International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	NEUROPSYCHOLOGICAL AND SOMATIC COMPLICATIONS OF ELECTROCONVULSIVE THERAPY (ECT) – A REVIEW OF CURRENT EVIDENCE AND CLINICAL IMPLICATIONS
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5019
RECEIVED	03 February 2026
ACCEPTED	11 May 2026
PUBLISHED	28 May 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

© The author(s) 2026.
This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

NEUROPSYCHOLOGICAL AND SOMATIC COMPLICATIONS OF ELECTROCONVULSIVE THERAPY (ECT) – A REVIEW OF CURRENT EVIDENCE AND CLINICAL IMPLICATIONS

Martyna Stanik *(Corresponding Author, Email: martyna.stanik@op.pl)*
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0001-6539-8932

Elżbieta Trzyna
University Children's Hospital in Cracow, Cracow, Poland
ORCID ID: 0009-0003-0746-6989

Klaudia Jurkowska
Medical University of Silesia in Katowice, Katowice, Poland
ORCID ID: 0009-0003-0205-6999

Joanna Górak
Clinical Department of Dermatology, AMiSNS St. Adalbert Hospital in Gdańsk
ORCID ID: 0009-0006-5536-8269

Eleonora Y. Mahoney
Medical University of Gdańsk, Gdańsk, Poland
ORCID ID: 0009-0003-0846-9727

Ewa Bełc
Intern doctor, USK nr 2, Medical University of Lodz, Żeromskiego ST. 113, 90-549 Lodz, Poland
ORCID ID: 0009-0004-0224-8162

Agata Reliszka
Provincial Hospital named after Maria Skłodowska-Curie, 95-100 Zgierz, ul. Parzęczewska 35, Poland
ORCID ID: 0009-0004-6744-8667

Aleksandra Stadnik
Medical Intern, University Clinical Hospital in Wroclaw, Borowska 213, 50-556 Wroclaw, Poland
ORCID ID: 0009-0005-4764-3980

Anna Gliwa
Wroclaw Medical University, Wrocław, Poland
ORCID ID: 0009-0006-0251-0591

Anastazja Gibowska
Collegium Medicum named after Ludwik Rydygier in Bydgoszcz, Poland
ORCID ID: 0009-0008-6799-3277

ABSTRACT

Electroconvulsive therapy (ECT) remains one of the most effective treatment modalities for severe psychiatric disorders, particularly treatment-resistant depression, catatonia, and schizoaffective disorders. Despite its well-established efficacy, ECT continues to generate controversy due to the potential for neuropsychological and somatic complications. The aim of this review is to provide a comprehensive analysis of ECT-related complications, incorporating the latest scientific findings, underlying mechanisms of cognitive deficits, risk factors, and strategies to minimize adverse effects. Evidence indicates that most cognitive deficits are transient; however, they necessitate careful monitoring, meticulous premedication, and individualized treatment planning in clinical practice.

KEYWORDS

Electroconvulsive Therapy, ECT, Cognitive Complications, Memory, Cardiovascular Disorders, Neuropsychology

CITATION

Martyna Stanik, Elżbieta Trzyna, Klaudia Jurkowska, Joanna Górak, Eleonora Y. Mahoney, Ewa Bełc, Agata Reliszka, Aleksandra Stadnik, Anna Gliwa, Anastazja Gibowska. (2026) Neuropsychological and Somatic Complications of Electroconvulsive Therapy (ECT) – A Review of Current Evidence and Clinical Implications. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5019

COPYRIGHT

© The author(s) 2026. This article is published as open access under the **Creative Commons Attribution 4.0 International License (CC BY 4.0)**, allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

1. Introduction

Electroconvulsive therapy (ECT) was introduced in the 1930s as a groundbreaking treatment for severe psychiatric disorders. From its inception, ECT has been controversial, both due to societal stigma and the early observed physical and cognitive complications. Contemporary procedures, conducted under general anesthesia and with the use of muscle relaxants, have significantly improved the safety of the treatment, allowing precise adjustment of stimulus parameters to meet the individual needs of patients. ECT is currently recognized as one of the most effective interventions for treatment-resistant depression, catatonia, and certain psychotic disorders, often providing rapid clinical improvement, which is particularly crucial in life-threatening cases.

