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Letter to the Editor

Integrated dexmedetomidine-sevoflurane algorithm for anesthetic induction – A viable asset for neurosurgery

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Dear Editor,

As per the existing literature, there is established evidence against the usage of propofol, thiopentone, and etomidate for anesthetic induction in patients with mitochondrial dysfunction.[6] These anesthetics require Gamma-amino-butyric acid A(GABAA) receptor enhancement, which is an energy dependent process, thereby utilizing mitochondria for metabolism and excretion. The current evidence shows that these anesthetic agents impair complex I of oxidative phosphorylation.[1] Propofol additionally also impairs complex III enzymes of this cycle,[9] which eventually leads to metabolic acidosis, arrhythmias, and neurotoxicity. There is recent emerging evidence regarding dexmedetomidine exerting a protective effect on enzymes of oxidative phosphorylation [11], along with added effects of neuronal preservation, anti-inflammation, and reduction in lipid peroxidation.[10] There is also evidence that the anti-inflammatory effect of dexmedetomidine is almost equal to that of methylprednisolone.[2] Dexmedetomidine also reduces tissue edema, inflammation, and apoptosis.[5] In the case of sevoflurane, the separation of mitochondria from anesthetic targets is not well defined.[3] Furthermore, it does not require metabolism for excretion. Therefore, it is exhaled, and this gives it a vantage point in our algorithm. We report the successful use of this integrated dexmedetomidine-sevoflurane algorithm [Figure 1] for anesthetic induction in spine surgeries under electroencephalography (EEG) guidance in three patients undergoing spine surgeries at our institute [Table 1]. The loading dose of intravenous infusion of dexmedetomidine for asleep-awake-asleep craniotomy procedures is 1 μg/kg/h. To attain the loss of consciousness within 10 min, this dose was calculated to be 0.6 μg/kg/min for 10 min. After elapse of 6 min, we gave fentanyl 2 μg/kg intravenously. At an elapse of 8 min, we applied the facemask on the patient and deepened the anesthetic plane further by administering sevoflurane 4%. The loss of consciousness was attained at 10 min, and to facilitate endotracheal intubation, we administered 0.15 mg/kg vecuronium intravenously. The intubation was performed in the first attempt without any adverse event. The quality of intubation was evaluated as per the modified Viby-Mogensen criteria[12] [Table 2], and the score came out to be 14.33 ± 0.471. The EEG trace was similar in all three patients during the anesthetic induction and showed a train track pattern [Figure 2]. The mean time of emergence from anesthesia was 7.37 ± 0.628 (mean ± standard deviation) min. In the dose finding trial conducted by Mu *et al.*[7] for effective induction of anesthesia using only dexmedetomidine as a sole agent, the authors used Dixon's up-and-down sequential method to determine the dose to achieve a loss of consciousness. Minimum effective dose $(ED)_{50}$ and ED_{95} of initial infusion

Table 1: Patient details.

S. No.	Age	Sex	Diagnosis	Surgery	Viby-Mogensen score	Emergence time (min)
1.	44	F	Burst compression fracture of D12 vertebra	Decompressive laminectomy and PSRF	14	6.717
2.	42	M	C4 vertebral fracture	ACDF	15	7.167
3.	28	F	D1 level Solitary Plasmacytoma	Decompressive laminectomy	14	8.217

