Electroconvulsive Therapy in Late-Life Depression

Harold A. Sackeim

Electroconvulsive therapy (ECT) plays a significant role in the treatment of late-life depression and other psychiatric conditions in the elderly. Compared to pharmacologic treatments, ECT is administered to an especially high proportion of elderly patients. For example, a survey of practice in California between 1977 and 1983 indicates that the probability of receiving ECT increases markedly with patient age (Figure 1). Of 1.12 persons per 10,000 in the general adult population treated with ECT, 3.86 per 10,000 are aged 65 years or older;[1] treatment with ECT was constant over this period and the high percentage of elderly patients is noteworthy. A national survey of inpatient psychiatric facilities conducted by the National Institute of Mental Health (NIMH) also indicates that patients aged 61 and older comprise the largest age group to receive ECT in 1975 to 1980.[2] In the mid-1970s when use of ECT in the United States had declined, ECT treatment of inpatients aged 61 and over remained constant.[2] Use increased again during the 1980s; in 1986 the national estimate was that 15.6% of inpatients aged 65 or older with mood disorders received ECT, compared with only 3.4% of younger inpatients with mood disorders.[3]

Data from the most comprehensive national study of factors associated with inpatient use of ECT, published in 1998, estimated that nearly 10% of a sample of nearly 25,000 depressed inpatients received ECT during their hospital stay.[4] (Figure 2) Factors most strongly predicting ECT use were age, race, insurance status, and median income of the patient's home zip code. Older patients, Caucasians, and those with private insurance living in affluent areas were most likely to be treated with ECT. Diagnosis rather than age, however, is the primary indication for the use of ECT. The vast majority of patients treated with ECT in the United States were experiencing an episode of major depression, either unipolar or bipolar. The NIMH national diagnostic survey conducted in 1986 reveals that 84% of patients who receive ECT are diagnosed with a major mood disorder.[3] The primary factors leading to consideration of ECT, regardless of age, are (1) a history of inadequate response or intolerance to antidepressant medication or (2) a history of good ECT response during prior depressive episodes.[5–7] ECT is administered less frequently for schizophrenia[8] and mania.[9]

Among patients of all ages, ECT is more effective and more likely to produce symptom remission than antidepressant medication.[10–15] The extent to which ECT is used early or late in the course of antidepressant treatment varies markedly from country to country and, within the United States, varies considerably among localities and practitioners.[16,17] ECT is particularly beneficial when elderly depressed patients are also medically ill, psychotic, or suicidal. Thus, ECT is most frequently administered to geriatric patients when anti-
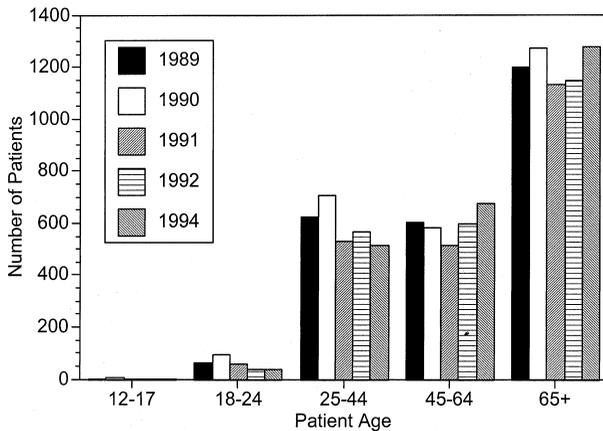
Figure 13.1. Patients treated with ECT in California, by age group and year; data for 1993 were unavailable.

depressant medications are too risky, have proven ineffective, or when ensuring a rapid or full clinical response is particularly important.

Indications for ECT

ECT is indicated for the acute treatment of depression as well as for maintenance treatment and prevention of relapse. Clinical outcome of ECT is more predictable in patients exhibiting particular characteristics of major depression. As with phar-macologic treatment, duration of the depressive episode consistently correlates with a positive ECT response: patients (of all ages) with longer duration of illness respond less well.[18–24] This relation between duration of illness and treatment response may reflect the impact of depression in CNS functioning. Duration of a depressed state correlates with the extent of hippocampal atrophy associated with chronic depression. Because the hippocampus has an established role in regulating the hypothalamic–pituitary—adrenal
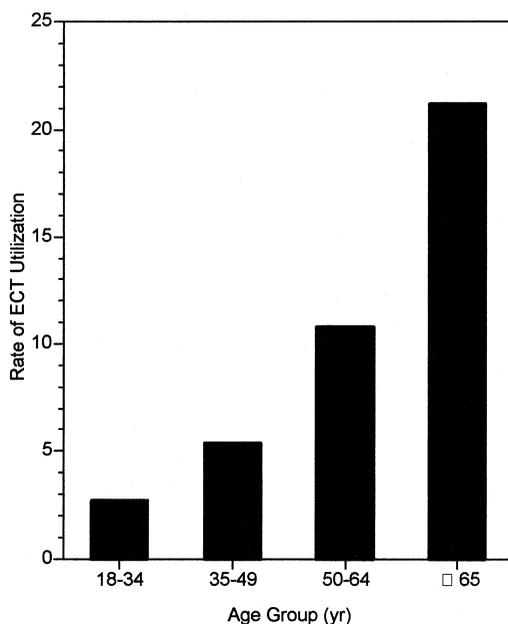

Figure 13.2. Rate of ECT utilization in a sample representative of inpatients in the US in 1993 with a diagnosis of recurrent, major depression. (From Olfson, et al. 1998.)[4]

(HPA) axis, the atrophic effects of depression may lead to increased vulnerability to stress and prolongation of the episode. Patients with hippocampal atrophy are especially resistant to antidepressant medications, particularly tricyclics. Poor response to antidepressants, in turn, predicts/correlates with inferior short-term response to ECT[18,25–27] as well as to other somatic treatments.[28,29] ECT is likely to be of greatest value when it is administered early in the course of a depressive episode and not as a last resort after all other treatments have failed.

Maintenance ECT, with treatments spaced over weekly to monthly intervals, is increasingly used for relapse prevention.[30–32] Unfortunately, continuation or maintenance ECT is commonly employed only after pharmacologic methods of relapse prevention have failed following successful ECT. At present, the vast majority of patients who respond to ECT are then treated with antidepressant medications despite evidence that failed medication regimens during the acute depressive episode are ineffective in preventing relapse following ECT.[24,26,30,33,34]

Efficacy

Overall, research observations and clinical experience indicate that ECT is particularly useful in the treatment of late-life depression. Prior to the introduction of ECT, elderly depressed individuals often exhibited chronic depression or died of intercurrent medical illnesses in psychiatric institutions.[35] A number of studies contrast the clinical outcome of depressed patients who received inadequate or no somatic treatment to that of patients who received ECT (Figure 3) While none of this work involves prospective, random-assignment designs, the findings are largely uniform. Contemporary ECT administered to elderly patients results in decreased chronicity, decreased morbidity, and possible decreased rates of mortality.[36–39]

Studies comparing ECT to other forms of antidepressant treatment[15,40] are relatively sparse. A metaanalysis of early comparative-age patient samples[11] reports that the average response rate to ECT is 20% higher when compared to tricyclic antidepressants (TCAs) and 45% higher when compared to monoamine oxidase inhibitors (MAOIs), although by modern standards, the pharmacologic treatments used were often suboptimal.[18,33,41,42] No study has ever found a pharmacologic regimen to be superior in antidepressant effects when compared to ECT. Rather, ECT consistently has had either equal or superior efficacy. In both young and elderly populations, ECT is superior to a standard antidepressant,[15] although the addition of lithium to an antidepressant results in more rapid onset of improvement compared to ECT in patients with treatment-resistant depression.[40,43,44]

Other differences between ECT and antidepressant treatments concern speed and quality of clinical response as well as residual symptoms. Residual symptomatology resulting from incomplete response to antidepressant medications may become chronic or lead to relapse.[45] Because remission is more likely following ECT, there is less chance of recurrence of chronic residual depressive symptoms.

