shunting, and possibly some degree of alveolar ventilation-perfusion mismatch and low cardiac output. Because peripheral oxygen saturation remained very low with nasal oxygen alone, the adjunction of NO was attempted. Inhaled NO concentration was certainly less than 15 ppm, however, sufficient NO delivery was achieved to increase Sa_{O_2} (29% to 71%) and obtain rapid clinical improvement. The systolic pulmonary artery pressure was reduced by 20%, indicating a pulmonary vasodilation and a lesser driving pressure for the shunting flow.[1,2] Other patients in whom inhaled NO improved pulmonary hypertension and systemic oxygenation were newborns with right-to-left shunting related to persistent pulmonary hypertension[3,4] and adults with patent foramen ovale and pulmonary hypertension.[5,6]

Few cases of long-term nasal NO delivery in spontaneously breathing patients with pulmonary hypertension have been reported.[7,8] This simple approach is effective, but the nasal route seems not very reliable, as emphasized by the episode of cardiac arrest that occurred when NO delivery was discontinued suddenly. This technique should be considered in selected patients, although medical personnel need to be very watchful.

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Anesthesiology
1998; 89:1587–9

An Unexpected Arousal Effect of Etomidate in a Patient on High-dose Steroids

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CATATONIA presents with stupor, excitement, or alternating stupor and excitement[1] and covers a broad group of movement disorders sometimes seen in psychotic illness. It is most often associated with schizophrenia but can also be found in connection with mania and depression.[2] Other conditions in which catatonia can be present include neurologic disorders, systemic metabolic disorders, and as a side effect of certain medications.[1]

Any two of the following signs manifests catatonia: motor immobility, excessive motor activity, negativism or mutism, peculiarities of voluntary movement, echolalia, or echopraxia.[2] Motor immobility may be manifested as a waxy flexibility. The excessive motor activity apparently is purposeless and is not influenced by external stimuli. There may be extreme negativism that is manifested by resistance to all instructions or the mainte-

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nance of a rigid posture against attempts to be moved. Additional features may include stereotypes, mannerisms, and automatic obedience or mimicry.[2]

What follows is a case in which etomidate produced an unexpected arousal effect, rather than sedation, in a patient with catatonia caused by a high dose of steroids.

Case Report

A 27-yr-old woman with a history of myasthenia gravis since 1994 was maintained with intermittent continuous positive airway pressure mask at home to facilitate ventilation. On the day of admission she complained of difficulty breathing and was brought to the hospital by her mother. *En route* to the emergency room, her mother realized that the patient was not responding to communication. At arrival in the emergency room, patient's physical examination revealed a mildly obese woman with cushingoid features. She was exhibiting rapid, shallow breathing and was unresponsive to verbal and painful stimuli (stupor); however, her eyes seemed to be moving under closed eyelids. Her initial arterial blood gas in room air was *p*H 7.43, arterial carbon dioxide tension (Pa_{CO_2}) 38, and arterial oxygen tension (Pa_{O_2}) 67. The emergency room physician thought that she was tiring and her breathing was becoming labored. It was then decided that intubation was warranted, after consultation with anesthesia personnel.

At this time, her oxygen saturation was 100% on continuous positive airway pressure mask with 100% oxygen, despite labored breathing. When the mask was removed for intubation, the patient was asked to open her mouth. Surprisingly, she complied. However, she quickly closed her mouth, with seemingly normal strength, when the laryngoscope blade was inserted into her mouth. Etomidate (20 mg) was administered to facilitate the laryngoscopy. A neuromuscular blocking agent was avoided because of her myasthenia gravis.

Less than 1 min after administration of the etomidate she opened her eyes and became animated. She was alert and oriented with muscle strength that was tested and rated as 5/5. She began to cry and talk steadily and with great emotion. The topic of her conversation was related to the events that had occurred since she became unresponsive on the way to the hospital. Her vital signs were stable. Her oxygen saturation was 100% with room air, and she seemed to be breathing more easily. Laboratory results were all within normal limits, except for white blood cell count of 18.0 1,000/mm^3 and glucose concentration of 327 mg/dl. Chest radiography showed questionable left lower-lobe infiltration.

A review of the patient's medications revealed a completed course of antibiotic therapy for an upper respiratory infection, which concluded 2 to 3 days before admission, and prednisolone for myasthenia gravis, which had been increased to 100 mg daily over the preceding 2 month period.

