PROPOFOL INDUCED MYOCLONUS MAY DISLODGE LARYNGEAL MASK AIRWAY
Dr. G. P. Rath1 Dr. P. K. Bithal2 Dr. H. Prabhakar3

SUMMARY
Propofol-induced myoclonus is not a new phenomenon. We present yet another case of myoclonus that was confined to neck and chest musculature, possibly caused by unchecked spinal segmental neuronal discharge as a result of disinhibition of supraspinal control. The patient was undergoing brachial plexus exploration under laryngeal mask airway (LMA) anaesthesia. There were two episodes of significantly increased airway pressure (Paw) that were subsided by relief from myoclonus with midazolam. We attribute the cause of increased Paw to LMA dislodgement by myoclonus of neck and possibly of pharyngeal musculatures.

Keywords: Propofol, Myoclonus, Laryngeal mask airway, Airway pressure.

Introduction
A wide range of excitatory events following induction of anaesthesia with propofol have been reported including opisthotonus, grand-mal seizures, and myoclonus.1,2 Perioperative myoclonus has been mainly reported involving limbs.3,4 We report yet another case of myoclonus which was atypical, not only because it involved muscles of neck and chest, but also, it caused dislodgement of laryngeal mask airway (LMA), resulting in significant increase in airway pressure (Paw).

Case report
A 19 year old, otherwise healthy male weighing 47 kg presented with inability to move his left upper limb since 2 months following a motor-vehicular accident. His medical and surgical history was unremarkable. All his routine investigations were within normal limits. A diagnosis of brachial plexus injury due to trauma was made. An exploration of brachial plexus was planned. In the operation theatre the patient was preoxygenated and anaesthesia was induced with fentanyl 100 g and propofol 100mg. An LMA # 4 was placed, successfully. After inflating the cuff with 30ml air, the patient was mechanically ventilated with tidal volume of 450 ml and respiratory rate 12 per minute. Paw at this time was 12 mmHg. Anaesthesia was maintained with propofol infusion (470 mg/hr) and intermittent fentanyl with O2 and N2O. Intraoperative monitoring included heart rate, non-invasive blood pressure, ECG, SpO2, end-tidal carbon dioxide (EtCO2) and Bispectral index (BIS). BIS value was kept between 40 and 60, titrating the infusion of propofol. Ten minutes later, a sudden rise in airway pressure to 41 mmHg was noted with leak around cuff. EtCO2 was also increased from 34 mmHg to 43 mmHg. Suspecting light plane of anaesthesia, propofol 40 mg bolus was purged from the infusion pump. However the condition did not improve. On auscultation of lungs there was diminished air entry but was not associated with any signs of bronchospasm; then, we suspected LMA displacement. At the same time, myoclonic movements of neck and upper part of chest were noticed. Midazolam 3 mg was given, intravenously. Twitching was abolished within 1 minute. The airway pressure also came down to 12 mmHg. Forty minutes later similar episode occurred again and was again relieved with midazolam. This prompted us to discontinue propofol, switching over to isoflurane along with O2 and N2O. Remaining period of anaesthesia was uneventful. At the end of the procedure because the patient was drowsy, LMA was removed in the postoperative ward after the patient became fully alert.

Discussion
Myoclonus is a movement disorder, which results from a lesion in any part of the neuraxis. Depending on the electroencephalographic (EEG) pattern, they may be epileptic or non-epileptic in origin. Anaesthetic agents like ketamine, etomidate, methohexitone, ether, and enflurane have been shown to produce seizure activity on EEG and have a potential to initiate perioperative seizures.5

In our patient myoclonus was probably, not an epileptic phenomenon as the BIS value was within 40 to 60 throughout the intraoperative period, not associated with any episode of gross fluctuation.5 Propofol seems to be the offending agent because similar episodes of myoclonus have been
reported in the past with this agent.\textsuperscript{3,4} However, the atypical observation in our case is involvement of neck and chest musculatures instead of limbs, as reported in the previous cases.\textsuperscript{3,4} The involvement of neck musculature and possibly, that of pharyngeal muscles caused the dislodgement of LMA resulting in increased Paw. Both the episodes of myoclonus were relieved by midazolam with normalisation of $P_{aw}$. This indicates myoclonus was the cause of increased $P_{aw}$. Propofol induced myoclonus has been reported in lighter plane of anaesthesia.\textsuperscript{8} In our case BIS value (40-60) was within plane of deep anaesthesia. Non-recurrence of myoclonus after discontinuation of propofol confirmed our suspicion that propofol was the offending agent. To counter the phenomenon of myoclonus, therapy aimed at potentiating inhibitory neurotransmitter glycine and $\gamma$-amino butyric acid (GABA) have been recommended using benzodiazepines, physostigmine, and chloromethiazole.\textsuperscript{9}

The involvement of neck and chest muscles in our case suggests spinal segmental myoclonus produced by unchecked spinal segmental neuronal discharge as a result of disinhibition of supraspinal control. A major role in the spinal form is played by abnormal discharges from interneurons and gamma motor neurons.\textsuperscript{10} Propofol is thought to have more subcortical potency than other anaesthetics do, and this effect on subcortical centers may persist at subhypnotic doses.\textsuperscript{11}

To conclude, we report yet another case of propofol induced myoclonus-involving neck and chest musculature that caused increased airway pressure probably due to dislodgement of LMA. The symptoms were relieved with use of midazolam, but only to recur later on. One should be cautious while maintaining airway by LMA under propofol anaesthesia.

References