

Anesthesia in Epileptic Patient Undergoing Temporal Lobectomy and Amygdalohippocampectomy with Electrocorticography: A Case Report

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Abstract

Introduction: Epilepsy is a chronic neurological disorder characterized by recurrent focal or generalized seizures. Despite the availability of various anti-seizure medications (ASMs), one-third of patients develop drug-resistant epilepsy, necessitating epilepsy surgery. In epileptogenic focus resection procedures, cortical mapping and intraoperative electrocorticography (ECoG) are frequently utilized to guide resection. Therefore, anesthetic depth, techniques, and agents selection must minimize cortical activity suppression and avoid interference with ECoG interpretation.

Case: A 30-year-old male with drug-resistant temporal lobe epilepsy was diagnosed with an epileptogenic zone in the right anterior temporal lobe, based on long-term electroencephalography (EEG) and magnetic resonance imaging (MRI). The patient underwent anterior temporal lobectomy with amygdalohippocampectomy under intraoperative ECoG guidance to determine resection extent. Classified as ASA III, the patient received general anesthesia with total intravenous anesthesia (TIVA) using propofol, remifentanyl, and rocuronium, with bispectral index (BIS) monitoring. Intraoperative hemodynamics remained stable, ECoG recordings were clearly interpretable, allowing accurate delineation of the epileptogenic zone. The patient was extubated successfully, and no postoperative seizures were observed.

Discussion: In epilepsy surgery involving ECoG monitoring, selection of anesthetic techniques and agents warrants careful judgement to avoid interference with intraoperative ECoG readings. Furthermore, it is equally important to consider the effects of anesthetic agents on epilepsy, brain protection, hemodynamic stability, and intracranial pressure management, while ensuring rapid postoperative recovery for early neurological assessment.

Conclusion: TIVA with adequate depth of anesthesia monitoring using BIS is a safe approach for epilepsy surgery with ECoG monitoring and is beneficial for early neurological function evaluation.

Keywords: Electrocorticography (ECoG), epilepsy surgery, general anesthesia, total intravenous anesthesia

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Introduction

Epilepsy is a chronic neurological disorder characterized by recurrent focal or generalized seizures. Although various anti-seizure medications (ASMs) are available, 30–40% of patients exhibit resistance, thereby necessitating non-pharmacological treatment options such as epilepsy surgery.^{1,2} Epilepsy surgery has become

the mainstay of treatment for patients with refractory or drug-resistant epilepsy, defined as resistance to treatment with two appropriately chosen and tolerated ASMs. However, only about 30.7% of adults with focal drug-resistant epilepsy and 15.5% of children with drug-resistant epilepsy are considered eligible for epilepsy surgery.³ In Indonesia, epilepsy surgery was first performed in July 1999, and the number of cases continuing to increase annually.⁴ Epilepsy surgery generally

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involves the resection of the epileptogenic focus to achieve seizure cessation or reduce seizure frequency. Although alternative methods such as vagal nerve stimulation (VNS), thalamic deep brain stimulation (DBS), and responsive neurostimulation (RNS) are increasingly being utilized, temporal lobectomy via craniotomy remains the most commonly performed surgical technique, particularly in patients with mesial temporal lobe epilepsy, due to its superior seizure-free outcomes.⁵ Anesthetic management plays a critical role in epilepsy surgery, as anesthetic agents may suppress cortical activity, directly affecting the accuracy of intraoperative epileptogenic zone (EZ) mapping. Electrocorticography (ECoG) is a neurophysiological technique used to monitor electrical activity in the cerebral cortex, enables direct, real-time cortical electrical recording during surgery, supporting precise EZ identification and functional mapping. Therefore, the anesthesiologist must have a thorough understanding of the available anesthetic agents, including their proconvulsant and anticonvulsant properties as well as their potential interactions with ASMs.^{5,6}

