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SAFETY AND UTILITY OF ACUTE ELECTROCONVULSIVE THERAPY FOR AGITATION AND AGGRESSION IN DEMENTIA

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Abstract

Objective—Agitation and aggression are among the most frequent and disruptive behavioral complications of dementia that contribute to increased cost of care, hospitalization, caregiver burden, and risk of premature institutionalization. This current study examined the safety and efficacy of electroconvulsive therapy (ECT) as a treatment for behavioral disturbances in dementia. We hypothesized that ECT would result in reduced agitated and aggressive behaviors between baseline and discharge.

Methods—Twenty-three participants admitted to McLean Hospital (Belmont, MA) and Pine Rest Christian Mental Health Services (Grand Rapids, MI), with a diagnosis of dementia who were referred for ECT to treat agitation and/or aggression, were enrolled in the study. We administered the Cohen-Mansfield Agitation Inventory (CMAI)-short form, Neuropsychiatric Inventory (NPI)-Nursing Home Version, Cornell Scale for Depression in Dementia (CSDD), and the Clinical Global Impression Scale (CGI) at baseline, during, and after the ECT course.

Results—Regression analyses revealed a significant decrease from baseline to discharge on the CMAI ($F(4, 8) = 13.3$; $p = 0.006$) and NPI ($F(4, 31) = 14.6$; $p < 0.001$). There was no statistically significant change in scores on the CSDD. The CGI scores on average changed from a rating of “markedly agitated/aggressive” at baseline to “borderline agitated/aggressive” at discharge. Treatment with ECT was well tolerated by most participants; discontinuation of ECT occurred for two participants due to recurrence of agitation and for three participants due to adverse events.

Conclusions—ECT may be a safe treatment option to reduce symptoms of agitation and aggression in patients with dementia whose behaviors are refractory to medication management.

Keywords

ECT; electroconvulsive therapy; dementia; agitation; aggression

Introduction

Agitation and aggression are among the most frequent and disruptive behavioral complications of dementia that contribute to increased cost of care, hospitalization, caregiver burden, and risk of premature institutionalization (Bartels et al., 2003; Cohen- Mansfield, 2009; Gilley et al., 2004; Lyketsos et al., 2002; Murman et al., 2002; Stern et al., 1997; Zahodne et al., 2013). Given the tremendous adverse impact of these behavioral symptoms for patients, caregivers, and the community, there is a critical need to identify effective treatment strategies for agitation and aggression in dementia.

Despite the high prevalence of these agitated and aggressive behaviors, there are currently no treatment options approved by United States Food and Drug Administration (US FDA) for this indication. Non-pharmacological interventions, including environmental and behavioral modification, are difficult to implement in nursing home settings due to low staff-to-resident ratios (Janzen et al., 2013). Clinicians commonly utilize off-label prescription of psychopharmacological agents, such as atypical antipsychotics, to manage behavioral disturbances in patients with dementia (Salzman et al., 2008; Sultzer et al., 2008). Atypical antipsychotics have been found to be only modestly helpful in addressing behavioral symptoms and unfortunately are associated with dangerous side-effects including tardive dyskinesia, cerebrovascular adverse events, sedation, and increased risk of mortality (Ballard et al., 2006; Dolder & Jeste, 2003; Schneider et al., 2005; Sultzer et al., 2008; Wang et al., 2005). All antipsychotics now contain a US FDA boxed warning for mortality risk in patients with dementia complicated by agitation and psychosis (US FDA, 2008). The persistence of agitated and aggressive behaviors refractory to pharmacological treatment often precludes safely maintaining an individual with dementia in the home or in a long-term care facility.

