

Airway Obstruction after Bilateral Carotid Endarterectomy

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Bilateral hypoglossal nerve palsy after a second carotid endarterectomy (CEA) has caused upper airway obstruction in the supine patient.¹ More recently, the importance of upper airway musculature in maintaining a patent airway during anesthesia has been reexamined.² We describe a case of airway obstruction during a second CEA that led to respiratory and cardiac arrests. We postulate that bilateral hypoglossal nerve deficit resulted in loss of upper pharyngeal muscle control.

REPORT OF A CASE

A 62-year-old, 66-kg man with a history of transient ischemic attacks and with angiographic demonstration of bilateral carotid stenosis underwent a right CEA under cervical plexus block anesthesia. The surgery was complicated by postoperative hematoma in the neck, which required emergency neck exploration under general anesthesia.

Approximately five weeks later, a left CEA was scheduled. At that time the patient was receiving captopril, 100 mg three times a day, and hydrochlorothiazide, 25 mg a day, for hypertension. His only complaint at that time was numbness in the right anterior neck, present since his previous neck surgery. Physical examination, including gross examination of the cranial nerves, was unremarkable except for decreased sensation in the distribution of the right anterior superficial branches of C2 and C3. Laboratory examination yielded hemoglobin 11.1 g/dl, hematocrit 31.5%, BUN 52 mg/dl, and serum creatinine 2.1 mg/dl. Chest roentgenogram and ECG were normal.

After premedication with diazepam, 10 mg orally, and morphine sulfate, 5 mg im, a left, deep cervical plexus block was performed by injecting 5 ml of a 1:1 mixture of 1% lidocaine and 0.5% bupivacaine at the transverse processes of C2, C3, and C4. Additionally, 10 ml of the local anesthetic mixture was injected subcutaneously along the posterior border of the sternocleidomastoid muscle.

Satisfactory anesthesia was obtained and the procedure progressed

for 120 min, during which time the patient received fentanyl, 150 μ g in increments of 50 μ g; diazepam, 5.0 mg in 2.5-mg increments iv, for complaints of generalized discomfort due to the surgical position (upper body elevated 20 degrees with head and neck held in flexion to the right); and oxygen via nasal cannula at 3 l/min. At that time the patient complained of pain at the surgical site, and the surgeons injected 10 ml of 1% lidocaine locally. The complaints of pain continued and an additional 5-10 ml of 1% lidocaine was injected around the left hypoglossal nerve, which was being dissected at that time.

About 10 min later, the patient began to complain of difficulty breathing and was noted to have a partial upper airway obstruction with little air movement at the mouth or nares and paradoxical motion of the thorax and abdomen. Until this point, the patient had had no complaints, except as noted, and had been oriented, talking appropriately, and following commands. Surgery was stopped immediately (the surgeons had not applied any vascular clamps); the airway was manipulated manually to alleviate the obstruction and positive-pressure assisted ventilation with an $F_{I_{O_2}}$ of 1.0 was administered by face mask, which failed to establish adequate ventilation. During this time, asystole occurred, and the radial artery tracing displayed no waveform. External cardiac massage and endotracheal intubation were performed (with evidence of adequate gas movement by chest auscultation) and, while the resuscitation cart was brought, ephedrine, 20 mg iv, was given. No airway abnormality was noted during laryngoscopy. After about 45 s of asystole (the actual time was not noted, except that it was longer than 15 s but less than 50 s), bradycardia developed and progressed to sinus rhythm and arterial blood pressure increased to 120/60 mmHg. Cardiac massage was stopped and spontaneous breathing returned. Arterial blood gases at that time were pH 7.36, $P_{a_{O_2}}$ 380 mmHg, $P_{a_{CO_2}}$ 37 mmHg, and base deficit 3 mEq/l. Five-lead ECG, including V5 chest lead, revealed sinus rhythm with no evidence of myocardial ischemia or infarction. The CEA was then performed with the aid of a shunt, and regional anesthesia was supplemented with 0.25-0.5% isoflurane in oxygen with controlled ventilation.

Approximately 60 min later, after completion of the surgery and resumption of adequate spontaneous breathing, the patient was awake and following commands and the trachea was extubated. Total upper airway obstruction ensued that could not be relieved by oral or nasal airway devices. During laryngoscopy, the obstruction was totally relieved when the tongue was lifted forward with the laryngoscope blade, and total obstruction followed relaxation of the tongue. The patient was unable to protrude his tongue, therefore, tracheal reintubation was performed. The pharyngeal and laryngeal structures appeared normal without edema or deviation.

The patient was transferred to the ICU, and the endotracheal tube was left in place for 48 h, after which time the trachea was extubated without further airway obstruction. When the patient was discharged after the fourth postoperative day, he still complained of bilateral numbness in the distribution of the anterior superficial branches of C2 and C3 and, although he still displayed weak tongue movements, he had no further airway obstruction.

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DISCUSSION

The advantage of performing CEA under cervical plexus block while the patient remains conscious and, thus, acts as a constant monitor of cerebral function is obvious.^{3,4} One distinct disadvantage may be that if airway problems arise, as during surgery of the neck, the patient lacks the advantage of having the airway protected by endotracheal intubation, as this case points out.

