Electroconvulsive therapy for persons with developmental disabilities: review, case report and recommendations

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Electroconvulsive Therapy for Persons With Developmental Disabilities: Review, Case Report and Recommendations

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Electroconvulsive therapy (ECT) is an effective, but under-utilized treatment for mood disorders, particularly recurrent depression. Its use is even rarer in persons with intellectual disability. The literature is sparse; we found only 14 reports which described the ECT treatment of 20 individuals with intellectual disability. All reports were anecdotal, but supported the efficacy and safety of ECT for such persons. The authors present a woman with intellectual disability and recurrent psychotic depression, who has been successfully treated with acute and maintenance ECT. The history and technique of ECT is described, and guidelines for its utilization in persons with intellectual disability are presented.

Clinical depression presents with a wide variety of symptoms in persons with intellectual disability. Several authors have described the prevalence and presentation of depressive disorders in individuals with intellectual disability. They suggest depressive disorders may be masked, and its presentation modified, by intellectual disability.

Similarly, as the diagnosis of depressive disorders in persons with developmental disability (DD) has become more widespread, so has the treatment become more standardized. There are now multiple reports of the use of pharmacologic and nonpharmacologic approaches to treating depressive disorders in persons with intellectual disability. Less often described, electroconvulsive therapy (ECT) is another major treatment option for major depressive disorders. This therapy is under-utilized in persons with intellectual disability; a literature review reveals only 14 reports of the use of ECT in persons with mental retardation (MR). We summarize these reports, describe a woman with intellectual disability and major depressive disorder with psychosis who was successfully treated with acute and maintenance ECT, and present guidelines for the use of this therapy in persons with intellectual disability.

BACKGROUND

History and Indications

Convulsive therapies in psychiatry were first utilized by Dr. Ladislas Meduna, who was among the first to note the apparent mutual exclusivity between epilepsy and schizophrenia, and attempt to treat schizophrenia by inducing seizures. In the pre-pharmacologic era of psychiatry, ECT was soon being investigated for a number of psychiatric, and even medical, conditions. These ranged from arthritis, malignancy and ulcer, to delinquency, marital discord and homosexuality. ECT was occasionally tried as a sedative for uncooperative or agitated patients, and as an aversive stimulus for the extinction of maladaptive behaviors. In recent decades, clinical experience, ethical safeguards, and the discovery of more effective pharmacological treatments have
relegated the use of ECT to a few fairly specific indications.\(^1\) Currently ECT is indicated and recommended for the following conditions:

1. major depressive disorder, single episode and recurrent
2. bipolar depression
3. bipolar mania
4. schizophrenia, particularly with catatonic or affective symptoms

In the above, ECT is most often utilized as an acute treatment, with the usual course of acute ECT consisting of five to ten treatments, spaced at intervals of one to two days. Often, at least the initial portion occurs while the individual is on an inpatient psychiatry service. Clinical features determining the initial or ongoing need for inpatient status may include the presence of suicidal ideation or activity, psychotic symptoms or inability to provide for basic activities of daily living.\(^1\) When these features are absent or enter remission, many persons are discharged to complete a course of acute ECT on an ambulatory basis.

Most of the reports demonstrating the use of ECT in persons with intellectual disability are single case reports or small case series. A literature review (searching for manuscripts specifically about ECT, and also reports of biological treatments of depressive illness in persons with MR), produced 14 articles reporting a total of 20 individuals. To date, there are no controlled studies regarding the efficacy of ECT in patients with intellectual disability, nor are there reports comparing the efficacy and safety of ECT to antidepressant medications in these individuals. This may reflect the difficulty in confidently diagnosing depressive illness in persons with intellectual disability, lack of acceptability in utilizing ECT and logistical problems in conducting this type of research.\(^1\,2\,20\)

Nearly all of the available case reports describe therapeutic success with ECT, as negative results are rarely reported in this fashion. Most report utilizing ECT after one or more unsuccessful trials of antidepressant medications; ECT is often similarly utilized in this fashion for depressed persons without intellectual disability.\(^1\)

In 1968, Payne\(^16\) described two adults who were considered to have mild intellectual disability. The first was a 38-year-old man, who developed manic depressive psychosis. He responded to ECT, and maintained his baseline cognitive ability after ECT, as measured by IQ testing. The second individual, who was diagnosed with hysterical aphony and primitive catatonic psychosis, failed ECT but responded to pharmacotherapy. This report was the only description of failure to respond to ECT in our literature search. Payne did not describe his ECT technique in either instance.