Case

Anamnesis/disease history

A 30-year-old male patient presented with a 19-year history of recurrent seizures. The seizures usually started with an uncomfortable sensation and nausea, followed by head movements and eye turning to the right (non-verse) which is then followed by verse head and eye turning to the left and tonic movements in all four extremities with an approximate duration of 1-2 minutes. After the seizure ceased, the patient looked confused or slept for 10-20 minutes. Once fully awake, he complained of a throbbing headache that resolved spontaneously within three hours. Seizure frequency ranged from three to ten episodes per week. The patient sought medical treatment and had undergone treatment trials with multiple ASMs, such as valproic acid, phenytoin, carbamazepine, and levetiracetam. Within the 19-year history of ASM treatment, the patient experienced a 2-year period of seizure freedom. However, the seizures returned after

ASM discontinuation, and since then, the patient has not experienced a period of seizure freedom, even with the optimal ASM dose. His most recent seizure occurred on January 25, 2025, two weeks before the scheduled surgery. At the time, the patient was on a regimen of valproic acid 250 mg three times daily, Carbamazepine 200-200-100 mg, and Levetiracetam 500 mg three times daily.

Physical Examination

On admission, the patient was fully conscious with a Glasgow Coma Scale score of 15. Both pupils were equal, round, and measured 3 mm, with normal direct and consensual light reflexes. Cranial nerve examination showed no abnormalities. Motor examination revealed normal muscle strength and tone in all extremities. Deep tendon reflexes were physiological and symmetrical (2+) in both upper and lower limbs, with no pathological reflexes. Sensory examination was unremarkable, and autonomic function was within normal limits.

Supporting Examinations

Before surgery, the patient underwent a presurgical evaluation that included routine electroencephalography (EEG), long-term video electroencephalography monitoring (LTVEM), brain MRI with contrast, and a HARNESS protocol, as well as a neurobehavioral examination. A multidisciplinary epilepsy team concluded that the patient was an appropriate candidate for right anterior temporal lobectomy with amygdalohippocampectomy under intraoperative ECoG guidance.

Management of anesthesia

The patient was diagnosed with right temporal lobe epilepsy associated with hippocampal sclerosis and was scheduled to undergo a temporal lobectomy and amygdalohippocampectomy with ECoG monitoring. The patient was classified as American Society of Anesthesiologists (ASA) Physical Status III due to temporal lobe epilepsy and obesity (Body Mass Index 35.5 kg/m²), with no predicted airway difficulties. The planned anesthetic approach included general anesthesia with endotracheal intubation, scalp nerve block for local anesthesia prior to Mayfield head frame

Table 1. Supporting Examination

Laboratory examination	Hemoglobin 15.8 g/dL, Hematocrit 45.3%, Leukocytes 8130 / μ L, Platelets 337.000 / μ L PT 10.3/11.1 s, APTT 36.5/30.4 s AST 33 U/L, ALT 37 U/L BUN 25.7 mg/dL, Creatinine 0.8 mg/dL, eGFR: 122 mL/min/1.73m ² Sodium 139 mmol/L, Kalium 4.7 mmol/L, Chloride 100.9 mmol/L Glucose 93 mg/dL
Chest X-ray	Within normal limits
MRI brain with contrast	- Right Mesial Temporal Sclerosis
Long-Term Video	- Ictal Phase: The seizure onset originated from the right mid-temporal region and propagated to both hemispheres, predominantly on the right side.
Electroence-phalography Monitoring	- Interictal Phase: Epileptiform activity observed in the right temporal region, accompanied by polymorphic slowing in the same area. The recording was conducted during wakefulness and continued into NREM stage II sleep.

placement, and arterial line insertion. Prior to induction, the patency of the intravenous access was confirmed, and standard monitors along with Bispectral Index (BIS) monitoring were applied. After 5 minutes of preoxygenation, anesthesia was induced with 80 mg of lidocaine and remifentanyl at a target-controlled infusion (TCI) of 6 ng/mL, followed by 200 mg titrated propofol. Rocuronium 50 mg was administered for muscle relaxation, and manual ventilation was continued for 3 minutes prior to intubation with a 7.5 mm endotracheal tube. A scalp block was performed using 0.25% bupivacaine, with a total volume of

20 mL (10 mL on each side), to provide regional anesthesia. Two 18G peripheral intravenous lines and an arterial line were inserted, after which Mayfield head pins were applied and the patient positioned in reverse Trendelenburg. Anesthesia maintenance was achieved using TCI of propofol (Schnider model) at 2 to 5.5 μ g/mL and remifentanyl (Minto model) at 2 to 4 ng/mL. BIS was maintained within a target range of 40–60, and end-tidal CO₂ (ETCO₂) was kept at 30–35 mmHg. Throughout the intraoperative period, the patient remained hemodynamically stable with no significant fluctuations in baseline EEG activity (Figure 1).