Electroconvulsive Therapy (ECT) has been found to be safe and effective in the treatment of depression and mania in elderly adults, with and without dementia (McDonald & Thompson, 2001; Nelson & Rosenberg, 1991; Rao & Lyketsos, 2000; Stoppe et al., 2006; Tew et al., 1999; Tomac et al., 1997; Weintraub & Lippman, 2001). The few published reports that investigated the use of ECT for the treatment of agitation in dementia have been case studies and retrospective chart reviews that provided support for the utility of ECT as a

safe and beneficial intervention strategy (Bang et al., 2008; Grant & Mohan, 2001; Roccaforte et al., 2000; Sutor & Rasmussen, 2008; Ujkaj et al., 2010). A major concern for using ECT in elderly patients, especially those with dementia, is its adverse effect on cognitive functioning. Research has found that most neurocognitive effects of ECT in the elderly without dementia are short-term and tend to resolve within a six month time period (McClintock et al., 2011). The few studies that have investigated the neurocognitive effects of ECT in patients with dementia found that performance on neurocognitive measures remained stable or modestly improved over time (Rao & Lyketsos, 2000; Hausner et al., 2011). To our knowledge, there have been no prospective clinical investigations that examined the safety and utility of ECT to treat behavioral disturbances in patients with dementia.

The current, naturalistic study examined the safety and efficacy of ECT in reducing agitation and aggression in dementia. We hypothesized that in elderly patients with dementia on an inpatient geriatric psychiatric unit, ECT would result in a reduction of agitated and aggressive behaviors from baseline to discharge. We also investigated the effect of ECT on cognitive functioning before and after an acute course of ECT.

Methods

Study Sample

We recruited participants for this study from the Geriatric Neuropsychiatry Unit at McLean Hospital (Belmont, MA) and the Older Adult Unit at Pine Rest Christian Mental Health Services (Grand Rapids, MI) from 2010 to 2012. Inclusion criteria included a diagnosis of dementia according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria (APA, 2000), and referral to ECT by the primary psychiatrist specifically for the treatment of agitation and/or aggression. Study entry criteria also included a Mini-Mental State Examination (MMSE) total score of ≥ 24 at baseline (Folstein et al., 1975). Minimal cutoff scores on the Cohen-Mansfield Agitation Inventory Short Version (CMAI-short form) were defined as ≥ 4 on at least one item, a score of 3 on at least 2 items, or a score of 2 on at least 3 items (Cohen-Mansfield et al., 1989). The treating psychiatrist, in consultation with the ECT service, made a clinical decision regarding the use of ECT treatment for agitation or aggression associated with dementia, irrespective of mood symptoms.

Exclusion criteria included a diagnosis of delirium at study entry, history of substance abuse in the past 12 months or ECT treatment during the 90 days prior to study enrollment. Patients whose authorized healthcare representative (AHCR) consented to ECT treatment for agitation and aggression were approached for study enrollment after the patient's clinical treatment team decided to proceed with ECT. We enrolled these patients in our study after we obtained written informed consent from the AHCR, and assent from the study participants. This study was approved by the institutional review board at each study site.

Measures

The CMAI-short form, Neuropsychiatric Inventory (NPI)-Nursing Home Version, Cornell Scale for Depression in Dementia (CSDD), and the Clinical Global Impression Scale were used to assess agitation, aggression and other neuropsychiatric symptoms (Alexopoulos et al., 1998; Cummings, 1997; Guy, 1976).

Measures to assess global cognitive functioning included the MMSE and the Severe Impairment Battery (SIB; Saxton et al., 1990). The SIB was included halfway through the period of enrollment after we determined it was necessary to have a cognitive measure for severely impaired patients with limited communications skills. The Alzheimer's Disease Cooperative Study Group-Activities of Daily Living (ADCS-ADL) scale was used to measure functional ability (Galasko et al., 1997).

