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Review Article

Neurocognition after Electroencephalography Guided Anesthetic Induction with Dexmeditomidine in Neurosurgical Patients: A Case Series

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Received date: January 02, 2025; **Accepted date:** Febuary 12, 2025; **Published date:** March 07, 2025

Citation: Kunal K. Sharma, Sudhir Sharma, Vikrom Takkar, Meena Devi, Sharath Krishnaswami (2025), Neurocognition after Electroencephalography Guided Anesthetic Induction with Dexmeditomidine in Neurosurgical Patients: A Case Series, *J. Neuroscience and Neurological Surgery*, 17(3); **DOI:10.31579/2578-8868/355**

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Abstract

The use of Dexmeditomidine as the primary agent for induction of anesthesia has only been sparingly described due to the concern of quality of anesthetic induction. Although the use of dexmeditomidine has been described for awake craniotomies during the initial phase of sleep-awake-sleep cycle, its use in other forms of neurosurgery has been sparingly described.

In this case series we explored the feasibility of combining dexmedetomidine with sevoflurane in an anesthetic induction algorithm. All patients achieved smooth anesthetic induction with introduction of sevoflurane after 8 minutes of dexmeditomidine infusion @ 0.6 mcg/kg/min. The whole induction process was carried out in continuous EEG monitoring using the double banana montage in all patients. Quality of anesthetic induction was evaluated by Viby-Mogensen's criteria and the postoperative cognitive parameters evaluated by Montreal Cognitive Assessment (MoCA) score and Confusion Assessment Method - Short form (CAM-S) score ruled out cognitive decline in this present case series.

We conclude that a structured anesthetic induction algorithm incorporating dexmeditomidine with sevoflurane followed by optimal intraoperative hemodynamics and anesthetic maintenance, can enhance patient outcomes and postoperative cognitive function.

Keywords: dexmedetomidine; neurocognition; induction; neurosurgery; electroencephalography

Introduction

Neurosurgery can result in transient or permanent changes in cognition due to a range of factors like the location of the tumor / aneurysm or the type of anesthetics used during the operation.[1] Even surgeries on the spinal cord can indirectly affect cognition.[2] While issues with memory arise after surgeries involving hippocampus and amygdala; the issues pertaining to comprehending language can arise from surgery around left frontal and temporal cortex.[3] Impairment in planning, organization and making decisions can arise from surgeries around pre-frontal cortex.[4] The damage to frontal cortex shall also result in increased impulsivity, mood swings and personality changes due loss of emotional regulation. Nevertheless, the recovery of cognition depends on the volume of brain tissue resected, the location of the lesion, age of the patient and the pre-operative cognitive status. Therefore, postsurgical neurocognitive rehabilitation plays a crucial role.

However, anesthesia has its own paradigm of memory impairment. Anterograde amnesia is associated with induction agents like propofol which acts on hippocampus in direct and indirect manner (by altering the blood flow to the cerebral cortex), thereby preventing formation of new

memories.[5] Alteration in processing of sensory stimuli during anesthetic induction, further alters the patient's perception and awareness to the environmental stimuli. Similarly, during emergence from anesthesia, the pandemonium continues as the subject suddenly transits from the state of unconsciousness to a state where they receive sensory and tactile stimuli all of a sudden, thereby confounding the memory.

An enhancement of inhibitory effects on GABAA receptors and the inhibition of excitatory effects of glutamate by volatile anesthetics and propofol result in alteration of key areas in brain associated with cognition and sensory input.[6] Fentanyl and morphine do not directly affect these key areas, however they alter perception and thereby decrease cognitive function.[7] The demographics of the patient alongwith the co-existing illnesses modulating the anesthetic metabolism, further influence the neurocognitive outcomes. Neurodegenerative diseases pose additional risk for postoperative delirium. Dexmeditomidine affects neurocognition by a different mechanism from that enlisted above. It inhibits norepinephrine release by binding to α_2-adrenergic receptors at brainstem and locus cereuleus, thereby causing sedation, anxiolysis and analgesia at lower doses without causing respiratory depression.[8] Although, higher doses do lead to anterograde amnesia, it still does not induce the level of cognitive suppression to the extent of other intravenous or inhalational anesthetics. This is attributable to its selective and localized action on brainstem, which curtails the disruption of cognition associated cortical structures. The analgesic effects of dexmeditomidine helps in reducing the dependence on opioids, which modulate cognition as per the mechanisms already stated above.[9] It is interesting to note that dexmeditomidine significantly reduces delirium and improves cognitive function after surgery in elderly patients. [10-13] Therefore, the term Enhanced Recovery of Cognitive Function (ERCF) has come into limelight due to this unique neuro-pharmacology of dexmeditomidine.

