

CASE REPORTS

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Factitious Disorder as a Cause of Failure to Awaken after General Anesthesia

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FAILURE of a patient to awaken after general anesthesia may involve a complex array of etiologies that includes several life-threatening disease states.¹⁻³ The potential for patient harm requires the anesthesiologist to rapidly diagnose and, if needed, treat (or arrange treatment for) any underlying pathology. Although factitious disorders are rarely the cause of delayed emergence, patient unwillingness to awaken after anesthesia is a recognized occurrence that is, by necessity, a diagnosis of exclusion.¹

We report two patients who failed to progressively awaken after general anesthesia for minor surgical procedures. Both patients had normal neurologic examination results, and serious complications related to the surgery and the anesthetic were ruled out. One patient had a history of psychiatric disorders; the other did not. In both, the failure to awaken was believed to be secondary to factitious disorders.

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Case 1

A 36-yr-old, 53-kg woman, ASA physical status 2, was scheduled for general anesthesia for an intranasal ethmoidectomy with middle meatal antrostomies for recurrent sinusitis. She had had juvenile onset diabetes mellitus for 21 yr with resultant retinopathy and peripheral neuropathy. Preoperative medications included Lente insulin 10 U subcutaneously every 8 h, terfenadine, and pseudoephedrine.

Seven years before surgery, the patient exhibited prolonged postoperative paresis after straightforward removal of a ganglion cyst

under general anesthesia. An underlying organic or pharmacologic disorder was never identified. Four years before surgery, she was diagnosed with a conversion disorder. This was based on three episodes of burning pain of her right extremities accompanied by unexplained, self-limited paresis. At that time, her Minnesota multiphasic personality inventory (MMPI)⁴ evaluation exhibited features consistent with histrionic behavior. Specifically, she demonstrated a tendency to exaggerate signs and symptoms of disease.

After placement of routine physiologic monitors, the patient was sedated with 6 mg intravenous morphine, and anesthesia was induced with 450 mg intravenous thiopental. After neuromuscular block with 80 mg intravenous succinylcholine, the trachea was intubated with a 7.0 mm-ID cuffed endotracheal tube. Ventilation was mechanically controlled, and anesthesia was maintained with inhaled enflurane and nitrous oxide in oxygen for the duration of surgery.

The operative course was uneventful. On conclusion of the surgical procedure, the patient breathed spontaneously with a normal respiratory rate and an adequate tidal volume (350 ml, estimated). The end-tidal enflurane concentration, measured by mass spectrometry, was 0.18%. Although the patient would not respond to verbal commands, she demonstrated clinically the ability to protect her airway by vigorous, repeated swallowing as well as purposeful motor activity (right upper extremity movement presumably directed to self-extubate her trachea). Accordingly, the trachea was extubated, by the anesthesiologist, without complication.

The patient was moved to a transport cart, and supplemental oxygen was supplied ($FiO_2 = 1.0$) for the duration of transfer to the post-anesthetic care unit (PACU). Once in the PACU, routine postoperative monitors were applied, and oxygen was delivered by a face shield ($FiO_2 = 0.4$). The patient continued to ventilate normally, however, she would not respond to verbal prompts, touch, or pain (sternal rub). Her hemodynamic variables remained stable. Pupil size was within normal limits, and funduscopic examination did not demonstrate the presence of papilledema. Serum electrolytes, blood glucose, and body temperature were normal. Hemoglobin oxygen saturation (measured by pulse oximetry) was >96% throughout the postoperative period.

After 90 min in the PACU, the staff anesthesiologist became concerned that the patient showed no signs of progressive awakening, and an underlying cause could not be identified. In response to a request for consultation, a neurologist performed a detailed neurologic examination and found that, although the patient was unresponsive, her neurologic status was otherwise within normal limits. At the conclusion of his examination, the neurologist thought he could see the patient looking surreptitiously at her surroundings when no one stood at her bedside. He repeated the neurologic evaluation with the same result: *i.e.*, the patient was completely unresponsive to noxious stimulation. He observed the patient closely as

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he wrote his note and, a few moments later, discovered that she covertly glanced around the PACU. He approached the patient again and injected ice water into her left ear, consistent with the vestibulo-ocular reflex test. The patient sat bolt upright on her cart and exclaimed, "What did you have to do that for?" as she demonstrated marked rightward fast-phase nystagmus and experienced nausea. Her neurologic status was closely monitored during her 72-h hospitalization and was normal. She was discharged from the hospital without neurologic deficit and was neurologically intact when seen on routine follow-up 2 weeks later.

