The Cognitive Effects of Electroconvulsive Therapy: A Critical Review

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ABSTRACT

In this article, we discuss the neurocognitive domains affected by electroconvulsive therapy (ECT), moderators of ECT-related cognitive change, and cognitive outcomes in high-risk populations, as well as compare the cognitive effects ECT to other common treatments for refractory depression. Despite ECT being one of the oldest and most common treatments for refractory depression, various approaches to ECT (ie, strength, wavelength, and electrode placement), use of clinical convenience samples, and employment of varied and often inadequate methods of neurocognitive assessment have contributed to ongoing confusion regarding the nature and severity of post-treatment cognitive side effects. Although findings suggest that most healthy adults return to neurocognitive baseline within a few days after treatment, older adults and those with premorbid neurological impairment may be at an increased risk of prolonged mental status changes post-ECT. Employment of comprehensive neuropsychological batteries versus screening measures may assist in further understanding the nature and course of post-treatment cognitive side effects. **[*Psychiatr Ann*. 2019;49(4):152-156.]**

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Disclosure: The authors have no relevant financial relationships to disclose.

doi:10.3928/00485713-20190313-02

Electroconvulsive therapy (ECT) is a well-established and highly effective treatment for depression, and it remains one of the most effective therapies for acute remittal of refractory psychotic symptoms.[1] Despite its safety and efficacy, however, psychiatric practitioners often report a lack of clarity regarding research findings concerning the cognitive sequelae of ECT, in part due to marked differences in approach to study design, methodology, clinical populations, neuropsychological assessment, and long-term follow-up. Consequently, neurocognitive findings can appear vague and at times contradictory, which can cause confusion among both patients and providers. In this review, we summarize the neurocognitive domains and treatment effects associated with ECT, moderating factors that may affect cognitive outcomes, and cognitive effects of ECT in special and high-risk populations. We also compare ECT to other treatments for refractory depression with respect to risk of neurocognitive side effects.

NEUROCOGNITIVE DOMAINS

A number of neurocognitive domains have been implicated in ECT, including processing speed, basic attention, working memory, mental flexibility, visuospatial processing, and anterograde memory,[1] potentially implicating a wide variety of cortical and subcortical neural networks. This variability in findings appears to primarily reflect methodological differences in ECT parameters (ie, waveform, placement, strength, duration of treatment) as well as in approach and operational definition of cognitive assessment. Several studies have used brief screening instruments such as the Mini-Mental Status Examination (MMSE), which although easy to administer and interpret, provides limited information regarding the nature and extent of cognitive impairment or improvement after ECT treatment. Accordingly, ECT studies using the MMSE to measure cognitive outcomes demonstrate small acute/subacute declines in global cognitive functioning, which may be useful in documenting transient symptoms in post-ECT delirium/confusion, but are uninformative in regards to specific domains

of reduced cognitive capacity.[1] In contrast, studies that have employed comprehensive neuropsychological batteries demonstrate domain-specific cognitive changes in anterograde verbal and visual memory, speed of processing, executive functions, attention, generative fluency, and visuospatial processing.[2-7] Although findings are somewhat varied, recent meta-analyses suggest the most prominent deficits are on measures of attentional/executive control (ie, tests measuring cognitive flexibility, inhibitory control, and processing speed) and auditory verbal learning/recall (ie, unstructured list learning), a memory task that is also strongly correlated with executive functioning.[1,8] In addition, one of the greatest concerns among patients and providers is the risk for memory loss after ECT treatment. Although updated findings suggest minimal changes in episodic visual and verbal memory, research suggests that autobiographical memories, especially those made within the last 6 to 36 months prior to treatment, may be especially vulnerable to ECT.[9,10] However, findings also suggest that symptoms generally resolve within 6 months post-ECT, although subjective complaints of autobiographical memory decline can persist further.[9]

DURATION OF COGNITIVE SIDE EFFECTS

It is now well accepted that ECT does not result in permanent cognitive changes; however, findings regarding the duration of post-ECT cognitive changes remain mixed. Acute transient disorientation has been well established across ECT modalities,[11,12] with one study suggesting a common reorientation sequence of first to person, then to place, and finally to time.[13] Disorientation usually resolves quickly after ECT treatment, although is likely to be prolonged with increased risk of post-ECT delirium in the context of advanced age and comorbid neurological disease.[13,14] Immediate decline in visual and verbal anterograde memory, increased reaction times, recall for autobiographical information, and impaired performance on cognitive screening measures have also been documented, although they appear to be more closely tied to electrode placement and width of electrical pulse.[3,15,16] Meta-analyses indicate subacute decline in processing speed and medium to large deficits in cognitive flexibility, inhibitory control, and verbal episodic memory up to 3 days after final ECT treatment, with greater impairments in spontaneous retrieval versus encoding of novel unstructured verbal information.[1] Findings also suggest that most other cognitive domains return to baseline functioning within 2 weeks after the conclusion of the ECT course.[1]

