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Case Report

Thiopental-Dexmedetomidine as Adjuvant Anesthesia for Craniotomy Tumor Removal: A Case Report

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Abstract

Brain tumor surgery requires special anesthesia to get a slack brain and perform perioperative brain protection. The selected anesthetic drugs and adjuvants have the ability of anesthesia sparring effect and have a brain protective effect. Not many have done the combination of thiopental adjuvant with dexmedetomidine. The purpose of this case report is to see the effect of the combination of thiopental with dexmedetomidine as an adjuvant anesthesia on hemodynamics and slack brain and successful removal of brain tumors. A woman, 32 years old, with meningiomas had surgery to remove a brain tumor at Santosa Bandung Central Hospital. Preoperative examination showed blood lab results within normal limits, the presence of large meningioma and midline shift. Induction of anesthesia with thiopental 5 mg/kgBW, rocuronium bromide 0.9 mg/kgBW, fentanyl 3 mcg/kg and anesthetic maintenance with sevoflurane below 1.5 MAC, oxygen/air, continuous rocuronium 0.5 mg/kgBW/hour, thiopental and continuous dexmedetomidine. The anesthetic adjuvant used was thiopental 1-3 mg/kg/hour and continuous dexmedetomidine 0.4–0.7 mcg/kg/hour. A slack brain is obtained, and 90% of the tumor could be removed, and transfused during surgery 4 units pack red cells (PRC), crystalloid liquid as much as 2,500 cc, and colloidal fluid as much as 2,000 cc. The length of surgery is 11 hours. Post-surgery was treated in the ICU for 5 days, then moved to the ward for 2 days then the patient could be discharged from the hospital. The use of thiopental and dexmedetomidine continuously can produce slack brain and almost the entire tumor can be removed.

Keywords: Alpha-2 agonist dexmedetomidine, adjuvant anesthesia, brain tumor, sevoflurane, thiopental

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I. Introduction

The basic principle of neuroanesthesia is to provide smooth induction, hemodynamic stability, and provision of optimal operative conditions such as relaxed brain, maintenance of cerebral perfusion pressure, and cerebral oxygenation. One of the peculiarities of neuroanesthesia is smooth emergence which is as much as important as smooth induction to allow early neurological assessment.[1-3] The target of anesthesia for brain tumor surgery, both for the first and redo surgery, is to get a slack brain, prevent a rise in intracranial pressure (ICP), and perform brain protection and resuscitation.[4,5]

Dexmedetomidine, an imidazole compound, is the pharmacologically active dextroisomer of medetomidine that displays selective dose-dependent α2-adrenoceptor agonist. Dexmedetomidine is a highly selective α2-agonist that has been shown to have sedative,

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anxiolytic, analgesic, sympatholytic effects, and anesthetic-sparing effects without significant respiratory depression.[1,6] The potential advantages of neuroprotection, minimal impact on neuronal function, stable hemodynamics, opioid and anesthesia sparing effects, and minimal respiratory depression during awake procedures render it an effective anesthetic adjuvant in various neurosurgical settings.[6] It acts at both spinal and supraspinal sites, modulates the transmission of nociceptive signals in the central nervous system. Even peripheral α2-adrenoreceptors may mediate nociception.[1,6] Numerous studies have shown that dexmedetomidine blunts the stress response, but these studies are mostly done in patients undergoing general or gynecological procedures, studies pertaining to neurosurgical procedures are less.[7-9] In addition, almost all studies have noted the effect of bolus dose of dexmedetomidine along with the infusion of the drug continuing after the bolus dose.[1,10,11,12] Dexmedetomidine has been shown to provide good perioperative haemodynamic stability with decreased intraoperative opioid requirements. It may have neural protective effects, and thus it may be a suitable anaesthetic adjuvant to neurosurgical anaesthesia.[12] Thiopental, a powerful cerebral vasoconstrictor, lowers cerebral metabolic rate for oxygen ($CMRO_2$), cerebral blood flow (CBF), cerebral blood volume (CBV), and ICP, reactivity to CO_2 is maintained, effective anticonvulsant, gamma amino butyric acid (GABA) agonist, free radical cleanser, membrane stabilization, n-methyl d-aspartate (NMDA) antagonist, Ca tunnel blockade, protein synthesis. The primary mechanism for brain protection is a decrease in $CMRO_2$ by 55–60% where electroencephalograh (EEG) becomes isoelectric. A greater decrease in $CMRO_2$ had no protective effect. Thiopental can cause the phenomenon of inverse steal because normal tissue vasoconstriction improves perfusion in ischemic areas that are unable to constrict. Thiopental decreases CBF and $CMRO_2$ paralel to the point of isoelectricity on the EEG. Change in CBF secondary to the change in $CMRO_2$ (a couple decrease in flow and metabolism). Thiopental, even in high dose, does not appear to abolish cerebral autoregulation or CO_2 reactivity.[13] Hence, this study was undertaken to see the effect of dexmedetomidine infusion combined with thiopental infusion on hemodynamics and its ability to act as anesthetic adjuvant in patients undergoing supratentorial tumor surgery.