Whether ECT reduces depressive symptoms more quickly than antidepressants in the elderly has not been adequately tested. Nonetheless, evidence suggests that no pharmacologic strategy results in as rapid symptomatic improvement as ECT.[10,12,13] Significant clinical improvement is usually seen within the first few treatments, with maximal gains seen by 3 weeks. This rapid improvement is less common with antidepressant medications.

Aging and Efficacy

The response rate to ECT is higher among older patients,[46–49] and a positive association is seen between patient age and degree of clinical improvement following

Sine Wave

Intensity

0 2 4 6 8 10 12 14 16
Time (msec)

Partial Sine Wave

Intensity

0 2 4 6 8 10 12 14 16
Time (msec)

Brief Pulse

Intensity

0 2 4 6 8 10 12 14 16
Time (msec)

Ultra Brief Pulse

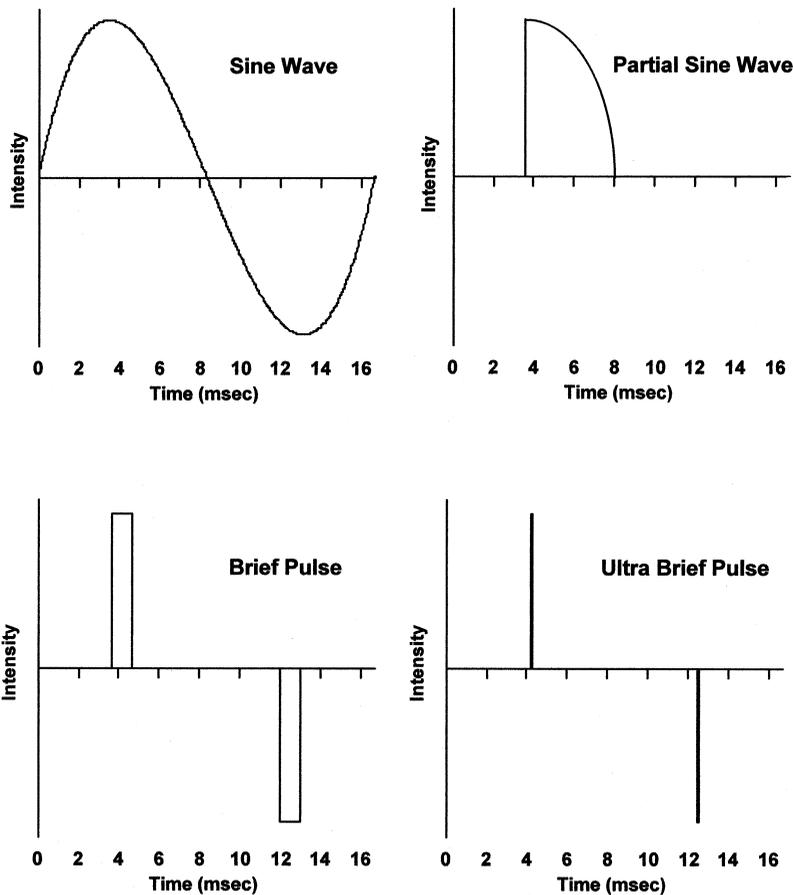Intensity

0 2 4 6 8 10 12 14 16
Time (msec)

Figure 13.3. Examples of waveforms used in ECT. Top left: sine wave. Top right: chopped, rectified sine wave. Bottom left: brief pulse, square wave. Bottom right: ultra-brief pulse, square wave. (From Sackeim HA, Long J, Luber B, et al. Physical properties and quantification of the ECT stimulus: I. Basic principles. *Convuls Ther* 1994;10:93–123).

ECT.[46–49] Table 13.1 provides a selective summary of studies addressing the relation between patient age and ECT outcome. Older individuals, however, may have a diminished response to unilateral, as opposed to bilateral, ECT[50,51] and may require longer courses of treatment to achieve the same level of remission as younger patients.[52,53] ECT may also be effective in the very oldest depressed patients.[54–57]

Predictors of Response

Two factors correlate with response to ECT in the elderly: intensity of the electrical stimulus, and the patient's diagnosis.

The extent to which the intensity of the electroconvulsive stimulus exceeds the individual patient's seizure threshold determines the efficacy of right unilateral ECT and speed of response regardless of electrode placement.[58–62] Age is one of the more reliable predictors of seizure threshold[62–64]: the oldest patients generally have the highest thresholds, shortest seizure duration, and lowest EEG seizure amplitude.[60,65,66]

In addition to stimulus intensity, evidence shows that among depressed patients, those with psychotic or delusional depression respond especially well to ECT.[25,30,67–72] Although not definitively

Table 13.1.   Relation Between Patient Age and ECT Outcome: Selected Studies

Study*	Patients	Study Design	ECT Treatment	Results/Comments
Prudic et al. (2004)	347 patients with major depression treated at 7 hospitals in the New York metropolitan area	Prospective, naturalistic study of ECT practices and outcomes in community settings; evaluations before, immediately after, and monthly for 6 months following ECT	Modified ECT given 3 times/week; diverse electrode placement and dosing practices	Age, treated as a continuous variable, not related to symptom improvement or categorical clinical outcomes
O'Connor et al. (2001)	253 patients with major depression, treated openly with titrated (50% above seizure threshold) bilateral ECT	Prospective naturalistic acute phase study followed by double-blind continuation trial comparing nortriptyline and lithium with continuation ECT; acute response to ECT contrasted in 3 patient groups: young adult (<45 years), older adult (46 to 64 years) and elderly (65 years and older)	Modified ECT given 3 times/week; patients titrated at first session and treated afterward with suprathreshold intensity 50% above threshold	Lower remission (70%) in youngest age group than either older adults (89.8%) or elderly (90%); when these dimensions were treated as continuous variables, age was also related to outcome.
Sackeim et al. (2000)	80 inpatients with major depression randomized to 4 groups (right unilateral at 1.5, 2.5, or 6 times seizure threshold or bilateral ECT at 2.5 times seizure threshold); free of all medications except lorazepam (up to 3 mg/day PRN)	Double-blind, prospective study of effects of electrical dosage and electrode placement; patients evaluated before ECT and regularly during and after treatment course	Modified ECT given 3 times/week; patients randomized to 3 forms of right unilateral ECT (1.5, 2.5, or 6 × seizure threshold) or bilateral ECT (2.5 × ST)	High dosage right unilateral ECT (6 × threshold) and bilateral ECT (2.5 × threshold) equal in efficacy and superior to lower dosage right unilateral ECT; no age-related effects

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Table 13.1. (*continued*).

