

Electroconvulsive Therapy for Obsessive-Compulsive Disorder: A Systematic Review

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ABSTRACT

Objective: Surgical therapies for treatment-refractory obsessive-compulsive disorder (OCD), such as deep brain stimulation or psychosurgery, remain unattainable for many patients. Despite the long-held view that electroconvulsive therapy (ECT) is an ineffective treatment for OCD, there is no systematic review to support or refute this claim, which is the basis of the current review.

Data Sources: A systematic search of MEDLINE, Web of Science, Scopus, and LILACS databases was conducted on December 22, 2013, using the terms *obsessive-compulsive disorder* and *electroconvulsive therapy*. Reference lists, specific journals, and clinical trial registries were also scrutinized. No date or language limitation was imposed on the search.

Study Selection: After irrelevant and redundant records from the 500 identified titles were excluded, the 50 articles reporting the acute treatment effects of ECT in OCD and related constructs (involving a total of 279 patients) were analyzed for this study.

Data Extraction: The relevant sociodemographic, clinical, and outcome data of individual cases were extracted. Data from individual cases were used to compare the characteristics of responders versus nonresponders to ECT.

Results: Most selected records were case reports/series; there were no randomized controlled trials. A positive response was reported in 60.4% of the 265 cases in which individual responses to ECT were available. ECT responders exhibited a significantly later onset of OCD symptoms ($P=.003$), were more frequently nondepressed ($P=.009$), more commonly reported being treated with ECT for severe OCD ($P=.01$), and received a fewer number of ECT sessions ($P=.03$). ECT responders were also less frequently previously treated with adequate trials of serotonin reuptake inhibitors ($P=.05$) and cognitive-behavioral therapy ($P=.005$).

Conclusions: Although 60% of the reported cases reviewed exhibited some form of a positive response to ECT, it cannot be stated that this provides evidence that ECT is indeed effective for OCD.

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Obsessive-compulsive disorder (OCD) is a frequent and debilitating condition¹ whose symptoms tend to group into 4 different thematic clusters: contamination/washing, taboo thoughts/checking, symmetry/ordering, and hoarding/collecting themes.² Available treatments for OCD include serotonin reuptake inhibitors (SRIs) administered in maximum tolerated doses for a sufficient period of time (minimum of 12 weeks)³ and/or cognitive-behavioral therapy (CBT) in the form of exposure and response prevention.⁴ In general, 40% to 60% of OCD patients show what have been considered favorable responses to these forms of treatment,⁵ usually defined as at least 25% decrease in the initial score on the Yale-Brown Obsessive-Compulsive Scale (YBOCS) plus a Clinical Global Impressions-Improvement scale (CGI-I) score of 1 or 2.⁶

Adequate management of OCD patients who do not respond to an initial trial with selective SRI and/or CBT involves the sequential administration of a different SRI (including clomipramine) that, if needed, can be augmented by CBT or dopamine blockers for those who were not exposed to it.^{7,8} However, experts have suggested that up to 10% of treatment-seeking OCD patients will show inadequate response to these strategies.⁹ Modern neurosurgical therapies for treatment-refractory OCD, such as deep brain stimulation¹⁰ or limbic system surgery,¹¹ remain unfeasible for many patients due to costs and/or access and have not been fully evaluated in regard to efficacy and safety. Theoretically, it may be that treatment-refractory OCD patients could be saved from more invasive procedures if a trial with electroconvulsive therapy (ECT) were undertaken.¹² However, it has been argued that the evidence for the efficacy of ECT in OCD remains sparse.^{13–15}

Some ECT experts are more optimistic about the occasional utility of ECT in treatment-refractory OCD,¹² although this mode of treatment is not mentioned in the official algorithms developed by OCD specialists.¹⁶ Historical reasons, lack of good quality data, and personal experiences may be at the core of this therapeutic inconsistency. For instance, some authors have suggested that defining and characterizing a subset of potential ECT responders should be subject to a definitive controlled study,¹³ while others have argued that the burden of experience does not suggest that controlled comparisons would be helpful.¹⁷ Indeed, despite many disparate opinions regarding the efficacy of ECT for OCD, we are not aware of any study that reviewed its efficacy in a systematic way.

In this review, we analyzed studies reporting the socio-demographic, clinical, and outcome features of OCD patients treated with ECT as described in the medical literature. Our objectives were 2-fold: (1) to assess the effectiveness and determine the rates of response to ECT among OCD patients and (2) to identify features related to a positive treatment response of OCD to ECT.

- Although it cannot be unequivocally stated that ECT is effective for OCD, 60% of the ECT-treated OCD cases exhibited some positive response.
- Our findings suggest that ECT responders had later onset of OCD symptoms, were more frequently nondepressed and treated for severe OCD, and received fewer ECT sessions.
- Clinicians using ECT in OCD, however, have frequently ignored effective doses and duration of SRIs and cognitive-behavioral therapy and may have prescribed ECT prematurely in a number of cases.

METHOD

Criteria for Considering Studies for This Review

We searched for randomized trials, observational studies, retrospective investigations, case series, and single case reports of ECT use in OCD (*DSM* or *ICD* diagnosed) and corresponding constructs (eg, obsessional neurosis, obsessional psychoneurosis, obsessional states, and psychasthenia). Studies in which OCD was comorbid to other psychiatric conditions were also included as long as the specific effect of ECT on OCD was detailed. Studies focusing on other OCD spectrum conditions (eg, body dysmorphic disorder, Tourette syndrome, self-mutilating behaviors), medication-induced OCD (eg, OCD due to clozapine), or OCD resulting from neurodegenerative conditions (eg, Pick disease, progressive supranuclear palsy, neuroacanthocytosis) were preliminarily excluded from our analysis.

The intervention of interest in this review was unilateral or bilateral ECT. Studies assessing the acute treatment effects of ECT in OCD were included regardless of concomitant reports on the effectiveness of maintenance ECT. We also included studies that maintained other forms of treatments during ECT (ie, SRI) or that included treatments starting simultaneously with ECT. The primary outcome measure was the authors' categorical report of significant reduction in the severity of obsessive-compulsive symptoms as a result of ECT. However, we also assessed whether patients were evaluated pretreatment and posttreatment with the Yale-Brown Obsessive Compulsive Scale (YBOCS).^{18,19}

Search Methods for Identification of Studies

A systematic search of MEDLINE, Web of Science, Scopus, and LILACS databases was conducted on December 22, 2013, using the terms *obsessive-compulsive disorder* and *electroconvulsive therapy* as MeSH major topics (in MEDLINE); topics (in Web of Science); article titles, abstracts, and keywords (on Scopus); and health sciences descriptors (in LILACS). Reference lists of selected articles, *Journal of ECT*, *Convulsive Therapy*, *Brain Stimulation*, and relevant books^{20–26} were also hand-searched and cross-referenced for any additional studies that could be included. The first author also searched the Cochrane Library and clinical trial

registries for ongoing or completed trials (ClinicalTrials.gov and World Health Organization International Clinical Trials Registry Platform). No date limit or language restrictions were applied. Editorials, commentaries, reviews, lectures, and letters not containing original data were excluded.