Nearly 15 years later, Bates and Smeltzer\(^2\) described a 25-year-old man with severe intellectual disability and life threatening self-injurious behavior (SIB), who was unresponsive to multiple medication trials. After ECT he was able to be free of restraints and helmet, and was SIB-free for four months. ECT was described as life-saving, and without adverse consequences, but legally difficult to administer because the individual had no guardian. Guze et al.\(^6\) described a 21-year-old man with mild MR, bipolar depression, and cerebral palsy, who received ECT after lack of response to nortriptyline. He then had a manic episode six weeks after ETC. Kearns\(^9\) reported a 67-year-old man with severe MR and psychotic depression who improved with ECT, after failing treatments with two antidepressants.

Zucker and Jensvold\(^25\) described a 68-year-old man with mild MR, alcoholism, and intractable depression. He developed a psychotic depression, and received ten
unilateral ECT treatments with complete remission. Post-ECT neuropsychiatric/neuropsychological testing at one week and three months post-ECT demonstrated no change compared to baseline.

Warren et al\textsuperscript{24} identified five adults with Down's syndrome who had presumptive Alzheimer's disease, surrounding severe deterioration of cognitive and vegetative function. All five were subsequently treated for possible depressive disorder, three with ECT after antidepressant failure or intolerance; the other two with antidepressants. All five patients responded to therapy, with improvement in cognitive function.

Merrill\textsuperscript{14} treated a woman with profound retardation and recurrent depression, who had failed to respond to multiple medication trials, including antipsychotics, antidepressants, and lithium. When she developed life-threatening symptoms, including tachycardia, fever, dehydration, and severe SIB, judicial approval was sought for ETC. After six treatments, the woman had a dramatic response. She completed ten treatments with remission of symptoms.

Lazarus et al\textsuperscript{11} presented two individuals with DD who responded to ETC. The first was a 32-year-old woman with cyclic depression who had not responded to multiple medication trials. ECT produced remission, but four months later, she relapsed following her mother's death. She was given three more treatments, then entered monthly maintenance treatment for six months. The same authors also treated a 50-year-old woman with mild MR who had been hospitalized 15 times for depression. She responded to ECT on five occasions over four years. Day\textsuperscript{4} also presented two adults, a 37-year-old woman and a 47-year-old man, both of whom had multiple episodes of psychotic depression treated successfully with antidepressants and ETC.

Puri et al\textsuperscript{18} described a man with mild MR and a strong family history of mood disorder (mother committed suicide and father alcoholic). At age 22, he had ECT for an apparent psychotic depression, with successful remission. At age 27, he attempted suicide on two occasions. After multiple medication failures, bilateral ECT produced a transient response. Over the next 1-1/2 years, he was given maintenance ECT every three weeks, and remained in remission until the maintenance interval was lengthened to six weeks. He relapsed with another suicide attempt. The interval of treatment was reduced again to three weeks, with another remission.

Karvounis et al\textsuperscript{8} reported a 69-year-old man with moderate intellectual disability and a history of depression, whose previous episodes had responded to antidepressant medication. During the described recurrence, he failed to respond to dothiepin or fluoxetine. He did improve with 11 outpatient bilateral ETC.

Jancar and Gunaratne\textsuperscript{7} presented two adults, both with moderate MR and dysthymic disorder, whose superimposed major depressive episodes responded to ETC. Both maintained chronic dysthymia in spite of ECT or medication management.

