

Brain relaxation score on craniotomy brain tumour removal with adjuvant thiopental and dexmedetomidine: A case report

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ABSTRACT

The term ‘brain relaxation’ is frequently used to describe the tension of brain tissue during craniotomy. The relaxation status of brain is an essential element of neuroanesthesia practice and is relevant to surgical condition, retractive injuries, and patient outcomes. The beneficial and detrimental effects of various interventions for brain relaxation should be considered in patient management.

A 63-year-old woman with a supratentorial tumor who weighed 70 kg and was 164 cm tall complained of a decrease in consciousness three days before hospital admission, and partial left-body paralysis for the last two months. The patient’s Glasgow Coma Scale (GCS) score was 12, feeling headache, nausea, vomiting, but not seizures. Blood pressure was 146/79 mmHg, mean arterial pressure (MAP) was 99 mmHg, pulse rate was 87 x/min, respiratory rate was 20 x/min, temperature was 36.9°C, and SpO₂ was 97% with air. She was induced with dexmedetomidine, followed by thiopental, vecuronium bromide, fentanyl, lidocaine, ventilation with 100% oxygen, and sevoflurane 2 vol%. Before intubation, a repeat dose of thiopental was given at half the initial dose. Dexamethasone and mannitol were administered, thiopental was continued, and the dose was then lowered to 1-3 mg/kg BW, sevoflurane 1-2 vol%, oxygen/air, and continuous vecuronium bromide were also given. A slack brain with a brain relaxation score of 1 was obtained. The operation lasted 5.5 hours. Using a drug combination from the neuroanesthesia drug club, a slack brain was achieved, allowing the tumor to be entirely removed, with minimal bleeding and without the need for blood transfusions.

Keywords: Adjuvant anesthesia, Brain tumor, Brain relaxation score, Craniotomy, Dexmedetomidine, Thiopental

Introduction

During craniotomy, the term “brain relaxation, is routinely use to describe the size and tightness of brain tissue. The relaxation status of the brain is an important aspect of neuro anesthesia practice and is relevant to surgical condition, retractive injuries and possible patients ourcomes [1, 2]. The relationship between the volume of intracranial content and the capacity of the intracranial space (i.e. the content-space relationship)

determines brain relaxation [3]. This is a concept related to intracranial pressure, but distinct from it. The beneficial and unfavorable effect of various interventions for brain relaxation should be taken into account in the treatment of patients [4, 5]. Brain swelling is common during craniotomy for brain tumors. Poor brain relaxation may increase the risk of cerebral ischemia, possibly worsening the outcome. The surgical team must identify risk factor that may cause perioperative brain swelling and decide which therapies are indicated to correct it [5, 6].

Various methods are employed to obtain a slack brain, including patient positioning, a clear airway, hyperventilation or hypocapnia, normotensive, normovolemia, isoosmolar or slight hyperosmolar, and drug administration. Osmotic diuresis with mannitol, NaCl hypertonic, sodium lactate hypertonic, or dexamethasone can induce pharmacological brain relaxation [6-10].

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Brain relaxation can be measured by calculating the brain relaxation score. Upon the opening of dura, the level of brain relaxation was rated on four-point scale (brain relaxation score: 1 completely relaxed; 2, sufficiently relaxed; 3, firm brain; 4, bulging brain) [6].