Procedures

Participants completed an inpatient acute ECT course as determined by their treating psychiatrist and the ECT treatment team. The CMAI, NPI, CSDD and CGI were completed at baseline (prior to the first ECT treatment), after the third, sixth, ninth and twelfth (where applicable) ECT sessions, and within 72 hours prior to discharge. The MMSE, SIB and ADCS-ADL were administered at baseline and within 72 hours prior to discharge. The use of as needed medications was tracked from baseline to final assessment for all study participants. The CMAI, NPI, CSDD, and ADCS-ADL were completed by a trained nurse on the inpatient unit based on an interview with direct-care specialists and their behavioral observations of the participant. The CGI was completed by the treating psychiatrist as part of standard clinical care. A neuropsychologist or nurse trained to assess neurocognitive function administered the MMSE and SIB. We also recorded concomitant medical and psychiatric diagnoses for each subject during the course of the study.

Electroconvulsive Therapy Procedure

Electroconvulsive therapy consultation was performed by an ECT-credentialed psychiatrist prior to treatment administration. The ECT device used to deliver the treatment was a MECTA SPECTRUM 5000Q ECT (MECTA Corporation, Tualatin, Oregon) or Thymatron System IV (Somatics, LLC, Lake Bluff, IL). Seizure threshold was titrated on the first treatment with the empirical dose titration method, and stimulus parameters were adjusted as needed during the ECT course based on seizure quality and treatment efficacy. The choice of initial electrode placement was determined by the treating clinician based on clinical assessment. For most participants, clinicians began with right unilateral electrode placement, as it is associated with less cognitive side effects, with a brief pulse width (0.5 to 1.0 milliseconds) at approximately 4 to 6 times the seizure threshold. If there was no clinical response, patients were transitioned to bitemporal electrode placement with a brief pulse width (0.5 to 1 millisecond) at approximately 1.5 to 2.5 times the seizure threshold.

The ECT course consisted of treatments administered three times per week, or less frequently if clinically indicated. Methohexital was used for anesthesia induction, and succinylcholine for muscle relaxation. Etomidate could be substituted for methohexital in patients with inadequate seizure duration or quality. Concomitant medications were

administered for treatment emergent effects as needed including ondansetron for nausea, ketorolac, ibuprofen or acetaminophen for headache, esmolol or labetalol for elevated blood pressure or heart rate, atropine or glycopyrrolate for bradycardia, and midazolam, lorazepam, and/or propofol to prevent post-ictal agitation. Electroencephalographic (EEG) monitoring was performed to ensure adequate seizure duration.

Statistical Analyses

Linear mixed-effects models, incorporating time as a fixed effect and subject as a random effect, were constructed for each of the behavioral scales. This design included all subjects enrolled regardless of treatment duration, similar to an intent-to-treat design. Our primary outcome measure (CMAI-short form) was tested using a multiple degree of freedom comparison of scores. Post hoc tests compared baseline CMAI, to CMAI at the third, sixth, ninth and twelfth (when applicable) ECT sessions. This same model was used to test the effect of ECT treatment on the secondary outcome measures (NPI and CSDD). Following the recommendations of Hollingworth et al. (2006), we also analyzed a four-factor model of the NPI (Hollingworth et al., 2006) using a similar approach.

Standing and as needed antipsychotic medications were converted to chlorpromazine equivalents (Baldessarini, 2012; Gardner et al., 2010) for an analysis of change in dosage and number across course of ECT treatment. A linear mixed effects model, utilizing subjects as a random effect and time as a fixed effect, evaluated the change in standing antipsychotic dosage. A separate linear mixed-effects model was constructed to examine summed use of “as needed” medication over three of the ECT trial weeks (these were defined as summed antipsychotic dosage contemporaneous with treatments 1–3, treatments 4–6 and treatments 7–9). Four subjects were excluded due to the use of haloperidol topical gel for which no calculation of chlorpromazine equivalents was possible due to the nature of the pharmacokinetics. Hypothesis tests were conducted using the Kenward-Rogers adjustment (Kenward and Roger, 1997; Kowalchuk et al., 2004). Bonferroni corrections were applied separately for our planned comparisons (CMAI, CSDD and NPI), and we accounted for our choice of three outcomes in our planned comparisons category by multiplying resulting p values by three. Therefore, our adjusted p-value required for significance in each category was 0.05. All models utilized an unstructured covariance matrix. All statistical tests were two-tailed. Data analysis was performed using SAS statistical software (version 9.3; SAS Institute, Cary, NC).

