—Review—

Anesthesia Management of Special Patient Populations Undergoing Electroconvulsive Therapy: A Review

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Electroconvulsive therapy (ECT) is the safe application of electricity to the scalp of a patient, using brief-pulse stimulation techniques under general anesthesia and muscle paralysis, inducing a series of generalized epileptic seizures. Principal indications for ECT are major depression (unipolar or bipolar) with a lack of response to medications, intolerance to medications due to side effects or coexisting conditions, the need for a rapid response because of other conditions such as catatonia, psychosis, suicidality, or clinically significant dehydration or malnutrition, mania, and schizophreniform disorder or schizoaffective disorder, and, medical disorders such as Parkinson's disease, neuroleptic malignant syndrome, and chronic pain. Anesthesia management of special patient populations undergoing ECT has been described in textbooks and guidelines, but some descriptions may be antiquated. Therefore, this review describes recent knowledge on anesthesia management of patients who require ECT, such as those with neurologic disorders, cardiovascular disorders, pregnancy, and other concurrent medical illness. Based on the findings of a recent paper, ECT may be safer than is widely reported. According to the American Psychiatric Association, ECT has no absolute contraindications; however, some conditions pose a relatively high risk, and there are many other kinds of complications associated with ECT that can lead to death. Understanding such complications and their management strategies can avoid unnecessary discontinuation of treatment due to manageable complications of ECT and, furthermore, ECT clinicians must also consider the risk-benefit ratio when treating high-risk patients.

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Introduction

Electroconvulsive therapy (ECT) is the safe induction of a series of generalized epileptic seizures in a patient for therapeutic purposes, using brief-pulse stimulation techniques under general anesthesia and muscle paralysis[1–7]. Since the earliest publications on the subject, the excellent therapeutic effectiveness of this method in the treatment of depression and other psychiatric disorders has been described in a variety of reviews and meta-analyses[5], and medical disorders such as Parkinson's disease[8,9], neuroleptic malignant syndrome[8], and chronic pain[10,11] can also be indications for ECT.

Although anesthesia management of special patient populations undergoing ECT has been described in textbooks and guidelines[12–15], some descriptions may be antiquated and, therefore, in this review I describe the recent knowledge on anesthesia management of such populations undergoing ECT by reviewing papers published after 2007 in English and Japanese that were retrieved through a bibliographic search of PubMed.

Neurologic Disorders

Neuroleptic Malignant Syndrome (NMS)

NMS is a relatively rare, but potentially fatal complication of the use of neuroleptic or antipsychotic drugs[16]. NMS typically consists of muscle rigidity, fever, autonomic instability, and cognitive changes such as delirium, and is associated with elevated plasma creatine phos-

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phokinase. Supportive therapy and pharmacological treatments for NMS, such as benzodiazepines, dopaminergic agents, and dantrolene are usually chosen, but ECT can also be effective[17,18]. The use of succinylcholine may have potentially deteriorative effects on the cardiovascular system and plasma potassium levels and cause adverse effects in patients with a known history of susceptibility to malignant hyperthermia. Thus, in patients with a history of NMS, the recommended alternative to the customary administration of succinylcholine is a non-depolarizing relaxant such as rocuronium[18,19]. The concomitant use of sugammadex, a reversal agent for rocuronium, is another alternative that may solve the problem of long-lasting effects caused by the other non-depolarizing relaxants that are substituted for succinylcholine in ECT for NMS without the adverse effects of the acetylcholinesterase inhibitors[18,19].

Malignant Catatonia, Catatonic Schizophrenia

Catatonic schizophrenia is characterized by at least two of the following: catalepsy or stupor, excessive motor activity; extreme negativism or mutism; peculiarities of voluntary movement as evidenced by posturing, stereotypical movements, prominent mannerisms, or prominent grimacing; and echolalia or echopraxia (American Psychiatric Association, 1994)[20]. Malignant catatonia, a particularly severe form, is the acute onset of excitement, fever, increasing autonomic instability (tachycardia, hypertension, fluctuating blood glucose levels, and hyperthermia), and delirium, and may be fatal[21,22]. ECT is widely used and shown to be effective and safe in patients with catatonic schizophrenia (American Psychiatric Association, 2001)[20]. Although the standard protocol can be applied in patients with catatonic schizophrenia[20], special anesthesia care is required in patients with malignant catatonia in accordance with symptoms of their condition (increasing autonomic instability) such as tachycardia and hypertension.