The adjuvant drugs used are an old drug, thiopental, and a relatively new drug, dexmedetomidine, an alpha-2 agonist. Both drugs, including those in the neuro anesthesia drugs club, have a positive and protective effect on the brain. A clear airway, hyperventilation, and the administration of mannitol, as well as the drug's effect, will increase the development of a slack brain. The effect of Thiopental are lowering the cerebral metabolic rate of oxygen (CMRO) and an improvement in the distribution of cerebral blood flow, suppressing seizures, suppressing catecholamines that cause hyperactivity, anesthesia, immobilization, and loss of thermoregulation, blocking Na channels, decreasing intracranial pressure, decreasing cerebrospinal fluid secretion, scavenging free radicals, membrane stabilization, and calcium channel blockage. Dexmedetomidine's effect is the anesthesia sparing effect, which has sedative, analgesic, blunt laryngoscopy-intubation, and brain-protective properties. Neuroprotection may be associated with the inhibition of ischemia-induced norepinephrine release; dexmedetomidine prevents delayed neuronal death after focal ischemia; dexmedetomidine decreases total ischemic volume by 40% compared to placebo; and dexmedetomidine enhances glutamine disposal by oxidative metabolism in astrocytes [6, 8]. This report examines the potential of thiopental and dexmedetomidine as adjuvant anesthesia to induce brain slack, as measured by the brain relaxation score.

Case

History

A 63-year-old female patient with a supratentorial tumor had decreased consciousness three days before hospital admission, accompanied by partial left-body paralysis for the past two months. Previously, patients complained of persistent and aggravating headaches that did not improve with anti-pain medication. Vomiting and seizure complaints were denied. A previous history of hypertension was identified. Diabetes history was denied. Despite the denial of a history of trauma, the left hand and foot were immovable.

Physical examination

The patient GCS was 12. She complaints were headaches, nausea, vomiting, blurred vision, but not seizures. Her Blood Pressure was at 146/79 mmHg, mean arterial pressure (MAP) was 99 mmHg, pulse rate was 87 beats per minute, respiration rate was 20 beats per minute, her temperature was 36.90 °C, SpO₂ was 97% with room air, body weight was 70 kg, and height was 164 cm.

Supporting examination blood lab examination

Table 1. Blood test result

Test	Result	Unit	References Range
HEMATOLOGY			
Hemoglobin	13.0	g/dL	11.7-15.5
Erythrocyte	5.56	mil/u	4.10-5.10
Hematocrit	41	%	35-47
Blood index			
MCV	74.5	fl	80.0-97.0
MCH	23.4	Pg	27.0-31.0
MCHC	31.4	g/dL	32.0-35.0
Leucocyte Count	7.03	Th/uL	3.60-11.00
Thrombocyte	197	Th/uL	150-450
MPV	10.2	fl	7.4-10.4
COAGULATION			
Bleeding time	1.30	Minute	1-3
Clotting time	9.00	Minute	5-15
CHEMISTRY			
Blood glucose at random	99	mg/dL	55-180
SGOT (AST)	27	U/L	<32
SGPT (ALT)	15	U/L	<31
Ureum	17	mg/dL	10-50
Creatinin	0.37	mg/dL	0.51-0.95
Total Calcium (Ca)	8.7	mg/dL	8.6-10.2
Sodium (Na)	135	mmol/L	135-153
Potassium (K)	3.1	mmol/L	3.5-5.3
Chloride (Cl)	102	mmol/L	98-109

Correction with KCl 25 meq 1 cycle: the result was Na 135 mMol/L, K 4 mMol/L, Ca 9 mg/dL

MRI head with contrast examination: Meningioma

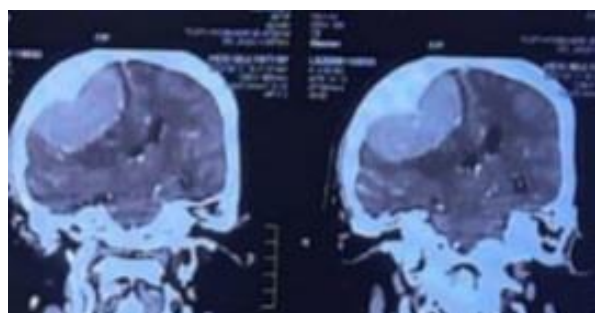


Figure 1. In the right parietal appears homogeneous lesion of extraxial isointens at T1, mild hyperintens at T2 and T2-Flair which is very strong after contrast administration with oval-round shape; which is accompanied by tail in anteriorly and posteriorly with size 6.0 x 5.6 x 5.0 cm accompanied by perifocal edema that constricts sulcy and gyri and obliterates the right and III lateral ventricles and a midline structure deviation of 1.2 cm to the left.

