American Psychiatric Association

TASK
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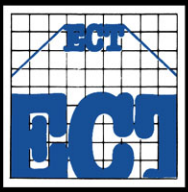

ELECTROCONVULSIVE THERAPY

Task Force Reports

This is the fourteenth in a series of reports approved by the Board of Trustees of the American Psychiatric Association to give wider dissemination to the findings of APA's many commissions, committees, and task forces that are called upon to evaluate the state of the art in a problem area of current concern to the profession, to related disciplines, and to the public.

The findings, opinions, and conclusions of the report do not necessarily represent the views of the officers, trustees, or all members of the Association. Each report, however, does represent the thoughtful judgment and findings of the task force of experts who composed it. These reports are considered a substantive contribution to the ongoing analysis and evaluation of problems, programs, issues, and practices in a given area of concern.

Jules H. Masserman, M.D.
President, APA, 1978-79

September 1978

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ELECTROCONVULSIVE THERAPY

Report of the Task Force on Electroconvulsive Therapy
of the American Psychiatric Association

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INTRODUCTION

This report on Electroconvulsive Therapy contains few simple solutions. We will be pleased if it helps to lead to a rational perspective.

An examination of the published literature since the early 1940s reveals the mixed reception accorded ECT since its introduction in 1938. Attitudes surrounding it capture the essence of the struggle between the rival philosophical approaches to emotional and mental illness, namely the psychological, the biological, and the social.

We believe that what follows in this report affirms that the last word has yet to be written on the etiology and treatment of psychiatric illness, as well as on the most useful role for government and the courts in attempting to prevent or manage it. Those who disagree have misled us in the past by exaggerating a single viewpoint; they will no doubt attempt to do so again.

In the fall of 1974, the Council on Research and Development resolved to establish a task force on ECT "with tenure of about two years, to have liaison with the Commission on Judicial Action and to include in its study consideration of legislative issues, consent, indications for its use and possible increasing use because of shortened length of stay in hospital."

The Task Force assembled for the first time in September 1975, with the following mandate approved by the Council:

Mandate to the APA Task Force on ECT

The task force is enjoined to:

(1) Review the current use of ECT, both typical and atypical, the prevailing criticisms of its use, and the recent and current research on the subject with special but not exclusive reference to studies of:
   (a) its indications in different clinical entities;
   (b) its benefits in relation to its adverse effects;
   (c) treatment techniques designed to minimize adverse effects, particularly memory loss and confusion;
   (d) the effectiveness of associated drug therapy;
   (e) associated neurochemical and neurophysiological changes;
   (f) long term effects, both beneficial and adverse;
   (g) comparisons with other treatment methods;

(h) comparable treatments with flurothyl;
(i) associated cerebral and behavioral events;
(j) induced seizures and convulsive shock in animals.

(2) Report its conclusions regarding the benefits of ECT in relation to the risks involved.
(3) Describe the indications for its use and the contraindications.
(4) Make recommendations regarding:

(a) general clinical considerations;
(b) pretreatment investigations and precautions;
(c) the use of anesthesia and related measures to reduce the dangers of treatment;
(d) techniques with reference to apparatus, schedules of administration including those of multiple and single treatments, appropriate numbers of treatments, location of electrodes, and after-care procedures;
(e) the simultaneous use of drug therapy;
(f) criteria of improvement and indications for the termination of treatment.

(5) Consider the civil liberties of patients and informed consent in the management of treatment programs.
(6) Comment and advise on related legislative issues.
(7) Recommend directions for future research and study.
(8) Report on any other issues it considers relevant.

In all, the Task Force held five two-day meetings through the two years, as well as an evening meeting and a two-day open forum at the time of the annual meeting in Miami in 1976.

Because the published literature, although considerably more helpful in recent years than previously, still provides only a limited number of well controlled studies, it was thought essential that the empirical evidence available be evaluated and given serious consideration. The members therefore decided on exploring four main avenues in order to accumulate the information needed by them to discharge their mandate. The methods selected were:

(1) a search of the literature;
(2) a national survey of a 20% random sample of the approximately 20,000 APA members to provide data on the extent of the use of ECT, the indications for its use, the methods and techniques in general use, and the general experience of its

effectiveness; these data were to provide the context within which to consider the questions raised in the mandate;

(3) an open forum for the discussion of ECT at the annual meeting in 1976. As not more than 20% of the membership would be polled in the national survey, an opportunity was to be provided for members generally to report on their views and experience to the members of the Task Force if they so wished. Announcements in *Psychiatric News* extended an invitation to the membership to arrange for an appointed time at which they could address the Task Force. Twenty four individuals had an opportunity to communicate personally in this way with the committee. Invitations were extended to a few highly experienced clinicians to share their views with us, as well as to two colleagues in neurology known to hold strong views against the use of ECT. A few of these people accepted the invitation and addressed us;

(4) data on the use of ECT at a selected series of psychiatric hospitals.

In addition to these planned sources of information, the Task Force attracted a large amount of correspondence, much of it helpful, from local surveys, psychiatric facilities, clinicians, and ex-patients.

All the information gathered has been considered by the members of the Task Force in the light of our joint but varied experience and applied to the questions that have to be addressed.

In the body of the report, we will commence with a summary of the data retrieved from the nearly 3,000 questionnaires returned by the polled membership; this will be followed by a series of chapters written by individual members of the Task Force, providing the material that supports the recommendations with which we conclude the report. In some instances the content of the chapters is detailed and technical, buttressed by a relevant review of the published literature. Although written by the individual members, the chapters have been reviewed by the Task Force, in some instances through several drafts, and reflect a consensus of the members. The topics covered in the chapters include:

data on the practice of ECT derived from the survey;
the efficacy of ECT in affective disorders and schizophrenia;
ECT in mental illness other than schizophrenia and the affective disorders;
adverse effects;
methods of administration;
physiological and biochemical concomitants of ECT;

social, ethical and legal aspects of ECT;
training and education;
areas requiring further study;
recommendations.

The recommendations are presented at the conclusion of the report. Those interested primarily in that section can proceed directly to it, referring to the supportive data of the individual chapters as they see fit. Others might prefer to become acquainted first with the reasoning in the sequence of the chapters. The recommendations have been carefully considered in detail by all the members of the Task Force. Our wish to avoid restrictions on clinical practice was more than balanced by our recognition of the fact that at this time in our history where the profession fails to provide its own guidelines, they will be created by others who may be less well-equipped to do so. We were encouraged in our resolve by almost two thirds of the questionnaire respondents who felt there was a need for more explicit guidelines for the proper use of ECT.

We are especially hopeful that the report and its recommendations will be viewed as an educational document. The procedures described and recommended include a model of ECT administration that is close to ideal. We recognize that there are other equally safe and effective procedures that differ in one or more aspects from those described here. Although no one is compelled to follow our model in all its aspects, we include it in the hope that those who administer ECT will examine it in a scholarly manner, prepared to be influenced by its recommendations if it appears that these will help improve the current standards of practice.

Before proceeding to the body of the report, we wish to draw attention to issues that we believe merit the closest attention of those interested in the subject. The report itself will elucidate them further:

(1) ECT in the 1970s, administered by competent clinicians, is vastly different from the procedures used and described in the preceding 30 years. Problems and confusion result from a failure to make this differentiation.
(2) There is no objective reason to couple ECT with psychosurgery or to compare the two treatment methods. Apart from the demonstrably irreversible changes that follow psychosurgery, which have not been shown in ECT, psychosurgery is an experimental procedure while ECT has been shown to be an effective therapy in well-selected cases; indications for their use and their modes of action differ.
(3) The Task Force, keenly aware of the importance of well-

controlled studies, has sought throughout its deliberations to integrate reasonable clinical practice and experience into its findings and recommendations.

(4) Recognizing the constant growth of knowledge and the likelihood of changing viewpoints in the future, we have recommended the establishment of an APA Committee on ECT which will act as a clearinghouse for new information and in an advisory capacity to the Association.

(5) Cognizant of the effect of language, the Task Force is concerned that the terms "shock therapy" and "to shock" are neither appropriate nor helpful descriptions of the treatment and elicit unnecessary anxiety in patients and families. We recommend that the use of such terms be discouraged. The treatment has undergone many changes, and while the muscular convulsion is no longer essential to the treatment and "convulsive therapy" is therefore not descriptive, we are recommending the terms "convulsive therapy" and "ECT" until a better name is formulated.

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CHAPTER I

A SURVEY OF THE AMERICAN PSYCHIATRIC ASSOCIATION MEMBERSHIP ON THE USE OF ELECTROCONVULSIVE THERAPY

In the process of developing its position on ECT, the Task Force felt that it would be useful to obtain systematically solicited input from the membership of the American Psychiatric Association (APA) regarding their attitudes toward and experiences with this treatment modality.

A survey of nearly 3,000 members of the APA was conducted by the Task Force to determine attitudes toward ECT, use of the treatment, and perceptions of the results obtained.

The questions to be answered from the survey data may be summarized as follows:

(a) For all psychiatrists responding:
  (1) What are their attitudes toward and beliefs about ECT?
  (2) For which diagnostic categories/problem behaviors do they feel that ECT is most appropriate?
  (3) What percentage use ECT?
  (4) To what extent is the use of ECT related to member characteristics (age, sex, certification, etc.), theoretical orientation to psychopathology, and attitude toward ECT?

(b) For those psychiatrists who use ECT:
  (1) With what frequency is each of the currently available techniques of administration used? In what settings?
  (2) What are the primary reasons for using ECT for each of the major diagnostic categories?
  (3) What are user perceptions of the efficacy of ECT for each of the major diagnostic categories and of the frequency of adverse effects?

Method

Respondents

Questionnaires were mailed to 4,013 randomly selected members of APA, 20% of the membership with addresses in the United States,

exclusive of Members-in-Training (residents), Inactive and Corresponding Members and Fellows, and Honorary and Distinguished Fellows. Some response was received to 3,049 (76%) of these questionnaires. In addition to partially completed and late questionnaires, responses were received saying that members had died, were too ill to practice, had had no recent experience with ECT, etc. Questionnaire data for 2,973 members (74.1% of the target sample) were punched on data processing cards and are included in the analysis reported here. Judged by the usual experience in mail survey research, the response rate must be considered excellent despite the length of the questionnaire. Two followup mailings of the questionnaire were made to those who had not already responded.

Questionnaire

A fixed alternative questionnaire of 140 items on 12 pages was used. This instrument was divided into two major sections: Section I for all respondents and Section II for those who reported using ECT. Section II was subdivided into sections dealing with (a) technique of administration, (b) patient selection, and (c) treatment outcome. A copy of the questionnaire appears in Appendix II.

Results

Sample Characteristics

The average age of the sample was 48 years; 88% were male; 67% had graduated from U.S. medical schools; 93% had three or more years of psychiatric residency; and 66% were board-certified in a medical specialty, predominantly psychiatry. Forty-nine percent characterized their predominant theoretical orientation to psychopathology as eclectic, 37% as psychological, psychoanalytic, and 5% or less fell into the remaining categories of organic, biochemical; organic, neurological; social/community; and behaviorist. Forty-nine percent were in private practice; 17% were in clinical practice in a public or private mental hospital; 15% were in clinical practice in other institutional settings; and 7% or less were in each of the six remaining categories of "primary work." Fifty-four percent practiced in very large urban areas and only 2% practiced in rural areas.

Attitudes/Beliefs about ECT

To obtain a measure of overall evaluation of ECT, the following item was included:

"Granting that the question is a gross over-simplification, which of the following *best* characterizes your attitude toward the use of ECT"

	Obtained response
1. Totally opposed to its use	2%
2. Generally opposed, but O.K. as a last resort in a few selected instances	22%
3. No really strong feeling, but tend to be more opposed than favorable	8%
4. Ambivalent; undecided	1%
5. No really strong feeling, but tend to be more favorable than opposed	6%
6. Generally favorable for appropriate patients	54%
7. Decidedly favorable to its use	7%

Thus, we see that 32% expressed some degree of opposition; one percent, ambivalence; and 67% some degree of favorable attitude.

Responses to more specific statements about ECT were distributed as follows (the difference to 100% reflecting response of no opinion/ambivalent/undecided):

	% Agree	% Disagree
1. There are many patients for whom ECT, either alone or in combination with other measures, is the safest, least expensive, and most effective form of treatment	72%	20%
2. Any psychiatric institution claiming to offer comprehensive care should be equipped to provide ECT	83%	12%
3. ECT should be used only when all else has failed	38%	57%
4. The introduction of antidepressants and phenothiazines has made the use of ECT obsolete	7%	87%
5. The use of ECT should be discontinued or at least should be curtailed	16%	75%

6. It is likely that ECT produces slight or subtle brain damage	41%	26%
7. There is a need for more explicit guidelines (perhaps from APA) for the proper use of ECT	69%	20%
8. The issuance of guidelines from any source for the use of ECT is likely to interfere with good patient care	22%	65%
9. ECT should not be administered to children 16 or under	57%	16%

Large percentages of the respondents feel that ECT is a valuable treatment technique and a majority would welcome explicit guidelines for its use.

Appropriate diagnosis/problem

Respondents were asked to rate the degree of appropriateness of ECT (assuming no physical contraindications) for 11 diagnoses/problems. Collapsing the six-point scale into "appropriate," "undecided" and "not appropriate," the following results were obtained (the difference to 100% reflecting rounding error and those who indicated opposition to the use of ECT for all patients):

	Appropriate	Undecided	Not Appropriate
Minor (non-psychotic) depression	6%	2%	88%
Major depression	86%	6%	7%
Schizophrenia (acute or chronic)	25%	15%	59%
Manic excitement	42%	13%	43%
Drug or alcohol abuse	1%	2%	94%
Personality disorders	2%	1%	93%
Sexual dysfunction	1%	1%	93%
Anorexia nervosa	11%	17%	70%
Intractable pain	8%	18%	72%
Unremitting hypochondriasis	11%	17%	70%
Toxic dementias	2%	3%	91%

In addition, 274 respondents (9% of the total sample) indicated ECT as appropriate for a variety of conditions which they filled in the space for "Other, please specify—." Correspondingly, 108 (4%) respondents went to the effort of specifying a variety of conditions for which they considered ECT inappropriate.

The preceding table provides reassuring evidence that the appropriateness ratings for most respondents are consistent with generally accepted practices and the available evidence regarding efficacy of ECT. On the other hand, it is evident that a small percentage of respondents view ECT as appropriate for conditions for which there is little evidence of efficacy and where the weight of professional opinion favors the use of other treatments. These points are further dealt with in Chapter III.

Percentage of Respondents Using ECT

After excluding 9% of respondents who indicated they did not spend at least 50% of a usual working week (35 or more hours) in activities related to psychiatry and/or neurology, the remaining respondents were categorized as Users or Non-Users according to whether or not they had either (a) personally treated patients with ECT in the last six months, or (b) recommended to residents under their supervision that ECT be used on patients in the last six months. According to this definition, 22% fell into the User category. Sixteen percent had personally treated at least one patient (within the past six months), and 11% had recommended that residents use ECT. Naturally, these two groups were not mutually exclusive.

Characteristics Distinguishing Users from Non-Users

The following member characteristics were cross-tabulated with the User vs. Non-User categorization: age, sex, attendance at U.S. vs. foreign medical school, certification status, type of practice, site of practice, geographical location of practice, theoretical orientation to psychopathology, and attitude toward ECT. It was found that a greater percentage of males than females (24% vs. 12%, respectively) were Users. Likewise, psychiatrists in clinical practice in mental hospitals, those in group private office practice, in private psychiatric hospitals, and those in large or medium-sized cities tended to be Users more than did members of other groups.

Of the eight "theoretical orientation" groups considered (organic, biochemical; organic, neurological; psychological, psychoanalytic; psychological other than psychoanalytic; social/community; behaviorist; eclectic; other), the Users more frequently characterized themselves as organic or eclectic. Further, the Users differed from the

Non-Users on all the attitude items listed above, in each case in the direction that would be expected. Thus, for example, Users agreed less often than did Non-Users that "ECT should be used only when all else has failed."

Techniques of Administration

The questionnaire contained items dealing with the use of anesthesia, muscle-relaxant drugs, type of machine, current used, and several other technique-related issues. Of the Users, 95% reported generally using a short acting anesthetic drug before administering ECT; 60% of these reported having had formal training in such administration. Sixteen percent reported never using the services of an anesthesiologist or nurse anesthetist, while 59% always used such services. Ninety-six percent reported that all of their ECT patients (during the preceding six months) had been given a muscle-relaxant drug before ECT was administered.

In response to a question about the type of machine generally used (Medcraft, Reiter, Mecta, other), 62% indicated Medcraft; 33% Reiter; and 1% Mecta. Type of current generally administered was distributed as follows:

Bipolar sine wave	69%
Unidirectional	16%
Pulsed unidirectional	4%
Pulsed bipolar	9%
Other	2%

Bilateral electrode placement was the most common technique used; 75% of the Users reported using it with all of their patients, followed in frequency by right unilateral, combined bilateral and unilateral, and left unilateral.

In regard to number of seizures per treatment session, 71% reported administering only one; 25% occasionally administered more than one; and 4% often or always administered more than one. For those who used more than one seizure per session, by far the most common number was two. The most common treatment schedule was three times per week (reported by 82% of the Users). In response to a question about the optimal number of treatments in a single course, the average recommendation for schizophrenic patients was 12; for major depressions, ten; and for minor depressions, four.

Over a third (35%) of respondents indicated they used mainte-

nance ECT. However, the number of patients being continued on ECT was relatively small, averaging out to 6% of all ECT patients so treated. When the average was recomputed excluding those who did not use maintenance ECT, it rose to 16%. That is, for those psychiatrists who reported using maintenance ECT at all, they reported using it for 16% of their total ECT patient load. The most common schedules for maintenance ECT were once a month (41%) and no fixed schedule (38%).

Opinions about the combined use of ECT and psychoactive drugs (antipsychotic agents and tricyclic antidepressant drugs) were distributed as follows:

Psychoactive medication. . .

Should almost always be stopped	8%
Should usually be stopped	22%
It probably makes little difference	16%
Should probably be continued	41%
Should almost always be continued	13%

While opinions varied, it can be seen that the weight of the responses was in the direction of continuing psychoactive drugs. In addition, an item was asked about the possible incompatibility of administering ECT to patients on five types of drugs. The percentages indicating such incompatibility were as follows:

	% considering drug incompatible with ECT
Lithium	20%
Reserpine	63%
Phenothiazines	19%
Antidepressants	16%
Thiazides	20%

Only 17% thought that ECT was contraindicated for elderly, depressed patients with cardiopulmonary problems who are receiving antihypertensive medication. This latter item was included in an attempt to posit a situation where many psychiatrists might be unwilling to use ECT; the response to this item suggests that the proportion of such psychiatrists is actually fairly low.

Respondents reported that 97% of their ECT treatments were administered in institutional settings accredited by the Joint Commis-

sion on Accreditation of Hospitals and that, on the average, 72% of ECT treatments were paid for by insurance companies.

Selection of Patients

Reasons for Use

It is difficult to write good items about, or for that matter for respondents to explicitly formulate their reasons for the use of ECT. This caveat notwithstanding, it will be seen that useful information was obtained regarding stated reasons for ECT use. The following question set was posed separately for schizophrenic patients, patients with major depressive illness, and patients with minor depressive illness. The response categories and average percentages for the three diagnostic groups were as follows:

> In approximately what percentage of ——— patients you treated with ECT in the past six months was ECT selected for each of the following reasons:

	Schizophrenia	Major Depression	Minor Depression
	Average percent		
It was the treatment of first choice (given either with or without psychoactive drugs)	8%	19%	3%
ECT was used in combination with other treatment methods with the expectation that its addition would yield a better quality of improvement	22%	23%	12%
Medication in large daily dosages (at least 1000 mg of chlorpromazine, 300 mg of imipramine or their equivalent) was tried and found ineffective	33%	40%	18%
Medication in smaller dosages was tried and found ineffective	11%	13%	8%

	ELECTROCONVULSIVE THERAPY		
	Schizophrenia	Major Depression	Minor Depression
	Average percent		
Patient was allergic to medication and/or medication led to side effects	4%	7%	3%
Psychotherapy (individual and/or group and/or family) was ineffective	7%	8%	6%
Large dosages of medication *and* psychotherapy were ineffective	46%	39%	37%
Smaller dosages of medication *and* psychotherapy were ineffective	(not asked)	10%	13%

These average percentages (based on patients treated by each User and irrespective of differences in absolute numbers of patients treated) suggest that ECT was not often used until other treatment approaches had been tried and found ineffective.

Another way of looking at the same issue is to refer to the number of separate individuals who responded positively to each of these eight indications. The following table reports how many individual Users cited the particular reason as having been an indication for ECT treatment for any of their patients so treated.

	Schizophrenia	Major Depression	Minor Depression
It was the treatment of first choice (given either with or without psychoactive drugs)	71	235	17
ECT was used in combination with other treatment methods with the expectation that its addition would yield a better quality of improvement	151	198	53

Medication in large daily dosages (at least 1000 mg of chlorpromazine, 300 mg of imipramine or their equivalent) was tried and found ineffective	167	309	64
Medication in smaller dosages was tried and found ineffective	68	104	29
Patient was allergic to medication and/or medication led to side effects	62	116	29
Psychotherapy (individual and/or group and/or family) was ineffective	45	62	31
Large dosages of medication *and* psychotherapy were ineffective	215	284	102
Smaller dosages of medication *and* psychotherapy were ineffective	(not asked)	86	41

Examination of the two preceding tables suggests that (1) ECT is only relatively rarely the treatment of first choice—*even in patients with major depressions;* (2) ECT is overwhelmingly used only when medication, usually in large dosages and often in combination with psychotherapy, has been ineffective; (3) ECT is fairly often used in combination with other treatment methods in hopes that the combination of therapies will yield a better quality of improvement than either the ECT or the other methods alone would be expected to yield; and (4) the use of ECT in instances where the patient is allergic to medication and/or medication has unwanted side effects is by no means frequent, but neither is it a rare event.

