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978-0-521-88388-7 - Electroconvulsive and Neuromodulation Therapies

Edited by Conrad M. Swartz

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Electroconvulsive and Neuromodulation Therapies

This ground-breaking new text is the definitive reference on electroconvulsive and neuromodulation therapies. It comprehensively covers the scientific basis and clinical practice of ECT as well as comparisons between ECT and medication therapies, including the new generation of antipsychotic drugs. It also provides readers with administrative perspectives and specific details for the management of this modality in clinical practice. The new forms of nonconvulsive electrical and magnetic brain stimulation therapy are also covered in detail, in a separate section. The chapter authors are leading scholars and clinicians.

DR. CONRAD M. SWARTZ is a board-certified psychiatrist, elected Fellow and past president of the Association for Convulsive Therapy, and two-time recipient of the Clinical Research Award from the American Academy of Clinical Psychiatrists for studies involving ECT. His extensive scholarly publications about electroconvulsive therapy and clinical pharmacology reflect his combining doctoral skills in engineering with medical psychiatry to find new practical solutions to clinical problems. He has directed ECT programs at several medical schools as well as research, education, and clinical programs. He is a co-author of the recently published title *Psychotic Depression* (Cambridge University Press, 2007).

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Preface

This book is dedicated to describing how electroconvulsive therapy (ECT) treats mental illness. Besides treating mental illness, ECT can prevent mental illness in several ways. First, ECT interrupts psychosis and catatonia and thereby prevents episodes of these from persisting and becoming chronic. Genetic data have identified similarities rather than distinctions between psychotic mood disorders and schizophrenia (e.g., Kishimoto et al., 2008; Taylor, 1992). These data complement epidemiologic–phenomenological studies that find continuous variation between psychotic mood disorders and schizophrenia, without a point of rarity to demarcate them. In other words, there is no known difference between schizophrenia and a psychotic (or catatonic) episode that has persisted. We should make every effort to interrupt these episodes before they become entrenched. In this way, ECT should prevent chronic schizophrenia.

Second, but just as important, ECT circumvents using antipsychotic drugs in patients who would otherwise receive them. As detailed in Chapter 23, “Electroconvulsive therapy or antipsychotic drugs (or benzodiazepines for catatonia),” these medications can cause a variety of serious psychiatric, neurological, and medical impairments. It takes a powerful lot of faith to believe that patients with psychosis who receive antipsychotic medications will indeed achieve remission and then maintain it after these medications are stopped. In reality, the data show the opposite. Virtually 100% of patients started on antipsychotic medications for psychosis eventually receive the diagnosis of schizophrenia on follow-up. Psychiatric diagnosis is simply not this reliable. It should not be this reliable for a diagnosis of exclusion such as schizophrenia, especially when the exclusion is not made rigorously. These data point to schizophrenia as often (but not always) the result of an “antipsychotic trap.” After all, what psychiatrist can boldly face the liabilities of discontinuing antipsychotic drugs on an outpatient? These issues are reviewed in detail in Chapter 23.

Third, if ECT is used early in a serious psychiatric episode, it prevents or abbreviates further threatening experiences associated with the illness, including stigmatization, loss of control over life course and job performance, self-injurious behavior, loss of control of thoughts, separation from home and family, and exposure to

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psychiatric wards. Decreasing these threats prevents or diminishes the development of anxiety disorders such as post-traumatic stress disorder (PTSD). Accordingly, ECT is a treatment that should be used early; it is not merely a last resort. Several chapters in this volume elaborate on this message.

Causation of anxiety disorder by serious psychiatric illness is a pervasive, serious, and underrecognized problem in psychiatric patients. It can fall under several different diagnoses and guises besides anxiety disorders. It is called resistant depression, residual depression, chronic depression, and institutional dependency. It has been called “bipolar depression,” a chronic depression that gradually develops and worsens among patients with bipolar disorder. This is not the rapid-onset episode with melancholic or psychotic features that was once known as the type of depressive episode that bipolar type I patients suffered. In my clinical experience, this PTSD from bipolar disorder often underlies the deteriorating course of bipolar disorder. I urge readers to evaluate for comorbid anxiety disorders in patients who have serious Axis-I diagnoses, including patients who receive ECT. Often, comorbid anxiety disorders are missed, and their symptoms are attributed to resistant depression or allegedly genetic panic disorder.

