

# Anaesthetic Management of a Patient with Lennox–Gastaut Syndrome

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## ABSTRACT

Lennox–Gastaut Syndrome (LGS) is a rare childhood-onset epileptic encephalopathy characterized by multiple seizure types, cognitive dysfunction, and a distinctive electroencephalographic pattern. Anaesthetic management in these patients is challenging due to altered drug metabolism, potential interactions with antiepileptics, and risk of perioperative seizures. We present the case of a 23-year-old woman with LGS undergoing vagal nerve stimulator (VNS) implantation under general anaesthesia. Thiopentone was chosen for induction due to its anticonvulsant and neuroprotective properties, and sevoflurane was used for maintenance owing to its smooth titration and hemodynamic stability. Intraoperative BIS monitoring guided anaesthetic depth. The perioperative course was uneventful with no seizure episodes. This case highlights the importance of individualized anaesthetic planning, vigilant monitoring, and multidisciplinary collaboration in patients with refractory epilepsy undergoing neurosurgical procedures.

**Keywords:** Lennox–Gastaut Syndrome, anaesthesia, vagal nerve stimulator, thiopentone, epilepsy, BIS monitoring

## Introduction

Lennox–Gastaut Syndrome (LGS) is a severe developmental and epileptic encephalopathy typically manifesting between 1 and 8 years of age. It is characterized by multiple seizure types—most commonly tonic, atonic, and atypical absence seizures—along with intellectual disability and an interictal EEG showing slow spike-and-wave discharges. The management of these patients poses significant anaesthetic challenges due to chronic antiepileptic drug (AED) use, altered pharmacokinetics, airway hypotonia, and a high risk of perioperative seizures. The increasing use of vagal nerve stimulators in medically refractory epilepsy necessitates understanding their anaesthetic implications.

## Case Presentation

A 23-year-old female with an assisted vaginal birth and perinatal antecedents had global developmental delay since birth. She started having polymorphic seizures in the form of atonic, tonic, and atypical absence seizures since 16 years of age, with regression in motor milestones. Seizure characterization was done in 2022, and video EEG data showed multifocal interictal epileptiform discharges (IEDs) and atypical generalized spike-wave discharges with increased activation and burst-attenuation patterns.

Poor background and sleep architecture suggested epileptic encephalopathy, and the overall clinical and electrical data favoured a diagnosis of

Lennox–Gastaut Syndrome. She underwent a trial of single-pulse intravenous methylprednisolone, to which there was no response, and was subsequently posted for vagal nerve stimulator implantation.

She had persistent, disabling seizures and was on multiple AEDs. On examination, she was conscious, with stable haemodynamics. Neurological evaluation revealed cognitive impairment, generalized hypotonia, and stereotypical hand movements. EEG demonstrated frontotemporal diffuse slow spikes, and MRI brain showed bilateral perihippocampal gliosis. Echocardiography was normal.

Preoperative optimization included neurology consultation and adjustment of AED dosages. After preoxygenation, general anaesthesia was induced with thiopentone (2.5%), fentanyl, and rocuronium, followed by controlled ventilation. Anaesthesia was maintained with sevoflurane 2% in oxygen and air. BIS monitoring was utilized to maintain a target value around 50. The intraoperative course remained stable, and there were no episodes of bradycardia or asystole during VNS lead testing. The patient was extubated uneventfully and transferred to postoperative monitoring, where no seizures occurred and vital parameters remained stable.

## Discussion

Lennox–Gastaut syndrome (LGS) is a lifelong, severe developmental and epileptic encephalopathy that most often presents in early childhood (typically between 3 and 5 years of age). The classical clinical triad comprises multiple treatment-resistant seizure types (especially tonic, atonic/drop attacks and atypical absence seizures), a characteristic electroencephalographic (EEG) pattern of interictal slow spike-wave complexes (usually <2.5 Hz) often accompanied by generalized paroxysmal fast activity during sleep, and variable degrees of cognitive impairment or developmental slowing. While a structural, genetic, metabolic or acquired brain abnormality is identified in a majority of cases (termed “symptomatic” LGS), up to 30–40 % of cases may have no identifiable cause (so-called

“cryptogenic”). Because seizures are frequently refractory to medical therapy, patients often require multimodal treatment approaches (including dietary therapy, device-based implants or surgery) and are at risk for long-term neurological, functional and psychosocial morbidity.

## Anaesthetic Challenges in LGS

Anaesthetic management in patients with Lennox–Gastaut Syndrome (LGS) is complex and demands meticulous planning because of the unique physiological and pharmacological challenges associated with the disorder. These patients are prone to frequent, unpredictable seizures that may be triggered perioperatively by factors such as hypoxia, hypercarbia, stress, or abrupt withdrawal of antiepileptic drugs (AEDs). To minimize the risk of intraoperative and postoperative seizures, it is essential that AED therapy be continued on the day of surgery, and anaesthetic agents known to reduce seizure threshold should be avoided.

