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Narcolepsy and Anesthesia

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NARCOLEPSY is a sleep disorder characterized by excessive daytime sleepiness, involuntary daytime sleep episodes, disturbed nocturnal sleep, sleep paralysis, and cataplexy (sudden loss of muscle tone without loss of consciousness).[1,2] Its treatment includes stimulants (e.g., amphetamines), tricyclic antidepressants, and behavioral therapy.[3,4] Anesthetic implications include increased sensitivity to anesthetic agents, increased risk of postoperative apneic episodes,[5] and interactions with treatment medications.[6] We report a case in which propofol and nitrous oxide were used to successfully anesthetize a patient with a history of narcolepsy and several episodes of prolonged emergence from inhalation anesthesia.

Case Report

A 51-yr-old woman required wide local excision of a recurrent right thigh mass during general anesthesia. The patient had a life-long history of sleepiness that worsened during the last 8 yr. Four years previously, she underwent evaluation at a sleep disorder center, which included a Multiple Sleep Latency Test, and was diagnosed with narcolepsy characterized by hypnagogic hallucinations, sleep paralysis, and sleep attacks. Family history was positive for daytime sleepiness, although to a lesser extent. The patient reported facial swelling after taking prochlorperazine, itching with use of penicillin, and hallucinations after morphine administration. She denied alcohol or tobacco use.

The patient underwent vaginal hysterectomy with general anesthesia in 1979, in which her emergence time was approximately 8 h. In 1983, she underwent removal of a right thigh lipoma during general anesthesia; after 30 min general anesthesia, she spent several hours in the postanesthesia care unit (PACU), and awoke 9 h later. In 1988, she underwent bilateral breast reduction with general anesthesia. The surgical procedure was 9 h, and the patient awoke 10 h later. In 1989, after a failed attempt to produce spinal anesthesia, she underwent tumor removal from the right leg during general anesthesia induced with thiopental, gallamine, and succinylcholine. Maintenance consisted of isoflurane and nitrous oxide. The patient also received 20 mg intravenous metoclopramide and 0.625 mg droperidol. She did not

receive narcotics, although she was administered 4 mg midazolam in divided doses immediately after the attempted spinal anesthesia. After 90 min general anesthesia, she remembered being in the PACU awake but unable to move. She remained in the PACU 8 h after the surgical procedure and awoke 4 h after leaving the PACU. In 1995, the patient underwent a 2-h low back surgery for a herniated disc during general anesthesia. She was extubated in the PACU and remained awake but unable to move for an additional 10 h. All these surgical and anesthetic procedures took place before the patient was diagnosed and treated for narcolepsy; but the anesthesiologists were warned of the history of prolonged emergence time after general anesthesia. The patient was prescribed diclofenac, dextroamphetamine, alprazolam, and a mixture of butalvital, acetaminophen, and caffeine. She had a history of migraine headaches, but denied loss of consciousness and spontaneous cataplectic episodes.

At examination, her blood pressure was 131/73 mmHg and her heart rate was 97 beats/min. Physical examination was unremarkable. The patient received only 20 mg dextroamphetamine by mouth preoperatively. Routine monitoring devices were placed, and the patient breathed 100% oxygen by face mask. Intravenous anesthesia was induced with 100 mg lidocaine, 200 mg propofol, and 50 mg rocuronium, followed by tracheal intubation. Anesthesia was maintained with an intravenous infusion of propofol ($100-150 \ \mu g \cdot kg^{-1} \cdot min^{-1}$), and ventilation was maintained with a 70:30% nitrous oxide:oxygen mixture. Muscle relaxation was accomplished with divided doses of intravenous rocuronium. The patient remained stable during the 2-h 30-min surgical procedure. Intravenous ondansetron (4 mg) and 30 mg ketorolac were administered before completion of the procedure. Muscle relaxation was reversed with 3.0 mg intravenous neostigmine and 0.6 mg glycopyrrolate. Fifteen min after the surgical procedure was completed, the patient was able to open her eyes and maintain a head lift for 5 s. She was extubated and transferred to the PACU, where she awoke 1 h later, alert and oriented. She received 50 mg intravenous meperidine for pain, and was transferred to the unit 1 h 40 min after extubation

Discussion

Narcolepsy is a sleep disorder that occurs in approximately 1 in 4,000 persons in the United States. Onset usually occurs during adolescence. Once established, the condition becomes chronic, without remission.[1,3] Narcolepsy appears to have a genetic basis and has been linked to human leukocyte antigens DR2 and DQwl.[2] Other factors, such as viral and bacterial agents, abrupt changes in awake–sleep cycles, illness, accidents, stress, drug use, and hormonal changes may act as triggers and determine whether narcolepsy will develop in a person who is genetically predisposed.[7,8]

