◇ This Month in

ANESTHESIOLOGY

■ Detecting Embolic Signals Intraoperatively Using Transcranial Doppler Sonography. Edmonds *et al.* (page 315)

Edmonds *et al.* used a transcranial Doppler probe to record embolic signals (ESs) to determine whether microemboli to the brain occur during total hip arthroplasty. The authors first describe a case report of a preliminary study in one patient, a 43-yr-old woman admitted for revision of a right total hip arthroplasty. A shower of ESs was noted during insertion of a cemented femoral prosthesis, at a time when the patient's mean pulmonary artery pressure increased from 13 to 20 mmHg.

The authors then prospectively studied 23 patients undergoing total hip arthroplasty. Patients were placed on the operating table in the lateral decubitus position, and the Doppler probe was secured over the middle cerebral artery. Throughout each surgery, a dedicated technician recorded ESs manually; these were verified by inspection of recorded tracings. ESs were defined as high-amplitude unidirectional transient signals lasting less than 0.1 s and associated with a characteristic chirping sound. Signal recordings were correlated with specific surgical events, such as reaming of the femoral canal and impaction of a cemented femoral component.

Three patients were excluded from final analysis; in the remaining 20 patients, in 8 patients ESs ranged in each patient from 1 to 200. In five of these patients, signals were noted during impaction of a cemented component, and in four patients, signals were noted after relocation of the hip joint. These two surgical maneuvers are also associated with intraoperative pulmonary emboli and with markers of thrombosis. One of two patients who had more than 150 ESs experienced mild chest pain and heaviness after surgery, but no further sequelae. None of the 20 patients exhibited signs of confusion or stroke after surgery; therefore, it is not clear whether these emboli are well-tolerated in the brain or whether the number of emboli is too few to result in cognitive deterioration. Further studies are needed to clarify the significance of these findings.

■ Desflurane's Bronchodilating Effects Compared with Those of Sevoflurane, Thiopental. Goff *et al.* (page 404)

Goff *et al.* hypothesized that, because of its ability to increase sympathetic nervous system activity, desflurane might offer an alternative to halothane as a bronchodilating agent after intravenous induction of anesthesia. Fifty patients scheduled for elective surgery with use of general anesthesia were recruited for the study and randomized for administration of 2.3% sevoflurane in oxygen–air (n = 20), 7% desflurane in oxygen–air (n = 20), or 0.25 mg · kg^{-1} · min^{-1} thiopental infusion (n = 10). Each group of patients contained approximately the same percentage of smokers: 42, 55, and 50%, respectively. The research team recorded baseline airway flow and pressure after thiopental induction, after tracheal intubation, and at 2.5, 5, 7.5, and 10 min after beginning volatile anesthesia. Respiratory system resistance was determined using the isovolume technique.

Instead of decreasing respiratory resistance (Rrs), desflurane increased Rrs by 5%. Thiopental also increased Rrs, by approximately 10%, but sevoflurane decreased Rrs by 15%. In patients administered desflurane, the greatest increases in Rrs occurred in those who were smokers. The authors did not explore whether the effects of desflurane on Rrs are different at lower or higher inspired concentrations than those used in this study, or whether desflurane may necessitate more time to induce bronchodilation.

■ Acute Opioid Tolerance Manifested by Increased Postoperative Pain. Guignard *et al.* (page 409)

To assess the clinical consequences of acute tolerance to remifentanil, Guignard *et al.* recruited 50 adult patients scheduled for major abdominal surgery. On the evening before surgery, patients were instructed regarding the use of a 10-cm visual analog scale, a four-point verbal rating scale, and a patient-controlled analgesia system. After induction of anesthesia with use of thiopental and tracheal intubation, patients were randomized for administration of either remifentanil titrated to autonomic responses (with desflurane kept constant at 0.5 minimum alveolar concentration [MAC]) or desflurane titrated to autonomic responses (with remifentanil 0.1 mg · kg^{-1} · min^{-1}). All patients were administered a bolus of 0.15 mg/kg morphine approximately 40 min before completion of skin closure. The total dose of remifentanil administered to each patient was recorded, as were any complications, such as respiratory depression or shivering. Behavioral pain assessment (0 = calm, 2 = intensive manifestation of pain) was performed 5, 10, and 15 min after tracheal extubation.