Presence of Cranial Metallic Objects

Gahr et al.[23] reviewed the safety of ECT in the presence of cranial metallic objects (cMO) such as medical devices or metallic foreign bodies (cerebral clipping systems, cerebral coils, deep brain stimulators, osteosynthesis materials or other metallic medical devices, and foreign bodies). The presence of cMO raises three theoretical concerns regarding the safety of ECT: (1) cMO may significantly alter the ECT-induced electrical field distribution in the brain regarding field strength and focality, (2) vascular complications at the location of the cMO due to the ECT-induced hyperdynamic state may occur, and (3) a prolonged seizure or status epilepticus may develop during ECT as a consequence of device-induced symptomatic epilepsy. However, Gahr et al.[23] found that no ECT-related complications related to the proposed theoretical concerns were reported, and concluded as follows: the absence of cMO-related complications during ECT in the reported cases implies that cMO might not represent an absolute contraindication for the performance of ECT. However, the indication for ECT should be carefully considered in patients with cMO. Further research is considered necessary for the adequate assessment of safety.

Dopa-Responsive Dystonia (Segawa Syndrome)

Segawa syndrome is an autosomal dominant form of guanosine triphosphate cyclohydrolase deficiency that results in decreased dopamine and serotonin levels and typically presents as a dopa-responsive dystonia, with marked diurnal fluctuation, and an onset in childhood or early adulthood[9]. Publications to guide the clinician in the treatment of psychiatric conditions in patients with this syndrome are lacking[9]. Sienaert et al.[9] reported a patient with dopa-responsive dystonia who presented with a delusional depression and anxiety. They concluded that ECT can be administered safely and effectively in a patient with dopa-responsive dystonia[9], and in any case, the possible benefits of ECT should be weighed against its possible risks.

Lycanthropy and Cotard Syndrome

Lycanthropy comes from the Greek *lykănthropos*, *lŷkos* (wolf) + *ănthrŏpos* (human), and describes the mythical condition of lycanthropy, a supernatural affliction in which people are said to physically transform into wolves[24]. In the psychiatric literature, lycanthropy is understood as an unusual belief or delusion that one has been transformed into an animal or that his/her behaviors or feelings are suggestive of such a belief, and besides being metamorphosed into a wolf, patients with this phenomenon have reported delusional transformation into a leopard, lion, elephant, crocodile, shark, buffalo, eagle, serpent, frog, bee, dog, gerbil, rabbit, horse, tiger, cat, bird, and other unspecified animal species[24]. The phenomenon of lycanthropy has been reported in patients with schizophrenia, psychotic depression, bipolar disorders, use of cannabis and alcohol, personality disorders, and also in organic conditions[24].

Cotard syndrome is a rare condition characterized by nihilistic delusions concerning body or life that can be found in several neuropsychiatric conditions. It is typically associated with depressive symptoms[25]. Grover et al.[24] effectively treated a patient presenting with lycan-

thropy and Cotard syndrome with the use of bilateral modified ECT.

Cardiovascular disorders

Recent Myocardial Infarction (MI) and/or Reduced Cardiac Output

There are limited reports on the use of ECT in patients with recent MI and in those with reduced cardiac output[26]. Use of ECT is associated with an increase in cardiac output, rise in blood pressure, and increase in heart rate lasting for a few minutes[1–3,6,7,26]. Accordingly, a thorough cardiac evaluation is considered to be mandatory prior to the use of ECT[7,26]. Generally, the guidelines given by the American College of Cardiology/American Heart Association for non-cardiac procedures requiring general anesthesia are also followed for ECT[27,28], and these guidelines suggest that acute MI within one week or severe MI within the last four weeks is associated with an increase in perioperative morbidity. Therefore, it is recommended to wait for 4-6 weeks after MI to perform elective procedures.