At the time of its occurrence, the cause of the upper airway obstruction was unclear. The patient remained conscious, oriented, and able to talk throughout the procedure until the time of airway obstruction, which clearly preceded the cardiopulmonary arrest. Over sedation therefore appeared doubtful as the cause. Although we felt that our monitoring was adequate (direct observation and verbal communication, five-lead ECG with V5 chest lead, and radial arterial pressure monitoring), transcutaneous pulse oximetry or PaO₂ monitoring might have indicated impending respiratory compromise if hypoxemia or hemoglobin desaturation were occurring gradually. However, airway obstruction occurred suddenly, and it was our inability to diagnose the cause of the obstruction and to reestablish adequate gas exchange with mask ventilation that most likely led to asystole. Indeed, with endotracheal intubation, adequate ventilation was restored.

Other possible causes of the sequence of events that occurred could have been primary cardiac, pulmonary, or central neurologic events. However, none of these seemed likely. Before the airway obstruction, the patient had no symptoms or complaints that suggested myocardial ischemia or impending infarction. His ECG revealed sinus rhythm with no acute changes in the V5 chest lead that suggested ischemia either before airway obstruction or after the arrest. He had no chest pain, dyspnea, diaphoresis, or cyanosis before the airway obstruction. Furthermore, he had no previous history of ischemic heart disease. The patient's preoperative evaluation for pulmonary disease was negative despite a history of smoking ½ a pack of cigarettes per day for almost 50 years. Preoperative lung examination, chest roentgenogram, and arterial blood gases immediately before surgery were all normal. There was no intraoperative suggestion of primary pulmonary compromise before the airway obstruction. Finally, no neurologic changes suggesting central nervous system ischemia, embolism, thrombosis, or hemorrhage occurred intraoperatively before the airway obstruction.

The patient had been awake, talking, and oriented; upper airway obstruction and secondary respiratory

compromise occurred suddenly and was not corrected by mask ventilation. Asystole ensued. With the establishment of an airway by endotracheal intubation and adequate ventilation, resuscitation was rapid and successful. The patient's inability to protrude his tongue at the end of surgery demonstrated a lack of muscle control of the tongue and upper pharynx. We postulate that upper airway obstruction occurred secondary to bilateral hypoglossal nerve dysfunction.¹

The hypoglossal nerves supply the motor fibers of the intrinsic muscles of the tongue as well as the subglottic upper pharyngeal muscles.⁵ Specifically, branches go to the styloglossus, hyoglossus, genioglossus, and the intrinsic muscles of the tongue. Other nerves connected with the hypoglossal trunk, but actually derived from C1, C2, and C3, occupy the same connective tissue sheath and supply the infrahyoid muscles, specifically the geniohyoid, thyrohyoid, sternothyroid, sternohyoid, and omohyoid muscles.⁵ During its course in the neck, the hypoglossal nerve lies in close proximity superficial to the carotid artery. During a CEA, this nerve is often subjected to the trauma of dissection, application of local anesthetic around the nerve may lead to a deficit, and traction on the nerve may produce pain because sensory fibers of C2 and C3 are in the same connective tissue sheath as the hypoglossal nerve.

There were several reasons for bilateral hypoglossal nerve deficits to have developed in this patient. In a period of little over a month he had undergone bilateral neck dissections with direct trauma to the nerves during two CEAs and, during the second CEA, had undergone a major nerve block of the left side of the neck as well as application of local anesthetic directly around the nerve. With bilateral hypoglossal nerve deficit, there would be a loss of motor function of the tongue and upper pharyngeal muscles and an inability to protrude the tongue. The tongue and subglottic musculature would relax distally into the upper pharynx and thus obstruct the airway. Our patient, although having no obvious abnormalities of tongue movement preoperatively, demonstrated exactly these events postoperatively.

Recently, Nishino *et al.* demonstrated decreasing hypoglossal nerve activity in cats subjected to increasing depths of anesthesia and related this to patency of the upper pharyngeal airway.² The importance of upper airway muscles supplied by the hypoglossal nerves in maintaining a patent pharyngeal airway during sleep⁶ and during anesthesia⁷ has also been demonstrated.

Another problem this case presents is continuing surgery in a patient who has an intraoperative arrest. We felt justified in continuing surgery after careful consideration of the facts. We observed a sequence of

events that led to cardiopulmonary compromise; the primary event was acute airway obstruction that persisted in spite of mask ventilation and led to asystole. With proper ventilation by endotracheal intubation, a rapid resuscitation was accomplished, without any evidence of prolonged tissue hypoxia or acidosis as revealed by analysis of arterial blood gases immediately after the restoration of sinus rhythm. Furthermore, we felt confident in ruling out a primary cardiac, pulmonary, or neurologic event based on monitoring. The presence of critical stenosis coupled with these facts led us to the conclusion that we could safely continue with surgery.

We believe that transient bilateral hypoglossal nerve deficit occurred after a second CEA and resulted in total upper airway obstruction. Anesthesiologists should consider this risk in any patient who is scheduled to undergo neck surgery twice within a relatively brief period, regardless of whether regional or general anesthesia is used.

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