Diagnosis necessitates the presence of the narcolepsy tetrad, consisting of excessive daytime somnolence, cataplexy, hypnogogic hallucinations at sleep onset, and sleep paralysis (an awareness that voluntary muscles are paralyzed, coincident with the onset of sleep).[2] The differential diagnosis should include sleep-disordered

breathing (e.g., sleep apnea), substance abuse, infections (e.g., mononucleosis), depression, sleep deprivation or circadian disorders, and Klein-Levin syndrome.[3] Treatment is symptomatic; sleepiness is treated with stimulants such as amphetamines, although use is limited because of side effects, patient intolerance, and the possibility of drug dependence. Other medications, such as methylphenidate and pemoline are options that have fewer side effects. Tricyclic antidepressants are the best management option for cataplexy, hypnogogic hallucinations, and sleep paralysis because they suppress rapid eye movement sleep. The serotonin reuptake inhibitor fluoxetine has been effective in the management of narcolepsy.[3,4] Some patients benefit from behavior modifications, such as therapeutic naps, group support, a regular sleep–awake schedule, and avoidance of caffeine and nicotine.[7]

Little information is available regarding the anesthetic treatment of these patients. Theoretically, prolonged emergence and postoperative hypersomnia after general anesthesia are serious concerns, although incidence and precipitating factors have not been researched seriously. Patients with narcolepsy seem to be at higher risk of having apneic episodes, cataplectic spells, and sleep paralysis when recovering from inhalation anesthesia.[5] However, following recommendations such as avoiding sedative premedication, continuing medical therapy on the day of surgery, and being aware of increased sensitivity to anesthetic agents may suffice to permit an uneventful outcome,[9] as we observed in this case. Spinal anesthesia is an attractive option; however, cataplectic spells have been reported in a narcoleptic patient who underwent spinal anesthesia.[6]

No clinical reports show the use of propofol in patients with narcolepsy. Propofol has a rapid onset of action and results in prompt patient recovery without residual sedation; probably because of its fast and extensive hepatic metabolism and rapid clearance.[12] Nausea and vomiting are concerns after anesthesia. Ondansetron has not been investigated in this particular group of patients; however, ondansetron retards the occurrence and shortens the duration of pinch-induced catalepsy in mice.[10] Our patient did not receive opioid analgesics during anesthesia because of the potential and theoretical risk of inducing postoperative apneic spells. She tolerated meperidine for postoperative analgesia without the untoward events she experienced after induction of previous anesthetics.

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Failure to Detect CO₂-absorbent Exhaustion: Seeing and Believing

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REBREATHING during general anesthesia is defined as an increased inspired pressure of carbon dioxide above 2 mmHg.[1] We report a case of rebreathing, with an unusual capnographic pattern caused by undetected exhaustion of the carbon dioxide absorbent.

Case Report

A 41-yr-old, 55-kg woman presented for nasal septoplasty, bilateral ethmoidectomies, and nasal scar revision. The patient's medical history was significant for anxiety disorder and prior cocaine abuse. Medication included loratidine and amoxicillin. General anesthesia was induced with 425 mg thiopental and 150 μg fentanyl. The patient received 120 mg succinylcholine to facilitate tracheal intubation. Place-

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ment of the endotracheal tube was confirmed by auscultation and the presence of end-tidal carbon dioxide on the capnograph. Positive-pressure ventilation was then instituted using the ventilator on our anesthesia machine (Narkomed 2B; Dräger Medical, Inc., Telford, PA). The initial end-tidal carbon dioxide value (40 mmHg) and the capnogram tracing were within normal limits, with an inspiratory carbon dioxide of zero. Fresh gas flow was 3 l/min (2:1, N₂O:O₂). Approximately 20 min into the procedure, the inspired end-tidal carbon dioxide value increased and the capnogram was noted to be abnormal (fig. 1). Shortly thereafter, the inspired carbon dioxide alarm sounded.

The carbon dioxide-absorbent canisters with soda lime were inspected visually. Both the upper and the lower canisters were not unduly warm, and no temperature difference was noted between them. Carbon dioxide absorbent in the upper compartment had a bluish cast, suggesting exhaustion of the carbon dioxide-absorbent granules. No color change was seen in the lower compartment (fig. 2). Because there was a dramatic color difference between the upper and lower canisters, the possibility, that the carbon dioxide absorbent was exhausted in both canisters seemed unlikely. Other causes of the abnormal tracing were sought (e.g., valve malfunction).

The expiratory and inspiratory valves were inspected and changed, without improvement in the capnogram. Rebreathing continued to be apparent, and the fresh gas flows were increased. The patient's lungs were manually ventilated while a machine exchange was performed. Although there was a decrease in carbon dioxide rebreathing with an increased fresh gas flow, we decided that changing the anesthetic machine would be the safest course of action because we were unsure as to the origin of the inspired carbon dioxide. The patient remained stable throughout this period, with normal blood pressure and a pulse oximetry reading of 100%. The capnogram was normal after the machine exchange. The remainder of the procedure was uneventful, and the patient was discharged the following day.