Bebchuk et al\textsuperscript{3} described a 44-year-old man with profound MR and three years of insomnia, agitation, crying, rumination, SIB, and 62 pound weight loss. He failed trials of neuroleptics, anxiolytics, antidepressants, and mood stabilizers, but his illness, thought to be either major depression or bipolar disorder with mixed features, responded to six unilateral ECT, with weight gain, improved sleep, decreased rumination, and increased verbalization. This response was maintained with monthly maintenance ETC. Following a relapse, he responded to six more bilateral ECT, and resumed twice-per-month maintenance ETC.
Renshaw et al reported a 37-year-old man with adult GM2 gangliosidosis (AGG), a very rare neuropsychiatric disease characterized by cerebellar and neuronal deterioration, associated with psychotic and affective symptoms. Although rare, it is known that antidepressant and antipsychotic agents are likely to aggravate the neurologic dysfunction in these individuals. The patient described has been treated over 15 years for multiple psychotic episodes with a number of psychotropic medications, and ultimately developed tardive dyskinesia. As a result, his psychotropic medications were discontinued, and he redeveloped major depression, mutism, and severe weight loss. Because of his medication sensitivity, ECT was undertaken; after two treatments he began to speak, and after five resumed eating.

Attempting to summarize the above anecdotal reports may reveal several trends. All patients were adults, ranging from 17-69 years. Most were relatively young (17-31), although three were in their late 60's. Fourteen of 20 were male. The majority had intellectual disability in the mild to moderate range, although ECT was also utilized for several individuals with severe, or even profound intellectual disability. All but two patients were thought to have depressive illness, many with psychotic features. Four individuals with severe or profound retardation were more difficult to diagnose, but three of these had significant self-injurious behavior and neurovegetative symptoms, as part of their illness. Twelve of 20 patients received a single course of ECT, eight others had multiple courses of acute ECT, and three reports describe the use of maintenance ETC. Only one individual reported did not respond to ECT; the other 19 were positive reports. This is typical for anecdotal literature. Two reports specifically noted that the patient's cognitive function was maintained after ECT treatment, and in three others it was reported to improve after ECT; these three patients had apparent depression-induced dementia that improved with ETC. When reported, the technique and number of treatments necessary for remission appeared to be similar to that reported for ECT in persons without intellectual disability. No report discussed specific negative effects of ECT, although two authors noted that successful ECT was followed by a manic episode; ECT and antidepressants are both known to induce mania in a minority of bipolar patients.

Case Report

Ms. B. is a 44-year-old single woman with intellectual disability in the mild range, who lives with her biological parents. Her family history is significant only for a paternal aunt who had depressive illness, and completed suicide. Ms. B.'s prenatal history and birth were unremarkable, and she talked at one year of age, walked at 18 months, and completed toilet training at 2-1/2 years. She attended kindergarten at age 5, where she was discovered to have intellectual disability. She suffered an initial major motor seizure at age 6, and was treated with phenytoin (DPH). She also was struck by a car at age 30, suffering a fractured hip, which required internal fixation.

Ms. B. first developed psychiatric symptoms around age 37, when she became disoriented and delusional, following the departure from the household of her ill grandmother, who had lived with the family. Ms. B. was seen by her neurologist, who prescribed haloperidol, on an as-needed basis, with resolution of the above symptoms. A repeat EEG at that time was unchanged, and she did well for several months, although she appeared to her parents to be mildly depressed.