II. Case

History

A 32-year-old woman came in. She had complaints of headaches that have disappeared since 5 months earlier and worsened one week later and was complaining of a very severe headache since 6 hours before she was admitted to the hospital. Initially, headaches improved with medications such as paracetamol/ibuprofen. Patients had a history of seizures as much as 1x a month. There is no history of visual field disturbances, blurry vision, limb weakness, fever, and trauma. The patient had a CT scan without contrast and was diagnosed a brain tumor in the left parietooccipital suspected meningioma. Currently the patient is on treatment of dexamethasone 3x5 mg and furosemide orally. The patient has never undergone surgery/anesthesia before.

Physical examination

The patient is a woman, aged 32 years, with a body weight of 59 kg, height 148 cm, body mass

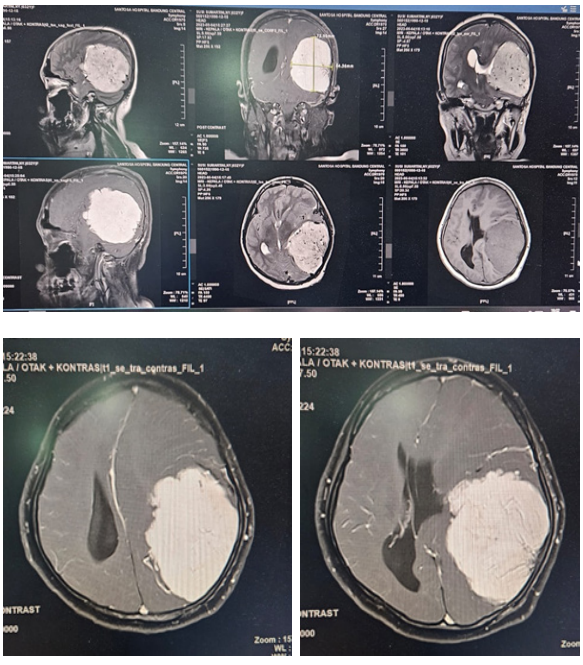

Figure 1: MRI Test Result

Supporting Examination
Blood test
Table 1. Result of Blood test and Reference Range

TEST	RESULT	UNIT	REFERENCE RANGE
HEMATOLOGY			
Hemoglobin	15.0	g/dL	11.7-15.5
Erythrocyte	4.92	Mil/uL	4.10-5.10
Haematocrite	45	%	35-47
MCV	91.5	Fl	80.0-97.0
MCH	30.5	Pg	27.0-31.0
MCHC	33.3	g/dL	32.0-35.0
Leucocyte Count	9.25	Th/uL	36.0-11.00
Thrombocyte	236	Th/uL	150-450
MPV	9.1	fl	7.4-10.4
COAGULATION			
Prothrombin Time	9.9	second	9.5-11.7
Control	10.9	second	
INR	0.97		
APTT	21.22	second	23.0-31.9
Control	23.8	second	
CHEMISTRY			
Blood glucose at Random	112	Mg/dL	55-180
SGOT	24	U/L	<32
SGPT	57	U/L	<31
Ureum	26	Mg/dL	10-50
Creatinin	0.44	Mg/dL	0.51-0.95
Total Calcium (Ca)	9.1	Mg/dL	8.6-10.2
Sodium (Na)	142	Mmol/L	125-153
Potassium (K)	3.9	Mmol/L	3,5-5,3
Chlorida (Cl)	99	Mmol/L	98-109

Hematology test showed blood test within normal limit

index (BMI) 26.93. GCS score of 15 (E4M6V5). Vital signs were measured and the following results were obtained: blood pressure: 110/80 mmHg, pulse rate: 82 x/min, respiratory rate: 20 x/min, temperature: 36.7°C, SpO_2: 94% with room air.