In this case series we used the integrated dexmeditomidine-sevoflurane algorithm [14] based anesthetic induction with the hypothesis of faster cognitive recovery, better orientation, alertness and attention in the patients. Sevoflurane was used to augment the anesthetic depth with dexmeditomidine for improving the quality of anesthetic induction assessed by modified Viby-Mogensen's score (table 2).[15]

Parameter		Score
Ease of Laryngoscopy (jaw relaxation)	Easy Average Difficult	3 2 1
Vocal cord position	Abducted Intermediate Closed (Adducted)	3 2 1
Vocal cord movements	Absent Moving intermittently Actively closing	3 2 1
Cough (Airway reaction to insertion of ETT)	None Diaphragmatic Sustained (>10 seconds)	3 2 1
Spontaneous limb movements	Absent Slight Vigorous	3 2 1

Table 2: Modified Viby-Mogensen's criteria for assessment of quality of intubation

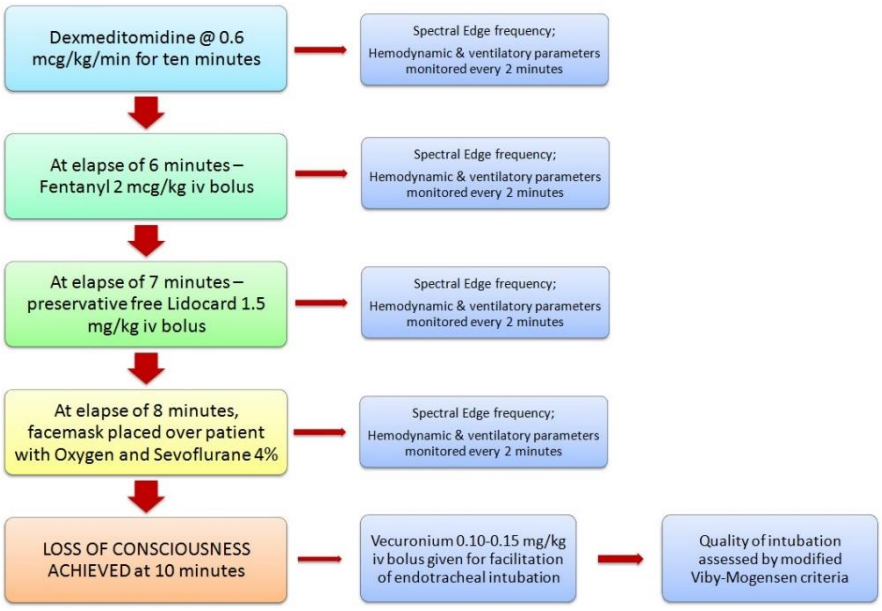

Figure 1: The integrated Dexmeditomidine-Sevoflurane algorithm (Sharma et al.)

Methodology

Our case series meets the CARE (CAse REport) guidelines and encompasses eight patients scheduled for neurosurgery at our institute,

who provided a written informed consent by themselves or next of their kin. 6 of these patients underwent anesthetic induction with integrated dexmeditomidine-sevoflurane algorithm, one patient underwent anesthetic induction with thiopentone, and one patient underwent the anesthetic induction with propofol. After the arrival of patients in the operating room, they laid supine on the operating table. Venous access was secured on dorsum of hand or forearm. Anesthetic induction was initiated with the integrated dexmeditomidine-sevoflurane algorithm.[12] Assessment of quality of intubation done as per modified Viby-Mogensen criteria. Additional data recorded pertained to that collected from the American Society of Anesthesiologists (ASA) standard monitoring; time of emergence from anesthesia; post-operative Montreal Cognitive Assessment (MoCA) score assessed at 3 hours after surgery and Confusion Assessment Method (CAM-S) scores assessed at 6 hours after surgery. The table 1 shows the patient demographics alongwith the collected data.

