INTRODUCTION

Electroconvulsive therapy (ECT), also known as electroshock therapy, is a unique treatment in patients with major depression, affective disorders, catatonia, schizophrenia, and other psychotic disorders for which pharmacological treatments do not produce adequate responses [1, 2]. Historically, ECT was first described in 1938 by Italian doctors Ugo Cereletti and Luigi Bini and was performed without anesthesia for almost 30 years, being referred to as “Unmodified ECT” [3, 4]. With the subsequent development of more advanced medications and their increased use in clinical applications, general anesthesia with an intravenous agent and neuromuscular blocking agent is now performed as an important part of the ECT protocol to improve patient safety, enhance treatment effects, and minimize complications.

Recently, ECT has been reported to produce symptom relief effects in 70–90% of cases, which is a superior outcome to the use of antidepressants and has a recurrence rate of approximately 20% [5]. Moreover, the United States Food and Drug Administration’s recent redesignation of ECT devices as Class II (from Class III) for certain indications may impact the application of this therapy, as this facilitates the continued availability of ECT devices worldwide and helps decrease the stigma associated with this procedure by acknowledging its safety and effectiveness [6]. Thus, the use of ECT is expected to increase.

The worldwide frequency of ECT interventions is approxi-
mately 4.9 (0.4–81.2) out of 10,000 people. In Asian countries, particularly China, Taiwan, and India, there has been a significant increase in the number of reported cases [7–9]. In Korea, some hospitals use ECT in both outpatient and inpatient settings; however, data on the overall clinical applications of this technique are currently lacking [10]. The incidence of ECT is increasing, and anesthesia is an essential component of its safe and successful use. Thus, more anesthesiologists will need to become familiar with the characteristics of this procedure. Our present review focuses on the clinical applications of ECT, anesthetic management during this procedure, pharmacological action of various drugs used in ECT, including anesthetics and neuromuscular blocking agents, possible complications, and postprocedural considerations. Evidence from a literature review and our own experiences are discussed.

**CLINICAL APPLICATIONS**

**Procedural aspects of ECT**

ECT involves the transmission of an electric current through the brain, causing generalized tonic-clonic seizures. During this procedure, the position of the electrode and the physical properties of the electrical stimulation affect the seizure threshold, which is related to the therapeutic effect and cognitive impairment. Three electrode positions (bitemporal, bifrontal, and right unilateral) are commonly adopted. Among these, the bitemporal position is the most widely used. In addition to the electrode positioning and physical properties of electrical stimulation, various factors can also affect the seizure threshold (Table 1).

The antidepressant effects of ECT are related to seizure duration, as measured using electroencephalography (EEG), electromyography, or muscle movement. Seizure duration assessment by muscle movement during general anesthesia is performed by placing a tourniquet on the arm or leg and blocking the blood flow to exclude the effect of muscle relaxants. The seizure duration on an EEG is approximately 5 s longer than the muscle movement [10]. The appropriate motor seizure duration was 25–50 s.

In the acute phase, the number of ECT treatments is not defined but must be performed until the symptoms are relieved or stabilized. Most patients who undergo ECT receive 6–12 treatments per course. However, patients with depression may require fewer patients, while patients with schizophrenia may require more treatment per course [11]. ECT is usually performed two or three times a week, but in certain urgent cases, such as patients with catatonia, daily courses may be used until symptoms improve [12,13]. In rare instances, ECT may need to be interrupted or discontinued due to tolerability issues, such as adverse cognitive effects, fear of anesthesia, headaches, or nausea.

Maintenance ECT (M-ECT) has been used for ongoing procedures to prevent the recurrence of a new episode of depression and can last for years, possibly indefinitely. In most cases, M-ECT has a schedule of 3–8 weeks, but some patients may require longer periods of weekly treatment [14].

**Indications for ECT**

Most guidelines recommend ECT as the first-line treatment for severe depressive episodes, such as the presence of psychotic features, catatonia, high suicide risk, and/or food or fluid refusal. A history of previous positive response and patient preference are also important considerations [15–17]. ECT is recommended as a second-line treatment for patients with severe major depressive episodes that are unresponsive to psychotherapeutic and/or pharmacological interventions. ECT is not recommended for personality disorders, drug abuse, or psychoneuroses. In children, the most common psychiatric indications are refractory depression, bipolar disorder, schizophrenia, catatonia, autism, and refractory status epilepticus [18].