In some case reports, authors reported the successful use of ECT after 10 days, 30 days, and 5 weeks after the MI[26]. Grover et al.[26] also commented that in terms of the use of ECT in patients with reduced cardiac output, again the data are sparse, and only one study has presented the data on such patients[29]. According to this study, ECT was used in 35 patients, aged 54-92 years with low cardiac output and median left ventricular (LV) ejection fractions ranging from 15 to 40% but without cardiac complications such as decompensated heart failure, myocardial ischemia, or MI, either during the ECT procedure or 24 hours thereafter. However, non-life-threatening cardiac arrhythmias were seen in 3 patients[29]. Those patients who developed marked hypertension during the ECT sessions were prophylactically given beta-blockers prior to the next ECT sessions[29].

Grover et al.[26] concluded that the experience with their patient with severe depression suggests that ECT can be safely given in patients with recent MI and/or reduced cardiac output with proper monitoring, and their case adds to the limited literature on the safe use of ECT in such patients. However, this author believes that ECT clinicians must consider the risk-benefit ratio when treating high-risk and medically complicated patients[7].

Unrepaired Abdominal Aortic Aneurysm (AAA)

Theoretically, ECT increases the risk of rupture of an unrepaired abdominal aortic aneurysm (AAA)[30,31]. The American College of Cardiology, the American Heart Association, and most screening studies consider AAA to be present when the minimum anteroposterior diameter of the abdominal aorta is 3.0 cm or more[30]. Currently, there are no guidelines for the clinical management of patients with an unrepaired AAA who require ECT, although some authors suggest that ECT may be performed safely in this population[31,32].

Mueller et al.[32] conducted a retrospective review of the medical records of all patients with unrepaired AAA who underwent ECT for severe depressive syndrome at their institution. They found that none of the patients died during the periprocedural period or experienced symptoms or signs suggestive of AAA expansion or rupture, and during the ECT procedure, the anesthesiologist administered intravenous medications as appropriate (e.g., esmolol hydrochloride or labetalol hydrochloride for severe hypertension and tachycardia). After ECT, patients at their institution are discharged from the post-anesthesia care unit when their vital signs are stable, spontaneous respirations via a patent airway are maintained without assistance, and the level of consciousness is adequate for returning to their hospital room or being discharged to home with an adult in attendance[32]. Nevertheless, they commented that one should not conclude from their findings and prior reports that all patients with unrepaired AAA can safely undergo ECT and, indeed, men with AAAs measuring ≥ 5.5 cm in diameter, women with AAAs measuring ≥ 5.0 cm, and patients with AAA expansion of ≥ 1.0 cm per year should be evaluated by a vascular specialist for surgical or endovascular AAA repair before undergoing ECT, because these patients are at increased risk for AAA rupture independent of the hemodynamic changes associated with ECT[32]. They also noted that several reports have been published of patients with repaired AAA who safely underwent ECT[32]. Furthermore, they recommended that patients' hemodynamic responses to prior procedures should be considered to determine ideal periprocedural management (e.g., the need for periprocedural β-adrenergic blockers for severe tachycardia and hypertension)[32]. Abrams[13] recommends that succinylcholine doses in such patients with AAA be adequate to provide full relaxation of the abdominal musculature to prevent mechanical stress to the aneurysm. Although Muller et al. ultimately concluded that ECT could be safely administered to patients with an unrepaired AAA[32], this author believes that ECT clinicians need to consider the risk-benefit ratio when treating such high-risk and medically complicated patients[7]. Use of the Nexfin HD monitor

(BMEYE, Amsterdam, The Netherlands)-now available as the ClearSight system (Edwards Lifesciences Corp, Irvine, CA, USA)-for hemodynamic management during ECT for the treatment of patients with an unrepaired AAA may also be considered[31].

Idiopathic Pulmonary Arterial Hypertension (IPAH)

IPAH is a progressive and fatal cardiovascular disease if left untreated[22]. In patients with IPAH with psychiatric illness or other complications, careful attention is required when administering medical therapies that may affect their hemodynamics[22]. Patients suffering from IPAH who undergo anesthesia and surgery experience high rates of mortality and morbidity, and pulmonary hypertensive crisis (PHC) is the most serious complication that can be induced by the sympathetic stimulation of ECT[22]. Hobo et al.[22] reported malignant catatonia in a patient with IPAH who eventually required ECT that could have affected the patient's hemodynamics. To avoid PHC, they administered oxygen using a laryngeal mask and administered remifentanil for anesthesia (chosen because it is a short-acting opioid with strong antinociceptive features and was expected to stabilize blood pressure), and they performed endotracheal intubation. Furthermore, standby nitric oxide inhalation was prepared in case of PHC secondary to dilation of the pulmonary arteries. During each ECT procedure, epoprostenol (PGI_2) was continuously infused to prevent PHC.