Frequency of Use by Diagnosis/Behavior.

Respondents were asked to indicate the approximate percentages of all of their ECT treated patients in the preceding six months who

fell into each of 11 categories of pathology. These categories, ranked by the average percentages, were as follows:

Major depressive illness	77%
Schizophrenia	17%
Minor depressive illness	5%
Manic excitement	3%
Unremitting hypochondriasis	<1%
Anorexia Nervosa	<1%
Drug or alcohol abuse	<1%
Intractable pain	<1%
Personality disorder	<1%
Toxic dementia	<1%
Sexual dysfunctions	<1%

On the average, and across all respondent users, 77% of all ECT treated patients had major depressions, 17% were schizophrenics, etc. As expected, the great bulk of ECT usage is in major depressive illness. Nevertheless, the data indicate some administration of ECT, albeit very rarely, for conditions for which ECT is not commonly considered appropriate.

Treatment Outcomes

Therapeutic Results

Respondents were asked to estimate the percentage of their ECT patients (treated in the last six months) who were at least moderately satisfied with the results of their treatment. Forty-five percent said that all their patients were at least moderately satisfied. The average report was that 88% of patients reported being satisfied to that degree. The report of the extent to which the psychiatrists themselves were moderately satisfied with treatment results closely paralleled their reports of patient satisfaction. Forty-one percent of the respondents themselves were at least moderately satisfied with the results for all of their patients; the average was satisfaction with the results in 83% of patients treated.

The *average percent* showing five possible levels of response for three diagnostic categories were as follows:

	Schizophrenia	Major Depression	Minor Depression
Excellent response	24%	62%	32%
Good response	42%	37%	29%
Fair response	31%	13%	24%
Poor response	13%	6%	7%
Very poor response	1%	1%	<1%

(Note: Percentages need not sum to 100%)

Adverse Effects

Users were asked to indicate the percentages of their ECT patients who had complained of various types of memory loss three months after treatment. The average percentages reported were as follows:

Temporary loss of memory for period of ECT course	46%
Temporary loss of memory for period immediately prior to ECT	34%
Permanent loss of memory for period of ECT course	27%
Slight confusion	18%
Spotty memory loss	16%
Permanent loss of memory for period immediately prior to ECT	15%
Temporary loss of distant memories	13%
Marked confusion	4%
Permanent loss of distant memories	1%

Ninety-two percent reported that *none* of their patients, as far as they knew, had been permanently prevented from performing specific vocational tasks after receiving ECT. Ninety-eight percent said that *none* of their patients had experienced spontaneous seizures shortly after ECT (who had not experienced them before). Ninety-seven percent reported that, over a period of five years, *no* deaths had occurred among their patients during or within 24 hours of the administration of ECT.

These findings are further referred to and discussed in the chapters which follow.

CHAPTER II

EFFICACY OF ECT IN AFFECTIVE DISORDERS AND SCHIZOPHRENIA

Introduction

Following its introduction in the late 1930s by Cerletti and Bini, ECT soon became a standard somatic therapy for severe psychiatric illnesses (1-3). Initially, the treatment was greeted with considerable enthusiasm, largely because it offered clearly superior results over the existing therapies—restraint, sedation, hydrotherapy, wet sheet packs, and seclusion. In more recent years, considerable controversy has surrounded ECT, stimulated in part perhaps, by a wide use of the treatment in a variety of disorders or by its use in large doses (4-7). From the replies to the questionnaire distributed by this Task Force, the consensus of American psychiatrists now appears to be that ECT is most appropriate for more severe affective disease and schizophrenia; this chapter examines the efficacy of ECT in these two conditions in some detail.

At the time that ECT was introduced, scientifically rigorous methods of conducting clinical trials using appropriately matched control groups were not in common use, and the strict guidelines established since for the demonstration of efficacy and safety in drugs have not been consistently applied to ECT. Nor have local quality control measures such as tissue committees for surgery been an element in the practice of the technique. ECT, therefore, has escaped many of the objective evaluations and controls which support other established surgical and medical procedures. The very nature of ECT renders the study of its efficacy elusive—since it is difficult to conduct completely blind clinical research with this technique. Yet many approaches to the study of the procedure have been made, some with very clever designs, and these will be discussed.

An important trend during the past 15 years has been the replacement of ECT by new drug treatments, primarily the MAO inhibitors and tricyclic antidepressants for depression (8, 9), and neuroleptics for schizophrenia (10). There is evidence, however, which strongly suggests that some severe depressions which fail to

respond to drugs will specifically respond to ECT (2, 11, 12), although a specific ECT response in schizophrenia is not so clear. Many studies have compared the efficacy of drugs and ECT in depression and schizophrenia, and these will be reviewed. However, no clear agreement has been reached concerning when in the course of therapy ECT should be employed or if and when it should be preferred to drugs. In this discussion, the evidence for efficacy of ECT in major affective disease and schizophrenia will be reviewed, with an emphasis on the role of ECT in and its pertinence to contemporary antidepressant and antipsychotic treatment.

Efficacy of ECT: Affective Illness

In addition to the basic question of antidepressant efficacy, other issues need to be examined in a critical review of the usefulness of ECT in contemporary antidepressant practice. These include:

1. Can it do what other treatments can't do?
2. What is the relationship between the presence of endogenous signs and symptoms to treatment outcome with ECT?
3. When in the course of therapy should it be employed?
4. How many ECT are sufficient to achieve adequate treatment?
5. What is the role of maintenance or prophylactic ECT in contemporary practice?
6. What is the evidence concerning the efficacy of unilateral treatments?
7. What are the clinical characteristics of patients who are medication resistant or intolerant yet responsive to ECT?

Another very important issue to be considered in the clinical decision to use ECT is the possibility of adverse effects. Although these will be touched on in this chapter, a more extensive discussion will be found elsewhere in this report. In this section on the efficacy of ECT in affective disease, comparison studies are discussed first, including more recent data on tricyclic drugs; next, outcome studies are reviewed with emphasis on mortality and suicide rates; then studies stressing descriptive elements and prediction of response will be presented. Finally, the evidence for efficacy of unilateral ECT will be explored.

I. Comparison Studies

One approach to the definition of efficacy of ECT has been by comparison to other treatments, usually drugs. A number of studies

performed in the 1960s all demonstrated the efficacy of ECT (2, 11, 13-21). Actually, in many of these, ECT was presented as the antidepressant standard against which the drugs were to be measured. An example of this approach is the work of Greenblatt, et al (2), who compared an MAO inhibitor (phenelzine), a tricyclic antidepressant (imipramine), and ECT in matched groups of manic-depressives, schizophrenics (schizoaffective type), involutional psychotic reactions, and psychoneurotic reactions. Overall, ECT was shown to be a superior antidepressant treatment for depressed patients diagnosed as manic-depressive and involutionally psychotic. Standard rating scales were employed in this study within a blind research design and generally adequate dosages of medication (200 mg. per day of imipramine; 45 mg. per day of phenelzine) were used. ECT was found to be effective in 78% of the more severely depressed sample in this study, compared to 59% effectiveness of tricyclic drugs. Signs and symptoms reflecting basic appetitive and vegetative dysfunction, including sleep disturbance, weight loss, libido change, and retardation or agitation, appeared particularly responsive to ECT. In addition, the authors concluded that ECT was the most effective antidepressant treatment and suggested that some patients respond to ECT while not responding to antidepressant drugs.

Later, well-designed studies by Wilson (13) and the Medical Research Council (MRC) in England (11) support the finding that ECT is a clearly efficacious therapy, and probably superior to medications, when antidepressant responses are compared. These researchers demonstrated that the vegetative symptoms and signs as measured by the Hamilton Depressive Index were most responsive to ECT. Their findings also relate to the question of whether subgroups of patients exist who will respond only to ECT and not to drug therapy. The MRC study was extensive and multicentered, and found that ECT was a significantly more effective antidepressant therapy when compared to MAO inhibitors and tricyclics (11). In addition, they found that when antidepressant non-responders were treated with ECT, an additional 50% of the sample responded whereas drug treatment of ECT non-responders yielded no further response. The tricyclic non-responders in this study were treated with an eventual dose of at least 200 mg. a day. It seems clear from these studies that ECT is a very effective therapy for severe depression, and it appears at least possible from the data presented by Greenblatt and the MRC group, that a "pure ECT-responsive/non-drug-responsive" population of depressed patients exists.

Other studies, including many from other countries, confirm the efficacy of ECT in severe depression and conclude that ECT is at least

equal to antidepressants, and, in most cases is a more effective treatment (14-18). Some comparison studies do show no difference between the two treatments (19-21). It is somewhat difficult to assess two of these studies by McDonald and Wittenborn because the samples are small. In the third study by Kristiansen interpretation is made difficult because the drug group was analyzed prospectively, whereas the ECT group data were gathered by a retrospective chart review method.

At this point, it might appear that ECT is a better treatment and it could be argued that it should be used as preferential treatment. Yet, over the past 15 years it has become clear that the tricyclic drugs also have proven efficacy in severe depression. Recent reviews, such as that by Morris and Beck (8), conclude that the tricyclics are effective in 66% of depressions. Drugs have advantages in that they are easier to prescribe, are less controversial, and are more readily accepted by patients. Furthermore, they usually do not affect memory adversely.

The recent evidence concerning drugs, particularly with a high dose regimen and serum level, and their relationship to antidepressant response would seem relevant to a discussion of the relative merits of the two treatments. This area is somewhat obscure at this time and while some investigators have shown that clinical improvement is enhanced by high dose drug therapy or appropriate serum levels (22), others have not (16, 23, 24). Individual patients vary in their absorption and enzymatic hepatic deactivation of these drugs (25). And more importantly, in studies where higher doses of these drugs have been used, the incidence of hypotension and atropinic side effects has been so noticeable that patients often could not be maintained on them (13). Elucidation of the relationship between serum and tissue levels of antidepressant drugs and clinical response will probably shed more light on the specificity of antidepressant response to ECT as well as to drugs.

It has recently been reported by Glassman that delusionally depressed patients do not appear to respond well to tricyclic antidepressants whereas they do respond to ECT (12). Hordern has also reported that delusional patients may not respond to antidepressant drugs (15). This might indicate one clear subgroup for preferential ECT. A recent report by Simpson, et al, however, questions the differential efficacy of ECT in the deluded group (26) and these investigators found that over 50% of deluded depressed patients *did* respond to tricyclics. There is some indication from their data that higher doses may be necessary in this delusionally depressed group. The question of possible ECT specificity in delusional depression deserves further examination.

In summarizing the comparison studies, ECT emerges as a very effective antidepressant treatment. It is sometimes difficult to decide whether ECT or antidepressant medication is the best *initial* treatment for severe depressive illness since the antidepressants have also shown clear efficacy for many similar patients. However, there is some evidence that certain depressed patients will respond only to ECT and not to antidepressant drugs.

II. Outcome Studies

Another approach to the definition of efficacy of ECT is by the study of the course of patients treated with this therapy. Early clinical studies of ECT outcome conducted in the 1940s and 1950s (27-31) were generally "open" trials (lacking matched control groups and blind evaluations of progress and outcome) and therefore anecdotal or, at times, frankly impressionistic. During this early period patient samples were often not carefully defined; results varied considerably and were often vague in their conclusions. Investigators enthusiastically concluded that ECT "shortened the length of the hospital stay" and resulted in a "fuller recovery" (28). These results are difficult to evaluate. However, one advantage of these studies was that retrospective control groups from the pre-ECT era were available and many important facts emerged from these data. For example, several earlier studies shed light on the question of the effect of ECT on mortality related to affective illness. Huston and Locher and other investigators observed that mortality was significantly less in ECT-treated patients compared to untreated groups from the non-ECT era (28). Death from suicide and other causes was found to be as high as 36% of untreated severely depressed patients, as compared to similar patients treated with ECT where the death rate was near zero. Other studies compared mortality in matched groups of ECT-treated and conservatively treated patients and reported similar results (27, 29-31). A large follow-up study performed recently by Avery and Winokur (32) confirms these findings of reduced mortality among depressed patients treated with ECT and adequate antidepressant therapy. In addition, these investigators reported that the mortality-reducing effects were more striking among the elderly, particularly men.

If one defines efficacy as reduced mortality and abbreviated depressive course, it is difficult to deny the efficacy of ECT in the treatment of severely depressed, suicidal, and debilitated patients. In the review of the early research, however, it is necessary to keep in mind the relevance of today's psychopharmacological practice. Except for the recent work of Avery and Winokur which found no significant difference in the mortality rates of ECT and adequately treated drug

groups, studies examining the course of ECT-treated and drug-treated patients appear to be limited.

Two additional outcome studies deserving mention are those of Sainz (33) and Ottosson (34-36). The former studied 20 patients suffering from the "depressed phase of manic-depressive disorder," or from "involutional depression." They were divided into two groups of 10 each, one group receiving ECT and the other "mock" ECT. At the end of the study, nine of the ECT patients had "recovered" and one was moderately improved, while the "mock" ECT group showed no improvement. The unimproved patients were then given actual ECT with the result that seven achieved full remission and two were substantially improved. Also, Ottosson's work with lidocaine and seizure threshold (34-36) demonstrated that outcome is related to the cerebral seizure itself and not externally applied electrical energy. Thus, as outlined elsewhere in this report, the efficacy of ECT appears to be related to its effects on limbic system mechanisms governing basic vegetative function and not "shock."

Research which was critical of ECT, including those outcome studies performed in the 1960s concluding that ECT was no better than "mock" or "sham" treatment (anesthesia without electrical stimulation), often relied on chronically ill patients including character disorders and chronic schizophrenics (37, 38). This type of study was hardly a fair test of the technique.

(a) ECT and Mania

Reports on ECT as a treatment for mania are sparse and usually anecdotal. Early studies utilizing retrospective control groups excluded manic patients (28) or contained inadequate sample size. In a recent outcome study McCabe (39) demonstrated that manic patients treated with ECT did remarkably well when compared to untreated controls. Although a controlled study comparing lithium, antipsychotic medications, and ECT has not been done, evidence does emerge from the work of McCabe that ECT has a very real role to play in severe mania where death from exhaustion is a considerable threat.

(b) Other Considerations

In a review of ECT outcome studies, it becomes clear that most measures of outcome tend to be clinical and retrospective in nature and long-term prospective outcome studies have not been performed. Since standardized rating scales have not been used, it is difficult to define what descriptive terms mean. In addition, data are sparse in several areas including long-term effects of ECT on natural history and maintenance studies. In regard to the former, little evidence was

found to suggest that ECT alters the long-term course or natural history of affective illness. Like the antidepressant medications, its effects appear limited to a reduction of symptoms of the illness (15). However it might be argued that work such as that by Avery and Winokur (32) does suggest alteration of the natural course of the affective illness since more ECT-treated patients survived.

Whereas exacerbations of depressive episodes diminish during longer term administration of tricyclics (40) and perhaps lithium (41), the efficacy of maintenance ECT has not been supported by controlled studies although it may perhaps have some usefulness in patients who do not tolerate even small amounts of drugs. Maintenance studies comparing ECT to tricyclics and combinations of tricyclics and antipsychotics are needed, especially in groups of patients such as the elderly who do not appear to tolerate medications well. There is some evidence that *adjunctive* use of medications during a course of ECT does not enhance the efficacy of the treatment (13), but some claim that adjunctive medication use is more likely to prevent relapse before the effect of the ECT is established. Others indicate, however, that since psychotropic drugs have potent venous pooling effects and can cause hypotension, there may be no uncomplicated advantage to using them simultaneously with ECT.

Finally, the relationship of outcome to the number of treatments remains unsettled. No "dose response curve" has ever been determined for ECT. In better studies where more severely depressed patients were treated, the average number tended to be between six and nine (2, 11, 13). Likewise, there is no evidence that extending a course of therapy will yield better clinical results or prevent relapse. In fact, a recent well-controlled study by Barton, et al (42) demonstrated no increased efficacy or prophylactic value in extended courses of ECT. These researchers assigned one group of patients with primary depressive illness to a course of ECT sufficient to elicit recovery and another group to two extra treatments after recovery. No difference in outcome between the two groups was demonstrated over a three month follow-up period. Most of the patients required about six treatments, which was close to the number required in other well-controlled studies.

III. Description and Prediction of Response Approaches

Since the 1950s, many efficacy studies have stressed descriptive and diagnostic elements. For example, Kahn, et al (43) studied a group of depressed patients treated with ECT and concluded that the patients who were "stereotyped, rigid, non-empathic, non-introspective, less educated, older, foreign-born," and who man-

ifested "euphoria and hypomania" were more often evaluated as recovered or improved. Conversely, patients who were "introspective, impassive, non-stereotyped, native-born, better educated, younger" and who manifested "somatization, paranoid withdrawal, and panic" tended to be rated as unimproved, or worse. The relevance to contemporary antidepressant practice of this type of study, performed prior to the introduction of antidepressants, is unclear. The items differentiating responders may also be forecasters of response to drugs.

The descriptive studies are closely related to a series of prediction of response studies performed over the past 20 years. Various methods have been described for prediction of response to ECT (44-48). These calculate a "prediction index" based on present clinical state and life history. Reported success in predicting treatment-responsive patients varied from 60-85% in these studies. The problem with virtually all of the prediction indices is that criteria for selection vary. Particularly important is that the introduction of antidepressant compounds has produced new samples of treatment-resistant patients who can hardly be compared to patients studied prior to the 1960s (48). Efficacy, therefore, when defined from earlier work is an elusive concept when applied to a modern sample. This is illustrated in the more recent prediction of response studies where patients treated with ECT are older. The probability exists that patients now treated with ECT are also more severely and delusionally depressed (12, 48). In a comparison of representative or typical predictive indices on a contemporary sample of depressed patients, no correlation was found between any of the indices and post-treatment clinical rating scores nor was there any correlation among any of the indices (47). Research studies using prediction indices have also failed to examine response to drugs in the samples studied. Some of the items (i.e., guilt, delusions, agitation) which have been reported in ECT prediction of response studies show considerable overlap when compared to tricyclic antidepressant predictors, and it does not seem possible to differentiate tricyclic responders from ECT responders from these studies. In fact, features which predict response to ECT appear to be quite similar to those related to outcome with tricyclic drugs (49, 50). While providing additional data for basic ECT efficacy, especially in more typically severely depressed patients, prediction of response studies have not clearly addressed the contemporary question of which treatment should be given to which patient.

IV. Unilateral ECT: Efficacy in Affective Disease

Unilateral ECT was introduced in 1954 by Pacella and Impastato

(51) to induce focal seizures in patients where generalized seizures were thought to be "too strainful." In 1958, Lancaster, et al (52) first reported the use of unilateral ECT producing generalized seizures. They found no statistical difference between the improvement caused by bilateral ECT and unilateral ECT but a marked improvement in orientation and short-term verbal memory with unilateral treatment.

Many subsequent comparison studies (53-56) have confirmed Lancaster's initial impressions that unilateral ECT with non-dominant hemispheric electrode placement produces less verbal memory impairment than bilateral ECT. In 1970, d'Elia (54) compared anterograde amnesia and subjective memory change in two randomized groups of patients with endogenous depressions (non-dominant unilateral ECT and bilateral ECT) using a double blind design. He found that non-dominant unilateral ECT had practically no adverse effect on the memory variables studied. He also found less retrograde amnesia and shorter orientation time after unilateral ECT in additional studies. Most recently, Reichert, et al (57) confirmed that bilateral ECT produced performance deficits while the deficits with unilateral ECT were minimal. While the subject of memory effects of ECT is complicated and there remain unanswered questions concerning dominant vs. non-dominant treatment (5), at this time it is clear that there is less disruptive effect on memory and shorter post-treatment confusion with unilateral ECT than with bilateral ECT. This subject of memory effects of ECT is covered in detail elsewhere in this report (Chapter IV).

Considerably more controversy exists concerning the therapeutic efficacy of unilateral ECT. This is probably due, in part, to variations in patient selection, treatment techniques including number and frequency, parameters of electrical stimulation, and electrode placement. Although most studies show that the two types of ECT are equivalent in therapeutic efficacy (52, 54, 56, 58-61), some authors have suggested that bilateral ECT produces quicker improvement (55), requires fewer treatments (59, 62), produces greater change in depression score (63), and shows a trend toward greater improvement.

In an attempt to resolve some of the confusion around the issue of therapeutic efficacy, d'Elia and Raotma (64) recently evaluated the literature on unilateral ECT for depression from a methodological perspective. They reviewed 29 studies from 1962-1973 which they divided into five groups according to various (methodological) criteria. They noted that the more precise the methodological conditions used in a study (i.e., incorporating simultaneous controls, random assignment with double blind comparison, homogeneous diagnostic groups and free number of ECT), the more unilateral and

bilateral ECT appeared to be equivalent in efficacy. Follow-up studies from one to 12 months to evaluate the stability of antidepressant efficacy showed unilateral ECT as effective as bilateral ECT (54, 55, 58, 61, 65).

D'Elia and Raotma also reported that patients subjectively reported the same feeling of improvement with unilateral ECT as bilateral ECT and they confirmed an earlier finding of Strömgren (66) that severe depressions respond as well with unilateral as with bilateral ECT. These researchers also commented on some popular misconceptions which may have contributed to the idea of unilateral ECT being less effective than bilateral ECT, including submaximal seizures (missed seizures due to inadequate stimulation and failure of the seizure activity to spread across the centrencephalic region). They concluded that non-dominant unilateral ECT has the same antidepressant effect as bilateral ECT; and that in endogenous depression requiring ECT, unilateral ECT is the treatment of choice.

