

Shigeru Saito *Editor*

# Anesthesia Management for Electroconvulsive Therapy

Practical Techniques and  
Physiological Background



Springer

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Background

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## Preface

Electroconvulsive therapy (ECT) was introduced in clinical practice based on the finding that psychiatric symptoms are improved after a seizure in a patient suffering from both schizophrenia and epilepsy. In spite of the great effort expended, the underlying mechanism has not been fully elucidated. ECT therapy has undergone several decades of criticism, accusations of inappropriate use, legal restrictions, and public protest. However, recent controlled studies have demonstrated clinical benefits of this therapy and many efforts have been made to reduce the special risks in ECT. Specifically, the introduction of general anesthesia using intravenous anesthetics and a muscle relaxant greatly reduced physical risks relating the muscular convulsive movement during the therapy. Currently, ECT for drug therapy-resistant depression and some other psychiatric disorders is widely accepted as a safe and effective therapy. Use of ECT for several psychiatric conditions is expanding, and the number of patients receiving ECT is increasing.

Although the basic mechanism of the clinical effects of ECT has not been clarified, several authorized medical societies, including the Royal College of Psychiatrists in the United Kingdom and the American Psychiatric Association, have published guidelines and audit-reports to promote the safe and effective use of ECT and to prevent its misuse. According to such guidelines, this therapy is applied under general anesthesia in most advanced countries. Apparently, this approach is more safe and effective compared to the original method without anesthesia. The most recent version of the guideline by the American Psychiatric Association clearly stated that anesthesia for ECT should be administered by a specially trained anesthesiologist, and that the anesthesiologists should have overall responsibility, not only for anesthesia itself but also for cardiopulmonary management and emergency care.

Under these circumstances, anesthesiologists who administer anesthesia for ECT should have sufficient knowledge regarding the physiologically and pharmacologically unique effects of ECT. However, the number of descriptions regarding the anesthesia management for ECT is limited, and most such descriptions are relatively short review articles. A handy practical guidebook is considered to be attractive for young doctors and co-medical staffs working on this therapy. This ECT anesthesia guidebook is prepared to respond to such requests from clinicians.

Seven experts in this field earnestly reviewed recent clinical and basic literature regarding ECT anesthesia and described most updated knowledge in plain language. As editor, I believe that anesthesiologists, psychiatrists, and the other medical staff working on ECT can master ECT anesthesia by reading this guidebook and by applying the techniques to their clinical cases.

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Shigeki Yamaguchi and Toshifumi Takasusuki

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## Abstract

Electric stimulus and seizure during the electroconvulsive therapy (ECT) make patients various impacts so that it is recommended to perform ECT under general anesthesia (modified ECT: mECT). To optimize the anesthetic management of patients under mECT, it is important to understand the physiologic responses to the electrical stimulus, even though under general anesthesia. And also, the early assessment of patient's physical condition is required, since there may be some physical problems because of psychiatric disorders, medications and others in patients required for mECT. Therefore, pre-procedure assessment, including inquiry, check of medications and usual examinations, is also important and required as normal general anesthesia. If needed, special examinations and special monitor during anesthesia should be ordered. In this chapter, some points for pre-procedure assessments and conditions for mECT were described.

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## Keywords

Depression • Schizophrenia • Antidepressant • Antipsychotic • QT prolongation

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## 1.1 Introduction

Electroconvulsive therapy (ECT) is a useful therapy for many psychiatric disorders, and the number of indications for which it is used is expanding (Table 1.1) [1–3]. Initially, ECT was performed without any anesthesia, resulting in serious problems such as fractures, tooth damage, and cardiovascular events, among others.

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**Table 1.1** Psychiatric diagnoses for which mECT has been claimed to be effective

1.	Major depression, single or recurrent episode
2.	Bipolar major depression, depressed or mixed type
3.	Bipolar disorder, mania or mixed type
4.	Schizophrenia
	Catatonia
	Schizophreniform or schizoaffective disorder
5.	Atypical psychosis
6.	Other conditions
	Organic delusional disorder
	Organic mood disorder
	Acute psychotic disorder
	Obsessive-compulsive disorder
	Dysthymia
7.	Miscellaneous conditions
	Parkinson's disease
	Neuroleptic malignant syndrome
	Secondary catatonia
	Lethal catatonia

As a result, it is now common to perform ECT while the patient is under general anesthetic [4].