MRI

The results of the MRI examination are shown below:

Large homogeneous hypointense mass, firm border, flat surface with dural tail, size 6.5 x 8.5 x 8.2 cm which in DWI/T2WI appears slight hyperintense in the left cortical temporo-occipital region which causes a shift of midline structure to the right as far as 1.6 cm which appears to urge the left posterior lateral ventricle. The MRI results show other cerebral parenchyma is still good, basal right left ganglia well, parenchyma cerebellum and good brain stem, the ventricular system in the center, symmetrical, does not appear dilated, sulcus corticalis and right left sylvii fissures are good, retroorbita no mass. MRI Conclusion are solid picture of large hypodense measuring about

6.5 x 8.5 x 8.2 cm in the temporo-occipital region, mass image of meningiomas with midline shift to the right currently no perifocal edema around the mass and parenchyma of cerebellum and brain stem is still good.

Anesthetic Management

The patient has been satisfied since 6 hours before surgery. Some treatments were carried out to monitoring of consciousness, vital signs. Informed consent was carried out regarding the patient's condition, also anesthesia plan with general anesthesia, as well as the risks of anesthesia, postoperative care plan in the intensive care room. The patient was positioned supine, oxygen supplementation using a 3 lpm nasal cannula connected to an anesthesia machine. Intravenous access installation with gauge size number G.18 was carried out, given ringerfundin balance solution. Hemodynamic monitoring was carried out using non-invasive blood pressure (NIBP), SpO_2, and ECG monitors. Preoxygenation with 100% oxygen through the facemask was carried out, the patient had a 30-degree head-up position, and asked to do volunteer hyperventilation. Induction was carried out with thiopental 300 mg, fentanyl 150 mcg, rocuronium 60 mg, and lidocaine 60 mg intravenously. Ventilation with 100% O_2 and 1.5 MAC sevoflurane, then intubation with spiral endotracheal tube no. 7, then connected to a ventilator machine with VCV TV mode 420 mL, rate 12 x / minute, PEEP 5, FiO_2 50%. Anesthesia maintenance was performed by administering sevoflurane/oxygen/air and continuous rocuronium 0.5 mg/kgBW/hour.

Anesthetic adjuvants were given continuous thiopental 3-5 mg/kg/hour, and dexmedetomidine 0.2–0.5 mcg/kg/hour. Dexamethasone was given 10 mg intravenously as a continuation of dexamethasone prior to induction of anesthesia. Monitoring was carried out using NIBP, ECG, oxygen saturation, temperature, urine output, amount of bleeding. During surgery, systolic blood pressure ranged from 72–138 mmHg, diastolic 38–102 mmHg, heart rate ranged from 85–140 times per minute, and SpO_2 ranged from 99–100%. Total bleeding of 3,000 cc. Patients were given PRC transfusions as much as 4 units,

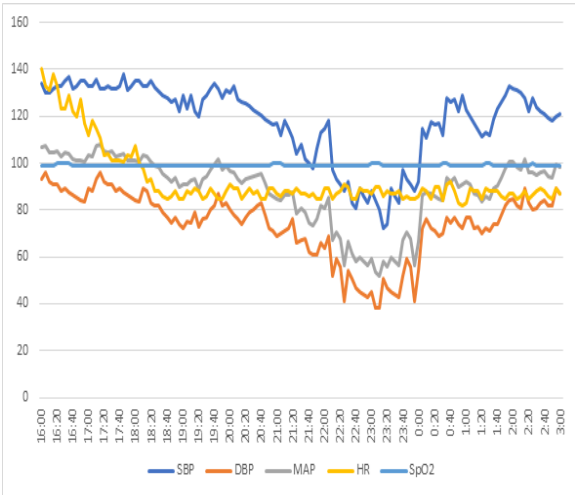

Figure 2. Monitoring Hemodynamic Intra-operative

SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial pressure, HR: heart rate, SpO_2: saturation, pheriperal oxygen

crystalloid liquid as much as 2,500 cc, and colloidal fluid as much as 2,000 cc during surgery. Diuresis volume of 1,500 cc. The operation lasted 11 hours. A slack brain was obtained, and 90% of the tumor could be removed. The amount of bleeding was relatively small compared to the size of the tumor removed and transfused during surgery 4 units pack red cells (PRC).