PSRF: Pedicle screw-rod fixation, ACDF: Anterior cervical discectomy and fusion

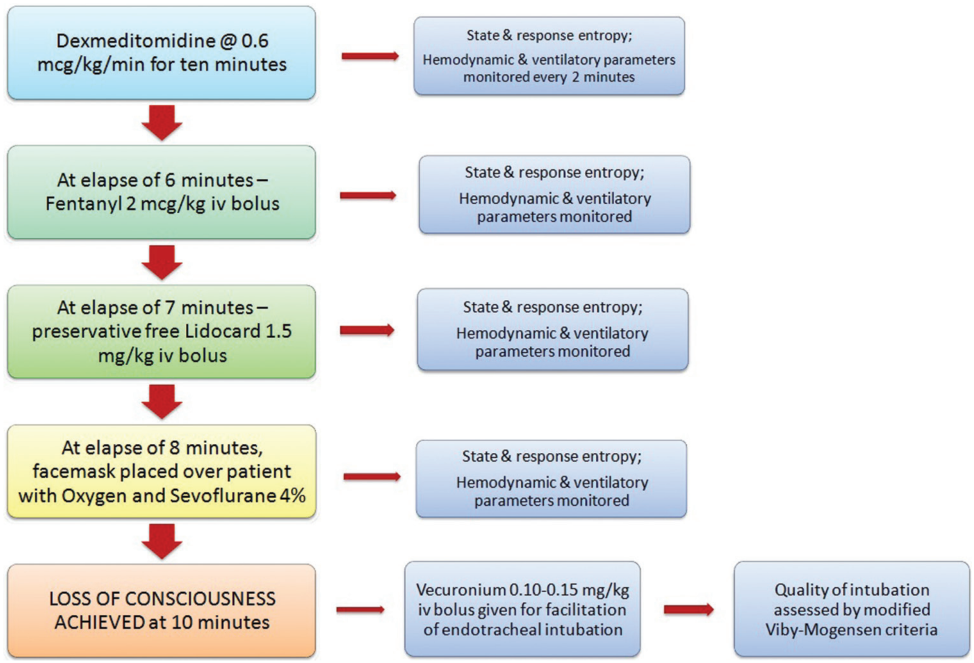

Figure 1: The integrated dexmedetomidine-sevoflurane algorithm.

rate of dexmedetomidine were 0.115 and 0.200 µg/kg/min. The mean induction time was 18.3 min. ED_{50} and ED_{95} of dexmedetomidine to achieve loss of consciousness were 2.899 (95% confidence interval [CI]: 2.703–3.115) and 5.001 (95% CI: 4.544–5.700) µg/kg, respectively. The mean patient state index on the loss of consciousness was 42.8 among the patients. During anesthesia induction, the hemodynamics, including blood pressure and heart rate, were stable, and the EEG monitor showed decreased α and β powers and increased θ and δ in the frontal and pre-frontal cortices of the brain. The authors concluded that continuous infusion of combined dexmedetomidine and remifentanil was an effective strategy for anesthesia induction. We, however, have not used remifentanil infusion, and our algorithm uses primarily dexmedetomidine, followed by a bolus dose of fentanyl and subsequent deepening of the anesthetic plane by sevoflurane. The use of an integrated dexmedetomidine-sevoflurane algorithm resulted in adequate parameters for anesthetic induction achievable in 10 min. Gao *et al.*[4] elucidated the neuroprotective effects of dexmedetomidine in the work on anesthetized rats with the right spinal cord contusion at the C5 level. These subjects presented with locomotor dysfunction. Analysis of collected data revealed that dexmedetomidine significantly decreased the inflammation and induration in these subjects. There was significant improvement in ipsilateral upper-limb motor dysfunction ($P < 0.0001$), decreased injury size ($P < 0.05$), spared white matter ($P < 0.05$), and reduced number of activated macrophages ($P < 0.05$) at the site of injury with the usage of dexmedetomidine. Even the tissue Ribonucleic acid expression exhibited significant down-regulation of pro-inflammatory markers and up-regulation of anti-inflammatory responses ($P < 0.05$). We did not have the facility to observe the advanced inflammatory parameters at our institute; however, the post-operative induration, as judged by clinical evaluation, was minimal. The patient remained afebrile and had an un-eventful course in the hospital until discharge. Ramsay and Luterman.[8] published a case series using dexmedetomidine as total intravenous anesthesia in three patients. However, their patients did not