Despite its established efficacy, one of the main areas of concern remains cognitive dysfunction, including memory and executive function impairments. Loss of autobiographical memory and difficulties in acquiring new information can affect patients' daily functioning and their overall satisfaction with therapy. Understanding the nature of these complications, the underlying mechanisms, and associated risk factors is essential for developing effective strategies to mitigate the adverse effects of ECT. Advances in neuropsychological assessment tools, neuroimaging studies, and biomarkers now allow for more precise monitoring of cognitive outcomes and evaluation of their temporal dynamics.

The aim of this review is to provide a comprehensive analysis of the scientific literature on complications associated with electroconvulsive therapy, with particular emphasis on neuropsychological and somatic aspects, and to discuss the clinical and ethical implications arising from these observations.

2. History and Development of ECT

Initially, ECT was administered without anesthesia or muscle relaxants, which was associated with a high risk of musculoskeletal complications and significant psychological stress for patients. In subsequent decades, the introduction of general anesthesia and pharmacological muscle relaxants substantially reduced the incidence of injuries and improved procedural comfort. Simultaneously, advances in medical technology enabled precise control of electrical stimulus parameters, including pulse duration, amplitude, and electrode placement.

In the 1980s, so-called brief-pulse stimuli were introduced, which proved to be cognitively less burdensome compared to the previously used sinusoidal pulses. The development of unilateral procedures, particularly with electrode placement over the non-dominant hemisphere, allowed for a reduction in the risk of memory deficits while maintaining high therapeutic efficacy. [1,2]

3. Characteristics of the ECT Procedure

During electroconvulsive therapy, electrodes are applied to the patient's scalp while they are under short-term general anesthesia and pharmacologically induced muscle relaxation. A controlled electrical stimulus with precisely defined parameters is then delivered, eliciting a generalized therapeutic seizure that typically lasts several to tens of seconds and is monitored via EEG. Following stimulation, the seizure activity ceases spontaneously.

Electrode placement may be unilateral or bilateral. Unilateral stimulation, most commonly over the non-dominant hemisphere, demonstrates a more favorable cognitive profile, although it may be slightly less therapeutically effective compared to bilateral stimulation, particularly bitemporal placement. Pulse parameters, including brief rectangular pulses, are associated with a lower cognitive burden than earlier methods. Standard ECT courses generally consist of two to three sessions per week over several weeks, depending on the patient's clinical condition and individual treatment response.

4.1 Cardiovascular Risk

Electroconvulsive therapy (ECT) is associated with transient hemodynamic changes resulting from the induced seizure and autonomic nervous system response. A biphasic reaction is observed: an initial parasympathetic-dominant phase, characterized by bradycardia and, rarely, brief asystole, followed by a sympathetic phase with tachycardia, elevated blood pressure, and increased myocardial oxygen demand.

The most common cardiovascular complications include transient cardiac arrhythmias, temporary hypertension, and nonspecific ECG changes. Serious events, such as myocardial infarction or persistent arrhythmias, are rare and mainly occur in patients with significant pre-existing cardiovascular disease. Risk factors include coronary artery disease, uncontrolled hypertension, cardiac conduction disturbances, and advanced age.

Contemporary ECT standards, including thorough pre-procedural assessment, ECG monitoring, and strict anesthesiological supervision, substantially reduce the risk of cardiovascular complications. When these standards are adhered to, ECT is considered a procedure with low and controllable cardiological risk.

4.2 Respiratory and Neurological Complications

Typical adverse effects related to anesthesia include nausea, vomiting, headache, and transient disorientation. With modern ECT techniques under general anesthesia and muscle relaxation, complications commonly associated with unmodified procedures—such as jaw dislocation, long-bone fractures, or compression fractures of vertebral bodies—are not observed. [3]

Neurological complications may include:

- Transient disorientation and balance disturbances,
- Sensory and coordination impairments in the post-procedure period,
- Rare tremors or seizures unrelated to ECT.

The ECT-induced seizure causes significant autonomic nervous system changes. The tonic phase of the seizure may lead to transient parasympathetic dominance, resulting in bradycardia, atrioventricular blocks, and, in exceptional cases, asystole. The risk of arrhythmia is particularly relevant in patients with pre-existing cardiac disease or conduction abnormalities. [4]

Severe neurological complications are very rare. Reported cases include brain herniation through the foramen magnum due to increased intracranial pressure, hemorrhagic or ischemic stroke. [5,6] It is likely that in these cases, pre-existing elevated intracranial pressure, hypertension, or cardiac conduction disturbances were overlooked before initiating ECT. Such complications could likely be prevented through appropriate pre-procedural evaluation and management.