Postoperative Care at ICU

After surgery, the patient was not extubated and then transferred to the intensive care with blood pressure 98/64 mmHg, heart rate 98 times per minute, breathing rate 18 times per minute with ventilator mode VC-SIMV VT 450, Rate 14, PS 15, PEEP 5, FiO_2 50%, VT 467, MV 5.8, SpO_2 100%, flat abdomen and palpable soft without nasogastric tube, and urination can not yet be assessed. Postoperative hemoglobin 7.7 g/dL, hematocrit 21, leukocytes 9,030/mm3, and platelets 85,000/mm3. Postoperative instructions include adjusting the position of the 30° head-up, observation of consciousness and vital signs, keeping the patient warm, patient continued fasting, and was given administration of maintenance fluid with balance solution crystalloid ringerfundin 100 ml/hour. Patient was given paracetamol 4x1 g intravenously and omeprazole 2x40 mg intravenously. Diuresis observation was carried out with a target of 1–2

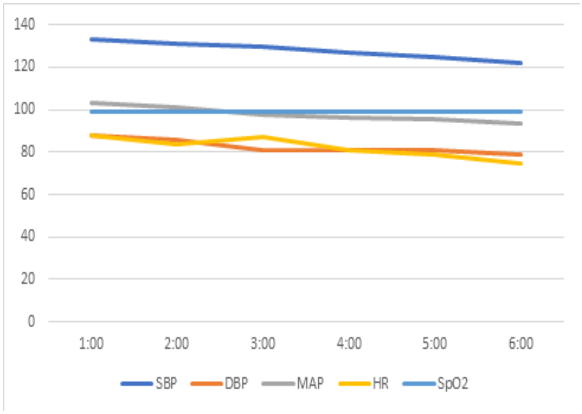

Figure 3. Monitoring Hemodynamic Post-Operative day-1

SBP: systolic blood pressure, DBP: diastolic blood pressure, MAP: mean arterial pressure, HR: heart rate, SpO_2: saturation peripheral oxygen.

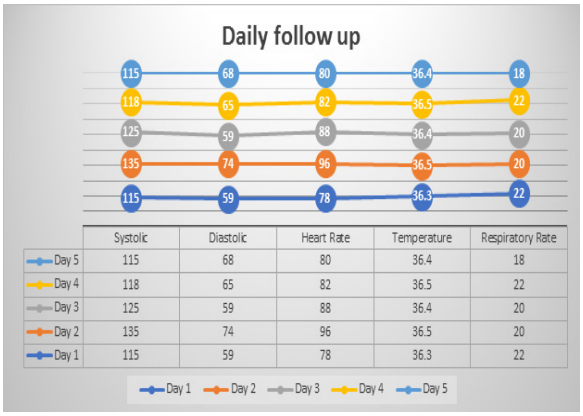

Figure 4. Daily Follow Up at ICU

much as 2 units with a target Hb 10 g / dL. During post-surgery patient was treated in the ICU for 5 days, then moved to the ward for 2 days then the patient could be discharged from the hospital.

Table 2. Daily Follow Up

	Day 1	Day 2	Day 3	Day 4	Day 5
	ICU	ICU	ICU	Ward	Ward
CNS	GCS E4 M5Vt Richmond Agitation Score 0	GCS E4 M6Vt Richmond Agitation Score 0	GCS E4M6V5 Richmond Agitation Score 0	GCS E4M6V5 Richmond Agitation Score 0	GCS E4M6V5 Richmond Agitation Score 0
C	BP: 115/59 mmHg	BP: 135/74 mmHg	BP: 125/59 mmHg	BP: 118/65mmHg	BP: 115/68 mmHg
V	Pulse: 78x/mnt	Pulse: 96x/mnt	Pulse: 88x/mnt	Pulse: 82x/mnt	Pulse:80x/mnt
S	36,3°C	36,5° C	36,4° C	36,5	36,4
Respiration	RR 22 x/mnt on ventilator PSV PS 5 PEEP 5 FiO2 40% Vte 385 MV 10 SpO_2 99%	RR 20 x/mnt on ventilator CPAP PEEP 5 FiO_2 40% Vte 375 MV 9,2 SpO_2 99%	RR 20-22 Simple mask 6 lpm SaO_2 100%	RR 22 Nasal canule 3 lpm SaO_2 100%	RR 18 x/ minute SaO_2 100% room air
GIT	Distention(-)	Distention(-)	Distention(-)	Distention(-)	Distention(-)
Fluids	Balance	+624cc/24 hour	+320cc/24 hour	+625cc/24 hour	
	Urine Output	1,7 cc/kg/hour	1,2 cc/kg/hour	1,94cc/ kg/ 24 hour	