Case 2

A 32-yr-old, 80-kg woman, ASA physical status 2, presented for a zygote intrafallopian tube transfer procedure for correction of infertility. Thirteen years before this surgery, she had had a left nephrectomy as a donor. Two years later she underwent partial pancreatectomy, also for donation, with subsequent splenectomy for bleeding complications postoperatively. The patient had been diagnosed 4 yr before surgery with Hashimoto's thyroiditis, treated with levothyroxine. Additionally, she was receiving erythromycin for prophylaxis of recurrent streptococcal pharyngitis. She had undergone MMPI testing several months before the current hospital admission to evaluate abnormal coping skills associated with the inability to have children. The results were consistent with features of histrionic behavior.

Routine physiologic monitors were placed, and the patient was sedated with 100 μ g fentanyl and 2 mg intravenous midazolam. Induction of anesthesia was accomplished with 180 mg intravenous propofol. After administration of 120 mg intravenous succinylcholine, the trachea was intubated with a 7.0 mm-ID cuffed endotracheal tube. Anesthesia was maintained with inhaled isoflurane and nitrous oxide in oxygen for the duration of the case.

Surgery proceeded uneventfully. The patient awakened following cessation of isoflurane (end-tidal concentration < 0.1%) and demonstrated adequate respiratory rate and tidal volumes (300 ml, estimated). The trachea was extubated without complication. She was fully alert, cognizant, and conversant on transfer to the outpatient recovery unit. Routine postoperative monitors were applied, and supplemental oxygen was delivered by face shield ($FiO_2 = 0.4$). Her body temperature measurements were within normal limits, and oxygen saturation, measured by pulse oximetry, was >97% throughout the postoperative period.

Ninety minutes after transfer to the outpatient recovery unit, while speaking on the telephone, the patient was observed to become suddenly flaccid and fall backward onto her bed. Further examination revealed that she was unresponsive to verbal commands. Hemodynamic variables and neurologic examination results, performed by one of us (C.H.L.), were otherwise within normal limits, with the exception that verbal and tactile stimulation would not elicit a response. Pupil size was normal bilaterally. The patient was emergently transferred to the PACU to obtain more specialized nursing care and closer observation.

The supervising staff anesthesiologist (C.H.L.) believed that, in the absence of additional neurologic, hemodynamic, and respiratory findings, this unusual presentation of altered consciousness could be attributed to a factitious disorder. Within the patient's field of vision, he administered an intravenous bolus of 1 ml of lactated Ringer's solution while declaring that this particular case called for the

use of a potent "experimental anesthesia reversing agent." Thirty seconds after this injection, the patient responded by moving slightly and speaking unintelligibly. A second milliliter of intravenous lactated Ringer's solution, coupled with further elaboration on the strength of this "medication," resulted in immediate, complete awakening.

The patient professed no memory of the syncope-like episode. She was monitored for another 90 min in the PACU without further complication. No neurologic deficit was found before hospital discharge the next day, nor was she found to have any neurologic sequelae on routine follow-up 3 weeks postoperatively.

Discussion

In the patient who experiences delayed awakening from general anesthesia, several etiologic factors must be considered, including the residual effects of anesthetic agents and preoperative medications, inadequately reversed muscle paralysis, extremes of hyper- and hypotension, electrolyte and glucose abnormalities, impaired oxygenation, hypothermia, ineffectual ventilation resulting in carbon dioxide narcosis, exogenous administration of psychoactive substances, organic cerebral insults (e.g., vascular thrombosis), and subclinical seizures.^{1-3,5} In the two patients we described, we were able to use historical information, physical examination, and direct physiologic measurements to rule out the majority of these and related etiologies. Only then did we entertain the diagnosis of another, rare cause of failure to progressively awaken: factitious disorder.

Diagnostic and Statistical Manual of Mental Disorders defines factitious disorder as the "intentional production of physical or psychological symptoms in order to assume the sick role."⁶ It is a diagnosis of exclusion (*i.e.*, one that may be considered only after more serious conditions have been eliminated). Although factitious disorder is a recently defined diagnosis, similar presentations have been cited for many years with labels including hysterical psychosis, the Ganser syndrome (hysterical pseudodementia), and pseudopsychosis.^{7,8}

The best known factitious disorder is the Munchausen syndrome, a condition in which patients are sociopathic impostors who attempt to assume the sick role by habitually, purposefully, and consciously misleading their health-care providers.^{6,9,10} The typical Munchausen syndrome patient is male, likely to have exhibited psychopathic traits, and prone to multiple, widespread hospitalizations that he may not voluntarily reveal.^{9,10} This condition, first described by Asher in 1951, is reported to account for only 10% of patients with facti-