Study[*]	Patients	Study Design	ECT Treatment	Results/Comments
Tew et al. (1999)	268 patients with major depression, treated openly with suprathreshold unilateral or bilateral ECT; free of all medications except lorazepam (up to 3 mg/day PRN)	Prospective naturalistic acute phase study followed by double-blind continuation pharmacotherapy trial; acute response to ECT contrasted in 3 patient groups: adult (59 years and younger), young-old (60 to 74 years) and old-old (75 years and older)	Modified ECT given 3 times/week; patients titrated at first session and treated with suprathreshold intensity selected by treating psychiatrist	More physical illness and cognitive impairment in both older age groups than in adult group. Both older groups had shorter depressive episodes and were less likely to be medication resistant. The adult patients had a lower rate of ECT response (54%) than the young-old patients (73%), while the old-old patients had an intermediate rate of response (67%).
Sackeim et al. (1993)	96 inpatients with major depression randomized to 4 groups (unilateral or bilateral ECT at low- or high-stimulus intensity); free of all medications except lorazepam (up to 3 mg/day PRN); age range, 22–80; mean, 56.4	Double-blind, prospective study of effects of electrical dosage and electrode placement; patients evaluated before ECT and regularly during and after treatment course	Modified ECT given 3 times/week; patients titrated at first session and treated either at just above threshold or at 2.5 times initial seizure threshold	Electrical dosage determined unilateral ECT efficacy and speed of response for unilateral and bilateral ECT; no age-related effects seen.
Black et al. (1993)	423 depressed inpatients between 1970 and 1981	Retrospective chart review using multiple logistic regression to identify response predictors	Modified ECT given 3 times/week; bilateral, unilateral, mixed courses included	Patients rated as recovered (n = 295) older than those rated as unrecovered (n = 128)

Study				
Sackeim et al. (1987a, b)	52 inpatients with major depression randomized to unilateral or bilateral ECT; patients free of all medications except lorazepam (up to 3 mg/day PRN); age range, 25–83; mean, 61.3	Double-blind, prospective study comparing low-dose, titrated bilateral and unilateral ECT; patients evaluated before ECT and following treatments 1, 3, 5, 6 and every treatment thereafter	Bilateral or right unilateral modified ECT 3 times/week; patients titrated and treated at just above threshold	Low-dosage right unilateral ECT ineffective; regardless of ECT modality, age unrelated to clinical outcome
Coryell & Zimmerman (1984)	31 patients with unipolar depression selected prospectively	Prospective study of ECT response predictors; ratings made on HAM-D at weekly intervals by blind raters	Most treatments unilateral; most patients had at least 6 treatments; patients with fewer than 4 treatments excluded	Age independently associated with outcome on more than 1 of 3 outcome measures; superior outcome in older patients
Rich et al. (1984)	Data from 2 groups of patients pooled: 66 with major depressive episode or organic affective syndrome; antidepressant medications either stopped prior to ECT or held constant	Prospective study of response rate to conventional ECT by identifying point of maximal improvement; patients rated on HAM-D before first ECT and at 36–48 hours after each treatment	Modified ECT given 3 times/week; right unilateral ECT used >80% of patients; mean no. of treatments for each of 2 groups 8.6 and 8.3	Age associated with longer time to achieve response; study flawed by use of different rating scales and different ECT devices for 2 groups
Fraser & Glass (1980)	29 depressed (Feigner criteria) elderly (64–86 years) randomized to unilateral or bilateral ECT	Prospective, double-blind, randomized study; postictal recovery times, memory changes, and clinical improvement assessed by HAM-D	Modified ECT with twice-weekly treatment until patient well or ECT stopped	No age difference between good and moderate outcome groups
Heshe et al. (1978)	51 patients with endogenous depression randomized to unilateral or bilateral ECT	Prospective blind evaluations before ECT, at end of ECT, and 3 months after final treatment	Either modified unilateral (average 9.2 treatments) or bilateral ECT (average 8.5 treatments), twice weekly, number of treatments decided by treating clinician	In patients over 60, significantly better therapeutic effect from bilateral than unilateral treatment; regardless of modality, satisfactory results in 75% of patients >60 years and 96% of patients <60 years–a significant difference

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Table 13.1.   (*continued*).

Study*	Patients	Study Design	ECT Treatment	Results/Comments
Herrington et al. (1974)	43 consecutive severely depressed patients (aged 25–69) randomized to ECT or l-tryptophan (up to 8 g/day); 40 patients included in efficacy analysis	Patients rated on day before treatment and weekly thereafter for 4 weeks	ECT given twice weekly for total of 6–8 treatments	Age unrelated to outcome
Strömgren (1974)	100 patients with endogenous unipolar or bipolar depression; aged 19–65; patients drug-free except for hypnotics and mild sedatives	Prospective, double-blind study contrasting unilateral and bilateral ECT	Minimum of 6 treatments given; duration of current individualized; average of 9 treatments given to younger patients, 8.7 to older patients	Of 53 patients aged 19–44, 17 were resistant; 7 of 47 aged 45–65 were resistant–a significant difference efficacy superior in older patients for both bilateral and unilateral ECT
Folstein et al. (1973)	118 consecutive patients who received ECT; diagnoses of schizophrenia, neurotic reactions, and affective disorders	Retrospective chart review: progress notes at time of discharge rated as to whether or not patient improved	Nature and duration of ECT not described	Improvement related to older age and shorter hospital stay; no significance tests provided; mean age of improved patients (n = 86) 50, compared with 31 inpatients rated not improved (n = 32)
Mendels (1965a, 1965b)	53 consecutive inpatients evaluated pre-ECT and 1 and 3 months post- ECT; age 21–76, mean 48.8	Prospective study: patients rated with HAM-D; evaluators not blind to treatment history	4–11 treatments (mean 6.4) with modified ECT	Superior outcome in patients over 50 at 3-month follow-up but not at 1-month follow-up
Carney et al. (1965)	129 depressed inpatients	Prospective study to establish predictive factors; patients scored for presence or absence of 35 features, followed up at 3 and 6 months post-ECT; outcome criteria defined	Patients received 3 or more treatments	Better response in endogenous depressives at 3 and 6 months (per factor analysis); in patients over 40, type of depression not associated with outcome

Study	Sample	Design	Treatment	Findings
Nystrom (1964)	2 series of patients: 254 in Gothenburg series, 188 in Lund; most cases depressed but other diagnoses included	Prospective, blind evaluation; outcome criteria specified	Modified bilateral ECT at 2 treatments/week initially; average number in Lund series 6.9, 4.4 in Gothenberg	Lund series: positive association between age and degree of improvement in females; Gothenberg series: age <25 years negatively related to outcome
Greenblatt et al. (1962)	128 patients randomized to 4 treatment groups; diagnosis of schizophrenia, psychoneurotic and psychotic depressive reactions, involutional psychosis; 28 received ECT; age 16–70, mean 46	Prospective study compared ECT and antidepressant medications; explicit outcome criteria used	ECT modified by succinylcholine given 3/weekly for 3 weeks minimum, more at discretion of psychiatrist	Medications and ECT equally effective in youngest age group; ECT significantly more effective than medications in oldest age group
Ottoson (1960)	44 (18 males, 26 females) with endogenous depression; age 36–70, mean 55.8	Prospective study with blind raters; efficacy evaluated by outcome 1 week after 4th treatment, 1 week after end of ECT course, and total number of treatments required	Modified bilateral ECT with intervals between first 3 treatments of 2–4 days and between following treatments of 3–7 days; dose adjusted upward for age; patients divided into 2 groups: one received stimulus grossly above threshold, one moderately above threshold	Age not significantly related to efficacy; therapeutic response later in older patients
Hamilton & White (1960)	49 hospitalized male patients with severe depression; age range 21–69, mean 51.7	Patients assessed prospectively and 1 month after end of ECT	Usual course 6 treatments, maximum 10; 14 patients had second course	Age unrelated to Outcome
Roberts (1959a,b)	50 patients, women 41–60 years	Prospective study of predictors of ECT response; patients scored on clinical features prior to ECT and presence or absence of symptoms at 1 and 3 months post-ECT	Twice weekly modified ECT until maximum benefit; averaged between 7 and 8 treatments	Symptom scores at 1 month: significant inverse correlation with age (older women more improved); no correlation at 3 months

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Table 13.1. (*continued*).

