

Literature Reviews

The efficacy of a subhypnotic dose of propofol in preventing laryngospasm following tonsillectomy and adenoidectomy in children. YK Batra, M Ivanova, SS Ali, M Shamsah, ARA Quattan, KG Belani. *Pediatric Anesthesia* 2005; 15:1094-1097

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Laryngospasm is a well known problem typically occurring following tracheal extubation. Propofol is known to inhibit airway reflexes. The authors have sought to assess whether the use of a subhypnotic dose of propofol, prior to emergence, will decrease the occurrence of laryngospasm following extubation in children. The study comprised of 120 patients, ASA status I and II, aged 3–14 years undergoing elective tonsillectomy with or without adenoidectomy. The study design was randomized, double blinded, and placebo controlled. Solid intake was allowed until midnight before the day of surgery, with clear liquids until 3 h before the start of surgery. All patients were premedicated with atropine 0.015 mg/kg and meperidine 1 mg/kg IM 30 min before anesthesia. Anesthesia was induced with thiopentone 4-5 mg/kg IV or with oxygen, nitrous oxide and Sevoflurane depending on patient preference. Each patient received 1.5 mg/kg of suxamethonium to facilitate tracheal intubation. Anesthesia was maintained with isoflurane 1.5-2.5% in 66% nitrous oxide in oxygen. Each received 1-2 mcg/kg of fentanyl IV. All patients were encouraged to breath spontaneously until completion of surgery. At the end of surgery the pharynx was suctioned and the child was allowed to breathe 100% oxygen. At the appearance of the first sign of tracheal reactivity the propofol group received 0.5 mg/kg, propofol IV and the randomized, double blinded control group received normal saline. Sixty seconds after the administration of the study drug tracheal extubation was performed and the children were administered oxygen. Occurrence of laryngospasm was recorded by another blinded investigator. Laryngospasm was graded as a condition occurring within 2 min after extubation, characterized by the following findings: (i) stridor; (ii) total occlusion of the cords, respiratory efforts with no air movement; (iii) cyanosis with evidence of airway obstruction at the level of vocal chords. Treatment of laryngospasm was standardized: 1) positive pressure ventilation with 100% oxygen with face mask; 2) aspiration of oropharynx and continued ventilation with 100% oxygen, if symptoms persisted; 3) administration of suxamethonium 1 mg/kg.

Comments: The authors noted an incidence of laryngospasm in 20% of the children in the control group (12 out of 60 patients). Seven had stridor, four demonstrated laryngeal occlusion and

one had cyanosis. Of these twelve children eleven responded to positive pressure ventilation with 100% oxygen via face mask and one required suxamethonium to break the spasm. In the propofol group 4 out of 60 had laryngospasm, an incidence of 6.6%. Three of these had stridor and one had occlusion. None required suxamethonium.

The authors have quoted several studies that show a 20% incidence laryngospasm following tonsillectomy. However an informal poll amongst anesthesiologists in private practice felt that the 20% reported incidence is too high. They suggested, although it is difficult to argue that as most of these studies came from teaching institutions, it may be that resident physicians learning the art may play a roll in the high incidence. By the same token the experienced private physician may underestimate the frequency of laryngospasm, as in the majority of these cases the problem is typically relieved by a modicum of positive pressure ventilation, which may then pass off as routine management. The etiology of the problem, which indeed is real, is still not well understood. Hence, several strategies and drugs have been suggested. These include injection of IV lidocaine 1 mg/kg or IV magnesium sulfate 15 mg/kg. Since laryngospasm does not occur under deep anesthesia, it has been suggested the patients be extubated while under deep anesthesia. Interestingly, there are recent studies quoted by the authors that report a 25% incidence of laryngospasm in patients extubated while under deep anesthesia and an incidence of between 21 and 27% when the patients were extubated wide awake. By contrast, there is a study that reports a zero incidence in wide awake patients, but there were only 20 patients in that group. The authors have demonstrated that a subhypnotic dose of propofol significantly reduced the incidence of laryngospasm.

The mechanism of action of propofol is not well understood but has been suggested that it is effective by attenuating laryngeal reflexes. It has been shown that afferent input from the vocal cords and larynx can activate N-methyl-D-aspartate (NMDA) receptors in the brain stem stimulating an efferent vagal response of vocal cord adduction. The authors suggest that propofol may have been effective in inhibiting NMDA receptors in the brain stem, thus stimulating an efferent vagal response of vocal cord adduction. There are reports that in some instances even a smaller dose of propofol, 0.25 mg/kg was effective in treating extubation laryngospasm. It may be that a larger, dose response study will be required to determine just how to prevent this common but life threatening problem.