Results

The sample (n=23) consisted of fourteen participants admitted to McLean Hospital and nine participants admitted to Pine Rest Christian Mental Health Services. Tables 1 and 3 show the demographic and clinical characteristics of the study sample.

Regression analyses revealed a significant decrease in the CMAI-short form scores from baseline to discharge ($F(4, 8)=13.31$; $p=0.006$). Post-hoc t-tests revealed a significant decline in CMAI –short form scores from baseline to the third ($t(22)=-2.63$; $p=0.045$), sixth ($t(22)=-5.22$; $p<0.001$), ninth ($t(20)=-7.41$; $p<0.001$) and twelfth ($t(3)=-3.71$; $p=0.012$) ECT treatments. As seen in Figure 1, CMAI scores declined from baseline to the ninth ECT

sessions and then remained fairly stable from the ninth to the twelfth ECT sessions and/or discharge.

Scores on the NPI significantly decreased (see Figure 2) from baseline to discharge ($F(4, 31) = 14.64$; $p < 0.001$). Post-hoc t-tests revealed a significant decline in NPI scores from baseline to the third ($t(52) = -4.32$; $p < 0.001$), sixth ($t(44) = -5.69$; $p < 0.001$), ninth ($t(33) = -6.14$; $p < 0.001$) and twelfth ($t(13) = -6.53$; $p < 0.001$) ECT sessions. As seen in Figure 2, scores declined from baseline to the third ECT session and continued to decline by the sixth and ninth ECT sessions. There was a significant decline in scores on the NPI subdomains of behavioral dyscontrol ($F(4, 17) = 11.76$; $p < 0.001$) and agitation ($F(4, 13) = 15.2$; $p < 0.001$) from baseline to discharge (see Figure 3). Post-hoc t-tests revealed a significant decline in the behavioral dyscontrol subdomain scores from baseline to the third ($t(22) = -5.22$; $p < 0.001$), sixth ($t(22) = -4.91$; $p < 0.001$), ninth ($t(21) = -5.64$; $p < 0.001$) and twelfth ($t(20) = -6.86$; $p < 0.001$) ECT sessions. Post-hoc t-tests revealed a significant decline in the agitation subdomain scores from baseline to the third ($t(22) = -3.26$; $p < 0.01$), sixth ($t(22) = -5.80$; $p < 0.001$), ninth ($t(22) = -7.48$; $p < 0.001$) and twelfth ($t(10) = -6.71$; $p < 0.001$) ECT sessions. There was no statistically significant decline in mean scores of NPI subdomains of mood ($F(4, 11) = 3.32$; $p = 0.053$) and psychosis ($F(4, 1) = 1.89$; $p = 0.467$) from baseline to discharge.

There was no significant change in mean CSDD scores from baseline to discharge ($F(4, 14) = 4.04$; $p = 0.07$). The CGI scores on average went from a rating of “markedly agitated/aggressive” at baseline to “borderline agitated/aggressive” by discharge.

As needed antipsychotic medication-use significantly decreased from an average chlorpromazine equivalent dosage of 7.8 ($SD = 14.2$) during the week between baseline and the third ECT session to 1.6 ($SD = 6.8$) during the week between the sixth and ninth ECT session ($F(2, 36) = 4.50$; $p = 0.018$). There was no significant change in antipsychotic standing medications (see Table 2) from baseline to discharge ($F(2, 36) = 0.2173$; $p = 0.764$).