The course of treatment with ECT is analogous to that with antibiotics in patients with acute infections. Treatment typically brings the patient to remission and is then stopped. Any side effects then disappear over days to weeks. The patient receives a persistent benefit, the removal of active disease. The illness can recur, but steps can be taken to prevent it.

ECT is also analogous to surgery. Surgery can be controversial, some surgeries have been overused, surgical procedures become more effective and safer with technological progress, and patients who undergo surgery wish there were an easier way. Throughout, there is no doubt of its frequent necessity, its effectiveness in professional hands, and the right of the patient and doctor to choose it. Indeed, I recommend a surgeon’s lecture, a recording of surgeon Sherwin Nuland, MD, eloquently describing his personal experience receiving ECT. This program clearly illustrates the value of ECT to our patients; it is on the Internet at: http://www.ted.com/index.php/talks/sherwin_nuland_on_electroshock_therapy.html (accessed January 17, 2009).

Several chapters here contend with obstacles to ECT, their development, and the influences of politics. In my professional lifetime, the strongest negative influence about ECT on the public is Milos Forman’s movie, *One Flew over the Cuckoo’s Nest*. Despite the completely fictional nature of this film, through dramatic suggestion it imparts a negative impression of ECT. Yet, even in the movie, ECT itself was not alleged to have caused injury or persisting brain changes. The issues were the crude appearance of unanesthetized ECT, forced nonconsenting administration, the absence of observable psychiatric illness, and a punitive environment. ECT was presented as a whiplashing in medical guise. This has nothing whatsoever to

do with modern ECT as medical treatment for a psychiatric condition that has seriously impaired the patient and caused him to appear sickly, disorganized, and emotionally drained.

Psychiatry is notorious for wide variations in concepts and practice, particularly concerning diagnostic formulations and its explanations. No one person's views encompass what is thought best or even what is proper in psychiatry. My own perspectives are influenced by extensive training in physical science before entering medicine and psychiatry. So it seemed that a wide variety of other authors should be included in this book, and they are. This book is divided into several major sections, and each section has several chapters. Ethics considerations are integrated into the book chapters, rather than collected from them into a separate or redundant chapter, just as ethics are integrated into our clinical and scientific work.

The section on “Scientific and experimental bases of electroconvulsive therapy” begins with my essay on ECT and electricity. Much of this material is new and perhaps surprising. Choosing efficient stimulus pulse width and frequency is included, with an evaluation of ultrabrief stimuli.

In describing historical events in Chapter 2, Niall McCrae reveals the details of how “Nonelectrical convulsive therapies” are experimental bases for ECT and specifically how electrical induction is preferable. Along the way this chapter reflects on many pearls about medical practice, for example, “diagnostic practice is determined by available treatment,” and it is much more than a history.

Writing on the neurochemical effects of seizure and implications for ECT mechanism in Chapter 3, Drs. Renana Eitan and Bernard Lerer and Ms. Galit Landshut will bring you up to date in this wide-ranging and fast-moving area. The numerous alterations in the hippocampus with ECT suggest its involvement in ECT mechanism, but this part of the brain is particularly given to change. This tendency to change together with its involvement in memory suggest that the hippocampus may be involved in ECT cognitive side effects as well as efficacy.

In Chapter 4, Dr. Nikolaus Michael explains and integrates the latest concepts in how seizure generalization, anatomical sites, and neuronal changes are implicated in ECT mechanism.

The photos and descriptions of Dr. Hal Blumenfeld and Ms. Kathy Peng correspond to localized brain effects of ECT stimulus placement and how anatomy is an important consideration in ECT. Their Chapter 5 evaluates the various technologies used in imaging the brain after ECT.

We know ECT works for depression, but the state of clinical evidence establishing that ECT is effective in mood disorders is a scientific matter. It is critically reviewed by Dr. Keith Rasmussen in Chapter 6.