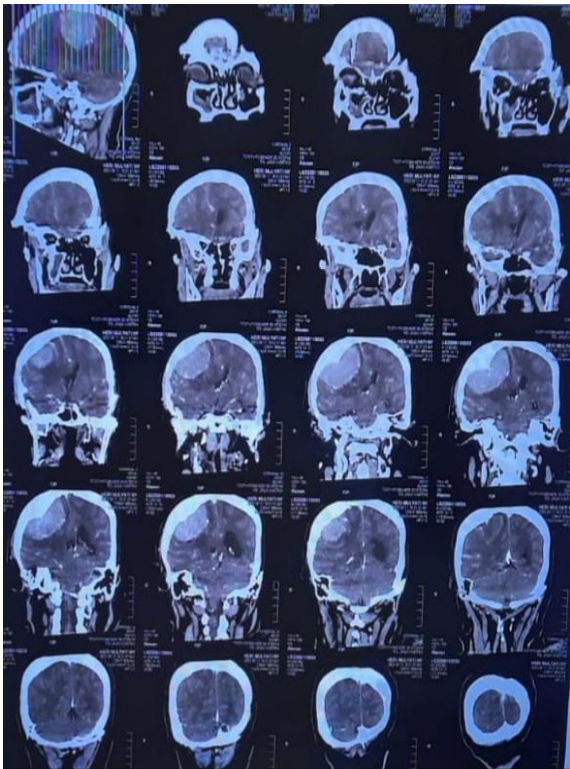


Figure 2. Appears extraxial isointens homogeneous at T1, mild hyperintens at T2 and T2-Flair in the right parietal very strong after contrast with oval-round shape; which is accompanied by tail anteriorly and posteriorly with a size of 6.0 × 5.6 x 5.0 cm accompanied by perifocal edema that constricts sulcy and gyri and right lateral ventricle and III and appears midline structure deviation of 1.2 cm to the left.

intramass appears central necrotic. Dining irregular mass slightly lobulated. Other ventricular systems show dilated and bilateral asymmetry, but the IV ventricular mash is good. Visible dilated left lateral ventricular temporal corn.

Anesthesia management

Patients were induced with dexmedetomidine 1 mcg/kg body weight for the next 15 minutes and then 0.6-0.8 mcg/kg body weight/hour, and thiopental 200 mg was injected. A relaxant with vecuronium bromide 8 mg, analgesic fentanyl 150 mcg, ventilation with 100% oxygen, and sevoflurane 2 vol% were given. Ninety seconds before the laryngoscopy intubation, lidocaine 70 mg was injected intravenously, and 30 seconds before the intubation, a repeat of thiopental at half the initial dose was given. She was administered dexamethasone 10 mg, mannitol 500 mg/kg BW, continuous thiopental 5 mg/kg BW then lowered to 1-3 mg/kg BW, sevoflurane 1-2 vol%, oxygen/air, continuous vecuronium bromide 0.1 mg/kg BW/hour. Ventilation with a tidal volume of 8 mL/kg BW, 14 times per minute, PEEP 5. A slack brain was obtained with a brain relaxation score of 1. The operation lasted 5.5 hours, and the amount of bleeding was 500 cc. The volume of ringerfundin crystalloid liquid was 2500 mL, and the urine discharge was 1500 cc. The patient was transferred to the ICU, where she received Ventilator.

