catheterization. Assisted ventilation represents always a great problem in pediatric sedation, particularly in neonates and infants, thus nonanesthetists need good training in airway management, practice, and protocols before drug administration. Some studies (4) recently demonstrated that patients sedated with propofol underwent significantly more airway manipulations to relieve obstruction than did patients sedated with pentobarbital (23% vs 0%, $P < 0.001$). More adverse respiratory events occurred in the propofol group than in the pentobarbital group (12% vs 0%, $P = 0.03$). Patients in the propofol group had a faster recovery profile than did patients in the pentobarbital group (34 min ± 17 vs 100 min ± 30, $P < 0.001$).

Neurodegeneration and drugs used for sedation is another debatable question. In animals, all currently available sedative drugs that have been studied, such as ketamine, midazolam, diazepam, clonazepam, propofol, pentobarbital, chloral hydrate, halothane, isoflurane, sevoflurane, enflurane, nitrous oxide, and xenon, have been demonstrated to trigger widespread neurodegeneration in the immature brain. In humans, recent preliminary findings from epidemiological studies suggest an association between surgery and anesthesia early in life and subsequent learning abnormalities, but this aspect has not yet verified for procedural sedation. Another study found that sevoflurane provided shorter induction and faster recovery times than intravenous propofol for sedation in children undergoing MRI. In this research, the percentage of MRI interruption in the propofol group was significantly higher than that in the sevoflurane group. Anyway, sevoflurane was associated with greater emergence delirium (5). In our clinical experience, sevoflurane is an ideal agent for sedation and diagnostic procedures especially in newborns and infants. Recent advances in monitoring of depth of sedation may contribute to a more precise dosing of halogens and concomitant decrease in the deleterious side effects. Sevoflurane presents many benefits with minimum inconvenience. It allows rapid inhalation induction, maintenance, and rapid recovery. It has little toxicity, and its hemodynamic and respiratory depressive effects are moderate and well tolerated. It is already widely used for sedation for a great number diagnostic procedures in children. Its use in pediatric patients could improve the management of pain and sedation.

Anyway, a recent research has demonstrated that there is a statistically significant increase in adverse events related to some drugs compared with others in ambulatory procedures. Propofol appears to have the lowest risk for adverse events. There is no statistically significant difference in the number of adverse outcomes between the administration of propofol for ambulatory surgery as an anesthetist/surgeon and the administration of propofol for ambulatory surgery as an anesthesiologist/nurse anesthetist. It remains critical that our specialty maintains the highest standards to provide safe sedation and to reduce adverse events (6). For what concern sevoflurane we are convinced that the drug need to be administered by anesthetist because of its implication on airway management.

Further clinical studies should be conducted in an attempt to provide answers to routine issues and questions, for example, how to tailor the level of sedation to the needs of the patient? how to stop it? which drug must be preferred? or what place for non-pharmacological approaches?

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Anti-NMDA receptor antibodies encephalitis

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Sir—We briefly describe the anesthetic management of a patient with a recently described, rare, and unusual

We report the presentation and management of a patient with anti-NMDA receptor antibodies. We briefly review the anesthetic implications of this condition.

acquired CNS illness because of the presence of anti-N-methyl d-aspartate (NMDA) receptor antibodies.

A 14-year-old, 65-kg female with recent history of encephalitis was scheduled to undergo a repeat diagnostic lumbar puncture (LP). She had been well until 10 months ago. After a febrile illness, she developed acute CNS symptoms secondary to encephalitis. At that time, she required an anesthetic for diagnostic LP. Anesthesia for the LP was induced with slow injection of 200 mg (3 mg·kg^{-1}) of i.v. propofol. Induction of anesthesia was rapid and produced profound hypotension as blood pressure (BP) fell from 110/70 to 60/20 mmHg. The BP remained around 80/40 mmHg for 15 min despite i.v. isotonic fluid boluses. The LP confirmed the presence of anti-NMDA receptor antibodies in the CSF. She was treated with steroids and immunosuppressive therapy with considerable symptomatic improvement. She was discharged after a month of treatment and was advised to continue oral prednisolone until further follow-up. She remained well until 6 months ago and then started having worsening of her neurocognitive function.