Although there are no double-blind randomized ECT studies of catatonia or schizophrenia, “catatonic features” is the only psychiatric syndrome in DSM that requires verifiable observable evidence – and nothing but – in making the diagnosis.

So studies of ECT efficacy in catatonia have an aspect of objectivity missing from other treatment studies in psychiatry, including those of major depression, and this objectivity provides clear evidence of ECT efficacy. The state of knowledge about ECT efficacy in catatonia (with or without schizophrenia) and schizophrenia is reviewed in Chapter 7 by Drs. Gabor Gazdag, Stephan Mann, Gabor Ungvari, and Stanley Caroff.

Bypassing speculations without evidence, I reviewed only the known effects and patterns of ECT-induced hormone changes in Chapter 8, “Hormonal effects of electroconvulsive therapy.”

Beginning the section on “Historical, societal, and geographic perspectives,” in Chapter 9 historian Edward Shorter summarizes both the fascinating history of ECT and variations in modern professional opinions about aspects of ECT practice. This is a living history of ECT, not merely a past. For more details, please see his book on the topic.

Movies mentioning ECT comprise most peoples’ entire awareness of it, which is one reason to read Chapter 10 by Drs. Andrew McDonald and Garry Walter on popular books and movies about ECT. They explain how such portrayals are impressionistic rather than factual.

Strong barriers to ECT stand within the professional world as well as outside it, as Dr. William Reid elucidates in Chapter 11. To confront them or even operate next to them, it is important to understand their nature. Moreover, Dr. Reid identifies several likely surprises, including the requirements for residency training in the United States.

Legislation in some countries and U.S. states deprives many patients of access to ECT, regardless of their medical needs. Notably, some of this legislation was motivated by followers of L. Ron Hubbard (“Scientology”) or was in reaction to ECT use that is now understood as not appropriate. In Chapter 12, Dr. Alan Felthous reviews these issues and explains how it remains useful to understand them.

The “International perspectives” section begins in the United States with a review of availability by Drs. Michelle Magid and Barbara Rohland in Chapter 13. Dr. Susan Benbow begins Chapter 14 by describing recent conflicts and misleading acronyms in the UK that represent an apparent assault on psychiatry by nonpsychiatric physicians. Famous for its tolerance, Scandinavia seems to have a more stable medical environment for ECT, as reviewed by Dr. Tom Bolwig in the conclusion of Chapter 14. Drs. Pascal Sienaert and Walter van den Broek similarly describe the generally receptive environment for ECT in Western Europe in Chapter 15. Ironically, the environment for ECT is difficult in Italy, where ECT originated.

In contrast, ECT availability and quality are widely variable in Asia, as noted by Dr. Sidney Chang in Chapter 16. In Russia ECT has been strongly influenced by national politics, according to details provided by Drs. Alexander Nelson and

Nataliya Giagou in Chapter 17. In South America, the ECT environment reflects variability in sociocultural and economic conditions, as reviewed by Drs. Moacyr and Marina Rosa in Chapter 18.

Although it is relatively brief, the “Administrative perspectives” section should provide valuable assistance in facing the bureaucratic expectations of administrating an ECT service. In Chapter 19, Dr. Barry Kramer offers detailed archetype documentation for “Electroconvulsive therapy hospital policy and quality assurance.” In Chapters 20 and 21, Dr. Jerry Lewis describes a salt-of-the-earth perspective on everyday concerns in “Staff management and physical layout for electroconvulsive therapy” and prototype forms for ECT service operation. These concerns include making privacy, efficiency, and completeness routine for both inpatient and ambulatory ECT.

The next section is a practical guide to the clinical aspects of ECT practice, in effect an ECT practice manual. As the first step is patient selection and ECT indications, this appears next as Chapter 22. In describing who is suitable for ECT and who is not, selectively, it differs fundamentally from the American Psychiatric Association (APA) Task Force Report, which aimed to allow rather than select.

Antipsychotic drugs are the medications most used in patients who should receive ECT but do not. I review how this differs from using these drugs in chronic schizophrenia in Chapter 23, “Electroconvulsive therapy or antipsychotic drugs (or benzodiazepines for catatonia).” This chapter elucidates how and why long-term antipsychotic drugs should be reserved as the last resort in psychiatric management.