Brugada Syndrome

Brugada syndrome is an inherited cardiac disorder initially described in 1992 by Pedro and Josep Brugada. It is traditionally identified by electrocardiogram (ECG) and is defined by right bundle branch block and ST-segment elevation in the right precordial leads (V_1-V_3), without evidence of any underlying structural heart disease[33]. Other symptoms can include: ventricular arrhythmias, syncope, and sudden death[33]. The genetic abnormalities that cause Brugada syndrome have been linked to mutations in the ion channel genes that encode the α-subunit of the cardiac sodium channel[33].

General anesthetic management with Brugada syndrome is limited. Careful preoperative evaluation and anesthetic management are important to avoid inducing malignant arrhythmias[33]. Sodium channel blockers such as procainamide and flecainide are contraindicated in patients with a Brugada ECG pattern[33]. Neostigmine and α-agonists augment ST elevation without coronary spasm. Therefore, class IA anti-arrhythmic drugs should be avoided, and special caution should be exercised when neostigmine and α-agonists are used[33]. In patients with Brugada syndrome, vagotonic agents have been shown to accentuate ST-segment elevation and may trigger ventricular fibrillation[34]. Therefore, succinylcholine also should be avoided, and a non-depolarizing neuromuscular blocking agent, having characteristics of cardiovascular stability, can be used as a substitute in these patients[34]. Reversal of residual non-depolarizing neuromuscular block is necessary after the short ECT procedure; however, the muscarinic effects of anticholinesterases such as neostigmine are also contraindicated[34]. Konishi et al.[34] reported that the combination of rocuronium and sugammadex was effectively and safely used to induce and antagonize neuromuscular block in a patient who underwent consecutive ECT sessions.

Prolonged QTc Intervals

The QT interval on the surface ECG represents the time of onset of ventricular depolarization to completion of repolarization[35]. This interval is usually reported as the QTc, which is the QT interval corrected for heart rate[35]. The QTc value printed on ECG reports actually represents the mean QTc of all ECG leads because the values vary slightly from lead to lead[35]. The degree of variance of the QTc intervals among the 12 leads is termed QTc dispersion[35]. Prolonged QTc intervals can be caused by intrinsic cardiac pathology or as an effect of medications and are associated with increased risks of ventricular arrhythmias that could trigger ventricular fibrillation and sudden cardiac death[35]. One previous case study has suggested that high-pretreatment QTc dispersion can predict arrhythmias during ECT treatments[36]. Another study showed that ECT treatment itself was directly associated with a temporary prolongation of QTc dispersion immediately after the treatments[37]. However, with the exception of one case report[38], there are no data addressing whether prolonged QTc intervals are associated with increased morbidity or mortality in ECT practice. Pullen et al.[35] investigated whether pretreatment prolongation of the QTc interval was associated with increased cardiac-related events in the ECT treatment of psychiatric disorders. They found that the presence of baseline QTc prolongation was not independent of an increased risk of cardiac-related events during ECT, and they concluded that careful consideration should be given to patients with QTc prolongation on their ECG who are undergoing ECT treatment, but this should not definitively exclude them from receiving ECT treatment.

Aortic Stenosis

Aortic stenosis is a risk factor for increased morbidity and mortality in patients undergoing procedures requir-

