3. Shaw WW: Edema of the larynx, a rare complication of endotracheal anesthesia—a case report. ANESTHESIOLOGY 7:416–418, 1946

4. Crispell LS, Hampton LJ: Laryngeal edema complicating endotracheal anesthesia in children: case report. Conn State Med J 14:98–99, 1950

5. Bryce DP, Briant TDR, Pearson FG: Laryngeal and tracheal complications of intubation. Ann Otolaryngol 77:442–461, 1968

6. Lindholm CE: Acute damage in the larynx and trachea following prolonged endotracheal intubation and its healing course. Acta Anaesthesiol Scand suppl XXXIII, 1969

Airway Obstruction Following Surgical Manipulation of the Posterior Cranial Fossa, an Unusual Complication

DANIEL W. GORSKI, M.D.,[*] TADIKONDA L. K. RAO, M.D.,[†] TIMOTHY B. SCARFF, M.D.[‡]

Removal of a tumor that necessitates neurosurgical manipulation with retraction in the posterior fossa may temporarily or permanently injure the delicate structures coursing through the posterior fossa. The following report describes an unusual cause of airway obstruction following extubation of the trachea in a patient who underwent subtotal removal of a tumor in the posterior cranial fossa.

REPORT OF A CASE

A 71-year-old man was admitted for investigation of headaches, syncope, and episodic losses of consciousness of three months' duration. Results of physical examination were essentially within normal limits. Neurologic examination revealed no cranial nerve dysfunction. Tandem gait was ataxic. Cerebral angiogram and pneumoencephalogram revealed a midline posterior fossa mass in the region of the cerebellum, extending into the fourth ventricle and inferiorly into the foramen magnum. The patient was scheduled for exploration of the posterior cranial fossa and removal of the tumor.

The patient was premedicated with phenobarbital, 100 mg, and atropine sulfate, 0.4 mg, im, one hour prior to the anticipated time of operation. In the operating room, cannulations of a peripheral vein, right internal jugular vein, and left radial artery were performed using local anesthesia. Anesthesia was induced with thiopental sodium, 300 mg, iv, given in incremental doses, and endotracheal intubation was facilitated with pancuronium, 8 mg, iv. Anesthesia was maintained with 60 per cent nitrous oxide in oxygen, supplemented by fentanyl citrate as needed. Throughout the operation, the electrocardiogram, direct arterial and central venous pressures, urinary output, esophageal temperature, and heart tones (through an esophageal stethoscope) were monitored. A Doppler ultrasonic detector was appropriately positioned on the anterior chest wall to detect air embolism. The sitting position was achieved without difficulty. Arterial blood pH and blood gases were monitored as needed throughout the operation, and Pa_{CO_2} was maintained around 30 torr. The entire intraoperative course was uneventful. The operation consisted of subtotal removal through a suboccipital craniectomy of an ependymoma arising from the floor of the fourth ventricle.

Upon conclusion of the surgical procedure, the patient was ventilated with oxygen for 5 min, and the residual neuromuscular block was reversed with atropine, 2 mg, and prostigmine, 5 mg, iv. Within 5 min, the patient was awake and responded appropriately to verbal commands, but was tolerating the presence of the endotracheal tube. Tidal volume was about 525 ml, with a forced vital capacity of 1,800 ml and a respiratory rate of 12–15/min. Nerve stimulation revealed normal responses to single and tetanic stimuli, with no posttetanic potentiation. The patient had good hand grasp and was able to perform a sustained head lift for more than 15 sec. Since the patient met all the criteria for extubation of the trachea, the endotracheal tube and oral cavity were suctioned and the trachea was extubated. Stimulation of oropharynx by suctioning did not provoke any gag or cough reflex. Immediately following extubation, upper airway obstruction with marked inspiratory stridor occurred. The obstruction remained in spite of forward subluxation of the mandible. Laryngoscopy revealed a normal-sized tongue and epiglottis. The vocal cords and the movements of vocal cords during inspiration and expiration appeared normal. The patient could not protrude the tongue on command, and did not cough or gag when the laryngoscope was placed in the mouth. The trachea was reintubated.