Historically, Ms. B. had worked since her graduation from school in several sheltered
settings, and at the time of illness onset, was working as a staff member in an Adult Training Center, providing care for individuals with more severe and profound intellectual disability. She was sufficiently independent that her parents could leave her at home alone for several days to a week when they traveled out of town, during which time they would call daily, and her siblings would check on her. In the year following her initial episode, a next door neighbor died, and Ms. B. became agitated and made some irrational statements, but no treatment was necessary. Several months later, when she was passed over for a hoped-for job promotion, she returned from work sobbing, and again delusional. Surrounding the severity of her symptoms, she was hospitalized psychiatrically, and treated again with haloperidol (doses of 15-20mg/day) and benztropine. Laboratory examinations and head CT were unremarkable. Following an episode of severe dystonia and oculogyric crisis, haloperidol was replaced with trifluoperazine. Ms. B. suffered similar side-effects, and eventually the second antipsychotic medication was discontinued as well. She was discharged significantly improved, on no medications. However, within one week she began pacing, and was unable to perform activities of daily living, requiring assistance in bathing, dressing and eating. She developed a poor appetite, lost 10 pounds, had sleep disturbance and decreased energy from her baseline. Ms. B. became withdrawn and nearly nonverbal, which was markedly different from her premorbid personality. She regressed, wanting to sit on her parent's lap, and became preoccupied with death. She exhibited delusions that her parents had died, and would often scream about their deaths, even in their presence. She reported a belief that she had been adopted, and was suspicious about the death of an aunt the previous year. She appeared internally stimulated, and described hearing non-command hallucinations of voices. On one occasion, she attacked her mother. There was no SIB behavior initially, but later she began scratching at her back to the point of excoriation.

Her mental status examination at the time of admission included restlessness with increased psychomotor activity, lack of spontaneity, flat affect and dysphoric mood. She would often lie in a fetal position, had decreased concentration, was easily distracted, and was disoriented to time and place. Her mother reported that she would sometimes fall in the hospital, which occasionally seemed to have a purposeful attention-seeking quality. During this hospitalization she was treated initially with thioridazine and alprazolam without benefit, and was subsequently treated with lorazepam and amoxapine, 350mg/day. On the latter regimen she improved, and was discharged with a diagnosis of major depression, recurrent, with mood congruent psychosis.

Ms. B. did well for 2-1/2 more years, when at age 40, she relapsed with signs and symptoms similar to her previous episode. Amoxapine was increased, but later discontinued, secondary to lack of response and serious tachycardia. A subsequent trial of sertraline combined with haloperidol failed to improve her status. She was then re-challenged with amoxapine, which provided no benefit at a dose up to 500mg/day, and was complicated again by serious tachycardia. An echocardiogram done at that time demonstrated an atrial septal defect, requiring cardiac consultation. Her head CT scan was again normal. Finally, after six weeks of continuous symptomatology, ECT was discussed with her parents. After discussions of the risks and benefits of ECT, and alternate therapies, her parents (who were legal guardians) approved. Ms. B. received bilateral brief pulse ECT using a
MECTA machine, which she tolerated well, and after the fifth treatment her symptoms improved. She began walking, resumed spontaneous verbalization, began participating in groups, eating and sleeping well, and she no longer displayed psychotic or depressed behavior. She was discharged taking fluoxetine, DPH, and lorazepam. With the recurrence of some psychotic symptoms, loxapine was added. Over the next two months Ms. B. became mildly hypomanic, and fluoxetine was decreased from 20mg/day, to 10mg every other day. When, after six months of treatment with loxapine, Ms. B. developed an orobuccolingual dyskinesia, loxapine was tapered, and within two months Ms. B. became symptomatic again, with agitation, loud and confused verbalizations, disorientation, decreased oral intake, and insomnia lasting several days at a time. She would talk to herself, and appeared to be hallucinating. Treatment with antipsychotic medication was resumed using risperidone, initially at low doses, with gradual titration to 6mg/day. When her symptoms failed to resolve, she was re-hospitalized and received a second course of ECT, this time receiving seven non-dominant unilateral treatments, with complete resolution of her depressive and psychotic symptoms. Following this second successful course of ECT, a decision was made to attempt continuation (or maintenance) treatment, surrounding her serious and debilitating symptoms, and lack of either acute or prophylactic response with combined psychopharmacologic treatments. For six months she received a single monthly ambulatory non-dominant unilateral treatment. She subsequently had another relapse, with crying spells, depression, auditory hallucinations, and paranoid delusions. On this occasion she was treated with five non-dominant unilateral, followed by three bilateral ETC. Although Ms. B. had seemingly responded to unilateral ECT administration on her previous episode, on this occasion five unilateral treatments produced an unsatisfactory response, but three more bilateral treatments seemed to produce remission. She was discharged to resume outpatient maintenance ETC.

