. Comparative electroencephalographic characteristics associated with dexmedetomidine and propofol sedation. The panelled figure summarizes the principal qualitative findings identified across comparative and mechanistic studies. Dexmedetomidine is commonly associated with slow-delta activity accompanied by frontal spindle- or alpha-range oscillations, comparatively higher BIS and SEF95 values at similar clinical sedation levels, and relative preservation of selected frontal coherence relationships. Propofol is associated with prominent slow-wave activity, state-dependent frontal alpha organization, comparatively lower processed index values, and greater disruption of selected long-range cortical interactions. Entropy, connectivity, and emergence findings remain less extensively replicated than spectral power and processed-index observations. The figure represents directions and patterns of evidence and does not present pooled effect estimates.

COHERENCE AND FUNCTIONAL CONNECTIVITY

Spectral power describes the amount of activity within a frequency range but does not fully capture communication among cortical regions. Coherence and functional connectivity analyses examine relationships between signals recorded at different locations and may therefore provide insight into network organization during altered consciousness. Akeju et al. reported distinct coherence patterns during dexmedetomidine- and propofol-associated sedation (3). Dexmedetomidine demonstrated stronger frontal spindle- or alpha-range coherence and relative preservation of selected network relationships. Propofol produced a different spatial organization and greater disruption of selected long-range interactions during reduced responsiveness. These findings offer a plausible explanation for differences in arousability. A sedated patient may exhibit substantial slow-wave activity while retaining selected network configurations that permit a more rapid response to stimulation. In contrast, disruption of long-range communication may be associated with a more profound reduction in integrated cortical processing. Nevertheless, connectivity findings should be interpreted cautiously. They are affected by electrode density, reference selection, volume conduction, preprocessing, artifact correction, frequency-band definitions, and analytic model. The strongest comparative connectivity conclusions remain dependent on a limited number of studies. Replication using standardized high-density EEG and predefined analytic pipelines is required before connectivity can guide routine clinical sedation.

EMERGENCE AND RECOVERY TRAJECTORIES

Emergence from sedation is not necessarily the reverse of induction. EEG recovery may follow distinct trajectories according to drug pharmacokinetics, receptor mechanisms, age, surgical stimulation, analgesia, and the duration of administration. Changes in behavioral responsiveness may also occur before complete normalization of spectral or network activity.

Hesse et al. examined EEG trajectories during emergence from dexmedetomidine or propofol sedation and identified differences in the redistribution of slow, alpha, and faster-frequency activity (9). Dexmedetomidine-associated recovery appeared comparatively gradual and oscillatory, whereas propofol emergence demonstrated a more marked transition from slow-wave dominance toward faster activity. These findings are clinically relevant because a patient may regain responsiveness while the EEG continues to display residual drug-related organization. Conversely, an abrupt processed-index increase may be influenced by electromyographic activity or stimulation rather than complete cortical recovery. Emergence monitoring should therefore consider the temporal pattern of spectral change rather than relying solely on a final numerical value.

The available emergence evidence remains limited, particularly because some analyses were secondary and involved heterogeneous surgical populations. Additional prospective studies are needed to determine whether specific recovery trajectories predict delayed awakening, agitation, delirium, postoperative cognitive dysfunction, or other clinically important outcomes.

EEG SIGNATURES AND POSTOPERATIVE DELIRIUM

Postoperative delirium is a multifactorial neurocognitive disorder associated with age, frailty, pre-existing cognitive impairment, illness severity, surgery, inflammation, medications, pain, sleep disruption, and excessive anesthetic exposure. EEG-guided reduction of unnecessarily deep anesthesia has been investigated as a strategy to reduce delirium, but results have varied across populations and protocols (10).

Dexmedetomidine has been associated with lower delirium rates in some clinical contexts, and its sleep-related oscillatory features and relative preservation of selected network relationships have been proposed as possible mechanisms. However, the current EEG evidence does not demonstrate that a specific dexmedetomidine-associated spectral or connectivity pattern directly prevents delirium. Such an inference would require mediation analyses and trials specifically designed to connect drug exposure, EEG state, and neurocognitive outcome.

Similarly, propofol-associated slow-wave dominance or reduced connectivity should not be interpreted as inherently pathological. These patterns may represent expected pharmacological effects at the administered dose. The clinical concern arises when unnecessarily deep or prolonged states occur in vulnerable patients or when processed monitors fail to reflect individual susceptibility.

Future research should therefore move beyond simple comparison of average BIS values and examine whether the duration, intensity, or spatial characteristics of specific EEG states predict delirium independently of age, dose, procedure, and baseline cognitive status.

CLINICAL IMPLICATIONS

The principal clinical implication of the available evidence is that dexmedetomidine and propofol should not be monitored through identical EEG assumptions. Both can achieve clinically adequate sedation, but their EEG manifestations differ because they engage different receptor systems and neural networks.

Clinicians should interpret processed indices according to the administered agent. A relatively high BIS during dexmedetomidine may coexist with adequate clinical sedation and expected frontal spindle- or alpha-range activity. A lower BIS or SEF95 during propofol may reflect the expected shift toward slow and frontal alpha oscillations rather than excessive drug administration. No single numerical target should replace clinical assessment. Raw EEG and density spectral array displays may improve interpretation when available. Recognition of expected oscillatory patterns can help distinguish pharmacological sedation

from artifact, cerebral hypoperfusion, seizure activity, or progression toward burst suppression. However, raw EEG interpretation requires appropriate training and should not be presented as a substitute for cardiorespiratory monitoring.