Postoperatively the patient did not regain either the motor function of the tongue or the gag and cough reflexes. Impairment of salivary secretion by the parotid gland was not evaluated. Clinically there was no injury to the vagus nerve. On the sixth postoperative day, a tracheostomy was performed, since the reflexes had not returned.

DISCUSSION

Laryngoscopy and visualization of the upper airway and normal movement of vocal cords in this patient excluded macroglossia, glottic edema, recurrent laryngeal nerve palsy, and external compression of the airway as the cause of the airway obstruction following extubation. Normal neuromuscular re-

sponses to twitch and tetanic stimulation, sustained head lift for more than 15 sec, and proper responses to verbal commands ruled out residual neuromuscular block or residual anesthetic drug action as the causative factor in the airway obstruction.

The acute onset of airway obstruction immediately following extubation of the trachea in this patient can be explained on the basis of trauma to the adjacent cranial nerves due to operative manipulation. Consideration of the anatomic location of the cranial nerves helps to identify the cause of airway obstruction in this patient. The glossopharyngeal nerve arises from the posterolateral sulcus of the medulla craniad to the fibers of origin of the vagus and accessory nerves. The afferent pathway of the pharyngeal or gag reflexes is through the sensory fibers of the glossopharyngeal nerve, and the efferent pathway is through the motor fibers of the vagus nerve.[1] The hypoglossal nerve supplies the motor fibers to the tongue. Trauma to the glossopharyngeal nerve results in absence of the gag reflex and impairment of parotid gland secretions. Inability to move or protrude the tongue results from injury to the hypoglossal nerve. Thus, when glossopharyngeal nerve is injured bilaterally, as occurred in this patient, pharyngeal or gag reflexes will disappear, resulting in inability to protect the airway, and injury to

hypoglossal nerve results in the tongue's falling back, leading to airway obstruction.

In retrospect, the lack of gag or pharyngeal reflexes with suctioning and stimulating the pharynx prior to extubation of the trachea should have aroused suspicion regarding the status of patient's cranial nerves. This case points out that airway obstruction could occur in spite of the patient's meeting all the criteria for tracheal extubation following surgical manipulation of the posterior fossa, in the absence of anatomic abnormalities of the tongue, pharynx, and larynx.

Since the tumor distorted the anatomy of the nerves, and as the tumor was removed piece by piece, the surgeon was not aware of the injury to the cranial nerves. The neuro-anesthesiologist should be aware of the surgical anatomy and the structures in the operative field, and should make a practice of inspecting the operative site before closure commences. This would help to predict the possibility of cranial nerve dysfunction in the immediate postoperative course.

REFERENCE

1. Rex MAE: A review of the structural and functional basis of laryngospasm and a discussion of the nerve pathways involved in the reflex and its clinical significance in man and animals. Br J Anaesth 42:891–898, 1970

Blood Levels of Bupivacaine after Injection into the Scalp with and without Epinephrine

PETER S. COLLEY, M.D.,* AND JAMES E. HEAVNER, D.V.M., PH.D.†

Infiltration into the scalp of a local anesthetic is often done for the surgical excision of a seizure focus. This will result in the appearance of the drug in the systemic circulation.[1] Since local anesthetics have anticonvulsant activity, systemic absorption might result in blood levels sufficient to decrease

* Assistant Professor.

† Associate Professor.

seizure activity and interfere with the localization of the seizure focus.[2] On the other hand, higher blood concentrations may be associated with symptoms of local anesthetic toxicity. In this report, we present observations on blood levels of bupivacaine following subcutaneous infiltration into the scalp of 0.125 per cent and 0.25 per cent solutions with and without epinephrine, 1:400,000.

METHODS

Twenty-one patients undergoing awake craniotomy for excision of a seizure focus were studied. On arrival in the operating room patients were sedated with 1–2 ml of either fentanyl–droperidol or fentanyl alone administered intravenously. Routine monitoring for these procedures included electrocardiography and continuous arterial pressure monitoring via the radial artery.