Delirium During Emergence From Anesthesia: A Case Study

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Table 1 Stages of anesthesia[1]

Stage	Description
I	Amnesia, induction of anesthesia to loss of consciousness
II	Delirium, excitation, potential for vomiting, laryngeal spasm, hypertension, tachycardia, uncontrolled movements, dilated pupils
III	Surgical anesthesia, constricted pupils, regular respiration, adequate anesthetic depth, prevention of hypotension and tachycardia, absence of movement
IV	Overdosage; shallow or no respiration; dilated, nonreactive pupils; hypotension

Note: The stages of anesthesia are not always obvious when modern anesthetic agents are used. The stages are used only as a guide for recognition of wakefulness from the anesthetized state.

Emergence is defined as the transition from the sleep state to full consciousness. Emergence from general anesthesia usually is a smooth and uneventful transition through the 4 stages of anesthesia (Table 1). Although uncommon (<10% of surgical cases), delirium may arise during emergence from anesthesia.[1]

Critical care nurses working in the postanesthesia care unit (PACU) should be aware that emergence delirium may occur after all types of anesthetic techniques. The focus of this article is delirium after anesthesia.[2] The case study deals specifically with the unusual behavior of a patient who received intravenous sedatives, not a general anesthetic, for plastic surgery.

Emergence delirium occurs most often in elderly patients and patients with psychiatric conditions such as depression or drug dependency. It may be associated with clinical conditions such as organic brain disease or with the medications used to treat the underlying conditions. Emergence delirium may occur in as many as 10% to 15% of elderly patients.[3,4] Emergence delirium also occurs in children and patients who are anxious about surgical findings and outcomes.[1,5] Patients who express marked fear of disfigurement or of a possible diagnosis of cancer preoperatively may have signs and symptoms associated with unusual emergence from anesthesia.[1,5]

Causes

Emergence delirium after anesthesia can be caused by a variety of physiological and pharmacological factors.[1,5] Before any intervention is started to manage emergence delirium, physiological causes must be investigated. Arterial hypoxemia and hypercapnia are potential causes of delirium or agitation after anesthesia. If sedating agents are given, respiratory depression may become severe to the point of apnea. If a patient's vital signs are stable and his or her oxygen saturation is normal, other physiological factors associated with untoward emergence from anesthesia should be considered: hypothermia, hypoglycemia, electrolyte imbalance, arterial hypoxemia, sepsis, embolism, sensory deprivation, and sensory overload.

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Central anticholinergic syndrome can produce delirium after anesthesia. This syndrome is caused by blockade of muscarinic cholinergic receptors in the central nervous system. Stimulation of the muscarinic receptors produces decreased heart rate and contractility, bronchial constriction, salivary secretions, and intestinal and bladder contraction and relaxation of sphincters.[4] The undesirable delirium or prolonged sleepiness that occurs after anesthesia is associated with use of atropine and scopolamine.

Pharmacological factors specifically associated with emergence delirium include the residual effects of several anesthetic medications, including droperidol, metoclopramide, benzodiazepines, opioids, ketamine, and atropine. Droperidol, a dopamine receptor antagonist used to prevent nausea and vomiting, may cause anxiety or prolonged sedation. Additionally, droperidol potentiates central nervous system depressants such as benzodiazepines and narcotics.

Metoclopramide, used to promote gastric emptying and to prevent emesis, is also associated with extrapyramidal effects such as involuntary muscle movements. This reaction is due to passage of the drug into the central nervous system, resulting in blockade of dopamine receptors.

Benzodiazepines and opioids occasionally cause prolonged recovery from anesthesia, particularly with higher doses.[1,5] Depression of the central nervous system by these drugs is more common with delayed awakening from anesthesia but must also be considered when emergence delirium occurs, particularly if the patient is using other central nervous system depressants.

Ketamine, a phencyclidine derivative, produces dissociative anesthesia that is defined as marked amnesia and profound analgesia.[1,5] Emergence delirium after ketamine administration may result in extreme restlessness and agitation. Hallucinations and unpleasant dreams may occur postoperatively. Combining benzodiazepines with ketamine lessens these untoward effects.

Signs and Symptoms

Signs and symptoms of emergence delirium or agitation after anesthesia include excitement and alternating periods of lethargy followed by excitement and disorientation. Inappropriate behavior such as screaming, kicking, and use of profanities also may occur. Also, the patients generally do not respond appropriately to commands.[1,4,6,7]

Treatment

Treatment of emergence delirium generally involves treatment of the signs and symptoms. The primary goal is to rule out possible physiological or pharmacological causes of the signs and symptoms (Table 2). Supplemental oxygen, fluid and electrolyte replacement, and adequate analgesia may be appropriate once the cause of the signs and symptoms has been determined. Medications used for emergence delirium include benzodiazepines for calming if the delirium is severe and antipsychotics, such as haloperidol and physostigmine, which reverse delirium associated with anticholinergics often used during general anesthesia.[6] Medications such as naloxone and flumazenil may also be used to reverse oversedative effects that may be due to narcotics and benzodiazepines used intraoperatively.[1,4,6,8]

Case Study

A 47-year-old woman with no known systemic disease was admitted to the PACU after a 5-hour facelift procedure during which she was given intravenous sedatives with monitored anesthesia care. Vital signs on admission included a blood pressure of 148/77 mm Hg, a heart rate of 90/min, spontaneous respirations of 16/min to 20/min, and oxygen saturation of 99%. The patient was sleeping and was nonresponsive to verbal or painful stimuli.

