Lingual and Buccal Nerve Neuropathy in a Patient in the Prone Position: A Case Report

RUTH WINTER, M.D.,* MARK MUNRO, M.D., PH.D.†

Proper positioning of the head of a patient in the prone position is essential to prevent neurovascular compromise.¹ Unlike the awake patient who will respond to compression pain with protective reflex movements of the head and neck, the anesthetized patient is susceptible to unprotected head compression. This can result in nerve injury to the dependent eye, ear, and facial structures. Many authors²–⁸ have reported cases of facial and mental nerve injuries secondary to improperly positioned head straps, excessive pressure from an anesthesia mask, or undue pressure behind the mandible to relieve airway obstruction. There have also been case reports of isolated lingual nerve injury resulting in hypohesthesia of the tongue⁷ and isolated paresis of the hypoglossal nerve and combined hypoglossal nerve and lingual nerve damage following tracheal intubation and direct laryngoscopy.⁸

The following case report describes a unique compression injury to both the lingual and buccal branches of the mandibular nerve occurring in an anesthetized patient in the prone position.

CASE REPORT

A 46-yr-old man with a history of low back pain and normal neurologic examination was scheduled for an elective lumbar laminectomy. His past medical history was significant only for an open head injury 30 yr earlier requiring craniotomy but without known neurologic sequelae and a 45 pack-yr history of smoking. Physical examination revealed no abnormalities of the head or neck. The remainder of the examination was also unremarkable.

The patient received 3 mg midazolam intramuscularly as a preanesthetic medication. Induction of anesthesia with 300 mg thiopental was uneventful, and 100 mg succinylcholine was given to facilitate tracheal intubation. The patient’s trachea was intubated on the first attempt under direct visualization using a number 3 Macintosh blade and a number 8.0 endotracheal tube. Breath sounds were equal and bilateral. No trauma was noted to the mouth or oropharynx. The endotracheal tube was placed on the right side of the mouth and secured at 22 cm. An oral airway and an esophageal stethoscope were then inserted. The eyes were protected. The patient was then placed in the prone position with his head face down and slightly rotated to the left on a doughnut ring support (fig. 1). The nose and dependent eye and ear were carefully checked to ensure that they were properly padded. The extremities were positioned to prevent neurovascular compromise. General anesthesia was continued with isoflurane, N₂O, O₂, and fentanyl. Vecuronium was used for muscle relaxation. The operative course was unremarkable. The patient was turned supine at the end of the surgery, and the trachea extubated in the operating room without incident. The patient was taken to the postanesthesia recovery room where the immediate postoperative course was uneventful. The total anesthetic time was 3 h 20 min.

On the first postoperative day the patient began to complain of numbness on the right side of his face and tongue. He stated that the numbness had been present from the immediate postoperative period. He had no other complaints or symptoms. Physical and neurologic examination revealed paresthesia confined to the buccal area of the right side of his face and the right distal and lateral surface of his tongue. There was no facial, oral, or tongue erythema, edema, lacerations, or contusions. Motor function of the tongue and face were intact. By the third postoperative day sensory function had returned to the lower facial region; however, the tongue paresthesia persisted. The patient was discharged on the fourth postoperative day with partial resolution of his symptoms. The residual tongue paresthesia slowly resolved with total resolution of his symptoms by the fourth postoperative week.

DISCUSSION

The clinical presentation of the patient’s sensory deficits suggests a nerve injury in both the lingual and buccal branches of the mandibular nerve (fig. 2). The distribution of the general somatosensory deficit and taste sensation loss on the tongue (i.e., lateral and planar surfaces of the distal tongue limited to the right side) coincide spatially and functionally with the lingual nerve. However, because the sensory innervation to the buccal surface of the face is not provided by the lingual nerve, a second nerve injury involving the buccal nerve, or a single lesion site on the mandibular nerve proximal to the branching of the buccal nerve, is implied. The lack of motor deficits in the muscles of mastication innervated by branches of the mandibular nerve distal to the buccal nerve branch point excludes the possibility of a single mandibular nerve lesion. Thus, the only anatomically possible lesion⁹ that would account for the noted sensory deficits, with the lack of motor involvement, would be isolated lingual and buccal nerve injuries.

Based upon the noted nerve lesions, anatomic considerations of the lower facial region, and the positioning of the patient during the procedure, the most reasonable

* Instructor in Anesthesiology.
† Resident in Anesthesiology.

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Address reprint requests to Dr. Winter: Department of Anesthesiology, University of Michigan Medical Center, 1500 East Medical Center Drive, Room 1G323, Box 0048, Ann Arbor, Michigan 48109-0048.

mechanism of injury is prolonged nerve compression\textsuperscript{10-12} between the pterygoid muscles. One must also consider the possibility of direct compression of both the buccal and lingual nerves between the endotracheal tube and the ramus of the jaw. This would require the endotracheal tube between the larynx and angle of the mouth to assume a rigid position intraorally. Because the endotracheal tube warms over time and becomes more pliable, it is mechanically and spatially unlikely to exert substantial pressure against the mandible.