Informed consent is required in the United States. Obtaining understanding by patients and families can involve psychological insight as well as knowledge about the procedures, and in Chapter 24, Dr. Peter Rosenquist aims to help achieve it. This chapter systematically presents ECT consent within the general considerations of informed consent.

Drs. Keith Rasmussen and Paul Mueller consider how to identify and reduce the wide variety of risks associated with concurrent medical conditions as part of the pre-ECT evaluation, in Chapter 25.

Anesthesia for ECT has basic differences from surgical anesthesia and cannot merely be delegated to an anesthesiologist because many details potentially influence the psychiatric outcome. In Chapter 26, Drs. Charles Kellner, Dongchen Li, and Limore Maron present what the psychiatrist needs to know about anesthesia in ECT.

Regarding the electrical stimulus, two main considerations are where to place it and how to dose it. I present placement in Chapter 27, and Dr. Vaughn McCall reviews dosing in Chapter 28, but these chapters cannot be entirely separate from each other or from Chapters 1 and 31. These chapters describe variations in opinion that correspond to common variations in clinical practice. Perhaps the reader

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should scrutinize my views most strongly because I have influenced this book as the editor.

Moving on to monitoring, the intricacies of the ECT seizure as displayed on the electroencephalogram (EEG) are demystified and systematically described by Dr. Hideki Azuma. Chapter 29 elucidates both the structure of the seizure and EEG terminology, which should assist communications about EEGs among ECT practitioners. In Chapter 30 on heart rate, I analogously discuss the pattern of heart rate acceleration and deceleration that accompanies the ECT seizure along with its clinical meanings and uses.

The cognitive effects and concerns associated with ECT are explained by Dr. James Stuart Lawson in Chapter 31, along with cognitive testing that can be performed to monitor it and identify when the patient has completed convalescence. Dr. Lawson discusses how cognitive effects from symptomatic psychiatric illness and medications can affect cognitive testing and how intellectual testing results can improve with ECT.

Lately, antipsychotic medications have been widely promoted for use in children and adolescents, even in those who do not have schizophrenia. These medications have never been established as safer than ECT in children and – as outlined in the chapter on antipsychotics earlier in this section – seem far more dangerous. So we include a timely review by Drs. Garry Walter, Colleen Loo, and Joseph Rey on ECT methods particular to adolescents and children in Chapter 32.

Post-ECT evaluation and medication prophylaxis and its consequences are elucidated by Drs. T. K. Birkenhäger and Walter van den Broek in Chapter 33. Drs. Charles Kellner and Unnati Patel note that ambulatory ECT usage is growing rapidly and might now be as common as inpatient ECT, especially in urban settings. Accordingly, their review of its efficacy and clinical specifics in Chapter 34 should be of interest to most readers. Some additional details that concern ambulatory ECT appear in Chapters 20 and 33.

The final section focuses on the newest somatic treatments in psychiatry. Transcranial magnetic stimulation (TMS) is explained and reviewed by Drs. Oded Rosenberg and Pinhas Dannon in Chapter 35. Vagus nerve stimulation is presented in Chapter 36 by Drs. Shawn McClintock and Mustafa Husain and Mr. Kenneth Trevino. Deep brain electrical stimulation through implanted electrodes is described by Drs. Thomas Schläpfer and Bettina Bewernick in Chapter 37. Perhaps less well-known than these other treatments, but also promising, transcranial direct current stimulation (tDCS) is presented by Ms. Julie Williams and Dr. Felipe Fregni in Chapter 38. Several studies comparing different methods of tDCS have reported varying efficacy and efficacy greater than sham treatment, and this suggests that tDCS may be useful clinically. The details here imply that tDCS differs from alternating (bidirectional) low-level currents, such as delivered by devices for transcutaneous electrical nerve stimulation (TENS).

I will take an editor's prerogative to state a few medical concepts in this preface. First is my rationalization of how and why ECT works, the "Reboot Theory." This is followed by my preferences in ECT clinical practice.