S. No.	Diagnosis	Surgery	Age (yr)	Sex	Induction anesthetic	Depth of anesthesia Monitoring	Viby Mogensen score	Emergence time (minutes)	MoCA score	CAM-S score
1	Right frontal glioma	Craniotomy and resection	65	M	Dexmeditomidine + Sevoflurane	EEG	15	13 minute 35 seconds	22	3
2	C3-C4 PIVD	ACDF	55	M	Thiopentone	EEG	14	11 min 15 seconds	26	1
3	Burst fracture of L1 vertebra	Decompressive laminectomy and PSRF	57	M	Propofol	EEG	15	12 minutes 24 seconds	25	2
4	Fracture of D12 vertebra	Decompressive laminectomy and PSRF	43	M	Dexmeditomidine + Sevoflurane	EEG	13	7 minutes 52 seconds	28	0
5	Fracture of C4 vertebra	ACDF	56	M	Dexmeditomidine + Sevoflurane	EEG	14	11 minutes and 8 seconds	26	0
6	Fracture of L1 vertebra	*Decompressive laminectomy and PSRF*	32	M	Dexmeditomidine + Sevoflurane	EEG	13	9 minutes 34 seconds	30	0
7	D12-L1 IDEM	*Decompressive laminectomy and gross total resection*	56	M	Dexmeditomidine + Sevoflurane	EEG	14	8 minute 16 seconds	29	1
8	D1 D2 vertebral body fracture	Decompressive laminectomy & resection of lesion	49	M	Dexmeditomidine + Sevoflurane	EEG	14	7 minute 16 seconds	27	1

Table 1. Patient demographics and details

Discussion

The multicentric prospective study by Numan and colleagues,16 aimed at evaluating the discriminative performance of EEG to detect delirium in 159 patients above the age of 60 years. All the patients underwent 5 minute EEG recording followed by video-recorded cognitive assessment during the first 3 days after the surgery. 1minute artifact-free EEG was used to calculate the delta power. Data analysis revealed delirium / possible delirium in 84 (23.3%) postoperative days. The area under the receiver operating characteristic curve (AUROC) of relative EEG delta power came out to be 0.75 [95% confidence interval (CI) 0.69-0.82]. The authors concluded that single-channel EEG recording can be used for detection of delirium. We used multichannel EEG recording as per 10-20 system in our case series followed by assessment of delirium using CAM-S scores.

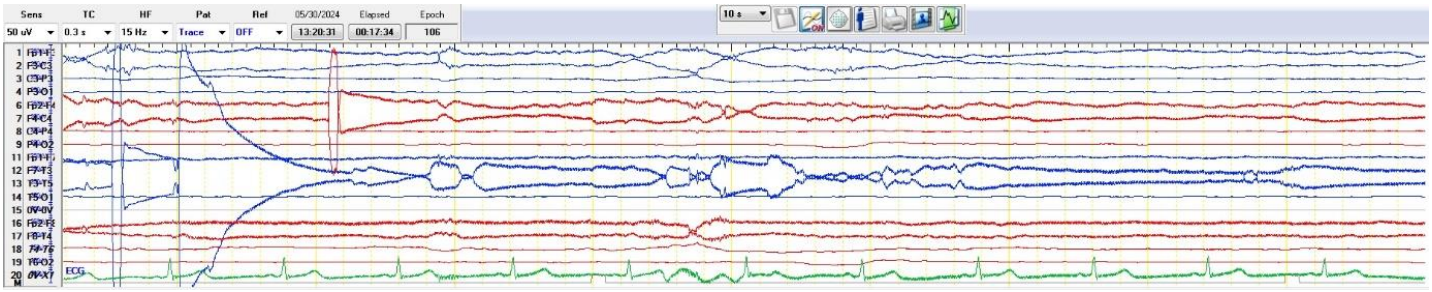

Figure 2: EEG trace of patient 5

In another prospective multicentric observational study on 246 patients by Ditzel and colleagues,17 the researchers used Trail Making Test B [TMT-B] and resting-state functional MRI (rs-fMRI), pre-operative and post-operatively, to study delirium. Data analysis revealed postoperative delirium in 16% of the patients. There was no difference in TMT-B scores in patients with or without delirium. However the postoperative rs-fMRI after 3 months of surgery revealed decreased functional connectivity

strength in patients with postoperative delirium (β= -0.015; 95% CI: -0.028 to 0.002; p = 0.023). This was in contrast to the rs-fMRI findings in the patients without delirium, where they had increased functional connectivity strength after surgery (β = 0.006; 95% CI 0.001-0.011; p = 0.013).