Study*	Patients	Study Design	ECT Treatment	Results/Comments
Herzberg (1954)	227 cases selected from all patients who had received ECT; diagnoses of schizophrenia, manic depressive psychoses, involutional melancholia	Retrospective chart review of patients rated for initial response to ECT, continued response, no relapse after discharge	Nature and duration of ECT not described	Superior outcome or sustained improvement in patients in 4th decade compared with patients in other age groups
Hobson (1953)	150 patients at Maudsley Hospital; no diagnostic criteria used, but almost all cases were depressed; 127 included in analyses	Prospective study to identify predictors of ECT response; patients categorized as either free of symptoms or still having marked symptoms after ECT	Nature and number of ECT treatments not described	Age unrelated to outcome; several other predictors identified
Rickles & Polan (1948)	200 private patients treated with ECT; diverse diagnostic categories included schizophrenia	Retrospective study of why patients failed ECT; treatment considered failed when improvement not maintained for at least 1 year	Usual course 10–12 treatments; patients with schizophrenia also received 24–40 subcoma insulin shocks	Authors felt that ECT failed if patient was menopausal or postmenopausal; statistics not presented
Gold & Chiarello (1944)	121 consecutive male patients, 103 diagnosed as schizophrenic; age range 15–60	Prospective study of outcome predictors and outcome; patients placed in 1 of 4 categories from *much improved* to *no change*	Type and number of treatments not described	Superior clinical outcome in older age groups

* Complete reference citations at end of chapter.

established, it is also probable that, among patients who receive ECT, the elderly have a lower rate of comorbid Axis II pathology (e.g., personality disorders), which further contributes to a superior ECT response rate.[24,73]

ECT Treatment of Psychotic Depression

ECT is a primary treatment for patients with psychotic depression due to the severity of the disorder, the high rate of response to ECT, and relative poor rate of response to antidepressant monotherapy.[5,30] In mixed-age samples, approximately 30 to 40% of depressed patients who receive ECT present with psychotic depression.[27,67] This rate is likely higher among the elderly, who are more likely to present with psychotic depression than younger patients.[74,75]

Between 20 and 45% of hospitalized elderly depressed patients present with psychotic depression.[76–78] Typically, the elderly patient with psychotic features has severe depressive illness, although the overall severity of late-life depression does not invariably indicate psychosis. Identifying psychotic features in elderly depressed patients is essential because these individuals are at considerably high risk for suicide.[21,79,80]

Psychotic depression is often underrecognized, particularly in the elderly. A telltale sign of psychotic depression is found in the elderly patient who denies being depressed despite psychomotor retardation, anorexia, markedly diminished social interactions, or other symptoms of depression. Further complicating identification of psychotic depression is the need to distinguish between overvalued ideas (''near-delusional states'') and true delusions. Delusions are significantly more common than hallucinations in the geriatric patient with psychosis. In the elderly patient, greater difficulty also occurs in distinguishing between hypochondriasis and somatic delusions because of the common preoccupation with health in older people. Mood-incongruent delusions or hallucinations, whose content is inconsistent with depressive themes, are a consistent feature of psychotic depression. Some elderly patients, however, deny delusions, making diagnosis of psychotic depression more difficult. Since mood-incongruent features may be more common among younger depressed patients and/or those with bipolar depression, the presence of mood-incongruent psychotic features in an elderly patient should trigger consideration of possible bipolarity or an organic affective disorder.

Evidence that the manifestations of psychotic depression tend to be consistent from episode to episode suggests a trait-like quality.[81–83] Furthermore, psychotic depression appears to be inherited, with relatives sharing the same psychotic content.[84,85] Psychotic depression is more frequent in bipolar compared to unipolar depression.[75] However psychotic depression that appears as a first episode after age 50 is frequently unipolar in course. Compared with unipolar depression, the elderly bipolar patient with psychotic depression more frequently experiences psychomotor retardation and sleep disturbance.

Particularly difficult to treat, late-onset psychotic depression is not only subject to a relapsing course; it may lead to later development of dementia.[82,86] Distinguishing between delusions of dementia as opposed to psychotic depression may be problematic. In contrast to the delusions of psychotic depression, the patient with an organic psychotic affective disorder usually has delusions that are less systematized and less congruent with depressive themes whereas the delusions accompanying psychotic depression are usually highly organized and reflect unrealistic or bizarre ideas about somatic illness, nihilism, persecution, guilt, or jealousy. However, the elderly patient with psychotic depression is particularly subject to gross global cognitive deterioration (''pseudodementia''), which reverses with successful treatment of the mood disorder.[87] Evidence also suggests that such

patients later develop a dementing illness.[88]

Elderly patients with psychotic depression respond less positively to pharmacologic treatment (particularly monotherapy) but more positively to ECT than nonpsychotic patients.[89] Specific delusions, in addition to vegetative or melancholic symptoms, predict favorable response,[19,70,90–93] as may psychomotor disturbance.[67,94] In elderly patients with psychotic depression, observation of early resolution of delusions, appetite and sleep disruption, with later improvement in subjective mood and feelings of self-worth is common. Certain delusional elements (bizarreness, effect on behavior, strength of delusional conviction, insight into delusional thoughts) may take longer to improve, with gradual recession during the course of ECT.

Traditionally, bilateral ECT has been the standard treatment for elderly patients with psychotic depression. However, recent experience suggests that high-dosage right unilateral ECT is at least as effective as bilateral ECT, with less long-term amnesia, which usually accompanies bilateral electrode placement. In the case of right unilateral ECT, high dosage is defined as treatment at least 6 times the seizure threshold. In the case of bilateral ECT, high dosage is defined as 2.5 times the seizure threshold (Figure 4).

The average number of ECT treatments given to patients of all ages in the United States in previous years for major depression was approximately 6; at present, the average is approximately 8 to 9 (possibly indicating increasing ECT treatment resistance) and the use of lower-intensity stimulation. Some depressed patients only begin to show clinical benefit after an extended ECT course, i.e., 10 to 12 treatments. Other elderly depressed patients with psychosis who do not im-
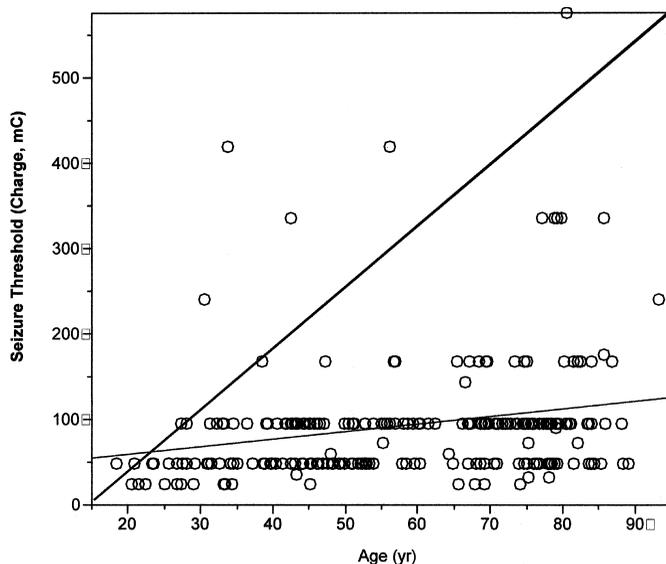

Figure 13.4. Initial seizure threshold as a function of age for 245 patients treated with right unilateral ECT (From Boylon LS, Haskett RF, Mulsant BH, et al. Determinants of seizure threshold in ECT: benzodiazepine use, anesthetic dosage, and other factors. *J ECT* 2000;16:3–18). The line at the diagonal represents the dosage patients would receive based on age-based dosing, e.g., 50% of device output for 50-year-old. The lower line is the fit of the regression of age on seizure threshold. While there is a significant relationship (r = 0.19 for raw values), there is marked variability. Dosing based solely on age provides a poor approximation of dosing needs and results in the greatest over-dosing in the oldest age patients.

prove after a standard course of bilateral ECT may subsequently show rapid improvement with extended treatment. For the elderly patient with psychotic depression who shows slow or insufficient response to ECT, the addition of a neuroleptic, especially the newer second-generation antipsychotic drugs clozapine and risperidone, may augment treatment response.[95–99]

Medical Complications and Relative Contraindications

Rates of medical complications among elderly patients during the course of ECT range from 0 to 77% for one or more complications.[20,46,54,57,100–114] This wide variability reflects different definitions of ''complication'' as well as differences in the medical status of patient samples. Nonetheless, ECT-related medical complications are considerably more likely in the elderly, particularly in the oldest age subgroups, especially among patients with reexisting medical conditions.

