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Anesthesia management for electroconvulsive therapy: hemodynamic and respiratory management

Shigeru Saito

Department of Anesthesiology, Gunma University Graduate School of Medicine, 3-39-22 Showa-machi, Maebashi 371-8511, Japan

Abstract
Recent guidelines have stated that anesthesia for electroconvulsive therapy (ECT) should be administered by a specially trained anesthesiologist, and that anesthesiologists have overall responsibility, not only for anesthesia itself, but also for cardiopulmonary management and emergency care. Accordingly, anesthesiologists who administer anesthesia for ECT should have sufficient knowledge regarding the physiologically and pharmacologically unique effects of ECT. Electrical current during ECT stimulates the autonomic nervous system and provokes unique hemodynamic changes in systemic and cerebral circulation. Excessive alterations in heart rate, blood pressure, and cardiac functions should be prevented by medications with anticholinergic and antihypertensive agents. Ventilation should be adequately maintained to ensure the efficacy of the therapy and to stabilize the hemodynamics immediately after the electrical stimulation. Reports of serious complications of this therapy are not frequent; however, patients with ischemic heart disease or cerebrovascular problems must be managed with special care to prevent myocardial infarction or neurological disorders. Safe physical management by anesthesiologists greatly contributes to the establishment of ECT under muscle relaxation. To maintain social confidence and to refine the therapy, anesthesiologists should play an essential role both in clinical activities and in laboratory research.

Key words Depression · Seizure · Ambulatory anesthesia · Psychiatrics

Introduction

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 [1,2]. ECT therapy has undergone several decades of criticism, accusations of inappropriate use, legal restrictions, and public protest [3]. However, recent controlled studies have demonstrated clinical benefits of this therapy, and many efforts have been made to reduce the special risks in ECT [4,5]. Specifically, the introduction of general anesthesia using intravenous anesthetics and a muscle relaxant greatly reduced physical risks related to 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.

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 [6] and the American Psychiatric Association [7], have published guidelines and audit reports to promote the safe and effective use of ECT and to prevent its misuse. 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 have overall responsibility, not only for anesthesia itself, but also for cardiopulmonary management and emergency care [7,8]. Accordingly, anesthesiologists who administer anesthesia for ECT should have sufficient knowledge regarding the physiologically and pharmacologically unique effects of ECT.

Hemodynamic management

Systemic circulation

Mechanism

Electrical current during ECT stimulates the autonomic nervous system and provokes unique hemodynamic

Address correspondence to: S. Saito
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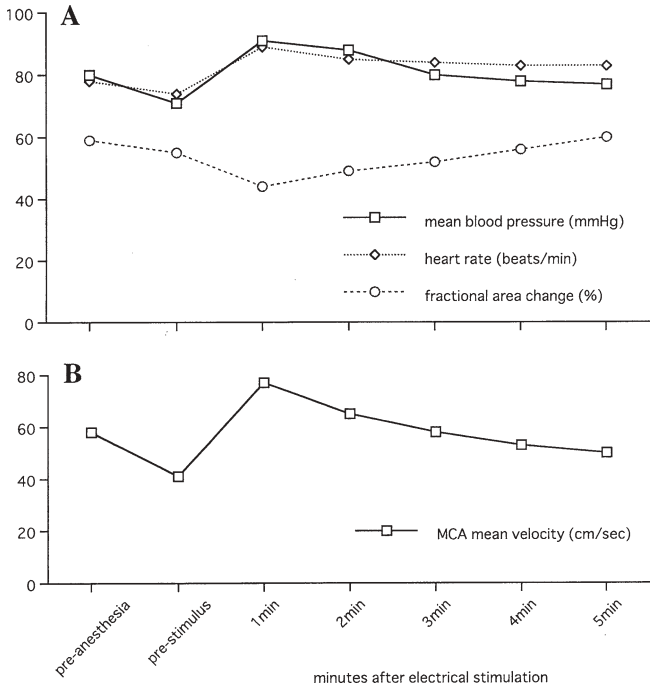