There was no significant change in ADCS-ADL scores from baseline to discharge ($F(1, 21) = 1.19$; $p = 0.289$). Due to significant agitation and/or inability to sustain attention, participants were unable to consistently complete the neurocognitive assessment at baseline and discharge. Of the patients ($n = 10$) who completed the MMSE at both baseline and discharge, the average MMSE score modestly improved from 8.2 ($SD = 5.4$) to 10.1 ($SD = 5.2$). Of the patients ($n = 6$) who completed the SIB, the average SIB total score improved from 55.8 ($SD = 16.0$) to 65 ($SD = 18.58$).

Based on the clinical judgment of the treating psychiatrist, ECT was discontinued due to poor treatment response for two participants (#7–8). Participant #8 died the day before he was to be discharged to a nursing home with palliative care. The treatment team determined that the patient’s death, which occurred approximately one month after his final ECT session, was related to end-stage dementia and was unrelated to ECT. ECT was discontinued for two participants (#2, 11) due to delirium that was thought to be secondary, in part, to treatment with diphenhydramine and a urinary tract infection, respectively. Participant #13 developed atrial fibrillation and was transferred to a general medical hospital so that she

could be medically monitored while continuing with ECT. See Table 3 for adverse events in our study sample.

Discussion

To our knowledge, this is the first naturalistic, prospective study to investigate the safety and efficacy of ECT as a treatment for agitation and aggression in patients with dementia.

Overall, our results demonstrated ECT to effectively reduce symptoms of agitation and aggression in elderly patients with dementia who did not respond to psychopharmacological intervention alone. This suggests that ECT may be a potential treatment option for patients with dementia who are refractory to medications for agitation and aggression. Importantly, there were significant reductions in behavioral disturbances by the third ECT session, and most participants showed a reduction in behavioral disturbances by the ninth ECT session. If borne out by subsequent trials, such rapid reduction in behavioral disturbances could have significant public health implications by improving the quality of life for patients with dementia, alleviating caregiver burden, and increasing residential placement options for patients.

The results of this study revealed a significant reduction in scores on the CMAI-short form and NPI from baseline to discharge, with the greatest decline seen from baseline to the ninth ECT treatment on both measures. Regarding the NPI, a significant decline was observed in the behavioral dyscontrol and agitation sub-domains. As needed antipsychotic medication use significantly decreased across the course of nine ECT sessions. There was no evidence for a significant decrease in the patients' ability to execute instrumental and basic activities of daily living from baseline to discharge.

The results of this study supported the findings of a retrospective chart review by Ujkaj et al. (2010) who found a significant reduction in agitation and aggression, from baseline to discharge, in patients with dementia referred for ECT due to behavioral disturbances. Also, our findings are consistent with other case series and retrospective chart review studies. Grant and Mohan (2001) reported a case series of four patients with dementia who showed reduced agitation and aggression after ECT. Sutor and Rasmussen (2008) found that nine of 11 participants with Alzheimer's disease and agitation had an improvement or remission of agitation symptoms and a decrease in hospitalizations during the year following ECT treatment. Most research on the use of ECT in patients with dementia involved treatment for co-morbid mood disorders. For example, Rao and Lyketsos (2000) evaluated 31 patients with dementia and they showed a significant decrease in depressive symptomatology after treatment with ECT. Weintraub and Lippman (2001) studied two patients with severe dementia, depression and mania, and found that mood significantly improved after five to seven ECT treatments, and its positive impact persisted for approximately two months post discharge.

In our study, ECT was discontinued for two participants (both with end-stage dementia) due to poor response, which was defined by recurrence of agitation and aggression that reached approximately baseline severity levels. Three participants, despite improvement in agitation and aggression, had adverse events that resulted in discontinuation of ECT.