ECT and Medical Illness Risks

The rate of ECT-associated mortality is very low among patients of all ages (estimated as about 1 per 10,000 mixed-aged patients treated), which is comparable to mortality rates from general anesthesia in minor surgery.[5,48,115] ECT may be a safer therapeutic treatment than the older TCA medications, particularly for the frail elderly.[5,47,116] Although there are no absolute medical contraindications for ECT,[5,117] risks for the elderly increase with the following conditions:

- space-occupying cerebral lesion
- recent intracerebral hemorrhage
- increased intracranial pressure
- recent myocardial infarction with instable cardiac function
- unstable vascular aneurysm or malformation
- pheochromocytoma

Cardiovascular Illness

Cardiovascular complications are the leading cause of mortality and significant morbidity with ECT, especially for geriatric patients.[5,100,118] The peripheral hemodynamic and cerebrovascular changes during and following the brief seizure are typically well tolerated, even in the frail elderly, despite their intensity. Prophylactic use of beta-adrenergic blocking agents, such as labetalol or esmolol, lessen the hypertensive and tachycardic effects of seizure induction.[119–124] Other agents that are similarly used include nitrates[125], hydralazine[126,127] calcium channel blockers,[128–132] diazoxide,[133] and ganglionic blockers (e.g., trimethaphan).[134] In recent years, a growing number of centers routinely use propofol as the anesthesia-induction agent, rather than methohexital or thiopental, partly because propofol results in less severe hemodynamic changes.[135–147]

Conservative clinical practice should guide the use of pharmacologic modifications of standard ECT in elderly patients. In 2001, an APA Task Force Report on ECT[5] recommended fully blocking the hemodynamic changes that accompany seizure induction for all treatments in patients who are unequivocally at increased risk for complications. In patients with unstable hypertension or cardiac conditions for whom ECT is not being considered an emergency treatment, clinicians should attempt to stabilize the medical condition before beginning ECT and closely monitor cardiovascular changes during initial treatments. If sustained hypertension and/or significant arrhythmia occur following seizure induction, prophylactic medication may be used for subsequent treatments.[100]

Cognitive Side Effects

Serious short- and long-term cognitive impairment is the primary side effect of ECT in the elderly, which argues against aggressive use in this population.[5,148,149] Prior to treatment, elderly depressed pa-

tients often exhibit deficits in acquiring information, which is mostly related to disturbances in attention and concentration as indicated by tests of immediate recall or recognition-of-item lists.[150–153]

Clinically, depressed elderly patients complain of pronounced problems with attention and concentration. ECT causes a new deficit in consolidation or retention so that newly learned information is rapidly forgotten[154] due to interrupted function of the medial temporal lobe.[155–161] During and following a course of ECT, elderly patients may also display retrograde amnesia (memory for events in the past, prior to receiving ECT). Deficits in the recall or recognition of both personal and general information are usually greatest for events that occurred closest to the treatment.[162–165] Both anterograde and retrograde amnesia are most marked for explicit or declarative memory, whereas no effect is expected on implicit or procedural memory.[166–168]

Patients vary considerably both in the severity of postictal cognitive changes and in speed of recovery. Specific postictal deficits may reflect a more intense form of the amnesia observed following the ECT course. For example, the disorientation with regard to identity, place, and time seen in the postictal state has been viewed as a form of rapidly shrinking retrograde amnesia (Figure 5).[169,170] Elderly patients often ''age'' with progressive recovery from disorientation. When first asked his or her age, the 80-year old patient frequently answers to being 20 years old; with repeated questioning, the correct age is eventually given, reflecting a remarkably rapid resolution of retrograde amnesia. Similarly, patients often revert to their mother tongue on awakening and only gradually return to English. Thus the severity of postictal disorientation predicts the degree of amnesia following termination of ECT.[169] Cognitive improvement after a course of ECT follows a sequential temporal pattern. Organic mental syndromes typically resolve within 2 to 10 days post-ECT.[171]
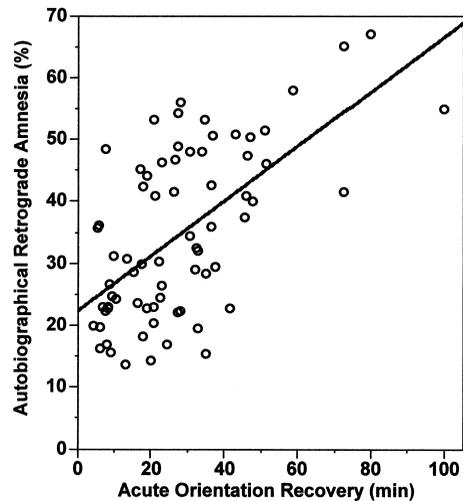

Figure 13.5. Relationship between the duration of acute postictal disorientation and retrograde amnesia for autobiographical information during the week following the ECT course. (From Sobin, et al. 1995, Reference 169.)

Recovery of cognitive function following a single ECT treatment is rapid, although in the immediate postictal period following ECT patients may manifest transient neurologic abnormalities, alterations of consciousness (disorientation, attentional dysfunction), sensorimotor abnormalities, and disturbance in the higher cognitive functions, particularly learning and memory.[148] Within several days following the course of ECT treatments, the cognitive functioning of an elderly patient slows or is typically unchanged. Occasionally immediate memory improves: change in clinical state is the critical predictor of the degree of subsequent improvements in cognition.[27,59,151,154] Following a typical course of ECT, patients of all ages often manifest a marked disturbance in their ability to retain information, reflecting ECT effects on impaired anterograde learning (the forming of new memories).[148] As the treatment series progresses, recovery of cognition in the elderly patient is often incomplete by the time of the next treatment,[20,110,170—173] causing progressive cognitive deterioration, and, in some

elderly patients, an organic mental syndrome characterized by marked disorientation.[170,174] The development of a severe organic mental syndrome often results in interruption or premature termination of ECT since patients, relatives, and clinicians are unwilling to risk further deterioration of mental status functioning.[175]

Within days of ECT termination, elderly depressed patients often manifest superior cognitive performance relative to their pretreatment baseline. Intelligence test scores for all age groups, including the elderly, may even be higher shortly after ECT relative to scores in the untreated depressed state.[148,176] More than a week or two following the end of the ECT course, differences in the cognitive effects of bilateral and right unilateral electrode placements are difficult to discern in domains other than retrograde amnesia.[27,59,148,164,165] Early evidence of improved cognition following ECT is manifested in patients' activities. After a few treatments with ECT, elderly individuals may begin to read books, attend group meetings, and become capable of following complex instructions. However, despite this improvement in attention and concentration, elderly patients still may not retain information after a brief time period. This anterograde amnesia typically resolves within a few weeks of ECT termination.[59,148] It is doubtful that ECT alone ever causes a persistent deficit in anterograde amnesia.[59,177] Not infrequently, elderly inpatients will repeatedly request information about a pass for the weekend or an expected visit from a relative whereas memory for more remote events is intact. Patients may have difficulty recalling events that occurred during treatment, and months or, in rare instances, years prior to the ECT course.[178]

Retrograde amnesia gradually disappears so that over time more distant memories, seemingly "forgotten" immediately following the treatment course, subsequently return.[163,165,177,179] However, in some patients amnestic effects of ECT persist,[27,163,165] most likely due to a combination of retrograde and anterograde effects. Patients vary considerably in the degree of cognitive impairment, regardless of how ECT is administered.