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tious disorders.¹⁰ The remainder of patients are typically younger, demographically stable individuals with benign disease states, often after a diagnosis of factitious disorder, externalization of symptoms (other than the patient) are absent, a prerequisite for diagnosis from that of malingering. A patient must have a clear motivation for symptom production (e.g., monetary compensation).^{6,9} A diagnosis that should be considered is somatoform disorder (factitious disorder). Somatoform disorders are physical complaints that are not explained by a general medical condition, but are produced (e.g., numbness in a limb). Patients with factitious disorder often have a "dramatic air" and a *logia fantastica* (fantastic aspects of their symptoms) and often have substantial knowledge of medical terminology and, in several cases, presented demographically by previous occupations.^{6,10,12,13} One of the (patient 1) was employed as a nurse and did not work in a medical field. The low prevalence of factitious disorder reflects the insidious nature of the disorder in diagnosis, and subsequent treatment. The average factitious disorder is diagnosed for 5-10 yr.¹⁰ Although severe cases of factitious disorder are believed to be more common, lesser manifestations are more frequent. Consistent with this view, both patients were female and suffered from the condition. Factitious disorder usually results in multiple hospitalizations and a lifelong pattern of behavior. In patient 1 by her right extremity burning was diagnosed as a conversion disorder. Factitious disorder starts in adolescence and is often associated with family or personal history of an abnormally high incidence of disorder.^{6,7,11} Although there

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tious disorders.¹⁰ The remainder of factitious disorder patients are typically younger, female, and more geographically stable individuals who only episodically feign disease states, often after serious life stresses.¹⁰

In factitious disorder, external incentives for the fabrication of symptoms (other than assuming the role of a patient) are absent, a prerequisite for distinguishing its diagnosis from that of malingering. Specifically, the malingering patient must have a clearly identifiable motivation for symptom production (e.g., the receipt of monetary compensation).^{6,9,11} Another possible diagnosis that should be considered in this patient population is somatoform disorder (including conversion disorder). Somatoform disorders also will present with physical complaints that are not fully attributable to a general medical condition, but they differ from factitious disorder in that symptoms are not intentionally produced (e.g., numbness in response to fear).^{6,11}

Patients with factitious disorder usually present their illness with a "dramatic flair" and may engage in *pseudologia fantastica* (fantastic lies) or confabulation of aspects of their symptoms and history.^{6,9} These patients often have substantial knowledge of medical tests and terminology and, in several studies, were over-represented demographically by persons with health-related occupations.^{6,10,12,13} One of the subjects we described (patient 1) was employed as a venipuncturist; the other did not work in a medical field.

The low prevalence of factitious disorder likely reflects the insidious nature of the condition, difficulty in diagnosis, and subsequent under-reporting of cases.⁶ The average factitious disorder patient is not accurately diagnosed for 5–10 yr.¹⁰ Although some authors disagree,¹² severe cases of factitious disorder generally are believed to be more common in males, whereas lesser manifestations are more likely in females.^{6,10} Consistent with this view, both of the patients we presented were female and suffered from a relatively mild form of the condition.

Factitious disorder usually follows a chronic course, resulting in multiple hospitalizations, and may become a lifelong pattern of behavior.^{6,10} This tendency was evident in patient 1 by her multiple hospitalizations for right extremity burning and paresis, eventually diagnosed as a conversion disorder. Traditionally, factitious disorder starts in adolescence, often after hospitalization for varied illnesses.⁶ Frequently, there is a family or personal history of psychiatric disorder^{6,7} and an abnormally high incidence of histrionic personality disorder.^{6,7,11} Although there was no known history of

psychiatric disorder in either of our patients' families, both patients underwent MMPI testing with results that were consistent with histrionic personality.

Historical information is important in determining whether a factitious disorder has contributed to delayed awakening or other anesthetic complications.⁸ Ideally, information obtained from the patient should be corroborated with other sources; however, this often is impossible.^{8,11} Nevertheless, in the preoperative interview, the anesthesiologist should be able to determine confabulation in all but the most medically sophisticated historians.¹⁰ The typical factitious disorder patient will seek the anesthesiologist's favor because he or she views acceptance by medical professionals as strong positive reinforcement.¹¹ This attitude will result in personable, accommodating behavior that is often incongruous to the circumstances leading to surgical intervention.⁹

Patients with factitious disorder usually stay within a given medical system until the hospital staff realizes, in light of fruitless examination and diagnostic procedures or lack of a response to medical and surgical treatment, that there is a high probability that the medical condition is voluntarily produced.^{7,10} Although some authors have reported a positive response to confrontational tactics,⁷ frequently the patient will leave a treatment setting, after positive reinforcement for assuming the sick role has been withdrawn, to locate more sympathetic surroundings.^{6,10} Accordingly, a long medical history involving multiple caregivers with poor continuity should signal the possibility of factitious disorder.¹⁰

In summary, we report two cases of failure to progressively awaken after general anesthesia because of factitious disorder. This is an uncommon, interesting etiology that should be considered only after other medical conditions have been ruled out as a cause of altered consciousness.