Ms. B. then remained on maintenance ECT for a period of two years, receiving a single monthly ambulatory treatment. Treatments have utilized bilateral stimulus, stimulus intensity 10, which has produced dynamic energy averaging 110 joules, and seizures ranging from 30-65 seconds (average 43 seconds), as measured by cuffed limb technique, and EEG. During this time she returned to work, and was able to tolerate several significant life changes without relapse. In 1997, she again relapsed with confusion, withdrawal and agitated behavior, decrease in her ability to perform ADLs, auditory hallucinations, persecutory delusions involving the death of relatives, poor appetite, and weight loss. Ms. B. was psychiatrically hospitalized and had a MRI of her brain, which was unremarkable. During this admission, she was again treated with acute ECT, began improving after her fourth treatment, and was discharged after six treatments. Following a two month remission, she was readmitted for a mild relapse and was given two treatments within the same week, which seemed to prevent further deterioration. At that time her primary medications were also changed, with valproate replacing DPH, and Olanzapine® replacing haloperidol. Until the present, Ms. B. had maintained another 12 month remission with single monthly ambulatory maintenance ETC. Over the last two years she has been able to accompany her parents on extended vacations out of her geographic area, through the arrangement of single ambulatory treatments through a University treatment center near where her family is
vacationing.

Ms. B. had undergone a neuropsychological evaluation at age 38, toward the end of a hospitalization, and prior to the initial ETC. At that time, her performance IQ on the Test of Nonverbal Intelligence (TONI) was 61. A repeated evaluation done within the last year (age 43) as part of a process of qualifying Ms. B. for more independent work, demonstrated a WAIS-R verbal IQ of 72, performance IQ of 62, with a full scale IQ of 66. It appears that Ms. B. has suffered no intellectual deterioration as a result of her illness or treatment, although her examiner noted that her independent adaptive skills were slightly lower, compared with her previous functional ability.

**DISCUSSION**

A brief description of the procedure of ECT can be found in Appendix I.

As noted, ECT is an effective treatment for persons with major depressive disorders, particularly those associated with psychotic or suicidal symptoms. The efficacy of a treatment, however, must be balanced against its risks and side-effects. These are described below.

*Risks*

There are no absolute contraindications to the use of ECT, but some situations increase ECT risk. Increased intracranial pressure (ICP) (as may occur in hydrocephalus, or CNS space-occupying lesions such as brain tumors), or the concurrent administration of ECT to a person receiving lithium pharmacotherapy, provide substantial risk. Several types of cardiac arrhythmias, as well as severe respiratory compromise, are relative contraindications, as they pose additional hazards to the use of anesthetic and paralytic premedications. Recent myocardial infarction (heart attack), CNS vascular event (stroke or hemorrhage), recent retinal detachment, and pheochromocytoma are also relative contraindications. Care must also be taken with severe illness of the cervical or thoracic spine (disc compression, or atlantoaxial instability). The latter malformation should be evaluated in a person with Down's syndrome prior to instituting ECT, as it would contraindicate neck hyperextension during ventilation.

Surrounding the use of anesthesia, there is a small risk of mortality (about 1 per 10,000 patients). In general, the morbidity and mortality currently seen with ECT is thought to be less than that associated with antidepressant therapy. ECT does not produce epilepsy, nor does a seizure disorder contraindicate using ETC. When treating individuals with known seizure disorders, clinicians maintain adequate anticonvulsant treatment throughout ECT, but modify the stimulus dose so as to produce an induced seizure. The previously significant orthopedic injuries seen with early ECT have been all but eliminated with the use of premedication. ECT can cause transient headache, nausea, and muscle soreness, all of which respond to symptomatic treatment.