Virtually all studies point to the equal efficacy of unilateral ECT in comparison with bilateral ECT; and in two independently conducted reviews of the literature regarding unilateral and bilateral ECT, the conclusion was reached that these treatments are equally efficacious in relieving depressive symptoms.

In Sweden, where there has been a long tradition of ECT research, 71% of all psychiatric hospitals that administer ECT currently use unilateral electrode placement (68). Nevertheless, some experienced clinicians in the United States currently view unilateral ECT as less effective than bilateral ECT (i.e., creating the need for a larger number of treatments in a course of ECT), and a few regard it as ineffective. Furthermore, 75% of those among the questionnaire respondents who use ECT currently use bilateral electrode placement.

There are several possible explanations for this disparity between published studies and individual experience. Special care is required to elicit a therapeutically effective cerebral grand mal seizure (see Chapter V) with the unilateral mode of administering ECT. Also, the experience of the individual psychiatrist may be more likely to reflect individual variation in the response to unilateral ECT than are the study populations in which these variations may be inconspicuous within the average group response.

The members of the Task Force have been persuaded by their joint experience and by the available data to favor the use of unilateral ECT, particularly as the memory disability following that method is considerably less than that following bilateral treatment. While this report presents the data supporting the advantages of unilateral ECT

in the average patient, the Task Force recognizes that the individual psychiatrist is in the most advantageous position to decide which is the best mode of ECT administration for any particular individual patient. Our report simply attempts to bring together the various factors which need to be considered in making this complex decision.

V. Additional Clinical Considerations and Summary

Although some research suggests that patients treated adequately with tricyclics may do just as well on standard clinical ratings as ECT-treated patients (26), it may also be true that severely depressed patients demonstrating endogenous features do not always respond to even high doses of tricyclics (2, 11-13). A significant number of these do respond to ECT (11, 49). Since the onset of the tricyclic antidepressant effect may take as long as three weeks to occur, patients who are severely suicidal, debilitated, agitated, or homicidal may also qualify as candidates for ECT. In these patients, the judgment must be made as to whether the prompt antidepressant response is worth the adverse effects of ECT, and in the case of a severely depressed and suicidal patient, the judgment is clear. However, many patients seen in clinical practice simply do not fall into a clear-cut category, and as yet no predictive measures are consistently helpful in determining how patients should be treated. Suicide indices are not in widespread use or generally reliable, and in practice a combination of diagnostic and severity criteria are used for this determination.

Patients who may be preferentially treated with ECT include those experiencing adverse reactions to drugs. Delirium, urinary retention, ileus with constipation and cardiac arrhythmias are the most common problems with the tricyclic drugs (13, 67). Delirium and other adverse effects secondary to tricyclics may be particularly troublesome in the elderly (48, 49).

Efficacy of ECT: Schizophrenia

Enthusiastic early reports, such as those by Kalinowski, Danziger, and others (69-73), led to widespread use of ECT for schizophrenia. The neuroleptics were introduced in the 1950s and early 1960s. Some comparison studies claimed superiority of ECT over neuroleptics (70-73) and may have formed the basis for the rationale of using ECT as a primary treatment in schizophrenia (3). In these early studies, however, experimental designs varied considerably and were usually retrospective. Evaluations were not double-blind, diagnostic and

selection criteria were unclear, and samples were probably biased. For instance, the series of reports by Baker, et al (71-73), which compared ECT with chlorpromazine, used patients from urban, highly mobile, low socioeconomic backgrounds and specified that chlorpromazine was not useful for this population because patients often demonstrated poor medication compliance following hospitalization. Long-acting phenothiazines may be more useful with such patients (74), but no recent studies were found which compared longer-term follow-up of matched groups treated with either ECT or long-acting phenothiazines.

In the early 1960s, a number of reports appeared which demonstrated no significant difference between ECT and antipsychotic drugs (75-77). Several reviews at this time found little proof of benefit from ECT in schizophrenia and heavily criticized statistical and design issues in previous studies (78, 79).

A number of difficulties emerge from the earlier data published on schizophrenia and ECT. Methods of establishing the diagnosis in this syndrome have varied tremendously over the past three decades, and standardized rating scales for descriptive elements of schizophrenia were not in use at the time of early work (71, 77). Yet standard psychiatric texts continue to speak favorably of ECT in the treatment of schizophrenia (3, 80). Recent data exist on the subject of ECT and schizophrenia and some informative though perhaps inconclusive studies will be reviewed here.

In a well-controlled, five-year follow-up study, May found that first admission schizophrenics (all diagnoses) treated with ECT fared significantly better than a psychotherapy group and about the same as neuroleptic treated patients when the time between discharge and further follow-up care was examined (81). The follow-up period was for at least three years, and comparison groups in this well-designed study included drug, psychotherapy, milieu, and psychotherapy plus drug treatment groups. When beneficial effects are defined in terms of hospital days, ECT appears to be effective in the treatment of schizophrenia. However, the situation is not clear since the same author concluded that in overall efficacy antipsychotic drugs are superior to ECT (82). This latest work examined all controlled pharmacologic and non-pharmacologic studies of schizophrenia treatment reported up to 1973 and classified and rated them. When weights were assigned to various treatments, including milieu, day treatment, aftercare, psychotherapy, drugs and ECT, medications demonstrated a significant advantage over ECT. Most authorities on the biological treatment of schizophrenia would agree that the antipsychotic medications are the preferred initial therapy (9, 83). This

attitude was also found among the psychiatrists who answered the questionnaire distributed by this Task Force. As can be seen from the findings of the questionnaire (see Chapter I), most United States psychiatrists use ECT for schizophrenic patients only when they have failed to respond to other treatment modalities.

Yet some evidence exists showing that ECT may be a very effective treatment in some forms of schizophrenia. Murillo and Exner have reported the use of "regressive" ECT in a group of chronic or "process" schizophrenics with apparently good results (84). They studied schizophrenic patients treated with an average of 26.3 treatments and compared outcome with a matched group treated with antipsychotic drugs and psychotherapy. On all measures of outcome including self-report, psychological tests (MMPI and Self Focus Sentence Completion), relatives' reports as measured by the Katz Adjustment Scale, and referring therapists' reports as measured by the Inpatient Multidimensional Psychiatric Scale, the ECT patients were found to be significantly more improved than the drug-treated group. Longer term follow-up by the same investigators appears to support the original findings (85). Although the work of Murillo and Exner does have some methodological difficulties (lack of random assignment, no independent blind raters) and has not been widely validated, the possible implications of their findings, like those of May (81), are not to be taken lightly. Further, well-designed studies of this technique may be warranted. This is underlined by what we now know about long-term treatment with antipsychotic drugs and the associated risk of irreversible neurological deficits—the tardive dyskinesias.

What about the efficacy of ECT in patients who have failed to respond to other treatments? Wells reported findings on 176 schizophrenic patients treated with ECT over a 10-year period (86). About half were first admissions and all had been given unsuccessful courses of neuroleptics and had failed to improve with milieu therapy. Seventy-five percent of this group with various schizophrenic diagnoses (catatonic, schizo-affective, paranoid, undifferentiated) showed good or moderate improvement—at least in the short term—following treatment with ECT. Follow-up data are not available from the study, descriptive features are not noted, and it is not known whether patients were on concomitant and/or follow-up antipsychotic drug therapy. There was some evidence that the best responses were obtained in schizo-affective and catatonic schizophrenia.

Folstein, et al, studied 110 consecutive patients, including schizophrenics who were given ECT, noting features of depression including hopelessness, worthlessness and guilt, as well as family history of affective disorder and suicide (87). They also described schizophrenic

features, if present, in these patients using the first-rank symptoms of Schneider (88, 89). They found that schizophrenia features alone were poor predictors of outcome in their patients, while even the presence of only one affective feature was a better forecaster of improvement than a schizophrenic diagnosis. Almost all schizophrenic patients who improved had affective features and responded to approximately eight treatments; the authors mention that many of the patients were rediagnosed as schizo-affective emphasizing the affective nature of the disorder.

In regard to the efficacy of unilateral ECT in schizophrenia, several studies have been reported but the data are insufficient at present to allow objective review (57, 90, 91). A recent study by Reichert, et al, (57) concluded that both bilateral and unilateral ECT were very effective in the relief of global pathology and specific symptoms associated with schizophrenia. However, their sample was diagnostically heterogeneous and the relationship between drugs and ECT was not clear. Additional study is needed to clarify the efficacy of unilateral ECT in schizophrenia.

Conclusions

It can only be concluded that the data are not adequate when considering the question of ECT and schizophrenia. Some data suggest that it is most useful in the treatment of affective target symptoms occurring independently of the diagnosis of schizophrenia and perhaps in cases carrying diagnoses of catatonia and schizo-affective disease. Some evidence supports its use in chronic or "process" schizophrenia, but data are sparse concerning its use as an initial treatment except when specific outcome variables are examined (81).

Positive results in "process" schizophrenia have been reported following the administration of 25-30 treatments (84, 85). The efficacy of unilateral ECT in schizophrenia has not been adequately studied.

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CHAPTER III

ELECTROCONVULSIVE THERAPY IN MENTAL ILLNESS OTHER THAN SCHIZOPHRENIA AND THE AFFECTIVE DISORDERS

I. *The Problem*

Contemporary methods for the convulsive therapies began to evolve in the late 1930s with the development of metrazol and later electroconvulsive therapy. Von Meduna was aware of eighteenth and nineteenth century clinical literature on camphor as a convulsant in the treatment of manias. He thought metrazol a better agent to induce seizures. Cerletti and Bini used the electrical method of inducing convulsions as they considered it simpler, with fewer complications than the chemical mode. In addition to the work of these authors in animals and man, animal work to test the safety and physiology of the convulsions was done in the previous century by Fritsch and Hitzig, and Ferrier (1). An important step in rendering the treatment more acceptable was the introduction of muscle relaxants by Bennett in 1941 (2).

Always the question has arisen—to whom should these treatments be given? The literature up to 1950 seems to indicate that patients receiving ECT were diagnosed as having neuroses, psychosomatic disorders, and manias, in addition to the major psychoses, if one can accurately translate the nosological categories. In part, based upon these experiences, more modern writings emphasize the use of convulsive therapies for the treatment of the affective disorders and the schizophrenias. However, as psychiatrists faced the challenge of helping patients, many psychiatric disorders or reactions were treated by ECT. In the past few years controversy and concern have arisen especially in regard to the use of ECT in mental illness not carefully diagnosed as schizophrenia or affective disorder. Here, not only are the usual problems concerning ECT involved, such as possible brain damage, memory loss, and the like; but for nonpsychotic patients serious questions are asked of any of the reasons given for its use (3, 4, 5, 6, 7, 8).

The purpose of this chapter is to review the literature of ECT use and efficacy in treating mentally ill patients other than those diagnosed as schizophrenia or affective disorder.

II. *Reports and Estimates of ECT Use in Nonpsychotic Disorders*

(See Tables I-VI at the end of this chapter)

APA recently polled a sample of members thought to be representative of the whole group. As can be seen in Table I, the majority of those responding to the questionnaire (2973 of 4000 polled) thought that ECT is probably not appropriate or totally inappropriate for use in mental illnesses other than affective disorder or schizophrenia. Only a small number, 29 to 136 (answer range depends upon the question asked), totally opposed ECT; only a small number totally approved it, 30 to 142. A slightly larger minority, 9 to 512, thought ECT was probably appropriate or was ambivalent to its use. Table II shows the psychiatrists who reported using ECT in nonpsychotic disorders. It seems from the table that fewer psychiatrists actually use it than those who mildly approved its use. Even for neurotic depressions, only 129 of 458 psychiatrists using ECT administered it to patients with this diagnosis. The psychiatrists reported the total ECT case load diagnosed neurotic in the one to five per cent range. Table III extrapolates, possibly inaccurately, from a report of six months' case loads. It suggests each working day 369 patients of all diagnoses received ECT from or under the supervision of APA members. One hundred and twenty-nine psychiatrists reported that one to five per cent of their case load who received ECT had a diagnosis of a non-psychotic mental disorder.

Table IV shows that in nonpsychotic disorders the number of ECT treatments ranges mostly from two to six, although there are cases of as many as 11 to 21 treatments. ECT is rarely the first choice of treatment. The reasons for use of ECT are explored in Table V. It seems that ECT is not a treatment of choice in minor depressions but is used after drugs and psychotherapy fail. Table VI shows reports from psychiatrists on how they saw response in their patients with minor depressive disorders. Most of the results were rated in the fair to excellent ranges.

Another epidemiological study was done by Grosser (9) who surveyed the state of Massachusetts for a period of one year after the introduction of the ECT guidelines. Twenty-five percent of patients in private hospitals who received ECT had a diagnosis of depressive

neurosis. Tien's smaller Michigan survey (10) noted much the same pattern. A few private practitioners with an atypical approach to ECT theory and therapy, as typified by Friedman (11), used ECT on a variety of neuroses, especially the obsessive compulsive or phobic types.

A survey of ECT use in Denmark (April 1972 to March 1973) (12) revealed that 18.8 per cent of departments of psychiatry surveyed use ECT for obsessive compulsive neuroses, depressive neuroses, and the symptomatic psychoses. A review of Toronto, Canada, hospitals during 1969 to 1973 (13) reported 28 per cent of patients receiving ECT had neurotic depressions. The authors believed, but did not demonstrate, that patients with neurotic depressions were often given ECT because of suicidal attempts.

From these incomplete data, it appears that ECT is and has been fairly widely used in the practice of psychiatry for patients with non-schizophrenic or non-affective diagnoses. However, caution should be used in interpreting these figures. Some private hospitals in the U.S. are reporting a decrease in patients given ECT. It is thought that the use of lithium and new medicines may be involved as well as other factors such as changing public awareness and growing government and mental health center programs (14, 15). The APA survey suggests ECT is used less for neuroses than would be implied by any of the above epidemiological studies. Moreover, some authors (9) noted social class differences in ECT use with higher social classes receiving more somatic therapy. Public hospitals seem to be abandoning the use of ECT.

III. *Reports on the Use of ECT in Patients with Psychiatric Disorders not Schizophrenic or Affective*

In Sections III and IV of this report, many different published papers will be mentioned. They include case reports, some retrospective clinical reviews, and a few projects of some substance. Much published ECT research, especially that applying to the nonpsychotic patient, is somewhat lacking in achieving modern standards of methodology. Such refinements as random samplings, prospective methods, blind ratings, specific hypotheses and instruments to rate them, multiple hospitals involved to achieve significant numbers, computer analysis of derived data, dissection of confounding variables, and the like have been used infrequently. ECT in nonpsychotic disorders has not had the benefit of rigorous research such as has been done for ECT in depressions or to a lesser extent schizophrenia, or for

the major tranquilizers and lithium. For these reasons, only the core ideas of most of the papers are extracted and any positive results must be reviewed with great care (16, 17, 18, 19).

A. ECT in Patients with Organic Brain Disorders

This section is important because of therapeutic and theoretical implications. It is also even more controversial than other portions of this report. A short introduction is in order. Firstly, because of the disputes about ECT causing an organic brain disorder, many would consider it strange to use it for the treatment of an organic brain disorder (20, 21, 22). However, as Cunningham (23) has pointed out, it is the considered opinion of some psychiatrists that ECT has an important place in the treatment of the more seriously disturbed patients suffering from psychiatric disorder, a category that certainly includes the organic brain disorders. Secondly, although ECT is considered by many a safe procedure, one of its quite rare but serious complications, as noted by Paulson (24), is the abrupt clinical presentation of an organic brain disorder during the course of treatment. He suggested that the ECT makes manifest an underlying latent organic disease. Smith (25), too, noted this problem and warned that severe cerebral damage may occur in patients who receive ECT within 30 days of recovering from carbon monoxide poisoning.

Despite the reviews noted above, many psychiatrists have reported good results with the use of ECT in a single case or small studies of several diverse clinical entities. They include such organic states as general paresis (26, 27, 28, 29); Parkinson's Disease (30); cerebral spastic paralysis (31); recovery from stupor following head injury (32); bizarre psychogenic movements (33); Alzheimer's Disease, Pick's Disease, steroid psychoses, and senile dementia with delirium (34, 35, 36).

Roberts (37) reviewed the sparse literature on the use of ECT in the acutely disturbed, delirious patient. His series of nine cases was taken from his hospital's records of delirious patients admitted over a 15 year period and treated with ECT. He thought ECT useful and safe in suppressing severe delirious symptoms and that its effect was not related to the cause of the delirium. Dudley and Williams (38) reported on a controlled study of the treatment of delirium tremens and thought that the use of ECT significantly shortened the time of hospitalization. However, the study had some methodological problems in choice of patients used for the matching and the fact that the retrospective review was done some years after the treatment.

ECT has been used for the treatment of drug induced psychotic or delirious conditions; for example, bromide intoxication (39), am-

phetamine psychosis (40), barbiturate intoxication (34), delirium due to tetraethyl lead (41), cannabis (42, 43), and LSD psychosis (44, 45, 46). Muller's paper (46) surveyed the use of ECT in LSD psychosis, noting particularly that the three cases he reported did not respond to more conventional drug therapy or to psychotherapy. Muller followed each of these three cases for a year. One interesting report in the French literature (47) told of the development of a stuporous state with delirium and hallucinations, probably induced by a monoamine oxidase inhibitor drug given for the treatment of depression, the patient recovering with ECT.

Epilepsy has also been treated with ECT. In an early work Caplan (48) noted that psychotic patients undergoing ECT who had epilepsy often seemed to have fewer seizures while they were receiving ECT. Wolff's work (49) was in a similar vein. Most clinicians feel that ECT on the whole tends to decrease spontaneous seizures. Many also think that ECT is useful in alcohol withdrawal states and for this reason have used it in other agitated withdrawal states, for example, barbiturate intoxication, opiate withdrawal, and the like. The results of these uncontrolled studies are variable (34, 36).

Lachman and Tuchman (50) reported a problem in the treatment of an epileptic patient. In their single case report, they withheld an anticonvulsant from a patient undergoing ECT with the consequence that she became stuporous and did not clear until the anticonvulsant was resumed. Assael, et al (51) reported a 30 year-old woman, previously in good health, suffering from a catatonic schizophrenia who developed epilepsy as a complication of ECT. Further doubts were expressed by Elmore and Sugerman (52). They reported on a series of three cases. In each, an organic type of psychosis developed during a brief series of ECT. Two patients recovered, another did not.

There is also some suggestion in the literature that ECT is useful for mentally retarded individuals. In the main, these are mentally retarded individuals who have affective disorders or thought disorders associated with their primary problem. In no sense can convulsive therapy be considered a primary treatment for mental retardation (53, 54).

Of the reports reviewed in this section, only the work of Dudley and Williams (38) used controls, but the methodology was flawed. Therefore, indications for the use of ECT in organic brain disorders lack substantive scientific validity.

B. ECT for Patients with Medical and Psychiatric Illness

This section is complex both to conceptualize and to categorize. There is a reciprocity between the presence or absence of mental

disorder and the degree of physical integrity of the human body. It is hard to describe this interaction or to understand or predict the articulated effects, among other factors, of dynamics, physical illness, external circumstances, and psychiatric treatment. Blachly (55) commented on this in his paper comparing ECT with cardioversion. Carlsson (56), in a recent academic lecture, noted that the drugs used to alter mood and behavior also may change such bodily functions as heart rate, its regularity, and liver and kidney action.

Another facet of this articulation was expressed by Goldfarb (57) who held the opinion that breast cancer, in particular, was associated with grief subsequent to a loss or frustration. For this reason, he used ECT on three women with breast cancer. All three experienced positive cancerocidal effects although in one case they were short-lived. He thought that further investigations were warranted.

In lupus erythematosus, Guze (58) reviewed 101 consecutive patients admitted to the hospital. He found psychiatric disorders were frequent and when present they began early in the course of the main disease. The relation to steroid administration was unclear but steroids were considered neither a necessary nor sufficient cause for the psychiatric disorder. He thought that the psychiatric illness was intrinsically related to the lupus. Three of the psychiatrically ill patients he studied received ECT and all benefited from it. An earlier study by Malamud and Sands (59) expressed similar views.

Hollender and Steckler (60) reported a case of multiple sclerosis and schizophrenia, combined with a review of the literature on the subject. Their patient received 15 electroconvulsive treatments with benefit. The report by Savitsky and Karliner (61) also was positive. Negative results were reported by Hart (62) in the treatment of rheumatoid arthritis with ECT. ECT has been tried with unclear results in such conditions as ulcerative colitis, neurodermatitis, and in bizarre psychogenic movements and other various chronic neuromuscular conditions of obscure origin (5, 6, 33, 34, 63).

ECT has been used for secondary psychiatric problems that are associated with medical illness. For example, it has been used to treat the depressions in patients with carcinoma or the grief reactions due to the loss of body parts and depressions associated with pellagra or pernicious anemia (5, 6, 34).

Pain also is considered a special indication for ECT (64, 65, 66, 67, 68, 69, 70, 71). There are reports in the literature of rapid and dramatic relief of physical pain when all other remedies have failed. Hohman and Wilkinson (72) even went so far as to say that ECT should be used prior to neurosurgical intervention. Boyd (73) thought that depression and neurosis combined with pain may render it

intractable. These opinions were written prior to some recent advances in neurosurgical treatment and theories of pain.

