Severe bradycardia during suspension laryngoscopy performed after tracheal intubation using a direct laryngoscope with a curved blade
-A case report-

Hyo Bin Ko, Dong Yeol Lee, and Yong Cheol Lee
Department of Anesthesiology and Pain Medicine, School of Medicine, Keimyung University, Daegu, Korea

There are a few reports about bradycardia or asystole caused by direct laryngoscopy. However, we encountered severe bradycardia in response to suspension laryngoscopy for laryngeal polypectomy after safely completing tracheal intubation using a direct laryngoscope with a curved blade. The tip of the curved blade of the direct laryngoscope is positioned at the vallecula (between the base of the tongue and the pharyngeal surface of the epiglottis) during tracheal intubation, while the blade tip of the suspension laryngoscope lifts the laryngeal surface of the epiglottis or supraglottic area during surgery. Therefore, suspension laryngoscopy can be said more vagotonic than curved-blade direct laryngoscopy. Because of the possibility of bradycardia induced by suspension laryngoscopy, clinicians must be careful about severe bradycardia even after safely completing intubation using direct laryngoscopy. (Korean J Anesthesiol 2010; 59: 116-118)

Key Words: Bradycardia, Remifentanil, Suspension laryngoscopy, Vagal reflex.
Case Report

A 49-year-old, 72 kg man with voice color change was diagnosed with laryngeal and vocal cord polyp. He had no history of other medication, surgery or significant illness. Microlaryngeal polypectomy was scheduled. Preoperative investigations were unremarkable. Monitoring methods included electrocardiography, noninvasive blood pressure monitoring, pulse oximetry, and capnography. The electrocardiograph showed a normal sinus rhythm of 85 beats per minute, and the patient’s blood pressure (BP) was 129/82 mmHg before anesthesia induction in the operating room. Glycopyrrolate, 0.2 mg, was given intravenously for premedication just before anesthesia induction. Anesthesia was induced with propofol, 140 mg, and followed by remifentanil, 210 μg; the patient’s lungs were ventilated with 100% oxygen. Propofol was mixed with 1% lidocaine 35 mg for reducing injection pain and the remifentanil was diluted to 10 ml with normal saline and injected slowly during a 90-second period.

One minute after administration of remifentanil, BP and heart rate (HR) were 89/60 mmHg and 57 beats per minute, respectively. The trachea was intubated with an endotracheal tube (ID 6.5 mm) using a curved-blade direct laryngoscope. Immediately after intubation, BP was 100/60 mmHg, HR was 64 beats per minute, and the oxygen saturation was 100%. There were no significant adverse events during intubation. We confirmed proper position of the endotracheal tube and adequate ventilation with stethoscope. Sevoflurane (1.0–2.5 vol%) was used to maintain anesthesia without a neuromuscular blocking agent. The BP and HR just before surgery began were 112/70 mmHg and 75 beats per minute, respectively.

However, when the surgeon placed a suspension laryngoscope (Laryngoscope 8590C, Karl-Storz, Tuttlingen, Germany) to secure a view of the vocal cords, HR progressively decreased to <30 beats per minute. Atropine, 0.5 mg, was given intravenously but was ineffective. The lowest heart rate was 18 beats per minute. At our request, the surgeon withdrew the suspension laryngoscope promptly, and then HR progressively increased to 70 beats per minute of itself; BP was 115/70 mmHg. On a second attempt of suspension laryngoscopy, HR decreased to <30 beats per minute, just as it did during the first attempt, so the surgeon again withdrew the suspension laryngoscope. The HR recovered to 68 beats per minute, and the BP was 120/79 mmHg. Five minutes later, we administered 0.5 mg of atropine intravenously again; then suspension laryngoscopy was performed without any problem. Surgery proceeded without further complications. The patient was discharged from ambulatory surgery 3 hours later without any problem.

Discussion

There are a few reports about bradycardia or asystole caused by direct laryngoscopy [6-9]. One factor in the occurrence of this phenomenon is the vagal reflex. Activation of afferent parasympathetic nerve fibers during stimulation of the lower pharynx, larynx, trachea, and epiglottis may result in bradycardia and asystole [9]. However, we encountered severe bradycardia caused by suspension laryngoscopy after safe completion of tracheal intubation using a direct laryngoscope.

This case report illustrates that although the tip of the curved blade of the direct laryngoscope is positioned at the vallecula (between the base of the tongue and the pharyngeal surface of the epiglottis) during tracheal intubation, the blade tip of the suspension laryngoscope lifts the laryngeal surface of the epiglottis or supraglottic area during surgery. The sensory distribution of the vagus nerve is more abundant in the supraglottic area and the laryngeal surface of epiglottis than at the base of the tongue and in the pharyngeal surface of epiglottis. Therefore, suspension laryngoscopy can be said more vagotonic than curved-blade direct laryngoscopy.