Rare complications also include prolonged apnea, which can occur in patients with slow metabolism of succinylcholine. In such cases, spontaneous resumption of respiration usually occurs within 30–60 minutes. During this period, adequate patient oxygenation is crucial.[7]

Most of these complications are transient and do not result in permanent neurological damage. [8]

5. Neuropsychological Complications of ECT

5.1 Memory Impairment

The most frequently reported cognitive adverse effects of electroconvulsive therapy (ECT) concern memory disturbances. These include both retrograde amnesia, characterized by difficulty recalling events that occurred prior to treatment, and anterograde amnesia, manifested by impaired encoding and retrieval of newly acquired information during the treatment course and in the immediate post-treatment period.[2]

Meta-analytical evidence indicates that post-ECT memory deficits are typically transient, resolving within several weeks or months following completion of therapy. Nevertheless, subjective complaints regarding autobiographical memory loss may persist for a longer duration.[9]

Declarative Memory (Explicit Memory)

Declarative memory refers to the conscious capacity to encode, store, and retrieve facts and events.[10] This system enables the recollection of dates, numerical data, temporal relationships, and self-referential information.[11] Disturbances of declarative memory following ECT are primarily associated with both retrograde and anterograde amnesia.

Retrograde amnesia generally encompasses a period of up to two to three years preceding the ECT course, whereas information consolidated earlier typically remains intact.[12] Notably, impersonal (semantic) memory concerning general knowledge about the world appears to be more vulnerable than personal (autobiographical) memory.[13] Empirical findings further suggest a temporal gradient effect: the shorter the interval between the occurrence or encoding of an event and the initiation of ECT, the greater the likelihood of subsequent forgetting.[14] Retrograde amnesia associated with ECT is usually reversible and tends to resolve within several weeks after completion of the treatment series.[2]

Anterograde amnesia induced by ECT involves impaired ability to acquire and recall newly learned or recently encoded information.[15],[16] These symptoms are typically short-lived and reversible, with the most pronounced deficits observed within the first three days following the conclusion of ECT.[2] In most cases, baseline learning capacity is restored within several weeks after treatment. It is noteworthy that anterograde memory disturbances are not unique to ECT and may also occur during pharmacotherapy with first-generation antidepressants. A comparative study involving 10 patients with depression treated with imipramine and 16 patients undergoing bilateral ECT demonstrated significant deterioration in anterograde memory in both groups relative to pre-treatment performance, while immediate memory remained unaffected. Retrograde memory impairment, however, was observed exclusively in the ECT group.[17] It is generally assumed that declarative memory functions return to pre-treatment levels within six months after the final ECT session.[2]

Non-Declarative Memory (Implicit Memory)

Non-declarative memory encompasses unconscious processes involved in the acquisition and retention of information. This system includes procedural memory (memory for skills and motor sequences), classical conditioning, and perceptual priming.[10] Available, albeit limited, research suggests that implicit memory remains largely unaffected by ECT.

Short-Term Memory

Short-term memory may be defined as the ability to reproduce, recognize, or recall presented material (e.g., facts, numbers, information) within approximately thirty seconds of exposure.[8],[10],[11] Working memory constitutes a component of this system and enables the temporary storage and manipulation of information necessary to perform ongoing tasks (e.g., maintaining task criteria).[19] Numerous studies have demonstrated that short-term memory impairment can be detected using neuropsychological assessments within hours after the first ECT session. Such deficits may persist for several days following completion of the treatment series.[20] However, within a few weeks after therapy, short-term memory generally returns to baseline levels. Some reports even suggest post-treatment performance exceeding pre-ECT levels, possibly reflecting improvement secondary to remission of depressive symptoms.[2],[21],[22]

5.2 Executive Functions and Attention

ECT has been associated with transient reductions in executive functioning, attentional capacity, and processing speed. However, evidence indicates that as mood symptoms improve, cognitive performance typically returns to baseline levels. In certain cases, cognitive functioning may even improve beyond pre-treatment levels, underscoring the complex interaction between affective state and cognitive processes.[23]

5.3 Affective and Psychiatric Disturbances Following ECT

Although ECT is recognized as an effective intervention for severe depression, certain affective and psychiatric symptoms may occur in a subset of patients, including:

- transient emotional lability,
- social disorientation, anxiety, and irritability,
- rare manic episodes, particularly in individuals with bipolar affective disorder.