Data Collection and Analysis

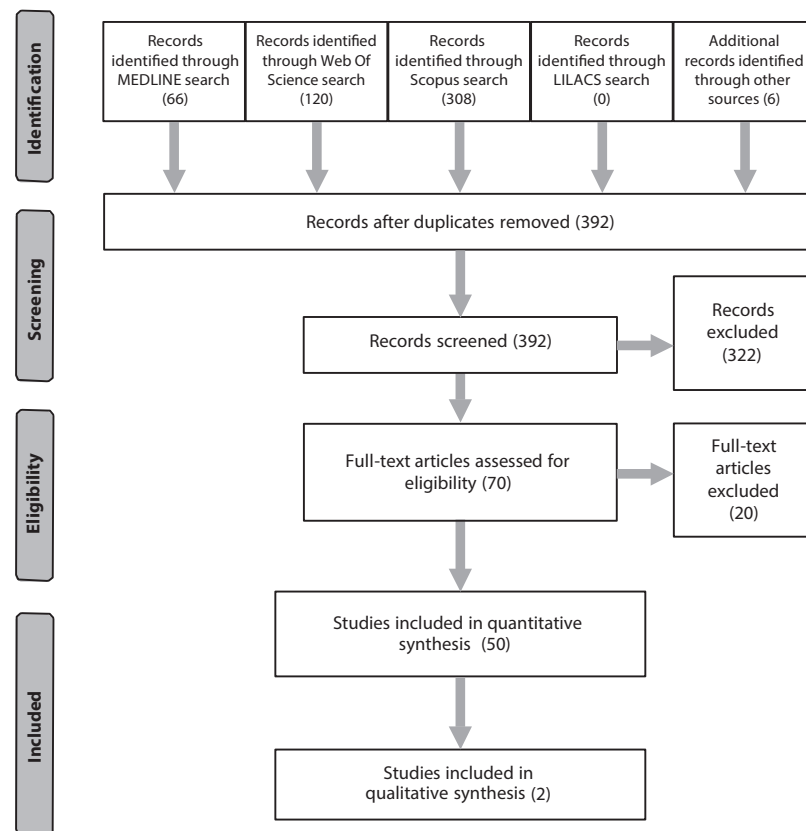
Selection of studies. All citations identified as potentially relevant by the literature search were closely inspected, and all redundant records identified through different databases searched were excluded from the review. Relevant articles were identified and screened for thematic suitability. This was followed by an assessment of eligibility according to inclusion and exclusion criteria. If this information was unclear, an attempt was made to contact the corresponding author(s). Discrepancies regarding inclusion and exclusion of the studies were resolved by consensus within the team of authors.

Analysis of individual studies. When appropriate, studies were assessed for quality following the Cochrane criteria²⁷ including (1) random sequence generation; (2) allocation concealment; (3) blinding of participants, personnel, and outcome assessors; (4) selective reporting; and (5) incomplete outcome data. We specifically described only studies with more than 1 treatment arm. We also assessed the likelihood of any publication bias by comparing the rates of positive treatment response between single case reports and case series versus studies including more than 1 treatment arm.

Analysis of individual cases. If studies were selected for inclusion, we collected and entered the individual data into an SPSS database (SPSS Inc, Chicago, Illinois), including demographic and clinical factors (ie, age, gender, age at OCD onset, OCD symptom dimensions, pretreatment YBOCS severity scores, course of illness, comorbidity patterns, and reasons for ECT administration), treatment history (ie, whether there was history of adequate treatment with an SRI in terms of dose and minimum duration and/or of CBT), ECT parameters (ie, whether ECT was bilateral or unilateral, the total number of ECT sessions, and the number of ECT sessions per week), and treatment response (ie, posttreatment YBOCS severity scores, the presence of response of OCD to ECT according to the author's opinion, the presence of response of major depression to ECT according to the author's opinion, duration of follow-up, and relapse or worsening of OCD).

A priori analyses of ECT responders versus nonresponders were planned based on the following variables:

1. age
2. gender
3. age at OCD onset
4. OCD symptom dimensions
5. course of illness
6. comorbidity patterns
7. reasons for ECT administration
8. previous exposure to an SRI

Figure 1. Description of Identification, Screening, Eligibility, and Inclusion of Studies in This Review According to the PRISMA Recommendations

9. previous exposure to an adequate trial with SRI in terms of either maximum dose tolerated or minimum duration of 12 weeks
10. previous exposure to CBT
11. bilateral or unilateral ECT
12. total number of ECT sessions
13. number of ECT sessions per week

RESULTS

Study Selection

The literature search identified 500 potential titles. After all redundant records were identified and removed, a total of 392 articles remained. These remaining articles were screened for thematic suitability by checking titles and abstracts, which generated 70 articles assessed as being suitable for eligibility based on the specific criteria. A total of 20 articles were excluded on the basis that they (1) failed to adequately describe the responses of OCD to ECT^{28–32}; (2) reported the ECT-related response of depression rather than OCD³³; (3) described responses of OCD symptoms to ECT in the context of neurodegenerative conditions,^{34–38} drug-induced OCD,³⁹ other OCD spectrum disorders,^{40,41} non-OCD spectrum psychiatric disorders,⁴² or unclear diagnoses⁴³; (4) reported cases already described in the selected literature⁴⁴,

or (5) merely reviewed the literature or provided an opinion concerning the use of ECT or other neurostimulatory approaches in OCD.^{45–47} For a description of the identification, screening, eligibility, and inclusion of studies according to PRISMA recommendations,⁴⁸ see Figure 1.

Analyses of Individual Studies

As no randomized controlled trial on the efficacy of ECT in OCD was found, we focused our systematic review on 1 non-randomized or quasi-randomized study,⁴⁹ 1 case-control study,⁵⁰ 1 cohort study,⁵¹ 22 case series, and 25 single case reports (see Table 1 for a description of different aspects of each selected study). As we have noted, only 2 studies included more than 1 treatment group, at least 1 describing response to ECT.^{49,51} Although the study by Ferrão et al⁵⁰ compared 2 groups (treatment-refractory vs non-treatment-refractory OCD patients) and provided data for individual case analyses, we decided not to describe it since it was not focused on the response to ECT.

The first study⁴⁹ was presented as a poster at a European conference meeting. We considered it a nonrandomized trial, as no clear information on allocation of study subjects to each therapeutic approach was provided. In this study, a total of 66 inpatients with OCD were assigned to

ECT (17 patients) versus SRI or “cyclic” antidepressant (49 patients) treatment arms. Efficacy of ECT was assessed with YBOCS, Hamilton Depression Rating Scale (HDRS), and CGI. According to the authors, CGI scores showed marked improvement in 60% of both groups. However, patients treated with ECT (average = 7.9 sessions/patient) showed more days of hospitalization (37.7 vs 30.1 days).

While this study suggests that ECT is as effective as an antidepressant (ie, improved CGI score was noted) in inpatients with severe OCD, a number of gaps make it difficult to judge the methodological qualities of the study. For instance, the study by Garrido⁴⁹ did not include information concerning the scores on the YBOCS and HDRS. Also, other than similar age and gender, there was too little information on possible differences between the 2 groups (ECT vs antidepressants) in terms of baseline characteristics. Therefore, it was not clear how interventions were allocated to participants and if there was allocation concealment. We were also unable to exclude systematic differences in the care that was provided (ie, duration of treatment was not available for each treatment arm and it is unclear when endpoint analyses were done).