According to the 2001 consensus statement of the American Psychiatric Association (APA), there are no absolute contraindications for ECT [19]. However, some conditions

<table>
<thead>
<tr>
<th>Table 1. Factors Affecting Seizure Thresholds</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Factors that increase the seizure threshold</strong></td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Skull thickness</td>
</tr>
<tr>
<td>Bilateral stimulation</td>
</tr>
<tr>
<td>Repeated stimulation</td>
</tr>
<tr>
<td>Drugs</td>
</tr>
<tr>
<td>Use of barbiturates, benzodiazepines, or anticonvulsants</td>
</tr>
<tr>
<td><strong>Factors that decrease the seizure threshold</strong></td>
</tr>
<tr>
<td>Genuine seizure</td>
</tr>
<tr>
<td>Hyperventilation/hypocapnia</td>
</tr>
<tr>
<td>Female sex</td>
</tr>
<tr>
<td>Hyperoxia</td>
</tr>
<tr>
<td>Drugs</td>
</tr>
<tr>
<td>Use of caffeine, antidepressants, or clozapines</td>
</tr>
</tbody>
</table>
Anesthetic care for ECT

such as uncontrolled hypertension, coronary artery disease, congestive heart failure, aortic stenosis, implanted cardiac devices, atrial fibrillation, obstructive lung disease, asthma, and increased intracranial pressure with or without mass lesions pose a relatively high risk and may result in death during ECT [20,21]. The details of the indications for ECT are summarized in Table 2 [22,23].

Preoperative evaluations

The preoperative evaluation of ECT was comparable to that used in other general surgeries. Medical histories relevant to these assessments and important for a successful ECT include psychiatric history; drug history, including the type, dose, response, compliance, and side effects of any psychiatric drugs; and physical and laboratory examination results such as electrocardiography, chest radiography, serum creatine, and electrolytes [24]. Airway evaluations are also necessary because some conditions, such as difficulties with mask ventilation, a higher risk of pulmonary aspiration, and prolonged ventilation, may require unplanned intubation. In most cases, patients take their usual medications until the morning of the procedure, except for theophylline, herbal medications, or oral diabetes drugs [21].

Although ECT is a low-risk procedure, certain hemodynamic abnormalities may increase the risk of complications in patients with cardiovascular disease. Abrupt hemodynamic changes during ECT typically spontaneously recover a few minutes after a seizure. However, these changes can cause serious complications in patients with cardiovascular disease, and careful monitoring and preparation, including cardiopulmonary resuscitation, are needed in these patients [21,25].

As in adult patients, appropriate preoperative evaluations should be performed prior to ECT in pediatric patients. If a child has comorbidities, additional examinations and an increased planning time for anesthesia are required. In children with central nervous system malignancies, hydrocephalus, or cardiopulmonary diseases, the anesthesiologist should prepare for the interaction of anesthetics and any immediate negative effects of the procedure [18].

Pregnancy testing should be performed in all women of reproductive age. Although pregnancy is not a contraindication for ECT, fetal exposure to anesthetics must be minimized [26].

Physiologic changes during ECT

The physiology of the patient changes dramatically during the ECT. Between the electrical stimulation and the onset of seizure, conditions such as hypotension, bradycardia, and asystole can occur because the parasympathetic nervous system becomes dominant during this period. Tachycardia and hypertension occur during seizures because of rebound sympathetic activity [21,23,27]. In this period, the rate-pressure product (heart rate × blood pressure) increases 2–4 times with a 30–40% increase in systolic blood pressure, a > 20% increase in heart rate, and an increase in the index of myocardial oxygen consumption [28,29]. After seizures, the heart rate and blood pressure normalize within a few minutes, and any serious cardiovascular and complications that arise usually occur during this period [30]. Acute hemodynamic changes during ECT can cause pulmonary edema, ventricular tachycardia, myocardial infarction, and, in rare instances, cardiac shock [31–33].

Cerebral blood flow, intracranial pressure, cerebral metabolic rate, and cerebral oxygen consumption increase during seizures because of transient cerebral ischemia and cerebral hemorrhage [34,35]. Acute neurological and cardiovascular changes, fractures, dislocations, and muscle pain also occur due to generalized convulsions [36,37]. Intraocular pressure also increases during convulsions but normalizes after seizures in most cases [38].