The spread of modified ECT (mECT) has increased the number of psychiatric patients undergoing ECT. Currently, more mECT is performed than coronary artery bypass or appendectomy in the USA [2]. Furthermore, meta-analysis data have indicated that mECT may be more effective than antidepressants [5, 6].

Although electric stimuli and seizures affect patients in a variety of ways, anesthetic management for mECT may not have been given sufficiently serious consideration. However, anesthetic management of mECT must be considered in the same way as other cases undergoing general anesthesia, because hypnotics and muscle relaxants are required. Particular attention must also be paid to patients requiring mECT, as their conditions can be complicated and high risk.

Therefore, mECT must involve anesthesiologists who specialize in anesthetic management. Guidelines by the American Psychiatric Association state that collaboration with trained anesthesiologists can make mECT safer [6]. This chapter describes points to note regarding pre-procedure assessments and specific conditions in mECT.

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## 1.2 Indications and Contraindications for mECT [7]

mECT is a useful therapy for many psychiatric and other disorders, especially drug-resistant conditions (Table 1.2). Table 1.3 lists symptoms that are responsive to ECT, indicating the wide variety of conditions for which mECT can be useful.

**Table 1.2** Criteria for primary use and mECT

Situations in which ECT should be considered prior to a trial of psychotropic agents include the following:

1.	Where a need for rapid, definitive recovery exists on either medical or psychiatric grounds
2.	When the risks of other treatments outweigh the risks of ECT
3.	When a history of poor drug response and/or good ECT response exists in previous episodes of the illness
4.	Patient preference

**Table 1.3** Symptoms responsive to mECT

1.	Symptoms associated with depression: suicidal ideation, depressed mood, delusions of guilt, hypochondriasis
2.	Symptoms associated with other functional psychoses: depression, stupor, psychomotor excitement, delusions or illusions, anxiety, or agitation
3.	Secondary depression or confusion associated with various physical diseases
4.	Muscular rigidity associated with parkinsonism or neuroleptic malignant syndrome

On the other hand, mECT is contraindicated in some conditions. For instance, intracranial hypertension is an absolute contraindication; relative contraindications include intracranial lesion, cerebral aneurysm, recent myocardial infarction, angina pectoris, heart failure, untreated glaucoma, severe fractures, thrombophlebitis, pregnancy, and retinal detachment, among others.

### 1.3 Physiologic Responses to mECT [4, 8–12]

To optimize the management of anesthesia in patients undergoing mECT, an understanding of the physiologic responses to electrical stimulus is important. An electrical current applied to the brain via transcutaneous electrodes induces a systemic tonic-clonic seizure, usually comprising a tonic phase lasting 10–15 s and a tonic phase for 30–60 s.

During the tonic phase, the parasympathetic nervous system is activated, resulting in bradycardia and hypotension. Immediately afterwards, the clonic phase activates the sympathetic nervous system, resulting in tachycardia and hypertension. Although changes that appear similar to myocardial ischemia on electrocardiogram occur immediately after electrical stimulus, cardiac enzyme levels usually do not increase.

Electrical stimulus and subsequent seizure increase both cerebral blood flow and cerebral metabolism, leading to intracranial hypertension. Intraocular pressure and gastric pressure also increase after mECT.

## 1.4 Assessment of Patients before mECT [4, 13, 14]

The pre-procedure examination is as important and necessary as for general anesthesia in healthy subjects. Since patients requiring mECT may also have physical problems, early assessment of their physical condition is necessary. Special examinations may be necessary.

### 1.4.1 Inquiry

Certain details, such as complications, medical history, anesthetic history, and medication lists, among others, must be carefully checked. Since communication with patients requiring mECT can be difficult, it may be necessary to ask the patient's family and attending physicians.

### 1.4.2 Pre-procedure Examination (Table 1.4)

A regular examination should be conducted and evaluated in the same way as for any normal general anesthesia. Abdominal X-ray is required because bowel dysfunction may occur in some patients. The QT interval must be carefully checked via electrocardiogram because many patients requiring mECT may exhibit QT interval prolongation. Brain computed tomography (CT), while not necessary before normal general anesthesia, is essential before mECT to check for the presence of intracranial occupying lesions.