In addition, the study by Garrido⁴⁹ was hampered by the fact that no sham ECT was mentioned in the antidepressant-treated group or placebo pill in the ECT group, a limitation that, together with the above mentioned differences in the

Table 1. List of Studies Included in the Present Systematic Review

Study First Author and Year	ECT Patients, N	Design	Principal Indication for ECT	Response Rate, %
Lins-Martins, 2014 ⁵²	5	Case series	MDE (4 patients) and mania/agitation (1 patient)	0
Bülbül, 2013 ⁵³	1	Case report	MDE	100
Sheehan, 2013 ⁵⁴	2	Case series	Treatment resistance	0
Grover, 2013 ⁵⁵	1	Case report	Treatment resistance	0
D'Urso, 2012 ⁵⁶	1	Case report	Catatonia	100
Raveendranathan, 2012 ⁵⁷	1	Case report	Treatment resistance	100
Makhinson, 2012 ⁵⁸	1	Case report	Catatonia	100
Riestra, 2011 ⁵⁹	1	Case report	Treatment resistance	0
Loi, 2010 ⁶⁰	1	Case report	Severe OCD	100
Tomruk, 2010 ⁶¹	2	Case series	Treatment resistance (1 patient) and severe OCD (1 patient)	100
Hanisch, 2009 ⁶²	1	Case report	Severe OCD	100
Nilsson, 2009 ⁶³	1	Case report	Severe OCD	100
Talaei, 2009 ⁶⁴	1	Case report	Treatment resistance	0
Fisher, 2008 ⁶⁵	1	Case report	Treatment resistance	100
Schindler, 2008 ⁶⁶	1	Case report	Treatment resistance	0
García Valls, 2007 ⁶⁷	1	Case report	Mania/agitation	100
Ferrão, 2006 ⁵⁰	4	Case-control study	Treatment resistance	0
Chaves, 2005 ⁶⁸	1	Case report	Mania/agitation	100
Strassnig, 2004 ⁶⁹	1	Case report	Severe OCD	100
Thomas, 2003 ⁷⁰	1	Case report	MDE	100
Polosan, 2003 ⁷¹	2	Case series	Treatment resistance	0
Fukish, 2003 ⁷²	1	Case report	Severe OCD	100
Chung, 2001 ⁷³	1	Case report	Treatment resistance	0
Ganesan, 2001 ⁷⁴	1	Case report	Unclear	100
Swartz, 1999 ⁷⁵	3	Case series	Severe OCD	100
Shusta, 1999 ⁷⁶	1	Case report	Unclear	0
Garrido, 1998 ⁴⁹	17	NRT	Unclear	58.8
Lavin, 1996 ⁷⁷	1	Case report	Treatment resistance	100
Filer, 1996 ⁷⁸	1	Case report	MDE	100
Wohlfahrt, 1996 ⁷⁹	1	Case report	Drug side effects	100
Beale, 1995 ¹²	3	Case series	Treatment resistance	100
Casey, 1994 ⁸⁰	1	Case report	MDE	100
Maletzky, 1994 ⁸¹	32	Case series	Treatment resistance	At least 56.2
Husain, 1993 ⁸²	1	Case report	Treatment resistance	100
Sichel, 1993 ⁸³	1	Case report	MDE	0
Schott, 1992 ⁸⁴	1	Case report	Treatment resistance	0
Soyka, 1991 ⁸⁵	1	Case report	Treatment resistance	100
Warneke, 1989 ⁸⁶	2	Case series	Treatment resistance	0.0
Khanna, 1988 ⁸⁷	9	Case series	Treatment resistance	77.8
Mellman, 1984 ⁸⁸	1	Case report	Previous response	100
Dubois, 1984 ⁵¹	19	Cohort study	Unclear	78.9
Walter, 1972 ⁸⁹	80	Case series	Treatment resistance	43.8
Grimshaw, 1965 ⁹⁰	32	Case series	Unclear	50
Korson, 1949 ⁹¹	1	Case report	Severe OCD	100
Bini, 1947 ⁹²	4	Case series	Unclear	75
Hamilton, 1947 ⁹³	9	Case series	Unclear	88.9
Milligan, 1946 ⁹⁴	11	Case series	Unclear	100
Kerman, 1945 ⁹⁵	1	Case report	Unclear	100
Moriarty, 1943 ⁹⁶	5	Case series	Unclear	100
Smith, 1943 ⁹⁷	7	Case series	Unclear	14.3

Abbreviations: ECT = electroconvulsive therapy, MDE = major depressive episode; NRT = nonrandomized trial, OCD = obsessive-compulsive disorder.

treatment provided, has the potential to affect blinding. Although outcomes were determined similarly (with CGI) in both treatment groups, the already mentioned lack of information on baseline and endpoint YBOCS and HDRS scores does not exclude the possibility of selective reporting of outcomes. Finally, no information on dropout rates was provided, which further limits the generalizability of the treatment effects.

The second investigation including a comparative group was a cohort study involving heterogeneous intervention

strategies in each treatment arm.⁵¹ More specifically, it included 14 patients treated with oral antidepressants (8 clomipramine, 4 chlorpromazine, 1 phenelzine, and 1 sulpiride), 10 patients treated with intravenous followed by oral antidepressants (9 clomipramine and 1 amitriptyline), 9 patients treated with intravenous plus oral antidepressants (specific drugs not mentioned) plus ECT, and 10 patients treated with oral antidepressants (9 clomipramine—associated with amitriptyline in 1 and imipramine in another—and isolated amitriptyline in another) plus ECT. At the end of 3 months of treatment, 12 (85.7%), 6 (60%), 7 (78%), and 6 (60%) patients, respectively, were described as “cured.” Perhaps because of methodological limitations inherent to their approach, these authors did not compare rates of response between the groups or discuss the superiority of any treatment dispensed. Instead, they described the improvement rates for the whole group as an average of 69.7%.

Predictably, the therapeutic flexibility reported in this cohort study by Dubois⁵¹ introduced systematic differences in the treatment provided, wherein patients received different combinations of therapeutic strategies (ie, oral antidepressants, intravenous antidepressants, hypnotics, and ECT) according to different clinical demands. In this study, treatment arms were defined a posteriori, thus hindering any attempt to perform reliable comparisons of response rates between groups. Apparently, in this study, patients whose illness was particularly severe were more likely to be treated with ECT and intravenous antidepressants, although this was not explicitly mentioned.

In terms of publication bias, there was no difference in positive response to ECT described in case reports and case series (60.0%) vs studies with more than 1 treatment arm (62.5%; $\chi^2_1 = 0.09$, $P = .76$). However, patients showing positive responses to ECT were more commonly described in recent studies (66.3%) as compared to older ones, albeit without statistical significance (57.5%; $\chi^2_1 = 1.84$, $P = .17$). Finally, we found an increased representation of case report descriptions of patients from more recent studies (ie, those published after 1990; 91.7%) compared to older ones (8.3%; $\chi^2_1 = 37.2$, $P < .001$).

Analyses of Individual Cases

A total of 279 OCD patients were treated with ECT: 17 in a nonrandomized or quasi-randomized trial, 19 in a cohort study, 4 in a case control study, 214 in case series, and 25 in case reports (Table 1). However, individual response rates to

ECT were available in only 265 cases. In these cases, 60.4% ($n = 160$) showed response to ECT according to their authors' opinion.* However, studies reporting YBOCS scores were almost negligible (ie, this information was available in only 7 cases of the total sample; 2.5%). If only cases published after the widespread availability of SRI were considered (ie, those published after 1990), the rate of treatment response was roughly comparable (ie, 66.3%). In addition, only 7.2% of the more recent cases included information on YBOCS scores. In these patients, YBOCS scores varied from 23 to 40, with a mean of 36.2 (SD = 6.3) points. The mean endpoint YBOCS score in the reported cases was 8.5 (SD = 7.9).