Table 2. Currently Used Indications for Electroconvulsive Therapy

| Major depression, single, or recurrent episodes |
| Bipolar major depression, depressed, or mixed type |
| Schizophrenia |
| Catatonia |
| Schizophreniform or schizoaffective disorder |
| Atypical psychosis |
| Other psychiatric conditions |
| Obsessive compulsive disorder |
| Pregnancy depression, severe postpartum depression, or psychosis |
| Miscellaneous conditions |
| Parkinson’s disease |
| Neuroleptic malignant syndrome |
| Status epilepticus |
| Delirium |
| Dementia with behavioral disturbances |
| Secondary catatonia |
| Dopa-responsive dystonia (Segawa syndrome) |
| Self-injurious behavior in autism |
GENERAL ANESTHESIA FOR ECT

Prior to ECT, patients should fast from solid food for more than 8 h. Clear liquids are permissible during this time to enable oral medications such as antihypertensive drugs to be taken up to 2 h before the procedure. To prevent post-ECT myalgia, patients can be pre-medicated with enteric-coated aspirin, acetaminophen, or intravenous ketorolac. Ventilation during ECT is assisted by a face mask with a standard simple bag-valve-mask system. Tracheal intubation is not recommended, except in very specific situations (e.g., late pregnancy or emergency treatments in which the patient has a full stomach), because ECT is typically performed frequently (two or three times a week for 3–4 weeks), and each procedure lasts only a few minutes. In obese patients with sleep apnea syndrome, an oral airway can be helpful in maintaining ventilation during the procedure.

Non-invasive blood pressure, pulse oximetry, electrocardiography, and capnography are recommended during an ECT procedure. A tourniquet technique or electromyographic monitoring should be employed to quantify the duration of the motor seizure activity. The tourniquet technique is used to isolate the distal circulation using a pressure of 160–200 mmHg before administering the muscle relaxant. Although sufficient muscle relaxation is necessary during ECT, forceful jaw clenching is still inevitable with this intervention because of the direct stimulation of the masticatory muscles, particularly the temporalis and masseter muscles, by electrical current. Hence, a bite block should be carefully placed before the application of the electrical stimulus to protect the patient’s teeth and minimize the risk of lacerating the tongue. Standard noninvasive hemodynamic variables and oxygen saturation should be monitored for 15–30 min after ECT [39]. Emergence agitation after ECT is usually treated by administering a small dose of midazolam or dexmedetomidine [40,41].

GENERAL ANESTHESIA DURING ECT FOR SPECIFIC PATIENT GROUPS

Children and adolescents

Although ECT is known to be safe in adults, it is not commonly used in children and adolescents because of the risk of damage to the nervous system at the early stages. However, the indications for ECT in the pediatric population have increased steadily over the past 20 years [18], the most common of which are refractory depression, bipolar disorder, schizophrenia, catatonia, autism, and pediatric refractory status epilepticus. Unique factors related to pediatric ECT include the potential need for a preoperative anxiolytic with dexmedetomidine, likely to be the most appropriate agent in this regard, as oral benzodiazepines are relatively contraindicated. Methohexital remains the gold standard anesthetic for pediatric ECT; although ketamine, propofol, and sevoflurane are becoming increasingly viable options [18,42,43].

Pregnant cases

ECT has been reported to be an effective and safe treatment for pregnancy-induced depression, unipolar depression, bipolar disorder, schizophrenia, and other psychiatric illnesses [44,45]. However, ECT can cause maternal complications such as aspiration and premature labor, as well as fetal complications such as spontaneous abortion and fetal death. Therefore, a multidisciplinary team approach is required to manage this treatment in pregnant cases [44,46].

When it is difficult to maintain the patient’s airway, or if fasting is insufficient, laryngeal mask airway or cricoid compression and endotracheal intubation can be helpful [47]. In addition, if there is a history of premature labor or uterine contractions following ECT, tocolytics can be used as prophylaxis. In addition, the use of inhaled anesthetics (e.g., sevoflurane) may reduce the risk of uterine contractions after ECT in late pregnancy [48]. Emergency cesarean section may be required in rare instances; therefore, treating clinicians should always be prepared for the possibility of premature delivery in relevant cases to ensure the child’s safety.