Preoperatively, no medications were given. Intraoperatively, the patient received 2 mg of midazolam intravenously, 100 µg of fentanyl intravenously, 50 to 75 µg/kg per minute of propofol via infusion, 0.625 mg droperidol intravenously, and 3 L/m of oxygen via nasal cannula. Her vital signs remained normal throughout the 5-hour operation.

After 30 minutes in the PACU, the patient remained somnolent and could not be aroused. Suddenly she began thrashing in the bed, screaming and kicking, and seemed uncontrollable. This behavior lasted approximately 2 to 3 minutes and was followed by somnolence lasting 10 to 13 minutes. Again, she began shouting profanities, then relaxed momentarily. Her demeanor was irrational and inappropriate. The certified registered nurse anesthetist and surgeon were notified of the change in status and were asked to assist. During an evaluation, the patient was unresponsive to commands. Vital signs were unchanged, and oxygen saturation remained at 98% to 100%, confirming an absence of hypoxemia.

Table 2 Causes of emergence delirium[1,5]

Physiological	Pharmacological
Hypoxemia	Ketamine
Hypercapnia	Droperidol
Hyponatremia	Benzodiazepines
Hypoglycemia	Metoclopramide
Intracranial injury	Atropine
Sepsis	Scopolamine
Alcohol withdrawal	
Airway obstruction	
Gastric dilatation	
Full bladder	
Pain	
Hypothermia	
Sensory overload	
Sensory deprivation	

The patient was given 1 mg of intravenous flumazenil and 0.4 mg of intravenous naloxone slowly over 5 minutes, but her behavior did not change. She continued to have intermittent episodes of the previously described inappropriate behavior that lasted approximately 1 to 3 minutes and were followed by rest periods of 1 to 15 minutes.

Preoperatively, the patient had said that she did not use prescription medications or street drugs. She also said that she did not use herbal remedies or diet drugs. In our practice, inquiry about the use of controlled and noncontrolled substances is standard and often extensive, depending on the patient's history. This patient said that she drank alcohol socially. Because of her unexplained behavior in the PACU, her family was called. Her husband disclosed that she was using the antidepressants fluoxetine and citalopram as well as lorazepam for sleep, all doses unknown. The bottles were sitting on her bedside table at home. Additional social history was significant for marital problems, including physical and emotional abuse and extramarital affairs. The family, including the husband and stepdaughter, expressed no surprise about the patient's unusual behavior during emergence from anesthesia. In fact, they shared personal psychosocial circumstances of the patient's lifestyle, including a "fight" with the husband the night before surgery.

After approximately 2 hours, the patient was awake and calm. Her vital signs remained stable, and she was deemed ready for discharge. For her safety, however, she was admitted to the hospital for observation and a psychiatric consultation.

Discussion

The patient in this case study experienced an unusual emergence from intravenous sedation. A thorough literature search revealed little information on emergence delirium occurring after intravenous sedation.

Hypoxemia, hypercapnia, and other physiological factors were ruled out because of her sustained stable vital signs and full spontaneous respiration. Steps to improve cognition for this patient included reversal of benzodiazepines and narcotics, neither of which changed the patient's demeanor. Constant monitoring of vital signs revealed an oxygen saturation of 99% to 100% throughout the entire perioperative cycle. Heart rate, blood pressure, respiratory rate, and body temperature were all within normal limits. Supportive nursing care included using warm blankets and staying close to the patient to protect her from injuring herself and others. Constant communication between the nursing staff, the anesthesia provider, and the surgeon provided a solid, safe approach to caring for this challenging patient.

When the unusual emergence from anesthesia occurred, communicating with the patient's family generated a wealth of information. Information was obtained about her use of antidepressants and sleeping pills as well as a strongly adverse psychosocial history. The interaction of anesthetic medications with the antidepressants, whose use was unknown at the time of surgery, along with psychosocial dysfunctional issues, markedly increased the likelihood of emergence delirium for this patient. The actions of benzodiazepines, narcotics, and central nervous system hypnotics were thought to have potentiated central nervous system effects of the patient's use of antidepressants.[8]

For personal reasons, some patients do not reveal their use of certain medications. Yet, the importance of obtaining a complete drug history is readily apparent in this case. In our outpatient surgery setting, a registered nurse, a surgeon, and a nurse anesthetist each elicit a drug history from each patient. Some patients reveal some information, some reveal no information, and some provide complete disclosure to 1 or all members of the healthcare team.

Delivery of anesthetic drugs tailored individually for each patient provides the safest form of care for surgical patients. When patients willingly withhold information, the entire perioperative team may see untoward outcomes, as occurred with this patient. Our goal to obtain the most complete history from each patient remains a priority.

Not all patients awaken smoothly and uneventfully after surgery and anesthesia. For this reason, all nurses who work with patients recovering from anesthesia should have a working knowledge of emergence delirium.

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