Myoclonic Movements Following Induction of Anesthesia with Propofol: A Case Report

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**Objective:** To report a case of myoclonic movements during an induction of anesthesia using propofol. Abnormal movements resulting from propofol are uncommon but there have been a number of such cases since propofol was introduced.

**Clinical features:** An 11-year-old boy with a diagnosis of obstructive sleep apnea was scheduled to undergo adenotonsillectomy. He demonstrated myoclonic movements during anesthetic induction using propofol. He was then given isoflurane and his airway was secured with an endotracheal tube after full muscle relaxation by succinylcholine. The anesthetic maintenance was uneventful as was the emergence. The patient recovered smoothly without neurological deficit.

**Conclusion:** Propofol, an intravenous anesthetic, with strong evidence of anticonvulsant property, could, in susceptible patients, under certain conditions, act as a proconvulsant, and should, thus, be avoided or cautiously used in some patients.

**Keywords:** Anesthetics: Intravenous, Propofol; Complications: Myoclonic movements

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The patient was a healthy 11-year-old boy with obstructive sleep apnea who presented for adenotonsillectomy under general anesthesia. He weighed 53.7 kg and was 150 cm in height. Apart from allergic rhinitis, there were no other underlying diseases, including epilepsy or movement disorders. He had not undergone any previous surgery. He was afebrile prior to and on the day of surgery.

The patient was not premedicated. He was monitored for blood pressure, ECG, arterial O₂ saturation and end-tidal CO₂. He was preoxygenated with 100% O₂. Fentanyl 50 mcg was given shortly before induction. Propofol 100 mg was given intravenously over 30 seconds. Soon after, the patient started to move his limbs which led to a concern that he was not fully anesthetized. More propofol was subsequently administered in increments of 20-40 mg. His involuntary movements became more vigorous, and were followed by myoclonic jerking. Propofol was terminated after the total dose of 220 mg. He was then given isoflurane by mask. Succinylcholine 50 mg was then given, following which his myoclonic jerking ceased. Tracheal intubation was performed once the patient was completely relaxed. Anesthesia was maintained with oxygen, nitrous oxide, isoflurane and vecuronium. The surgery was uneventful. His recovery from general anesthesia progressed satisfactorily, with a smooth emergence. He was extubated. The patient exhibited no neurological deficit. He was closely observed in a recovery room for 60 minutes before being transferred to a ward. The next day, he was discharged home.

**Discussion**

Propofol (2, 6 diisopropylphenol) is a rapid-onset and short-acting intravenous anaesthetic(3), first introduced in 1980(2). Since then, it has been widely used to induce and maintain anesthesia as well as to sedate patients in intensive care units(1-4). Propofol provides anesthetic effects through a potentiation of γ-aminobutyric acid A (GABAₐ) transmission(1,2). It also appears to inhibit glutamate release, possibly as a...
result of a complex activation on the GABAergic system.

The first case reports of seizure-like phenomena (SLP) in relation to propofol emerged in 1987(4,5). Since then, there has been an ongoing flow of similar reports(6). It was estimated by the Committee on the Safety of Medicines in the United Kingdom that the incidence of seizures after propofol administration was 1 in 47,000 exposures(6). SLP are classified as: 1) generalized tonic-clonic seizures, 2) focal motor seizures, 3) events presented as increased muscle tones with twitching and rhythmic movements not perceived as generalized tonic-clonic seizures, 4) opisthotonos, and 5) involuntary movements. Categories 1 to 3 are most suggestive of an epileptic origin whereas categories 4 and 5 are most likely non-epileptic in nature(5). SLP have been reported both in non-epileptic and epileptic patients either during induction of, maintenance of, emergence from, or following anesthesia(2-6). Spontaneous movements related to propofol have been seen with a higher incidence in children than in adults(6,9).

A systematic review indicates a predominance of SLP during induction of, emergence from, or following anesthesia, with a few occurrences during maintenance. Three causative factors have been advanced to explain this pattern. First, muscle relaxants are often used during maintenance of anesthesia, thereby preventing movements. Second, propofol plasma and cerebral concentrations remain stable during maintenance, suggesting that SLP are likely to occur during changes of propofol levels in the blood or brain. Third, there is frequently no alteration of consciousness during maintenance and, thus, probably less cerebral excitations which may predispose the patient to seizures(5).