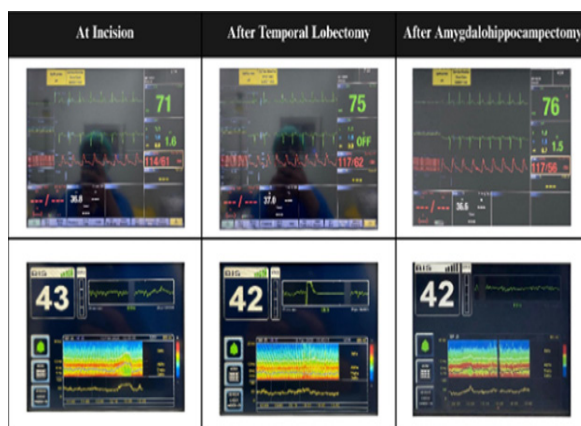


Figure 1. Baseline wave from Cz-A1 and Cz-A2

The neurology team were able to identify the epileptogenic activity and delineated the boundaries of the EZ for resection by the neurosurgical team. After the resection was completed and assessment for normal brain activity was performed, the Mayfield head pins were removed, followed by airway suctioning by the anesthesiologist. At this point, propofol infusion was stopped, while remifentanyl was maintained at a reduced target of 1 ng/mL until extubation. The patient was extubated while fully awake after demonstrating adequate spontaneous

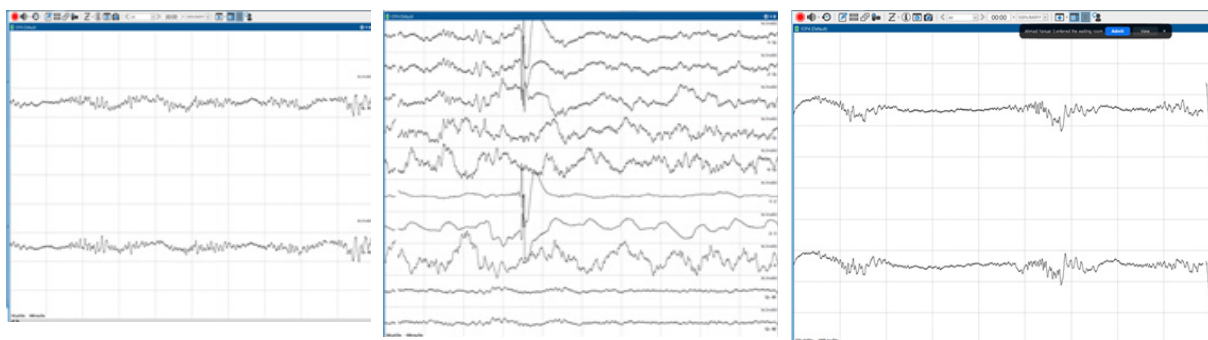


Figure 2. Baseline Wave from Cz-A1 and Cz-A2

ventilation. The total duration of anesthesia was 640 minutes. Intraoperative blood loss was estimated at 250 mL, and the patient received 3000 mL of crystalloid fluids. Urine output remained adequate throughout the procedure, averaging 1.98 mL/kg/h, and the intraoperative course was stable overall. Following surgery, the patient was admitted to the High Care Unit (HCU) for postoperative monitoring. During the intraoperative period, reference electrodes were placed at Cz-A1-A2 using a screw and twisted needle, and secured the ground electrode on the left shoulder. Before surgery, we checked the impedance and found it to be within normal limits. A baseline recording was performed at electrode Cz-A1, which revealed alpha waves with a frequency of 9-10 SPD and several vertex waves (Figure 2).