5.4 Biological Basis of Cognitive Disturbances Following Electroconvulsive Therapy

The occurrence of cognitive disturbances after ECT does not appear to reflect structural damage to the central nervous system (CNS). This conclusion is supported both by the transient and reversible nature of these deficits and by studies assessing biomarkers of CNS injury during and after treatment. In a study involving 19 patients undergoing bilateral ECT, plasma concentrations of neuron-specific enolase (NSE) and S-100 protein—established markers of CNS damage—were measured. No statistically significant increases in the levels of these biomarkers were observed either during the course of therapy or following its completion.[24]

Similarly, Zachrisson et al.[25] examined a group of nine patients diagnosed with depression who were treated with ECT. Concentrations of neuronal injury markers—tau protein, neurofilament light chain (NFL), and S-100 protein—were analyzed in cerebrospinal fluid samples obtained prior to the first session and after six ECT treatments. No significant differences in the levels of these markers were detected between pre- and post-treatment assessments.[26]

Available biochemical, structural, and neurophysiological data indicate that the mechanism of action of ECT is complex and multifactorial. The therapeutic effects of ECT are known to involve robust activation of multiple neurotransmitter systems, including noradrenergic, serotonergic, dopaminergic, and GABAergic pathways, as well as stimulation of brain-derived neurotrophic factor (BDNF) production.[27] Conversely, ECT has been associated with a reduction in central cholinergic activity. It has therefore been hypothesized that inhibition of cholinergic neurotransmission may represent one of the principal mechanisms underlying post-ECT cognitive impairment.[28]

Another proposed explanation for postictal memory disturbances is based on the glutamatergic model. According to this hypothesis, ECT induces hyperstimulation of N-methyl-D-aspartate (NMDA) receptors, leading to excessive calcium influx into neurons and potentially triggering excitotoxic processes.[28] Support for this model is derived in part from studies examining the use of the NMDA receptor antagonist ketamine as an anesthetic agent during ECT procedures (see below). The use of ketamine may additionally be justified by animal studies indicating that NMDA receptor blockade enhances BDNF expression, a factor implicated in the antidepressant effects of ECT.[29]

6. Risk Factors for Cognitive Complications

Multiple factors influence the course of cognitive functioning following ECT. Procedural parameters, including electrode placement, stimulus amplitude, and pulse duration, significantly affect the severity of memory deficits. Bilateral stimulation—particularly bitemporal electrode placement—is associated with more pronounced retrograde amnesia compared to unilateral stimulation. Higher stimulus intensity and increased treatment frequency have also been correlated with greater cognitive impairment.

Individual patient characteristics play an equally important role. Advanced age, pre-existing memory impairment, and comorbid neurological disorders are associated with increased risk of post-treatment cognitive complications. Furthermore, concomitant pharmacotherapy with anticholinergic agents, lithium, or neuroleptics characterized by strong sedative properties may exacerbate memory deficits. Severe underlying illness, treatment-resistant depression, and the presence of psychotic symptoms have likewise been identified as factors increasing vulnerability to transient cognitive deterioration.[1]

Genetic factors may additionally modulate susceptibility to cognitive side effects. Polymorphisms in genes such as *BDNF* and *COMT* have been implicated in differential vulnerability to post-ECT cognitive impairment. Variations in neuroplasticity, neurotransmitter metabolism, and stress hormone regulation may further influence both the severity and duration of observed deficits.

7. Assessment and Monitoring of Cognitive Function

Effective management of cognitive complications associated with ECT requires systematic neuropsychological assessment conducted prior to treatment initiation, throughout the treatment course, and following completion of the ECT series. Standard evaluation tools include comprehensive neuropsychological test batteries designed to monitor memory, attention, and executive functioning.[2]

Regular assessment enables the identification of both short- and long-term cognitive deficits and facilitates the implementation of supportive interventions when necessary. Such measures may include structured memory training, cognitive rehabilitation programs, or modification of treatment parameters (e.g., electrode placement or stimulus intensity).[2] Continuous monitoring is therefore considered an essential component of safe and individualized ECT practice.