It is also very difficult at times to administer properly indicated ECT to patients who have a psychiatric illness and a concomitant severe medical illness. Kardener (74) reported in 1968 concerning a patient with thrombocytopenic purpura. In his opinion the patient had life threatening depression but the medical consultant advised against ECT and psychotropic drugs. On his own Kardener proceeded to use ECT, relieved the depression, and did not hurt the patient. Another example was reported by Hoaken, et al (75) wherein a patient with Klinefelter's syndrome developed a serious psychosis and was helped only after a course of ECT which was probably lifesaving. Ananth and Beszterczey (76) recommended ECT be considered in the treatment of a phenothiazine-induced agranulocytic psychosis. Dressler and Folk (77) noted that ECT may not necessarily be contraindicated in the case of a brain tumor. In selected cases, it possibly was beneficial. They gave some guidelines and one instance. Patients with severe cardiovascular disease who required ECT were reviewed by Rubin and Atkinson (78). They stressed the general safety of ECT and how under modern conditions with modified ECT procedures the heart can be protected by atropine so that the blood pressure does not rise significantly because of the use of succinylcholine. The work of the Smalls (79) found that the number of premature ventricular contractions in patients without heart disease does not significantly increase after modified ECT. Bodley and Fenwick (80) reviewed the effects of ECT on patients with essential hypertension. Their conclusion from their own study and review of the literature was conservative. They stated it would be presumptuous to advise which patients would or would not stand the unique burden of the treatment. Weinstein and Fischer (81) have used it in patients who have artificial aortic and mitral valves. Selzer and Inderbitzin (82) did not find renal function directly related to ECT.

Others worry about the intraocular changes during anesthesia and electroshock therapy. Epstein, et al (83) recently reviewed this matter and thought the changes that occur are not of sufficient magnitude or duration to cause concern.

There is, too, the complex syndrome of anorexia nervosa. ECT use has been reported by Bernstein (84), and Moldofsky and Garfinkel (85) were somewhat skeptical of their own results. More recent work stresses drug treatment for severe forms of this condition (86).

Blazer, et al (87) have a new report. They reviewed their work with affective psychoses arising subsequent to renal transplant. They used ECT, found that the patients responded well, and that it did not

interfere with immunosuppressive drugs. These affective psychoses did not tend to recur after the ECT induced remission.

The papers in this section are uncontrolled and often retrospective. In the main they are case reports and any positive results must be conservatively evaluated. The short section on interaction with physical illness is included to stress the great clinical complexities encountered and the knowledge and judgment required to make serious medical decisions when few hard data are available. Mention is made of the practical problems that arise in the treatment of the mentally ill when both mind and body require care.

C. ECT for Patients with Neuroses

Practicing psychiatrists have used ECT for years in the depression, hysterical, phobic, and compulsive neuroses. To a smaller extent, they have used it in the anxiety neuroses. In the main, a clinical dictum exists that ECT increases neurotic anxiety and the associated memory loss upsets anyone who inclines toward a hysterical personality. Another common clinical dictum states that the greater the affective elements, especially of a depressive neurosis type, the better the ECT response. The theoretical basis for its use has been to this time empirical (88).

In 1968 Prendergast (89) reviewed the practical treatment of anxiety neuroses. He did not recommend ECT. Davison (90), reporting on episodic depersonalization, found in the literature conflicting reports on the value of electroconvulsive therapy. One of his cases responded, two showed no change, and one was made worse with ECT. Noreik (91) did a five-year follow-up of 81 neurotics admitted to the hospital over a 22 year period. Of these, 53 received ECT but at follow-up no long-term benefit from ECT was found. The long-term outlook for severe neurosis was poor; 47 per cent improved; 36 per cent remained unchanged; 17 per cent deteriorated; and 4 per cent died. Carney and Sheffield (92), in a study of 53 endogenous and 22 neurotic depressives, found 72 per cent of endogenous depressives recovered with ECT, whereas 32 per cent of the neurotic depressives recovered. Patients were rated subjectively by themselves and objectively by the authors. The patients' subjective reports in the neurotic group did not indicate that ECT was of much benefit. Pilowsky (93) reported a series of 147 cases with hypochondriasis followed for at least two years. Thirty-five patients received ECT. There were no marked differences among the treatments given to the three outcome groups—good, fair, or poor—so no usefulness could be shown for ECT. Goldney and Simpson (94) found in a severe case of hysteria that it was not helpful.

Some reports gave more positive results. Grief reactions, especially unresolved, were reported as responding well to ECT (95, 96, 97, 98, 99). Edwards (33) reported dramatic success in the treatment of severe hysteria with ECT. His note also reviewed the literature and suggested a method of treatment. In 1966, Sargent, et al (100) reported unusually good results in the treatment of chronic tension states using antidepressant drugs, narcosis, and ECT. Seventy-three patients were studied for five months to three years. Thirty-seven received drugs and ECT; and 36 drugs, ECT, and continuous narcosis. Forty-nine were symptom free at follow-up. The ECT narcosis group had only three referred for leucotomy compared to 15 for the other group. He was not at that time able to explain his unexpected results.

Folstein, et al (101) reviewed the charts of 118 patients who received ECT. Patients with a family history of mood disorders, suicide, or symptoms of hopelessness, worthlessness or guilt improved after ECT, regardless of diagnostic label. Patients without these features did not improve, especially if diagnosed schizophrenic or neurotic. The authors attempted to validate the study by the external criteria of rating checks from a chart review and measuring length of stay. Improved patients on chart review stayed 38 days in hospital after ECT; those not improved, 93 days.

Slavney and McHugh's small study (102) was interesting. They reported on 32 inpatients diagnosed as hysterical personality (DSMII) who were compared to an age, sex matched sample of inpatients having diagnoses of personality disorders, psychosis, or neurosis other than hysterical. The index patients showed a high incidence of depressive symptoms and suicidal reactions. They were not treated as primary depressions but rather as individuals who demonstrated traits of emotional instability and self-dramatization. The hospital course of the neurotic group was significantly longer than that of the control population, and significantly fewer received antidepressant therapy for their suicide attempts. Bibb and Guze (103) in another study reviewed all females admitted to their hospital in 1968 (N-911). Index patients were those females meeting Perley and Guze's criteria for Briquet's syndrome—essentially a chronic polysymptomatic disorder with dramatic features and no conversion symptoms. The 95 index patients were matched with 83 primary depressives on wards or private services by age and race. Study of the two groups cross-sectionally revealed similar problems with depression, suicide, and suicidal threats. However, the index patients were found to have received significantly less ECT for these depressions. The authors noted that secondary affective disorders may be considered different from primary affective disorders in their response to treatment.

Haddock (104) reviewed his private patients who suffered from obsessions, compulsions, and phobias. He selected 77 of these patients from 1933 consecutive patients. No controls were used. Twenty per cent of the obsessives, 45 per cent of the compulsives, and 25 per cent of the phobics received ECT. He thought ECT was quite effective for the obsessive and depressive groups, somewhat so for the compulsives, and not at all effective for the phobics.

Friedman and Andrus (11) have recommended Sedak Therapy for phobic neuroses. They particularly like this technique of unidirectional stimulation because it does not cause a full convulsion. Most psychiatrists using ECT would not agree.

The studies in this section are often more than case reports but do not give enough data to provide scientific documentation. Both positive and negative data are found so it is likely that many factors other than ECT must be included to explain the results. Scientific documentation of the use of ECT in the neuroses is still elusive.

D. ECT in Patients with Behavior Disorders

Disorders of behavior are not ordinarily given ECT. Authors such as Darling (105) have reported on it but results have not been good. Textbooks do not recommend it (6, 36). Sachs and Titevsky (106) in a mildly positive report described two homosexuals treated with ECT. Sexual impulses were "released" by ECT. Thompson (107) reported suggestive results in a series of six cases. These studies and case reports are negative and give no grounds for ECT use in disorders of behavior.

IV. *Reports on the Use of ECT in Special Circumstances*

A. ECT in Patients who are Children and Adolescents

The largest European study of ECT use in children was published in 1947. Heuyer (108) described work with 29 children, age five to 15 years. The results were clinically unimpressive. No problems were found with ECT from a physical, mental, or developmental viewpoint. The patients who responded somewhat were mainly described as depressives, manics, or confused. These latter probably were early onset adolescent schizophrenics in current nosological terms.

Hift, et al (109) also reviewed ECT in children. Even in their study in 1960, they noted that it is unlikely that anyone will ever be able to redo this kind of work because of opposition to ECT treatment, especially as it affects children. They reported on 23 children with

differing types of schizophrenic diagnoses. Eight received electroshock therapy, while five received electroshock therapy plus insulin coma. The number of treatments varied from two to 16. The ages of the patients were from seven to 14. They, too, did not believe ECT had significant or long lasting value. On the other hand they thought it did no harm.

Arajärvi, et al (117) reported a study of psychoses in childhood with one case of childhood schizophrenia receiving electroshock. The long-term results were equivocal. This work reviewed all the children seen between 1952 and 1960 at the neurological and psychiatric clinics of the University of Helsinki.

In the American literature, the most prolific writer has been Lauretta Bender (110, 111, 112). For many years she followed her schizophrenic children for a longitudinal view of personality development. She stated that in schizophrenic children the use of ECT was justified as it improved the quality of remission compared to patients who did not receive ECT. In 1955, Gillis (113) also reported use of ECT and insulin for one child.

A case report of an adolescent was made by Warneke (114) who described a 12-year-old with a manic depressive psychosis who received ECT and benefitted. The late onset of psychoses of childhood beginning in early adolescent life was reviewed by Kolvin (115). In his paper, the author thought it the consensus that these patients did not respond well to ECT. However, Hudgens (116) recommended ECT for adolescents, particularly those who are suicidal or unresponsive to drugs or psychotherapy. His opinion on this matter was quite strongly expressed. He stated that it is cruel to withhold treatment because of prejudice against ECT.

Most of the reports of the use of ECT in children do not tell of good clinical results. Probably the strongest positive position is taken by Bender. She followed her children using clinical, psychological, and EEG testing for many years. Yet most child psychiatrists would probably not agree, as there is much concern regarding inducing convulsions in a young person whose nervous system is possibly more vulnerable than that of adults. The point concerning suicide raised by Hudgens is important but other treatments for this reaction are available.

B. ECT for the Quieting of Homicidal States

Almost any severe psychotic state, especially agitated depression, catatonia, paranoid schizophrenia, the manias, and the severe organic brain disorders, may result in a furor. Under these conditions behavior that is seriously homicidal or suicidal may ensue. Judicious use of

ECT or ECT combined with drugs might help to end the process early.

The existence of a catatonic state with hyperpyrexia is one very specific indication for ECT. The condition is considered one of high, rapid mortality but almost instantly reversible with ECT (118). Rainey (119) advocates stricter rules for diagnosis, EKG studies (an increase of the QT interval), and the use of i.v. cortisone and phenothiazines, combined with ECT. Recently Shader (120), too, suggested the use of ECT to calm violent schizophrenics.

C. ECT in Patients with Psychoses other than Thought Disorders or Affective Disorders

On the borderline of schizophrenia and depressive disorder are the problems of schizo-affective disorders, the cycloid psychoses, or the schizophreniform, psychogenic, or atypical psychoses. Many of them are considered to be quite responsive to ECT. Wålinder (121) has reviewed the literature and outlined the theories behind the diagnostic criteria for schizo-affective psychosis. He emphasized the uniqueness of these disorders and their genetic background. He urged the use of ECT for its acute treatment and lithium for prophylaxis. Dempsey, et al (122) also have presented case studies, reviewed the literature on diagnosis and treatment of schizo-affective disorders, and recommended for acute treatment the use of ECT and/or lithium. Perris' monograph (123) is a comprehensive study of 60 patients with cycloid psychoses. Most patients were followed over several years. Their relatives were studied, too. He especially recommended ECT for treatment of acute confusional cases. He was of the opinion that in time lithium offers a prophylactic treatment and neuroleptics may significantly reduce the amount of ECT used. He referred to an ongoing cooperative Scandinavian study that may give a better answer to the issues of definitive treatments. Perris also reviewed the work of Scandinavian, German, American, and Japanese workers on schizo-affective disorder. He expected most would concur with his remarks on therapy. Other psychoses, too, may respond to ECT. Among these are the late paraphrenias, the depressions of the aged, and the periodic catatonias. No control studies were available (124, 125, 126).

V. *Further Considerations*

A. Mode of Induction of ECT may be Related to Treatment Outcome

Soon after the introduction of ECT into the United States, Liberson (127) tried to reduce the current required to induce a seizure. He thought if this were done the treatment could be less

impairing of memory and its range extended diagnostically. Blachly and Gowing (128), with an updated, specially designed low energy machine, have evolved the multiple monitored mode of treatment. They think this technique reduces the time required for treatment, diminishes the usual side effects, and enhances the therapeutic effect. Others (129) have not always replicated the results. For similar reasons, Weaver, et al (130) have worked in this area too, designing an experimental ECT machine. They and others are studying in a controlled method effects of current intensity as related to memory loss.

Others, such as Reiter and Freidmen, recommended that the Reiter stimulating machine be used with various types of unidirectional currents and considerable alterations in the placements of electrodes. It is their opinion that almost any diagnostic group of patients can be treated with this machine, generally after failure of drugs and psychotherapies. For the most part the subconvulsive currents are used without anesthesia. Endogenous depressions only are thought to require convulsive currents (11). This work, too, is highly controversial and violates Ottosson's finding (131) that the convulsion is a necessary therapeutic ingredient of ECT. These later opinions are reported as the views of a minority of psychiatrists whose work has been presented in case reports but not in controlled studies.

B. An Animal Model is Needed

The effect of electrically-induced convulsions in species other than man has always posed a problem. The differences in species, mode of induction, and patient management have always been too great to show conclusions. Yet the idea is intriguing, for if animal work could be used, the prediction of response to treatment might be much more easily studied. We should then be on firmer scientific ground in predicting patient response to treatment.

Recently Lewis and McKinney (132) worked with monkeys using methods paralleling human conditions. They found the ECT response could be predicted from previous life experience and training. Monkeys reared in a deprived environment showed increases in environmental activity and tended to decrease self-disturbance behaviors. Those reared in a normal environment showed decreases in activity and social behaviors and increases in self-disturbed behavior. This new work offers the possibility of an experimental model to study ECT which is an exciting prospect.

Summary

It appears from this review that there are many opinions and conflicting studies concerning the use of ECT in conditions other

than the affective disorders or the schizophrenias. This conflict exists in the context of the larger problem of what is the risk-benefit equation of ECT. Some ask, "Even if the risk-benefit equation balances in the direction of help for the patient, should ECT be used at all?" Further, how can ECT be used in non-affective or non-schizophrenic conditions on such slim indications when we have not answered to satisfaction the issue of amnesia and the topic of informed consent?

It has been estimated from available data that 15 to 30 per cent of patients treated in this country with ECT are not diagnosed as depressed or schizophrenic. The APA survey does not support this estimate and indicates that one to five per cent of case loads receiving ECT are diagnosed neurotic. It appears that the use of ECT in private hospitals may be declining, probably due to social pressures and the development of new treatments. Both Aden and Blachly have noted this trend (14, 15).

This review cites many reports of the use of ECT in patients with organic brain disorders. However, insofar as efficacy is concerned the data base is thin and at times conflicting. It is interesting from a clinical point of view to note that psychoses, probably of organic origin, are reported in some instances to be favorably influenced by ECT. Similarly, ECT in medical disorders is a complex prescription. The published case reports seem to indicate safety and usefulness as well as dangers but here, too, we can rely only on sparse clinical data. The key issue here seems to be, "Should ECT be used when properly indicated in a psychiatric condition that is combined with a severe medical problem?" At this time, the best answer is complex: judge each case on its own merit and obtain consultative assistance or follow channels used in the community for special or research studies.

The subject of ECT in the neurotic disorders is another area of controversy. Much psychiatric opinion is against its use and several studies of follow-up populations support this view. The mildly positive data found are some studies that emphasize the affective components of the neurotic condition as predicting a good response. However, even here Bibb and Guze (103) noted that the response to treatment of the neurotic affective disorders may be different from the response to treatment of the more pure affective disorders.

With children and adolescents even more disparity exists. Existing studies are not satisfactory and the recommendations by authorities differ. Certainly from the published literature and task force hearings few, if any child psychiatrists are now using ECT in the United States. However, its use has been acceptable to a small minority on rare and exceptional occasions.

For adults in life threatening situations, especially fulminating catatonia, ECT can be considered invaluable. But again not all psychiatrists agree and controlled studies are lacking. Data for the use of ECT in the schizo-affective psychoses are not compelling but better than for many areas and come from diverse sources.

It is not possible to consider ECT use without studying how it is used. Different currents, machines, electrode placements, modifications, and ancillary treatments are thought to effect the outcome of treatment. It is hoped that more controlled studies such as those of Abrams and Taylor (133), d'Elia and Raotma (134). Lewis and McKinney (132), Weaver, et al (130), and others will cast more light on the matter.

The weight of the research data in the published literature leaves little room for doubt that ECT is an effective and valuable treatment in some types of affective disorders and probably effective in the treatment of some cases of schizophrenia. The consensus of clinicians who use ECT, reflected in the answers to our questionnaire, supports this view. However, there exists among some psychiatrists the opinion that ECT is an appropriate treatment for conditions other than the affective disorders and schizophrenia.

This apparent difference of opinion is not unknown in medical practice. It does seem to us, however, that if such use of ECT is continued, care should be taken to study its effectiveness in a research setting, or consultation should be sought with colleagues who use a different type of therapy. No individual is compelled to comply with these suggestions, but if we aim to increase knowledge or enrich psychiatric practice generally by means of an exchange of opinions among the various schools of thought, the measures suggested above are highly desirable.

A model for the investigative studies of ECT referred to above has already been developed for the investigation of new drugs.

TABLE I

APA Survey

Psychiatrists' Opinions

Indications for ECT

Response Rating	*Totally Appropriate* 1	*Probably Appropriate* 2	*Ambivalent* 3	*Probably Not Appropriate* 4	*Totally Inappropriate* 5	*Opposed to ECT* 6
		INDICATION		N	MEDIAN RESPONSES (1-6)	
		Minor Depressive Disorder		2924	4.76	
		Drug and Alcohol Abuse		2971	4.78	
		Personality Disorders		2922	4.85	
		Sexual Dysfunction		2920	4.90	
		Anorexia Nervosa		2916	4.08	
		Intractable Pain		2912	4.19	
		Unremitting Hypochondriasis		2920	4.10	
		Toxic Dementia		2915	4.78	
		Other		432	2.70	

(See App. II, Questions 20-31)

TABLE II

APA Survey
On the Use of ECT in Non-psychotic Disorders

Diagnosis	*Per Cent of Case Load Receiving ECT*		
	Mean	*Median*	*N-Responders*
Minor Depressive Illness	5.485	0.196	458
Drug and Alcohol Abusers Treated	0.298	0.015	383
Personality Disorders Treated	0.159	0.012	384
Sexual Dysfunctions Treated	0.021	0.005	383
Anorexia Nervosa Treated	0.315	0.033	391
Intractable Pain Treated	0.338	0.032	385
Hypocondriasis Treated	0.556	0.048	396
Toxic Dementia Treated	0.116	0.009	378
Per Cent of Others (nonpsychotic) Treated	1.743	0.031	292

(see App. II, Questions 87-98)

TABLE III

APA Survey

Number of Patients Reported Treated in U.S.A.
in Six Month Period

467 Psychiatrists report treating
6,652 Patients treated in six months
Data multiplied by 6.66 as 15% of APA members surveyed responded (20% were polled)
∴ Calculate – treat 44,302 patients in six months
– treat 7,282 patients in one month
– treat 369 patients per working day (20 days/month)

(see App. II, Questions 37 and 38)

TABLE IV

APA Survey

Average Number of ECT Treatments Given
Patients with Minor (Non-Psychotic) Depressive Illness

Optimal Number of Treatments	*N - Responses*	*Per Cent Reporting*
0	319	29%
2 - 6	139	44%
7 - 10	69	22%
11 - 21	15	5%

(see App. II, Question 57)

TABLE V

APA Survey

Reasons for ECT Use in Minor Depressive Disorders

Reasons for Use of ECT	*Mean Per Cent of Patients*	*N - Responders*
ECT is first choice of treatment	2.6	154
ECT is better combined with other treatments	11.9	159
Large Dose Drugs Ineffective	18.4	160
Small Dose Drugs Ineffective	7.5	150
Side Effects of Drugs	3.1	151
Psychotherapy Ineffective	6.0	148
Large Dose Plus Psychotherapy Ineffective	37.2	178
Small Dose Plus Psychotherapy Ineffective	12.9	155

(see App. II, Questions 79-86)

TABLE VI

APA Survey

Minor Depressive Disorder
Response of 50% or More of Cases Treated

	Mean Per Cent of Cases	*N*
EXCELLENT	32.4	140
GOOD	29.0	151
FAIR	23.6	141
POOR	7.4	128
VERY POOR	0.13	117

(see App. II, Questions 117-121)

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CHAPTER IV

ADVERSE EFFECTS OF ECT

Section I. *Memory and ECT*

Memory loss has long been recognized to be a prominent effect of electroconvulsive therapy (ECT). For a decade or two after the introduction of ECT, loss of memory was believed to contribute to ECT's therapeutic effect (1). Today the view is considerably different. First, several investigators have demonstrated that the extent of memory impairment is not correlated with clinical improvement (2, 3, 4). Second, following the development of right unilateral ECT (5), it became clear that this mode of convulsive therapy results in markedly less memory impairment than conventional bilateral ECT (6, 7, 8, 9). Yet right unilateral ECT is clinically as effective, or nearly as effective, as bilateral ECT (10). Accordingly, all available evidence supports the contention that memory loss is an undesirable side effect of ECT, not related to therapeutic efficacy.