Information on gender was available in 106 valid cases, the slight majority of patients being female (51.9%). Age at OCD onset was accessible in 126 patients and varied from 8 to 81 years, with a mean (SD) of 27.1 (8.5) years. Among the valid cases, the patient's main symptom dimensions were described as taboo or forbidden thoughts (with aggressive, sexual, or religious content) and/or checking compulsions in 49 of 61 patients (80.3%), contamination thoughts and/or washing compulsions in 36 of 61 (59.0%), symmetry/ordering symptoms in 11 of 42 (26.2%), hoarding/collecting symptoms in 2 of 42 (4.8%), and other OCD symptoms (eg, somatic obsessions) in 16 of 62 (25.8%). Major depression at the time of ECT was reported in 56 of 97 cases (57.7%), nonaffective psychosis (eg, schizophrenia) in 5 of 38 (13.2%), and mania in 1 of 51 (2.0%). It was possible to determine the course of OCD in only a small minority of cases (12), which revealed that it was episodic in 66.7% ($n = 8$) and chronic in 33.3% of patients ($n = 4$).

According to the reviewed studies, "treatment-resistant" OCD was the most frequent indication for ECT, being reported in 147 (90.2%) of the 163 valid cases. However, previous exposure to CBT or to a trial with an SRI was described in only 37.6% (59/157) and 21.5% (59/275) of the cases, respectively; of note, in only 16.9% of SRI-treated patients (10/59) was there enough information to consider patients as being adequately treated, based on dose and duration of treatment. Even if one focuses on studies published after the widespread availability of SRI on the market (post-1990), the rates of SRI use remain considerably low; in these studies, only 52.7% of OCD patients (49/93) were reported as having been treated with an SRI, and, among these, only 18.4% (9/49) were described as having been treated with adequate doses of SRI for at least 12 weeks.

Besides treatment-resistant OCD, other reasons for prescription of ECT were assessed in our study. For instance, ECT was prescribed for severe OCD in 114 of 186 patients (61.3%); major depression with suicidality in 96 of 163 (58.9%); severe mania, psychosis, or agitation in 5 of 163 (3.1%); previous response to ECT or seizure in 4 of 163 (2.5%), catatonia in 3 of 163 (1.8%), and drug-related side effects in 1 of 182 (0.5%). From 90 valid cases, ECT was bilateral in 75 of 90 (83.3%), unilateral in 6 of 90 (6.7%), and

alternating between unilateral and bilateral in the remaining 9 of 90 (10.0%). Total number of ECT sessions was available in 121 cases, with a mean (SD) of 9.1 (5.4) sessions per patient. Information on the weekly frequency of ECT was reported in 104 cases: it varied from 1 to 7 per week, with a mean (SD) of 3.7 (0.7) weekly sessions being employed.

Data on duration of follow-up, if any, were available in 67 cases; it varied from 1 to 24 months, with a mean (SD) of 9.9 (4.6) months. In 68 of 279 cases (24.4%), there was information on follow-up. Among these patients, 32.2% (19/59) showed relapse or worsening of OCD symptoms after ECT. The analysis of the 57 cases in which individual responses to ECT were available showed that responders displayed later onset of OCD symptoms, were more frequently nondepressed, and more commonly reported being treated with ECT for severe OCD symptoms. However, patients who responded to ECT were less frequently previously treated with adequate doses of SRI prescribed for sufficient time and less frequently provided CBT. Finally, patients who responded to ECT were treated with a lower number of ECT sessions. For detailed information on these results, see Table 2.

DISCUSSION

In this review, we were unable to find unequivocal evidence supporting the efficacy of ECT in OCD, as no randomized controlled trial (RCT) has been conducted to date. Even observational studies with comparative groups were rare. However, positive responses to ECT were reported in at least 60.4% of the total sample. Arguably, this rate of treatment response could result from the inclusion of older studies, published before the availability of more effective treatment strategies, such as SRIs and CBT and hence the inclusion of non-"treatment-resistant" patients. According to this view, focus on more recent studies would result in lower response rates to ECT. However, if only cases published after the widespread availability of SRIs are considered (ie, articles published after 1990), the rate of treatment response was roughly similar or slightly higher (ie, 66.3%).

Despite these high treatment response rates, we also found evidence that OCD patients treated with ECT have frequently received inadequate treatment prior to ECT. For instance, in just 52.7% of the most recent studies mentioned above, patients were clearly previously treated with an SRI. In addition, in only 16.9% of SRI-treated patients was enough information available on dose and duration of administration, thus suggesting that clinicians reporting ECT use in OCD have frequently ignored effective doses and duration of SRI treatment and may have prescribed ECT prematurely in a number of reported cases. This finding is particularly worrisome in the light of current doubts regarding the true efficacy of ECT in OCD. It also suggests that many patients treated effectively with ECT had more benign forms of OCD and were inaccurately labeled as "treatment resistant."

It is also worth noting that information on maintenance of gains was frequently not available, as only 24.4% of published cases were accompanied by relapses rates, with periods

*Among the 279-patient sample, information on responses to ECT was unclear in 14 of 32 patients from a single study.⁸¹

Table 2. Comparison Between Sociodemographic, Clinical, and Therapeutic Features of Patients With OCD Who Showed Negative vs Positive Responses to ECT^a

Variable	Negative Response to ECT; n=up to 20 ^b	Positive Response to ECT; n=up to 36 ^b	Statistics
Age, mean (SD), y	38.6 (12.0)	40.00 (15.1)	$Z = -0.06, P = .95$
Gender			$\chi^2 = 0.4, df = 1, P = .50$
Female	9 (47.4)	21 (56.8)	
Male	10 (52.6)	16 (43.2)	
Age at OCD onset, mean (SD), y	20.6 (4.8)	34.3 (16.7)	$Z = -2.88, P = .003$
Predominant OCD symptoms ^c			
Taboo thoughts/checking	12 (80.0)	19 (70.4)	Fisher test, $P = .72$
Contamination/washing	5 (33.3)	14 (51.9)	$\chi^2 = 1.3, df = 1, P = .24$
Symmetry/ordering symptoms	6 (40.0)	5 (18.5)	Fisher test, $P = .16$
Hoarding/collecting symptoms	0 (0.0)	2 (7.4)	Fisher test, $P = .53$
Other OCD symptoms	4 (26.7)	11 (39.3)	$\chi^2 = 0.7, df = 1, P = .41$
Course of OCD			Fisher test, $P = .09$
Chronic	2 (100.0)	2 (20.0)	
Episodic	0 (0.0)	8 (80.0)	
Major comorbidities at time of ECT			
Major depression	12 (80.0)	12 (38.7)	$\chi^2 = 6.9, df = 1, P = .009$
Schizophrenia related disorders	1 (8.3)	4 (15.4)	Fisher test, $P = 1.0$
Mania	1 (8.3)	0 (0.0)	Fisher test, $P = .3$
Indication for ECT			
Treatment resistance	10 (62.5)	16 (61.5)	$\chi^2 = 0.004, df = 1, P = .9$
Severe OCD symptoms	2 (11.8)	15 (48.4)	$\chi^2 = 6.4, df = 1, P = .01$
Major depression with suicidality	6 (37.5)	10 (38.5)	$\chi^2 = 0.004, df = 1, P = .9$
Catatonia symptoms	0 (0.0)	3 (11.5)	Fisher test, $P = .2$
Mania, psychosis, or agitation	2 (12.5)	3 (11.5)	Fisher test, $P = 1.0$
Previous response to ECT	0 (0.0)	4 (15.4)	Fisher test, $P = .2$
Drug-related side effects	0 (0.0)	1 (3.8)	Fisher test, $P = 1.0$
Treatment history			
SRI prescribed	15 (75.0)	21 (56.8)	$\chi^2 = 1.8, df = 1, P = .17$
With adequate dose and time ^d	7 (46.7)	3 (14.3)	Fisher test, $P = .05$
Antipsychotic prescribed	14 (93.3)	20 (83.3)	Fisher test, $P = .63$
CBT	10 (83.3)	8 (33.3)	$\chi^2 = 8.0, df = 1, P = .005$
ECT-related variables			
Total no. of sessions, mean (SD)	12.4 (4.4)	10.2 (7.9)	$Z = -2.1, P = .03$
Frequency of weekly application, mean (SD)	2.3 (0.5)	3.1 (1.6)	$Z = -1.2, P = .20$
Mode of application			Fisher test, $P = .6$
Unilateral	1 (11.1)	5 (21.7)	
Bilateral	8 (88.9)	18 (78.3)	

^aValues shown as n (%) unless otherwise noted.