Ten months after her initial illness, she presented with disinhibition, worsening aggressive behavior, and fluctuating level of consciousness. She required sedation and physical restraints. Her current medications included quetiapine fumarate (Seroquel^{TM}; AstraZeneca Pharmaceuticals LP, Mississauga, ON, Canada) 50 mg twice daily, sodium valproate 250 mg thrice daily, and clonazepam 0.5 mg twice daily. Her recent laboratory investigations were within normal range. On examination, it was found that she was a moderately obese teenager (65 kg), conscious, oriented, and generally cooperative with unremarkable general and systemic examination. Her mood was labile, and she could suddenly, without apparent cause or stimulus, become angry, agitated, or just active and excessively verbose.

Diagnostic LP was again to be performed under anesthesia. Routine medications were continued. After standard monitors were attached in the operating room, i.v. access was obtained. She remained calm and cooperative. Anesthesia was induced with 50 mg of propofol (<1 mg·kg^{-1}). The anesthetic was then maintained with the patient spontaneously breathing sevoflurane (0.5–0.7 MAC in an air–oxygen mixture) through a facemask using the standard circle breathing system. Her BP and heart rate remained within 10% of baseline throughout the procedure that lasted approximately 15 min. She woke up comfortably, and the rest of her recovery was without any adverse incident or event.

Anti-NMDA receptor antibodies have been recently described in patients with severe forms of encephalitis (1). While many of the cases have been described in adults, the youngest patient reported in this series was a 5-year-old (1). Patients may present with psychiatric symptoms, memory problems, seizures, unresponsiveness, decreased consciousness, dyskinesias, autonomic instability, and/or hypoventilation (1). These are considered to be part of a constellation of paraneoplastic neurological syndromes and associated with ovarian teratomas in over half of the patients (1,2). The anti-NMDA receptors antibodies have previously been implicated in the neuropsychiatric dysfunction in SLE (3). The condition is potentially treatable with steroids or immunoglobulins, and patients may recover completely (4). Those associated with tumors may have better prognosis as surgical removal of the tumor often results in complete recovery (5). Our patient had no identifiable tumor and, unfortunately, had a relapse with prolonged period of hospitalization and poor response to treatment.

To our knowledge, there are no previous published reports that describe the effects of the presence of anti-NMDA antibodies on anesthetic requirements. The NMDA receptors play an important role in the mechanism of action of many anesthetic drugs. Centrally acting NMDA antagonists (such as the well-known ketamine) decrease anesthetic requirements in analogous animal models (and also in clinical practice) (6). Even propofol may also have some actions mediated via the NMDA receptors (7). The potential for exaggerated responses to i.v. anesthetics observed during this patient's first anesthetic can therefore be explained anecdotally. Also, the presence of antibodies to the NMDA receptors may explain the preoperative presentation that will be familiar to anesthesiologists having observed patients who have received ketamine.

Our experience, although limited to this one patient; suggests that the presence of anti-NMDA antibodies may increase the sensitivity of patients to some anesthetic agents. Overall, this is a fascinating, coexisting illness with hitherto unknown, but possible serious anesthetic implications that require further clinical evaluation.

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Anesthetic consideration for radiofrequency ablation of a suspected paraganglioma metastasis in a child

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Sir—The need to pretreat patients with pheochromocytoma or paraganglioma (1) as well as children with neuroblastoma and documented excessive catecholamine secretion (2) prior to surgical resection is well established. Pretreatment is typically performed with alpha and beta adrenergic receptor blockade (α- and β-blockade). Massive catecholamine release and hypertensive crisis have also been reported during chemotherapy of neuroblastomas (2) or during radiofrequency ablation (RFA) of tumors of or near the adrenal (3) and of pheochromocytoma metastases (4–6), and pretreatment of these patients with antihypertensives prior to RFA has been recommended.