ing general anesthesia and, although the absolute risk of ECT to patients with aortic stenosis receiving ECT is unclear, given the physiologic changes occurring in aortic stenosis, it is reasonable to postulate that such patients are at an increased risk from adverse events[39]. Severe aortic stenosis is associated with several physiologic and hemodynamic changes[39]. In response to the chronically elevated LV pressures necessary to maintain adequate cardiac output, patients typically develop increased LV pressure and hypertrophy with a commensurate increase in myocardial oxygen consumption, particularly when the heart rate or blood pressure increases[39], which can be commonly induced by the ECT procedure. A severe pressure gradient across the aortic valve exists, placing patients at risk of hypotension when LV pressure falls and when arrhythmias occur[39]. Patients with severe aortic stenosis (defined as an aortic valve area ≤ 1.0 cm^2, mean aortic transvalvular pressure > 40 mmHg, or an aortic valve velocity > 4.0 m/s[40]) undergoing ECT could be expected to be at risk for severely decreased cardiac output in the event of hypotension, bradycardia, or dysrhythmia, or secondary to anesthetic agents that decrease inotropy[39]. In addition, prolonged periods of tachycardia and elevated blood pressure, which the ECT procedure commonly induces, could result in ischemia or infarction because of the increased oxygen demands placed upon the left ventricle[39]. In a patient with severe aortic stenosis undergoing ECT reported by Sutor et al.,[39] pre-treatment with phenylephrine and glycopyrrolate or atropine may have prevented hypotension and bradycardia but might have put the patient at an increased risk of infarction because of the potential for increased oxygen demand resulting from the increased heart rate and inotropy. Atropine, given its more rapid onset of action, is preferable to glycopyrrolate in managing bradycardia acutely and could have been used in such patients instead when vagolysis was needed rapidly[39]. Treatment considerations also include surgical correction or valvuloplasty before ECT[39]. Furthermore, ECT treatment teams must be prepared to quickly assess and manage life-threatening cardiovascular complications when they arise[39].

Heart Failure and Decreased Left Ventricular Systolic Heart Function

Because little is known about the safety of ECT in patients with a history of heart failure and decreased LV systolic heart function, Rivera et al.[29] conducted a retrospective review of the medical records of 35 patients at their institution with such a history and assessed the safety of ECT in these patients. The median age was 77 years, the median LV ejection fraction was 30%, and the median number of sessions per patient was 10 (range, 1-44). Before undergoing ECT, none of the patients had symptoms of heart failure and all of the patients tolerated ECT well, none died or experienced decompensated heart failure, myocardial ischemia, or MI during or within 24 hours after an ECT session[29]. Prophylactic intravenous β-blockers were given to patients who had experienced marked hypertension or had a heart rate ≥ 100 bpm during previous ECT sessions. Overall, this prophylaxis was used in 26 patients during 413 ECT sessions (80% of the total number of ECT sessions). Only three patients experienced temporary, non-life-threatening cardiac arrhythmias.

Rivera et al.[29] offered some suggestions for planning the care of patients with a history of heart failure and reduced LV systolic heart function and who are undergoing ECT. A dedicated and complete interview and clinical evaluation should be documented before any patient is considered for this type of therapy, and the following tests should be performed to follow adequate periprocedural guidelines, especially if cardiac symptoms arise: basic blood work to assess liver and renal function, red blood cells, and electrolytes, and non-invasive cardiac studies (12-lead ECG and echocardiogram). The anesthesiology team should also be involved in advance to design adequate cardiovascular management of the patient's cardiac condition[29], and a preoperative medical evaluation that follows standard of care recommendations is advised[41].

However, Rivera et al.[29] commented that ECT may not be safe in some patients with a history of heart failure and reduced systolic function and, finally, their findings cannot be generalized to patients with heart failure due to diastolic dysfunction, which is a pathophysiological condition different from that of heart failure due to systolic dysfunction.

Peripartum Cardiomyopathy

The incidence of psychotic illness in the puerperal period is low[42]. Peripartum cardiomyopathy is a rare life-threatening cardiomyopathy of unknown cause that occurs in the peripartum period in previously healthy women[43]. Diagnosis is confined to a narrow period and requires echocardiographic evidence of LV systolic dysfunction[43]. Symptomatic patients should receive standard therapy for heart failure, managed by a multidisciplinary team[43]. If subsequent pregnancies occur, they should be managed in collaboration with a high-risk perinatal center[43]. Many of the antipsychotics are contraindicated dur-

ing pregnancy, and ECT cannot be administered due to the added risks involved with regard to anesthesia[42].