Individual Correlates of Cognitive Dysfunction

Two key clinical questions arise regarding ECT-induced cognitive impairment: (1), are there signs during the ECT course that predict which patients will develop more severe and/or persistent short- and long-term cognitive deficits and (2), can we identify the patients most at risk for severe and/or persistent amnesia prior to the start of ECT?

Over the 70-year history of convulsive therapy, numerous investigations of the technical factors that influence the degree of cognitive side effects have been conducted. Surprisingly, only in the last few years has investigation focused on the patient factors that predict the variability in these deficits. Some patients will take twice or three times as long to reorient and be capable of leaving the recovery room; others will develop an organic mental syndrome, a continuous confusional state.[170,171] Although rapid improvement in global cognitive status immediately following termination of ECT will occur, patients with prolonged postictal disorientation are likely to develop the most severe and persistent retrograde amnesia.

A range of retrospective studies indicates that patient age and medical status are also predictors of the development of persistent confusion during the ECT course.[20,57,103,110,111,113,175,180] Older patients and those with compromised medical status are most at risk for prolonged confusion during the course of ECT. Older depressed patients experience more severe anterograde and retrograde amnesia immediately following the end of ECT relative to younger patients, with some differences persisting at one-month follow-up.[181] Elderly patients with preexisting cognitive impairment, even outside the context of frank neurologic disease, are at risk for more prolonged retrograde amnesia and require appropriate modification of ECT technique to lessen cogni-

tive deficit (see Table 13.2). Global cognitive impairment seen in the depressed state also increases vulnerability for the amnestic effects of seizure induction. For example, elderly pseudodemented patients[87] often show dramatic improvement in global cognitive status during and following ECT but are at increased risk for more prolonged and deeper amnesia. Consequently, baseline cognitive impairment in the elderly depressed patient may denote a subgroup whose memory function is more fragile and likely to be affected by ECT.

Technical Administration

A variety of technical factors associated with ECT administration determine the degree and persistence of the cognitive side effects. These include the nature of electrical waveform, anatomic positioning of stimulating electrodes (electrode placement), electrical stimulus intensity, spacing or frequency of treatments, total number of treatments, duration of seizures, type and dosage of anesthetic agent, adequacy of oxygenation, and use of concomitant medications.[5,148] Table 13.2 summarizes the steps that can be taken to minimize cognitive side effects by altering ECT technique.

In recent years, sine wave stimulation has been replaced by standard brief-pulse stimulus, which dramatically reduces the acute cognitive side effects of ECT. (see Figure 6) Another recent modification, ultrabrief pulse stimulation, reduces adverse cognitive effects.[182–185] Ultrabrief pulse (0.3 ms) right unilateral ECT administered at 6 times initial seizure threshold is comparable in efficacy to standard pulse width (1.5 ms), bilateral (2.5 × ST), or right unilateral (6 × ST) ECT. In contrast, ultrabrief pulse (2.5 × ST) bilateral ECT lacks efficacy and has markedly inferior therapeutic effects than right unilateral ECT. Because ultrabrief right unilateral ECT (0.3 ms and 6 × ST) is highly effective and has a profoundly reduced side-effect profile, it is likely to become widely adopted as the ''standard'' ECT treatment.

Electrode Placement and Cognitive Dysfunction

Over the past 30 years, one of the most controversial aspects of ECT administration has been the anatomic positioning of stimulating electrodes, specifically the use of bilateral and right unilateral ECT. This debate has centered on possible differences in efficacy as well as experience suggesting that bilateral ECT accentuates long-term amnesia.[48,149,186,187] That bilateral ECT results in more profound acute and short-term cognitive impairment rather than right unilateral ECT is widely recognized.[148] In the immediate postictal period, the duration of disorientation will be considerably longer after bilateral relative to right unilateral ECT positioning.[59,169,170,188] During treatment and in the days following ECT termination, bilateral ECT will result in greater retrograde amnesia for personal and general information.[59,163–165] Anterograde amnesia—verbal memory in particular—will also be greater following bilateral ECT.[59,151,165,189] Compared to depressed patients treated with medications, patients treated with right unilateral ECT do not show greater retrograde amnesia for autobiographical information 6 months after the ECT course.[165]

Bilateral ECT is usually reserved for psychiatric or medical emergency or for medically high-risk patients for whom the number of treatments must be minimized. When bilateral ECT is administered, a switch to right unilateral ECT should be considered for patients exhibiting substantial clinical progress but unacceptable cognitive side effects. When right unilateral ECT is ineffective, increased stimulus dosage should be considered before a switch back to bilateral ECT.

Stimulus Dosing and Seizure Threshold

Three factors reliably predict seizure threshold: electrode placement, gender, and age.[60–62,190–192] In males relative to females, and in older patients, seizure threshold is higher with bilateral place-

Table 13.2.   Treatment Technique Factors and Severity of Cognitive Side Effects

Treatment Factor	Effects on Cognitive Parameters	Methods to Reduce Cognitive Side Effects	References[a]
Stimulus waveform	Sine wave stimulation grossly increases cognitive side effects	Use square wave, brief pulse stimulation	Weiner et al. (1986) Daniel & Crovitz (1983a) Valentine et al. (1968)
Electrode placement	Standard bilateral (bifrontotemporal) ECT results in more widespread, severe, and persistent cognitive side effects	Switch to right unilateral ECT	McElhiney et al. (1995) Sackeim et al. (2000, 1993, 1996) Weiner et al. (1986) Daniel & Crovitz (1983b)
Stimulus dosage	Grossly suprathreshold stimulus intensity increases acute and short-term cognitive side effects	Adjust stimulus intensity to needs of individual patients by dosage titration	Sobin et al. (1995) Sackeim et al. (1993) Sackeim et al. (1986) Squire & Zouzounis (1986)
Number of treatments	Progressive cognitive decline with high-intensity treatments (sine wave, bilateral, or grossly suprathreshold)	Limit treatments to number necessary to achieve maximal clinical gains	Calev et al. (1991) Sackeim et al. (1986) Daniel & Crovitz (1983a) Fraser & Glass (1978, 1980)
Frequency of treatments	More frequent treatments (3–5 per week) result in greater cognitive deficits	Decrease frequency of ECT	Lerer et al. (1995) McAllister et al. (1987)
Oxygenation	Poor oxygenation can result in hypoxia and increased cognitive deficits	Pulse oximetry to monitor oxygen saturation and administer 100% O_2 prior to seizure induction	APA (2001, 1990) Holmberg (1953)
Concomitant medications	High anesthetic dose may increase cognitive effects, which some psychotropics can augment	Reduce anesthetic dose to produce light level of anesthesia; decrease or discontinue psychotropic dosage; discontinue lithium prior to ECT	Mukherjee (1993) APA (2001, 1990) Small & Milstein (1990) Miller et al. (1985)

Adapted from American Psychiatric Association Task Force on ECT. The practice of ECT: recommendations for treatment, training, and privileging. Washington, D.C.: American Psychiatric Press, 2001, with permission.
[a] Complete reference citations at end of chapter.

ment than with right unilateral electrode placement. However, the combined predictive power of these features is insufficient to base choice of electrical dosage on a formula.[5,192,193] Regardless of dosage choice, patients with the highest seizure thresholds are predominantly elderly males, especially those with cardiac disease.[194] The use of ultrabrief stimulation may partially redress this issue. Since this form of stimulation is considerably more efficient, seizure thresholds are much reduced, allowing greater effective range for dosing relative to threshold.

