pulmonary artery catheters, and thus could not directly measure the shunt fraction. An increase of pulmonary shunt normally accompanies general anesthesia and thoracic surgery. An increased shunt fraction may be caused by the presence of an open chest, mediastinal compression of the lower lung, elevation of the inferior hemidiaphragm, and interstitial edema in lung regions lying below the level of the heart.[13] An increase of pulmonary vascular resistance in the ventilated lung or a decrease of cardiac output would also increase the pulmonary shunt during one-lung ventilation.[15] In addition, intraoperative hypoxemia may result from improper location of the double lumen tube or the accumulation of secretions.

In summary, we used quantitative ventilation/perfusion lung scans to estimate the relative perfusion and ventilation of each lung in 30 patients who subsequently underwent one-lung anesthesia for thoracic surgery. We found that the degree of preoperative perfusion and ventilation of the operative lung correlated inversely with intraoperative oxygenation during one-lung anesthesia. Unimpaired preoperative perfusion and ventilation of the operative lung appeared to be major risk factors for developing intraoperative hypoxemia during one-lung anesthesia.

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Exacerbated Spinal Neurologic Deficit during Sedation of a Patient with Cervical Spondylosis

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New or worsened neurologic deficits associated with "awake" tracheal intubation in patients with instability or narrowing of the cervical spinal canal are generally attributed to injudicious movement of the neck. However, we recently encountered a patient with cervical spondylosis who became substantially weaker after receiving diazepam and droperidol prior to intubation, despite neck stabilization, normal arterial blood pressure, and normal arterial blood gases. Consequently, we speculate that, under some conditions, sedative-hypnotics and tranquilizers can exacerbate or unmask underlying spinal cord dysfunction.

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Key words: Anesthetics, intravenous: diazepam; droperidol. Complications: neurologic. Spinal cord: weakness.

Report of a Case

A 74-yr-old woman with a long history of osteoarthritis (treated with aspirin), type II diabetes (well-controlled on NPH and CZI insulin),

and ECG evidence of an old inferior myocardial infarction presented with tingling in the hands and feet and leg weakness that had progressed over the previous 3 months to the point where she needed a walker to ambulate. Pertinent neurologic findings included 4/5 strength in both upper and lower extremities, increased leg muscle tone bilaterally, and decreased vibratory sense in the lower extremities. Deep tendon reflexes in the upper extremities were mildly exaggerated, the knee jerks were normal, ankle jerks were absent, and the plantar reflexes were bilaterally flexor. The neurologist attributed some of her symptoms and findings to diabetic neuropathy, but suspected spinal cord compression as well. In fact, nerve conduction studies and somatosensory evoked potentials were compatible with a high cervical block, and spinal cord compression was confirmed with a myelogram which demonstrated an osseous bar compressing the spinal cord at C_3-C_4. Therefore, the patient was scheduled for cervical laminectomy.

Because of her symptoms and narrow cervical canal, an "awake" tracheal intubation was planned. On physical examination, neck extension was limited to about 75% of normal, but neither extension nor rotation of the head exacerbated her symptoms. Diazepam 7.0 mg po was given for premedication. In the operating room, approximately 2.5 h later, an intravenous was started; heart rate and arterial blood pressure were monitored with ECG and an intra-arterial catheter, respectively. The patient received droperidol 1.25 mg and diazepam 7.5 mg iv over 10 min, and the oropharynx was anesthetized topically with 4% lidocaine. Orotracheal intubation under direct vision was accomplished easily on the first effort without neck extension or coughing. Nevertheless, neurologic examination after intubation revealed that, although the patient was cooperative, she was much weaker in the left hand and both lower extremities. Her systolic blood pressure during this interval was 120–140 mmHg, well within her normal range of 100–140 mmHg. Although we suspected a drug effect, surgery was cancelled. Dexamethasone 10 mg iv was administered and the trachea was extubated. She was then taken to the recovery room; strength in her extremities returned to baseline over the subsequent 2-h period.

Four days later, the patient was placed in halo traction and scheduled for surgery the next day. We again planned an "awake" intubation and used a similar premedication and intravenous sedation regimen. Diazepam 7.0 mg po was given and, when she arrived in the induction room, about 1.5 h later, strength in her extremities was both objectively (examination) and subjectively (her report) unchanged from hospital admission. She could elevate and hold both legs off the bed, flex her legs at the hip, and wiggle her toes vigorously. Her hand-grasps were moderately weak, and she could hold her arms off the bed, but only for brief periods. An intravenous and an arterial line were started and ECG leads were applied; her systolic blood pressure was 120–130 mmHg. We proceeded to sedate her with diazepan 5 mg iv, given in increments over 20 min. Repeat neurologic assessment showed that the patient was awake and cooperative, could move her legs but not elevate them against gravity, could only partially flex her legs at the hip, and was considerably weaker in the arms. Droperidol 1.25 mg iv was administered and, within about 5 min, her legs became so weak that she was virtually unable to move them; we could detect no further decrease in arm strength, however. At this point, arterial blood pressure was 103/80 mmHg and Pa_{O_2} was 86 mmHg, Pa_{CO_2} 34 mmHg, and *p*Ha 7.47. The patient was only lightly sedated and felt subjectively weaker; in fact, she asked spontaneously, "Why can't I do it (move) now? I could do it before!"