Intra-operative monitorin

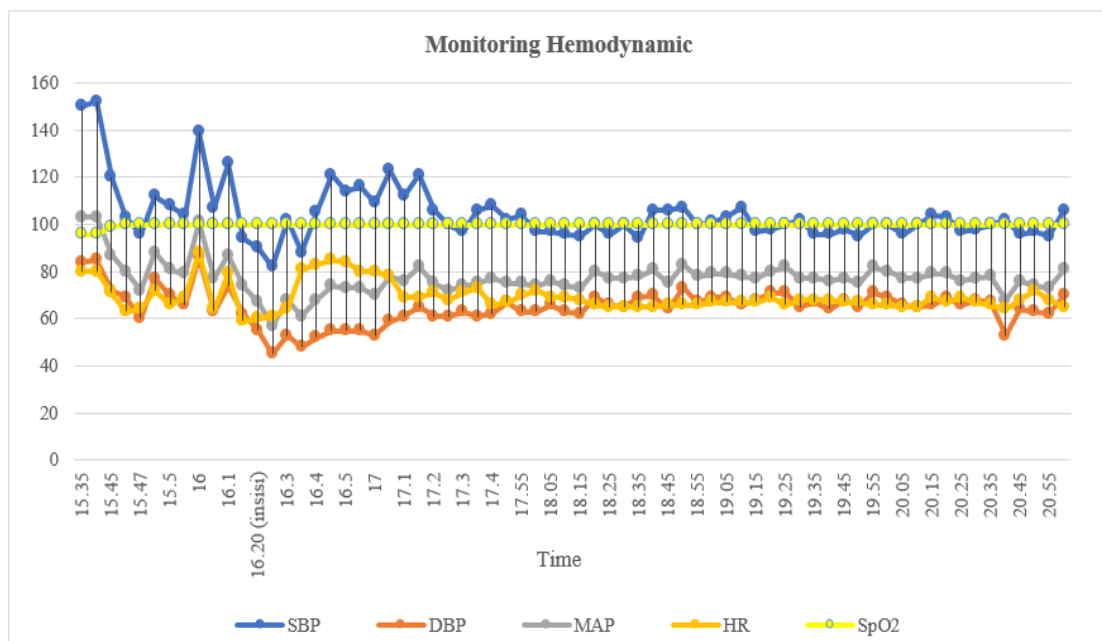
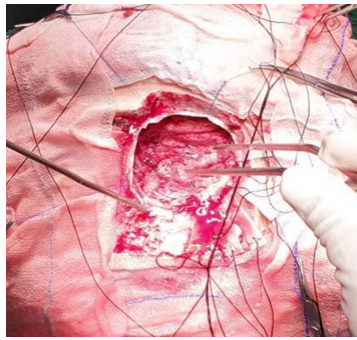
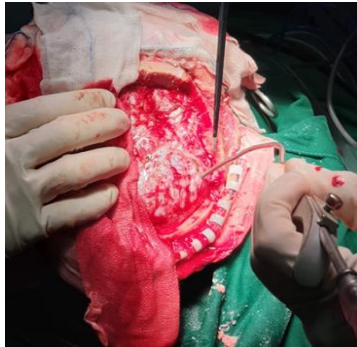


Figure 3. Intraoperative Monitoring Hemodynamic

Note: SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; SpO₂ = saturation peripheral oxygen



a)



b)

Figure 4. This picture illustrates slack brain condition



Figure 5. Tumor mass: the tumor was 100% removed.

Postoperative at ICU

Day 1

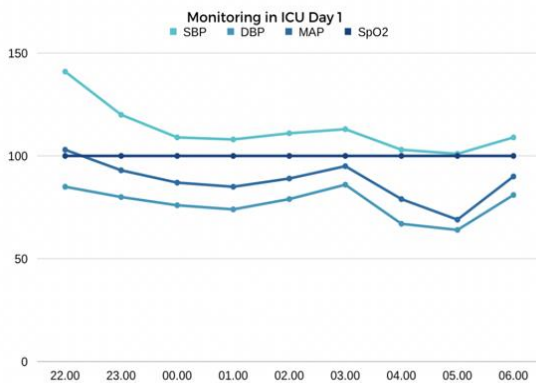


Figure 6. Postoperative Monitoring Hemodynamic at ICU
 Note: SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; SpO₂ = saturation peripheral oxygen