The efficacy of right unilateral ECT is especially sensitive to electrical dosage. When stimulus intensity is near the seizure threshold, right unilateral ECT lacks
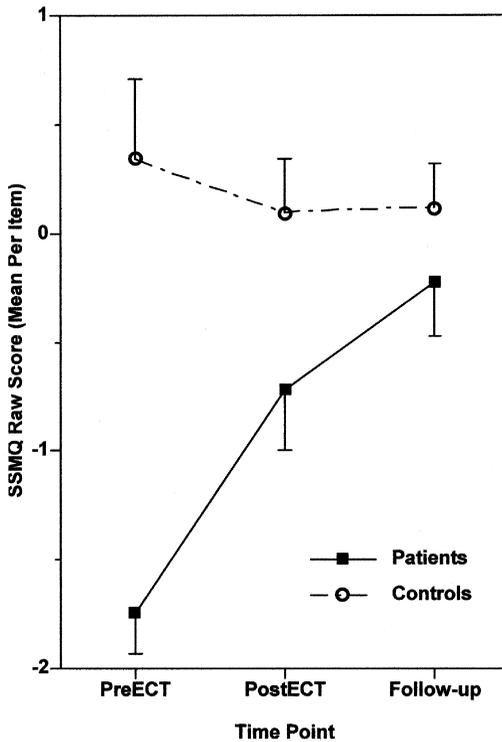
Figure 13.6. Score on the Squire Subjective Memory Questionnaire before and after the treatment course in ECT responders and nonresponders. Scores of '0' indicate no change in memory relative to before the episode of depression. (From Coleman EA, Sackeim HA, Prudic J, et al. Subjective memory complaints before and after convulsive therapy. *Biol Psychiatry* 1996;39:346–356).

therapeutic properties.[58,59] Since advanced age correlates with higher seizure threshold, older patients are less likely to benefit from standard electrical dosage.[60,62,190,191,195] The efficacy of right unilateral ECT improves with escalation of intensity of electrical stimulation relative to seizure threshold.[27,59] At markedly suprathreshold dosing (e.g., 6 times the initial seizure threshold), right unilateral ECT achieves an efficacy that is equivalent to that of robust forms of bilateral ECT (e.g., 2.5 times the initial seizure threshold).[27,59] Even at grossly suprathreshold stimulus intensities, right unilateral ECT retains significant advantages with respect to cognitive parameters.[27] The high sensitivity of geriatric patients to the cognitive side effects of bilateral ECT suggests that suprathreshold forms of right unilateral ECT should be routine in this population. Indeed, given the marked cognitive benefits of ultrabrief stimulation, optimal treatment might involve dose-titrated, ultrabrief stimulation, using markedly suprathreshold right unilateral ECT.

Efficacy, speed of response, and cognitive side effects of ECT depend on the degree to which the ECT stimulus exceeds the seizure threshold. Suprathreshold dosing will improve the efficacy of right unilateral ECT, enhance speed of clinical improvement with right unilateral and bilateral ECT, and will result in more severe acute and short-term cognitive impairment.[59,169,188,196]

Number and Schedule of Treatments

Cognitive effects of ECT are proportional to the frequency with which treatment is administered as well as to the total number of treatments given.[197–199] This is particularly true when the most intense form of ECT, suprathreshold, bilateral treatment is used. The most common schedule in the United States involves 3 treatments per week, whereas in England, 2 treatments per week is more common. The U.S. schedule results in more rapid improvement but increased short-term cognitive impairment.[198,200,201] Elderly patients may be more sensitive to the

frequency and number of treatments.[20,110,172,188,202] Some clinicians reduce the frequency of treatment to twice weekly in elderly patients who show progressive clinical improvement but excessive cognitive deficit.

Concomitant Medications and Cognitive Effects

Evidence suggests that the dose of anesthetic agent may contribute to the severity of cognitive impairment during the postictal recovery period.[203] Not surprisingly, excessive anesthetic dose may result in prolonged postictal disorientation. For this reason, older patients should receive lower doses of anesthetic agents than younger patients. This is particularly important since the dose of the anesthetic may also alter seizure duration and intensity.[60,204]

A small dose of a muscarinic anticholinergic agent (0.4–0.8 mg atropine or 0.2–0.4 mg glycopyrrolate) is commonly administered intravenously in ECT, just prior to the anesthetic agent. The anticholinergic agent serves to block vagal outflow and limit the bradycardia produced by the ECT stimulus. This is especially necessary whenever the possibility of subconvulsive stimulation exists, or when patients are administered a β-blocker.[100] Atropine is preferred to glycopyrrolate since protection against bradycardia is less certain with glycopyrrolate. In addition, incidence of postictal nausea is also higher with glycopyrrate,[205,206] and glycopyrrolate holds no advantage with respect to cognitive effects during ECT.[205–211]

In sensitive elderly patients, a variety of psychotropic agents may intensify the adverse cognitive effects of ECT. Lithium carbonate, for example, causes acute confusion during ECT in approximately 1 in 15 patients; more rarely, status epilepticus occurs.[212–216] Lithium should be discontinued prior to the start of an ECT series, or it can be withheld the night and morning before an ECT treatment. In the elderly, concurrent use of benzodiazepines, neuroleptics, or other sedating psychotropic agents may increase cognitive side effects. Benzodiazepines and anticonvulsant medications may also interfere with efficacy by raising seizure threshold.[217,218]

ECT for Depressed Patients with Neurologic Disorders

Increasingly, ECT is used to treat psychiatric manifestations in a variety of patient populations with frank neurologic illness, including Parkinson's disease,[219–225] poststroke depression,[226–228] and to a lesser extent, dementing disorders.[229,230] Across a variety of neurologic disorders, ECT is effective in the treatment of primary or secondary mood disorders. In the case of Parkinson's disease, ECT frequently exerts beneficial effects on aspects of the movement disorder and has been used as a primary treatment for the neurologic condition. Duration of the antiparkinsonian effects is, however, unpredictable; some patients lose benefit within days, while others maintain improvement in the movement disorder for months or longer.[231] The role of continuation or maintenance ECT in sustaining improvement in the movement disorder is largely undocumented, although clinical experience indicates that such long-term treatment can be highly effective. Patients with Parkinson's disease who receive ECT may be at increased risk for prolonged confusion or delirium.[232,233] ECT is also effective in treating poststroke depression and major depression in the context of dementing illness,[229,230] although there is risk of increased severe cognitive side effects.

CLINICAL VIGNETTES

Case 13.1

Ms. A., a 71-year-old married retired schoolteacher, was seen in consultation regarding a serious chronic depression. Over the course of her adult life, she had suffered many such depressions that were always characterized by extreme anergia, anhedonia, and a sense of

pointlessness. Ms. A. was never psychotic and never suicidal, but her diminished energy would often reach such severe proportions that she was unable to get out of bed for long periods during the day. She maintained her weight by forcing herself to eat, but no longer enjoyed the preparation or taste of food or, indeed, anything else. What was most striking to her was her subjective loss of interest in her grandchildren. Untreated, these depressive periods could last up to one year. Typically, however, although Ms. A. would begin to feel some symptomatic relief after a few months, a chronic pessimism and dysphoria persisted even in the absence of serious depressive symptoms (double depression).

Over the course of her life, Ms. A. had been treated with at least one antidepressant from each class of medication and had responded at least once to each. She had successful trials of imipramine, fluoxetine, venlafaxine; her response to monotherapy with nefazodone, mirtazapine, and bupropion was nontherapeutic. She never took an MAO inhibitor and never had lithium augmentation.

