

Unilateral Hypoglossal Neurapraxia Following Endotracheal Intubation for Total Shoulder Arthroplasty

Bryan Haslam, CRNA, MSN, CCRN
Shawn Collins, CRNA, PhD, DNP

A case is described of postoperative right hypoglossal neurapraxia after general anesthesia and interscalene block with endotracheal intubation for left total shoulder arthroplasty. Postoperative hypoglossal neurapraxia has been reported in cases, yet it remains a rare complication of anesthesia-related interventions. In this case report, postulated causes of hypoglossal neurapraxia are presented. A review of the literature pertaining to anesthesia-related causes of hypoglossal nerve injury is

included. Anesthesia providers should be aware of the course of cranial nerve XII as it relates to the position of the head and neck and use of airway instrumentation. In suspected cases of hypoglossal neurapraxia, conservative therapeutic interventions may be warranted.

Keywords: Beach chair position, cranial nerve XII, endotracheal tube, hypoglossal nerve, laryngeal mask airway.

The hypoglossal nerve (cranial nerve XII) is the sole motor nerve innervating the tongue. It exits the skull through the hypoglossal canal in the occipital bone and travels a course through the neck, ultimately reaching the mylohyoid and hypoglossus muscles (Figure), placing it in close proximity to airway instrumentation devices. Iatrogenic hypoglossal nerve injury has been described following laryngeal mask airway (LMA) and endotracheal intubation, but it remains an extremely rare neurologic injury related to anesthesia care. Other causes of hypoglossal nerve injury include malignancy, trauma (eg, gunshot wounds), stroke, multiple sclerosis, Guillain-Barré syndrome, infection, and even hysteria.¹ Idiopathic 12th-nerve palsy was found to be an ominous sign in a large case series by Keane,¹ yet Sharma et al² found that 60% of patients with isolated unilateral hypoglossal nerve palsy had substantial clinical recovery.

Case Summary

A 56-year-old woman was scheduled for left total shoulder arthroplasty. Her surgical history included gastric bypass surgery with a subsequent 57.2-kg (127-lb) weight loss, several orthopedic surgeries, hysterectomy, bladder surgery, bilateral carpal tunnel release, and tonsillectomy and adenoidectomy. She had a 10-pack per year history of smoking, but quit 10 years before this surgery. Neuromusculoskeletal history included arthritis and fibromyalgia. Preoperative Mallampati assessment revealed no neurologic deficits of the tongue. The findings of other physical systems appeared normal. Medications included acetaminophen and oxycodone (Percocet), glucosamine, hydrochlorothiazide, dulox-

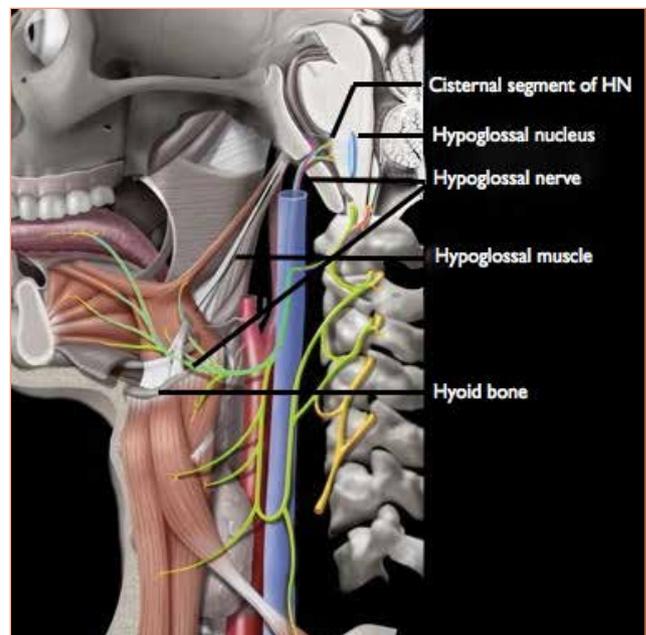


Figure. Lateral graphic depicting entire course of hypoglossal nerve (HN). Used with permission from *Diagnostic and Surgical Imaging Anatomy: Brain-Head and Neck-Spine*, published by Amirsys Inc, 2006.

etine (Cymbalta), diclofenac, misoprostol, amitriptyline, cyclobenzaprine, aspirin, ranitidine, vitamin C, vitamin B₁₂, calcium citrate, and vitamin E. Laboratory results were normal. The patient reported no complications with previous general anesthetics.

An ultrasound-guided left interscalene block was performed without difficulty after premedication with midazolam, 2 mg, intravenously (IV) and fentanyl, 100

µg, IV. Thirty milliliters of 0.5% ropivacaine was injected for postoperative pain control. An effective block was demonstrated before induction of general anesthesia.

