

detailed discussion of risks before anesthesia, but it is equally clear that some do not. At least for some patients, discussion of risks and the increased physician dialog that goes with it may be beneficial. However, our data are also compatible with the hypothesis that certain patients are unable or unwilling to tolerate a detailed discussion of possible anesthetic risks, and that such a discussion may not be in their best interest. At the present time, we feel the most reasonable approach is to tell all patients that there are serious, although remote, risks of anesthesia, but to allow the individual patient to decide how much additional information he or she wishes to obtain about these risks.

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## Anesthesia, Sleep Paralysis, and Physostigmine

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Sleep paralysis, a relatively unknown and uncommon disease, is characterized by an inability to execute any voluntary activity while fully awake.<sup>1</sup> During such an episode, loss of muscle tone, decreased blood pressure, and hypoventilation, often with irregular respiration, are observed. Episodes usually occur upon awakening (hypnapompic or postdormital) or, more rarely, on falling asleep (hypnagogic or predormital). Sleep paralysis may occur as a single entity, but commonly is found together with the narcolepsy syndrome.

### REPORT OF A CASE

A 33-year-old white woman was admitted to the hospital with pain in the right lower quadrant of the abdomen of several weeks' duration, initially slight but recently increasing in intensity. In the past the patient had received general anesthesia at least five times with no untoward sequel. Past medical history was remarkable only in that the patient had had recurrent episodes of sleep paralysis, for which she had received unsuccessful treatment. The patient had no allergy, took no drug, did not smoke, had minimal alcohol intake, had no other diseases, and had no history of psychiatric illness.

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Physical examination was non-contributory with the exception of tenderness in the right lower quadrant and slight rebound tenderness. Results of laboratory examinations, including chest x-ray, ECG, and IVP, were within normal limits. The patient was scheduled for exploratory laparotomy the next day.

During the preanesthetic interview, the patient expressed considerable anxiety over the possibility that one of her episodes of sleep paralysis might be misinterpreted as a cardiac arrest and unnecessary cardiopulmonary resuscitation begun, as had occurred during a previous hospital stay. This had not occurred in the preanesthetic period, but the patient was determined to alert everyone to the possibility.

On the morning of operation, the patient was premedicated with morphine, 10 mg, diazepam, 10 mg, and atropine, 0.4 mg, im. She arrived in the anesthesia area sedated and in good spirits. She was prepared for anesthesia, taken to the operating room, and anesthetized. The surgical procedures consisted of abdominal exploration and left ovarian cystectomy. Anesthesia was maintained with nitrous oxide, oxygen, fentanyl, 0.1 mg, and pancuronium, 4 mg. Following the operation the residual action of pancuronium was reversed with neostigmine, 3.0 mg, and atropine, 1.0 mg, iv. Response to electrical nerve stimulation indicated minimal paralysis, and the patient was able to generate inspiratory force to 50 torr. The trachea was extubated and the patient was taken to the recovery room. In the recovery room she manifested an unusual glassy-eyed stare, and was unresponsive to either verbal command or noxious stimuli. Respirations were very irregular, with alternating periods of apnea, sighs, and tachypnea without evidence of airway obstruction. The patient's husband, a physician, said she appeared as though she were having an episode of

sleep paralysis. She was given 2 mg physostigmine, iv, and within a minute was awake, alert, and breathing normally. She reported that she had been conscious and having an episode of sleep paralysis. Forty minutes later, she lapsed into a second episode of sleep paralysis, and physostigmine again effected a dramatic recovery. The remainder of her stay in the recovery room was uneventful.

Four days later, at 1:30 A.M., the anesthesia department was called to see the patient. The resident who had attended the patient in the recovery room responded and found her in the same state, glassy-eyed, unresponsive, breathing irregularly. Physostigmine, 2 mg, iv, was administered, with the same immediate response. The patient recalled the entire episode and had clearly had another episode of sleep paralysis, terminated by physostigmine. The remainder of her hospital stay was uneventful.

### DISCUSSION

In our experience we have occasionally seen in the recovery room patients who have had signs and symptoms similar to those manifested by this patient, who have responded to treatment with physostigmine. With most, there is little understanding of the cause of the problem, and treatment with physostigmine is often purely empiric.<sup>2-7</sup> The etiology of our patient's symptoms was clearly sleep paralysis, confirmed by her own report. Being familiar with sleep paralysis, she was able to recognize the recovery room episodes as such.

Sleep paralysis is not nearly so uncommon as it is unknown and unrecognized. In one study, 359 unselected medical students, nursing students, and inpatients were interviewed, with the result that nearly 5 per cent were found to have experienced one or more episodes of sleep paralysis.<sup>1</sup> The usual course shows infrequent attacks, of brief duration, and lack of sequelae. The common association with hallucinations during sleep is probably responsible for patient's lack of concern; consequently the disease is seldom recorded in routine medical histories.

Little is known about primary sleep paralysis. What information there is commonly comes from patients who have narcolepsy with associated sleep paralysis. Sleep paralysis occurs in about 24 per cent of narcoleptic patients and usually is associated with a reversal of EEG sleep patterns. Sleep paralysis in these cases occurs with onset of rapid-eye-movement (REM) sleep,<sup>8,9</sup> whereas during normal nocturnal sleep, sleep onset is characterized by a non-REM EEG and followed after about 70 minutes with the first REM pattern. It is theorized

that sleep paralysis may represent a dysfunction in the reticular activating system. Others have considered it a dissociation between the physical and mental components of sleep, where the physical aspects of sleep can exist briefly without the mental. Other than its association with narcolepsy, sleep paralysis is not known to be associated with intracranial lesions, epilepsy, psychiatric disorders, or drug use. There appears to be no effective prophylaxis.

Since encountering the above-reported case, we have speculated whether the anesthetized state or drugs used in the perianesthetic period could induce a first episode of sleep paralysis in susceptible individuals or initiate an occurrence in an individual with previously unrecognized sleep paralysis.

Of six patients in whom we observed unexplained postoperative depression who responded to physostigmine, one gave a history indicating that the depression was almost certainly sleep paralysis, and that he had probably had several previous brief episodes of sleep paralysis without recognizing them as such.

Based on our experience and the unexpected high incidence (5 per cent) of sleep paralysis in the normal population, we feel that undiagnosed sleep paralysis may be the antecedent cause of unexplained postoperative depression more frequently than previously suspected.

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