^bData for all variables were not available in all studies.

^cPercentages do not add up to 100 because individuals typically have more than 1 symptom dimension.

^dWith a history of being treated with adequate doses of SRI for at least 12 weeks.

Abbreviations: CBT = cognitive-behavioral therapy, ECT = electroconvulsive therapy, OCD = obsessive-compulsive disorder, SRI = serotonin reuptake inhibitor.

forms of treatment in OCD (including SRI^{99–103} and CBT^{103,104}). However, severe (ie, delusional) depression has been described as a predictor of good responses to ECT,²³ an observation that is in apparent contrast to our findings. Indeed, there is evidence that depression in OCD is likely to involve different neural pathways than depression in other contexts,¹⁰⁵ a phenomenon that could explain its lower ability to predict treatment response. For instance, previous studies have shown that depression related to OCD is more responsive to serotonergic rather than noradrenergic drugs.¹⁰⁶ Finally, the fact that effective ECT was more likely to be recommended for severe, but not treatment-resistant OCD dovetails with the idea that ECT is more likely to work in noncomplicated forms of OCD.

The observation that patients who responded to ECT were less frequently previously treated with adequate trials of an SRI and less frequently provided CBT supports the earlier interpretation that ECT was more likely to treat OCD that would be otherwise treatable by conventional antiobsessional treatments, if they were prescribed. Although this finding indicates that clinicians may be misinformed about adequate treatment of OCD,^{107,108} it could still be argued that ECT may be used in situations in which SRIs and/or CBT is potentially problematic or not feasible, such as in the presence of bipolar depression^{109–115} associated with paralyzing fears about the consequences of exposure and response prevention

of follow-up that have been extremely heterogeneous. Unfortunately, deterioration in OCD symptoms was seen in more than a third of effectively treated OCD patients. While these relapse rates are somewhat lower than the 51% reported 1 year after ECT in depression,⁹⁸ they probably represent an underestimate, as information on follow-up was not available in most patients. Indeed, increased rates of relapse in ECT-treated OCD patients were already reported and suggest that, besides RCTs, studies including longer follow-up periods and treatment arms that contain relapsing prevention strategies are also needed.

Analysis of cases in which individual responses to ECT were available showed that ECT responders exhibited later onset of OCD symptoms, were more frequently nondepressed, and more commonly reported being treated with ECT for severe OCD symptoms. In fact, higher age at onset has already been associated with better responses to other

sessions.¹¹⁶ However, as reported above, comorbid depression was associated with resistance to ECT in OCD. Patients who were effectively treated with ECT were also submitted to a lower number of ECT sessions. Rather than suggesting that fewer ECT sessions are sufficient to treat OCD, this finding could equally reflect that patients who do not respond to ECT are treated more frequently with a greater number of ECT sessions in an attempt to overcome resistance.

Although we did not find an RCT, we detailed 2 studies reporting 2 or more treatment arms, at least 1 of which involved ECT administration, combined or not with other forms of treatment. Both have several limitations. First, the study by Garrido⁴⁹ comparing ECT to different antidepressants had incomplete information on a number of features and thus was likely to be affected by a range of methodological problems, including selection (unclear method of allocation to treatment), performance (poor

blinding to treatment), reporting (selective outcome reporting), and attrition bias (incomplete outcome data). In addition, the cohort study by Dubois⁵¹ had limitations that are inherent to an observational design (eg, in these studies, severely ill patients may be more likely to receive multiple treatments).

Our review has its own limitations. For instance, inclusion criteria were broad, encompassing nonrandomized trials, observational studies, and, most critically, case series and single case reports. Further, by selecting studies with OCD, obsessional neurosis and, psychasthenia, we may have included individuals who, despite having overlapping conditions on a symptom level, are conceptually different. In addition, treatment response was loosely defined, based exclusively on authors' consensual personal opinions. However, restricting our review to high-quality studies, such as those involving RCT design, *DSM*-diagnosed cases, and reporting of scores measured by valid scales (such as the YBOCS), would impede or critically limit our ability to amass a reasonable number of studies and perform appropriate statistical analyses, particularly on a topic characterized by substantial missing information.

Given that OCD has been considered by some a contraindication to ECT,^{17,117} that current treatment guidelines exclude ECT from their algorithms,¹⁶ and that modern treatments for treatment-refractory OCD cases are either invasive and irreversible (neurosurgery) or expensive (deep brain stimulation),¹¹⁸ we predicted that there would be overreporting of positive findings in case reports/series, as clinicians would feel stimulated to report their isolated cases of success using ECT in OCD. However, we did not find clear evidence for publication bias.

In spite of our findings, it is possible that the heterogeneity of OCD cases treated with ECT and the variation in outcomes reported by observers using its *DSM* definition may have obfuscated the potential role of ECT in OCD. For instance, since catatonia shows very high rates of response to ECT,¹¹⁹ it has been suggested that some low-order repetitive behaviors¹²⁰ (such as self-injury behaviors in autism¹²¹ and tics in Tourette syndrome⁴⁰) can be alternate catatonic signs that may potentially predict response to ECT. Therefore, any future systematic study of ECT in OCD should investigate whether different natural history features (eg, acuteness of onset, periodicity of relapse, duration of illness), target symptoms (eg, ticlike compulsions, motor or vocal tics, self-injurious stereotypies), or associated syndromes (eg, catatonia, mania, psychosis) might, at least theoretically, predict response to ECT in OCD.

In sum, the present state of knowledge suggests that ECT has no role in the routine treatment of OCD. Although nonrandomized and cohort studies, case series, and some single case reports have suggested beneficial effects of ECT in OCD under special circumstances, these studies are limited by a lack of standardized assessment of results, history of less than optimal treatment of OCD, and poorly defined treatment resistance. In fact, cases of OCD argued to have been effectively treated with ECT (almost 60% of the sample) were probably more benign than those described as resistant according to modern criteria. Thus, our findings suggest that OCD patients labeled treatment refractory according to current criteria would be unlikely to show any response to ECT. In patients who show some response to ECT acutely, maintenance of gains is unclear at best, as most studies did not include information on treatment follow-up.

Drug names: clomipramine (Anafranil and others), clozapine (Clozaril, FazaClo, and others), imipramine (Tofranil and others), phenelzine (Nardil).