8. Mortality Associated with Electroconvulsive Therapy

Mortality associated with ECT is rare and results from a combination of risks related to general anesthesia and the physiological consequences of the electrically induced seizure.[1,30,31] Contemporary ECT standards—including comprehensive anesthetic evaluation, continuous monitoring of vital parameters, and strict control of stimulation parameters—have substantially reduced the incidence of life-threatening complications.[32]

Available epidemiological data indicate that most reported deaths temporally associated with ECT have been attributable to cardiovascular complications.[31,33] The most frequently described events include severe cardiac arrhythmias, acute coronary syndromes, and cerebrovascular complications, particularly in patients with significant underlying medical comorbidities. ECT induces transient biphasic autonomic changes: an initial parasympathetic predominance (manifested by bradycardia and, in rare cases, transient asystole), followed by pronounced sympathetic activation (tachycardia and elevated blood pressure). In high-risk individuals, these hemodynamic fluctuations may precipitate cardiovascular destabilization.[30,34]

The estimated mortality rate directly attributable to ECT ranges from approximately 1 per 10,000 to 1 per 80,000 treated patients.[31,33] This risk is comparable to that associated with brief surgical procedures performed under general anesthesia.[30] Importantly, among individuals with severe depression, the overall mortality risk—including death by suicide—substantially exceeds the risk associated with ECT.[32,35] Some analyses further suggest that mortality among patients treated with ECT may be lower than among those receiving tricyclic antidepressants, which are associated with clinically significant cardiotoxic risk.[35]

9. Strategies for Minimizing Complications

Minimizing complications associated with electroconvulsive therapy (ECT) requires an integrated approach encompassing technical, pharmacological, and rehabilitative strategies. Optimization of treatment parameters—such as selecting unilateral rather than bilateral electrode placement and employing brief-pulse stimulation—may reduce the severity of memory impairment while maintaining therapeutic efficacy. Individualized adjustment of stimulus intensity and session frequency is likewise essential in order to limit adverse cognitive effects. In addition, cognitive rehabilitation, psychoeducation for patients and their families, and structured attention and memory training programs may facilitate post-treatment functional recovery. Lifestyle factors, including regular physical activity and a diet rich in antioxidants and omega-3 fatty acids, have also been proposed as supportive measures for executive functioning and cerebral perfusion following ECT, although empirical evidence remains limited.[37]

Various pharmacological strategies have been investigated in an attempt to mitigate ECT-related cognitive impairment. A meta-analysis of randomized controlled trials suggested that certain agents—such as memantine and liothyronine (triiodothyronine, T3)—may confer modest benefits in short-term cognitive outcomes; however, the overall quality of evidence was low.[38] Acetylcholinesterase inhibitors, piracetam, and melatonin have also demonstrated positive effects in selected cognitive domains in some studies, yet the strength and consistency of supporting data remain limited.[38]

Piracetam, a nootropic agent believed to modulate neurotransmission, has been evaluated for its potential role in preventing cognitive dysfunction during ECT. However, a randomized, double-blind, placebo-controlled trial failed to demonstrate statistically significant protection against memory impairment or improvement in neuropsychological test performance among patients receiving piracetam compared with placebo during ECT.[39]

Another line of investigation has focused on the administration of thyroid hormone (triiodothyronine, T3) as a potential means of counteracting suppression of the hypothalamic–pituitary–thyroid axis, which has

been hypothesized to contribute to post-ECT memory deficits. Preliminary findings indicated that adjunctive T3 administration might improve memory performance during ECT; nevertheless, further research in larger samples is required to confirm these observations.[40]

The role of the endogenous opioid system has also been explored. Studies examining opioid receptor antagonists, such as naloxone, reported that high doses administered immediately prior to ECT sessions were associated with reduced severity of anterograde amnesia and improvement in selected cognitive functions, including verbal fluency, compared with placebo or lower doses of naloxone. These findings suggest a potential mechanistic involvement of the opioid system in ECT-related memory impairment, while also underscoring the complexity of this interaction.[41]

Intranasal vasopressin has likewise been evaluated as a possible intervention for reducing memory disturbances associated with ECT. However, results from a randomized placebo-controlled trial did not demonstrate significant cognitive benefits compared with placebo.[38]

In summary, although multiple technical, rehabilitative, and pharmacological strategies have been proposed to attenuate cognitive adverse effects of ECT, none has yet demonstrated unequivocal clinical efficacy. The most promising approaches—such as memantine and liothyronine—require further validation in well-designed, adequately powered clinical trials.[38]