Fig. 1. Typical change of systemic and cerebral hemodynamics in a patient. **A** Typical changes in heart rate, blood pressure, and fractional area change. **B** Typical change in cerebral blood flow velocity measured at middle cerebral artery (*MCA*)

changes in systemic circulation [9,10]. First, a parasympathetic discharge upsurge occurs immediately after application of the electrical current; this phase ensues within a few seconds. Second, sympathetic systems are stimulated and catecholamines are released from the adrenal medualla and sympathetic nerve terminals [11]. This phase continues for 10 min approximately. Several unique hemodynamic changes are provoked by the sequential autonomic activation (Fig. 1).

Heart rate

Patients presented for ECT may have tachycardia before the therapy due to dehydration induced by difficulty in oral intake [7,12]. Vasodilative intravenous anesthetics, such as thiopental, provoke a further increase in heart rate [13]. In contrast, propofol maintains a stable heart rate, or decreases the heart rate to some extent [13]. Parasympathetic discharge immediately after electrical stimulation suppresses the heart rate [14]. In some patients, the bradycardia is severe, and temporary asystole might be observed [15–17]. However, this phase is completed within a minute and the following sympathetic activation induces tachycardia. If a patient has an arrhythmogenic tendency, dysrhythmia might be provoked in this phase [18]. This tachycardia is augmented by excessive hypocarbia induced by intended hyperventilation [19], hypercarbia induced by inappropriate ventilation [20], use of vasodilative antihypertensive drugs [13], or elongated seizure activity [21]. In adequately managed patients, this tachycardia phase is completed within 2–5 min [20].

Blood pressure

Many patients using psychotropic drugs are relatively hypotensive [22]. If the patient has a problem in food intake, the hypotension can be aggravated by dehydration. Intravenous anesthetics used in ECT, except for ketamine and etomidate, induce temporary hypotension due to their vasodilative and myocardial depressant properties [13]. However, sympathetic stimulation by electrical current and the following catecholamine increase overwhelm the hypotensive effects and elevate blood pressure by 20%–50% as compared to a prestimulus value [9,23]. The elevated blood pressure gradually decreases within 2–5 min when the patient is appropriately ventilated [20]. If hypercapnea and/or hypoxia exist because of inappropriate respiratory management, sympathetic stimulation may not ensue, and the elevated blood pressure is maintained until full recovery of spontaneous breathing [20].

Cardiac functions

Although the parasympathetic dominant phase immediately after electrical stimulation suppresses cardiac function, this phase is completed within a minute and the effect on cardiac risk is limited [7]. Only when the parasympathetic discharge is not counteracted by a sympathetic surge due to subconvulsive stimulation does severe bradycardia or asystole persist. In contrast, tachycardia and hypertension provoked by the following sympathetic stimulation increase myocardial oxygen demand profoundly for several minutes and possibly induce myocardial ischemia in patients with compromised coronary circulation [24]. Rate pressure product, which is the product of heart rate and systolic blood pressure and considered an indictor of myocardial oxygen demand, is increased by 50%–400% during the sympathetic dominant phase [19,25]. Electrocardiographical ischemia is also observed in some cases [26,27]. An echocardiographical study [26] demonstrated regional wall motion abnormalities immediately after ECT. Also, systolic performance detected by fractional area change is suppressed by tachycardia, increased systemic vascular resistance, and hypertension [28,29].

Reported complications

Because ECT provokes drastic changes in hemodynamic variables, as noted above, several mortal complications have been reported in previous literature. Cardiac arrest probably induced by parasympathetic hyperactivity and use of a beta-blocking agent was reported by Decina et al. [15]. Myocardial infarction and

cardiac rupture were independently reported by Lopez-Gomez et al. [30] and Ali and Tidmarsh [31]. Although the incidence of such severe complications is rare [32], anesthesiologists who bear responsibility for hemodynamic management during ECT should pay special attention to cardiac risk factors of ECT patients in the preanesthesia physical assessment. Also, patients and/or their responsible relatives should be notified of the unique hemodynamic effects and risks of ECT.