COVID-19 era

ECT units have faced certain challenges during the COVID-19 pandemic. These issues include screening, personal protective equipment, airway management, and maintenance of recovery rooms and facilities to prevent the spread and transmission of COVID-19 [49,50]. However, the most challenging of these issues is airway management. ECT requires close supervision by an anesthesiologist and the patient’s oral and airway secretions. Commonly administered mask ventilation and hyperventilation without reliable airway protection increase the risk of aerosolization, which poses a serious risk to health care staff [51]. To overcome this drawback, Luccarelli et al. [52] performed ECT without bag-mask ventilation by applying adequate preoxygenation. The
use of a second-generation supraglottic airway with a viral filter is also helpful in preventing viral transmission. In addition, Limoncelli et al. [53] reported the use of a Jackson–Rees circuit instead of an ambu-bag to provide leakage-free spontaneous ventilation, thus minimizing air emissions.

**DRUGS FOR ECT**

**Anesthetics**

The ideal characteristics of an anesthetic to be used for ECT include rapid onset, attenuation of ECT-induced physiological changes, minimal anticonvulsant effects, and rapid recovery. Although most of the currently available anesthetic agents can be used for ECT, seizure duration, hemodynamic stability, recovery time, antidepressant effect, and cognitive side effects must be considered when selecting this drug. Most anesthetics have a dose-dependent anticonvulsant effect; therefore, the minimum effective dose should be used during ECT [54]. The effects of commonly used anesthetics for ECT are summarized in Table 3.

1. **Methohexital**

Methohexital is the gold standard drug among the established anesthetics [55,56]. The routine dosage of this agent for ECT is 1.5 ± 0.3 mg/kg, but the Royal College of Psychiatrists (0.75–0.9 mg/kg) and APA (0.75–1.0 mg/kg) have recommended a dose reduction [56]. It remains the drug of choice for ECT except where there are barbiturate contraindications (e.g., acute intermittent porphyria) because they have few hemodynamic effects and low anticonvulsant properties [1,23]. However, methohexital is currently unavailable commercially in Korea.

2. **Thiopental sodium and thiamylal**

Thiopental sodium (1.5–2.5 mg/kg) and thiamylal (1.5–2.5 mg/kg) reduce the seizure duration and have a slower recovery compared to methohexital (0.5–1.0 mg/kg). Both of these agents also increase the incidence of arrhythmias such as sinus bradycardia and premature ventricular contraction, as well as increase the blood flow in the middle cerebral artery after ECT compared with propofol [57,58]. Moreover, they produce more hemodynamic changes than sevoflurane [59]. Hence, the use of thiopental and thiamylal as intravenous anesthetics for ECT is not advantageous.

3. **Etomidate**

Etomidate (0.15–0.3 mg/kg) is effective in patients with a short seizure duration (i.e., < 20 s) even under maximum stimulation because it prolongs this duration compared to methohexital, thiopental, or propofol [55,60]. However, etomidate also increases the incidence of confusion, delirium, nausea, and vomiting after ECT compared to other anesthetics such as propofol, methohexital, and thiopental [1,23,60]. Etomidate-induced myoclonic jerks should be differentiated from seizures after ECT, as long-term use of etomidate can cause adrenal insufficiency [61].

4. **Propofol**

Propofol is the most commonly used intravenous anesthetic owing to its rapid recovery and antiemetic mode of action. However, the seizure duration after ECT is shorter with this drug because it has stronger anticonvulsant effects than other intravenous anesthetics [61–63]. Propofol is thus preferred for use in adolescents and young adults receiving ECT because they typically have a lower seizure threshold and longer duration of seizures than adults [64]. The routine dosage of propofol is 1.0–1.5 mg/kg. If the minimum hyp-

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**Table 3.** Effects of Commonly Used Anesthetics in Electroconvulsive Therapy Protocols and Comparisons of the Physiologic Changes Before and After Electrical Stimulation (Before/After)