Since the patient was still in the halo apparatus, we attributed her worsened neurologic function to a drug effect and, in an effort to eliminate her anxiety, proceeded (after consulting the surgeon) to induce general anesthesia with thiopental and halothane. Her trachea was intubated easily thereafter with the aid of a fiberoptic bronchoscope. Anesthesia was maintained by inhalation of halothane and nitrous oxide, and intravenous morphine. Systolic blood pressure remained between 120–160 mmHg during the procedure. A very narrow spinal canal between C_3-C_5 was noted at surgery, and laminectomies were performed. In the immediate postoperative period, the patient was awake and could wiggle her toes, but had significant lower extremity weakness. By the forth postoperative hour, however, her arm and leg strength had returned to preoperative levels. She continued to improve and, at the time of discharge to a rehabilitation hospital 10 days later, was ambulating with the aid of a walker and her lower extremity strength was slightly better than on admission.

Discussion

Exacerbation of an existing neurologic deficit or creation of a new one is a recognized complication of endotracheal intubation in patients with unstable or narrowed cervical spinal canals. Mechanical changes (*i.e.*, flexion or extension) are usually implicated, but seem unlikely in the situation we describe. The patient could tolerate considerable neck extension and flexion preoperatively; the trachea was intubated easily (*i.e.*, without multiple efforts, head position changes, etc.) the first time; and she developed weakness before intubation while in a halo when she returned to the operating room. Therefore, other mechanisms must be considered. Spinal cord perfusion and, consequently, function could be compromised by side effects of sedation, such as hypotension or hypoxemia. This too seems unlikely, because arterial blood pressure remained normal and there was no evidence of hypercarbia (which might cause spinal cord swelling) or hypoxia, either clinically or by arterial blood gases. In addition, our patient remained quite awake during both episodes, so her increased weakness cannot be attributed to heavy sedation or lack of cooperation. Indeed, somatosensory evoked potential monitoring (which is available but not used routinely during such cases at our institution) would have added little to our clinical management at this point, because loss of movement in a cooperative patient is an ominous sign, regardless of the status of the evoked potentials. It would have been interesting, nevertheless, to have correlated neurologic examination and evoked potentials during these events and, perhaps, to have monitored somatosensory evoked potentials intraoperatively.

Since common causes of worsened neurologic function can be excluded with reasonable certainty, we hypothesize that a primary drug effect might explain the weakness our patient experienced. Drug-induced decreases in spinal cord blood flow, for example, might exacerbate cord ischemia, but this seems only a remote possibility. Diazepam and droperidol, particularly in large doses or when combined with nitrous oxide or narcotic, reduce brain blood flow,[1] but the effect of sedative doses on spinal cord blood flow, although unknown, is probably minimal. A specific neurochemical

effect of diazepam and/or droperidol on movement is a more likely possibility. Diazepam acts on spinal cord gamma amino butyric acid (GABA) receptors to enhance presynaptic inhibition and, as a result, reduces release of excitatory transmitter from primary afferents.[2] This effect probably accounts for diazepam's muscle relaxant properties (reduced gain of the stretch reflex) and efficacy in the treatment of spasticity.[2] Diazepam also depresses spinal polysynaptic reflexes, but, since higher doses of diazepam are needed to do so in animals with cord transections, this effect apparently depends partially on inhibition of a supraspinal descending facilitory system.[2] Moreover, presumably by acting on the spinal cord itself, intrathecally administered benzodiazepines produce analgesia,[3,4] and epidural administration may cause weakness. That is, although rats given diazepam epidurally showed no evidence of weakness, accidental epidural administration of diazepam (2 mg) to a patient was associated with profound weakness and sensory blockade.[5] Droperidol also has spinal neurochemical effects which could influence movement. It is a weak alpha adrenergic and dopaminergic antagonist and, as demonstrated electrophysiologically and behaviorally, these neurotransmitters are involved in motor responses.[2,6] In addition, neuroleptic binding sites exist in the spinal cord dorsal horn, and there is evidence that some neuroleptics act directly on motor neurons to reduce motor output.[6]

Thus, we speculate that the weakness our patient experienced during tracheal intubation relfects a neurochemical effect of diazepam and/or droperidol on spinal or supraspinal neuronal circuits subserving movement. For this effect to become manifest clinically is evidently rare, and probably depends on the dose and route of drug administration (oral diazepam alone did not worsen our patient's weakness), and probably requires the coexistence of other as yet undefined conditions (*e.g.*, increased drug access due to an altered blood-spinal cord barrier, or already severely compromised spinal cord function). Consequently, we do not believe one case of this sort warrants abandoning diazepam and droperidol for "awake" tracheal intubation. We do recommend, however, that a neurologic examination be performed after sedation, but prior to intubation, so that drug-induced weakness will not subsequently be confused with neck movement-related cord injury. Clinically, however, it is most essential to recognize that, if a spinal neurologic deficit occurs during sedation or intubation of a patient at risk, one must initially assume it is due to a more serious and potentially correctable problem, such as hypotension or neck hyper-extension, and act accordingly.

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Accidental Injection of Epidural Methohexital

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Occasional reports have appeared describing mistaken administration of drugs into the epidural space.[1–3] We describe the accidental injection of 1% methohexital *via* an epidural catheter.

Case Report

A 25-yr-old, 63-kg woman was to undergo laparotomy for suspected massive ovarian tumor. She was ASA class I with no significant past medical history. Apart from abdominal distension, physical and labo-

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