Long-term AED therapy, particularly with drugs such as valproate, carbamazepine, and phenytoin, alters the metabolism of many anaesthetic agents and neuromuscular blockers through induction of hepatic microsomal enzymes. This enzymatic induction can cause relative resistance to nondepolarising muscle relaxants like rocuronium, necessitating higher doses to achieve adequate paralysis. Additionally, valproate therapy is associated with thrombocytopenia and coagulopathy, which underscores the importance of assessing platelet count and coagulation profile in the preoperative evaluation.

Airway management can also be challenging in LGS patients because of generalized hypotonia, reduced airway tone, and associated cognitive impairment. These factors increase the likelihood of airway obstruction and delayed emergence from anaesthesia. Postoperative airway maintenance and monitoring, therefore, require special attention. Furthermore, preexisting neurological deficits can complicate the assessment of recovery from anaesthesia, as the patient’s baseline mental status may already be impaired.

The choice of anaesthetic agents should be guided by their effects on seizure threshold and interaction with AEDs. Thiopentone is often preferred for induction due to its strong anticonvulsant and neuroprotective properties, as it suppresses epileptiform discharges and maintains EEG stability. Sevoflurane is a suitable volatile agent for maintenance because it allows smooth titration, rapid recovery, and has minimal impact on seizure threshold when used at standard concentrations. However, high concentrations of volatile anaesthetics should be avoided as they may provoke epileptiform activity. Fentanyl is advantageous for analgesia as it provides haemodynamic stability and minimal interference with EEG activity. Propofol, while effective and possessing anticonvulsant properties, should be used cautiously in patients receiving valproate because of the potential risk of mitochondrial dysfunction. The use of Bispectral Index (BIS) monitoring helps to maintain adequate anaesthetic depth, reducing the likelihood of both intraoperative awareness and excessive anaesthetic suppression.

Intraoperatively, it is important to maintain physiological stability by ensuring normocapnia, normoxia, normoglycaemia, and normothermia. Hyperventilation and the resulting hypocapnia can reduce cerebral blood flow and increase the risk of seizure activity, and thus should be avoided. Postoperatively, patients benefit from close observation in a high-dependency or intensive care setting to detect delayed seizures, respiratory depression, or adverse effects from ongoing AED therapy.

## Vagal Nerve Stimulator (VNS) Implantation & Anaesthetic Implications

Vagal nerve stimulation is a palliative surgical therapy for medically refractory epilepsy, designed to reduce seizure frequency and severity by delivering intermittent electrical stimulation to the left vagus nerve. The mechanism of action is thought to involve modulation of neuronal excitability through projections from the vagus nerve to the nucleus tractus solitarius and onward to other limbic and cortical structures involved in seizure generation.

The procedure involves dissection of the left cervical vagus nerve—the left side is chosen to minimize cardiac side effects—and placement of a subcutaneous pulse generator in the upper chest wall, connected via leads. Anaesthetic management during this procedure requires vigilance because intraoperative stimulation of the vagus nerve during lead testing can cause bradycardia, hypotension, or even asystole. Continuous ECG and haemodynamic monitoring are therefore essential throughout the procedure, and the surgical and anaesthetic teams should be prepared to immediately terminate stimulation and administer atropine or initiate resuscitative measures if significant bradyarrhythmias occur.

General anaesthesia with endotracheal intubation is the preferred technique for VNS implantation, as it provides optimal airway control and immobility during the delicate dissection. Neuromuscular blockade aids surgical exposure of the vagus nerve in the neck. Succinylcholine should be avoided in patients with hypotonia or neurological impairment due to the risk of hyperkalaemia. Maintenance of anaesthesia can be achieved using either sevoflurane-based inhalational techniques or total intravenous anaesthesia (TIVA), depending on the institutional preference and patient's clinical stability.

Following implantation, the VNS device is typically activated a few weeks postoperatively. For any subsequent surgeries or diagnostic procedures, it is crucial to remember that electrocautery and MRI can interfere with the device's function; thus, the stimulator must be deactivated beforehand. Anaesthesiologists should also document the presence of the VNS device in the patient's medical records and ensure that caregivers are aware of its implications for future anaesthetic care and emergency management.

## CONCLUSION

Vagal nerve stimulator implantation can be safely performed in patients with Lennox–Gastaut Syndrome when anaesthetic management is tailored to the patient's neurological and pharmacologic profile. Key factors for success include

meticulous preoperative optimization, judicious choice of anaesthetic agents, vigilant intraoperative monitoring, and coordinated teamwork between anaesthesiologists and neurologists.

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