<table>
<thead>
<tr>
<th></th>
<th>Heart rate</th>
<th>Blood pressure</th>
<th>Cerebral blood flow</th>
<th>Seizure duration</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methohexital</td>
<td>↓ / ↑</td>
<td>↓ / ↑↑</td>
<td>NE</td>
<td>→</td>
<td>Standard anesthetics for ECT</td>
</tr>
<tr>
<td>Thiopental</td>
<td>↑ / ↑</td>
<td>↓ / ↑↑</td>
<td>↓ / ↑↑</td>
<td>↓</td>
<td>Histamine release</td>
</tr>
<tr>
<td>Etomidate</td>
<td>→ / ↓</td>
<td>↓ / ↑↑</td>
<td>NE</td>
<td>↑</td>
<td>Injection pain, slow recovery</td>
</tr>
<tr>
<td>Propofol</td>
<td>↓ / ↑ →</td>
<td>↓ / ↑</td>
<td>↓ / ↑</td>
<td>↓</td>
<td>Injection pain</td>
</tr>
<tr>
<td>Ketamine</td>
<td>↑ / ↓</td>
<td>↓ / ↑↑</td>
<td>↓ / ↑↑</td>
<td>↓</td>
<td>Psychotic action</td>
</tr>
<tr>
<td>Benzodiazepine</td>
<td>→ / ↑</td>
<td>↓ / ↑</td>
<td>NE</td>
<td>↓</td>
<td>Long acting</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>↑ / ↓</td>
<td>↓ / ↑</td>
<td>↓ / ↑↑</td>
<td>↓</td>
<td>Slow induction</td>
</tr>
</tbody>
</table>

ECT: electroconvulsive therapy, NE: not evaluated.
notic dose (0.75 mg/kg) is used, the seizure duration is similar from that seen with methohexital [62]. Although propofol produces a shorter duration of seizures, analysis of the antidepressant effects of ECT, such as the Hamilton rating efficiency and Beck Depression Inventory score, show that the use of propofol has similar outcomes to those achieved with methohexital [65]. As propofol has cardiovascular inhibitory properties, it can suppress acute hemodynamic changes immediately after ECT. Hence, it is preferred in patients with hypertension, tachycardia, or expected hemodynamic changes after ECT [64].

5. Ketamine

Ketamine is an intravenous anesthetic with both hypnotic and analgesic effects. The recommended dose of ketamine (1–2 mg/kg) can help achieve the desired ECT effects, but a low dose of this drug (0.4–0.8 mg/kg) leads to a shorter seizure duration on an EEG compared to methohexital. Because ketamine can also increase blood pressure, heart rate, and intracranial pressure, it is not generally preferred over methohexital or propofol for use in ECT procedures [54,66]. Moreover, it can induce psychiatric side effects such as agitation, confusion, delirium, and disorientation [64,67]. However, as ketamine has antidepressant properties, it is preferred in patients with depression [66].

6. Benzodiazepine

Benzodiazepines, such as midazolam and lorazepam, can alter the threshold and duration of seizures after ECT. In patients who have been taking benzodiazepine over the long term, seizures may not occur owing to its anticonvulsant effects [68]. Recently, remimazolam, a novel ultra-short-acting benzodiazepine, has been approved in many countries. Although there are no published reports on the effects of remimazolam as part of an ECT protocol, it may have anticonvulsant effects similar to those of other benzodiazepine drugs [69].

7. Dexmedetomidine

Dexmedetomidine is rarely used alone; in combination with other intravenous anesthetics, it can reduce the acute hemodynamic changes that are possible after ECT. Moreover, if dexmedetomidine at a 1 µg/kg dose is administered 10 min prior to ECT, it can reduce the extent of post-ECT agitation without affecting seizure duration or patient recovery time [70,71].

8. Inhalation anesthetics

Most ECT procedures are performed outside the operating theatre, and intravenous anesthesia is generally preferred over inhalational anesthesia. However, as possible inhalation anesthetics, sevoflurane (1.7%) and nitrous oxide (50%) can more potently reduce acute hemodynamic changes following ECT than thiopental [59]. The seizure duration and recovery times with these drugs were similar to those with thiopental. Inhalation anesthetics require a longer induction time than intravenous agents but can reduce the risk of uterine contractions after ECT in late pregnancy cases [72].

Neuromuscular blocking agents

Neuromuscular blocking agents are necessary to prevent possible musculoskeletal complications of ECT, such as myalgia, dislocation, and fracture, and are effective because they typically have a fast onset and a short duration of action [36,37].