Contemporary research does not support permanent cognitive deficits related to ECT, although there is a paucity of studies whose follow-up periods extend beyond 6 months.[6,11] In fact, several studies demonstrate neurocognitive improvement relative to baseline at short-term and long-term follow-up in the domains of memory, processing speed, and attention/working memory.[1,17-19] As depression causes a neurocognitive profile of reduced executive functioning, psychomotor retardation, and decreased encoding and retrieval of novel information,[20] (the same domains that appear to improve after ECT treatment) the improvement is likely secondary to relief of depressive symptoms. Conversely, studies demonstrating lasting neurocognitive deficits or post-ECT cognitive decline may reflect true ECT-related neurocognitive impairment, but they might instead demonstrate residual cognitive deficits associated with depression, which have been shown to persist beyond the remittal of depressive symptoms.[21] At present, many of the studies of ECT-related cognitive change are drawn from small samples of patients with treatment-resistant depression and other psychiatric comorbidities, thus complicating interpretation of research findings.

MODERATING FACTORS: WAVEFORM, PLACEMENT, STRENGTH, AND NUMBER OF TREATMENTS

As mentioned above, several moderating factors related to treatment parameters, cognitive measurement, and nonrandom sampling likely contribute to the heterogeneity in findings regarding cognitive side effects of ECT. With respect to treatment waveform, studies report associated risks and benefits for each administration type (ie, sine wave vs brief pulse vs ultrabrief pulse) in terms of treatment efficacy and potential cognitive harm. Historically, ECT was administered using longer sine waves, although this has since largely been abandoned due to increased risk of cognitive side effects, including greater visual memory impairment and slowed reaction times.[3] Instead, most current treatment is performed using brief (0.5-1.5 ms) or ultra-brief (0.3 ms) pulse width ECT. Although data have been variable, there has been some evidence to suggest that ultrabrief ECT may have fewer cognitive side effects, particularly in the domains of visual and verbal retentive memory and autobiographical information, although it may be somewhat inferior in reducing depressive symptoms and thus may require more treatment sessions.[22,23]

In addition to treatment waveform, cranial electrode placement (unilateral vs bilateral, frontal vs fronto-temporal) has also been implicated as a moderator of cognitive outcomes. Historically, bilateral electrode placement has been shown to have the greatest acute cognitive side effects, including more severe postictal confusion and poorer performance on cognitive screeners and tasks of visual and verbal memory,[10,16,24] including in comparison to unilateral treatment or medication alone.[25] Furthermore, one study found that compared to patients who received right unilateral ECT and experienced post-treatment cognitive improvement, patients receiving bilateral ECT did not experience any significant cognitive gains and quickly returned to

pre-ECT cognitive baseline.[3] In contrast, many studies have found the bi-temporal and right unilateral methods to be comparable in efficacy and degree of cognitive side effects, although one meta-analysis found bi-temporal placement to be associated with superior MMSE scores at short-term follow-up and superior working memory performance at long-term follow up.[1,26] Comparison of bi-temporal versus bi-frontal versus right unilateral placement indicates similar efficacy in reducing depression, and with only modest to negligible differences in cognitive outcomes.[27-29]

The effect of charge dosage on cognition remains less clear and varies among patients, although it appears that the application of supra-seizure threshold treatment is an important moderator of cognitive outcomes. Significant supra-threshold dosing is associated with numerous cognitive issues, including reduced reorientation and anterograde and retrograde memory deficits.[15,30] Finally, the effects of repeat dosing, number of treatments, and long-term treatment/maintenance ECT also remain largely unknown due to the lack of controlled follow-up studies. Assessment of cognitive side effects of maintenance ECT has been limited, with most studies only employing gross cognitive screening measures. Despite this, most studies suggest no cognitive deterioration related to ECT, including in comparison to maintenance pharmacotherapy and among patients receiving up to 12 years of maintenance ECT.[31-33]

SPECIAL POPULATIONS

Over the course of ECT's history, there have been concerns regarding an increased risk of cognitive side effects for select populations, including youth, women, and older adults. The use of ECT in pediatric populations is considerably less prevalent and occurs rarely in children younger than age 12 years (as ECT for people younger than age 13 years is not FDA-approved and therefore is "off label"), although it may be considered an option should comorbid neurodevelopmental disorders be ruled out and refractory psychiatric symptoms persist. Large-scale randomized controlled trials investigating the cognitive effects of ECT in children and adolescents are scant, and case studies have yielded mixed findings. Cohen et al.[34] found no long-term side effects associated with ECT among an adolescent population, whereas Zhand et al.[35] found that although depression symptoms remitted, adolescents receiving ECT treatment reported subjective long-term cognitive complaints.