The reboot theory of ECT mechanism and its implications

How electrical current induces seizure is presented in Chapter 1. The clinical question is how the seizure produces psychotropic benefits. I will first summarize the "reboot theory" and then explain it; I believe its initial mention was by Swartz (1984). This mechanism of ECT effect is inherently tied to the phenomenon of seizure, and it has two phases: seizure and recovery from seizure. In the first phase, ECT grand mal seizure depletes brain neurotransmitters by causing neuronal depolarization and neurotransmitter release. In the second phase, this depletion is corrected by replenishment of neurotransmitters according to gene transcription. The induction of replenishment is stimulated by homeostatic mechanisms operating in response to the depletion. This mechanism meshes closely with the details of Chapter 4 and is compatible with the other scientific chapters.

By definition, there is no ECT therapeutic effect without preexisting psychiatric illness. In this mechanism, the psychiatric illness is mediated by a pathological pattern of neurotransmitters in the brain. This pattern resulted from interactions between the patient's genes and the environment. These interactions are life experiences that affect body physiology, such as activation of the sympathetic nervous system. The patterns of illness that result from this interaction can result from combining severely pathological genes with mild life stresses. Such defective genes represent a fragile patient and a highly heritable illness. Illness can also result from combining mildly pathological genes with severe life stresses; this combination represents severe or repeatedly severe adverse life experiences. Alternatively, illness can result from moderate gene predisposition and moderate life stresses.

Eventually, if not interrupted, this pathological pattern of neurotransmitters can begin conversion into a structural pattern, and in time this structural pattern can become established more deeply, essentially worn in. The stronger the structural pattern, the more resistant it is to change, including the changes that ECT can make. When the pathological pattern is primarily in neurotransmitters, the illness has a good prognosis if treated. When the pattern is strongly established in cell structure, it is, for example, chronic schizophrenia. Even when a psychiatric illness is established structurally it should have some neurotransmitter components that can be mitigated by depletion and replacement.

The basic characteristic of grand mal seizure, as in ECT, is the widespread depletion of central nervous system (CNS) neurotransmitters until a change occurs to stop the seizure. Seizure termination is apparently related to neurotransmitter depletion or to the extensive release of neurotransmitters, itself concomitant with

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neurotransmitter depletion. As the ECT seizure depletes neurotransmitters, their pathological patterns and networks are disrupted and so is the expression of illness they mediate. With repeated ECT seizures, the pathological patterns are progressively depleted and disrupted. Eventually the pathological pattern fades, and so does the illness.

This therapeutic effect is buttressed by replenishment of neurotransmitters according to gene transcription that is activated by neurotransmitter depletion, neurotransmitter release, or both. With repeated ECT seizures, this replacement and its patterns become stronger and more extensive, and they gradually displace the pretreatment pathological pattern of neurotransmitters and eventually become dominant. This pattern of replenishment and replacement reflects only the genes, not effects of the environment or any interaction with the environment, and thereby differs from the pretreatment pathological pattern.

The benefit of neurotransmitter depletion and replacement should persist as long as the gene–environment interaction effects do not accumulate sufficiently to overcome it. Maintenance ECT should work by depleting accumulations of gene–environment interaction effects on neurotransmitters. Perhaps lithium works by diminishing the effect of the environment on neurotransmitter patterns; such a mechanism for lithium is consistent with its effect of decreasing second messenger activity.

The gradual and progressive processes of neurotransmitter depletion and replacement correspond to the gradual improvement and eventual achievement of remission along a course of several ECT sessions. It is somewhat analogous to rebooting (restarting) a computer, suggesting a name for this mechanism. In this analogy, psychiatric illness resembles errors accumulating in random access memory during computer operation. These errors eventually produce malfunctions. ECT is analogous to clearing the computer memory by restarting it from the bootstrap memory (read-only memory [ROM]), in other words, rebooting. This is a nondestructive process.

In this mechanism, ECT restores brain neurotransmitter patterns and function to a normal preillness state. This mechanism differs from the mechanisms of medical interventions that obstruct aspects of brain function, for example, psychosurgery, deep brain electrical stimulation, antipsychotic drugs, and slow TMS. It also differs from treatments that stimulate aspects of brain operation, such as vagal nerve stimulation, stimulant drugs, and rapid TMS (rTMS).