Recommended medication protocols (anticholinergic and antihypertensive agents)

Hemodynamic changes during ECT are drastic, but mostly predictable. To reduce the risks of ECT, several medications have been examined to ameliorate the hemodynamic alterations. Anticholinergic agents are used to prevent bradycardia immediately after electrical stimulation. Intravenous atropine (0.4–0.8 mg) is effective in preventing bradycardia [7,33]. Previous studies demonstrated that intravenous administration of atropine several minutes before electrical stimulation is more reliable than intramuscular injection [34]. Glycopyrrolate (0.2–0.4 mg) is also effective in preventing bradycardia immediately after electrical stimulation [35]. When glycopyrrolate is used as the anticholinergic agent during ECT, tachycardia following seizure is less common than when atropine is used.

The hyperdynamic state induced by sympathetic activation can be ameliorated by ganglion blocker [27], beta blocker [36,37], alpha-/beta blocker [38,39], alpha-2-agonist [40], Ca^{2+}-channel antagonist [41,42], or nitrate [43,44] (Table 1). Recent guidelines recommend use of the ultrashort-acting beta blocker esmolol and the alpha-/beta-blocker labetalol as antihypertensive medications for patients with cardiovascular complications [9,35]. Ca^{2+}-channel antagonists, such as diltiazem [41], or nicardipine [42], are also effective for hemodynamic management. Nicardipine provokes temporary tachycardia because of its vasodilative action. Glyceryl trinitrate [43,44] and sodium nitroprusside [45] may be especially appropriate for use when a patient has ischemic heart disease.

Some anesthetics can ameliorate the hemodynamic changes during ECT (Table 2). Hemodynamic alteration under propofol anesthesia is more stable than that under barbiturate anesthesia [46–48]. Additional use of sevoflurane has been proposed to blunt the hemodynamic activation during ECT [49], although it requires a relatively long induction time and induces relatively short seizure.

Previous reports noted that several antihypertensive regimens, including use of esmolol or labetalol, induce a relatively short seizure [50] (see Table 1). Use of propofol [51] and supplemental use of lidocaine [36] or sevoflurane [49] also decrease seizure duration. However, recent psychiatric studies demonstrated that seizure duration is not necessarily related to therapeutic efficacy [52]; only abortive or extremely short seizures (of less than 15 s) are problematic [7]. Anesthesiologists should weigh the risks and benefits of antihypertensive regimens in their ECT management, especially for patients with cardiac or vascular complications.

Cerebral circulation

Electrical stimulation to the brain provokes unique alterations in cerebrovascular dynamics. In the 1970s,

Table 1. Effects of hemodynamic modulators when used during electroconvulsive therapy (ECT)

	Cerebral blood flow	Seizure duration
Nicardipine	↑*	→
Verapamil	N.E.	→
Diltiazem	N.E.	↓
Trinitroglyceline	↑ →	→
Alprenolol	↑→	→
Prostaglandin E_1	↑*	→
Labetalol	N.E.	↓ →
Landiolol	↑*	→
Esmolol	↑*	↓ →

N.E., not evaluated
This table summarizes descriptions in references 7, 35, 58, and unpublished data from the author's institute
* elevation as non-medication

Table 2. Effects of anesthetics when used during ECT

	Heart rate	Blood pressure	Cerebral blood flow	Seizure duration	Others
Methohexital	→/↑	↓/↑ ↑	N.E.	→	Standard anesthetic for ECT
Thiopental	↑/↑	↓/↑ ↑	↓/↑ ↑	↓	Histamine release
Propofol	↓/↑ →	↓/↑	↓/↑	↓	Injection pain
Diazepam	→/↑	↓/↑	N.E.	↓ ↓	Long acting
Ketamine	↑/↑	↑/↑ ↑	↑/↑ ↑	↑ ↓	Psychotic action
Etomidate	→/↑	→/↑ ↑	N.E.	↑	Injection pain, slow recovery
Sevoflurane	↑/↑	↓/↑	↑/↑ ↑	↓ ↓	Slow induction