In the postanesthesia care unit and in the surgical unit, nurses blind to patient group assignment or intraoperative remifentanil dose assessed patient pain intensity using a visual analog scale and a verbal response scale, and they initially treated patient postoperative pain with morphine administered intravenously at a rate of 3 mg at 5-min intervals until the behavioral pain score was less than 1. Time to first request for postoperative morphine was also recorded, as were anesthetic-related complications, such as nausea, vomiting, or pruritus. Within 3 h after tracheal extubation, patients were connected to a patient-controlled analgesia device set to deliver 1 mg morphine as an intravenous bolus, with a lock-out interval of 5 min.

Patients in the remifentanil group had higher verbal response scores for pain, and greater visual analog scale scores, at 30 min and 3 h and 4 h after extubation, than did those in the desflurane group. The cumulative dose of morphine administered intravenously by nurses in the postanesthetic care unit was significantly greater in the remifentanil group; by adding the cumulative patient-controlled analgesia morphine dosages, it can be seen that patients in the remifentanil group received almost double the amount of morphine.

■ Will Combining Intravenous Zaprinast with Inhaled NO Improve Gas Exchange in ARDS? Adrie *et al.* (page 422)

To test the effectiveness of intravenous Zaprinast (M&B 22948; 2-*o*-propoxyphenyl-8-azapurin-6-one; Rhône-Poulenc Rorer, Dagenham, Essex, United Kingdom) in lung-injury models, Adrie *et al.* studied two groups of sheep in which saline lavage was used to induce lung injury. After collection of baseline hemodynamic measurements, the authors performed bilateral lung lavage with use of 0.5% (vol/vol) polyoxythylene–sorbitan monooleate in 37°C saline in 15 healthy sheep. In the first group of 10 sheep, 1, 5, 10, and 20 ppm inhaled nitric oxide (NO) was administered in random order before and after an intravenous Zaprinast infusion (2 mg/kg bolus, followed by 0.1 mg · $kg^{-1}min^{-1}$). In the second group of five sheep, inhaled NO was administered, in the same concentrations, before and after an intravenous infusion of Zaprinast solvent (0.05 M NaOH).

The authors found that intravenous administration of Zaprinast alone decreased pulmonary artery pressure but worsened gas exchange. Furthermore, the Zaprinast infusion abolished the ability of inhaled NO to improve pulmonary gas exchange and to reduce pulmonary artery pressure.

■ Histologic Damage Assessed after Forebrain Ischemia in Anesthetized Rats. Nellgård *et al.* (page 431)

Nellgård *et al.* designed a randomized study to evaluate histologic outcome in rats subjected to different durations of severe forebrain ischemia and to explore the anesthetic neuroprotection of halothane *versus* isoflurane. Sixty Sprague-Dawley rats were anesthetized with 0.75 minimum alveolar concentration (MAC) isoflurane and 60% N_2O or with 0.75 MAC halothane and 60% N_2O. Rats in the isoflurane group were subjected to either 6.5 or 8.0 min of ischemia, and rats in the halothane group endured 6.5 min of ischemia. Four days later, histologic damage was assessed. The forebrains of rats in the isoflurane group with 6.5 min of ischemia had fewer dead hippocampal CA1 neurons compared with those found in the rats in the halothane group. However, the increase in the ischemic interval to 8 min in isoflurane-anesthetized rats resulted in the same amount of neuronal necrosis as was found in the halothane-anesthetized rat group. Although they provided evidence that cerebral metabolic rate reduction can have a beneficial effect on outcome of severe brain ischemia, the results also suggest that such benefit is small.

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