Probably the most common side-effect of ECT today is post-ECT confusion and memory dysfunction. Post-ECT confusion is brief, time-limited, and appears consistent with the post-ictal state, with perhaps some contribution of anesthetic effects. ECT may cause some permanent loss of memory for events that occur immediately prior to, or during the course of ECT, but does not appear to have any negative effect on remote memory, or prevent the ability to learn and remember new information. None of these areas of cognitive function have been studied surrounding ECT use in persons with intellectual disability. Memory dysfunction is clearly more common with ECT utilizing bilateral electrode placement and sine wave
stimulus, and treatments given in rapid succession (more than three per week). With the use of non-dominant electrode placement, brief pulse stimulus, and spacing of treatments, memory dysfunction is significantly attenuated.

We did not find any report of ECT use in persons with MR which described significant cognitive or memory morbidity as a result of ETC. However, such results would be unlikely to be reported, based on the rarity of inclusion of persons with MR in general studies of ETC.

Continuation Treatment of Depression

Because depression is a chronic disorder, with predisposition to relapse, a number of techniques are undertaken to prevent the re-emergence of symptoms (prophylactic treatment). These primarily involve the continuation of the treatment which produced the remission of the acute depressive episode. It is now widely accepted that individuals who suffer a depressive episode have a 50% chance of having a second episode. Having a second episode predicts future episodes in more than 80% of persons.

A continuation therapy in medicine is defined as any form of treatment undertaken to prevent a relapse, after successful treatment of the acute condition. It is now widely recognized that the use of continuation treatment in depression significantly reduces the risk of illness recurrence. While antidepressant medications are the most commonly utilized form of continuation therapy, ECT can also be utilized in this fashion.

Continuation ECT

ECT is most likely to be utilized as continuation treatment for the following persons:

1. Those whose acute depression was responsive to ECT, particularly if ECT had been successful after unsuccessful antidepressant pharmacotherapy.
2. Persons for whom continuation therapy with antidepressants poses some additional risk (dangerous drug-drug interactions, intolerable side-effects).
3. Persons who had failed previous continuation therapy with antidepressants, and had suffered recurrences, requiring a second series of acute ETC.
4. Noncompliance with other continuation therapies, with relapse.

Maintenance ECT

When continuation ECT is continued for longer than six months after a specific depressive episode, it is usually referred to as maintenance ECT (ECT-M). Because studies of ECT-M have varied widely in terms of persons treated, diagnoses, concurrent treatments with medications, and ECT technique, no consistently agreed-upon data exist regarding how ECT-M should be administered. The APA Task Force on the use of ECT, and the ACT (Association of Convulsive Therapy), have published a general set of recommendations for continuation and maintenance ECT, however, which are summarized in Appendix II.

Summary

ECT remains misunderstood, sometimes feared, and as a result, generally under-utilized as a treatment for persons with major depression and other psychiatric disorders. This is particularly true for individuals with intellectual disability, for whom there has been additional reluctance to recommend this form of treatment. This reluctance appears twofold. The first reason is a global under-treatment of persons with MR and psychiatric disorders. The second involves a specific reluctance to utilize a treatment that has
traditionally been associated with cognitive and memory dysfunction, in persons who already have cognitive/intellectual problems. As a result, there are only a handful of reports of ECT use in persons with intellectual disability; our review found only 20 individuals. Of these, 12 received a single course of ECT, eight others more than one course of acute ECT, and three reports describe ECT used as a maintenance form of treatment. Ms. B's clinical course adds to this literature. She has now received 65 ECT over a 4 year 8 month span. It is informative that on most recent neuropsychological testing, she had had no apparent decrement in intellectual ability. This is reassuring, in that the literature seems to support that certain individuals with MR and chronic depression do not respond to prophylactic treatment with pharmacotherapy. This appears to mirror the experience in chronically-depressed persons without intellectual disability - there is a subgroup of depressed individuals who appear to require ECT in order to remain in remission.