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REFERENCES

1. Ruscio AM, Stein DJ, Chiu WT, et al. The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Mol Psychiatry*. 2010;15(1):53–63.
2. Mataix-Cols D, Rosario-Campos MC, Leckman JF. A multidimensional model of obsessive-compulsive disorder. *Am J Psychiatry*. 2005;162(2):228–238.
3. Fineberg NA, Reghunandanan S, Brown A, et al. Pharmacotherapy of obsessive-compulsive disorder: evidence-based treatment and beyond. *Aust N Z J Psychiatry*. 2013;47(2):121–141.
4. Olatunji BO, Davis ML, Powers MB, et al. Cognitive-behavioral therapy for obsessive-compulsive disorder: a meta-analysis of treatment outcome and moderators. *J Psychiatr Res*. 2013;47(1):33–41.
5. Pallanti S, Quercioli L. Treatment-refractory obsessive-compulsive disorder: methodological issues, operational definitions and therapeutic lines. *Prog Neuropsychopharmacol Biol Psychiatry*. 2006;30(3):400–412.
6. Goodman WK. Obsessive-compulsive disorder: diagnosis and treatment. *J Clin Psychiatry*. 1999;60(suppl 18):27–32.
7. Fontenelle LF, Nascimento AL, Mendlowicz MV, et al. An update on the pharmacological treatment of obsessive-compulsive disorder. *Expert Opin Pharmacother*. 2007;8(5):563–583.
8. Simpson HB, Foa EB, Liebowitz MR, et al. Cognitive-behavioral therapy vs risperidone for augmenting serotonin reuptake inhibitors in obsessive-compulsive disorder: a randomized clinical trial. *JAMA Psychiatry*. 2013;70(11):1190–1199.
9. Denys D. Pharmacotherapy of obsessive-compulsive disorder and obsessive-compulsive spectrum disorders. *Psychiatr Clin North Am*. 2006;29(2):553–584, xi.
10. de Koning PP, Figee M, van den Munckhof P, et al. Current status of deep brain stimulation for obsessive-compulsive disorder: a clinical review of different targets. *Curr Psychiatry Rep*. 2011;13(4):274–282.
11. Sheth SA, Neal J, Tangherlini F, et al. Limbic system surgery for treatment-refractory

- obsessive-compulsive disorder: a prospective long-term follow-up of 64 patients. *J Neurosurg*. 2013;118(3):491–497.
12. Beale MD, Kellner CH, Pritchett JT, et al. ECT for OCD. *J Clin Psychiatry*. 1995;56(2):81–82.
 13. Rudorfer MV. *Electroconvulsive Therapy in Treatment-Refractory Obsessive-Compulsive Disorder*. London, UK: Lawrence Erlbaum Associates, Publishers; 2000.
 14. Dell'Osso B, Altamura AC, Allen A, et al. Brain stimulation techniques in the treatment of obsessive-compulsive disorder: current and future directions. *CNS Spectr*. 2005;10(12):966–979, 983.
 15. Blom RM, Figue M, Vulink N, et al. Electroconvulsive therapy, transcranial magnetic stimulation and deep brain stimulation in OCD. In: Zohar J, ed. *Obsessive Compulsive Disorder: Current Science and Clinical Practice*. Chichester, UK: John Wiley & Sons; 2012.
 16. Bandelow B, Zohar J, Hollander E, et al; WFSBP Task Force on Treatment Guidelines for Anxiety, Obsessive-Compulsive and Post-Traumatic Stress Disorders. World Federation of Societies of Biological Psychiatry (WFSBP) guidelines for the pharmacological treatment of anxiety, obsessive-compulsive and post-traumatic stress disorders—first revision. *World J Biol Psychiatry*. 2008;9(4):248–312.
 17. Fink M. ECT in anxiety: an appraisal. *Am J Psychother*. 1982;36(3):371–378.
 18. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale, 2: development, use, and reliability. *Arch Gen Psychiatry*. 1989;46(11):1006–1011.
 19. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale, 2: validity. *Arch Gen Psychiatry*. 1989;46(11):1012–1016.
 20. Kalinowsky LB, Hippis H. *Pharmacological, Convulsive, and Other Somatic Treatments in Psychiatry*. New York, NY: Grune & Stratton; 1969.
 21. Fink M. *Convulsive Therapy: Theory and Practice*. New York, NY: Raven Press; 1979.
 22. Coffey CE. *The Clinical Science of Electroconvulsive Therapy*. Washington, DC: American Psychiatric Press; 1993.
 23. Abrams R. *Electroconvulsive Therapy*. Oxford, New York, NY: Oxford University Press; 2002.
 24. American Psychiatric Publishing. *The Practice of Electroconvulsive Therapy: Recommendations for Treatment, Training, and Privileging (A Task Force Report of the American Psychiatric Association)*. Arlington, VA: American Psychiatric Publishing; 2008.
 25. Swartz CM. *Electroconvulsive and Neuromodulation Therapies*. New York, NY: Cambridge University Press; 2009.
 26. Mankad MV, Beyer JL, Weiner RD, et al. *Clinical Manual of Electroconvulsive Therapy*. Arlington, VA: American Psychiatric Publishing; 2010.
 27. Higgins JPT, Green S. *Cochrane Handbook for Systematic Reviews of Interventions*. Chichester, UK: Wiley; 2011.
 28. Stengel E. Intensive electro-convulsive therapy. *J Ment Sci*. 1951;97(406):139–142.
 29. Rosenberg CM. Complications of obsessional neurosis. *Br J Psychiatry*. 1968;114(509):477–478.
 30. Bridges PK, Goktepe EO, Maratos J. A comparative review of patients with obsessional neurosis and with depression treated by psychosurgery. *Br J Psychiatry*. 1973;123(577):663–674.
 31. Biçakci S, Bozdemir H, Över F, et al. Spontaneous intracerebral hemorrhage in a adult young female: migraine? ECT?? [article in Turkish]. *Türk Serebrovasküler Hastalıklar Dergisi*. 2004;10(2):97–99.
 32. Medda P, Mauri M, Fratta S, et al. Long-term naturalistic follow-up of patients with bipolar depression and mixed state treated with electroconvulsive therapy. *J ECT*. 2013;29(3):179–188.
 33. Wetzler AJ, Elias R, Fostick L, et al. Suicidal ideation versus suicidal obsession: a case report. *CNS Spectr*. 2007;12(7):553–556.
 34. Tonkonogy JM, Smith TW, Barreira PJ. Obsessive-compulsive disorders in Pick's disease. *J Neuropsychiatry Clin Neurosci*. 1994;6(2):176–180.
 35. Karnik NS, D'Apuzzo M, Greicius M. Non-fluent progressive aphasia, depression, and OCD in a woman with progressive supranuclear palsy: neuroanatomical and neuropathological correlations. *Neurocase*. 2006;12(6):332–338.