ECT use During Pregnancy

Pregnancy is a special situation in which pharmacotherapy during the first trimester risks major congenital malformations and pharmacotherapy during the last trimester risks drug toxicity or withdrawal effects. In addition, the use of some psychotropic drugs during pregnancy has been associated with fetal neurodevelopmental effects in some studies[44]. ECT has been suggested as an alternative to pharmacotherapy for pregnant women with major mental illness; the advantage is that the fetus is minimally exposed to transplacental transfer of chemical substances[44]. The subject of the safety of ECT during pregnancy has been reviewed in different manners by different teams of authors, resulting in differing results and conclusions. Therefore, Sinha et al.[44] recently compared these reviews with regard to search strategy, study selection criteria, total number of studies identified, total number of patients included, findings related to safety and adverse events, and interpretation of results. They found four systematic reviews that examined the safety of ECT in pregnancy[45–48] and concluded that the recommendations by Anderson et al.[46] and Pompili et al.[47] seemed to be the most balanced, and readers would do well to consider their conclusions.

Anderson et al.[46] concluded that ECT did seem to be effective in treating major mental illness during pregnancy, and the risks of adverse events were low. Accordingly, they suggested that ECT should be strongly considered in pregnant women with severe symptoms of mental illness, such as psychotic symptoms, catatonia, or strong suicidal urges.

Pompili et al.[47] found that severe psychiatric disorders are relatively common during pregnancy, with some studies reporting a morbidity rate of 15-29% among all pregnant women[49]. Pharmacological treatment of severe psychiatric disorders during pregnancy is complicated by the potential harmful effects to the fetus (e.g., teratogenicity, toxicity, and withdrawal syndromes).[47] Many authors have indicated the efficacy and safety of ECT during all trimesters of pregnancy.[47] Pompili et al.[47] found the following results: depressed pregnant women treated with ECT had a full or partial response to treatment in 84% of cases, and the rate of response in schizophrenic pregnant women treated with ECT was 61%. The most common adverse effects in mothers were confusion, memory loss, muscle soreness, headache, hypertension, vaginal bleeding, placental abruption, uterine contractions, and the induction of premature labor. The most common adverse effects in the fetus were transient decrease in fetal heart rate, multiple cortical infarcts, ascites, transposition of the great vessels, stillbirth, and neonatal death. Pompili et al.[47] concluded that the indication for ECT in pregnant women is to replace drug therapy in those who cannot continue pharmacological treatment for their disorder (major depression, bipolar disorder, suicidal crises) or to prevent the side effects of psychiatric drugs in the mother and teratogenicity and toxicity in the fetus, and the efficacy and safety of ECT appeared to be good when compared with pharmacotherapy.

Concurrent Medical Illness (Other Disorders)

Chronic Obstructive Pulmonary Disease (COPD)

Recent guidelines on the care of medically ill patients with ECT recommend administration of the patients' prescribed inhalers on the morning of the ECT treatments[50] and caution is recommended in the use of ECT in patients taking theophylline, which has been associated with prolonged seizures and status epilepticus in ECT treatment[51]. However, Schak et al.[51] reviewed the case files of patients with COPD receiving ECT at their facility because little has been written about the safety of ECT in such patients. These patients might be at a heightened risk of complications because the ECT procedure involves the administration of general anesthesia and the need for assisted ventilation[51].

Based on their experiences, Schak et al.[51] offered the following clinical recommendations. To provide guidance for further pre-anesthesia management, the medical history should be reviewed and also a physical examination should be performed before anesthetizing a patient with COPD. The patient should be questioned regarding the frequency of COPD exacerbations, the usual intensity of the treatment required for exacerbations (such as emergency room visits, hospitalization, or intensive care unit treatment), current medication regimen (type, dose, and frequency), past and current use of corticosteroids (both oral and inhaled), smoking history, current symptoms, and recent history of upper respiratory tract infections. If, on the basis of these inquiries, the physician feels the patient's COPD is not optimally managed, then ECT should be postponed until such a result is achieved.

Pulmonary function testing, particularly measures of obstruction (e.g., peak expiratory flow and forced expiratory volume in 1 sec.) are useful in the general management of COPD but are not specifically indicated before

ECT, and a chest X-ray should be performed to screen for infections. Concomitant psychotropic medications should be minimized[51]. Schak et al.[51] empirically suggested that it seems reasonable to administer inhalers before ECT treatments prophylactically, but there is no evidence in the literature to support this practice. In addition, patients who are wheezing should receive inhaled β_2-agonists and parenteral corticosteroids and, furthermore, unless the psychiatric indication for ECT is life-threatening, significant exacerbations of COPD should be treated first before administering ECT[51].