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Successful Pharmacologic Treatment of Massive Atenolol Overdose: Sequential Hemodynamics and Plasma Atenolol Concentrations

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ATENOLOL is a non-lipid-soluble β -adrenergic blocker, with greater selectivity for β_1 -adrenergic receptors.¹ It lacks intrinsic sympathomimetic activity or properties.¹ Only eight cases of atenolol poisoning have been reported in the English-language literature,²⁻⁹ and in a recent report, hemodialysis was advocated to treat atenolol overdose.² We report a case of massive atenolol overdose and successful treatment by aggressive pharmacologic intervention only, and present sequential

plasma atenolol concentrations and hemodynamic data. Our results show a marked improvement resulting from the administration of glucagon and call into question the need for instituting hemodialysis instead of aggressive pharmacologic management of such patients.

Case Report

A 50-yr-old, 98-kg man with a history of depression, coronary artery disease, hypertension, and asthma was brought to the our institution's emergency department after having allegedly ingested 20 atenolol tablets (50 mg/tablet) 30 min earlier. On arrival, he was alert and cooperative. Blood pressure was 130/80 mmHg, heart rate was 72 beats/min, respiratory rate was 24 breaths/min, and tympanic temperature was 36.6°C. Physical examination was otherwise unremarkable. An electrocardiogram showed sinus rhythm with a ventricular rate of 72 beats/min. A chest roentgenogram was normal. Laboratory data revealed sodium 141 mmol/L, potassium 4.9 mmol/L, chloride 104 mmol/L, HCO_3^- 18 mmol/L, blood urea nitrogen 12 mg/dL (4.28 mmol/L), creatinine 1.2 mg/dL (106 $\mu\text{mol/L}$), glucose 116 mg/dL (6.44 mmol/L), and hemoglobin 160 g/L. Routine toxicologic screening was negative. Gastric lavage was instituted without recovery of tablet fragments. An external pacemaker was applied. Electrocardiogram and oscillometric blood pressure monitoring were instituted, and for the next 90 min, a sinus rhythm at a rate of 60 beats/min and blood pressure between 110/70 mmHg and 120/80 mmHg were observed. Two hours after admission, the patient collapsed while having a bowel movement. Despite a total of 2 mg atropine, 1 mg glucagon, and 1,900 ml crystalloid, systolic blood pressure remained less than 80 mmHg. The electrocardiogram showed

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sinus rhythm at a rate between 40 and 60 beats/min. The patient's consciousness varied from unresponsive to responsive. Three hours after admission, his trachea was intubated and he was transferred to the intensive care unit. A total of 2 mg atropine, 1 mg glucagon, and 1,900 ml crystalloid were administered. A 13 $\mu\text{g/min}$ norepinephrine infusion was started immediately after intubation (mean arterial pressure 60 mmHg, PaO_2 74 mmHg, PaCO_2 206 mmHg, and pH 7.23). A balloon-tipped pulmonary catheter was inserted into the radial artery, and arterial blood gas analysis was started. Arterial blood gas analysis revealed a respiratory rate of 16 breaths/min, tidal volume 400 ml, PaO_2 333 mmHg, PaCO_2 44 mmol/L. A balloon-tipped pulmonary catheter was inserted. Pharmacologic management consisted of glucagon infusion, and isoproterenol infusion via a metered dosed inhaler. The patient was transferred to the intensive care unit transfer (6 hours after admission).

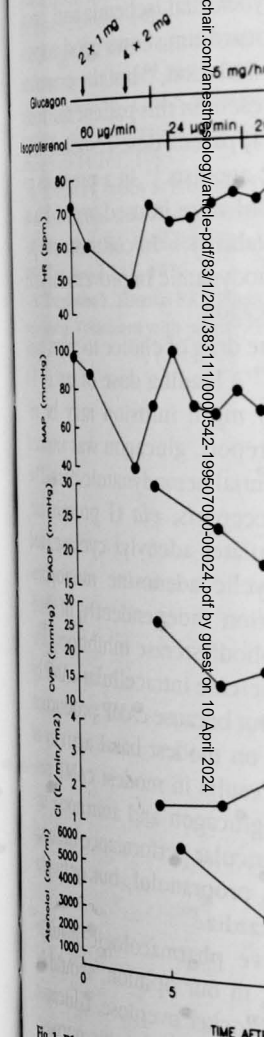


Fig 1. Pharmacologic management and plasma atenolol concentrations.