10. Ethical and Legal Considerations

Informed consent constitutes the cornerstone of the ethical application of electroconvulsive therapy (ECT). Comprehensive patient education regarding potential complications—both objectively measurable and subjectively experienced—is essential. Active involvement of the patient in the decision-making process enhances autonomy, improves treatment adherence, and reduces procedural anxiety. In selected cases, specialized support in clinical neuropsychology or psychiatry may be required to monitor and mitigate persistent cognitive deficits.[1]

11. Future Research Directions and Innovations in ECT

Contemporary developments in ECT aim not only to maximize therapeutic efficacy but also to further minimize cognitive and somatic adverse effects. One principal area of investigation involves refinement of electrical stimulation parameters to enable more precise tailoring of the stimulus to individual patient characteristics. Research exploring modulation of pulse frequency, stimulus duration, and waveform configuration seeks to reduce the impact of ECT on autobiographical memory and executive functioning while preserving full antidepressant effectiveness.[2]

Another significant domain concerns advances in neuroimaging and neurophysiological monitoring technologies. Techniques such as functional magnetic resonance imaging (fMRI) and magnetoencephalography (MEG) permit real-time observation of neuronal activity and structural changes within the hippocampus and prefrontal cortex. Integration of neuroimaging data with neuropsychological outcomes may facilitate the development of predictive models capable of identifying patients at increased risk of transient or persistent cognitive deficits.[42]

Parallel research efforts have focused on low-energy ECT modalities, including ultra-brief pulse stimulation, which utilize shorter pulse widths and lower electrical intensity. Preliminary findings suggest that such modifications may significantly reduce the incidence and severity of retrograde amnesia while maintaining rapid antidepressant effects. Moreover, investigations into neuronavigation and stereotactic electrode placement techniques aim to enhance targeting precision and thereby minimize unwanted cognitive consequences.[43]

Substantial progress is also being made in the integration of pharmacotherapy with ECT. Studies examining neuroprotective agents, modulation of the cholinergic system, and interventions targeting oxidative stress seek to protect neuronal integrity from potential adverse effects of electrical stimulation. The combination of ECT with individualized pharmacological regimens offers promising prospects for achieving high therapeutic efficacy with reduced cognitive burden.[1]

An additional innovative direction involves the development of hybrid treatment approaches combining ECT with modern neuromodulation techniques such as transcranial magnetic stimulation (TMS) or transcranial direct current stimulation (tDCS). Research into the potential synergistic effects of these modalities aims to shorten treatment duration, reduce the number of ECT sessions required, and enhance cognitive safety.[9]

Finally, increasing attention is being directed toward personalization of therapy based on genetic and epigenetic factors predisposing patients to cognitive complications. Investigation of polymorphisms in genes

associated with neuroplasticity and neurotransmitter metabolism may, in the future, enable more accurate prediction of individual treatment response and risk profiles. Such efforts align with the broader paradigm of personalized medicine, in which therapeutic strategies are optimized for the individual patient.[42]

12. Discussion

The available evidence portrays ECT as a highly effective therapeutic intervention, albeit one associated with the potential for transient cognitive side effects. Memory impairment and executive dysfunction are generally reversible, and serious somatic complications remain rare when appropriate monitoring standards are applied.

In clinical practice, particular importance should be attributed to patients' subjective experiences, which may not always correspond directly to objective neuropsychological findings. Careful adjustment of treatment parameters and individualized therapeutic planning are therefore essential. Ethical implementation of ECT requires transparent communication, valid informed consent, and flexibility in adapting treatment strategies to patient-specific needs. The integration of clinical data, neuropsychological assessments, and patient-reported outcomes is crucial for optimizing both therapeutic efficacy and procedural safety.

13. Conclusions

Electroconvulsive therapy remains one of the most effective treatments for severe psychiatric disorders, frequently producing rapid and, in some cases, life-saving therapeutic effects. The most common cognitive complications—particularly disturbances of memory and executive functioning—are typically transient and reversible. Somatic complications are rare when patients are appropriately screened and monitored.

Key components of safe and effective ECT practice include individualized adjustment of stimulation parameters, systematic cognitive assessment, and the provision of psychological support. Future research should focus on elucidating neurobiological mechanisms, developing neuroprotective interventions, and incorporating patient-reported outcomes to further enhance the safety profile and overall quality of ECT treatment.