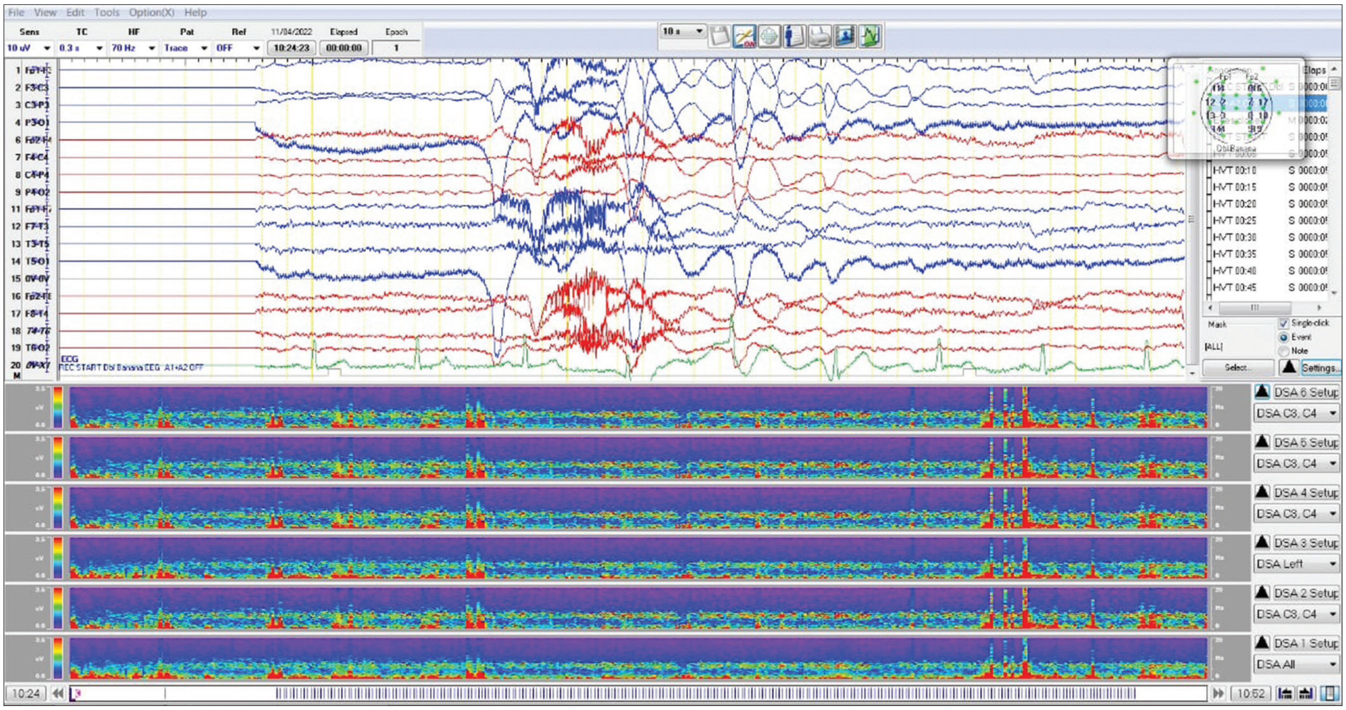

Figure 2: Electroencephalography trace of patient 1 during anesthetic induction.

Table 2: Quality of intubation criteria.[11]

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

ETT: Endotracheal tube

include any neurosurgical patients, and they needed more duration and a higher dose of dexmedetomidine to attain sufficient conditions suitable for anesthesia in their patients. This case series demonstrates that the algorithm achieved optimal quality of intubation in these patients. This algorithm will serve as a viable tool in neuroanesthesia practice, where we can also come across patients with mitochondrial dysfunction.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Acco A, Comar JF, Bracht A. Metabolic effects of propofol in the isolated perfused rat liver. Basic Clin Pharmacol Toxicol 2004;95:166-74.
2. Can M, Gul S, Bektas S, Hanci V, Acikgoz S. Effects of dexmedetomidine or methylprednisolone on inflammatory responses in spinal cord injury. Acta Anaesth Scand 2009;53:1068-72.
3. Driessen J, Willems S, Dercksen S, Giele J, van der Staak F, Smeitink J. Anesthesia-related morbidity and mortality after surgery for muscle biopsy in children with mitochondrial defects. Paediatr Anaesth 2007;17:16-21.
4. Gao J, Sun Z, Xiao Z, Du Q, Niu X, Wang G, *et al*. Dexmedetomidine modulates neuroinflammation and improves outcome via alpha2-adrenergic receptor signaling after rat spinal cord injury. Br J Anaesth 2019;123:827-38.
5. He H, Zhou Y, Zhou Y, Zhuang J, He X, Wang S, *et al*. Dexmedetomidine mitigates microglia-mediated neuroinflammation through upregulation of programmed cell death protein 1 in a rat spinal cord injury model. J Neurotrauma 2018;35:2591-603.
6. Liang Y, Huang Y, Shao R, Xiao F, Lin F, Dai H, *et al*. Propofol produces neurotoxicity by inducing mitochondrial apoptosis. Exp Ther Med 2022;24:630.
7. Mu B, Xu W, Li H, Suo Z, Wang X, Zheng Y, *et al*. Determination of the effective dose of dexmedetomidine to achieve loss of consciousness during anesthesia induction. Front Med 2023;10:1158085.
8. Ramsay MA, Luterman DL. Dexmedetomidine as a total intravenous anesthetic agent. Anesthesiology 2004;101:787-90.
9. Rigoulet M, Devin A, Avéret N, Vandais B, Guérin B. Mechanisms of inhibition and uncoupling of respiration in isolated rat liver mitochondria by the general anesthetic 2,6-diisopropylphenol. Eur J Biochem 1996;241:280-5.
10. Schwarz A, Nossaman B, Carollo D, Ramadhyani U. Dexmedetomidine for neurosurgical procedures. Curr Anesthesiol Rep 2013;3:205-9.
11. Sun L, Niu K, Guo J, Tu J, Ma B, An J. Dexmedetomidine attenuates postoperative spatial memory impairment after surgery by reducing cytochrome C. BMC Anesthesiol 2023;23:85.
12. Viby-Mogensen J, Engbaek J, Eriksson LI, Gramstad L, Jensen E, Jensen FS, *et al*. Good clinical research practice (GCRP) in pharmacodynamic studies of neuromuscular blocking agents. Acta Anaesthesiol Scand 1996;40:59-74.

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