Before resection, a baseline recording was performed using a 1×6 ECoG electrode in the right temporal region. Periodic spikes were detected, particularly in the superior and middle anterior temporal region (within electrode 1-2-3) (Figure 3). The team elected to perform a right anterior temporal resection with amygdalohippocampectomy. Post-resection revealed no recurrence of periodic spikes in the area surrounding the resection (Figure 4).

Post-surgical Management

Following surgery, the patient was admitted to the High Care Unit (HCU) for postoperative monitoring. No seizure activity was observed during the postoperative period, and the patient did not report any complaints of nausea or vomiting. The patient remained well-oriented with no signs of memory impairment. Given the stable

clinical condition, the patient was transferred to the general ward on the first postoperative day. The patient exhibited satisfactory postoperative recovery and was discharged from the hospital on the third postoperative day.

Discussion

Epilepsy surgery may be performed under general anesthesia or local anesthesia with sedation, depending on the procedure and patient characteristics. The primary objective is resection of the epileptogenic zone (EZ) or disruption of epileptogenic networks responsible for seizure generation.⁶ Surgical options include temporal lobectomy and amygdalohippocampectomy, as performed in this case, as well as extratemporal cortical excision, hemispherectomy, corpus callosotomy, and neuromodulation techniques.¹ The choice of procedure is individualized based on epilepsy type, EZ localization, and preservation of neurological function.

Intraoperative electrocorticography (ECoG) is commonly used to directly record cortical electrical activity, assisting in EZ localization, defining resection margins, and preserving eloquent cortex. This is particularly important in temporal lobe epilepsy involving functionally critical regions, where accurate mapping reduces the risk of postoperative language and cognitive deficits. In this patient, the EZ had been localized preoperatively; however, intraoperative ECoG was used to confirm EZ boundaries and optimize resection precision. The patient underwent anterior temporal lobectomy with amygdalectomy and partial hippocampectomy under ECoG guidance, a

procedure shown to provide superior seizure control compared with medical therapy alone. Patients should be counseled regarding potential postoperative complications, particularly memory impairment, which may occur in approximately 25% of cases.^{7,8} The anesthetic management in this case was consistent with established neuroanesthesia principles for epilepsy surgery. The primary anesthetic goals include maintenance of cerebral perfusion, control of intracranial pressure, hemodynamic stability, facilitation of early postoperative neurological assessment, and preservation of neurophysiological monitoring quality.^{6–10} In the present case, these objectives were achieved using TIVA with propofol and remifentanyl, invasive arterial blood pressure monitoring, BIS-guided anesthetic titration, and scalp block analgesia. Unlike many other neurosurgical procedures, epilepsy surgery with ECoG requires anesthetic management that balances adequate hypnosis and analgesia while minimizing suppression of cortical electrical activity to allow accurate localization of the epileptogenic zone.^{7–10}

The anesthesiologist plays a central role during ECoG-guided epilepsy surgery. Anesthetic agents can significantly influence cortical electrical activity, potentially suppressing epileptiform discharges and compromising EZ identification. In addition to maintaining hemodynamic stability, cerebral perfusion pressure, and intracranial pressure control, anesthetic depth must be carefully adjusted to facilitate accurate ECoG recording.^{7,9,10} The effects of anesthetic agents on ECoG must be considered when planning anesthesia for epilepsy surgery. Benzodiazepines are generally avoided because they suppress epileptiform activity and may reduce the diagnostic value of ECoG recordings.^{7,9} Volatile anesthetics can alter cortical electrical activity in a dose-dependent manner and may affect interpretation of ECoG findings, particularly at deeper anesthetic levels.^{7–9} Propofol also influences cortical electrical activity; however, because of its rapid titratability and predictable pharmacokinetic profile, it remains one of the most commonly used anesthetic agents

during epilepsy surgery.^{8,9} Opioids generally have less effect on ECoG recordings than hypnotic agents, and remifentanyl may be useful because of its short context-sensitive half-life and ease of titration.^{7,8}