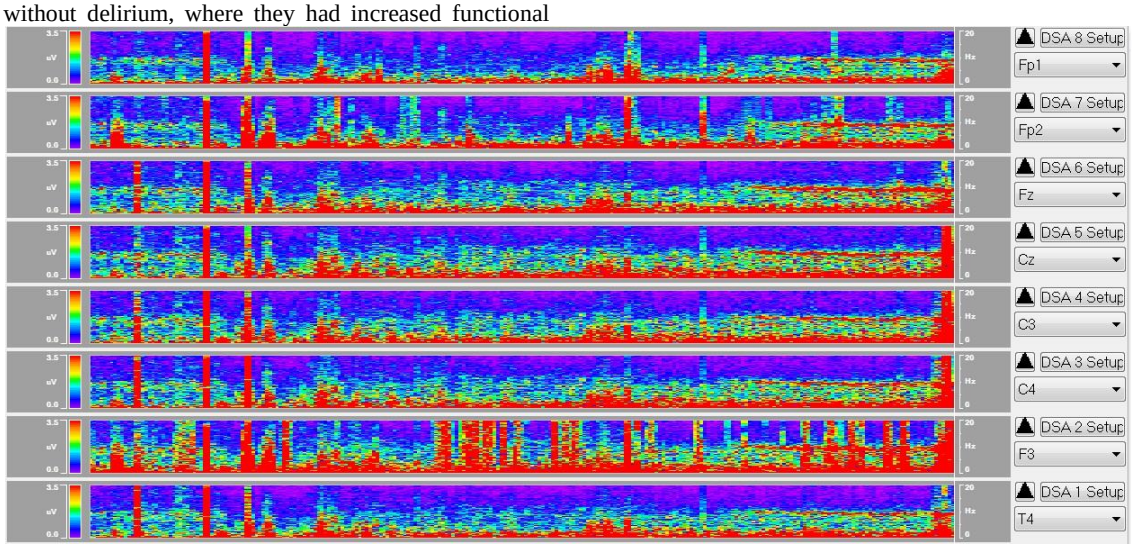

Figure 3: Density spectral array trace of patient 5

In order to test a cognitive disintegration model, Tanabe and colleagues18 recruited 70 patients who underwent cognitive testing, EEG, blood biomarkers and preoperative MRI before and after the surgery. On data analysis adjusted for multiple corrections (MC), the patients with postoperative delirium had impaired structural connectivity (increased radial diffusivity; MC $p < 0.05$) on diffusion tensor imaging. However, the EEG revealed higher alpha power and increased alpha band connectivity (MC $p < 0.05$). A positive correlation of these connectivity effects was observed ($r2 = 0.491$; $p = 0.0012$). The researchers also found that postoperative delirium was associated with breakdown in functional connectivity alongwith increased slow wave activity (SWA) in occipito-parietal and frontal cortex ($r2 = 0.257$; $p < 0.0001$). There fore they concluded that delirium occurs when SWA progresses to involve posterior brain regions, with an associated reduction in functional connectivity. Due to financial constraints we could not perform the MRI in the patients of present case series.

The prospective study by Ballweg and colleagues19 on 114 surgical patients revealed that glial fibrillatory acid protein (GFAP) concentrations showed no relationship to delirium. However change in tau from baseline to post-surgery was positively correlated with the severity of delirium ($r = 0.39$, $p < 0.001$). This change in tau also correlated with elevation in interleukin IL-8 ($p < 0.001$) and IL-10 ($P = 0.0029$). Tau remained significantly associated with severity of delirium even after adjusting age, sex, preoperative cognition, and change in IL-8 ($p = 0.026$). Even linear mixed effect models showed tau as a predictor for recovery from delirium ($p < 0.001$).