A 10-year-old boy weighing 41 kg had had a malignant retroperitoneal paraganglioma resected and was diagnosed with the associated germline mutation of the gene coding for the subunit B of succinate dehydrogenase (SDHB). He was scheduled for RFA of a presumed paraganglioma metastasis. Because of the available literature and expert recommendation, perioperative adrenergic blockade was discussed. On review, his blood pressure was usually around 120/80; he reported occasional palpitations but was otherwise asymptomatic for catecholamine release. A $1.2 \times 0.8 \times 0.8$ cm lesion in the mid-diaphysis of the left femur was visible on MRI, CT, and 2-[18F]-fluoro-2-deoxy-D-glucose (FDG) positron emission tomography (PET), but not on I-123-metaiodobenzylguanidine (MIBG)- or 6-[(18)F]-fluorodopamine (FDA) PET scan. His levels of chromogranin A, catecholamines, metanephrine, normetanephrine, and vanillylmandelic acid were not elevated. Finally, his anesthetic course during a difficult resection of the retroperitoneal paraganglioma prior to histologic diagnosis and without antihypertensive pretreatment had been free of hemodynamic signs of catecholamine release. Based on these data, it was agreed that he had a low likelihood for a catecholamine producing metastasis and it was decided to proceed with RFA without antihypertensive pretreatment.

The patient was premedicated with midazolam, anesthesia was induced with propofol, and he was intubated following muscle relaxation with vecuronium. Anesthesia was maintained with sevoflurane and fentanyl 2 $\mu g \cdot kg^{-1} \cdot h^{-1}$. Large bore venous and intraarterial access were established. α- and β-blocker (phentolamine, esmolol, labetalol) and vasodilator infusions (sodium nitroprusside) were prepared and in line. The patient was hemodynamically stable during induction, intubation, esophagogastroduodenoscopy (performed to rule out gastrointestinal stromal tumor associated with SDHB mutation), dynamic CT scan, needle placement into the femur, and biopsy of the femoral lesion by the radiologist. Heart rate and blood pressure increased mildly from 80 to 110 min^{-1} and from 80/40 to 110/60 mmHg during RFA which was performed to 90°C for 6 min each in three adjacent locations to cover the length of the lesion. No vasoactive substances were required. Catecholamine levels were sent and the patient was extubated at the end of the procedure. He had increased blood pressure to 150/80 mmHg and tachycardia to 140 min^{-1} in the recovery room. He received additional analgesia with hydromorphone and was transferred to the ward where normotension and normalization of his heart rate were documented for 24 h before discharge home. Catecholamine levels returned normal and the biopsies were free of tumor, confirming the absence of a catecholamine producing metastasis.

As with manipulation during surgical resection, acute tissue necrosis during RFA of catecholamine-secreting tumors or their metastases carries the risk of massive catecholamine release with development of acute and severe hypertension and justifies the same perioperative management including antihypertensive pretreatment, invasive monitoring, and immediate availability of vasoactive agents. Our patient had a history of a histologically proven paraganglioma, a suspicious lesion by MRI, CT, and FDG PET and an increased risk for metastatic disease based on his SDHB mutation (1). Although positive FDG PET in the presence of negative MIBG and FDA PET scans is compatible with pheochromocytoma metastasis (7), the catechol metabolites metanephrine and normetanephrine levels would be expected to be elevated in a tumor producing increased amounts of catecholamines which if not secreted would undergo intracellular metabolism. Because there was no evidence of increased catecholamine synthesis or secretion by the femoral lesion, and because the patient had not shown any signs of catecholamine release during his initial surgery which had required several hours of tumor manipulation, we decided to proceed without pretreatment, but with close invasive