 36. Vázquez MJ, Martínez MC. Electroconvulsive therapy in neuroacanthocytosis or McLeod syndrome. *J ECT*. 2009;25(1):72–73.
 37. Gadit AM, Smigas T. Efficacy of ECT in severe obsessive-compulsive disorder with Parkinson's disease. *BMJ Case Rep*. 2012;2012(apr04):bcr0120125675.
 38. Rutherford M. Use of electroconvulsive therapy in a patient with chorea neuroacanthocytosis and prominent delusions. *J ECT*. 2012;28(2):e5–e6.
 39. Rao NP, Antony A, Raveendranathan D, et al. Successful use of maintenance electroconvulsive therapy in the treatment of clozapine-associated obsessive-compulsive symptoms in schizophrenia: a case report. *J ECT*. 2011;27(1):e37–e38.
 40. Dhossche DM, Reti IM, Shettar SM, et al. Tics as signs of catatonia: electroconvulsive therapy response in 2 men. *J ECT*. 2010;26(4):266–269.
 41. Mantovani A, Leckman JF, Grantz H, et al. Repetitive transcranial magnetic stimulation of the supplementary motor area in the treatment of Tourette Syndrome: report of two cases. *Clin Neurophysiol*. 2007;118(10):2314–2315.
 42. Kelly VC, Chan YC. Obedience thwarted with electroconvulsive therapy. *J ECT*. 2004;20(4):273–274.
 43. Haddock JN. The place of obsessions, compulsions and phobias in psychiatric diagnosis and treatment. *South Med J*. 1965;58(9):1115–1121.
 44. Fontenelle LF, Mendlowicz MV, Bezerra de Menezes G, et al. Asperger Syndrome, obsessive-compulsive disorder, and major depression in a patient with 45,X/46,XY mosaicism. *Psychopathology*. 2004;37(3):105–109.
 45. Gruber RP. ECT for obsessive-compulsive symptoms. (Possible mechanisms of action). *Dis Nerv Syst*. 1971;32(3):180–182.
 46. Selinski H. The selective use of electro-shock therapy as an adjunct to psychotherapy. *Bull NY Acad Med*. 1943;19(4):245–252.
 47. Dupin L. Obsessive compulsive troubles in shock. *Choc sur les TOC*. 2003;229:10.
 48. Liberati A, Altman DG, Tetzlaff J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate healthcare interventions: explanation and elaboration. *BMJ*. 2009;339(1):b2700.
 49. Garrido A. Electroconvulsive therapy in severe obsessive-compulsive disorder. *Eur Psychiatry*. 1998;13(supplement 4):2365–2375.
 50. Ferrão YA, Shavitt RG, Bedin NR, et al. Clinical features associated to refractory obsessive-compulsive disorder. *J Affect Disord*. 2006;94(1–3):199–209.
 51. Dubois JC. Obsessions and mood: apropos of 43 cases of obsessive neurosis treated with antidepressive chemotherapy and electroshock [article in French]. *Ann Med Psychol (Paris)*. 1984;142(1):141–151.
 52. Lins-Martins NM, Yücel M, Tovar-Moll F, et al. Electroconvulsive therapy in obsessive-compulsive disorder: a chart review and evaluation of its potential therapeutic effects [published online ahead of print August 8, 2014]. *J Neuropsychiatry Clin Neurosci*.
 53. Bülbül F, Copoglu US, Alpik G, et al. Maintenance therapy with electroconvulsive therapy in a patient with a codiagnosis of bipolar disorder and obsessive-compulsive disorder. *J ECT*. 2013;29(2):e21–e22.
 54. Sheehan JP, Patterson G, Schlesinger D, et al. y knife surgery anterior capsulotomy for severe and refractory obsessive-compulsive disorder. *J Neurosurg*. 2013;119(5):1112–1118.
 55. Grover S, Malhotra S, Varma S, et al. Electroconvulsive therapy in adolescents: a retrospective study from north India. *J ECT*. 2013;29(2):122–126.
 56. D'Urso G, Mantovani A, Barbarulo AM, et al. Brain-behavior relationship in a case of successful ECT for drug refractory catatonic OCD. *J ECT*. 2012;28(3):190–193.
 57. Raveendranathan D, Srinivasaraju R, Ratheesh A, et al. Treatment-refractory OCD responding to maintenance electroconvulsive therapy. *J Neuropsychiatry Clin Neurosci*. 2012;24(2):E16–E17.
 58. Makhinson M, Furst BA, Shuff MK, et al. Successful treatment of co-occurring catatonia and obsessive-compulsive disorder with concurrent electroconvulsive therapy and benzodiazepine administration. *J ECT*. 2012;28(3):e35–e36.
 59. Riestra AR, Aguilar J, Zambito G, et al. Unilateral right anterior capsulotomy for refractory major depression with comorbid obsessive-compulsive disorder. *Neurocase*. 2011;17(6):491–500.
 60. Loi S, Bonwick R. Electroconvulsive therapy for treatment of late-onset obsessive compulsive disorder. *Int Psychogeriatr*. 2010;22(5):830–831.
 61. Tomruk NB, Saatcioglu O, Ugurlu E, et al. ECT use in refractory obsessive-compulsive disorder. *Klinik Psikofarmakoloji Bulteni-Bulletin of Clinical Psychopharmacology*. 2010;20(2):167–170.
 62. Hanisch F, Friedemann J, Piro J, et al. Maintenance electroconvulsive therapy for comorbid pharmacotherapy-refractory obsessive-compulsive and schizoaffective disorder. *Eur J Med Res*. 2009;14(8):367–368.
 63. Nilsson BM, Ekselius L. Acute and maintenance electroconvulsive therapy for treatment of severely disabling obsessive-compulsive symptoms in a patient with Asperger syndrome. *J ECT*. 2009;25(3):205–207.
 64. Talaei A, Morteza-Nia M, Jafar-Zadeh M, et al. Dramatic response of resistant obsessive compulsive disorder to repeated transcranial magnetic stimulation on right supplementary motor area. *Iran J Med Sci*. 2009;34(4):295–298.
 65. Fisher CE, Sporn AL, Mantovani A, et al. Electroconvulsive therapy as an alternative to deep brain stimulation for medication-refractory Tourette syndrome. *J ECT*. 2008;24(1):103–104.
 66. Schindler F, Angelescu I, Regen F, et al. Improvement in refractory obsessive compulsive disorder with dronabinol. *Am J Psychiatry*. 2008;165(4):536–537.
 67. García Valls JM, Romero Balsalobre N, Vilaplana Pérez A, et al. Effect of ECT on a case of intermittent explosive disorder, refractory to pharmacological treatments. *An Psiquiatr*. 2007;23(6):301–303.
 68. Chaves MPR, Crippa JAS, Morais SL, et al. Electroconvulsive therapy for coexistent

