Ketamine and Paralysis Agitans

To the Editor: —It has been suggested that ketamine be avoided in patients being treated with L-dihydroxyphenylalanine (L-dopa) because of an exaggerated sympathetic response.¹ We administered ketamine anesthesia to a 75-year-old man who had severe paralysis agitans (Parkinson's disease) and was receiving L-dopa, and found no hypertension but, instead, a salutary alleviation of his disease. The patient had a ten-year history of paralysis agitans and was totally incapacitated by the disease: he had extreme akinesis, rhythmic tremor, and general enfeeblement. He used eye signals to communicate, and could withdraw in response to painful stimuli. Surgical closure of a large trochanteric decubitus ulcer was planned. We chose ketamine anesthesia to avoid a difficult intubation. Ketamine, 200 mg, iv, was given over three hours, supplemented with nitrous oxide. The tremor ceased and the muscle rigidity decreased. This effect persisted for several hours postoperatively.

The postulated biomolecular defect in paralysis agitans is a decrease in dopaminergic neurons in the nigrostriatal pathways.² Treatment consists of increasing dopaminergic activity. Experimentally, ketamine has been shown to alter dopamine levels in rat brain.³ Whether it does so in man is unknown; our clinical observation suggests it may.

Paralysis agitans is a common disabling disease (1.57 cases per 1,000 population) presenting multiple problems for the anesthesiologist.⁴,⁵ Musculoskeletal deformities can make intubation traumatic, pharyngeal dysfunction increases the potential for aspiration, and impaired baroreceptor reflexes can produce unpredictable blood pressure responses. Although ketamine may be theoretically contraindicated in patients receiving L-dopa therapy, we found its use in this particular case to be advantageous.

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REFERENCES

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