Hospital Course

The patient was transferred to the medical intensive care unit for careful observation. A psychiatric evaluation was requested. Approximately 8 h later, she was once again immobile and unresponsive to verbal and painful stimuli. Anesthesia personnel were again consulted. The attending psychiatrist requested another dose of etomidate to see whether the patient would respond as before. Less than 1 min after receiving etomidate (10 mg), she became alert and oriented with a muscle strength of 5/5. She again started to cry and talk incessantly. After evaluation by the psychiatrist, the behavior of the patient was explained as a brief atypical episode of catatonic psychosis[3] caused by the increased dose of prednisolone. There was no personal or family history of psychiatric disorder. Neurologic examination and computed tomography scan of the brain were normal.

The patient's dose of prednisolone was decreased to 30 mg three times a day and tapered down 5 mg every 5 days. The patient remained in the hospital for 4 days without further episodes.

Discussion

The patient exhibited catatonic behavior. Her exhibition of immobility, unresponsiveness to verbal and noxious stimuli, and eyeball movement under closed eyelids (negativism) fit the criteria of a brief catatonic psychosis.[3] Her unusual response to the verbal command to open her mouth and subsequent clamping down was consistent with "automatic obedience"' seen infrequently in catatonic patients.[2]

The patient's symptoms and clinical course were consistent with steroid-induced catatonic psychosis.[2,4,5] The mechanism of action of corticosteroid psychosis is not understood completely, but a stimulating effect on the reticular system by the corticosteroid is strongly suggested.[5] Stimulation of this system usually is coupled with inhibition of gamma-aminobutyric acid (GABA) receptor function, resulting in mood changes similar to those described in schizophrenia. These side effects of corticosteroid usage range from alteration in mood to overt delirium. The frequency of steroid-induced psychiatric disorders increases with the daily dose of medication, ranging from 4.2% for those prescribed 40–80 mg prednisone to 18% for those prescribed more than 80 mg.[5] Dividing the dosage of drug can diminish this side effect, whereas a previous psychiatric illness increases the incidence of corticosteroid psychosis.[6] Any drugs or factors such as etomidate that antagonize the stimulation of the reticular system may enhance GABA receptor function, which could moderate the psychotic symptoms.[7]

The effect of etomidate in this patient is similar to the effect of amobarbital. An "amobarbital interview" consists of three stages. In the first stage, patients become drowsy and dizzy. In the second stage, patients are still drowsy but become increasingly responsive to questions, which is the ideal period for an interview. In the third stage, patients lose corneal reflex, indicating that the patient has become oversedated. Patients with psychiatric disorders ranging from major depression to catatonic psychosis often will become verbal during the

second stage of an amobarbital interview. This is not usually the case if there is no psychiatric disease.[8] The mechanism of amobarbital has been shown to be the enhancement of the GABA receptor function.[9]

In this case report, the patient had a brief atypical catatonic psychosis, most likely resulting from stimulation of the reticular formation with antagonism of the GABA receptors function by the high dose of steroids. Her stuporous condition, together with her myasthenia gravis, resulted in respiratory difficulties. We believe that etomidate reversed this effect by antagonizing the steroid-induced changes. Hence, etomidate caused arousal rather than sedation in this particular patient.

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Anesthesiology
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Fatal Pulmonary Fat Embolism in the Early Postoperative Period

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PULMONARY fat embolism is a well-recognized complication of major orthopedic surgery.[1–5] Most cases occur intraoperatively during pressurization and manipulation of long-bone intramedullary canals.[1–3,5,6] Embolic events that occur in the postoperative period may be insidious, with their clinical sequelae evolving progressively.[4] We report an unusual case of acute, massive pulmonary fat embolism that occurred in the early postoperative period.

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Key words: Hip arthroplasty; osmium tetroxide stain; respiratory arrest.

Case Report

A 76-yr-old man was scheduled for left total hip arthroplasty during combined regional–general anesthesia. Two previous left total hip arthroplasties were uncomplicated. He had a history of hypertension, a 6-cm infrarenal abdominal aortic aneurysm and hypothyroidism. There was no documented history of coronary vascular disease. Medications included isosorbide dinitrate, metoprolol, and L-thyroxine. Results of physical examination were within normal limits; baseline preoperative systolic blood pressure was 120–150 mmHg and heart rate was 50–60 beats/min. Preoperative laboratory data were normal. The electrocardiogram showed only sinus bradycardia. Chest radiograph was normal.

Lumbar plexus psoas compartment and sciatic Mansour approach nerve blocks were performed using bupivacaine. General endotracheal anesthesia was induced and maintained with fentanyl, isoflurane, nitrous oxide, and oxygen. Arterial and central venous pressure monitoring and intraoperative autologous blood salvaging were used.

Despite an estimated 3,000-ml blood loss, the patient remained