ECT is considered safe and effective for women, even during pregnancy and the perinatal period, although there is some evidence that women may be more sensitive to cognitive changes associated with ECT. Sackeim et al.[3] noted that retrograde amnesia associated with bilateral ECT was more pronounced for women, whereas Brus et al.[36] noted significantly higher subjective memory complaints among women after ECT. One theory as to why there are significant differences in cognitive effects by sex suggests that due to women's generally lower seizure threshold, they may be more susceptible to greater supra-threshold dosing compared to men.[24]

Similar to the above-noted populations, findings detailing cognitive side effects of ECT among older adults are heterogeneous, likely secondary to small sample sizes, limited cognitive screening measures, and lack of control samples. A retrospective review of studies documenting cognitive changes associated with single-course and maintenance ECT among the elderly found that most participants reported improvement in cognition after ECT treatment, including those with documented premorbid cognitive impairment; however, although cognitive screening scores remained stable at 1-year follow-up among maintenance ECT patients, findings also suggested persistent deficits in encoding of verbal information and executive control.[37] Similarly, the review by Gardner and O'Connor[38] from 2008 reported interictal confusion and decreased processing speed, with otherwise mixed neurocognitive findings among older adults. Among studies that identify patients with mild cognitive impairment or dementia, findings indicate that healthy older adults and those with mild cognitive impairment experience cognitive improvement post-ECT, whereas patients with dementia who were receiving pharmacological treatment for cognitive decline experienced some cognitive improvement after the sixth ECT session;[39] among those with a history of dementia and no pharmacological treatment of cognitive symptoms, cognition generally declined post-ECT.[39] Notably, baseline MMSE scores appeared to be the best predictor of cognitive decline among the dementia group at short-term and long-term follow-up,[39] suggesting that ECT may be contraindicated for older adults with preexisting cognitive impairment.

EFFICACY AND RISKS COMPARED TO OTHER TREATMENTS FOR REFRACTORY DEPRESSION

To our knowledge, no systematic investigations regarding long-term cognitive effects of ECT versus pharmacological therapy have been conducted. Although selective serotonin reuptake inhibitors (SSRIs) are generally considered relatively safe and effective, some studies have indicated cognitive decline after treatment with SSRIs among older adults with increased risk of dementia.[40] In contrast, a recent meta-analysis of randomized controlled trials investigating the cognitive effects of SSRIs, selective norepinephrine reuptake inhibitors, tricyclic antidepressants, and norepinephrine–dopamine reuptake inhibitor found an overall positive effect on psychomotor processing speed and delayed memory recall,[41] and population studies suggest no independent contribution of antidepres-

sants to cognitive decline in older adults when accounting for psychiatric status, medical comorbidities, and anticholinergic effects.[42] Taken together, these findings suggest that ECT is associated with relatively greater cognitive side effects than pharmacotherapy, although neither treatment is associated with permanent cognitive deficits.

With respect to transcranial magnetic stimulation (TMS), a systematic review comprised of a total of six studies found that ECT and TMS were largely comparable with respect to acute cognitive changes, and ECT may be the favored treatment given relatively minimal cognitive side effects and greater improvement of depressive symptoms.[43] However, given the small scope of the review, it is clear that significantly more research is needed.

DISCUSSION AND AREAS FOR FUTURE RESEARCH

Overall, although research findings regarding the cognitive sequelae of ECT are limited by convenience samples and variability in ECT and cognitive measurement methodology, several broad trends do emerge. Evidence suggests that cognitive side effects are characterized by acute postictal confusion followed by residual impairment in one or more aspects of cognition (ie, anterograde memory, processing speed, executive functions, attention, generative fluency, or visuospatial processing) that typically resolves within several days to 2 weeks after treatment. Further, whereas some patients may experience retrograde amnesia for autobiographical events that occurred in the months preceding ECT, findings suggest that the decline is generally mild and resolves within 6 months post-ECT, although subjective complaints can persist further. Advanced age and the presence of untreated dementia or neurologic disease at baseline appear to be risk factors for greater post-ECT cognitive decline. There is some evidence that cognitive sequelae may be mitigated with certain treatment parameters such as ultra-brief pulse width and right unilateral electrode placement.

Although a degree of heterogeneity in findings is expected given the use of clinical samples, future research efforts would greatly benefit from the use of a standardized neuropsychological assessment approach to better understand ECT-related cognitive changes over the course of treatment, and to better differentiate between cognitive side-effects of ECT and cognitive changes related to improved or refractory psychiatric symptoms. This is particularly relevant in addressing questions related to maintenance and long-term ECT, as only a handful of studies addressed cognition beyond 6 months of follow-up. Furthermore, employment of normed comprehensive cognitive assessment tools (as opposed to screening measures) in ECT clinical research should result in improved understanding of the possible risks and advantages of treatment in high-risk or special populations, including older adults or those with comorbid neurologic disease. Finally, although ECT poses no clear risk of cognitive decline in healthy people, studies comparing ECT to other methods of treatment for refractory depression should be encouraged.

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