Effects of anesthetics before and after electrical stimulation (before electrical stimulation/after electrical stimulation)
This table summarizes descriptions in references 7, 35, 59, and unpublished data from the author's institute

Brodersen et al. [53] first reported cerebral hyperemia following the electrically induced seizure using xenon-133. In the 1990s, we [54,55] and Vollmer-Hasse et al. [56] continuously measured intracranial blood flow during ECT, using real-time monitoring systems such as transcranial Doppler ultrasonography and near-infrared spectrophotometry. In these studies, it was demonstrated that cerebral blood flow decreased for a few seconds immediately after electrical stimulation and then increased drastically for a few minutes (see Fig. 1B). Because these responses are observed even after abortive stimulation, electrical current itself (rather than seizure activity) may provoke the responses [54]. This augmented cerebral blood flow is possibly induced by the increased oxygen demand of brain tissues and the increased cerebral perfusion pressure secondary to the systemic hyperdynamic responses [55]. The degree of cerebral hyperemia has positive correlation with the degree of systemic blood pressure elevation [55]. Recently, Fabbri et al. [57] demonstrated that this phenomenon is not only observed in bilateral ECT but also in unilateral ECT.

Because cerebrovascular hemodynamic alteration during ECT is partly provoked by systemic hemodynamic changes, antihypertensive regimens that prevent systemic hypertension during ECT ameliorate cerebrovascular hemodynamic alteration to some extent [58]. However, antihypertensive medication that can prevent systemic hypertension cannot completely suppress the reactive acceleration in middle cerebral artery blood flow. Relative effects on systemic and cerebral circulation are not identical among antihypertensive drugs (see Table 1). For example, a beta-blocking agent, alprenolol, and glyceryl trinitrate suppress both systemic and cerebral hyperdynamics; in contrast, a calcium channel blocker, nicardipine, and prostaglandin E_1 suppress mainly systemic hemodynamics and have minimal effects on cerebral hemodynamics. Choice of anesthetics also influences cerebrovascular reaction during ECT. An increase in middle cerebral artery blood flow velocity is minor under propofol anesthesia compared to thiopental anesthesia [59]. Several authors [60–62] have speculated that the reactive hyperemia may be necessary to compensate for the increased oxygen and energy demand after the electrical stimulation. Therefore, complete suppression of cerebral hyperemia may be unnecessary or inadequate in hemodynamic management during ECT.

Cerebrovascular complications

Considering abrupt changes in cerebral circulation after electrical stimulation, the presence of cerebral aneurysm and intracranial mass lesion are listed as relative contraindications of ECT in some articles [9,63,64]. However, case reports describing unsuccessful management of ECT for the patients with cerebral complication are limited [65]. In contrast, safe completion of the therapy schedule with deliberate hemodynamic management has been reported [45,66–71]. Even though there are few previously reported catastrophic cases, anesthesiologists should take special effort to stabilize hemodynamics and cautiously inspect neurological symptoms when ECT is administered for such patients.

Respiratory management

Although respiratory care during ECT can be completed within 10 min, inappropriate management interferes with the efficacy of the therapy and increases the risk of complications [7,20,72]. Following anesthesia induction with an anesthetic agent and a muscle relaxant, the airway should be secured and ventilation should be supported manually. Hypocarbia induced by hyperventilation may be required in some patients to ensure appropriate seizure duration, because hypocarbia prolongs seizure duration [73,74]. Our previous study [20] demonstrated that end-tidal carbon dioxide measurement at a nostril is effective in maintaining the requested carbon dioxide level before the electrical stimulation. In some patients for whom mask ventilation is difficult because of oromandibular anatomic reasons, airway devices including laryngeal masks can be applicable for adequate ventilation [75]. Because regurgitating accidents are very rare, intubation is not necessary in most cases [76]. Some authors recommend intubation during ECT when this therapy is applied for depression during late pregnancy [77,78].

During electrical stimulation, even under the use of a muscle relaxant, facial muscles are electrically contracted. Dental and lingual protection should be considered and administered, either by use of a specially designed mouthpiece or by fixing the mandibular joint at the maximally closed position [7].

