Morphine and Tramadol Compared for Effects on Visceral Sensory Function, Gastrointestinal Motility. Wilder-Smith *et al.* (page 639)

Wilder-Smith *et al.* recruited 50 women scheduled for abdominal hysterectomy for a randomized double-blind study to compare the effects of morphine and tramadol used for postoperative analgesia. Two weeks before surgery, all patients (ASA I-III) underwent tests to assess pain scores, rectal distension and skin electrical sensation, pain tolerance thresholds, and gastrointestinal transit times. The battery of tests was repeated on the second postoperative day and 1 month after surgery. Patients were premedicated with diazepam 10 mg orally 2 h before surgery and received a routine isoflurane- or halothane-based anesthetic, with a small dose of fentanyl administered at the beginning of the procedure. Surgical incisions were infiltrated with 20 ml 0.25% bupivacaine. Patients were then randomized to receive either tramadol or morphine in a continuous infusion postoperatively. The patients in the tramadol group received a loading bolus at time of wound closure of 2 mg/kg by slow intravenous injection, then an intravenous infusion pump of 0.5 mg·kg⁻¹·h⁻¹ for the first 24 h and 0.25 mg·kg⁻¹·h⁻¹ for the next 24 h. The loading bolus of morphine at the time of wound closure was 0.1 mg/kg intravenously, followed by continuous intravenous infusion of 0.05 mg·kg⁻¹·h⁻¹ in the first 24 h and 0.025 mg·kg⁻¹·h⁻¹ in the next 24 h.

Pain intensity scores decreased similarly in patients who received either morphine or tramadol, and pain tolerance thresholds in the incisional dermatome were similar with both drugs. At the 1-month follow-up, researchers observed no visceral sensitization in either group of patients. However, during morphine infusions, pain tolerance thresholds in the shoulder (included in the dermatome receiving visceral input via the C5 spinal segment) increased in the first few postoperative days. The postoperative return of gastrointestinal and bowel function was more rapid with tramadol than morphine.

Dissecting the Mechanisms of Postintubation Laryngeal Trauma. Paulsen *et al.* (page 659)

Impaired movement of the cricoarytenoid joint (CAJ) is thought to be responsible for postintubation hoarseness and immobility of the vocal ligament. In an attempt to simulate the laryngeal complications associated with endotracheal intubation, Paulsen *et al.* intubated and extubated 37 unfixed larynges obtained from cadavers of people aged 25–89 within 48 h of death. The group specifically attempted to simulate the force of an endotracheal tube impinging on the arytenoid cartilage during intubation to reproduce the clinical result of arytenoid subluxation.

The research group conducted a series of intubation trials in the hand-held larynges. The first injury mechanism was tested in nine right arytenoids and nine left arytenoids by first introducing the endotracheal tube under view into the larynx until the tip of the left or right arytenoid was inside the lumen of the tube. The tube was then inserted with a jerky motion. A second injury mechanism was tested in another nine larynges by introducing the tube into the larynx under view until the cuff had passed the subglottic region. The cuff was filled with air, and the tube was then pulled back through the cords with a jerky motion. In the final nine larynges, the researchers manually attempted to subluxate the arytenoid joint by squeezing the CAJ and applying pressure in the anterior-inferior direction (right CAJ) and in the posterior-lateral direction (left CAJ).

Interestingly, none of the attempts using intubation or manual squeezing was successful in producing persistent subluxation of the arytenoid cartilage. Histologic analysis did show injuries in the synovial folds of some CAJs as a result of intubation attempts. Several CAJs showed chondrocyte clusters near the joint surface or a roughening of the entire cartilaginous joint surface. In a healthy state, the strong posterior cricoarytenoid ligament prevents dislocation of the arytenoid cartilage. Therefore, intubation trauma is unlikely a result of subluxation *per se*, but is more likely a consequence of the formation of a hemarthros or fractures of the joint bodies, leading to fixation of the joint surfaces in an abnormal position.

