◇ This Month in

ANESTHESIOLOGY

■ Minimizing Impact of Endotracheal Intubation during Cervical Spine Surgery. Audu *et al.* (page 898)

Some researchers have suggested that dysfunction of the vocal folds, a well-documented complication of anterior cervical spine surgery, may be the result of the endotracheal tube (ETT) compressing the laryngeal nerve. Audu *et al.* compared a deflation/reinflation maneuver of ETT cuffs to no manipulation after placement of the ETT to see whether the former could reduce cuff pressure and the incidence of postoperative vocal fold immobility after anterior cervical spine surgery.

The authors recruited 100 patients scheduled for anterior cervical spine surgery, and randomized them into two groups. The control group received no manipulation of the ETT cuff after confirmation of placement. For patients in the intervention group, cuff pressures were maintained at 20 mmHg or less. After placement of self-retaining retractors, the ETT cuff was deflated for 5 s and then reinflated. Cuff pressures for both groups were recorded before skin incision and after placement of self-retaining retractors. Vocal fold motion was evaluated by indirect laryngoscopy before and after surgery, and reviewed by an otolaryngologist blinded to group assignment. The reviewer graded postoperative vocal fold motion as normal, paretic, and paralyzed. In the 94 evaluable patients, the authors found a 3.2% incidence of vocal fold paralysis. The incidence of paralysis was greater in patients undergoing a right-sided as opposed to a left-sided surgical approach. The deflation/reinflation maneuver of the ETT cuff reduced cuff pressure but had no effect on the incidence of vocal fold immobility, suggesting that nerve compression by the ETT may not be the primary mechanism of injury.

■ How Does Xenon Induce Anesthesia? Rex *et al.* (page 936)

To investigate whether xenon reduces cerebral metabolic rate, like volatile anesthetics and propofol, or increases it, like nitrous oxide and ketamine, Rex *et al.* conducted a neuroimaging study in 12 healthy volunteers. The participants fasted for at least 8 h before the study period and avoided caffeine for 24 h before undergoing a positron emission tomography scan. Regional cerebral metabolic rate of glucose was measured in the awake state using ^{18}F-fluorodeoxyglucose as a tracer. After induction of anesthesia with propofol and placement of a laryngeal mask airway, six of the study volunteers received 1 minimum alveolar concentration xenon, while anesthesia for the control group of volunteers was maintained with propofol only. Then, regional cerebral metabolic rate of glucose was reassessed in both groups of participants, and quantified in 10 cerebral volumes of interest.

Compared to the group receiving propofol only, the group of six receiving xenon showed a 26 ± 7% reduction in whole brain metabolic rate of glucose and a significant decrease of regional cerebral metabolic rate of glucose in all volumes of interest. The effects of xenon on cerebral metabolism were not uniformly distributed, however. Voxel-based analyses revealed the greatest decreases in several cortical areas and the thalamus. Xenon did not induce any detectable increases in regional cerebral metabolic rate of glucose. The results suggest that NMDA antagonism, which would be expected to increase cerebral metabolism, is not the primary mechanism of anesthetic action for xenon in the brain.

■ Epinephrine's Effect on Lidocaine-induced Central Nervous System Toxicity. Takahashi *et al.* (page 984)

To assess whether epinephrine augments the central nervous system toxicity of intravenously administered local anesthetics, Takahashi *et al.* conducted a series of experiments using awake, spontaneously breathing rats. Before the study period, the animals were instrumented during general anesthesia with catheters for monitoring blood pressure and heart rate, blood sampling, and drug infusion. Microdialysis probes were also inserted, and the animals were allowed to recover in semidark cardboard boxes before experiments began.

One group of 10 rats received lidocaine alone, whereas the other group received lidocaine with epinephrine. Animals were observed for onset of convulsions, defined as tonic/clonic movements, by an author blinded to group assignment. Arterial blood samples were drawn before infusion of lidocaine, at the onset of partial muscle twitch preceding convulsions, and at regular intervals thereafter to determine plasma concentrations of lidocaine. At the onset of convulsions, oxygen was supplied to the animals' containers to prevent convulsion-induced hypoxia. The authors then quantitatively measured unbound concentrations of lidocaine in the brain's

extracellular fluid using a microdialysis technique. After completion of the experiment, brain specimens were prepared for histologic analysis to confirm location of the microdialysis probes.

The rats receiving the combination of epinephrine and lidocaine had higher mean arterial pressure and heart rates during infusion than those receiving lidocaine alone. After termination of infusion, these values decreased and there were no differences between the control and experimental groups. Rats in the epinephrine/lidocaine group had more convulsions and higher overall concentrations of unbound lidocaine than those in the lidocaine-only group. The concomitant administration of epinephrine enhanced the toxicity of intravenously administered lidocaine to the central nervous system and suggested an increased risk of seizures from intravascular injection of lidocaine if epinephrine were added.

■ Effects of Gabapentin on Postamputation Pain Studied. Nikolajsen *et al.* (page 1008)

In an attempt to reduce the incidence of chronic amputation pain, Nikolajsen *et al.* examined the effect of oral gabapentin compared to placebo in 46 patients scheduled for lower limb amputation. Surgeries were conducted with either spinal or general anesthesia, and bupivacaine was given epidurally until 2 or 3 days after amputation. Opioids were used to supplement epidural pain treatment and for treating pain when the epidural catheters were removed.

Patients received one oral capsule (300 mg gabapentin or placebo) on the first postoperative day. The dose was gradually increased until postoperative days 13–30, when patients received eight capsules (2400 mg gabapentin). If patients experienced intolerable side effects, they were allowed to stay on lower doses of the drug for the remainder of the study period. Patients who did not tolerate a minimum dose of 900 mg gabapentin were withdrawn from the study.

Incidence and intensity of stump pain and phantom pain were assessed for 30 days postsurgery using a numeric rating scale. The authors conducted patient interviews after 7, 14, and 30 days and at the 3-month and 6-month marks. Results from 41 patients were included in data analysis. Gabapentin administered in the first 30 days after amputation did not reduce the incidence or intensity of postamputation pain. It is possible that for patients in this study, who experienced high levels of preoperative pain, central sensitization of second-order neurons in the dorsal horn had already occurred and rendered them more resistant to postoperative treatment with the study drug.

Gretchen Henkel