We were unable to consistently collect neurocognitive data for participants at baseline and post-ECT time points due to severity of agitation. Based on the data from participants who completed the neurocognitive measures before and after ECT, there was an approximate two-point increase in the average MMSE score and a 10-point increase in the average SIB score from baseline to discharge. This is consistent with a previous study by Rao and Lyketsos (2000) who found a modest improvement in MMSE average scores of 1.6 points from baseline to discharge in patients with dementia treated with ECT for depression. Hausner et al. (2011) demonstrated stability of the MMSE total score in patients with dementia treated with ECT for depression, from baseline to 6 weeks and 6 months post treatment, suggesting that global cognitive function neither improved nor declined across time. In our study, it is unclear if treatment with ECT directly resulted in improved global cognitive functioning. A possible explanation is that ECT improved agitation and aggression, which resulted in enhanced cooperation with neurocognitive testing. However, further research in larger sample sizes is warranted to better understand the effects of ECT on neurocognitive function in patients with dementia who are treated with ECT for aggression and agitation.

A limitation of this naturalistic study design was the lack of a control group of participants treated only with pharmacotherapy. We were unable to recruit an adequate control group of individuals who were referred for ECT, but whose AHCR did not give permission (n=1). The majority of healthcare proxies were generally supportive of the study and the use of ECT to treat behavioral disturbances. In this study, ECT treatment was initiated for those participants for whom multiple medications were found to be ineffective. Patients had been on an inpatient unit for an average of 28 days, minimizing the likelihood that medications alone were responsible for reducing behavioral disturbances. As a result, this study was unable to compare the effectiveness of ECT and pharmacotherapy. It may be possible that the reduction in agitation and aggression may have been due to a synergistic effect between ECT and pharmacological treatment. Another important limitation of this study was that it was open label and non-randomized, which may have influenced rating behavior by the study staff. As this was the first naturalistic, prospective study of the use of ECT to treat agitation and aggressive symptoms in patients with dementia, the goal was to collect preliminary data for the development of a randomized, double-blinded, controlled clinical investigation.

No consistent clinical algorithm was in place to determine the number of required medication trials before participants were referred for ECT. Each participant received psychotropic medications at different dose strengths and for varying duration prior to study enrollment, which may have impacted the clinical response to ECT. In addition, as this was a naturalistic study that did not control factors such as concomitant medications, participants were on varying psychotropic medication regimens while they participated in the study. Participants were diagnosed with dementia of various etiologies, which contributed to diagnostic heterogeneity and raised the question of the relationship of dementia neuropathology and the mechanism of action of ECT. However, including participants with varying etiology of dementia and treatment regimens with various pharmacological agents improved the generalizability of the results.

Despite these limitations, our results are encouraging and suggest that ECT may be a rapidly acting, safe and effective treatment for certain patients with dementia and behavioral disturbances that do not respond to or tolerate standard behavioral interventions and pharmacotherapy. Future research is warranted that includes a randomized, double-blinded study, with a control group adhering to a medication algorithm, in order to investigate the efficacy of ECT as a treatment for behavioral disturbances in dementia. Such research could be followed by investigations to determine the combination of ECT parameters (e.g., electrode placement, pulse width) that will result in high efficacy and low side effects. Longitudinal evaluation of behavior, mood, neurocognitive function, and functional ability is necessary to assess the long-term effects of ECT on behavioral disturbances. Measures of quality of life and caregiver burden could also be included as part of future clinical investigations.

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Key Points

1. Agitation and aggression are common behavioral complications of dementia.
2. Off-label prescription of atypical antipsychotics to treat agitation and aggression in dementia are associated with dangerous side-effects.
3. ECT may be a potential treatment option for patients with dementia who are refractory to medications for agitation and aggression.