The patient was brought to the operating room, and general anesthesia was induced with propofol, 130 mg; lidocaine, 60 mg; rocuronium, 50 mg; and fentanyl, 50 µg. A student registered nurse anesthetist performed oral intubation with a 7.0 Parker Flex-Tip endotracheal tube (ET) by direct visual laryngoscopy using a Macintosh No. 3 blade and sweeping the tongue to the left. The tube was inserted on the right side of the tongue and mouth and was secured in that position for the duration of the case. The intubation was reported to be easy, with a grade 1 view of the glottic opening. Intubation was accomplished on the first attempt; no trauma was noted.

The patient was positioned in a beach chair position with the head supported in a padded head support. The head and neck position were verified as neutral without pressure points. The head position was rechecked at frequent intervals during the procedure with no discernible change of position of the head, neck, or ET. General anesthesia was maintained with sevoflurane without nitrous oxide. At the end of the procedure, the patient was extubated while under deep anesthesia. Tidal volume and respiratory rate were adequate. An oropharyngeal airway was inserted. The patient emerged from anesthesia uneventfully, at which time the oral airway was removed. After full recovery in the postanesthesia recovery unit, she was transferred to a medical floor.

Several hours after the procedure, the patient reported difficulty swallowing solid food. She had no difficulty swallowing liquids. Examination by an anesthesiologist displayed tongue deviation to the right. Her speech was slurred. Mentation was normal. There were no signs of ischemic events affecting the central nervous system. An otolaryngologist consultation was ordered on the following day.

The otolaryngologist found no obvious lesions or ecchymosis in the oral mucosa. The patient's tongue appeared swollen on the right side, with a deviation to the right of midline on protrusion. No facial droop or eyelid lag was noted. No vocal cord paresis was seen on indirect laryngoscopy. She reported no pain.

It was surmised that the patient had suffered a compression injury to cranial nerve XII, possibly from the head position and/or ET position. Factors determined to be unrelated to the injury included the surgery itself and the interscalene block, which likely would be demonstrated by ipsilateral injury and probable involvement of the vagus nerve as in Tapia syndrome.^{3,4} Transient ischemic attack or hypoperfusion of the vertebrobasilar arterial system was deemed unlikely to cause isolated unilateral hypoglossal nerve injury without other cranial nerve involvement or signs of central nervous system deficits. We decided that the head cradle was not a cause

of compression because the head position on the cradle was well padded and was seen to support the posterior cranium with no pressure points near the course of cranial nerve XII.

Discussion

The literature describes few cases of unilateral hypoglossal nerve injury related to intubation. An integrated review of the literature was conducted using Academic Search Premier, Cumulative Index of Nursing & Allied Health (CINAHL), Cochrane Database of Systematic Reviews, EBSCO Host Electronic Journal Service (EJS), and MEDLINE. Keyword search terms included *cranial nerve XII* or *CN XII* or *hypoglossal and injury* or *neuropraxia* or *palsy*. Case reports that describe hypoglossal neurapraxia after endotracheal intubation are summarized in the Table.

Suspected mechanisms of unilateral hypoglossal nerve injury related to intubation include compression of the hypoglossal nerve against the hyoid bone,⁵ compression of the base of the tongue by the ET or laryngoscope blade,⁶⁻⁹ stretching of the nerve against the lateral process of the first cervical vertebra if the neck is hyperextended,^{7,9} or direct trauma.¹ The anatomical course of the 12th nerve places it in relatively protected tissues with the exception of close proximity to the hyoid bone. Inflated cuffs or ETs may compress tissues, and laryngoscope blades may compress or stretch lateral roots of the hypoglossal nerve in the tongue.

Several reports describe hypoglossal neurapraxia or palsy secondary to compression injury caused by the inflated cuff of the LMA.¹⁰⁻¹² The reports typically describe bilateral nerve involvement. Brain¹³ illustrated (by use of a cadaver) the proximity of the 12th cranial nerve to the inflated cuff of the LMA and the greater cornu of the hyoid bone.

Our otolaryngology consultant believed that the ET might have compressed the nerve. Without signs of ecchymosis in the oral or pharyngeal mucosa, however, it seems possible that the injury could have occurred to lateral roots of the nerve by the laryngoscope blade during brief laryngoscopy for intubation. A second-year student registered nurse anesthetist performed the intubation and had previously completed approximately 350 anesthetic cases with approximately 200 oral intubations using similar equipment. Both a nurse anesthetist and an anesthesiologist directly monitored the student. Improper technique during the intubation did not occur. Laryngoscopy was performed quickly with a single attempt, passing the ET atraumatically between the vocal cords. The ET depth was sufficient to inflate the cuff below the vocal cords (22 cm at the lips), placing the cuff well below the cornu of the hyoid bone.