Note: CNS: central nervous system; CVS: cardiovascular system; GIT: gastrointestinal tract

cc/kg BW per hour. Other medications given were ceftriaxone 2x1 g intravenously, tranexamic acid 3x500 mg intravenously, vitamin K 3x10 mg intravenously, dexamethasone 3x8 mg intravenously, mannitol 4x150 cc intravenously, phenytoin 4x100 mg intravenously, and patients was planned to be given PRC transfusions as

II. Discussion

The principle of neuroanesthesia in brain tumor removal is to slack the brain, reduce bleeding and protect the brain. Everything can be done

by performing the ABCDE neuroanesthesia technique. ABCDE is acronym from Airway, Breathing, Circulation, Drugs and Environment (temperature). Regulating $PaCO_2$, PaO_2 and circulation is important to prevent increased cerebral blood flow, increased intracranial pressure.[2,3] Maintaining hemodynamic stability during intracranial surgery is one of the most important tasks because hypertension may lead to hemorrhage and vasogenic edema and a low blood pressure may resulted in cerebral ischemia in areas of impaired autoregulation.[1-3] Moreover, systemic hypertension associated with emergence from anesthesia has long been believed to contribute to intracranial hemorrhage and cerebral edema following craniotomy.[1-3] Hence, one should not only focus on maintaining the stability of vitals intraoperatively but also at the time of emergence. Dexmedetomidine is a potent α2-adrenoceptor agonist with 8 times higher affinity for α2-adrenoceptor than clonidine, superselective α2-adrenergic with a 1600:1 selectivity (α2: α1), sedative, analgesic and anxiolytic effect, no evidence of respiratory depression, anesthesia sparring effect, produced hemodynamic decline decreased arterial blood pressure and heart rate, patients was alerted when stimulated, decrease plasma catecholamines, centrally mediated bradycardic and hypotensive effects, diuresis due to inhibition of anti diuretic hormone (ADH) release and antagonism of ADH tubular effects, decongestant and antisialogogue effects.[14]

Infusion of dexmedetomidine reduces the amount of thiopentone required for induction.[1,15,16] Dexmedetomidine is the drug with opioid-sparing effect, and it has been observed in various studies in humans.[1,14,15,16] Anesthesia sparing effect dexmedetomidine, in addition to reducing the dose of thiopental, dexmedetomidine also reduces the dose of intravenous narcotics. With reduced need for intravenous anesthetic, narcotic analgetic, and sevoflurane inhalation anaesthetics, it will cause cardiovascular stability and if needed the patient can wake up immediately to facilitate postoperative neurological evaluation.[14] Dexmedetomidine may cause hypotension and bradycardia. Thiopental also causes hypotensive effects and a decrease or increase in heart rate.[13] The combination of these two drugs is likely to cause a greater drop in blood pressure and heart rate. With the regulation of the dose, in this case blood pressure and pulse rate remain stable, hypotension and bradycardia do not occur. The use of thiopental adjuvants and dexmedetomidine will cause brain slack, making it easier for surgeons to resection large brain tumors. (Figure 2).

Effect of dexmedetomidine on cerebral blood flow: In animal models dexmedetomidine causes a reduction in cerebral blood flow up to 45%, has no effect on the $CMRO_2$, produces the concentration-dependent constriction of pial arteries and veins, limits hypercapnea-and hypoxia-induced cerebral vasodilation.[1,7-9] In human study (TCD) dexmedetomidine showed mean cerebral blood flow velocity decreased with an increase in plasma concentration of dexmedetomidine, pulsatility index increased at higher level of dexmedetomidine (indicates an increase in CVR).[15,16] During post-surgery, the patient was admitted to the ICU and was given mechanical ventilation due to heavy bleeding and prolonged surgery (11 hours). In handling this case, tiopental and dexmedetomidine adjuvants were used with the results of a slack brain, stable hemodynamics.