According to this mechanism of ECT, the seizure and its therapeutic effects are inseparable. It is hard to imagine how a medication could accomplish similar neurotransmitter depletion and replacement actions without inducing seizure, and there is no conceivable equivalent treatment or substitute for convulsive therapy (or ECT). Aspects of the convulsive therapy procedure might change in how it

is induced, its intensity, and its location, but seizure – neurotransmitter depletion and replacement – is the essence of ECT therapeutic effect and not just its means.

This mechanism accounts for a broad range of ECT phenomena, including providing therapeutic benefits in several different neuropsychiatric disorders. Presumably each disorder has its own pathological pattern of neurotransmitters. The nonspecific nature of neurotransmitter depletion should disrupt any neurotransmitter-mediated illness. It corresponds to the gradual and cumulative clinical effect of giving several ECT treatments and the possible involvement of multiple neural networks, anatomic locations, and neurotransmitters. It is consistent with the stronger therapeutic effect generally seen with greater seizure generalization, including bilateral ECT and higher stimulus doses. The mechanism accounts for ECT cognitive side effects, as recent memory resides in labile neurotransmitters whereas remote memory is structural. It meshes with the genetic–environmental interaction theories of mood disorders and the greater resistance to treatment of longer-lasting illnesses. It explains the corrective therapeutic action of ECT, in which remission typically continues despite discontinuation of the treatment, analogous to antibiotic treatment of infection. Finally, this mechanism and its aspects can be tested and developed further, and its elements can be explained briefly and simply to patients and their families.

ECT surely causes neurochemical changes besides depleting neurotransmitters, such as generating anticonvulsant activity and briefly releasing hormones from the pituitary and other glands. The anticonvulsant activity might help to relieve psychopathology caused by seizure disorders or focal brain irritability. Because several anticonvulsant medications treat mania or diminish somatic tension anxiety, so might the anticonvulsant effects of ECT. However, anticonvulsant activity from ECT persists for only several weeks and so does not explain the persistent remission that typically continues after ECT is stopped.

My ECT clinical preferences

Approach to informed consent

I aim to harness the ECT strength so that it specifically helps patients whose illness is visible. Even seriously ill patients are concerned about personal appearance and privacy. Together, these observable features provide concrete, accurate, and persuasive information about the benefits of ECT.

First, I examine for noticeable signs of illness. These include poverty of speech, motor retardation, masked facies, exhaustion, pained expression, difficulty in solving problems, withdrawal, and malnutrition. Then I describe what I see back to the patient. I typically describe deficits in initiating conversation, physical movement,

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and emotional expression. A patient only rarely disputes my saying that other people easily see that he or she is ill, and that his or her illness is not private.

I mention that we have a treatment that should help him or her look normal, feel healthier and stronger, and manage his or her own life as before the illness. This is usually persuasive.

I believe that my obtaining informed consent is helped by my maintaining good outcomes with only rare side effects. These good outcomes require strict patient selection and avoidance of bitemporal ECT as a routine. On the ward, patients watch each other. When obviously ill patients improve, other patients notice the difference. When ECT patients show confusion, they also notice – and can feel threatened. When I started using left anterior right temporal (LART) placement, my ECT patients improved without showing confused behavior, and other patients began requesting ECT.

Sedation for sleep on the night before ECT

Promethazine (Phenergan) 25 to 50 mg at bedtime. It is a highly sedating antihistamine and a mild antiadrenergic and antidopaminergic. Yet, it does not inhibit seizure. At 50 mg (25 mg in elderly patients), mild drowsiness can continue in the morning. This drowsiness helps to blunt pre-ECT anxiety. I avoid benzodiazepines because they are anticonvulsant. Zaleplon (Sonata) is too weak for inpatients but should be useful for ambulatory ECT patients.

Pre-ECT intramuscular medication

Glycopyrrolate 0.0044 mg/kg, 1 to 3 hours prior to ECT. When I omit it, saliva secretion usually interferes with treatment and seemingly risks aspiration. I prefer intramuscular (IM) to intravenous (IV) because patients seem drier and some anesthesiologists object to IV.