Positioning of the patient was performed with a team effort. Several members of the surgical team, includ-

Source, year	Conclusions
Dearing, ⁶ 1998	Reported to be the first case in the literature of hypoglossal nerve injury following nasotracheal intubation. Possible mechanisms of injury include direct trauma due to excessive pressures applied to the throat pack or by hemorrhage caused by either the throat pack or the laryngoscope. Treatment included prednisolone, 50 mg/d, for 5 days followed by rapid tapering of the dosage. Recovery of function occurred by the 10th postoperative day.
Hong & Lee, ⁷ 2009	Following laparoscopic cholecystectomy, authors postulate that pressure to the lateral roots of the tongue during routine intubation caused a compression injury to the hypoglossal nerve. Conservative treatment included vitamin B and systemic corticosteroids for 2 weeks. Progressive recovery of function occurred over 3 weeks; however, tongue deviation was still evident.
Keane, ¹ 1996	Of 100 cases reviewed, the sole surgery-related case resulted from carotid endarterectomy with direct injury to the nerve. The author cites that 5% of carotid endarterectomies cause transient hypoglossal nerve injury. Other neck operations and even tonsillectomies have had reports of hypoglossal neurapraxia.
Mullins et al, ⁸ 1992	Postulated cause of hypoglossal neurapraxia was compression of the nerve by the ET with a change in position of the head while in beach chair position. No specific treatments were administered. Complete recovery of symptoms occurred by the eighth postoperative week.
Rubio-Nazábal et al, ⁹ 2002	Following emergent intubation for repair of ruptured abdominal aortic aneurysm, the patient displayed signs of bilateral hypoglossal neurapraxia after extubation. The authors postulate that hypotension combined with excessive pressure from the ET cuff may have caused the injury. Other suspected causes included pressure from the ET itself or from the Macintosh laryngoscope blade, or a change in the position of the head causing stretching of the nerve against the first cervical transverse process. No specific treatments given. Symmetrical extension of the tongue occurred at 1 month. Dysphagia resolved by 2 months. Normal tongue movements were documented by 6 months.
Venkatesh & Walker, ⁵ 1997	Surmised cause of hypoglossal neurapraxia was inadvertent extubation with inflated ET cuff causing compression or stretching of the nerve against the hyoid bone. Treatment included only observation in the neurosurgical ICU for 24 hours followed by observation on the hospital ward. Symptoms resolved over 6 days.

Table. Hypoglossal Neurapraxia and Transoral Intubation: Sample of Case Reports in the Literature

Abbreviations: ET, endotracheal tube; ICU, intensive care unit.

ing the surgeon, supervised the position of the head, neck, and torso on the operating bed in the beach chair position. Mullins et al,⁸ in 1992, described hypoglossal neurapraxia following shoulder arthroscopy and repair of the rotator cuff with the patient in the beach chair position. The authors commented that unilateral hypoglossal nerve compression by the ET had not been previously reported. They presumed that the reversible injury occurred during a change in position of the bed from 70 degrees to 30 degrees, causing the nerve to be compressed against the angle of the mandible. In our patient, however, we noted no changes in position of the head and neck. Nor did we note a change in position of the ET throughout the case. It is possible that a position change in the bed could have caused some torsion or pressure to the neck in such a manner that could cause a stretching or compression injury to the nerve, but this is without supportive observation.

Conclusion

Our patient demonstrated spontaneous progressive recovery of motor control of her tongue over a 6-day period. She was discharged from the hospital after a normal course of recovery. She showed no sequelae when assessed 4 weeks later.

Hypoglossal neurapraxia caused by intubation of the trachea has been suspected in several case reports. The true cause of hypoglossal neurapraxia remains unproved and illustrates the complexity of defining a mechanism of injury. Nonetheless, anesthesia providers should be familiar with the anatomy of cranial nerve XII as it relates to the position of the head and neck and as it relates to the use of airway instrumentation. Despite meticulous care and technique, injury to the 12th cranial nerve may occur as a result of the normal course of anesthetic care. The extremely low likelihood of this rare complication, however, gives it low relevance in

discussions with the patient for informed consent.

If hypoglossal neurapraxia is suspected, conservative management may be warranted, as 2 of the reported cases resolved after pharmacologic interventions and 3 of the neurapraxic injuries resolved spontaneously.

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AUTHORS

Bryan Haslam CRNA, MSN, CCRN, is a recent graduate of the Nurse Anesthesia Program at Western Carolina University, Enka, North Carolina. Email: bryanhaslam@gmail.com.

Shawn Collins, CRNA, PhD, DNP, is program director of the Nurse Anesthesia and DNP programs at Western Carolina University.

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