Sedation assessment should remain multimodal. Clinical responsiveness, airway patency, ventilation, oxygenation, hemodynamics, procedural stimulation, analgesia, patient age, neurological history, drug dose, and EEG information should be integrated. The relative importance of each component depends on the clinical context.

STRENGTHS AND LIMITATIONS OF THE EVIDENCE

The principal strength of this narrative synthesis is its focused examination of neurophysiological outcomes rather than limiting comparison to hemodynamic or recovery endpoints. The review integrates spectral power, processed monitoring, entropy, density spectral arrays, connectivity, and emergence patterns, thereby providing a broader framework for understanding drug-specific EEG behavior.

The inclusion of both controlled volunteer studies and clinical investigations allows mechanistic findings to be considered alongside real-world sedation. Volunteer experiments provide precise control over drug administration and responsiveness testing, whereas clinical studies demonstrate how EEG patterns behave in the presence of surgery, regional anesthesia, procedural stimulation, and patient heterogeneity.

The evidence nevertheless has important limitations. The number of detailed head-to-head EEG studies remains limited, and many samples were relatively small. Procedures and populations varied substantially. Some studies involved healthy volunteers, while others included patients undergoing major surgery or intracranial intervention. The effects of age, comorbidity, baseline cognition, regional anesthesia, opioid administration, and procedural stimulation were not uniform. EEG acquisition and analysis also differed. Some studies used limited frontal montages associated with commercial processed monitors, whereas others used broader recordings and advanced coherence analyses. Frequency-band definitions, recording intervals, artifact rejection procedures, and statistical methods were not standardized. These differences prevented formal pooling and limit the generalizability of any single numerical comparison.

As a structured narrative review, literature selection was not intended to provide the exhaustive reproducibility of a prospectively registered systematic review. Although multiple databases and citation sources were searched, relevant unpublished studies or publications indexed under different terminology may have been missed. Formal study-level risk-of-bias grading and certainty-of-evidence assessment were not undertaken. The narrative conclusions should therefore be interpreted as a critical synthesis of the available evidence rather than as pooled estimates of treatment effect.

Publication bias is also possible. Studies demonstrating clear differences between drug-associated EEG patterns may be more likely to be published than studies showing minimal or inconsistent distinctions. Commercial EEG algorithms are proprietary, which further limits full evaluation of why processed values differ across pharmacological states.

FUTURE RESEARCH

Future studies should use adequately powered, prospective head-to-head designs with predefined EEG outcomes. Dexmedetomidine and propofol should be titrated to clearly specified behavioral targets, with simultaneous recording of drug dose, plasma or effect-site concentration where feasible, stimulation, analgesia, respiratory parameters, and hemodynamic variables.

EEG reporting should include electrode montage, reference, sampling frequency, filters, artifact rejection procedures, frequency-band definitions, analysis windows, and whether investigators were blinded to treatment allocation. Raw or minimally processed data should be reported alongside proprietary indices to permit mechanistic interpretation (14).

High-density EEG studies are needed to replicate connectivity and coherence findings. Standardized methods should examine frontal–parietal, interhemispheric, and thalamocortical relationships while

minimizing the effects of volume conduction and reference selection. Studies should distinguish drug-related effects from state-related effects by comparing participants at matched levels of responsiveness. Age-specific investigations are particularly important because aging alters alpha power, slow-wave activity, cortical connectivity, and sensitivity to sedatives. Evidence from younger volunteers cannot be assumed to apply directly to frail older adults or patients with pre-existing neurological disease (15).

Clinical trials should evaluate whether drug-specific EEG-guided titration improves outcomes compared with conventional clinical assessment or non-specific processed-index targets. Relevant endpoints include respiratory complications, hemodynamic instability, recovery time, agitation, delirium, postoperative cognitive function, patient satisfaction, procedural conditions, and resource utilization. Research should also determine whether machine-learning models trained on raw EEG can classify drug, sedation depth, and transition state more accurately than existing generalized indices. Such systems should be externally validated across devices, institutions, age groups, and clinical procedures before implementation.

CONCLUSION

Dexmedetomidine and propofol produce distinct but context-dependent electroencephalographic states during adult sedation. Both agents increase low-frequency activity, but they differ in the organization of slow, spindle- or alpha-range oscillations, processed index behavior, selected connectivity measures, and emergence trajectories. Dexmedetomidine commonly produces slow-delta activity accompanied by frontal spindle- or alpha-range oscillations and may preserve selected coherence relationships, whereas propofol produces prominent slow-wave activity with state-dependent frontal alpha organization and greater disruption of some long-range interactions. These patterns do not establish that one drug produces a universally more physiological or clinically superior brain state. They demonstrate that equivalent behavioral sedation does not necessarily represent equivalent neurophysiology. BIS, SEF95, entropy, and other processed measurements should therefore be interpreted in relation to the administered sedative, clinical responsiveness, and raw or spectrally resolved EEG findings. Standardized prospective research is required before agent-specific EEG targets can be recommended for routine precision-guided sedation.

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