REFERENCES

1. American Psychiatric Association. (2023). The practice of electroconvulsive therapy: Recommendations for treatment, training, and privileging (3rd ed.). APA Publishing.
2. Semkovska, M., & McLoughlin, D. M. (2010). Objective cognitive performance associated with electroconvulsive therapy for depression: A systematic review and meta-analysis. *Biological Psychiatry, 68*, 568–577.
3. Verwijk, E., Comijs, H. C., Kok, R. M., et al. (2014). Short- and long-term neurocognitive functioning after electroconvulsive therapy in depressed elderly: A prospective naturalistic study. *International Psychogeriatrics, 26*, 315–324.
4. Stein, C., et al. (2018). Cognitive outcomes after electroconvulsive therapy: A review of recent evidence. *Frontiers in Psychiatry, 9*, 123.
5. McClintock, S. M., Choi, J., Deng, Z. D., et al. (2014). Multifactorial determinants of the neurocognitive effects of electroconvulsive therapy. *Journal of ECT, 30*, 165–176.
6. Verwijk, E., Comijs, H. C., Kok, R. M., et al. (2014). Short- and long-term neurocognitive functioning after electroconvulsive therapy in depressed elderly: A prospective naturalistic study. *International Psychogeriatrics, 26*, 315–324.
7. Squire, L. R., & Zola, S. M. (1997). Amnesia, memory and brain systems. Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 352, 1663–1673.
8. Kellner, C. H., Knapp, R., Petrides, G., Rummans, T., Husain, M. M., & Rasmussen, K. (2012). Continuation electroconvulsive therapy vs pharmacotherapy for relapse prevention in major depression: A randomized clinical trial. *JAMA Psychiatry, 69*(4), 377–384.
9. Brus, M., et al. (2017). Cognitive effects of electroconvulsive therapy in patients with depression: A systematic review. *European Psychiatry, 41*, 61–69.
10. Squire, L. R., & Zola, S. M. (1997). Amnesia, memory and brain systems. Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 352, 1663–1673.
11. Borkowska, A. (2002). Ocena neuropsychologiczna. In A. Bilikiewicz, S. Pużyński, J. Rybakowski, & J. Wciórka (Eds.), *Psychiatria 1* (pp. 539–550). Urban & Partner.
12. Meeter, M., Murre, J. M., Janssen, S. M., Birkenhager, T., & van den Broek, W. W. (2011). Retrograde amnesia after electroconvulsive therapy: A temporary effect? *Journal of Affective Disorders, 132*, 216–222.