Figure 1.
Cohen-Mansfield Agitation Inventory
*=p<0.05; ***=p<.001

Figure 2.
Neuropsychiatric Inventory
***= $p < .001$

Figure 3.
Percent of Symptoms Remaining in NPI Subdomains Following ECT Treatment
*=p<.001

Table 1

Demographics and Clinical Characteristics (n=23)

	Mean (SD)	N (%)
Age	73.8 (9.2)	
Education	13.9 (2.6)	
Gender		
Female	n/a	14 (60.1)
Race		
Caucasian	n/a	23 (100)
Diagnoses	n/a	
Alzheimer's disease		13 (56.5)
Vascular dementia		4 (17.4)
Mixed dementia		2 (8.7)
Frontotemporal dementia		1 (4.3)
Dementia NOS		3 (13.0)
No. ECT Treatments	9.4 (range: 5–14)	
RUL		17 (73.9)
RUL to BL		2 (8.7)
BL		4 (17.4)
No. Days on Unit pre-ECT	27.9 (20.1)	
Total No. Days on Unit	57.3 (28.2)	
Continuation ECT recommended	n/a	15 (65.2)

NOS=Not otherwise specified, ECT=Electroconvulsive therapy, RUL=right unilateral, BL=bilateral

Table 2

Standing Medication Classes Upon Admission, Before ECT, and at Discharge (% Participants)

	<u>Upon Admission</u>	<u>Before ECT</u>	<u>Discharge</u>
Atypical Antipsychotics	61	65	61
Conventional Antipsychotics	0	4	0
Anticonvulsants	30	35	30
Benzodiazepines	26	17	22
Antidepressants	70	61	52
Cholinesterase Inhibitor	35	22	13
NMDA receptor antagonist	30	22	4

ECT=Electroconvulsive therapy

Table 3**Medical and Psychiatric History and Adverse Events**

Subj #:	Axis I and III diagnoses	Adverse Events
1	History of complex partial seizure	None
2	Bipolar affective disorder, benign prostatic hypertrophy, HTN	ECT discontinued due to delirium and skin rash
3	Schizoaffective disorder, hyperlipidemia, seizure disorder	None
4	Mood Disorder NOS, HTN, hypothyroidism, macular degeneration	None
5	Osteoporosis	None
6	Renal insufficiency, h/o lymphoma	Developed hypertension, treated with esmolol and continued ECT
7	Post-Traumatic Stress Disorder, Generalized Anxiety Disorder, HTN, history of subdural hematoma	ECT discontinued due to poor response
8	History of traumatic brain injury, s/p cardiac arrest, HTN, GERD, h/o alcohol dependence	ECT discontinued due to poor response
9	Mood Disorder NOS, history of alcohol dependence, history of TBI, encephalopathy, acute renal failure, HTN, cellulitis, dermatitis	None
10	Osteoarthritis, GERD	None
11	MDD, CVA, GERD, diverticulitis, HTN, hyperlipidemia, CAD	ECT discontinued due to delirium secondary to UTI
12	Mood Disorder NOS, history of CVA, hyperlipidemia, HTN, GERD	None
13	Mood Disorder NOS	ECT discontinued after participant developed atrial fibrillation on two occasions after ECT
14	HTN	Developed delirium secondary to UTI after final ECT treatment
15	HTN, osteoarthritis, GERD	None
16	COPD, hypothyroidism, osteoarthritis, remote history of alcohol dependence	None
17	None	None
18	Anemia, glaucoma, CLL, diabetes, GERD, hypercholesterolemia	None
19	Duodenal ulcer, benign prostatic hypertrophy, history of stroke	Delirium secondary to ECT, and visual hallucinations two days after final ECT treatment
20	head trauma, benign essential tremor, COPD	Delirium secondary to ECT, incontinence and ataxia
21	HTN, history of CHF, COPD, hyperlipidemia, vitamin B12 deficiency, osteoporosis, osteoarthritis	Delirium secondary to ECT
22	Asthma	None
23	Urosepsis, diabetes, GERD, HTN	Participant fell without incident and was medically cleared to continue ECT

ECT=Electroconvulsive therapy, UTI=Urinary tract infection, CHF=Congestive heart failure, COPD= Chronic obstructive pulmonary disease, HTN=hypertension; GERD= Gastroesophageal reflux disease; CVA= cerebrovascular accident, CL= Chronic lymphocytic leukemia, NOS=Not otherwise specified;