After the completion of electrical stimulation, oxygen consumption and carbon dioxide production are elevated by the seizure activity [62] (Fig. 2). Fasciculation that is induced by succinylcholine also contributes to the elevated oxygen demand and carbon dioxide production [20]. To overcome the elevated oxygen demand and carbon dioxide production, anesthesiologists should increase ventilation volume adequately. Hypoxia and/or hypercarbia induced by inadequate ventilation after the electrical stimulation aggravate hypertension and tachycardia after seizure [20]. Incidence and intensity of postictal excitement and headache may also be pronounced by long-lasting hypercarbia [75].

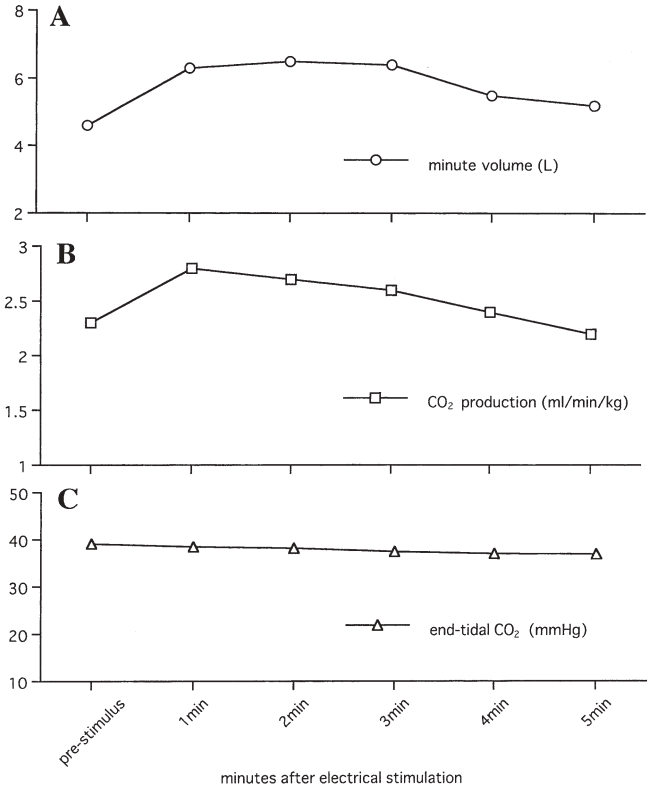

Fig. 2. Typical respiratory management during electroconvulsive therapy (ECT) in a patient. **A** Minute volume trend when the anesthetist tried to maintain the end-tidal carbon dioxide partial pressure at 35–40 mmHg. **B** Typical carbon dioxide production during ECT. **C** End-tidal carbon dioxide partial pressure trend when airway gas was sampled from laryngeal mask airway and the anesthetist tried to maintain the end-tidal carbon dioxide partial pressure at 35–40 mmHg

Anesthetics and muscle relaxants

Anesthetics

Selection of anesthetics and determination of dose of the selected anesthetics are crucial in ECT management, because these factors have a potent impact on seizure induction in ECT (see Table 2). Currently, methohexital is the most recommended anesthetic for ECT [7,35]; however, thiopental or thiamylal can be an alternative barbiturate although these drugs provoke relatively short seizure and more frequent arrythima when compared to methohexital [79]. Propofol is also used in many reports [48,80,81], because systemic and cerebrovascular hemodynamic changes under propofol anesthesia are more stable than under barbiturate anesthesia. Although seizure duration under propofol anesthesia is shorter than that under barbiturate anesthesia, the pshychiatric efficacy of ECT under either anesthetic is reportedly comparable [82,83]. Etomidate [84] and ketamine [85] are applicable for ECT and ensure longer seizure duration when compared to both barbiturates and propofol. However, hemodynamic changes are more pronounced, and recovery after therapy is delayed when these anesthetics are used. Intracranial pressure elevation may be another concern when ketamine is used in ECT [86]. Use of sevoflurane has been examined and demonstrated as effective to stabilize hemodynamics during ECT [49,87,88]; however, its time-consuming induction, relatively short seizure, and the requirement of an anesthesia machine make this technique impractical in most cases.

