Optimizing ECT Technique in Treating Catatonia

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Catatonia is an acute movement disorder characterized by mutism, negativism, posturing, staring, rigidity, repetitive acts, verbalizations, and approximately 20 other signs. In systematic studies in academic centers, approximately 10% of patients in psychiatry, neurology, emergency department, and medical units meet criteria for its presence.[1–4]

It was first characterized in 1874 by Karl Kahlbaum in his sanitarium at Gorlitz, Germany. In 1899, the German psychiatrist Emil Kraepelin delineated the *dementia praecox* syndrome and incorporated catatonia as a marker. A decade later, the Swiss physician Eugen Bleuler relabeled the syndrome as *schizophrenia*, accepting catatonia as its marker. For the next century, catatonia was regarded as a type of schizophrenia and treated as such. Its rapid relief by barbiturates was described by William Bleckwenn in Wisconsin in 1930 (a finding confirmed in a randomized controlled trial by McCall et al in 1992[5]). Four years after the report on the efficacy of barbiturates, the Hungarian neuropsychiatrist Ladislas Meduna reported that chemically inducing seizures relieved catatonia. By 1938, the seizures were induced with electricity, the present method of electroconvulsive therapy (ECT).

Although barbiturates and seizures relieved catatonia, they were ineffective in other forms of schizophrenia, casting doubt on Kraepelin's incorporation. By the 1970s, catatonia was increasingly identified among patients with mood disorders, infections of syphilis and typhoid fever, the epilepsies, and the neurotoxicity induced by neuroleptic drugs (identified as the *neuroleptic malignant syndrome*.) The neuroleptic treatments of schizophrenia were ineffective in catatonia (often worsening the condition), and by 1990, catatonia was considered a syndrome independent of schizophrenia.[6] However, Kraeplin's incorporation was firm, and it took 2 additional decades until catatonia was finally divorced from schizophrenia in *Diagnostic and Statistical Manual of Mental Disorders*, *Fifth Edition*, in 2013. Catatonia as a type of schizophrenia was deleted from the classification and is now cited mainly as secondary to a systemic medical condition.[7]

During these decades, the signs of catatonia were identified, rating scales and methods of examination to assist the diagnosis developed and effective treatment protocols established on the basis of ECT and the benzodiazepines (these replaced the barbiturates for considerations of safety).[8,9] Benzodiazepines are prescribed as soon as the syndrome is recognized. For patients with the sedated stuporous form of catatonia marked by mutism, withdrawal, and inhibition of movement, the dosage of lorazepam begins at 1 to 3 mg orally, progressing rapidly until symptom relief is seen. For severely ill patients, dosages of 15 to 30 mg/d have been necessary (Diazepam dosages are calculated at ratios of 5 to 1 mg of lorazepam. Zolpidem dosages are reported up to 40 mg/d). In systematic studies of catatonia, approximately 80% of the recognized cases respond to benzodiazepine treatment, leaving fewer than 20% requiring referral for ECT.[1,2]

How is catatonia recognized? The syndrome is best defined by 2 or more signs for 24 hours or longer from among the 23 signs on the Catatonia Rating Scale (CRS). A prompt reduction in the CRS score by an intravenous injection of lorazepam (1–2 mg) or diazepam (5 mg) or an oral dose of zolpidem (10 mg) verifies the diagnosis. Successful relief by high doses of a benzodiazepine or a course of ECT validates the diagnosis.[1,8,9]

It is the treatment with ECT that is of interest here. Catatonia appears in many forms, some retarded and relatively benign, but many malignant and life-threatening. The bread and butter of ECT practitioners is the treatment of middle aged and elderly depressed patients.[10] In these patients, fear of cognitive deficits has led practitioners to weaken the electrical stimulus with decreasing treatment efficacy. Dosing with unilateral electrode placement, ultrabrief currents, low total stimulus charge, and twice weekly treatments are often offered. However, when these types of ECT are applied in catatonia, they are likely to fail to relieve the catatonia syndrome.

The retarded form of catatonia is characterized by withdrawal, mutism, negativism, refusal of food, and dehydration leading to a malignant life-threatening illness. A more robust treatment is necessary. To

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the common protocol of oxygenation, sedation with an anesthetic (methohexital, etomidate, propofol), administering an agent to block vagal activity (atropine, glycopyrrolate), it is necessary to administer the benzodiazepine antagonist flumazenil in patients who have been heavily dosed with benzodiazepines.[11] For the manic patient requiring restraints, intramuscular ketamine is a useful alternative anesthetic.[12]

Treatments are best administered daily in febrile, dehydrated, and overly excited delirious patients. Bitemporal electrode placement with age-based suprathreshold dosing offers the best outcomes. The fear of memory loss that plagues treatment in the depressed elderly patients is not relevant because the issue is saving life and reversing negativism or delirium to assure a living patient.

A principal hurdle in applying ECT in catatonia is the requirement for individual consent, especially in the states with legislated restrictions on ECT practice. Practitioners need to be well acquainted with each state's laws and be agile in convincing their courts to approve treatment.[13]

The prescription of a set number of treatments is a common practice that is particularly dangerous in catatonia. No physician can anticipate how many treatments are necessary, and the guesstimate of a fixed number is associated with treatment failure and early relapse. Continuation ECT is necessary for all patients until the patient returns to the preillness state.[1,2]

Catatonia scholars have identified many forms beside the retarded mute form, the malignant form of neuroleptic malignant syndrome, and the excited delirious form.[1,4] The most interesting recent delineation is that of repetitive self-injurious behavior in young adolescents identified as suffering from autism or mental retardation.[3,14] Many patients with signs of catatonia and suffering anti-N-methyl-D-aspartate receptor encephalitis, lupus, or other autoimmune diseases respond well to ECT.[4] The repetitive behaviors of Tourette syndrome meet catatonia criteria and improve with ECT.[4]

For decades, ECT has been commonly prescribed for patients with major depression, less often for those with bipolar disorder, and infrequently for other conditions. These are the principal accepted indications for ECT. Because catatonia has been divorced from schizophrenia, many treatable varieties have been identified, its recognition verified, and guidelines for effective treatment defined. Catatonia is a malignant disorder that requires the specialized treatments of benzodiazepines and ECT for effective outcomes. The prescription of neuroleptics is best interdicted. Practitioners are urged to consider the special needs in catatonia patients as central to the services that they offer in clinical practice.

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