Like the organic amnesias that result from head trauma (11), Korsakoff psychosis (12), diencephalic tumor (13), or temporal lobe dysfunction (14), the amnesia associated with ECT is both antero-

grade and retrograde. Amnesia occurs for the events prior to each seizure and an impairment in the ability to commit new events to long term memory is evident following each seizure. This impairment in learning new material diminishes gradually following each seizure (15) and is cumulative with successive treatments.

Several general reviews of the amnesic effects of ECT are available (16, 17, 18). This review will summarize the current evidence regarding the nature and extent of memory impairment. First, the anterograde amnesic effects of bilateral ECT will be compared to the effects of right unilateral ECT. Second, retrograde effects of ECT on remote memory will be reviewed. Third, the effects of extended treatments of ECT on memory will be summarized. Fourth, recent findings involving the reinstatement procedure will be described. Results with this procedure in animal studies suggested that learned material not ordinarily affected by electroconvulsive shock (ECS) may be forgotten if a reminder of the material is presented just prior to ECS (19, 20, 21). These reports raised the possibility that eliciting depressive ideation just prior to ECT could be therapeutically advantageous since ECT might produce amnesia for such ideation. Finally, memory capacity many months after ECT will be considered, in terms of objective and subjective estimates of ability.

Anterograde amnesia: bilateral vs. right unilateral ECT

It has been demonstrated that bilateral ECT produces a greater impairment of new learning capacity than right unilateral ECT (6, 7, 8, 9). Typically, however, learning ability has been assessed with verbal memory tests of the type particularly sensitive to dysfunction of the left cerebral hemisphere. The possibility has therefore remained that, if memory were assessed with nonverbal tests designed specifically to detect dysfunction of the right hemisphere, the amnesic effects of right unilateral ECT might be similar to or greater than the amnesic effects of bilateral ECT. In two studies of patients receiving bilateral or unilateral ECT (6, 22), impairment of "nonverbal" memory associated with bilateral ECT was slightly greater than the impairment associated with right unilateral ECT. However, in the absence of information about how patients with identified unilateral cerebral lesions would perform on these "nonverbal" tests, it is difficult to be sure how specifically sensitive these tests are to right unilateral hemispheric dysfunction.

Recently, verbal and nonverbal memory before and after ECT has been assessed in patients receiving bilateral or right unilateral treatment (23). To assess verbal memory, patients were read a short story and immediately thereafter were asked to recall as much of it as

possible. Delayed recall was tested on a second occasion 16 to 19 hours later. Patients with identified dysfunction of the left temporal lobe are known to perform more poorly on this test than patients with similar dysfunction of the frontal, parietal, or right temporal regions (24). To assess nonverbal memory, patients were asked to copy a complex geometric design (25, 26). Sixteen to 19 hours later, without forewarning, they were asked to copy it from memory. Patients with right temporal lesions are known to be deficient on this memory task, whereas patients with left temporal lesions exhibit no impairment (27). Tests were administered one to two days before ECT and again with equivalent forms six to 10 hours after the fifth treatment of the series.

Figure 1 indicates that patients about to begin a course of bilateral or unilateral ECT were nearly identical in delayed recall of the story and in delayed reproduction of the geometric figure. After ECT, bilateral ECT caused a greater impairment in both verbal and nonverbal memory than unilateral ECT. Delayed recall of the story was markedly impaired by bilateral ECT ($p < .01$), but not affected by right

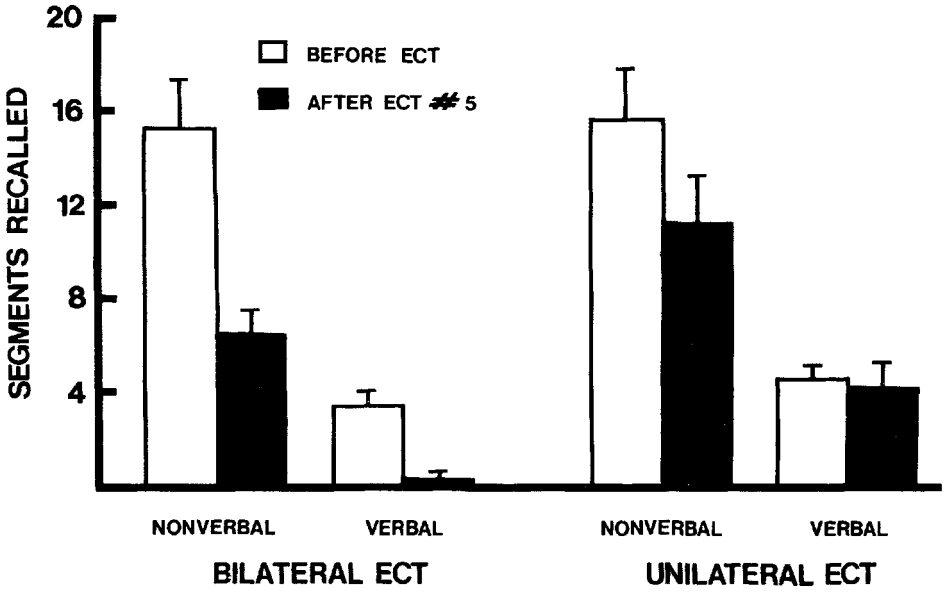

Figure 1. ECT and Memory Loss

Delayed retention scores on verbal and nonverbal memory tests for patients receiving bilateral ECT (N = 15) or right unilateral ECT (N = 9). Brackets indicate S.E.

unilateral ECT ($p > .3$). Scores of the bilateral and unilateral groups were significantly different ($p < .01$). Delayed reproduction of the geometric figure was significantly impaired by bilateral ECT ($p < .01$). In this case, the difference between the scores of bilateral and right unilateral groups was short of significance ($p < .09$).

It has sometimes been assumed that right unilateral ECT causes as much memory impairment as bilateral ECT on those aspects of memory function identified with the right hemisphere. The present results clearly indicate that bilateral ECT impairs memory to a greater extent than right unilateral ECT, regardless of whether the tests used to assess memory are more sensitive to left or right hemispheric dysfunction. This finding may mean that following unilateral ECT the unaffected hemisphere can always contribute to some extent to performance. This notion is supported by the observation that bilateral medial temporal surgery affects both verbal and nonverbal memory to a greater extent than left or right unilateral temporal surgery (27).

Retrograde amnesia for remote events: Information about public events

It has been reported frequently that convulsive therapy can cause retrograde amnesia for events that occurred close to the time of treatment (16, 17, 18). It is now clear that retrograde amnesia can also extend to events that occurred many years previously (8, 28, 29, 30). In such studies, remote memory is assessed with objective tests that ask about relatively familiar past events. In one test (29) patients were asked to recognize the names of former television programs that were broadcast for a single season from 1957 to 1972 (31). The programs selected from different time periods were apparently exposed to national audiences to about the same extent, and memory for these programs was acquired close to the time the programs were on the air (31). Patients prescribed a course of bilateral ECT took one form of this test before ECT and another form one hour after the fifth ECT. Figure 2 indicates that ECT caused a temporal gradient of impairment in long-term memory. Programs broadcast one to three years previously were forgotten; programs broadcast four to 17 years previously were remembered as well after ECT as before. The memory loss associated with bilateral ECT largely recovered by one to two weeks after the completion of treatment. Further work indicated that right unilateral ECT caused no deficit in remote memory, as measured by this test (29).

Other tests have confirmed the clinical impression that ECT produces a greater loss of temporal order information than other

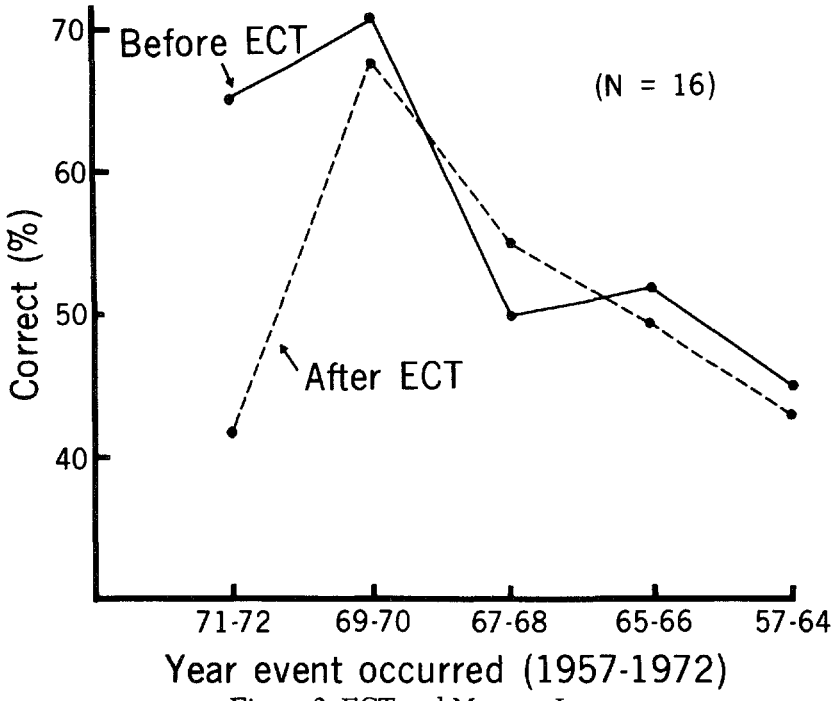

Figure 2. ECT and Memory Loss

A remote memory test was given before bilateral ECT and one hour after the fifth treatment. ECT selectively impaired performance on questions covering the 1971-1972 period. The test was given in early 1974.

aspects of memory (30). Patients saw sets of three television progam names and were asked to choose which was broadcast most recently. In each set of three the correct program name was broadcast for one season from 1962 to 1973 and the other two (incorrect) program names were broadcast five years previously, from 1957 to 1968. Following five bilateral treatments, patients developed a marked impairment in their ability to make temporal judgements about this material. The impairment was temporally graded, extending to events that occurred four to seven years before treatment, but not to events that occurred eight to 16 years before treatment. The deficit for temporal order information was more persistent than the deficit for recognition of program names, and remained unchanged at one to two weeks after the completion of treatment. Work is in progress to determine how long this deficit remains.

The clinical relevance of these findings is twofold. First, these results indicate clearly that the amnesia associated with bilateral ECT affects not only recent events, but can also extend to events that

occurred many years previously. Second, they indicate that bilateral ECT produces greater retrograde amnesia than right unilateral ECT. Right unilateral ECT caused no measurable loss of memory for remote events; by contrast, following a standard course of bilateral ECT, an impairment in memory for remote events persisted for at least two weeks.

Retrograde amnesia for remote events: Information about autobiographical material

The formal tests described above provide information about the effect of ECT on the ability to recognize public events that occurred from one to 16 years prior to treatment. There have also been some investigations of the effect of ECT on the ability to recall autobiographical material. In 1950, Janis (32) reported the results of a study frequently quoted by persons interested in and concerned about the effects of ECT on memory. Nineteen psychiatric patients who had been prescribed bilateral ECT, and 11 control patients not receiving ECT, were given a series of probing autobiographical interviews. These interviews concerned events relating to early schooling, job history, travel, the history of their mental problems, and other life experiences. The interviews were scheduled prior to ECT, four weeks after the completion of ECT (mean of 17 treatments), and again for five of the patients at 14 to 18 weeks after ECT. During the second interview, all patients who had received ECT exhibited amnesia for some of the material they produced in the first interview. Control patients exhibited virtually no amnesia. Finally, the five patients interviewed 14 to 18 weeks after ECT exhibited some recovery, but remained amnesic for some experiences (i.e., eight to nine experiences out of an unspecified number that had been produced during the initial interview). Since it has long been recognized that patients receiving ECT have persistent and probably permanent amnesia for much of the time period immediately surrounding the hospitalization period, it is important to ask to what time period did the memories belong that could not be recalled by Janis' patients. Unfortunately, because of limitations in this methodology and because of the design of the study, no general answer to this critical question is available. However, Janis did include five protocols as samples of persistent amnesia for pre-ECT events. An examination of these protocols indicates that for four out of five, the lost memories belonged to the time period just prior to hospitalization. For the remaining example, the lost memory seemed to relate to events that occurred sometime during the year preceding hospitalization. Accordingly, the

results of this study provide no evidence that memories acquired many years before ECT can be permanently lost as a result of ECT. The critical question remaining then is: How far back in time before ECT can the permanent amnesic effects of ECT extend? This question will be considered in the next section: Issues of test sensitivity.

Issues of test sensitivity

Studies with objective memory tests for remote events (28, 29, 30) show that ECT can cause a temporal gradient of retrograde amnesia covering the past one to seven years and that, where information is available about recovery, this effect can reverse spontaneously (with no reason to suspect relearning) within a few weeks after the completion of treatment. However, the multiple-choice tests on which these conclusions about recovery are based are not as sensitive to amnesia as tests that ask subjects to recall as much as possible about a past event (33). Tests are needed that ask subjects to recall specific, time-dated memories before and after ECT. Studies of this type now in progress confirm (1) that memory for more recent events (one to three years prior to ECT) are much more affected by ECT than memory for more remote events; (2) memory for remote events can be affected by ECT but clearly recover; (3) memory loss for very recent events may be permanent.

All the studies on retrograde amnesia for remote events following a conventional course of bilateral ECT can be summarized in the following way:

1. ECT can affect memories acquired many years prior to treatment;
2. the effect on memory is greater for recent memories and less for more remote memories;
3. very remote memories appear to recover fully following ECT in a manner that suggests that recovery is spontaneous and does not require relearning;
4. memories acquired during the days prior to a course of ECT may be permanently lost;
5. there is as yet no evidence to suggest that ECT produces permanent loss of memory for events occurring during the one or two years preceding ECT; indeed, there is some evidence to indicate that memories acquired during this period do recover. Nevertheless, a fully satisfactory study of this issue with maximally sensitive tests has not yet been accomplished.

Memory loss in patients receiving extended ECT

Most modern studies of ECT and memory loss concern patients receiving a conventional course of eight to 12 treatments. Accordingly, such studies do not speak to possible long-term effects on memory of an extended course of ECT (e.g., more than 50 treatments). Three studies have been reported that do assess memory capacity and other cognitive functions in patients who have received an extended course of ECT (34, 35, 36). These studies are retrospective investigations of patients who have in previous years received a total of more than 50 treatments. Such patients were compared to other patients matched as closely as possibly for age, sex, and psychiatric diagnosis. The results indicated that those patients who had received ECT performed worse on a variety of memory tests and other cognitive tests than the control group. However, these patients were either chronic schizophrenic inpatients who had been long-term inpatients or severely ill patients who had received cingulotomy in addition to ECT. In retrospective studies of this type, it is always difficult to know if differences between groups are attributable to ECT, or if those patients selected for extensive ECT were different from control patients before ECT in ways that affected their subsequent performance on neuropsychological tests. Accordingly, these studies cannot provide a conclusive answer to questions concerning possible permanent effects of extensive ECT.

Reinstatement

Normally, the severity of retrograde amnesia is inversely related to the time interval between learning and amnesic treatment. Several animal studies have suggested, however, that material not ordinarily affected by convulsive stimulation may be forgotten if a reminder of previously learned material is presented just prior to treatment (19, 20, 21).

To assess the reinstatement phenomenon with human subjects (37), inpatients receiving bilateral ECT learned material 18 hours before ECT or about ten minutes prior to ECT. Alternatively, they learned 18 hours before ECT and then were given a reminder a few minutes before ECT. Retention was always tested six to ten hours after ECT. Figure 3A presents results for a 32-item recognition task, and Figure 3B presents results for 18 paired associates. Patients learning 18 hours before ECT consistently exhibited better retention than patients learning only a few minutes before ECT ($p < .05$). Patients

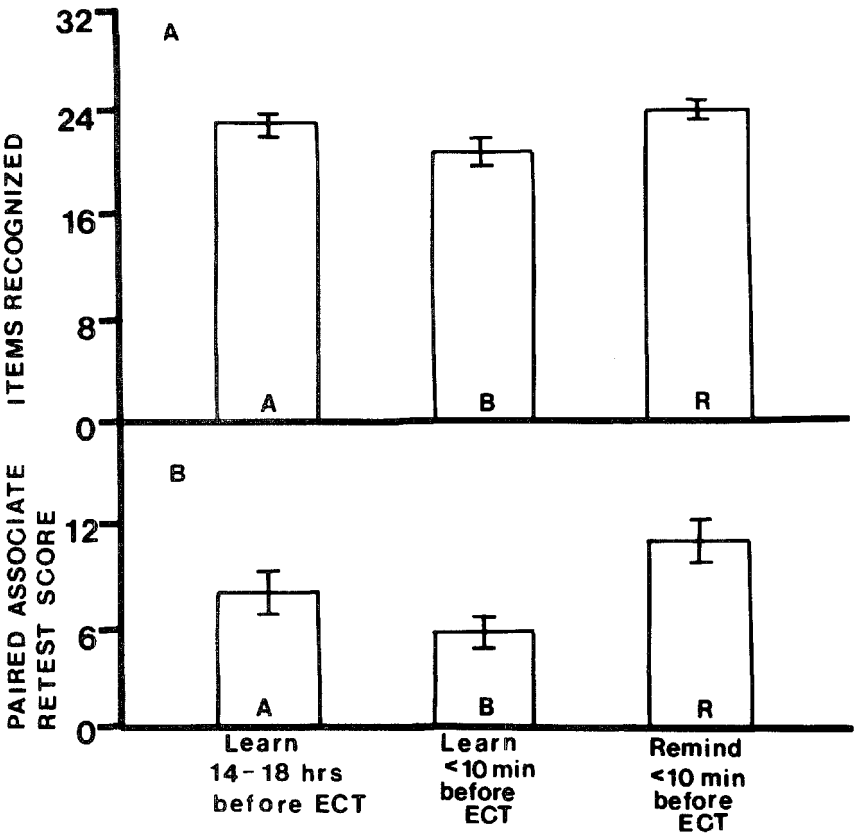

Figure 3. ECT and Memory Loss

Retention scores for 12 patients taking a recognition memory test (A) and a paired associate learning test (B). Learning occurred 14-18 hours before bilateral ECT (Conditions A and R) or just prior to ECT (Condition B). In Condition R, a reminder was given just prior to ECT. Retention was tested 6-10 hours after ECT.

given a reminder of previously learned material just prior to ECT retained this material as well or better than patients not given a reminder. Thus, recalling material from memory just prior to ECT did not produce amnesia. If anything, the reminder procedure improved retention. The results cannot rule out the possibility that amnesia might have been demonstrated if the interval between the reminder and ECT had been shorter than the three minutes required for administration of medications. Nevertheless, it is clear that amnesia need not occur even when a reminder is given at a time before ECT when memory for newly learned material is disrupted. This finding is of clinical interest because of the possibility that the reminder

procedure might be used advantageously with depressed psychiatric patients to improve the effectiveness of electroconvulsive therapy. The results of the present study provide no evidence that such a procedure would be effective in a clinical population.

Long-term effects of ECT on memory

A recent study reviewed the available literature on follow-up studies of ECT and memory and reported the results of a long-term follow-up study of patients who had received bilateral ECT, right unilateral ECT, or hospitalization without ECT six to nine months previously (38). Memory functions were assessed with six different tests of learning and remote memory capacity, and self-ratings of memory functions were obtained from all subjects. A group of inpatients was also included, who at the time of testing were receiving a course of bilateral ECT. This study can be summarized by stating that the three follow-up groups did not differ from each other on any of the memory tests. However, the group tested a few hours after the fifth bilateral treatment was consistently impaired. Figure 4 presents results for one of the memory tests. As might be expected, the inpatients performed more poorly than the other groups. Considerable forgetting occurred in all groups at one day and two weeks after learning, but there was no measurable difference between the retention scores of the three follow-up groups ($p > .3$).

Although no objective evidence could be obtained for persistent memory impairment long after ECT, subjects who had received bilateral ECT frequently felt that their memory was not as good as it used to be (38). Figure 5 presents additional data on memory complaints for a larger sample of subjects who had received bilateral ECT or right unilateral ECT six to nine months before. Of 55 persons who had received bilateral ECT (mean number of treatments = 9.9), 37 (67%) indicated that their memory was not as good as it used to be. By contrast, of 15 persons who had received right unilateral ECT (mean number of treatments = 9.4), only four (27%) felt that their memory was impaired. Such an asymmetry in the distribution of memory complaints of bilateral and right unilateral groups could have occurred by chance less than one in fifty times. Most persons with complaints felt that ECT was the cause of their memory problems. Eleven of the 37 persons who had complaints after bilateral ECT selected from four statements the one they felt best described their circumstances. None felt that they had "severe memory problems that interfere with almost everything I do"; two felt that they had "many memory problems that

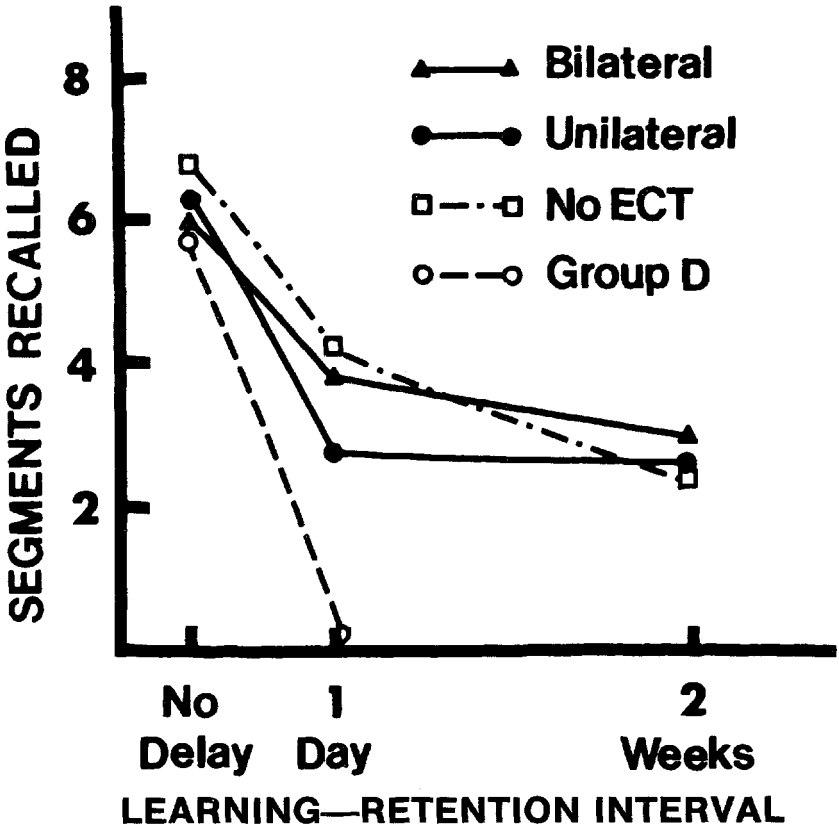

Figure 4. ECT and Memory Loss

Immediate and delayed recall of a short paragraph learned six to nine months after bilateral (N = 16), right unilateral (N = 10), or no ECT (N = 12). A group of inpatients, group D, (N = 15) was also tested 6-10 hours after their fifth bilateral treatment.