1. Succinylcholine

Succinylcholine is the oldest and most commonly used neuromuscular blocking agent in ECT protocols [56,73]. The recommended dosage is 0.5 mg/kg, but higher doses (0.75–1.5 mg/kg) are also used in clinical practice [74]. Therefore, higher doses of succinylcholine should be avoided in patients with bradycardia [75]. Even at low concentrations, there is a risk of side effects (e.g., myalgia, malignant hyperthermia, hyperkalemia) in patients who are susceptible to malignant hyperthermia, neuroleptic malignant syndrome, catatonic schizophrenia, or organophosphate poisoning [76–78]. Because its duration of action may be prolonged, it must be used cautiously in patients with pseudocholinesterase deficiency or any kind of muscular dystrophy [54].

2. Atracurium and cisatracurium

In patients receiving intravenous atracurium, a 0.3 mg/kg pretreatment leads to significantly more ECT-induced moderate and vigorous convulsions (86 vs. 16%) and a shorter recovery time (4.2 ± 0.4 min vs. 9.2 ± 0.8 min) when compared with patients receiving 0.3 mg/kg [79]. Therefore, a low dose of atracurium is recommended when succinylcholine cannot be used. However, even small doses of atracurium (10–15 mg) can cause delayed recovery in patients with atypical plasma cholinesterase [80]. Clinically, cisatracurium is now starting to replace atracurium, but few studies have addressed its effectiveness using ECT.
3. Vecuronium and rocuronium

Vecuronium and rocuronium are non-depolarizing neuromuscular blocking agents with an aminosteroid structure that can also be used as part of the ECT protocol. Although the long duration of action has been a problem with these treatments, the development of sugammadex could make them useful in ECT. Sugammadex is a cyclodextrin-based compound with an antagonistic mode of action against aminosteroid nondepolarizing neuromuscular blockers. If sugammadex was used in conjunction with rocuronium during ECT, rapid onset of action and recovery could be expected. Hence, this potential drug combination has attracted attention as a possible alternative to succinylcholine [81,82]. In addition, calabadian, a new antagonist of benzylisoquinoline-based neuromuscular blocking agent, and a combination of gantacurium (CW002) and L-cysteine are anticipated to become part of future ECT procedures [83].

Drugs for the treatment of cardiovascular reactions during ECT

As acute cardiovascular reactions following ECT can cause serious complications, cardiovascular drugs are used to relieve acute parasympathetic and sympathetic reactions [29,84]. Some of these agents may affect the duration of seizures; however, the choice of drug should be made carefully [85].

1. Anti-cholinergics

Pretreatment with anticholinergics as part of the ECT protocol has been reported to reduce the incidence of premature atrial contracture, bradycardia, and asystole, as well as decrease secretion and salivation [57]. Glycopyrrolate (0.1–0.3 mg, i.v.) is the preferred agent in this regard because it can reduce salivation and bradycardia after ECT without side effects such as cognitive impairment [86].

2. β-blockers

β-blockers, such as esmolol and labetalol, attenuate the sympathetic and cardiovascular responses following ECT. Pretreatments of ECT patients with esmolol (1.0 mg/kg) or labetalol (0.3 mg/kg) are more effective than those with fentanyl (1.5 mg/kg) or lidocaine (1.0 mg/kg) [27]. Because esmolol can also decrease the duration of seizures, it is recommended to be administered immediately before or immediately after ECT [1,23].

3. Calcium channel blocker

Nicardipine (1.25–5 mg, i.v.) has a rapid hemodynamic control effect without impact on the cardiovascular inhibitory action of methohexital due to its rapid onset. Moreover, small doses of nicardipine have little effect on the duration of seizures [85]. Rebound tachycardia can occur after bolus administration of this drug, but intravenous administration of labetalol can attenuate this. A nicardipine and labetalol combination has also been reported to lower the mean arterial pressure immediately after ECT in comparison to labetalol alone [87].

4. Vasodilators

Nitroglycerin (NTG, 3 μg/kg, i.v.) can reduce hemodynamic changes after ECT compared to esmolol (2 mg/kg, i.v.) [27]. NTG has no effect on the duration of seizures [88]. In addition to the intravenous administration of this drug, a sublingual, patch, and ointment delivery method also reduces the onset of hemodynamic changes after ECT [89,90]. Nitroprusside is preferred in patients with intracranial aneurysms, dissecting aortic aneurysms, or aortic stenosis [91–93]. A β-blocker combined with nitroprusside lowers the incidence of tachycardia and hypertension and increases the blood flow velocity in the middle cerebral artery [34]. Nitroprusside also has no effect on seizure duration in ECT [94].