Effects of Sevoflurane and Isoflurane on Cerebral Blood Flow Examined. Matta *et al.* (page 677)

Using propofol to induce an isoelectric electroencephalogram, Matta *et al.* compared the intrinsic vasodilatory effects of 0.5 and 1.5 minimum alveolar concentration (MAC) sevoflurane and isoflurane in 20 patients who required analgesia for routine spinal neurosurgical pro-
cedures. Anesthesia was induced with 2.5 mg/kg propofol, 2 μg/kg fentanyl, and 0.5 mg/kg atracurium. A propofol infusion was used to achieve electroencephalogram isoelectricity. In addition to routine monitoring, transcranial doppler was used to measure blood flow velocity in the middle of the cerebral artery, providing an indirect measure of cerebral blood flow. Matta et al. accomplished this using a specially designed frame to hold the transcranial doppler probe in position so that the angle of insonation and vessel diameter remained constant during the study period.

All measurements were performed before surgery began. Cerebral blood flow, blood pressure, and heart rate were recorded after 20 min of isoelectric electroencephalogram, and patients were then allocated to receive either age-adjusted 0.5 MAC (0.8–1%) or 1.5 MAC (2.4–3%) end-tidal sevoflurane; or age-adjusted 0.5 MAC (0.5–0.7%) or 1.5 MAC (1.5–2%) end-tidal isoflurane. Measurements were performed 15 min later. Both agents increased cerebral blood flow at 0.5 and 1.5 MAC, but the increase was significantly less with sevoflurane. Because of its “weak” intrinsic vasodilatory action, sevoflurane is unlikely, the authors report, to cause significant increases in intracranial pressure.

Will Combining Partial Lung Ventilation and Prone Position Improve Arterial Oxygenation after Acute Lung Injury? Max et al. (page 796)

Strategies for improving hypoxemia in acute respiratory distress syndrome include partial liquid ventilation (PLV) and placing patients in the prone position. Max et al. induced acute lung injury via saline lung lavage in 21 pigs to evaluate the possible additive value of combining PLV with prone position to treat acute respiratory distress syndrome. After induction of anesthesia, gas exchange and hemodynamic parameters were determined in all animals in both supine and prone positions. Then one group of 10 pigs was assigned to receive PLV with two sequential doses of 15 mL/kg perfluorocarbon after acute lung injury, while another 11 pigs received gaseous ventilation. Gas exchange and hemodynamic parameters were determined at set time points in both groups in prone and supine positions.

Placing pigs in the PLV group in the prone position resulted in an increase of PaO₂ before and during PLV with both doses of perfluorocarbon when compared with values after acute lung injury. PLV in the supine position was only effective at the 30-ml/kg dose. In the gaseous ventilation group, PaO₂ increased when animals were prone. A significant additive improvement of arterial oxygenation was observed during combined therapy with 30 ml/kg perfluorocarbon and prone position in the PLV group compared with either therapy administered separately.

Responses of Wide Dynamic Range and High Threshold Dorsal Horn Neurons Studied. Zahn et al. (page 772)

Using their rat model, Zahn et al. designed a set of experiments for human postoperative pain to determine whether mechanically insensitive areas of the receptive field of the dorsal horn neuron could be converted to areas responsive to weak mechanical stimuli. The researchers induced anesthesia with 4% halothane in 59 adult male Sprague-Dawley rats, prepared for intravenous administration of fluids and drugs, and then performed limited laminectomies at the cervical and thoracicolumbar level. Electrodes were inserted into the C1 segment for antidromic stimulation, and action potentials of the dorsal horn neurons were recorded. Cells were classified as wide dynamic range (WDR) or high threshold (HT) depending on responses to brushing or pinching of the rat’s left foot. HT cells responded to pinch only, whereas WDR neurons responded to brushing with von Frey filaments and even more strongly to pinch stimuli.

Of 50 neurons recorded (29 WDR and 21 HT cells), only 9 showed a sustained increase in background activity after incision. In 9 of 28 WDR neurons, there was a marked decrease in threshold to von Frey filaments applied adjacent to the wound. A blunt mechanical stimulus (a 5-mm plastic disk) applied directly on the incision activated 18 of 22 WDR neurons, but HT cells were not excited by this stimulus after incision. The expansion of pinch receptive fields outside the injury area was common and similar in both WDR and HT neurons. The data suggest that conversion of mechanically insensitive areas of WDR neurons to areas responsive to weak mechanical stimuli could contribute to pain behaviors caused by punctate and blunt mechanical stimuli, but do not explain secondary hyperalgesia, the reasons for which must be examined further.