	Impairment	No Impairment
Bilateral ECT	37	18
Unilateral ECT	4	11

Figure 5. ECT and Memory Loss

Self-ratings of memory function six to nine months after bilateral or right unilateral ECT. Patients with memory complaints related only to the period of hospitalization were not scored as having perceived memory impairment.

are disturbing and that occur frequently"; six felt that they had "minor memory problems that occur frequently"; and three indicated that they had "only an occasional minor problem".

Unfortunately, the discrepancy between subjective and objective measures of memory function cannot be conclusively resolved. Three possible explanations of this discrepancy will be considered here. (1) The possibility cannot be ruled out that failures of recall persist after ECT that are not detected by conventional memory tests. (2) The possibility also cannot be ruled out that patients receiving bilateral ECT were different from patients receiving unilateral ECT in some way that favored the development of memory complaints. For example, patients receiving bilateral ECT might have initially been more depressed than patients receiving unilateral ECT, or they might initially have had different expectations about memory impairment. Thus, it should not be concluded that bilateral ECT will cause persistent memory complaints in any depressed patient. Whichever explanation is correct it seems clear that memory complaints long after ECT are common in persons judged clinically appropriate for bilateral treatments. (3) Finally, bilateral ECT might itself lead to a lingering sense of memory impairment. Thus, the marked impairment of recent and remote memory initially associated with bilateral ECT might cause some individuals to be more sensitive to subsequent failures in recall, even if they occur at a normal frequency. By this hypothesis, unilateral ECT, which causes less memory impairment than bilateral ECT, would not be expected to lead to memory complaints. Put in its strongest form, this explanation of memory complaints supposes that bilateral ECT might lead many individuals (with or without psychiatric illness) to have persistent illusion of memory impairment.

Summary

The findings reviewed above lead to the following general conclusions about ECT and memory loss:

1. bilateral ECT is associated with greater anterograde amnesia than right unilateral ECT, even when memory is assessed with tests known to be particularly sensitive to dysfunction of the right cerebral hemisphere;
2. bilateral ECT also produces more extensive retrograde amnesia for remote events than right unilateral ECT;
3. extensive ECT (e.g., more than 50 treatments) may lead to long-lasting or permanent impairment in memory capacity or

cognitive function, but a definitive conclusion is not yet possible;

4. the activation of previously learned material just prior to ECT does not cause amnesia for that material;
5. new learning capacity substantially recovers by six to nine months after the completion of bilateral or right unilateral ECT, but persisting memory complaints are common in individuals who receive bilateral treatment;
6. memory for events that occurred long prior to ECT substantially recovers by six to nine months after ECT; memory for events that occurred days prior to ECT may be permanently lost.

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Section II. *CNS (Central Nervous System) Sequelae of ECT: Risks of Therapy and their Prophylaxis*

Introduction

Epilepsy is a frightening illness for which robust therapies are generally prescribed. In the neurological literature, the negative effects of seizures are emphasized and concern is great to prevent them. In folk-lore, seizures are marks of ill-omen and epileptics are often segregated.

Yet in clinical psychiatry seizures are induced under controlled conditions for their effects on behavior. Since the mid-1930s, various ways have been used to elicit grand mal convulsions in mentally ill patients. Seizures are induced a number of times a week, and a treatment course may consist of four to eight seizures for depressive illnesses and up to 30 seizures for patients with more severe psychoses (1). The principal effects of repeated seizures (ECT) are a reduction in depressive affect, suicidal preoccupation, and delusional thoughts. Insomnia, anorexia, weight loss, irritability and loss of libido are also responsive to ECT. The efficacy and safety of ECT for appropriate psychiatric disorders are well described (2, 3, 4, 5, 6).

There is a discrepancy in attitudes between the psychiatrists who use ECT and the public and many professionals who do not. Anxiety concerning the hazards and potential misuses of ECT has led to legal restrictions in its use in some jurisdictions. When one reviews the criticism of ECT, few deny the efficacy of treatments for depressive psychosis, although there is less assurance in other conditions (4). The principal causes of concern are the risks of impairment of memory functions after ECT and alleged abuses, particularly the use of ECT to diminish complaining, negativistic, and hostile behavior. This latter concern is considered briefly elsewhere in this report.

The principal neurologic complications not infrequently attributed to ECT resemble the effects of head trauma to which ECT has been compared (7). In addition to headache and impairment of memory, spontaneous seizures, brain damage, and death are also alleged. This section examines the data in an attempt to answer the questions:

What are the central nervous system risks of ECT?

Are the neurological effects central to the therapeutic process of ECT?

Can these be reduced or modified?

The evaluation of ECT is complicated by historical factors. In the early decades, treatments were given without sedation, patients being brought to the treatment room, placed on a pallet, protected against the convulsions by two to four aides, and treated. Missed seizures occurred and patients experienced the panic and pain of unmodified electric currents or subconvulsive pentylenetetrazol (Metrazol). Treatment today is modified by special electrode placements, muscle relaxants, sedatives, anesthesia, and hyperoxygenation so that missed seizures, panic, fracture, and death are exceedingly rare. These modifications have been gradually introduced over four decades. As it became known that the cerebral seizure is necessary for the therapeutic response and that neither the motor convulsions, nor fear, nor memory loss was essential, treatments were modified and the use of sedation, anesthesia, and muscle paralysis using succinylcholine was generally accepted. Memory loss was also reduced by non-dominant unilateral placement of electrodes and by hyperoxygenation before and after the seizure.

The principal studies of the neurologic sequelae of ECT were done during the period of initial enthusiasm for the treatment when amnesia, apnea, hypoxia, and punishment were believed to be part of the therapeutic process. Instead of reducing their effects, these states were encouraged, with inestimable effects on neurologic functions. These data, although derived from different methods of treatment, are discussed in this section and their relevance for present methods are reviewed.

A. Data Regarding Sequelae of ECT

1. Impairment of Memory

Memory impairment is a consequence of seizures although its degree and persistence may be influenced by the currents with which the seizure is induced. During and immediately after equal numbers of treatments, changes in memory test scores are usually found to be equivalent when the effects of seizures induced electrically (ECT) are compared with the effects after the inhalant flurothyl (8, 9, 10, 11). But after the treatment course, there may be a greater improvement after flurothyl (12, 13, 14). Laurell (12) found equivalent effects on immediate and delayed recall tests but less retrograde amnesia after flurothyl, suggesting that the electric current had a small but measurable effect on forgetting. Differences in the degree of memory

impairment are also related to changes in current characteristics during ECT. An example is less memory impairment when brief, pulsed stimuli are used to induce a seizure (15) or when threshold currents are used (16).

In electrical inductions, the location of the electrodes has a direct effect on the type and extent of memory loss. Conventional electrode placement is bitemporal with currents concentrated in the anterior temporal lobes and the brain stem. Seizures may also be induced by currents through electrodes on one side of the head, (unilateral placement); and these may be placed over the dominant or the non-dominant hemisphere. The clinical efficacy of treatments using unilateral electrodes is approximately equal to the efficacy of bilateral placements, but the effects on memory are less (17, 18, 19, and Section I, this Chapter). Further, the impact on memory tasks differs depending on whether electrodes are placed over the dominant or the non-dominant hemisphere.

Missed seizures are one identifiable difficulty with these analyses. Small and Small (13, 20) and Laurell (12) suggested that the differences in efficacy of unilateral and bilateral electrode placements may be related to the number of partial or missed seizures which occur and which may occur more frequently with unilateral electrode placement. They suggest that EEG monitoring of each seizure would be one way to obviate this source of difference.

2. Spontaneous Seizures

Spontaneous seizures are convulsions that occur outside the treatment setting, days or weeks after the last treatment (21). Up to 1955, 51 cases were reported, when Blumenthal (22) added 12 cases. He estimated the incidence of spontaneous seizures after ECT to be 0.5%, similar to that recorded for epilepsy in the population. Karliner (23) reported six additional cases in patients without a history of epilepsy. He noted that the seizures remitted within three years and viewed their appearance as evidence of persistent brain dysfunction. More recently Assael, Halperin, and Alpern (24) reported a single case of a 30-year old woman without a personal or family history of seizures who developed a typical grand mal convulsion two weeks after the successful treatment of a catatonic stupor with four ECT. Seizures recurred once or twice weekly until anticonvulsant drugs were given.

It has been suggested that ECT may "kindle" an epileptic focus (25). "Kindling" is a phenomenon described in some animal species when small electric currents are passed through implanted electrodes in the brain stem, lowering the threshold to seizures such that incidental stimuli elicit spontaneous seizures (26). Pinel and Van Oot

(25) implanted electrodes aimed at the center of the amygdaloid complex, and stimulated male rats with 45, 1-second, 400 μ-Ampere, 60-Herz stimulations, three times a day, five days a week. They then intubated the animals and administered large amounts of alcohol for 45 intubations. When alcohol administration ceased, the withdrawal symptoms of hyperreactivity, clonus, and body jerks were intensified. Similar observations were made using repeated administrations of pentylenetetrazol and sub-convulsive amygdaloid stimulations. From these data they hazard warnings that similar increased sensitivity to seizures may occur in patients receiving ECT.

Inherent in the definition of kindling is a lowering of the cerebral threshold so that a seizure may be elicited by low, incidental stimuli. This explanation is inconsistent, however, with the observations by Brockman, et al (27) and Green (28) who found that the threshold for the currents necessary to elicit a seizure in ECT rises during the course of the treatment. Green found that the current necessary to elicit a seizure rose in 24/39 cases and showed no change in 15. In no case did the threshold for a seizure fall in his series. Brockman, et al (27) found that patients needed greater amounts of intravenous Azozol to induce seizures as the course of treatment progressed. The difference between ECT and the animal experiments cited in the studies by Pinel and Van Oot may be in their use of depth electrodes, the absence of anesthesia, the large amplitudes of their currents, the frequency at which the brain was stimulated, and the species used to study the phenomenon (1).

3. Organic Psychosis

The characteristics of an organic psychosis are impairment in orientation, memory, intellectual functions, and vigilance, with confusion, perceptual defects, lability of affect, and defective judgment as occasional symptoms. In a technical sense all induced seizures are associated with an "organic" impairment since memory loss and change in affect are common. But a severe organic psychosis has been described as occurring in some subjects (29). The psychosis disappeared within a few days, and the observers suggested that its appearance should not be interpreted as an adverse sign; particularly that the treatment should not be discontinued. These attitudes to the organic psychosis after ECT may have been conditioned by the earlier experience with insulin coma where a prolonged coma resulted in a prolonged confusional state, often with good clinical results when the confusion resolved.

These observations led some authors, notable Glueck (30) and more recently Murillo and Exner (31), to treat severely ill schizo-

phrenic patients three times daily to induce an organic psychosis in a process termed "regressive ECT." Glueck noted regression to be complete when a patient manifested memory loss, confusion, disorientation, lack of verbal spontaneity, slurring of speech to dysarthria or muteness, and apathy. In this special form of ECT the psychotic state is reversible, the long term clinical benefits to schizophrenia being equal to or better than those following drug therapy (31). A detailed description of the recovery process from a severe dementia due to frequent ECT has been reported by Regenstein, Murawski, and Engel (32). They noted that with cessation of ECT mental functions and behavior recovered over a period of more than one year.

A modification of regressive ECT was suggested by Blachly and Gowing (33) in which multiple seizures (up to six) were induced daily under conditions of hyperoxygenation. It is not clear why this method, termed MMECT (Multiple Monitored ECT) by these authors, rarely produced the severe organic psychosis noted by Glueck. In a series of 40 cases on MMECT, Abrams and Fink (34) noted two cases of confusion, and Strain and Bidder (35) reported a single such case, suggesting that oxygenation does not fully protect the patient.

An interesting aspect of the organic psychosis is seen in the syndrome of denial of illness (36). In neurological patients with brain damage, a syndrome of anosognosia, perceptual distortion, and changed language patterns may be elicited by barbiturates and defined as a measure of brain dysfunction (37). The same patterns of denial language and perceptual errors are seen after ECT, indicating that the post-ECT syndrome is qualitatively similar to other forms of diffuse cerebral dysfunction (38).

Another description of the organic mental syndrome was found in the report by a psychiatrist of his experiences through two courses of ECT. He noted the types of memory effects, particularly the differential impact on recent events, the topographical disorientation, and the changes in mood accompanying these treatments in eloquent detail (39).

4. "Brain Damage"

The evidence for sustained pathologic changes with repeated seizures comes from three sources: examination of brain tissues from patients who died after ECT and from epileptic patients who died in status epilepticus; tissues from animals subjected to repeated seizures; and the psychologic and physiologic tests which are usually interpreted as measures of an organic mental syndrome in man.

(a) Human brain tissue

The assessment of the pathology of ECT is complicated by the time between ECT and the time of death. In some studies, tissues are examined after seizures which are clearly proximal to the death; while in others, ECT is a distant event which may or may not be related to the pathologic examination. With this caveat, brain tissues have been reported to show increased gliosis (40); diffuse degeneration (41); petechial hemorrhages in the brain stem with fat embolism (42); and more commonly edema and subarachnoid hemorrhage (43, 44, 45). Will, et al (46) found the brain of a patient who died 15 minutes after the twelfth ECT to be edematous and to show neuronal damage and increased lipofuscin pigmentation. Madow (47) reported one case of intraventricular hemorrhage and three who died of cardiovascular disease in four autopsies of patients who died after ECT.

These cases raise the question as to the basis for death in ECT. The incidence of death varies from none in 8500 treatments in 870 patients (6) to 0.06, 0.08, 0.3 and 0.8% of patients treated (1, 48, 49, 50). Death is usually ascribed to cardiac complications, frequently occurring after the seizure during the recovery period and rarely during the seizure itself. The records of death rates show these to be higher in the early treatments which were usually given without modification. Some early studies used curare or flaxedil to relax musculature. But both these agents were unpredictable in their onset and duration of action, leading to irregular modification of seizures and the frequent occurrence of "missed" or incomplete seizures. In such events, the passage of electric current was not followed by a seizure or by post-ictal amnesia. Patients experienced pain, fear, and panic—psychologic events that may contribute to a cardiac death (51). In the pathologic reports cited above, missed seizures were associated as a prelude to a number of deaths (44, 46, 52).

Missed seizures and subsequent panic, fear, and excitement are no longer a feature of clinical ECT. Pretreatment sedation and anesthesia are routine, probably contributing to the reduction in the number of deaths occurring with ECT.

These pathologic data may also be compared to the findings in patients who died in status epilepticus despite the fact that it is difficult to separate the lesions which may be the cause of the seizure state from the anatomic changes which result from repeated seizures. A common finding in epileptics is sclerosis of the pyramidal cell layer of Ammon's horn (53), or diffuse necrosis, neurophagia, and gliosis (54). Norman, et al (55) suggest that repeated seizures lead to impaired

blood and oxygen supply and that the sequence of pathologic changes are secondary to hypoxia. Oxygenation is another factor in the present treatment procedure which differs from the early studies of ECT. Hypoxia and cyanosis were exaggerated to increase memory impairment since such impairment was believed to be the basis for the therapeutic efficacy of ECT. It is now clear, however, that when oxygenation is not provided the neurological sequelae of ECT are considerably greater than when oxygenation is provided (see Section III). Moreover reports of therapeutic efficacy for ECT using unilateral electrode placement with a reduction in memory impairment has made hypoxia no longer a feature of therapy, and indeed, hyperoxygenation is encouraged (33, 34). We can find no neuropathologic studies of ECT given with anesthesia, muscle relaxants, and hyperoxygenation (see Section III).

(b) Animal experimental data

When animals are subjected to experimental seizures, punctate hemorrhages and subarachnoid bleeding are seen (43, 56). Ferraro, et al (57) found degeneration of brain cells and gliosis in monkeys subjected to four to 18 seizures at a rate of three per week. Ferraro and Roizin (58) studied monkeys receiving an extensive course of 32 to 100 seizures with a follow up of ½ hour to 18 months and reported gliosis and cellular degeneration soon after treatment, but none after months. They concluded that these changes were reversible. Hartelius (59) studied the effects of seizures in cats and noted disintegration of nerve cells, neuronal loss, and glial reactions. These changes were not extensive and were related to the age of the animal and to the number of seizures. However, other workers, carrying out as extensive studies, failed to find either vascular or glial reactions to repeated seizures (60, 61, 62, 63).

It is difficult to determine the relevance of these studies since none of them involved the use of hyperoxygenation in conjunction with ECT and many of the parameters of the treatment (seizure rate and number, current strength, physiologic state, age, and anesthesia) are not comparable to ECT in man.

(c) Physiologic indices of Organic Brain Syndrome

The indices frequently used to define an organic impairment of brain-function are tests of orientation, memory, recall, confabulation, body image, and psychomotor performance; language measures; and electrophysiologic measures (EEG). These indices are highly intercorrelated and some have been discussed earlier.

The scalp-recorded EEG is a reliable and easily recorded index of

cerebral activity. Characteristic patterns have been defined and related to the diagnosis of epilepsy, organic confusional states, and cerebral impairment due to trauma, mass lesions, and cerebrovascular deficiency. These conditions are characterized by diffuse high voltage slow waves of two to six Hz in runs and bursts and in focal, asymmetric, or symmetric patterns (64, 65).

Following ECT, EEG slow wave activity increases, appearing in bursts and runs, more prominent in the frontal and temporal leads (66). Fast frequencies decrease and disappear (67). The degree of slowing, the increase in amplitudes, the duration of burst activity, and their persistence after the last treatment are directly related to the number and the frequency of seizure inductions and to the time of recording in relation to a seizure. Following the last seizure, the slow waves rapidly disappear, amplitudes decrease, and the mean frequency increases. Within four weeks of the last treatment, the EEG is filled with regular, rhythmic alpha activity, usually in amounts greater than that recorded before ECT (7, 66, 68, 69, 70). Occasionally, slow waves persist and may be recorded up to ten months after the last treatment (71).

The persistence of increased theta/delta burst activity has been related to the clinical efficacy of the treatment (66). Roth (72) and Roth, et al (73) reported that the early development of bilateral synchronous slow wave bursts elicited by intravenous thiopental was prognostic of a good clinical outcome with ECT. Ottosson (74) found that pretreatment with lidocaine reduced the amount of delta/theta activity developed with ECT and also reduced the therapeutic efficacy of the seizures. He asserted that the clinical efficacy was related to the duration of the seizure and not to the currents used to induce the seizure—a finding later confirmed in studies of flurothyl and the unilateral placement of electrodes. Ottosson noted that the severity of the memory deficits was related to the intensity of the currents used to elicit a seizure and not to the seizure itself. These studies suggested that there was an association between the duration of the seizure's electrographic effects and the clinical outcome, while the memory effects were more related to the other factors in the treatment. Analyses find the relationship between EEG slowing and improvement to be coincidental, not causal (70, 75, 84). From a theoretic viewpoint, synchronous slow wave activity in bursts is related to activity of the brain stem and it is to this region of the brain that much study is directed to understand the basis of the therapeutic process of ECT.

Studies of language measures (76, 77) and psychomotor performance (78, 79) find increasing deficits related to the number and frequency of seizures and a return to pre-treatment levels in the

weeks after the last seizure. Few studies relate the changes in these measures to therapeutic outcome, although the language measures were the focus of one novel hypothesis of the action of ECT. Weinstein, et al (37) found that following amobarbital, speech patterns of neurological patients exhibited the language of denial and predicted that the organic mental state elicited by ECT was conducive to the expression of explicit and implicit verbal denial—an anosognosia for the illness. Kahn, et al (38) and Kahn and Fink (77) found that denial language patterns did increase during ECT, that the denial language was related to pretreatment personality and diagnosis, and that denial early in treatment was prognostic of good clinical outcome. The changes in perception and language, like the changes in memory capacity, may be additional signs of the organic mental syndrome which may be uncorrelated to therapeutic outcome. (1).

B. Modification of Neurologic Effects

The repeated induction of seizures elicits a defined, though apparently reversible, neurologic syndrome which has many characteristics of the post-traumatic state including impairment of memory, organic psychosis, and spontaneous seizures. There is limited evidence for persistent brain damage and death from cerebral causes is rarely reported. The severity of these effects is related to the number, frequency, and mode of induction of seizures, the clinical diagnosis, and age. Frequency and number of seizures are the most critical.