Based on the above, we agree with Lund, that ECT should be considered for persons with MR who meet the clinical criteria for utilization of ECT in the general population. These would include persons with:

1. major depressive disorder, particularly with psychosis or suicidality
2. bipolar depression
3. bipolar mania
4. schizophrenia, with catatonic or affective symptoms

where there is:

1. a need for a rapid, definitive response, or
2. a risk of other treatment that may outweigh ECT risks, or
3. a history of poor medication response, and/or good ECT response on previous episodes, or
4. a history of treatment failure with medications, or
5. a history of adverse side-effects with medications.

We also recommend consideration of the use of maintenance ECT for those persons who respond to ECT utilized acutely, and who have the features noted above, with lack of medication response, and/or adverse effects. Appropriately utilized, ECT can be a safe, effective, and at times, life-saving treatment for these persons.

Appendix I

Procedure of ECT

A number of modifications have been made in the technique of ECT since its inception 60 years ago, making the procedure not only safer, but also more acceptable to individuals. The most important has been the use of premedication, and cardiac and respiratory monitoring, which now require the attendance of practitioners with advanced anesthesiology training. In "premedicated" (also known as "modified") ECT, the individual is given three medications intravenously prior to the seizure stimulus. First is an anticholinergic agent to dry oral secretions, help maintain an airway, and prevent bradyarrhythmias. The second medication is a short-acting anesthetic (generally a short-acting barbiturate such as methohexital), which produces unconsciousness. Once asleep, the patient receives succinylcholine, which paralyzes the muscles of the limbs and trunk. This relaxation has essentially eliminated the previous orthopedic injuries that were sometimes seen with vigorous seizures. The muscles of respiration are also temporarily paralyzed, so that the anesthesiologist must ventilate the individual with mask, bag, and oxygen for 5-8 minutes.

Following anesthesia and relaxation, the
treating psychiatrist administers an electrical stimulus to the patient's head, through two stimulus electrodes. This is followed by a 30-60 second major motor (Grand mal) seizure, which is observed (most persons move only fingers or toes), and recorded on a single channel electroencephalogram (EEG). Following the seizure, the individual requires assisted ventilation for another 3-5 minutes. After another 5-10 minutes, he/she regains consciousness. Throughout the pre-treatment and post-treatment process, individuals are monitored with EKG, VS, and often pulse oximetry (measurement of blood oxygenation).

Two other modifications involve the location of stimulus electrodes on the head, and the form of electrical stimulus utilized. Unilateral stimulus (with both stimulus electrodes on the same, non-dominant side of the brain) is associated with less post-treatment confusion and memory dysfunction. Most individuals given ECT today are treated initially with unilateral non-dominant stimulus, but there are individuals who respond better (or only) to the traditional bilateral approach.

Most ECT machines today are able to deliver either sine wave, or brief pulse stimuli, which reduces the total electrical stimulus, and is associated with a reduction in post-treatment confusion and memory dysfunction. Most individuals given ECT today are treated initially with unilateral non-dominant stimulus, but there are individuals who respond better (or only) to the traditional bilateral approach.

APPENDIX II

Recommendations for Continuation or Maintenance ECT

1. The individual has a recurring episodic illness (depression) which has been responsive to ETC.
2. Pharmacotherapy has proven ineffective, or cannot be safely administered.
3. The person is agreeable to receive continuation ECT, and is capable, with the assistance of significant others when necessary, of complying with the treatment plan.
4. The timing of treatment should be individualized, and should be adjusted as necessary.
5. Maintenance ECT should be administered at the minimum frequency compatible with sustained remission; in general, this frequency will be one treatment every one to three months.
6. The continued need for ECT maintenance should be reassessed at least every three months, considering both beneficial and adverse effects.
7. Treatment facilities should develop procedures for the pre-ECT evaluation for ECT maintenance, which should include:
   a. interval psychiatric evaluations
   b. interval medical history and physical examinations, focusing on specific systems at risk from the ECT
   c. at least every three months:
      1. determination that continuation ECT is indicated
      2. serum electrolytes and hemoglobin/hematocrit
      3. an assessment of cognitive function
   d. at least every six months: updated consent for ECT
   e. at least annually: EKG

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