Therefore, TIVA using propofol and remifentanyl is frequently recommended for procedures requiring intraoperative ECoG monitoring.^{6–10} Anesthetic agents exert complex effects on neuronal transmission, acting as anticonvulsants or proconvulsants depending on dose and anesthetic depth. As anesthesia deepens, EEG activity typically transitions from faster alpha and beta waves to slower theta and delta patterns, reflecting progressive cortical suppression.⁸ In this case, general anesthesia was administered using a total intravenous anesthesia (TIVA) technique, with invasive arterial monitoring and bispectral index (BIS) monitoring to guide anesthetic depth.^{8,10}

Preoperative Management

Preoperative management requires careful review of long-term anti-seizure medication (ASM) therapy. In routine epilepsy surgery, ASMs are generally continued perioperatively. Dose reduction is more commonly considered during prolonged EEG monitoring to facilitate seizure capture but must be supervised to avoid breakthrough seizures or status epilepticus.^{9,10} Long-term ASM use may be associated with metabolic, hematologic, hepatic, and cardiovascular effects, particularly with enzyme-inducing agents. Accordingly, preoperative laboratory assessment and cardiovascular evaluation should be individualized based on patient risk factors. In this case, given the patient's young age and absence of cardiac symptoms, extensive cardiac evaluation was not required.^{7–10}

Enzyme-inducing ASMs accelerate the metabolism of anesthetic agents, potentially increasing requirements for drugs such as propofol and non-depolarizing neuromuscular blockers. These pharmacokinetic interactions highlight the importance of individualized anesthetic planning. In the present case, the patient was receiving chronic carbamazepine therapy, an enzyme-inducing ASM that may increase

the metabolism of several anesthetic agents and reduce responsiveness to non-depolarizing neuromuscular blockers.^{11,12} Consequently, anesthetic requirements may differ from those of other neurosurgical patients who are not receiving enzyme-inducing ASMs. Furthermore, because intraoperative ECoG was planned, anesthetic management required additional consideration of the effects of anesthetic agents on cortical electrical activity, making anesthetic titration more complex than in routine neuroanesthesia cases.¹³⁻¹⁷

Intraoperative Monitoring

During intraoperative ECoG, benzodiazepines are generally avoided due to their strong EEG-suppressive effects. Short-acting opioids may be used when activation is required to enhance epileptiform activity. Close, real-time communication between the anesthesiology, surgical, and neurophysiology teams is essential to optimize ECoG recording quality while maintaining patient safety.^{9,10,18} Anesthesia in this case was maintained with propofol and remifentanyl. Propofol provides reliable anticonvulsant effects, rapid titratability, and minimal interference with neurophysiological monitoring, making it the preferred agent for epilepsy surgery.^{8,9} Volatile anesthetics were avoided due to their potential to suppress cortical activity and provoke seizures at higher concentrations, particularly under hypocapnic conditions.⁹ We also established large-bore peripheral intravenous access and inserted an arterial line for invasive hemodynamic monitoring. Routine placement of a central venous catheter (CVC) is generally unnecessary for this procedure. Patient positioning must be handled with caution to avoid excessive head rotation, which can impede cerebral venous drainage via the jugular veins.^{7,8}

Induction in this patient was achieved using propofol, an agent with well-documented anticonvulsant properties. In patients with long-term ASM use, particular attention is required due to the potential for resistance to NMBs. Chronic use of ASMs—particularly enzyme-inducing agents such as carbamazepine—has been associated with reduced sensitivity to

non-depolarizing NMBs like rocuronium and vecuronium. Proposed mechanisms include increased hepatic metabolism, enhanced protein binding, and upregulation of acetylcholine receptors at the neuromuscular junction. Atracurium is thought to demonstrate less resistance in patients treated with carbamazepine; however, its metabolite laudanosine has been associated with a potential reduction in seizure threshold, and is capable of crossing the blood-brain barrier. Nevertheless, clinical studies indicate that laudanosine concentrations produced by standard atracurium doses typically remain below the threshold for proconvulsant activity.^{19,20}