Table 2. Postoperative lab test

Hemoglobin	13,4 g/dl
Haematocrit	42 %
Leucocyte	7.340 uL
Thrombocyte	205.000/ uL
Ureum	19 mg/ dl
Creatinin	0,42 mg/dl
Natrium	134 mEq/ L
Kalium	4,2 mEq/ L
Chloride	105 mEq/ L
Magnesium	2,24 mEq/ L
Calcium ion	1,31 mEq/ L

In the ICU, the patient was positioned head-up at 30 degrees, neutral. The drug given in the ICU was a thiopental sedative of 0.5 mg/kg/hour because the patient was still under ventilator and intravenous fluid maintenance with a balanced solution of ringerfundin (30 mL/kgBW/24 hours). Postoperative Hb was 13.4 g/dL, so no blood transfusion was required.

Results and Discussion

Experienced neuro anesthesiologists and surgeons regularly integrate the concept of ‘brain relaxation’ into perioperative communication. After the opening of the skull and dura, surgeons frequently assess how tight or relaxed the brain is. If the brain is tight or enlarges, various maneuvers are performed to soften in to enhance operating conditions and to protect the brain from retraction injuries and ischemia caused by compression [4, 8-10].

Brain relaxation is a term specifically used to describe the brain condition during craniotomy; however, the concept of brain relaxation has expanded over time to integrate a variety of terms. Additionally, the term, such as, brain relaxation, brain bulging, and brain herniation have been used. The term brain relaxation should be used to describe clinical entities that have anesthetic, surgical, physiological and outcome implication. When the skull and dura are opened by the neurosurgeon, brain relaxation defines the relationship between the volume of intracranial contents and the capacity of the intracranial space. Therefore, it is a concept that must be applied specifically during intracranial procedures when direct assessment of content-space relationships is possible. If the volume of intracranial content is equal to or less than the capacity of the intracranial space, then relaxation of the brain is adequate. However, if the volume of cerebral content in connection with the capacity of the intracranial space that gives the best operating circumstances for open intracranial surgery and patient outcomes should be described as the current notion of brain relaxation. Relaxation of the brain is sufficient if the volume of intracranial content is equivalent to or smaller than the capacity of the intracranial space. The modern concept of brain relaxation should be defined as the ideal volume of intracranial contents in conjunction with the capacity of the intracranial space

that provides optimal operating conditions during open intracranial surgery and patient outcomes [4, 8, 9].

It is important to distinguish the concept of brain relaxation from the practice of measuring intracranial pressure (ICP). Intracranial pressure is pressure with a closed skull, that is measured objectively by a transducer. Brain relaxation, by contrast, is a more subjective assessment by surgeons primarily based on intracranial content-space relationship when the skull and dura are opened. Intracranial pressure decrease to the atmospheric level (referred to as zero) when the dura is opened, but the degree of relaxation of the brain may not change, or may worsen if brain tissue suddenly expands. ICP physically results from the pressure that intracranial contents place on the skull's walls. It makes sense that the ICP would be higher and vice versa, depending on how much cerebral space was occupied by intracranial substances. Research demonstrating a link between raised ICP and a greater frequency of clinical brain edema following dural opening supports this connection. Physically, ICP is the result of the force exerted by intracranial content on walls of cranium. Intuitively, the greater the volume of intracranial space, the higher the ICP, and vice versa. This association is confirmed by research showing that elevated ICP correlates with a higher incidence of clinical brain swelling after dural opening [4, 8, 9].

