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# Adrenocortical Function Following Multiple Anesthesia Inductions with Etomidate

*To the Editor:*—Etomidate is an intravenous anesthetic agent whose pharmacologic profile is characterized by cardiovascular stability[1] and rapid recovery. However, etomidate may produce adrenocortical suppression by enzyme inhibition of the cortisol synthetic pathway. This suppression has been demonstrated following both short and prolonged infusions and single doses of etomidate and may last as long as 22 h.[2] The effect of repeated exposure to etomidate over a period of days or weeks has not been documented previously. We describe a case in which etomidate was chosen as the induction agent in a patient with severe cardiovascular disease undergoing a course of electroconvulsive therapy.

A 63-year-old white woman with a psychotic depressive illness manifest by agitation, anxiety, weight loss, and poor response to therapy with psychotropic drugs was scheduled to undergo a course of electroconvulsive therapy. Her medical history was significant for severe coronary artery disease. She was symptomatic for angina and shortness of breath on exertion and had had a previous myocardial infarction. Coronary angiography revealed a dilated left ventricle with inferior akinesis, global hypokinesis, mitral regurgitation, and an ejection fraction of less than 0.25. Current therapy comprised sublingual glyceryl trinitrate tablets as required and nifedipine 10 mg orally tid.

Electroconvulsive therapy was performed on alternate days for a total of 10 treatments. General anesthesia was induced with a dose of 0.25 mg/kg of etomidate followed by 1 mg/kg of succinylcholine to provide muscle relaxation. The patient's lungs were manually ventilated by mask with 100% oxygen until return of adequate respiration. Pulse, blood pressure, and ECG were recorded in the routine fashion. Seizure activity was documented clinically and by chart recording of the EEG. There were no untoward cardiovascular events during the treatments. The cortisol response to ACTH stimulation was assessed 24 h following the final treatment. The baseline cortisol level was 31 μg/dl (normal range 7–25 μg/dl), and the level at 1 h after injection of ACTH was 42.5 μg/dl. An increase of 10 μg/dl or more of cortisol is regarded as a normal response of the adrenal cortex to ACTH stimulation. The high baseline cortisol level observed in our patient probably resulted from central stimulation of the hypothalamic pituitary adrenal axis, which occurs often in the depressive psychotic state.[3]

The presence of the normal response to ACTH stimulation in this patient suggests that recovery of adrenocortical function is complete by 24 h and that the endocrine effect of multiple doses of etomidate is not different in magnitude or duration than following a single dose.

The use of etomidate for multiple inductions of anesthesia in our patient appeared to be associated with the same risks and benefits as a single administration.

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