Pre-ECT oral medication (two to four hours prior to ECT with water sip)

Many patients develop sore muscle headache after ECT. For them, I first try either ibuprofen (400 mg) or acetaminophen (500 to 1,000 mg). Headache patients usually assert their preference, and I order accordingly. Heat and massage can help.

Atropinic agent IV

If glycopyrrolate was not given IM, I give it IV.

Routine narcosis agent, dose range

My preference is methohexital (0.4 to 0.7 mg/kg). My aim is only to obtund the patient from awareness of succinylcholine paralysis. Higher doses can inhibit seizure, but patients with past heavy drinking often require them. If methohexital

is not available, I prefer etomidate. For patients younger than 30 years, for the first few ECT sessions I don't mind propofol, about 1 mg/kg. I avoid propofol in elderly patients because of anticonvulsant effects and aspiration risk if the patient takes an antipsychotic or has parkinsonism.

Routine muscle relaxant, dose

In the succinylcholine routine, I prefer 1 mg/kg in muscular or lean patients, 0.7 mg/kg for average patients, and 0.4 mg/kg in the morbidly obese. If the patient has past postictal excitement, I use 1.1 mg/kg (more if the anesthesiologist is willing).

Other common pre-ECT IV agents

When pre-ECT acetaminophen or ibuprofen does not control post-ECT headache, I use ketoprofen (Toradol; 15 to 30 mg). I rarely give other drugs pre-ECT. For patients who show hypertension or tachycardia after ECT seizure, esmolol or labetalol given 30 seconds poststimulus does not inhibit the seizure. Given before the stimulus they can shorten seizure.

Extra steps (if any) for patients taking benzodiazepines

I have not seen flumazenil help obtain a seizure in patients taking benzodiazepines, so I avoid it. I prefer gradually discontinuing benzodiazepines (to avoid withdrawal) but not delaying ECT for this. As the first ECT treatments show the most vigorous seizures, it is still timely to stop benzodiazepines by the third or fourth ECT treatment. In older patients, I additionally compensate for recent benzodiazepines by increasing stimulus dose.

Oxygenation

I prefer maximum hyperventilation from the time of obtundation until the stimulus, regardless of oximeter readings. Higher oxygen and lower carbon dioxide levels promote seizure. Ventilation according to oxygen saturation alone unfortunately allows carbon dioxide levels to accumulate and inhibit seizure. After the stimulus, I aim for a pink patient and an oxygen saturation of at least 97%.

Other anesthetic considerations

Minimizing antipsychotic use in elderly patients cuts aspiration from parkinsonian dysphagia. I avoid propofol in patients on an antipsychotic, those with Parkinson's disease, those receiving no atropinic agent, and elderly patients in general. In patients with postictal excitement despite a succinylcholine dose of 1.1 mg/kg, I administer an additional methohexital (30–50 mg) or propofol bolus immediately after the motor seizure.

xxviii Preface**Electrode placement**

LART placement is my routine. If I avoid excessive electrical dosage, cognitive side effects are usually impalpable. Lower side effects should result from both its asymmetry and its physical separation from the working memory functions of the left dorsolateral prefrontal cortex.

However, if the patient is actively suicidal or violent, I use bitemporal ECT. When the patient has a history of severe ECT confusion, I use right unilateral placement unless it produced this problem. If the patient shows no improvement after four good-quality ECT treatments, I usually switch to bitemporal ECT.

Stimulus dose method

I use the Benchmark Method to adjust the stimulus dose along the course of treatment, with peak heart rate and tonic motor seizure as the physiological indicators. The stimulus dose at the first ECT is “half age %Energy” at 900 mA and 0.5-ms pulse width with LART or bitemporal placement. This dose has a charge of 2.5 mC/year of age at a 900-mA current. I increase this dose if seizure-inhibiting influences are present, for example, anticonvulsants and benzodiazepines. For unilateral ECT, I start with “full age %Energy” (5 mC/year). At 800 mA the equivalent charge is 4 mC/year for any bilateral ECT and 8 mC/year for unilateral ECT.