13. Kroes, M. C., Tendolkar, I., van Wingen, G. A., et al. (2014). An electroconvulsive therapy procedure impairs reconsolidation of episodic memories in humans. *Nature Neuroscience, 17*, 204–206.
14. O'Connor, M., Lebowitz, B. K., Ly, J., et al. (2008). A dissociation between anterograde and retrograde amnesia after treatment with electroconvulsive therapy: A naturalistic investigation. *Journal of ECT, 24*, 146–151.
15. McClintock, S. M., Choi, J., Deng, Z. D., et al. (2014). Multifactorial determinants of the neurocognitive effects of electroconvulsive therapy. *Journal of ECT, 30*, 165–176.
16. Verwijk, E., Comijs, H. C., Kok, R. M., et al. (2014). Short- and long-term neurocognitive functioning after electroconvulsive therapy in depressed elderly: A prospective naturalistic study. *International Psychogeriatrics, 26*, 315–324.
17. Fernie, G., Bennett, D. M., Currie, J., Perrin, J. S., & Reid, I. C. (2014). Detecting objective and subjective cognitive effects of electroconvulsive therapy: Intensity, duration and test utility in a large clinical sample. *Psychological Medicine, 24*, 1–10.
18. Rami-Gonzalez, L., Boget-Llucia, T., Bernardo, M., et al. (2002). Selective alteration of the declarative memory systems in patients treated with a high number of electroconvulsive therapy sessions. *Revista de Neurología, 35*, 805–808.
19. Baddeley, A. (1986). Recent developments in working memory. *Science, 232*, 1612–1619.
20. Polster, J. D., Kayser, S., Bewernick, B. H., Hurlemann, R., & Schlaepfer, T. E. (2014). Effects of electroconvulsive therapy and magnetic seizure therapy on acute memory retrieval. *Journal of ECT, 21*.
21. McClintock, S. M., DeWind, N. K., Husain, M. M., et al. (2013). Disruption of component processes of spatial working memory by electroconvulsive shock but not magnetic seizure therapy. *International Journal of Neuropsychopharmacology, 16*, 177–187.
22. Verwijk, E., Comijs, H. C., Kok, R. M., et al. (2012). Neurocognitive effects after brief pulse and ultrabrief pulse unilateral electroconvulsive therapy for major depression: A review. *Journal of Affective Disorders, 140*, 233–243.
23. Vasavada, M., et al. (2017).
24. Zachrisson, O. C., Balldin, J., Ekman, R., et al. (2000). No evident neuronal damage after electroconvulsive therapy. *Psychiatry Research, 96*, 157–165.
25. Bolwig, T. G. (2011). How does electroconvulsive therapy work? Theories on its mechanism. *Canadian Journal of Psychiatry, 56*, 13–18.
26. McCall, W. V., Andrade, C., & Sienaert, P. (2014). Searching for the mechanism(s) of ECT's therapeutic effect. *Journal of ECT, 30*, 87–89.
27. Chamberlin, E., & Tsai, G. E. (1998). A glutamatergic model of ECT-induced memory dysfunction. *Harvard Review of Psychiatry, 5*, 307–317.
28. Chen, A. C., Shin, K. H., Duman, R. S., & Sanacora, G. (2001). Induced mossy fiber sprouting and BDNF expression are attenuated by ketamine pretreatment. *ECS-ECT, 17*, 27–32.
29. Tew, J. D., Jr., Mulsant, B. H., Haskett, R. F., et al. (2002). A randomized comparison of high-charge right unilateral electroconvulsive therapy and bilateral electroconvulsive therapy in older depressed patients who failed to respond to 5 to 8 moderate-charge right unilateral treatments. *Journal of Clinical Psychiatry, 63*, 1102–1105.
30. Freeman, C. P., & Kendell, R. E. (1986). Patients' experiences of and attitudes to electroconvulsive therapy. *Annals of the New York Academy of Sciences, 462*, 341–352.
31. Weiner, R. D., & Reti, I. M. (2017). Key updates in the clinical application of electroconvulsive therapy. *International Review of Psychiatry, 29*(2), 54–62.
32. UK ECT Review Group. (2003). Efficacy and safety of electroconvulsive therapy in depressive disorders: A systematic review and meta-analysis. *The Lancet, 361*, 799–808.
33. Tørring, N., Sanghani, S. N., Petrides, G., Kellner, C. H., & Østergaard, S. D. (2017). The mortality rate of electroconvulsive therapy: A systematic review and pooled analysis. *Acta Psychiatrica Scandinavica, 135*(5), 388–397.
34. Abrams, R. (2002). *Electroconvulsive therapy* (4th ed.). Oxford University Press.
35. Prudic, J., & Sackeim, H. A. (1999). Electroconvulsive therapy and suicide risk. *Journal of Clinical Psychiatry, 60*(Suppl. 2), 104–110.
36. Kranaster, L., & Zachrisson, H. (2015). Modern ECT: Techniques and cognitive safety considerations. *Journal of ECT, 31*(1), 41–46.
37. Verdijk, J. P. A. J., van Kessel, M. A., Oud, M., Kellner, C. H., Hofmeijer, J., Verwijk, E., & van Waarde, J. A. (2022). Pharmacological interventions to diminish cognitive side effects of electroconvulsive therapy: A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica, 145*(4), 343–356.
38. Andrade, C. (2022). Reconsideration of the benefits of pharmacological interventions for the attenuation of the cognitive adverse effects of electroconvulsive therapy. *Journal of Clinical Psychiatry*.
39. Tang, W. K., Ungvari, G. S., et al. (2002). Effect of piracetam on ECT-induced cognitive disturbances: A randomized, double-blind, placebo-controlled clinical trial. *Journal of ECT*.
40. Tremont, G. (1997). Use of thyroid hormone to diminish the cognitive side effects of ECT. *Biological Psychiatry*.

41. Prudic, J., Fitzsimons, L., Nobler, M. S., & Sackeim, H. A. (1999). Naloxone in the prevention of the adverse cognitive effects of ECT. *Biological Psychiatry*.
42. Kranaster, L., & Zachrisson, H. (2015). Modern ECT: Techniques and cognitive safety considerations. *Journal of ECT, 31*(1), 41–46.
43. Vasavada, M., Prudic, J., & Sackeim, H. A. (2017). Electroconvulsive therapy and cognitive effects: Recent developments and clinical considerations. *Current Psychiatry Reports, 19*(12), 101.