When Succinylcholine is Contraindicated or Not Recommended

Although succinylcholine has been used during ECT for many years because of its rapid onset and short duration of action to prevent injury during seizures,[52] succinylcholine is contraindicated in patients with pseudocholinesterase deficiency[53], muscular dystrophy[53], Brugada syndrome[34], and succinylcholine allergy[52]. González et al.[53] asserted that mivacurium is a suitable alternative to succinylcholine, based on its relatively short duration of action, because mivacurium is metabolized by plasma pseudocholinesterase; however, its use is not recommended in patients with pseudocholinesterase deficiency. They also noted that although the use of rocuronium and sugammadex, which is an expensive drug whose high cost would limit its repeated use during ECT procedures, is attractive, it is probably the best option for ECT when succinylcholine is contraindicated[53].

Systemic Lupus Erythematosus (SLE)/Neuropsychiatric SLE (NPSLE)

SLE is an autoimmune disorder that requires long-term management and can have a profound impact on patient quality of life[54]. Neuropsychiatric manifestations are fairly common, occurring in up to 75% of the patients with SLE[54]. However, NPSLE, in which patients with SLE present with psychosis, is less common, with a prevalence of up to 11%[54].

In 1999, the American College of Rheumatology (ACR) Neuropsychiatric Lupus Nomenclature Committee criteria consensus accepted five different psychiatric syndromes: cognitive dysfunction, delirium (acute confusional state), anxiety disorder, mood disorder, and psychosis[54]. The ACR defines psychosis in patients with neuropsychiatric symptoms of SLE (NPSLE) as the presence of four categorical criteria: the presence of delusions and/or hallucinations without insight, the presence of functional impairment as a consequence, the absence of delirium, and the absence of any secondary cause (e.g., a concomitant neurological disorder) or condition primarily associated with psychosis (such as schizophrenia)[54].

Tan et al.[54] described three patients who presented with psychosis during a lupus relapse, and were treated safely and successfully with ECT after their symptoms did not improve with medical treatment (antipsychotics and immunosuppressants). Hussain et al.[55] also described a patient with NPSLE presenting with catatonia that did not respond to benzodiazepines or immunosuppression but showed improvement with ECT. ECT may play an important role in the treatment of neuropsychiatric manifestations of SLE and should always be considered as a treatment option if the patient is resistant to medications[55].

Patients Taking Steroid Medication

Patients who take chronic steroid medication are often prescribed extra "stress doses" before procedures involving general anesthesia[56]. The rationale for this practice is that their chronic steroid use has suppressed the ability of the endogenous hypothalamic-pituitary-adrenal steroid stress-reactivity system to handle the systemic stress of surgery[56]. Rasmussen et al.[56] described their experience treating 27 patients with ECT taking prednisone to investigate whether the stress of treatments was sufficient enough to warrant this practice in ECT, which has not been broached in the literature. They concluded that the use of extra "stress doses" of steroid medication is unnecessary in ECT practice and recommended that patients receive their usual morning dose of steroid before ECT treatments[56].

Addison Disease

Addison disease, or primary adrenocortical insufficiency, is a rare disorder with a mean incidence of approximately 6 new cases per million per year[57,58]. Major depression is a frequent occurrence in patients with Addison disease[57,58]. Although glucocorticoid replacement may alleviate major depression in these patients, some have persistent major depression despite adequate replacement therapy[58,59]. Major depression is associated with both hyper- and hypocortisolemia, with hypocortisolemia being more prevalent in patients with chronic major depression[58]. Whether the efficacy of antidepressants in patients with depression with Addison disease is similar to that in physically healthy patients with depression is unknown[58]. Nevertheless, Heijnen et al.[58] described a patient who had one previous episode of major depression, which responded favorably to treatment with tricyclic antidepressants, but after the development of Addison disease, the patient experienced a new episode of

severe major depression that failed to respond to adequate treatment with imipramine and was subsequently successfully treated with ECT.

Heijnen et al.[58] commented as follows: There are several possible pathways to an increased risk of depressive disorders in patients with Addison disease. Psychological and mood impairment has been documented in patients with primary adrenocortical insufficiency. This could result in an increased risk of depressive disorders. Another explanation is that patients with Addison disease have continuous dysregulation of the hypothalamic-pituitary-adrenal axis that might not be reversed by glucocorticoid hormone substitution. Glucocorticoid replacement therapy may improve depressive symptoms in Addison disease, but some patients have persistent depressive symptoms despite adequate replacement therapy. In addition, it is not always possible to give an optimal dose of hydrocortisone, for instance, because of adverse effects.