These sequelae were more severe and more prevalent in the early days of convulsive therapy—when the techniques were being developed; and when the similarities and distinctions among ECT, Metrazol, insulin coma, psychosurgery, histamine shock, and atropine coma were blurred. Indeed, these different treatment approaches are still confused. Yet, ECT is as effective, or more so, in depressive psychoses than other treatments; it is as effective as antipsychotic drug therapy in acute schizophrenia; and in chronic schizophrenia its efficacy is suggestive (4; and see Chapter II). These neurologic sequelae are the principal deterrents to the use of ECT limiting its use to instances where the benefits outweigh the risks. Considering the advances in the understanding of the ECT process, we may materially reduce the risks by a more general application of the technical developments of the past two decades.

Panic, fear, and their probable associations with death may be reduced by pre-treatment sedation, as with secobarbital or diazepam. Anesthesia should be used as it is prophylactic for the pain, discomfort, and panic of a missed seizure.

Fracture and other consequences of muscular contractions may be relieved with succinylcholine. Its proper use provides the patient with a cerebral seizure without motor manifestations.

Sedation and anesthesia may also reduce spontaneous seizures. These seizures have become less frequent during the past few decades; while the reason for this is not entirely clear, the difficulty of inducing kindling in animals pretreated with barbiturates suggests that sedatives may reduce the possibility of spontaneous seizures.

Memory dysfunction and other manifestations of an organic psychosis remain the chief hazards of ECT. Hyperoxygenation, selective locations of ECT electrodes, and modifications of the currents used to elicit the seizure should be considered to reduce these manifestations.

The differences in the incidence of organic psychosis after multiple seizures is striking when one compares regressive ECT as used by Glueck (30) and Multiple Monitored ECT of Blachly and Gowing (33). The principal difference between the techniques lies in the importance Blachly and Gowing placed on hyperoxygenation—the need to maintain the patient's color as pink throughout the treatment—and their use of threshold currents. In the descriptions of treatments of the earlier period, patients usually became cyanotic and remained so during and after the seizure until they breathed spontaneously.

The differences in memory effects between unilateral and bilateral electrode placements are well documented (18, 19, 80, 81, Section I). Despite the experimental evidence, however, clinical reports emphasize a small difference in the clinical response between unilateral and bilateral electrode placements, asserting that unilateral ECT is slower and requires one or two additional seizures for equal efficacy. It is not clear whether the hazards of extra seizures and additional time for a satisfactory clinical response outweigh the benefits of reduced memory deficit, but at the least unilateral ECT provides a way to reduce memory dysfunction and organic psychosis.

Impairment in memory functions may also be reduced by modifying the parameters of the electric currents (15, 82, 83). These technical aspects are still under investigation but, if their promise is fulfilled, current type may be an additional parameter to reduce the adverse effects of ECT.

Summary

The effects of repeated inductions of seizures (ECT) are mood elevation, the relief of depression, and modification of vegetative

(hypothalamic) symptoms. These are usually accompanied by changes in memory, perception, and language.

The recognized adverse effects of ECT on the CNS include usually transient memory loss, a rare confusional state, and extremely rarely, death. Fear prior to each treatment and a mild headache following it are also sometimes associated with ECT.

The therapeutic process for depression lies in biochemical events which accompany or result from seizures, and not in the convulsion, memory loss or other neurologic sequelae. Modifications of the treatment process which maintain therapeutic activity and reduce side-effects are sedation, anesthesia, muscle relaxation, selective electrode placements and electric currents, and hyperoxygenation.

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Section III. *ECT: Possible Neurological Side-effects*

This brief commentary will concentrate on how electroconvulsive therapy (ECT) *might* injure the brain. Because relevant studies are inconclusive, it largely begs the prior question of whether modified ECT does in fact produce neurological damage. While a transient amnestic-confusional state follows most induced seizures, there is no firm consensus about ECT's long-term effects on cognitive functions. Rarely, acute and neurologically disabling strokes occur (1), but these are not the focus of recent controversy about the alleged adverse effects of ECT.

Current practice is to use paralytic agents and ventilatory assistance as adjuncts to ECT, the goals being to prevent fractures and dangerous hypoxemia. If respiratory function is not protected, one can of course predict brain injury and need not speculate as to why it occurs. Moreover, if patients are not well-attended during their post-ictal deliria, falls and head trauma may lead to brain injury. Apart from these rather gross departures from accepted standards, however, is the question of how modified ECT may produce permanent disturbances in memory or other higher brain functions. This is where the problem lies.

Clinicopathological studies

Comparatively old neuropathological studies of patients dying shortly after ECT disclose a variety of findings, ranging from no gross or microscopic changes through ischemic cell changes and small petechiae to major brain infarcts and hemorrhages (2, 3). Many deaths apparently resulted from cardiorespiratory problems, and most of the patients had not received muscle relaxants and systematic respiratory care. Thus, hypoxemia and the physical trauma of the unmodified generalized seizure may have been important contributions to the findings at autopsy. To our knowledge, no extensive neuropathological study of the long-term effects of "modern" ECT has been published.

A recent study of paralyzed and ventilated patients undergoing ECT indicated that no cerebral hypoxia occurred during the induced seizures (4). Thus, jugular venous oxygen tension remained constant and there was only a small decrease in venous pH, which could be accounted for by the observed rise in jugular venous CO_2 tension.

Jugular venous lactate rose slightly late in the seizure but levels of creatine phosphokinase remained stable. These data imply that during induced seizures cerebral metabolic demands did not outstrip cerebral oxygen delivery.

Patients who experienced spontaneous seizures less than three hours before evaluation had only a transient mild metabolic acidosis and a small rise in lactate levels in cerebrospinal fluid (CSF) (5). The rise in CSF lactate persisted for several hours or even a few days, but this was not associated with impaired consciousness or other abnormal neurological findings. In other words, a single spontaneous seizure, unmodified by paralytic drugs or ventilatory support, produced only minor chemical evidence of brain hypoxia and no clinical residua. While even a slight degree of brain hypoxia during a spontaneous seizure might injure some particularly susceptible neurons (e.g. those in the hippocampus), this hypothesis lacks satisfactory proof.

Possible mechanisms of brain damage

Because available clinicopathological studies do not clarify the basis of any long-term neurological side-effects of induced seizures, speculation dominates any formulation of pathophysiological mechanisms.

The most plausible mechanism seems to be ischemia-hypoxia. Apnea regularly occurs during generalized seizure and hypoxemia may follow. Respiratory depression is usually transient, however, and the above-noted studies of Brook and Adams (5) indicate that post-ictal biochemical changes ordinarily are slight. But if the apneic phase is unusually prolonged or if respiratory obstruction occurs during the seizure, the consequences may be more profound. Plum and co-workers found that adequate oxygenation allows the organism to meet the metabolic demands which seizures impose (6, 7). Thus, single generalized seizures in paralyzed but unventilated animals produced a substantial fall in cerebral venous oxygen tension, a metabolic acidosis in CSF, and a sharp rise in the concentration of lactate in brain tissue. If ventilation was provided, however, these changes were prevented.

Only where experimental animals are subjected to repeated generalized seizures over periods of from 30 minutes to five hours are persistent cerebral metabolic and morphologic abnormalities demonstrable (8-12). In primate studies, Meldrum and co-workers found that repetitive seizures induced by bicuculline or allylglycine produced ischemic cell changes, particularly in hippocampus (8, 9). But these

changes were not observed in all the animals, and tended to occur only during the phase of seizure when hyperpyrexia, hypotension, hypoxemia, acidosis, and occasionally hypoglycemia were present. These studies are consistent with findings in smaller animals suggesting that permanent disruption of cerebral energy metabolism occurs only after a prolonged series of repetitive seizures (10-12). There is no convincing experimental evidence that a single, brief, generalized seizure in a ventilated subject causes ischemic cell change or more than a transient disruption of cerebral energy metabolism.

Even if prolonged apnea or respiratory obstruction are averted, the circulatory accompaniments of a generalized seizure could lead to brain ischemia. While cerebral blood flow ordinarily rises substantially during seizures (13), this compensatory adjustment may not occur if cardiac output is impaired. In addition to clinical and experimental reports of sometimes fatal cardiac dysrhythmias following electrical stimulation of the brain (14, 15), a recent study in paralyzed and ventilated cats suggests that the marked rise in total peripheral vascular resistance which occurs during a seizure may reduce cardiac output (16). Thus "pump failure," whether due to cardiac dysrhythmia or left ventricular overload, may create a situation where cerebral blood flow is inadequate to meet heightened cerebral metabolic demands. Ischemic injury to cortical neurons might then follow. However, there are no studies which demonstrate that significant "pump failure" occurs in humans undergoing modified ECT.

Intracranial hypertension is another hypothetical mechanism of brain injury. Cerebral blood flow, and hence intracranial blood volume, increase during a generalized seizure (13, 17, 18). Also, there is evidence that the function of the "blood-brain barrier" (BBB) is disturbed during electrical stimulation (19). This combination of factors might conceivably lead to vasogenic brain edema, and perhaps even perivascular hemorrhages. But even if one assumes that a substantial increase in intracranial pressure occurs, the rise apparently does not persist after an uncomplicated seizure. There is no evidence to support the notion that repeated brief bouts of intracranial hypertension damage the BBB enough to adversely affect metabolism of cortical neurons.

One can further speculate that a series of induced seizures might produce morphologically undetectable injury to cortical neurons by altering properties of neuronal membranes or neuronal enzyme systems. Studies demonstrating that some areas of brain in experimental animals may be made epileptogenic by repeated electrical stimulation, the so-called "kindling" phenomenon, suggest that a large number of induced seizures might beget significant functional changes

in neurons (20). But the kindling model, which involves use of intracerebral electrodes, bears so little relationship to modified ECT that its relevance to the issue of cognitive change after ECT is dubious.

Summary

While the respiratory depression, heightened cerebral energy requirements, and dramatic cardiovascular changes that accompany even a modified generalized seizure could produce a gap between the brain's need for oxygen and the delivery of this essential fuel, no published study has established that such a gap develops. Indeed both clinical and experimental studies indicated that where adequate oxygenation is provided, brain tissue hypoxia does not occur during induced seizures. Mechanisms of neurological injury other than ischemia-hypoxia are even less easy to envision. Nevertheless, it seems proper to continue to investigate the question of whether ECT produces adverse morphological or metabolic effects in brain.

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CHAPTER V

ECT: METHODS OF ADMINISTRATION

Introduction

This chapter deals with the procedure by which ECT is administered. It presents information about the physiological and neuropharmacological events which are known to occur when a seizure and convulsion are evoked by an electrical stimulus. The adverse responses associated with the seizure and with the drugs employed in the treatment are discussed. And the psychological events which appear to be related to this form of treatment are presented.

The objective of this discussion is to present information which will enable ECT to be given in an effective, safe, and compassionate manner. This information, which is scattered throughout the medical literature, needs to be known to those who are involved in the treatment. The material should be available to psychiatrists who prescribe and administer ECT and who need to evaluate risks as well as anticipate and treat complications. This information should also be in the hands of internists and cardiologists who are frequently asked to clear patients for the procedure. And, finally, this material about ECT should also be known to anesthesiologists who are increasingly being involved as part of the team which carries out the treatment procedure.

This chapter concludes with a suggested step-by-step procedure for administering ECT. It is presented with an acknowledgement of the fact that there are other equally acceptable and safe procedures for ECT which differ in one or more details from that which is given here.

Physiological Considerations

This section is concerned with the physiological, neurophysiological and neuropharmacological events which are known to occur when a therapeutic seizure is electrically induced (1, 2, 3). These events can be described in two categories: central reactions and peripheral responses. The mechanisms in the brain responsible for the therapeutic and memory dysfunctional effects of the seizure are not fully understood (see Chapters IV and VI). However, the *known*

peripheral consequences of an electrically-induced seizure have one noteworthy attribute; they can be eliminated without, apparently, influencing the therapeutic response. This is indeed fortunate since most of the *acute* adverse reactions (e.g. cardiovascular, musculoskeletal) result from peripheralization of the central seizure process.

The known effects of an electrically-induced major seizure consist of an intensification of normally-occurring central and peripheral neuronal processes and the end-organ responses which result therefrom. These can be described with the aid of Figure 1:

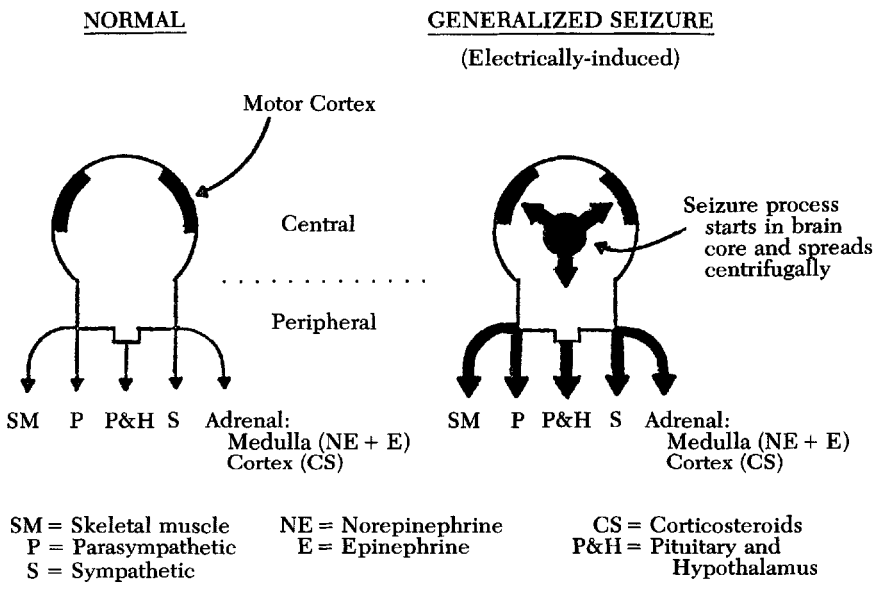

Figure 1

1. *Central Effects Of An Electrically-Induced Seizure*

(a) *Electrocorticographic effects*

Changes in the electrocorticograph are among the most striking manifestations of an electrically-induced seizure.

There is an immediate, striking change in the electrocorticographic pattern from the desynchronized resting configuration characteristic of the level of anesthesia being employed to a high-amplitude synchronized pattern (1).

Preconvulsive pattern

Immediate postconvulsive pattern

Depth recordings indicate that the seizure process is initiated in the central part of the brain and spreads centrifugally to the cortices (4). This is of clinical importance in view of the empirical observation that the therapeutic effect of the seizure is dependent upon involvement of a major portion, or even of the entire brain, in the seizure process. The occurrence of a tonic → clonic muscle response is indicative of the strength of the electrical stimulus being adequate to generate seizure activity of sufficient intensity to spread from the point of initiation, deep in the brain, to the motor cortices on its surface.

(b) *Effects on the cerebral circulation: acute responses*

Results from both animals and man indicate that there is a prompt increase in cerebral blood flow which reaches a maximum approximately three minutes after the seizure and progressively returns to normal during the ensuing 30 minutes (5, 6). The maximum increase in cerebral blood flow is of the order of 100% or more. These acute cerebral circulatory responses are chiefly the result of the systemic hypertension which characteristically accompanies an electrically-induced seizure (see below).

(c) *Effects on the cerebral circulation: sustained responses*

In patients whose psychiatric disorders are relieved by ECT, the clinical response is usually sustained. This fact has led to much work attempting to find physiological and biochemical changes which have a time-course parallel to the therapeutic response.

By employing cerebral impedance plethysmography, a non-invasive technique for measuring changes in cerebral blood flow, Lovett-Doust (7) demonstrated that increased cerebral blood flow induced by each treatment persisted for at least one week, especially in patients with depressive disorders who achieved a satisfactory response to ECT. Most of the schizophrenic patients in this study did not have a therapeutic response to ECT and did not get these persistent increases in cerebral blood flow.

(d) *Effects on cerebral metabolism: acute responses*

The significance of many animal studies on the short-term changes in brain metabolic parameters produced by electrically-

induced seizures has been complicated by the occurrence of varying degrees of hypoxia. These studies have indicated that the convulsion is associated with marked reduction in brain ATP and phosphocreatine as well as with an accumulation of lactate. The latter finding is interpreted as signifying that brain metabolism has shifted to an anaerobic pattern as a result of the marked increase in activity which exceeds the ability of the increased cerebral blood flow to supply oxygen in sufficient quantity.

The work of Plum and his co-workers (5, 8) marked a significant advance since they studied the changes occurring with an electrically-induced seizure in animals which were adequately ventilated with oxygen-enriched mixtures so that hypoxemia and hypercarboxemia were prevented. They found that, in anesthetized and paralyzed animals adequately ventilated with oxygen-enriched mixtures, no significant brain hypoxia occurs and there is no shift to anaerobic metabolism. This is in striking contrast to the severe brain hypoxia, marked depletion of high-energy metabolic intermediates, and concomitant anaerobic metabolism which is characteristic of animals that develop hypoxemia.

These results have much practical significance for the administration of ECT. In view of the fact that substantial hypoxia is not a requirement for ECT to exert its full therapeutic effect, there is no need to tolerate hypoxemia. And there are at least two positive reasons *not* to have hypoxemia and hypercarboxemia occur if at all possible. First is the uncertain relationship of severe cerebral hypoxemia to the memory dysfunction and confusion which occur with ECT. Second is the fact that hypoxemia and hypercarboxemia have a potent cardiac dysrhythmiagenic action (see below).

The foregoing considerations constitute a strong recommendation that those who administer ECT possess the skills and employ the proper equipment, so that adequate ventilation—preferably with oxygen-enriched gases—is maintained at all times during the procedure except, of course, during the period when convulsive muscular movements render this impossible.

(e) *Effects on cerebral metabolism: sustained responses*

Most of this work has involved brain monoamines and the enzymes associated with their synthesis and disposal. Increased turnover of brain norepinephrine and increased tyrosine hydroxylase activity in norepinephrine-rich areas of the brains of experimental animals have been reported to occur with a series of

electrically-induced convulsions (9, 10, 11). However, these changes are of comparatively short duration.

More persistent changes have been reported. After a series of convulsions, an increase in brain monoamine oxidase activity persists for several weeks after the last convulsion (12, 13). In rats given once-daily, electrically-induced seizures for a week, increased tyrosine hydroxylation persists for a week following the final convulsion (14). The same investigator observed a sustained increase in brain tryptophane concentration which occurred without significant changes in the synthesis, turnover, or uptake of 5-hydroxytryptophane.

2. *Peripheral Effects Of An Electrically-Induced Seizure*

Many of the effects of an electrically-induced seizure occurring external to the central nervous system can be viewed as being consequences of the peripheralization of the seizure process via the available neuronal circuits (Fig. 1).

(a) *Musculoskeletal effects*

The skeletal muscle movements, which are the hallmarks of a grand-mal convulsion, result from involvement of the motor cortices in the seizure process. These peripheral motor manifestations can be totally obliterated by means of succinylcholine and other muscle paralysants without discernible effects on the therapeutic action of ECT.

Skeletal muscle convulsive movements have both negative and positive attributes in the patient receiving ECT. Thus they are responsible for the musculoskeletal injuries which were such disturbing adverse effects of ECT before the introduction of succinylcholine (15).

However, the occurrence of a sequence of tonic → clonic skeletal movements constitutes a reliable, simple, and practical method of ascertaining that the seizure process has been sufficiently generalized to evoke a therapeutic response. But this end-point is not infrequently obscured when the estimated dose of succinylcholine is too large. This situation is very likely to occur when larger doses of the drug are being used in attempts to prevent fractures in elderly patients with osteoporosis.

The positive, informational aspects of the convulsive muscle movements can be retained while their negative, injurious potentialities can be obliterated by a simple maneuver based on a

principle described by Claude Bernard in 1856 (16, 17). The maneuver consists of the implacement of a blood pressure cuff so as to occlude an artery in the leg (popliteal fossa) or in the arm (around the humerus). The cuff is inflated to, or slightly above, the arterial pressure just prior to injecting the succinylcholine. This procedure will insure that the "cuffed" extremity will clearly and reliably show the following sequential muscle movements:

(i) *tonic* contraction occurring during the time that the electrical current is flowing. This muscle movement by itself does not signify that a therapeutic seizure has been, or will be, evoked.
(ii) *tonic* contraction occurring, or continuing, after the electrical stimulus has ceased. This is replaced by a . . .
(iii) *clonic* contraction. The occurrence of movements (ii) and (iii) indicates that the electrical stimulus has been of an intensity sufficient to evoke a generalized, therapeutically-effective seizure.

By using this maneuver the convulsive muscle movements in the rest of the body can be completely eliminated, if desired, by use of sufficiently large doses of succinylcholine. This degree of immobilization is essential if ECT is to be used in patients with disease (e.g. aseptic necrosis of the femoral head) or injury (e.g. fracture, laceration) of one or more extremities.

(b) *Parasympathetic effects*

There is peripheralization of the seizure process over cardiac parasympathetic nerve circuits. The most visible evidence of this discharge is the bradycardia which appears concomitant with the onset of an electrically-induced convulsion in unmedicated animals and man (18, 19, 20). This bradycardia, which can be severe enough to produce asystole of several seconds' duration, can be completely eliminated by adequate cholinergic block with atropine or scopolamine.

The bradycardia is of importance because it is a manifestation of parasympathetic (cholinergic) inhibition of the S-A Node. Other potential atrial pacemakers (e.g. A-V Node) or ectopic pacemakers (e.g. ventricular irritable foci associated with myocardial disease) are not significantly inhibited by cardiac vagal activity. The result is that seizure-induced reduction in S-A nodal activity can eventuate in its cardiac pacemaker function being usurped by an ectopic focus.