5. Ganglionic blocking agents

Although trimethaphan is not currently the preferred drug in clinical practice, its bolus administration at 5–15 mg can control hemodynamic changes after ECT without affecting the duration of the seizure [95]. Moreover, there were no side effects after ECT, such as rebound hypertension, arrhythmia, or hypotension [1,85].

6. Local anesthetics

Lidocaine can also attenuate the onset of hemodynamic changes after ECT, but it also decreases seizure duration in a dose-dependent manner [27,96].

7. Opioids

Opioids can act as a “seizure enhancers” by reducing the required hypnotic dose. Hence, short-acting opioids are effective in patients with an insufficient duration of seizures following ECT [1]. However, the effects of ECT have not been reported to be greater than those of hypnotics alone [97]. Fentanyl (1.5 μg/kg, i.v.) shortens the seizure duration and does not alleviate ECT-induced hemodynamic changes [27].
Remifentanil (0.05–1.0 μg/kg/min) can prolong the duration of seizure by 27–38 s without impact on hemodynamic changes or recovery time [98,99]. Pethidine and tramadol are not recommended for use with ECT because they may interact with other antidepressants (e.g., monoamine oxidase inhibitors or selective serotonin reuptake inhibitors), potentially leading to hypertensive crises and/or serotonin syndromes [100].

**Table 4. Physiologic Changes and Adverse Events Associated with Electroconvulsive Therapy**

<table>
<thead>
<tr>
<th>System</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central nervous system</td>
<td>Increases in the cerebral blood flow, intracranial pressure, and cerebral metabolic rate</td>
</tr>
<tr>
<td></td>
<td>Dizziness, headache, amnesia, agitation, cognitive impairment, delirium, cerebral hemorrhage</td>
</tr>
<tr>
<td>Cardiovascular system</td>
<td>Increases in blood pressure, heart rate, and cardiac output</td>
</tr>
<tr>
<td></td>
<td>Arrhythmia, hypertension, myocardial infarction, stress-induced cardiomyopathy</td>
</tr>
<tr>
<td>Musculoskeletal system</td>
<td>Tonic - clonic seizure</td>
</tr>
<tr>
<td></td>
<td>Myalgia, dislocation, fracture</td>
</tr>
<tr>
<td>Others</td>
<td>Increased salivation</td>
</tr>
<tr>
<td></td>
<td>Nausea, vomiting, dental fractures, lacerations of the gum, gingiva, and tongue</td>
</tr>
</tbody>
</table>

8. Magnesium sulfates

Magnesium sulfates can reduce ECT-related hypertension and have no effect on seizure duration. The combined use of these compounds with remifentanil may delay the recovery of spontaneous respiration but can also prevent tachycardia and hypertension in elderly patients with ischemic heart disease [101].

ECT is a safe procedure in patients with minimal comorbidities. However, cardiovascular changes and psychiatric complications may occur following treatment. Pulmonary aspiration, respiratory failure, and residual neuromuscular blockade must be considered as possible complications of ECT interventions because neuromuscular blocking agents are used [102]. Although cognitive impairment is common after ECT, it is not permanent. Osler et al. [103] reported that ECT is not associated with dementia. Postictal delirium or agitation may also occur after ECT but should respond to small amounts of midazolam or propofol [104]. An extreme increase in cerebral blood flow due to sympathetic stimulation is also associated with intracranial hemorrhage. Succinylcholine-related myalgia responds well to nonsteroidal anti-inflammatory drugs such as ketorolac [18]. Prophylactic antiemetics may be recommended for high-risk patients or drugs such as sevoflurane and etomidate. Typical physiological changes and adverse events associated with ECT are summarized in Table 4.

**CONCLUSION**

ECT is a safe and effective treatment for various psychiatric disorders, and accepted indications for its use has steadily increased over time. Anesthesia during ECT should ideally provide deep hypnosis, ensure muscle relaxation to reduce injury, have minimal effects on seizure duration, and allow for rapid recovery to a baseline neurological and cardiopulmonary status. Multiple anesthetic agents are acceptable for use during ECT, and the choice of this drug should be considered for any underlying comorbidities that the patient has.

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None.

**CONFLICTS OF INTEREST**

No potential conflict of interest relevant to this article was reported.

**DATA AVAILABILITY STATEMENT**

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

**AUTHOR CONTRIBUTIONS**

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