III. Conclusion

Thus, it can be concluded that during neurosurgery dexmedetomidine infusion started before surgery maintains hemodynamic stability intraoperatively, reduces the amount of anesthetic drug required for induction, decreases the requirement of analgesic drug, and increases the time to rescue analgesia postoperatively without any residual sedation.

References

1. Batra A, Verma R, Bhatia VK, Chandra G, Bhushan S. Dexmedetomidine as an anesthetic adjuvant in intracranial surgery. Anesth Essays Res 2017;11(2):309-13. Doi: 10.4103/0259-1162.194555

2. Bisri DY, Bisri T. Anestesi Untuk Operasi Tumor Otak: Supratentorial dan Infratentorial. Bandung: Fakultas Kedokteran Universitas Padjadjaran; 2016

3. Gaudet JG, Schoettker P, Bruder NJ. Supratentorial mass: anesthetic consideration. In: Cottrell JE, Patel PM, Soriano SG, eds. Cottrell and Patel's Neuroanesthesia, seventh ed, Philadelphia: Elsevier; 2025, 206

4. Bisri DY, Hallis IK, Saputra TA, Bisri T. Brain relaxation score on craniotomy brain tumor removal with adjuvant thiopental and dexmedetomidine: a case report. J Adv Phar Educ Res 2023;23(3):73-8. Doi: https://doi.org/10.51847/CTKVDUSitR

5. Bisri DY, Septiani GAP, Limawan MA, Bisri T. Anesthesia management of patients with redo craniotomy: cases of supratentorial recidive tumors. Interdisciplinary Social Studies, May 2023;2(8):2215. Doi: https://doi.org/10.55324/iss.v2i8.450

6. Ebert TJ, Hall JE, Barney JA, Uhrich TD, Colinco MD. The effects of increasing plasma concentrations of dexmedetomidine in humans. Anesthesiology 2000;93(2):382–94. Doi: 10.1097/00000542-200008000-00016

7. Soliman RN, Hassan AR, Rashwan AM, Omar AM. Prospective, randomized study to assess the role of dexmedetomidine in patients with supratentorial tumors undergoing craniotomy under general anaesthesia. Middle East J Anaesthesiol 2011;21:325–34.

8. Ilhan O, Koruk S, Serin G, Erkutlu I, Oner U. Dexmedetomidine in the supratentorial craniotomy. Eurasian J Med 2010;42:61–5. Doi: 10.5152/eajm.2010.19

9. Tanskanen PE , Kyttä JV, Randell TT, Aantaa RE. Dexmedetomidine as an anaesthetic adjuvant in patients undergoing intracranial tumour surgery: a double-blind, randomized and placebo-controlled study. Br J Anaesth. 2006; 97(5):658-65. Doi: 10.1093/bja/ael220

10. Basar H, Akpinar S, Doganci N, Buyukkocak U, Kaymak C, Sert O, et al. The effects of preanesthetic, single-dose dexmedetomidine on induction, hemodynamic, and cardiovascular parameters. J Clin Anesth 2008;20:431–6. Doi: 10.1016/j.jclinane.2008.04.007

11. Keniya VM, Ladi S, Nahpadi R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. Indian J Anaesth. 2011;55:352–7. Doi: 10.4103/0019-5049.84846

12. Bekker A, Sturaitis M, Bloom M, Moric M, Golfinos J, Parker E, Babu R, Pitti A. The effect of dexmedetomidine on perioperative hemodynamics in patients undergoing craniotomy. Anesth Analg.2008;107(4):1340-7. Doi: 10.1213/ane.0b013e3181804298

13. Matsumoto M, Yamashita A. Effects of anesthetic agents and other drugs on cerebral blood flow, metabolism, and intracranial pressure. In: Cottrell JE, Patel PM, Soriano SG, eds. Cottrell and Patel's Neuroanesthesia, seventh ed, Philadelphia: Elsevier; 2025,

14. Tasbihguo SR, Barends CRM, Absalom AR. The role of dexmedetomidine in neurosurgery. Best Practice & Research Clinical Anesthesiology 2021;35(2):221-29

15. Lin N, Vuskits L, Bebawy JF, Gelb A. Perspective on dexmedetomidine use for neurosurgical patient. Journal of Neurosurgical Anesthesiology 2019;31(4):366-77

16. Peng K, Wu S, Liu H, Ji F. Dexmedetomidine as an anesthetic adjuvant for intracranial procedure: metaanalysis of randomized control trial. Journal of clinical neuroscience 2014;21: 1951–58