### 1.4.3 Check of Medications

Many patients requiring mECT have drug-resistant conditions and therefore receive pharmacotherapy with typical or atypical antipsychotics and/or many types of antidepressants. In patients with drug-resistant conditions, medications—not only current but also past—should be checked in detail, as follows.

**Table 1.4** Pre-procedural examination list

1.	Minimum requirements Chest and abdominal X-ray, ECG, brain CT, SpO <sub>2</sub> , laboratory examination (Alb, BUN, Cr, Na, K, Cl, AST, ALT, LDH, ChE, CPK, Glu, CRP, WBC, Hct, Hb, Plt, Hb-Ag, HCV-Ad, STS)
2.	Adequate requirements Brain-MTI, brain-MRA, EEG, SPECT, respiratory function, cardiac function, echocardiography, holter-ECG

## 1.5 Preparation for Anesthetic Management of mECT

Although the duration of anesthetic management for mECT is short, preparation must be the same as for any normal general anesthesia, including instructions regarding nil by mouth and internal use as well as venous lines and various monitors.

### 1.5.1 Monitoring

Table 1.5 lists recommendations on monitoring during anesthesia published by the Japanese Society of Anesthesiologists [15]. This monitoring is necessary to quickly evaluate physiological changes as a result of mECT.

### 1.5.2 Instructions Regarding Nil by Mouth

As described, patients requiring mECT may have bowel dysfunction, meaning aspiration is a risk during anesthesia. Therefore, enough time should be provided for nil by mouth to take effect; ideally, patients are required to stop eating from 9 pm the day before receiving mECT and to stop drinking 6 h before mECT.

### 1.5.3 Instructions Regarding Internal Use

All medications, except cardiovascular agents, must be discontinued for a certain period before mECT. The timing for stopping each drug is shown in Table 1.6. In some patients, if discontinuing medication is difficult, internal use may be continued until 1 day before the procedure.

**Table 1.5** Recommended monitoring during anesthesia, published by the Japanese Society of Anesthesiologists

①	An anesthesiologist must remain by the patient's side during anesthesia and must constantly observe the patient
②	Oxygenation must be checked via skin color and/or pulse oximeter
③	Evaluation of respiration via thoracic motion, capnography, or ventilation monitor is recommended
④	Hemodynamics must be checked via heart sound, palpation of the arteries, or pulse oximeter
	Electrocardiogram must be monitored
	Blood pressure must be checked at 5-min intervals
	Invasive arterial pressure measurement may be required as necessary
⑤	Body temperature must be checked
⑥	Degree of neuromuscular block may be checked as necessary

**Table 1.6** Timing of discontinuation of each drug before mECT

	Timing of discontinuation	Notes
Atypical antipsychotics (phenothiazine, butyrophenone, etc.)	2 weeks before mECT	The $\alpha$ -receptor-blocking function of atypical antipsychotics may induce hypotension during anesthesia
Monoamine oxidase inhibitors	10 weeks before mECT	Hypertensive crisis may occur if an indirect vasoconstrictor such as ephedrine is administered in patients receiving monoamine oxidase inhibitors
Lithium carbonate	24 h before mECT	Lithium may be a risk factor for cognitive deficit after general anesthesia. Lithium may also increase muscle relaxation by suxamethonium or rocuronium
Anticonvulsants	2–3 times the half-life of each anticonvulsant before mECT	Seizure threshold may be increased in patients receiving anticonvulsant therapy. Phenytoin and carbamazepine may reduce muscle relaxation properties of non-depolarizing muscle relaxants

## 1.6 Pre-procedure Assessment of Patients with Depression [16–27]

In mECT for patients with depression, a detailed investigation of pharmacotherapy, cardiovascular disease, QT prolongation, and diabetes, among others, is necessary before the procedure.

### 1.6.1 Pharmacotherapy

A list of medications for depression is shown in Table 1.7. Antidepressants vary widely, with the first line usually being selective serotonin reuptake inhibitors (SSRIs), then tricyclic antidepressants (TCAs) or tetracyclic antidepressants (TeCAs) may be selected for moderate to severe depression. Patients requiring mECT are usually receiving pharmacotherapy with TCAs or TeCAs. Table 1.8 lists details to note regarding these medications.