I monitor heart rate during seizure and motor seizure. If no tonic motor activity occurs, it is not a good benchmark seizure and I increase stimulus dose. I also increase the electrical dose if peak heart rate is less than 140 bpm, unless the patient is older than 80 years or has a specific medical reason for low heart rate (e.g., propofol anesthesia, atherosclerotic cardiovascular disease). Otherwise, the peak heart rate becomes the initial benchmark. If peak heart rate at later ECT treatments is within 6 bpm of that benchmark and at least 140 bpm, the stimulus dose is high enough. If peak heart rate is lower, I increase stimulus dose. Likewise, if there is no tonic motor activity, I increase the electrical dose. I follow EEG signs of seizure intensity (such as postictal suppression), but it only rarely adds guidance.

Stimulus potentiation (after maximum stimulus dose is reached)

My first approach is basic: medication discontinuation, clear airways, maximal hyperventilation, minimizing methohexital dosage, and considering promethazine at bedtime before ECT. Next, I switch to etomidate anesthesia. If this is not enough, I usually give two stimulus doses, one right after the other. Traditional ECT devices do not prevent this. Even a gap of 5 seconds between the two stimuli should not prevent their effects from combining. In this double stimulus method, each stimulus should not exceed a four-second duration, so the total does not exceed eight seconds. If this is inadequate, I switch to ketamine anesthesia and use a single maximum stimulus dose. The next step after this is a double stimulus. My final

step, in addition to everything else, is IV caffeine pre-ECT, 500 mg for an elderly patient or 1,000 mg for other patients.

Caffeine is last because of adverse histological CNS neural effects (Enns et al., 1996). This toxicity occurred with high caffeine doses alone, so it is probably the same effect caused by any exposure to amphetamine or methylphenidate and should not prevent IV caffeine when truly needed.

Physiological monitoring (and specific signs)

I monitor tonic motor activity and peak heart rate during the seizure. If there is no tonic motor activity, total motor duration is less than 18 seconds, or peak heart rate decreases as described in “Stimulus dose method,” I strongly consider increasing stimulus dose. If peak heart rate is within 10 bpm of baseline heart rate and motor duration is less than 10 seconds or equivocal, I restimulate under the same anesthesia. I do not restimulate for weak EEG morphology alone, but if it weakens I increase the dose at the next session.

If motor seizure exceeds 60 seconds or EEG seizure exceeds 120 seconds, I administer IV methohexital, propofol, or midazolam to terminate the seizure.

Recovery from ECT

Unpleasant feelings of agitation from postictal excitement dispose patients to withdraw consent, so I treat this vigorously, but with drugs eliminated from the body by the next ECT. I use midazolam (1 mg IV), monitoring carefully for apnea. After the patient can swallow, I give oral oxazepam, with another dose four to five hours later. For the next ECT, I plan to prevent postictal excitement with higher doses of succinylcholine and sometimes a second dose of methohexital at the end of the seizure, as noted earlier.

Ward management

With any catatonic or moderately or severely depressed inpatient, I weigh him or her daily; monitor orientation, initiation of speech, psychomotor activity, and emotional expression; and describe changes since ECT was started.

Discharge considerations

I try to enforce a minimum ECT course of six sessions. My median is seven. I treat to plateau, with one to two extra sessions for patients who are risky, resistant, or long ill. Then I evaluate for anxiety disorder. This evaluation is routine because patients can develop PTSD from the experience of having a severe psychiatric illness. I investigate discrepancies between improvements I see and patient self-assessments. If psychomotor activity normalized but the patient complains of continuing “depression,” low mood, dissatisfaction, apprehension, or persistent

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unpleasant thoughts, then anxiety disorder is likely. ECT probably temporarily decreases somatic tension anxiety, but in patients with anxiety disorder, somatic tension still needs treatment and selective serotonin reuptake inhibitors do not reliably mitigate it.

I advise that patients avoid making major life or financial changes within two months of the end of an ECT course because ECT can temporarily induce an orbital–frontal syndrome or dysexecutive syndrome and it can be subtle, but it should fade within five to six weeks.

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Conrad M. Swartz

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