Regardless of the type of anesthetic, the anesthetic dose is important in inducing adequate seizure in ECT. For most anesthetics, seizure duration becomes shorter with an increase in anesthetic dose [89]. Some articles have described hypnosis as satisfactory for ECT [47,90], because electrical stimulation itself has potent neurosuppressive action and induces retrograde amnesia in the postictal phase. The minimum anesthesia depth required may be unconsciousness at the time of muscle relaxation and before electrical stimulation, because muscle relaxation under an extremely shallow anesthetic state is considered ethically problematic.

Recently, bispectral index (BIS) is widely used to monitor the anesthetic depth [91,92]. Several reports have described the value as unreliable under special conditions including after electrically induced seizure [93–96]. Some patients are completely awake even at a very low BIS value after ECT. However, consciousness before electrical stimulation can be monitored by this system. We [95] and White et al. [96] demonstrated that the index can be used to titrate the dose of propofol or methohexital in ECT. By referring to the BIS value immediately before electrical stimulation, operators might be able to control seizure duration to some extent. However, this approach is applicable on a condition that propofol or methohexital is used consistently throughout ECT sessions. Lemmens et al. [97] reported that when different types of anesthetics are used, comparison across anesthetics can not be attained by this system.

Muscle relaxant

Succinylcholine at 0.5–1 mg/kg has been commonly used in ECT because of its short action [7]. Although several ultrashort-acting nondepolarizing muscle relaxants have been developed, none of these are shorter acting than succinylcholine [98]. Also, the potent muscle contraction impulse during electrically induced seizure may not be satisfactorily inhibited by nondepolarizing agents [99]. Muscle relaxation can be temporarily reversed by electrical stimulation (Fig. 3). Therefore, even now, use of a nondepolarizing muscle relaxant is limited in cases suspected of having malignant hyperthermia (MH)

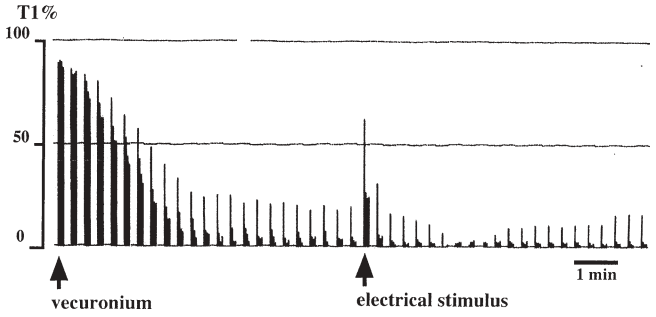

Fig. 3. Electrical reverse of the effect of a nondepolarizing muscle relaxant. T1 and T4 ratio after administration of 0.08 mg/kg vecuronium bromide in a patient treated by ECT

[100]. Patients with a history of neuroleptic malignant syndrome (NMS) are safely managed by succylcholine in many case reports, and recent guidelines have reported that succinylcholine can be safely used in ECT [101]. However, when hyperthermic episodes cannot be conclusively diagnosed as NMS, avoiding the use of succinylcholine is recommended. In patients with either NMS or MH, body temperature should be carefully monitored, and therapeutic drugs and cooling strategies should be ready to use throughout the therapy schedule.

Conclusion

ECT is a unique therapy that intentionally provokes seizure by applying electrical current to the human central nervous system. In general, seizure is believed to be detrimental to the nervous system, and it is recommended that it be suppressed as early as possible. This general concept may be the main reason for public resistance to this therapy. However, the clinical effectiveness of this therapy for several types of psychiatric disorders overcomes the opposition to it and promotes its clinical use worldwide. Safe physical management by anesthesiologists greatly contributes to the establishment of ECT under muscle relaxation. To maintain social confidence and to refine the therapy, anesthesiologists should play an essential role both in clinical activities and laboratory research.

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