As previously described, the constellation of neurologic findings and the clinical course of recovery suggest a compression nerve injury to both the buccal and lingual nerves. The origin of the buccal nerve is in the pterygoid fossa, just medial to the lateral pterygoid. (Just proximal to its origin, the motor branches to the lateral pterygoid, temporalis, and masseter muscles branch off from the mandibular nerve. Because motor function of these muscles was unaffected, the lesion site must lie distal to this point.) Close to its origin, the buccal nerve passes through the two heads of the lateral pterygoid muscle and then descends along the medial surface of the temporalis muscle tendon to the buccal surface of the face (fig. 2). After emerging from the pterygoid muscle, there are no points along its course where the buccal nerve is likely to become compressed between rigid structures. Therefore, nerve compression as it passes through the lateral pterygoid—while the muscle is in tension—is the most likely point of injury.

The lingual nerve originates distal to the buccal nerve. It passes between the lateral and medial pterygoid muscles and then continues between the medial pterygoid muscle and the mandible. It then passes medially through soft tissue structures to the inferior surface of the tongue. Compression injury to the lingual nerve would be possible as it passes either between the medial and lateral pterygoid muscles and/or between the medial pterygoid and the mandible. In either case, the medial pterygoid muscle would have to be in tension for a compression injury to occur (fig. 2).

Based upon these considerations, for a compression nerve injury to occur to the ipsilateral buccal and lingual nerves, both the lateral and medial pterygoid muscles must be in tension. For this to occur in a paralyzed and anesthetized patient, the position of the mandible would have to be such that both muscles would be in passive tension. Because the mandibular motion for which both muscles are synergistic is mandibular protrusion, lower jaw retraction would place both muscles in tension.

The patient described herein was in the prone position with his head face down and slightly rotated to the left. The weight bearing points for the head were along the mandible and the forehead, with these structures resting on a foam ring support (fig. 1). In this position, the mandible would have been retracted, thus placing both the pterygoid muscles in passive tension. Therefore, compression injuries to both the buccal and lingual nerves can be explained based on positioning. However, involvement of the contralateral lingual and buccal nerves might have also been expected. A possible explanation for the lack of involvement of the contralateral nerves is asymmetric retraction of the mandible due to the slight rotation of the head to the left.

We have, to the best of our knowledge, presented a unique nerve compression injury resulting both from the presence of an endotracheal tube and the use of the prone position. In addition, we have described a possible mechanism to explain the sensory deficits exhibited by our patient. It should be noted that the patient's symptoms occurred within hours of the surgical procedure and were completely resolved by the fourth postoperative week. This is consistent with other reported cases of similar nerve compression injuries.\textsuperscript{3-6,10,11,13}
Pulse Oximetry and Patient Positioning: A Report of Eight Cases

ALEC R. HOVAGIM, M.D.,* W. WALTER BACKUS, M.D.,* GERARD MANECKE, M.D.,* ROBERT LAGASSE, M.D.,†
UPINDERJT SIDHU, M.D.,* PAUL J. POPPERS, M.D.‡

Pulse oximetry, used to measure arterial hemoglobin oxygen saturation, can also serve to monitor the adequacy of arterial blood flow.1–5 The use of pulse oximetry helps to ensure proper positioning of patients on the operating table. Eight cases are presented in which the use of pulse oximetry alerted the anesthesiologist of a patient’s improper position.

CASE REPORTS

Case 1. A 27-yr-old obese woman (90 kg, 1.7 m) with persistent pelvic pain was scheduled for diagnostic laparoscopy. In addition to other monitors, a pulse oximeter sensor (Nellcor N-100, software version 68-1, Hayward, California) was applied to the patient’s left index finger. After induction of general anesthesia, the patient was positioned in the lithotomy position with the upper extremities at 90° angles to her body. Immediately thereafter, the pulse oximeter was unable to detect a pulse. Palpation confirmed the absence of left forearm pulses; they remained palpable in the right forearm. No observable change in skin color or skin temperature was detected at that time. Repositioning the armboard to less than 90° was followed by prompt return of peripheral pulses as detected by the pulse oximeter and palpation.

Case 2. A 49-yr-old man with a right middle lobe lung mass was scheduled for thoracotomy. In addition to other monitors, a pulse oximeter sensor was applied to the patient’s left index finger. General anesthesia was induced and the patient was positioned in the left lateral decubitus position. After placement of an axillary roll, the pulse oximeter did not detect a pulse. Palpation confirmed the absence of left forearm pulses. The axillary roll was noted to be located high in the axilla and immediately repositioned further caudad, after which there was a prompt return of peripheral pulses.

Case 3. A 42-yr-old woman with persistent hemoptysis and fever was scheduled for an open lung biopsy. In addition to other monitors, a pulse oximeter sensor was placed on the left index finger. After induction of general anesthesia, the patient was placed in a 45° semilateral position supported by folded sheets under the left chest. The left (upper) arm was suspended from the anesthesia screen, which was subsequently angled cephalad to aid surgical exposure. At that point, the pulse oximeter was unable to detect a pulse. Palpation confirmed the absence of left forearm pulses; they remained palpable in the right forearm. It was then noted that the left arm was hyperabduced greater than 90°. Return of the screen to its original position was followed by return of peripheral pulses.

Case 4. A 33-yr-old man, a victim of a motorcycle accident, was scheduled for an emergent exploratory laparotomy. In addition, he also suffered bilateral clavicular fractures. His chest x-ray revealed a right lung contusion and a right upper lobe hematoma near the fractured and markedly dislocated right clavicle. In addition to other monitors, a pulse oximeter sensor was placed on the right index finger.

References


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