

ANESTHESIOLOGY

■ Anatomical Mechanics of Successful Airway Management Studied in Conscious Subjects. Calder *et al.* (page 799)

Difficulties in performing direct laryngoscopy in patients with cervical spine disease led Calder *et al.* to explore the mechanisms involved in normal full mouth opening. The team recruited 10 female and 10 male volunteers, who were asked to perform maximal mouth opening while their heads were held in four positions: slight flexion, in which a line joining the tragus of the ear and the canthus of the eye was horizontal; the “neutral” position naturally adopted by the subject; an “extension allowed position,” in which the subject attempted maximal mouth opening without restraint on head position; and a full head extension. The authors also measured the interdental distance between the upper and lower incisions with a Willis bite gauge to determine whether this measurement also changed during mouth opening in the various positions.

In previous pilot studies, the authors found that subjects unconsciously extended their heads when asked to fully open their mouths. The research team devised a system consisting of a laser pointer attached to a cycling helmet. They and instructed the subjects to aim the laser beam at a target mark on the wall, thus restricting the extension movements.

The participants' Mallampati scores improved with extension from the neutral position. They achieved full mouth opening by extending their heads approximately 26 degrees from the neutral position. The interdental distance increased from 28 mm in a slight flexion position to 46 mm at full extension. However, when cranio-cervical extension beyond the neutral position was prevented, the subjects' mean interdental distance was 12 mm less than at full extension. The authors conclude that cranio-cervical extension is an integral part of complete mouth opening in their subjects. Even though the experiments were performed in awake subjects and anesthesiologists may be able to more fully open the mouths of anesthetized patients, the study suggests that airway management in patients with cranio-cervical rigidity may be adversely affected.

■ Does Sevoflurane Provide Myocardial Protection during Off-pump Coronary Artery Surgery? Conzen *et al.* (page 826)

Conzen *et al.* randomized 20 patients scheduled for elective off-pump coronary artery bypass surgery to re-

ceive general anesthesia with either sevoflurane or propofol. The aim was to determine whether the beneficial effects of sevoflurane shown in laboratory studies—providing protective effects on the myocardium after ischemic insult—would be reproducible in humans.

Only patients with one-vessel or two-vessel coronary artery disease suitable for repair without cardiopulmonary bypass were included. For induction of anesthesia, patients randomized to the sevoflurane group received an intravenous bolus of etomidate; in the propofol group, patients received a target-controlled infusion started at 2 $\mu\text{g/ml}$. Following tracheal intubation and for the duration of the procedures, anesthesia was maintained with either sevoflurane 2 vol% end tidal (approximately 1 minimum alveolar concentration) or propofol 2–3 $\mu\text{g/ml}$ *via* target-controlled infusion.

For quantification of tissue injury during coronary artery bypass, troponin I and creatine kinase were measured. Procalcitonin, interleukin-6, and C-reactive protein were used as indicators of systemic inflammatory reaction. In addition, standard hemodynamic variables were recorded after skin incision, during preparation of anastomosis (after approximately 50% of the ischemic period), 15 min after reperfusion, and during skin closure at the end of the procedure. The authors found that troponin I concentrations increased significantly more in patients receiving propofol than in those receiving sevoflurane. The sevoflurane-treated patients also experienced decreased inflammatory response, as measured by C-reactive protein and interleukin-6. Although the study was performed in a small group of patients, the authors believe that its results supply further evidence of the possible cardioprotective effects of sevoflurane.

■ A Neuroprotective Role for Xenon during Cerebral Ischemia? Homi *et al.* (page 876)

To further explore the neurologic benefits of xenon shown in both *in vitro* and *in vivo* experiments, Homi *et al.* evaluated histologic injury and functional neurologic outcome after transient middle cerebral artery occlusion in mice. Three groups of 21 male C57BL/6 mice, 8 weeks old, were first surgically prepared for middle cerebral artery occlusion and then randomized to one of three groups, receiving (1) 70% Xe + 30% O₂, (2) 70% N₂O + 30% O₂, or (3) 35% Xe + 35% N₂O + 30% O₂. To conserve xenon, the authors used a closed-loop ventila-

tion system custom-designed for this protocol. After an equilibration period of 15 min after initiation of the closed-loop gas delivery system, artery occlusion was achieved by inserting a 6-0 nylon blunted monofilament through the external carotid artery stump into the circle of Willis, thus occluding the middle cerebral artery.

Arterial blood samples were collected 30 min into the occlusion period; any animal with an arterial blood gas out of range for P_{CO_2} was excluded from further analysis. Reperfusion was begun after 60 min of occlusion, and after 24 h an observer blinded to group assignment evaluated the animals using a four-point overall neurologic score and determined the presence or absence of other general or focal neurologic deficits. After completion of the neurologic evaluations, the animals were reanesthetized and killed by decapitation. Their brains were frozen for histologic analysis to determine cerebral infarct size.

The researchers demonstrated that the xenon-anesthetized mice, despite having higher intraschismic serum blood glucose levels than the 70% N_2O group, had better neurologic scores and histologic outcomes. Both Xe groups had significantly lower total infarct volumes. The group receiving the 70% Xe demonstrated the most improved functional neurologic outcomes. Although limited by lack of a control, awake group of animals and a short outcome assessment period (only 24 h postprocedure), this study does seem to validate the potential neuroprotective effects of xenon.

■ Suppressing Electrocerebral Activity with Intracarotid Injection of Propofol. Wang *et al.* (page 904)

In a rabbit model, Wang *et al.* compared doses of intracarotid and intravenous propofol required to produce transient and sustained electrocerebral silence, and monitored the hemodynamic side effects of each delivery method. In the first study protocol, seven rabbits randomly received boluses of either intracarotid or intravenous propofol, and then crossed over to the alternate mode of drug delivery following a 30-min recovery period. Boluses of study drugs were administered every

30 s until electrocerebral silence was sustained for at least 10 s. Systemic and cerebral hemodynamic parameters were evaluated at four points: (1) baseline, (2) during electrocerebral silence, (3) on return of burst suppression, and (4) on return of electrocardiographic activity with amplitudes and frequency composition comparable to baseline values.

During the study's second protocol, eight animals each received intravenous or intracarotid boluses of propofol designed to maintain electrocerebral silence for 1 h. The experimental protocol was identical to that of protocol 1, except that repeat doses of the drug were administered whenever electrical spikes appeared on the electrocardiograph trace. Four animals from the intracarotid administration group were euthanized, and their brains were harvested for histologic examination to determine the extent of neuronal injury.

In protocol 1, the intracarotid injection of propofol produced electrocerebral silence at one-tenth the dose required by intravenous propofol to produce the same effect. Intracarotid propofol produced sustained silence at one-fifth the intravenous dose in protocol 2. Compared to baseline, mean arterial pressure and ipsilateral cerebral blood flow, as assessed by laser Doppler flowmetry, remained unchanged or declined transiently during electrocerebral silence with intracarotid propofol. Intravenous propofol, however, resulted in systemic hypotension and a decrease in ipsilateral cerebral blood flow. The brains harvested from the four animals in the intracarotid group showed no evidence of acute neuronal injury. The animals that received intracarotid anesthetic producing transient silence also recovered more quickly than those receiving intravenous anesthetic. This did not hold true, however, following the sustained (60-min) silence produced by intracarotid propofol injections. The study suggests that the hemodynamic effects of propofol are due largely to direct systemic effects, *e.g.*, on the heart and vasculature, rather than a consequence of the drug's action on the central nervous system. They also raise the possibility that intraarterial administration might be used for neurologic purposes with a lower risk of systemic side effects.

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