Although non-depolarizing NMBs do not directly interfere with cortical electrical activity, appropriate selection and dosing remain important during procedures involving ECoG.^{7,8} In the present case, rocuronium 50 mg was administered during induction to facilitate tracheal intubation, and no additional neuromuscular blocker was administered during the ECoG recording period. Adequate anesthetic depth was achieved using propofol and remifentanyl, and a bilateral scalp block with 0.25% bupivacaine was performed before Mayfield head-pin placement to attenuate nociceptive stimulation and associated hemodynamic responses.^{7,8,10} BIS monitoring was used throughout the procedure to guide anesthetic depth and facilitate conditions favorable for ECoG interpretation.^{8,10}

During the intraoperative period, the patient received remifentanyl for analgesia at a target-controlled concentration of 6 ng/mL, along with administration of 80 mg lidocaine. Opioids have short durations of action and exert minimal effects on cortical activity when administered via continuous infusions. Remifentanyl, administered as a low-dose 1 mcg/kg bolus, has been shown to increase interictal spike activity, potentially facilitating the localization of ictal cortex regions during ECoG. However, at higher doses, remifentanyl may exhibit anticonvulsant properties, potentially suppressing epileptiform activity and interfering with accurate mapping. Careful titration of remifentanyl is essential to optimize ECoG interpretation. In patients with

seizure disorders, the proconvulsant potential of low-dose remifentanyl further underscores the need for cautious and closely monitored administration.^{7,8,10}

During epilepsy surgery, intraoperative seizures may occur and must be anticipated. Risk factors include younger age, recent preoperative seizure activity, frontal or parietal lobe lesions, and certain epilepsy etiologies, such as oligodendroglioma. Under general anesthesia, seizure activity may manifest indirectly through autonomic and physiologic changes, including tachycardia, hypercapnia, pupillary dilation, and increased muscle rigidity. BIS monitoring can assist in recognition of seizures under general anesthesia by detecting seizure-related fluctuations in cortical electrical patterns. Management requires prompt communication with the surgical team, irrigation of the surgical field with cold saline, and administration of a 10–30 mg bolus of propofol. If seizures persist, additional agents such as midazolam (1–2 mg) or thiopental (25–50 mg) may be administered. Avoidance of seizure-provoking factors, particularly intraoperative hypocapnia, is essential, and continuation of ASM therapy throughout the perioperative period is strongly recommended to reduce the risk of intraoperative seizures.^{7,8} In this patient, intraoperative hemodynamics remained stable, and no seizure activity was observed.

Postoperative Management

Delayed anesthetic emergence may occur in patients receiving long-term ASMs, which is noted in individuals receiving phenytoin intraoperatively. In this case, the propofol infusion was discontinued after Mayfield head pins removal, while remifentanyl was maintained at 1 ng/mL until the patient regained consciousness. Extubation was performed in the operating room before transfer to the HCU.

Postoperative monitoring focused on early detection of seizures, hemorrhage, and neurological deficits. Following temporal lobe surgery, potential complications include memory impairment, visual field defects, and aphasia. Accordingly, close neurological and

hemodynamic monitoring is recommended.¹⁰ The patient remained neurologically stable during the first 24 hours in the HCU and experienced no postoperative seizures. He was transferred to the general ward and discharged after an additional two days. Given the potential impact of GABAergic anesthetics on postoperative cognition, early neurological assessment and cognitive monitoring are recommended. Targeted rehabilitation strategies may be beneficial in patients at risk of cognitive decline.^{8,12} A multidisciplinary approach is essential to optimize long-term outcomes following epilepsy surgery.

Conclusion

In epilepsy surgery requiring resection of the EZ, a coordinated, multidisciplinary approach involving the anesthesiologist, neurosurgeon, and neurologist is essential for achieving favorable intraoperative outcomes while ensuring patient safety. This collaboration supports the use of TIVA without volatile agents under BIS guidance, enabling high-quality ECoG recording and accurate cortical mapping. In this case, the approach demonstrated a favorable perioperative course, characterized by the absence of intraoperative seizures, an uncomplicated postoperative recovery, and no occurrence of postoperative nausea and vomiting.

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