- schizophrenia and obsessive-compulsive disorder. *J Clin Psychiatry*. 2005;66(4):542–543.
69. Strassnig M, Riedel M, Müller N. Electroconvulsive therapy in a patient with Tourette's syndrome and co-morbid obsessive compulsive disorder. *World J Biol Psychiatry*. 2004;5(3):164–166.
 70. Thomas SG, Kellner CH. Remission of major depression and obsessive-compulsive disorder after a single unilateral ECT. *J ECT*. 2003;19(1):50–51.
 71. Polosan M, Millet B, Bougerol T, et al. Psychosurgical treatment of malignant OCD: three case-reports [article in French]. *Encephale*. 2003;29(6):545–552.
 72. Fukuchi T, Okada Y, Katayama H, et al. A case of pregnant woman with severe obsessive-compulsive disorder successfully treated by modified-electroconvulsive therapy [article in Japanese]. *Seishin Shinkeigaku Zasshi*. 2003;105(7):927–932.
 73. Chung MS, Huang SC, Tsai SJ, et al. Mania associated with electroconvulsive therapy in a patient with obsessive compulsive disorder. *Int Med J*. 2001;8(3):185–186.
 74. Ganesan V, Kumar TCR, Khanna S. Obsessive-compulsive disorder and psychosis. *Can J Psychiatry*. 2001;46(8):750–754.
 75. Swartz CM, Shen WW. Is episodic obsessive compulsive disorder bipolar? a report of four cases. *J Affect Disord*. 1999;56(1):61–66.
 76. Shusta SR. Successful treatment of refractory obsessive-compulsive disorder. *Am J Psychother*. 1999;53(3):377–391.
 77. Lavin MR, Halligan P. ECT for comorbid obsessive-compulsive disorder and schizophrenia. *Am J Psychiatry*. 1996;153(12):1652–1653.
 78. Filer ADJ, Brockington IF. Maternal obsessions of child sexual abuse. *Psychopathology*. 1996;29(2):135–138.
 79. Wohlfahrt A. Successful ECT in obsessive-compulsive disorders: a case report [article in German]. *Nervenarzt*. 1996;67(5):397–399.
 80. Casey DA, Davis MH. Obsessive-compulsive disorder responsive to electroconvulsive therapy in an elderly woman. *South Med J*. 1994;87(8):862–864.
 81. Maletzky B, McFarland B, Burt A. Refractory obsessive compulsive disorder and ECT. *Convuls Ther*. 1994;10(1):34–42.
 82. Husain MM, Lewis SF, Thornton WL. Maintenance ECT for refractory obsessive-compulsive disorder. *Am J Psychiatry*. 1993;150(12):1899–1900.
 83. Sichel DA, Cohen LS, Dimmock JA, et al. Postpartum obsessive compulsive disorder: a case series. *J Clin Psychiatry*. 1993;54(4):156–159.
 84. Schott K, Bartels M, Heimann H, et al. Results of electroconvulsive therapy in restrictive indications: a retrospective study of 15 years [article in German]. *Nervenarzt*. 1992;63(7):422–425.
 85. Soyka M, Niederecker M, Meyendorf R. Successful treatment of a therapy-refractory compulsive syndrome by electroconvulsive therapy [article in German]. *Nervenarzt*. 1991;62(7):448–450.
 86. Warneke L. Intravenous chlorimipramine therapy in obsessive-compulsive disorder. *Can J Psychiatry*. 1989;34(9):853–859.
 87. Khanna S, Gangadhar BN, Sinha V, et al. Electroconvulsive-therapy in obsessive-compulsive disorder. *Convuls Ther*. 1988;4(4):314–320.
 88. Mellman LA, Gorman JM. Successful treatment of obsessive-compulsive disorder with ECT. *Am J Psychiatry*. 1984;141(4):596–597.
 89. Walter CJ, Mitchell-Heggs N, Sargent W. Modified narcosis, ECT and antidepressant drugs: a review of technique and immediate outcome. *Br J Psychiatry*. 1972;120(559):651–662.
 90. Grimshaw L. The outcome of obsessional disorder: a follow-up study of 100 cases. *Br J Psychiatry*. 1965;111(480):1051–1056.
 91. Korson SM. The successful treatment of an obsessive compulsive neurosis with narcosis followed by daily electroshocks. *J Nerv Ment Dis*. 1949;109(1):37–41.
 92. Bini L, Bazzi T. L'elettoshocterapia col metodo dell' "annichilimento" nelle forme gravi di psiconevrosi. *Rass Neuropsichiatr*. 1947;1:59–70.
 93. Hamilton DM. The use of electric shock therapy in psychoneurosis. *Am J Psychiatry*. 1947;103(5):665–668.
 94. Milligan WL. Psychoneuroses treated with electrical convulsions; the intensive method. *Lancet*. 1946;248(6424):516–520.
 95. Kerman E. Electroshock therapy, with special reference to relapses and an effort to prevent them. *J Nerv Ment Dis*. 1945;102:213–242.
 96. Moriarty JD, Weil AA. Combined convulsive therapy and psychotherapy of the neuroses. *Arch Neurol Psychiatry*. 1943;50(6):685–690.
 97. Smith LH, Hastings DW, Hughes J. Immediate and follow up results of electroshock therapy. *Am J Psychiatry*. 1943;100(3):351–354.
 98. Jelovac A, Kolshus E, McLoughlin DM. Relapse following successful electroconvulsive therapy for major depression: a meta-analysis. *Neuropsychopharmacology*. 2013;38(12):2467–2474.
 99. Ackerman DL, Greenland S, Bystritsky A, et al. Predictors of treatment response in obsessive-compulsive disorder: multivariate analyses from a multicenter trial of clomipramine. *J Clin Psychopharmacol*. 1994;14(4):247–254.
 100. Ravizza L, Barzega G, Bellino S, et al. Predictors of drug treatment response in obsessive-compulsive disorder. *J Clin Psychiatry*. 1995;56(8):368–373.
 101. Rosario-Campos MC, Leckman JF, Mercadante MT, et al. Adults with early-onset obsessive-compulsive disorder. *Am J Psychiatry*. 2001;158(11):1899–1903.
 102. Fontenelle LF, Mendlowicz MV, Marques C, et al. Early- and late-onset obsessive-compulsive disorder in adult patients: an exploratory clinical and therapeutic study. *J Psychiatr Res*. 2003;37(2):127–133.
 103. Jakubovski E, Diniz JB, Valerio C, et al. Clinical predictors of long-term outcome in obsessive-compulsive disorder. *Depress Anxiety*. 2013;30(8):763–772.
 104. Lomax CL, Oldfield VB, Salkovskis PM. Clinical and treatment comparisons between adults with early- and late-onset obsessive-compulsive disorder. *Behav Res Ther*. 2009;47(2):99–104.
 105. Fontenelle LF, Harrison BJ, Pujol J, et al. Brain functional connectivity during induced sadness in patients with obsessive-compulsive disorder. *J Psychiatry Neurosci*. 2012;37(4):231–240.
 106. Hoehn-Saric R, Ninan P, Black DW, et al. Multicenter double-blind comparison of sertraline and desipramine for concurrent obsessive-compulsive and major depressive disorders. *Arch Gen Psychiatry*. 2000;57(1):76–82.
 107. Denys D, Van Meegen H, Westenberg H. The adequacy of pharmacotherapy in outpatients with obsessive-compulsive disorder. *Int Clin Psychopharmacol*. 2002;17(3):109–114.
 108. Blanco C, Olfson M, Stein DJ, et al. Treatment of obsessive-compulsive disorder by US psychiatrists. *J Clin Psychiatry*. 2006;67(6):946–951.
 109. Keck PE Jr, Lipinski JF Jr, White K. An inverse relationship between mania and obsessive-compulsive disorder: a case report. *J Clin Psychopharmacol*. 1986;6(2):123–124.
 110. Jefferson JW, Greist JH, Perse TL, et al. Fluvoxamine-associated mania/hypomania in patients with obsessive-compulsive disorder. *J Clin Psychopharmacol*. 1991;11(6):391–392.
 111. Steiner W. Fluoxetine-induced mania in a patient with obsessive-compulsive disorder. *Am J Psychiatry*. 1991;148(10):1403–1404.
 112. Vieta E, Bernardo M. Antidepressant-induced mania in obsessive-compulsive disorder. *Am J Psychiatry*. 1992;149(9):1282–1283.
 113. Berk M, Koopowitz LF, Szabo CP. Antidepressant induced mania in obsessive compulsive disorder. *Eur Neuropsychopharmacol*. 1996;6(1):9–11.
 114. Diler RS, Avci A. SSRI-induced mania in obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry*. 1999;38(1):6–7.
 115. Philip J, Janaki R. Antidepressant-induced mania in obsessive compulsive disorder. *Indian J Psychiatry*. 2012;54(2):194–195.
 116. Santana L, Fontenelle JM, Yücel M, et al. Rates and correlates of nonadherence to treatment in obsessive-compulsive disorder. *J Psychiatr Pract*. 2013;19(1):42–53.
 117. Yaryura-Tobias JA, Neziroglu FA. *Obsessive-Compulsive Disorder Spectrum: Pathogenesis, Diagnosis, and Treatment*. Washington, DC: American Psychiatric Press; 1997.
 118. Hariz MI, Hariz GM. Therapeutic stimulation versus ablation. *Handb Clin Neurol*. 2013;116:63–71.
 119. Rohland BM, Carroll BT, Jacoby RG. ECT in the treatment of the catatonic syndrome. *J Affect Disord*. 1993;29(4):255–261.
 120. Hollander E, Poskar S, Gerard A. Subtypes and Spectrum Issues. In: Zohar J, ed. *Obsessive Compulsive Disorder: Current Science and Clinical Practice*. Chichester, UK: Wiley; 2012.
 121. Wachtel LE, Dhossche DM. Self-injury in autism as an alternate sign of catatonia: implications for electroconvulsive therapy. *Med Hypotheses*. 2010;75(1):111–114.