The glossopharyngeal sensory nerve fibers innervated in the nasopharynx, the caudal half of the soft palate, the caudal one third of the tongue, the vallecula, the pharyngeal surface of epiglottis, and the pharyngeal wall. Internal branch of the superior laryngeal nerve originated from the vagus nerve innervated in the laryngeal surface of the epiglottis, the laryngeal vestibulum, posterior wall of the glottis, caudal aspect of the vocal fold, subglottis and joining with the recurrent laryngeal nerve [10].

Other factors that may have contributed to the bradycardia in this case included administration of a relatively high dose of remifentanil (3 μg/kg) combined with propofol during anesthesia induction. It is unlikely that the bradycardia in our patient was due to remifentanil alone, because the peak effect of remifentanil is within 1 to 2 minutes, yet bradycardia occurred approximately 6 minutes after tracheal intubation and 7 minutes after remifentanil administration. Moreover, there was no bradycardia during direct laryngoscopy. Nevertheless, we could not completely exclude the influence of remifentanil. Although the peak effect time of remifentanil was past, remnant plasma remifentanil could influence occurrence of bradycardia in regard to context-sensitive half- time and terminal elimination half-times of remifentanil are 2–5 minutes and 9 minutes, respectively [11-13]. There is also abundant evidence of the potential of remifentanil and propofol to cause severe bradycardia and asystole, possibly through a centrally mediated decrease in sympathetic tone and vagally mediated bradycardia [2-5,14-16].

We ruled out hypoxia-induced bradyarrhythmia by pulse
Severe bradycardia during suspension laryngoscopy

oximetry (the oxygen saturation was 100%), capnography (continuous normal graph of 35 mmHg), and adequate ventilation. We ruled out severe hypotension-induced brady-arrhythmia by the presence of normal blood pressure during bradycardia.

In summary, clinicians must be careful about the occurrence of severe bradycardia induced by suspension laryngoscopy even after safely completing intubation using direct laryngoscopy. Although pretreatment with anticholinergics may prevent or attenuate vagus-mediated bradycardia, atropine could be insufficient for treating bradycardia once it occurs. Cessation of vagus nerve stimulation by withdrawing the suspension laryngoscope must first be considered.

References

1. Stevens JB, Wheatley L. Tracheal intubation in ambulatory surgery patients: using remifentanil and propofol without muscle relaxants. Anesth Analg 1998; 86: 45-9.
2. DeSouza G, Lewis MC, TerRiet MF. Severe bradycardia after remifentanil. Anesthesiology 1997; 87: 1019-20.
3. Briassoulis G, Spanaki AM, Vassilaki E, Fytrolaki D, Michaeloudi E. Potentially life-threatening bradycardia after remifentanil infusion in a child. Acta Anaesthesiol Scand 2007; 51: 1130.
4. Reid JE, Mirakhur RK. Bradycardia after administration of remifentanil. Br J Anaesth 2000; 84: 422-3.
5. Elliott P, O’Hare R, Bill KM, Phillips AS, Gibson FM, Mirakhur RK. Severe cardiovascular depression with remifentanil. Anesth Analg 2000; 91: 58-61.
6. Podolakin W, Wells DG. Precipitous bradycardia induced by laryngoscopy in cardiac surgical patients. Can J Anaesth 1987; 34: 618-21.
7. Cheong KF, Manivannan GK, Yau GH. Asystole following laryngoscopy and endotracheal intubation: a case report. Ann Acad Med Singapore 1996; 25: 283-5.
8. Mizuno J, Mizuno S, Ono N, Yajima C, Arita H, Hanaoka K. Sinus arrest during laryngoscopy for induction of general anesthesia with intravenous fentanyl and propofol. Masui 2005; 54: 1030-3.
9. Sutera PT, Smith CE. Asystole during direct laryngoscopy and tracheal intubation. J Cardiothorac Vasc Anesth 1994; 8: 79-80.
10. Yoshida Y, Tanaka Y, Hirano M, Nakashima T. Sensory innervation of the pharynx and larynx. Am J Med 2000; 108(Suppl 4a): 51S-61S.
11. Shim HS, Park TS, Park SH, Lee SH, Kim IK, Shin MK. Intubating condition and hemodynamic changes during tracheal intubation with propofol followed by remifentanil without the use of muscle relaxant. Korean J Anesthesiol 2005; 49: 617-23.
12. Kang TU, Shin HC, Lim HS, Ko SH, Han YJ, Kim DC. Optimal dosages of propofol and remifentanil for minimizing hemodynamic changes during laryngeal microscopic surgery. Korean J Anesthesiol 2008; 55: 314-9.
13. Beers R, Camporesi E. Remifentanil update: clinical science and utility. CNS Drugs 2004; 18: 1085-104.
14. Altermatt FR, Muñoz HR. Asystole with propofol and remifentanil. Br J Anaesth 2000; 84: 696-7.
15. Thompson JP, Hall AP, Russell J, Cagney B, Rowbotham DJ. Effect of remifentanil on the haemodynamic response to orotracheal intubation. Br J Anaesth 1998; 80: 467-9.
16. Krassioukov AV, Gelb AW, Weaver LC. Action of propofol on central sympathetic mechanisms controlling blood pressure. Can J Anaesth 1993; 40: 761-9.