### 1.6.2 Cardiovascular Disease

Patients with depression have a high risk of cardiovascular disease, regardless of pharmacotherapy; reasons for this are shown in Table 1.9. Depression itself is

**Table 1.7** List of medications for depression

First generation (tricyclic)	Imipramine, amitriptyline, trimipramin, nortriptyline, clomipramine
Second generation (tricyclic)	Amoxapine, lofepramine, dosulepin
Second generation (tetracyclic)	Maprotiline, mianserin, setiptiline
Second generation (others)	Trazodone, sulpiride
Third generation (SSRI)	Fluvoxamine, paroxetine
Fourth generation (SNRI)	Milnacipran, duloxetine
Fourth generation (NaSSA)	Mirtazapine

**Table 1.8** Points to note about tricyclic antidepressants and tetracyclic antidepressants

1.	Risk for myocardial infarction
2.	Tachycardia and increase in myocardial oxygen consumption due to cholinergic effects
3.	Hemodynamic events, such as hypotension, reduction of cardiac contractility, or sudden death, among others, because of depletion of noradrenaline at nerve endings and down-regulation of $\beta$ -adrenergic receptor
4.	QT prolongation and risk for Torsade de Pointes

**Table 1.9** Reasons for cardiovascular diseases in patients with depression

1.	Risk for arrhythmia due to heart rate variability by depression
2.	Risk for myocardial infarction due to platelet aggregation by dysregulation of the serotonin system
3.	Acceleration of atherosclerotic lesions due to enhancement of the immune system
4.	Deterioration of lifestyle, high smoking rates, alcohol polydipsia, and decrease in physical activities, among others, related to reduced motivation for healthy behaviors
5.	Hypotension, decrease in cardiac contractile force and prolongation of QT interval due to tricyclic antidepressant and tetracyclic antidepressant
6.	High risk for diabetes

considered a risk factor for ischemic heart disease and myocardial infarction. A report has indicated that the TCAs and TeCAs increase the risk for cardiovascular events. On the other hand, SSRIs are safe for use in ischemic heart disease and may provide a preventive effect for cardiovascular events.

### 1.6.3 QT Prolongation

It is well known that TCAs and TeCAs increase the QT interval, resulting in Torsade de Pointes. Therefore, the QT interval must be evaluated carefully before mECT. Table 1.10 shows special risks regarding QT prolongation in patients receiving TCAs or TeCAs. SSRIs and serotonin-norepinephrine reuptake inhibitors (SNRIs) may pose less risk than TCAs and TeCAs.

**Table 1.10** Special risks regarding QT prolongation

1.	Congenital long QT syndrome
2.	Heart failure
3.	Bradycardia
4.	Electrolyte imbalance
5.	Excessive administration of psychotropic
6.	Excessive administration of antidepressant
7.	Female
8.	Elderly
9.	Liver or renal dysfunction

### 1.6.4 Diabetes

Patients receiving antidepressants may be at risk for diabetes. A report has indicated that taking antidepressants for more than 2 years increases the risk of diabetes. Furthermore, in patients with diabetes and depression, glycemic control may be unstable, resulting in high mortality. Therefore, risk of diabetes in a patient with depression must be evaluated before the procedure. Blood glucose level must be controlled carefully during mECT therapy.

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## 1.7 Pre-procedure Assessment of Patients with Schizophrenia [28–42]

Patients diagnosed with schizophrenia and requiring mECT may also have many physical diseases. Furthermore, the presence of these diseases may go unnoticed because communication with patients with schizophrenia may be difficult.

Patients with schizophrenia are usually known to have a higher risk for peri-operative complications than healthy subjects. Table 1.11 shows possible problems that may occur during mECT therapy.

### 1.7.1 Pharmacotherapy

Table 1.12 shows a medication list for schizophrenia. Antipsychotics are varied and are classified as typical or atypical antipsychotics. Usually, patients requiring mECT have been receiving multiple typical antipsychotics over a long period. Furthermore, these patients have often also received other types of medications, such as antiparkinson agents to prevent parkinsonism. These agents have side effects such as ileus, hydrodipsia, and cognitive decline, among others.