Adrenal crisis in patients with Addison disease is a life-threatening event that might occur due to the physiologic stress of ECT. On the basis of Suzuki et al.'s[59] experience, Heijnen et al.[58] treated their patient with 100 mg hydrocortisone intravenously as steroid cover and prophylaxis against adrenal crisis without the patient experiencing any severe adverse effects or adrenal crisis and suggested that this regimen for prophylaxis is safe.

After Eye Surgery

ECT can increase intraocular pressure (IOP). Because data on ECT safety in patients following eye surgery are scarce and those on the influence of modern anesthetic drugs on IOP during ECT are not available, Sienaert et al.[60] described a patient treated with ECT for depression 13 days after phacoemulsification and intraocular lens implantation. Baseline IOP dropped after the administration of propofol, increased after succinylcholine, and then further increased during the ECT-induced seizure. Their patient ultimately underwent 9 treatments until remission and experienced no complications. Sienaert et al.[60] concluded as follows: taking a reasonable period for wound healing into account, cataract surgery should not be seen as a contraindication to ECT. It is advisable to use an anesthetic regimen known to reduce IOP significantly, such as etomidate, or propofol, either in monotherapy or in combination with remifentanil.

Multiple Sclerosis (MS)

The prevalence of major depression among patients with MS is higher compared with that in the general population, and it occurs in approximately 50% of patients. Importantly, depressive symptoms such as a lack of energy, fatigue, and cognitive disorders can be misdiagnosed as MS-related symptoms[61]. The knowledge of the safety and efficacy of ECT in depressive patients with MS is based solely on case reports[61]. It was suggested that ECT might cause the deterioration of the neurological state (new MS lesions in follow-up magnetic resonance imaging [MRI]), and moreover, there were also data indicating some anesthesiological complications and difficulties in patients with MS[61]. So far, all case reports except one recommended ECT to be a safe and effective treatment for depressive symptoms in patients with MS[61]. Mattingly et al.[62] described three depressive patients with MS whose psychiatric symptoms were treated effectively with ECT, although one of the patients deteriorated neurologically as was indicated by gadolinium contrast MRI scans. The mechanism of potential exacerbation of MS symptoms over the course of ECT is unknown[62].

Urban-Kowalczyk et al.[61] experienced two seizure episodes that occurred after the 14th episode of ECT in their patient. They suggested that about 3-4% of patients with MS experienced seizures[61]. Seizures can occur at any point during the course of MS and may sometimes be the first symptom of the disease[61].

The safety of ECT in patients with MS raises doubts not only because of the possible neurological deterioration but also because of unusual reactions to anesthesia: some authors suggest that general anesthesia itself might be the cause of the exacerbation of neurological symptoms and, moreover, autonomic dysfunction manifesting as abnormal thermoregulatory and sweating responses and heart rate variations in patients with MS was described[61]. Anesthesiologists recommend mivacuronium in patients with MS during ECT as a safer muscle relaxant than the commonly used succinylcholine, which is related to a higher risk of hyperkalemia in this population[61].

Conclusions

ECT procedures continue to have risks, and these risks are associated with general anesthesia, tonic-clonic seizures and convulsions, the interaction between concomitant medications and ECT, and other procedural aspects of ECT. Based on the findings of a recent paper, ECT may be safer than is widely reported[7]. The reported adverse events are mostly rare and generally minor in severity. According to the American Psychiatric Association, ECT has no absolute contraindications; however, some conditions pose a relatively high risk, and there are many other kinds of complications associated with ECT

that may cause death during the treatment. Understanding such complications and their management strategies can avoid unnecessary discontinuation of treatment due to manageable ECT complications and, furthermore, ECT clinicians must always consider the risk-benefit ratio when treating high-risk and medically complicated patients. Moreover, anesthesiologists must evaluate risk factors carefully prior to ECT and pay attention to modifications in patient management or ECT techniques that may diminish the level of risk. To avoid complications, it is very important that the anesthesiologist perform a pre-ECT evaluation. Proper anesthetic care also allows safe administration of ECT in patients with multiple coexisting medical conditions.

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