The Chemical Brain Retractor concept for obtaining a slack brain is:

- Mild hyperosmolality (use NaCl 0.9% [304 mOsm/kg] as baseline infusion; give 20% mannitol [1245mOsm/kg] 0.5 to 0.75g/kg or hypertonic saline [7.5%, 2498mOsm/kg] 2 to 4mL/kg before bone flap removal)
- Intravenous anesthetic agent (propofol), adequate depth of anesthesia
- Mild hyperventilation, mild hyperoxygenation
- Mild controlled hypertension: mean arterial blood pressure maintained around 100 mmHg to decrease cerebral blood volume and intracranial pressure
- Normovolemia; no vasodilators
- Mild hyperoxia

Together with:

- Head-up positioning with unimpeded cerebral venous drainage; no compression of the jugular veins
- Minimal positive end-expiratory pressure
- Adequate anesthetic depth or muscle relaxant to prevent bucking on the ventilator
- Lumbar drainage
- Avoidance of brain retractors

Picture. The Chemical Brain Retractor concept [6].

Propofol-based intravenous anesthesia has been used with positive results [8]. As well as the combination of the inhalation anesthetic sevoflurane with propofol [5]. The use pharmacological brain retraction, such as perioperative dexamethasone, was associated with improved brain relaxation,

however, the presence of a preoperative middle shift and a higher haemoglobin level were associated with poor brain relaxation [6].

In this case, thiopental, fentanyl, sevoflurane, vecuronium, mannitol, and dexamethasone were used as anesthetic drugs and adjuvants. The effects of anesthetics on Cerebral Blood Flow (CBF), cerebral metabolic rate (CMRO₂), and intracranial pressure (ICP) are illustrated in the table below.

Table 3. The effects of anesthetics on Cerebral Blood Flow (CBF), Cerebral Metabolic Rate (CMRO₂), and Intracranial Pressure (ICP) [7-12].

Drugs used in this patients	CBF	CMRO ₂	ICP
Thiopentone	Decrease	Decrease	Decrease
Fentanyl	0/Decrease	0/Decrease	0/Decrease
Vecuronium	0	0	0/Decrease
Lidocaine	Decrease	Decrease	Decrease
Sevoflurane	Increase	Decrease	Increase
Mannitol	0/increase	-	Decrease
Dexamethasone	0	-	Decrease
Dexmedetomidine	Decrease	-	Decrease

Note: CBF = cerebral blood flow, CMRO₂ = cerebral metabolic rate for oxygen, ICP = intracranial pressure

In this case, a dose of 0.5 g/kg BW of mannitol as a standard osmotic diuretic was given within 20 minutes.

The incidence of poor intraoperative brain relaxation, as is manifested by brain oedema (the brain swelling). A spectrum review of elective neurosurgery patients found an incidence of 0.7% to 6.1% of severe brain swelling. Mild and moderate brain enlargement is relatively common, occurring in nearly 30% of patients undergoing supratentorial brain tumor resection [4]. In this case, although the brain tumor was large, 100% of the tumor could be removed because the brain slack was achieved.

The main cause of tight brain is the excessive volume of intracranial contents. Various brain lesions, such as tumor, cyst, hematomas, and traumatic injuries, together with cerebral oedema, enlarge the volume of intracranial contents without changing the capacity of the intracranial space, leading to a space-content mismatch. Other factors that contribute to brain tightness include excessive cerebrospinal fluid (CSF, e.g., hydrocephalus), increased cerebral blood volume (CBV). Elevated CBV may result from increased CBF (e.g., hypercapnia, high minimum alveolar concentration (MAC) of volatile anesthetic agents) or reduced cerebral venous drainage (e.g., headward tilt, extreme head/neck rotation, neck compression) [4, 7, 9, 12, 13]. In this case, the problems of CBF, CBV, patient position, fluid intake, and anesthetic effects on cerebral dynamics were calculated.

Conclusion

Brain relaxation during a surgical removal of brain tumor plays an important role in the success of procedure. The combination of sevoflurane < 1.5 MAC, oxygen/air,

dexmedetomidine, and the thiopental adjuvant produced a slack brain with a brain relaxation score of 1 so that brain tumor removal could be performed in a relatively short time. 100% of the tumor was removed and the amount of bleeding was so minimum that it did not require a blood transfusion.

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Ethics statement: None

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