The linkage between SLP and propofol is most prominent during induction in patients without neurological abnormalities because no conceivably confounding factors such as psychotropic drugs or surgical interventions are present(5). However, many patients are co-administered with other agents prior to or during propofol induction. Fentanyl has been acknowledged to increase plasma propofol concentrations by as much as 50%(9).

The majority of previously published case reports did not have simultaneous EEG monitoring to determine whether each propofol-related SLP was caused by actual cortical epileptiform activity. The lack of such EEG reports is a substantial limitation for the definite diagnosis of SLP in relation to propofol(2).

Propofol suppresses all parts of the central nervous system efficiently and uniformly(9). It causes marked cortical suppression presenting with isoelectric EEG in most cases(9). However, it appears to suppress subcortical structures in a different manner from cortical regions in which subcortical structures are influenced at lower concentrations and for much longer periods. This is manifested in such a way that subhypnotic doses of propofol have a direct antiemetic effect and lessen pruritus related to neuraxial morphine(4,8).

Throughout the central nervous system, inhibitory centers are more susceptible to anesthetic depression than are excitatory centers. Moreover, propofol has a greater subcortical effect than other anesthetics. Under these circumstances, propofol logically seems to cause a greater subcortical excitatory-inhibitory imbalance than other anesthetics. This imbalance exhibits as excitatory movements of the subcortical origin. According to this explanation, dystonic movements would be a result of midbrain and brainstem imbalance, whereas opisthotonos would be caused by a similar mechanism at the spinal level(8).

Myoclonus and opisthotonos have also been postulated as occasional side effects of propofol because propofol probably has a primary action on the GABA receptors with a much lower degree of glycine antagonism at subcortical levels. In addition, it has been suggested that propofol exerts an antidopaminergic effect which may be associated with the events observed(8).

A small group of epileptic patients experience true seizures which may be precipitated by propofol(9). Propofol has been shown to create epileptiform discharges on an EEG in patients with treatment-resistant temporal lobe epilepsy, even in regions not previously identified as epileptiform foci(3,9).

Alternatively, propofol is used to treat intractable status epilepticus in humans(5-9). It has been used successfully to terminate experimental status epilepticus in rabbits(3,6). Propofol infusions can produce burst suppression in both adults and children undergoing cardiopulmonary bypass. In patients undergoing surgery for unresponsive epilepsy, an EEG study showed no increase in seizure activity, but demonstrated significant burst suppression following propofol boluses. In patients with intractable partial epilepsy, propofol was shown not to induce increased seizure activity(4). Experimental studies in mice and rabbits documented that propofol was effective in aborting electrical and drug-induced seizures(4,6). Propofol shortens seizure duration when used for electroconvulsive therapy in comparison with methohexital(4,6).
It is thought that propofol, an anesthetic with strong evidence of anticonvulsant properties, might, in susceptible patients, under certain conditions, act as a proconvulsant\(^2-6\).

SLP induced by propofol may predispose patients to injure themselves in such ways as biting their tongues or falling from the operating table. Costs of health care are likely to be higher overall in propofol-related SLP patients because they may need to be admitted to the intensive care unit or have an ongoing follow-up requiring elaborate neurological investigations\(^2\).

It is suggested that the reporting of propofol-related SLP should cover the following: medical history, personal or family history of epilepsy and movement disorders, previous anesthetic history, current medications, drug and alcohol use, chemical dependency, emotional status prior to induction, occurrence of fever or hyperventilation, descriptive details of the SLP including rate of propofol injection and amount administered, onset of seizure, characteristics of signs, parts of the body affected, duration, postictal phase, any changes of the cardiovascular system, any potential complicating factors such as premedications, post seizure tests including temperature, blood sugar and electrolytes, arterial blood gas, neurological examination, EEG, CT scan\(^6\), and MRI study.

Propofol should be avoided or used with caution in epileptic patients\(^2,5\), especially those with poor control\(^4\), patients at risk of developing seizures\(^2,4\), patients who have previously experienced propofol-related SLP\(^6\), where antiepileptic drugs have been discontinued\(^6\), and patients taking tricyclic antidepressants or other antidepressants\(^7\).

References