At the time she presented for evaluation, Ms. A. was in a state of profound melancholic, nonpsychotic depression. Out of desperation, she requested a consultation regarding ECT and agreed to a course of treatment. She initially received 2 bilateral treatments, and then was given 4 more treatments applied to the unilateral nondominant hemisphere on a thriceweekly basis. Ms. A.'s response was rapid and dramatic. After the first 2 treatments, she no longer remained in bed and began actively to participate in family life. By the sixth treatment she proclaimed herself to be ``back to normal.'' Based on evidence regarding high rates of relapse following ECT, Ms. A. was placed on a low dose of lithium carbonate (600 mg; blood level 0.4) for maintenance. She remained depression-free at one-year followup.

This case illustrates the importance of considering this most useful antidepressant treatment even for older patients who have had chronic depression over the course of a lifetime. Her treatment also illustrates the usefulness of post-ECT lithium maintenance to prevent relapse. Although Ms. A. experienced impairment of recent recall for the period during and prior to the ECT, she reported that this memory loss was a small price to pay for the dramatic improvement in her mood. Like other older patients whose depression has responded to ECT, she also indicated that the quality of the response to treatment was better than that from chemical antidepressants. She stated that her mood and thinking felt ``clearer'' following the ECT

during the memory loss. This is consistent with observations of clinicians experienced with use of ECT who have noted that some patients' response to ECT does seem to produce a better remission than chemical antidepressants, which may produce only a response or a partial response.

Case 13.2

Mr. B., a 76-year-old widowed attorney, developed a classical syndrome of severe major depression with melancholia. His first symptoms of depression appeared at the age of 73 after partial retirement from his law firm. Treatment with desipramine 85 mg daily (blood level of 140 ng/mL) led to dry mouth and mild urinary hesitancy; intravenous pyelogram revealed no significant residual urine. After approximately 4 weeks on desipramine, Mr. B.'s symptoms of depression remitted, although a feeling of ``mild uneasiness'' remained. He was able to return to work for a few hours a week and resumed most of his social activities. Approximately 3 months after the initial response, Mr. B.'s ``uneasiness'' intensified and became particularly prominent in the morning. Finally, depressed mood and feelings of hopelessness as well as insomnia and appetite loss developed over a period of 2 months despite maintenance therapy with desipramine together with supportive psychotherapy.

Severe exacerbation of his depressive symptoms followed some changes in Mr. B.'s law firm. Desipramine dosage was raised to a blood level of 182 ng/mL; later, thyroid augmentation was attempted with triiodothyronine (up to 50 mg daily) for 2 weeks. Because no change in his mental status occurred, triiodothyronine was discontinued, and lithium augmentation was attempted, with dosage gradually increased to 600 mg daily (blood level of 0.75 mEg/L). Depressive symptoms were ameliorated approximately 3 weeks after the introduction of lithium, but he developed tremor and unstable gait that required reducing the dosage to 300 mg daily (blood level of 0.44 mEg/L). Mr. B. remained partially symptomatic with mildly anxious and depressed mood, particularly in the morning, with early morning awakening and complaints of poor concentration.

Approximately 2 months after the improvement induced by lithium, Mr. B.'s depression worsened severely; suicidal ideation developed, and he was hospitalized in a geriatric psychiatry unit. Psychotropic drugs were discontinued, and 10 unilateral ECTs were administered, resulting in complete remission of his depression. Sertraline, 50 mg daily, was started immediately

after the last ECT and increased to 75 mg 5 days later. This drug was chosen because the tricyclic antidepressant desipramine had failed to maintain Mr. B.'s remission. However, 3 weeks after the last ECT, Mr. B. began again to experience depressed mood, early morning awakening, and suicidal ideation. Sertraline was discontinued a week later, and three additional unilateral ECT treatments were administered, with excellent response. Although therapy with MAOIs was considered, maintenance ECT was chosen because his rapid development of suicidal ideation and lack of supervision after discharge placed him at risk. Compliance with MAO diet was also a concern, especially during the period after ECT when his memory was impaired. Maintenance ECT was given every 2 weeks during the first 2 months and then monthly. Nine months after completion of the initial ECT trial, Mr. B. was still asymptomatic.

Some depressed geriatric patients respond well to antidepressant treatment but cannot sustain remission despite continuation therapy with antidepressant drugs. Mr. B.'s major depression with onset in late life responded favorably to desipramine, desipramine combined with lithium, and a trial of ECT at various times. However, approximately 1 to 3 months after initial improvement, his depression returned, necessitating additional antidepressant treatment. Patients like Mr. B. often are difficult to treat, particularly if they cannot tolerate particular antidepressants or therapies. ECT is usually effective in such cases and should be considered, especially when the patient becomes disheartened by the repeated failures. The rollercoaster of hope and disappointment, coupled with the pessimism of the depressive syndrome, may cause the patient to give up and facilitate development of suicidal ideation.

Maintenance ECT needs further investigation. Many patients, however, remain in remission from depression while receiving ECT every 4 to 6 weeks. ECT appears to be a reasonable option for patients with severe depression who fail to remain in remission while on an adequate dosage of a heterocyclic antidepressant, a serotonin-reuptake inhibitor, or an MAOI. Only depressed patients who are able to tolerate and respond to a trial of ECT should be considered for maintenance ECT.

Case 13.3

Mrs. C., a 76-year-old widow, was diagnosed with Alzheimer's disease. Although her dementia was moderate, she was still able to function in her own home with a 24-hour companion.

After a fall, Mrs. C. sprained her right ankle, and her mobility decreased for 2 to 3 weeks. During this time, she became apathetic, lost interest in television or socialization, and developed insomnia and appetite loss. After a diagnosis of depression, imipramine was begun, with dosage increased by 25 mg every other day up to 75 mg daily. After 6 days on imipramine 75 mg, Mrs. C. developed agitation, confusion, inability to sustain her attention, and incoherent speech. Her symptoms were significantly worse at night; she appeared frightened and kept saying that her neighbors were coming to ``put her away.'' Her face was flushed, her skin dry, and her pulse was 120 beats per minute.

Mrs. C. was admitted to an acute psychiatric unit with a diagnosis of anticholinergic delirium. Imipramine was discontinued, a course of hydration was begun, her vital signs were monitored closely, and her pulse decreased to 95 beats per minute within 24 hours. Three days later, her confusion and agitation lessened, but the symptoms of depression were even more apparent. She gradually developed severe psychomotor retardation and began refusing to eat or drink. Treatment with desipramine, 10 mg daily, began and was increased by 10 mg every 3 days. A week later, Mrs. C. required tube feeding. At 40 mg of desipramine daily, her pulse rate ranged between 100 and 110 beats per minute. At this point, desipramine was discontinued, and 5 days later unilateral ECT was begun. ECT was administered twice a week, and after a total of eight treatments, Mrs. C.'s depression was in complete remission.

Elderly patients are sensitive to the anticholinergic effect of heterocyclic antidepressants. Delirium, persistent sinus tachycardia, or urinary retention often lead to discontinuation of these drugs. When the diagnosis of anticholinergic delirium is in doubt, physostigmine 1 mg diluted in 10 mL of normal saline should be administered intravenously over 5 to 7 minutes. The mental status of patients with anticholinergic delirium improves almost immediately. However, physostigmine should be avoided for very old patients or those with cardiac disease because it may cause sinus brachycardia or transient sinus arrest. Patients with bronchial asthma may develop bronchospasm after administration of physostigmine.

ECT is the treatment of choice in a rapidly worsening depressed elderly patient. Although ECT-induced memory dysfunction may be more severe and prolonged in demented than in nondemented patients, there is no evidence that ECT worsens the course of dementia.

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