One of the features of delirium is post-operative inattention. In 71 patients aged 60 years and above, Acker and colleagues20 recorded awake 32-channel EEG pre-operatively to see the association between alpha attenuation and postoperative inattention. The data analysis revealed that preoperative alpha attenuation with eyes opening was inversely associated with postoperative inattention (odds ratio [OR] 0.73, 95% CI 0.57, 0.94; P=0.038). Alpha-attenuation magnitude showed an inverse relation with the inattention chronicity (OR 0.76, 95% CI 0.62-0.93; P=0.019). Interestingly, due to this feature of inattention, the preoperative alpha-attenuation magnitude was inversely associated with postoperative delirium severity (OR 0.79, 95% CI 0.65, 0.95; p = 0.040), predominantly as a result of the inattention feature. The authors concluded the alpha attenuation in EEG to represent a neural biomarker for risk of postoperative attention impairment.

The meta-analysis by Patel and colleagues21 of 30 trials incorporating 4090 patients undergoing cardiac surgery who received intravenous

dexmedetomidine in perioperative period, having primary outcome as incidence of postoperative delirium revealed that dexmedetomidine was associated with reduction of postoperative delirium in comparison to control group (12.4% vs 16.2%; RR= 0.62; 95% CI 0.44–0.86; p = 0.005). They also observed that dexmedetomidine was not significantly associated with bradycardia or hypotension. Neither did they observe it to be associated with increase in the duration of mechanical ventilation.

Sevoflurane acts by potentiating GABA (gamma-aminobutyric acid) receptor activity and inhibiting NMDA (N-methyl-D-aspartate) receptors. This combination results in sedative, hypnotic, and amnesic effects.22 It is characterized by a low blood-gas partition coefficient of 0.69, allowing for rapid induction and emergence from anesthesia with a minimal risk of airway irritation.23 In brain it inhibits cortical and subcortical processes that are involved in attention, memory and executive function. Thereby reducing the synchronization of neural networks involved in cognition.24 The purported mechanism as per the animal studies is that sevoflurane may increase oxidative stress, which could damage neurons and contribute to long-term cognitive dysfunction. Another mechanism hypothesized is that it can trigger neuroinflammation.25

However, the study conducted by Taylor and colleagues26 incorporating patients aged 65 years or above, undergoing non-intracranial surgery, aimed to observe the impact of sevoflurane on delirium in 118 patients, revealed that the severity of delirium, measured with 3-minute Diagnostic Confusion Assessment Method (3D-CAM) or CAM-ICU, did not correlate with age-adjusted median sevoflurane (AMS) ($r = -0.014$, $p=0.89$) or area under the sevoflurane time-dose curve (AUC-S) ($r = 0.093$, $p=0.35$). Likewise, it was observed that the incidence of delirium did not correlate with sevoflurane (AMS Wilcoxon p=0.86, AUC-S p=0.78). Also, no association was observed for AUC-S in unadjusted (log (IRR) = 0.00, p=0.054) or adjusted models (log (IRR) = 0.00, p=0.832). Therefore the authors concluded that biological mechanisms of delirium, such as inflammation and neuronal injury, appeared more plausible than the dose of sevoflurane as a cause for delirium. Perhaps, it is the surgery itself which acts as a significant contributor for neuroinflammation, leading to post-operative cognitive dysfunction. And this effect gets pronounced in the elderly patients with pre-existing low-grade inflammation. Traditional anesthetic induction agents like propofol and

thiopentone, also compromise regional cerebral oxygenation27 therefore research for better induction agents is the need of the hour.

Conclusion:

Strategies like minimizing the duration of anesthesia, ensuring good postoperative care, avoidance of iatrogenic hypovolemia, ensuring adequate pain management, early mobilization, and cognitive monitoring during surgery can support faster cognitive recovery. Cognitive rehabilitation or stimulation may also exhibit significant impact.

Our case series does not exhibit neurocognitive impairment on usage of dexmeditomidine based anesthetic induction. However translation of this research to routine clinical practice shall require a larger study cohort and adequately powered randomized clinical trial.

Conflicts of Interest: None

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Journal of Anaesthesiology Clinical Pharmacology 41(1):90-97.

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