(c) *Sympathetic effects*

Peripheralization of the seizure process over sympathetic nerves results in release of norepinephrine in the heart. The resulting adrenergic effects summate with those resulting from release of catecholamines from the adrenal medulla. The combined adrenergic effects emanating from these two sources will be described in the next section.

(d) *Adrenal medullary effects*

Peripheralization of the seizure process over fibers innervating the adrenal medulla results in release of catecholamines into the venous circulation and, within one circulation time, adrenergic effects on heart and blood vessels. These effects, which summate with those resulting from direct cardiac sympathetic discharge (see above), include hypertension (average increases of 50-100 mm Hg) and tachycardia (average increases of 20-50 beats per minute) (20). Plasma norepinephrine and epinephrine concentrations are markedly elevated (10-50-fold) within one minute after the seizure is initiated (20). There is a rapid decline in the second minute with a more gradual return to near pre-seizure concentrations within the next five minutes. The post-seizure hypertension, tachycardia, and cardiac dysrhythmiagenic susceptibility decline parallel to the falling plasma concentrations of catecholamines.

These pressor responses can cause certain adverse reactions (e.g. stroke), particularly when they are exaggerated or when they occur in patients whose blood pressure is already elevated. It is of interest, therefore, to know that their intensity can be significantly reduced by the use of adrenergic blocking agents and with ganglionic blockers (20).

(e) *Adrenal cortical effects*

Peripheralization of the seizure process over nerves innervating the adrenal cortex is one cause for the elevation of blood cortical steroids which occurs in the human during the two to four hour period following an electrically-induced seizure (21, 24). The other contributing stimulus is the elevation in blood corticotropin concentrations which occurs following an electrically-evoked seizure (see below).

The clinical consequences, if any, of these elevated corticosteroid concentrations are apparently not significant even when convulsive therapy is administered thrice weekly for 20 or more total treatments. It is of interest that convulsive therapy can retain

its therapeutic effectiveness in surgically adrenalectomized individuals (22).

(f) *Pituitary effects*

Elevations in blood concentration of several pituitary hormones in the period immediately following an electrically-induced seizure have been documented. These elevations can be viewed as possible consequences of the "peripheralization" of seizure activity through the pituitary gland.

The following pituitary hormones have been measured in the blood following an electrically-induced convulsion:

(i) *adrenocorticotrophic hormone* (corticotropin, ACTH)
An eight-fold increase in plasma ACTH concentration occurs following single or multiple convulsive treatments in patients who were ventilated with 100% oxygen, anesthetized with a short-acting barbiturate, and totally paralyzed with succinylcholine (23). The ACTH concentration peaks 2.5 minutes after the electrical stimulus is applied and returns to normal in the following 45 minutes. Virtually identical results were obtained in anesthesized patients given a single convulsion without the use of muscle relaxants (24).

(ii) *human growth hormone* (HGH)
A number of studies of human serum growth hormone concentrations following single or several convulsive treatments have disclosed no significant changes (23, 24, 25, 26, 27). However, one group studying an all-female population of patients given convulsive therapy without *any* drugs demonstrated marked (5-10-fold) increases following each of several seizures. Peak concentrations were attained 30 minutes after the convulsion and normal levels were reached two and one half hours later (28).

(iii) *other pituitary hormones*
No changes in plasma concentrations of thyrotropin (TSH), follicle-stimulating hormone (FSH) or luteinizing hormone (LH) have been observed in the time interval immediately following an electrically-induced convulsion in man (23, 24).

(g) *Hypothalamic effects*

(i) *Prolactin*
A 10-50-fold increase in serum prolactin concentration occurring 15 minutes following an electrically-induced convulsion was found in eight of nine female patients given atropine,

thiopental, and succinylcholine (29). Age, concomitant psychotropic drug administration, duration of the seizure, number of treatments, and therapeutic response did not interfere with this prolactin response

Adverse Responses Associated With ECT

The following discussion will deal with a select group of adverse reactions which occur during the treatment and immediate post-treatment periods. Adverse responses occurring in these two periods can be conveniently divided into those which are related to the electrically-induced seizure and those associated with the drugs commonly used in the treatment. This separation is not absolute: several adverse reactions have their genesis in both the drugs and the seizure.

1. *Adverse Responses Related to the Electrically-induced Seizure*

(a) *Cardiac Dysrhythmias*

There is substantial evidence that circulatory disturbances are the most important and dangerous complications of ECT (30, 31) and that their incidence and seriousness is disproportionately increased in older individuals and in persons with recognized or unrecognized cardiovascular disease (20).

The occurrence of cardiac dysrhythmias with modified convulsive therapy (i.e., where intravenous anesthesia and succinylcholine are used) is a function of the various factors to be discussed presently—but principally of the use, or non-use of anticholinergic drugs, of the adequacy of pulmonary ventilation and of the presence or absence of significant coronary artery and myocardial disease. Not surprisingly, then, the reported incidence of cardiac dysrhythmias varies widely. Pitts and his co-workers reported an 8-41% incidence of ventricular dysrhythmias depending on the barbiturate used and upon the presence or absence of cardiac disease (32).

Richardson (33) found cardiac dysrhythmias occurring in 30% of patients receiving ECT under general anesthesia. In a series of 21 guarded-risk patients (20) given 220 treatments with electrocardiographic monitoring and expert anesthetic management, cardiac dysrhythmias were encountered in 75% of the patients and in 47% of the treatments. Fifteen per cent of the dysrhythmias were mild, 50% were of moderate severity, 27% were severe and 8% were very severe.

Cardiac arrest appears to be a leading, if not the major, cause of deaths occurring with the administration of convulsive therapy (35, 36, 37).

Rather than trying to arrive at some precise value for the incidence of cardiac dysrhythmias with ECT, it would appear to be more useful to emphasize the importance and consequences of this as a potentially serious adverse reaction which can complicate the treatment—even of individuals not judged to be guarded cardiovascular risks (20, 34, 37, 38, 39, 40, 41, 42, 43).

Of equal importance with a recognition of the potential for cardiac dysrhythmiagenic complications in ECT is an understanding of the mechanisms whereby these particular adverse effects are generated. This understanding is of particular value since it makes possible prophylactic strategies, anticipation and early recognition of these adverse responses and, most important, effective therapy.

The major mechanisms involved, either singly or in combination, in the genesis of cardiac dysrythmias are the following:

(i) *those involving inhibition of S-A nodal activity*

Cardiac muscle contractions initiated by the normal pacemaker, the S-A Node, provide optimal hemodynamic work. Inhibition of S-A nodal activity can result in take-over of pacemaker activity by the A-V Node or by irritable ectopic pacemakers in the ventricular myocardium with the potential for hemodynamic incompetence (e.g. ventricular fibrillation). S-A nodal activity can be inhibited by the following:

The intense outflow over the cardiac parasympathetic nerves which accompanies the electrically-induced convulsion;

The cholinergic agonist activity which precedes the cholinergic blocking action of certain drugs:

atropine and related anticholinergic drugs

These agents, which effectively block cholinergic inhibition of the S-A Node produced by nerve action when used in adequate doses will, paradoxically, produce a bradycardia in many patients when used in smaller doses (< 0.4 mg) (44).

succinylcholine

The cholinergic agonist action of this agent, which is so visibly apparent as the muscle fasciculations occur-

ring prior to paralysis, is manifest on the S-A Node as the bradycardia which the drug characteristically produces prior to its paralytic action. This bradycardia, which is likely to be especially severe with successive doses of succinylcholine, can be effectively reduced or prevented by adequate doses of scopolamine or atropine (45, 46).

(ii) *those producing localized or generalized ventricular myocardial irritability*

coronary artery disease or insufficiency

This may produce relative or absolute ischemia of the myocardium. Relative ischemia is produced when coronary perfusion is not adequate to keep pace with the increased metabolic needs of hyperdynamic myocardial activity (e.g. during the tachycardia associated with seizure activity). Absolute ischemia occurs with gross coronary arterial insufficiency (e.g. occlusion with infarction). Ischemic myocardial tissue, even when small in size, becomes irritable and may assume ectopic pacemaker activity and generate cardiac dysrhythmias (e.g. premature ventricular contractions, ventricular fibrillation).

increased cardiac sympathetic or adrenergic activity

This may be generated by peripheralization of seizure activity over cardiac sympathetic nerves or by the catecholamines liberated from the adrenal medulla (see above).

hypoxemia and/or hypercarboxemia

Either alone or especially in combination these are potent stimuli for increasing ventricular irritability and triggering cardiac dysrhythmias (47).

When pulmonary ventilation is inadequate (e.g. assisted respiration not employed prior to the convulsion), both hypoxemia and hypercarboxemia develop. When oxygen-enriched mixtures are used but where *ventilation* is inadequate (e.g. due to poor airway management), dangerous degrees of hypercarboxemia may exist in the absence of cyanosis and other visible signs of hypoxemia.

hyperkalemia

Under certain circumstances, the hyperkalemic response which occurs with succinylcholine is sufficiently marked so as to produce cardiac arrest (see below).

2. *Adverse Reactions Associated with Drugs Used in ECT*

Three types of drugs are commonly used in the administration of ECT: an anticholinergic agent, an intravenous central nervous system depressant, and a short-acting muscle paralysant. Each of these drug types has the capacity for evoking significant adverse reactions.

(a) *Anticholinergic Agents*

The major use of an anticholinergic agent in ECT is to prevent the bradycardia-asystole accompanying the seizure. The capacity to reduce the volume of pharyngeal secretions is a useful secondary property of these drugs.

The actions of cholinergic blockers on the S-A Node are not only therapeutic—they can contribute toward the development of adverse responses as well. Thus, the initial bradycardia which can occur with low doses may intensify the cardiac slowing which occurs during the initial phase of the action of succinylcholine, and which is associated with the seizure. However, the increase in heart rate is the most prominent action of an anticholinergic drug. This tachycardia can cause difficulties in individuals with significant coronary disease, especially when the heart rate increase is marked (i.e. dose of atropine excessive) and when it summates with the adrenergic cardiac effects occurring with the seizure. In these individuals and under these circumstances, the increased rate of cardiac contraction can increase the need for blood supply beyond a coronary vessel's capacity to deliver it. As a result, focal areas of the myocardium may develop relative ischemia and consequent hyperirritability and serve as a focus for ectopic cardiac dysrhythmias. When a biotitrative technique (i.e. administering incremental doses of the anticholinergic drug until a 10% increase in heart rate has been attained) is employed, excessive tachycardia and the complications which it engenders can be minimized.

A second adverse reaction, which is restricted to atropine sulfate and other anticholinergics which cross the blood-brain barrier, is the development of a post-treatment Central Anticholinergic Syndrome or toxic psychosis. This is particularly likely to occur in patients who are also receiving other drugs which have cholinergic-blocking actions (e.g. antipsychotic drugs, antiparkinson agents, tricyclic antidepressants). The possibility of this side-effect can be reduced by using quarternary anticholinergics which, being charged molecules, do not cross the blood-brain barrier (48, 49).

(b) *Intravenous Anesthetics*

These are almost always ultra-short-acting barbiturates such as thiopental (Pentothal) and methohexital (Brevital). The use of intravenous anesthetics is associated with all the formidable risks of general anesthesia. It is particularly important to appreciate the fact that light levels of anesthesia produced with low doses of anesthetic agents are particularly likely to be accompanied by such complications as laryngospasm. Little doses do not necessarily beget little troubles!

Two additional adverse responses to intravenous anesthetics are rare but serious. The first of these is accidental intra-arterial injection of the barbiturate with subsequent tissue necrosis and severe deformity of the hand (50, 51, 52). The second is a rapidly-developing hyper-sensitivity reaction. Anaphylactic reactions to thiopental are well-documented (53, 54). They have also been reported with methohexital (55).

(c) *Muscle Paralysant*

Succinylcholine is the near-universal choice of a drug to attenuate the force of convulsive skeletal muscle contractions and thereby markedly to reduce the incidence of musculo-skeletal injuries.

There are several important and potentially serious adverse reactions which can occur with succinylcholine even when the drug is used in very small doses.

(i) *modification of duration of action of succinylcholine*

The usually brief (five to seven minutes) duration of the paralytic action of succinylcholine is a consequence of the rapid hydrolysis of the drug by plasma pseudocholinesterase(s). Drug-induced modification of the enzyme's hydrolytic activity as well as genetic variation in the enzyme's structure and function are known to occur with resulting clinically significant modification of the duration of action of succinylcholine. In response to these factors, the skeletal muscle paralysis produced by the drug may be substantially lengthened (for hours!), or significantly shortened, in comparison with the five to seven minute duration of action which occurs in the great majority of patients who receive intravenous succinylcholine.

increased duration of action

This occurs with two general types of etiological factors

which impair the hydrolytic activity of plasma pseudocholinesterase(s): drug inhibition and genetic factors.

drug inhibition

A number of potent blockers of plasma pseudocholinesterases (i.e. anticholinesterases) are currently employed in medicine in the therapy of myasthenia gravis (neostigmine, pyridostigmine [Mestinon], amebonium [Mytelase]) and in the treatment of glaucoma (diisopropyl fluorophosphate [DFP], [Isoflurophate], tetraethylpyrophosphate [TEPP], echothiopate [Phospholine], bisneostigmine [Demecarium, Humorsol]). In addition, a number of insecticides are potent anticholinesterases (e.g. parathion, malathion). The duration of anticholinesterase action varies greatly. The agents used for the therapy of myasthenia gravis generally have a duration of action of a few hours and, when stopped two or more days prior to the administration of succinylcholine, will not significantly inhibit its hydrolysis or prolong its paralytic action.

The organophosphate anticholinesterases used in the therapy of glaucoma and those employed as insecticides are very long-acting agents whose effects last from a few days to several weeks! There have been reports that significant systemic effects resulting in marked prolongation of the action of succinylcholine have occurred when drugs of this type have been instilled into the conjunctival sac of humans! (56).

genetic factors

The structure and, consequently, the functional activity of plasma pseudocholinesterases are under genetic control (57, 58). The following three general types of plasma pseudocholinesterase are clinically important in determining the duration of action of succinylcholine:

(a) *typical form*

The great majority of humans have an enzyme which is exclusively of this type—which accounts for the fact that, with very few exceptions, the muscle paralyzing actions of intravenous succinylcholine last only five to seven minutes. The typical enzyme's hydrolytic capacity is so great that, in individuals

who are homozygous for this type, amounts of the drug considerably in excess of the usual 0.5-1.0 mg/kg can be given without appreciable prolongation of its paralytic action.

(b) *atypical form*

This type is essentially devoid of hydrolytic action on succinylcholine when it is administered in the usual therapeutic doses. About one individual in 3,200 is homozygous for this form of the enzyme; administration of even minute doses of succinylcholine (e.g. 2-4mg) to these patients can result in respiratory insufficiency or apnea lasting 15 minutes or longer (59).

(c) *C-5 Form*

This type is a rare type of plasma pseudocholinesterase which hydrolyzes succinylcholine at a rate 30% greater than that of the typical enzyme (58).

decreased duration of action

Patients with substantial amounts of the C-5 form of the enzyme are apparently those rare individuals who get virtually no paralytic action from succinylcholine because of the great rapidity with which the drug is hydrolyzed. Although this is a rare variant, its existence should be known to those administering ECT since, in these patients, usual therapeutic doses of succinylcholine will not significantly attenuate the force of muscle contractions occurring during the convulsion.

additional facts about genetic factors

First, there is evidence that abnormal rates of succinylcholine hydrolysis are much more common in patients who receive the drug for ECT than in those who get the drug for general surgery (60). Second, individuals can be homozygous for any one of the forms (i.e. her/his pseudocholinesterase is of one type) or else heterozygous (i.e. has a mixture of the various forms described above). Heterozygotes involving the atypical enzyme are particularly hazardous since, in these individuals, succinylcholine can produce respiratory muscle *weakness* rather than the readily recognized complete apnea which

occurs when all, or nearly all, of the plasma pseudocholinesterase is of the atypical type. This emphasizes the need for careful monitoring of tidal volume in the immediate post-treatment period and for ventilatory assistance when required.

The third fact involves the unchanging genetic nature of an individual's plasma pseudocholinesterase throughout life. Thus, an individual who has previously responded in a normal manner to succinylcholine can be expected to react similarly in the future unless, of course, there has been recent exposure to an anticholinesterase.

laboratory determination

Fortunately, there is a relatively simple and inexpensive laboratory test which can be used to measure an individual's plasma pseudocholinesterase-hydrolyzing capacity and, thereby, to estimate the duration of the paralytic action of succinylcholine (61). Two laboratory values can be obtained: the rate of hydrolysis in the absence and in the presence of certain inhibitors such as dibucaine or fluoride. The typical and atypical forms of the enzyme are inhibited in characteristic manner by these agents (62, 63).

Some therapists routinely obtain plasma pseudocholinesterase activities on all patients who are to receive succinylcholine. Others reserve the determination for the following types of patients:

(a) in guarded-risk patients (e.g. very old individuals and those with significant cardiovascular and/or bronchopulmonary disease) in whom a prolonged period of artificial ventilation might prove hazardous;

(b) blood relatives of individuals known to have had abnormal responses to succinylcholine;

(c) patients who have had, or who are suspected of having had, abnormal responses to past administration of succinylcholine;

(d) patients who have recently been taking anticholinesterase medications for myasthenia gravis or glaucoma as well as those who may have had occupational or other types of exposure to these agents.

(ii) *hyperkalemic response to succinylcholine*

Potassium liberation occurs as part of the normal action of succinylcholine. However, under certain conditions this release is so marked that it produces hyperkalemia sufficient to evoke cardiac dysrhythmias and even cardiac arrest (64, 65). Succinylcholine-induced hyperkalemia becomes a serious problem when the drug is administered to patients 14-21 days after massive thermal body burns, direct muscle trauma, upper motor neurone lesions (associated with multiple sclerosis, spastic paralysis from brain injury) and to those who have been immobilized for long periods of time. This exaggerated response to succinylcholine may persist for up to six months after an injury. It is not rare to consider the use of ECT in patients who have one of these conditions predisposing to the development of succinylcholine-induced hyperkalemia and resulting cardiac complications. In such cases it is advisable to carry out the treatment with electrocardiograph monitoring and with the immediate availability of drugs, equipment, and personnel necessary to manage cardiac dysrhythmias and cardiac arrest adequately.

(iii) *interaction with lithium*

There is some evidence that lithium prolongs the duration of the paralytic action of succinylcholine (66). The activity of plasma pseudocholinesterase does not appear to be changed by lithium.

(iv) *anaphylaxis* has been reported with succinylcholine (67).

Psychological Considerations

There is virtually no attention given in the psychiatric literature to patients' psychological reactions to the ECT treatment *procedure.* This lack of formal attention to emotional responses stands in marked contrast to the intense feelings about this form of treatment which exist within and without the psychiatric profession.

The evidence for concluding that ECT is an emotionally stressful and frightening experience for many or most patients to whom it is administered is, unfortunately, largely anecdotal. Those individuals who have contact with patients receiving ECT become very familiar with the apprehension and fear which surround the procedure; and these patient responses are reflected in the opinions of some psychia-

trists who, despite evidence to the contrary, remain convinced that the treatment works *solely* through this aversive action which, in effect, terrorizes the patient into a "flight into health."

However, there is some objective support for the observed emotional stress associated with ECT. Thus studies have shown that the plasma non-esterified fatty acid concentrations become markedly elevated in the period immediately prior to a treatment in comparison with concentrations on days when no ECT is given (68). Elevations in non-esterified fatty acids are considered to result from increased plasma concentrations of catecholamines evoked by fear and stress (69).

Despite the paucity of objective data, it is desirable to try to describe some of the specific fears which appear to be important in patients receiving ECT. First, though, a word should be said about the relationship of these fears to the memory dysfunction which is associated with currently used techniques of administering ECT. It is common to encounter patients who have marked anxiety prior to a treatment session but who later had no recollection of these feelings. This experience has led to the widespread assumption that *all* patients have retrograde amnesia for events occurring in the pre-treatment period.

There is some evidence that *complete* retrograde amnesia does not occur in all patients who are given ECT. Thus some patients—and especially young individuals—can clearly remember the exact time displayed on a clock which is viewed just before unconsciousness is produced by the anesthetic, approximately 90 seconds prior to application of the electrical stimulus (70). Some incompletely anesthetized patients report the terror associated with being paralyzed while in an altered state of consciousness; others report verifiable fragments of conversations carried on by members of the treatment team.

The emotional stress associated with ECT appears to be derived from multiple sources. There is the fear which is associated with procedures such as general anesthesia in which consciousness is lost. To this is added the impact of the lurid information about ECT which has been widely disseminated through books, movies, and the news media. The confusion and memory dysfunction produced by the treatment causes additional emotional discomfort as a treatment series progresses.

The following appear to be some of the major elements comprising the emotional stress associated with ECT:

1. fear of dying;
2. fear of separation and abandonment; feeling of isolation from family, friends, and staff;

3. concern that the electrical current will damage , or is injuring, the brain and the mind. The confusion and memory deficits, as they are observed in other individuals or experienced by the patient, frequently heighten this concern.
4. fear that while under the influence of the anesthetic normal behavioral controls will be lost and that unacceptable behavior and utterances will occur;
5. concern and anxiety about being helpless and passive;
6. feelings associated with the inadequacy of anesthesia which results in patients being conscious, or semi-conscious, but paralyzed and unable to react.

While these fantasies, fears, and concerns are primarily those of the patient who is being treated, they are also shared to some degree by the family and even by the staff. These feelings can sabotage the quality of care that the patient receives, and when the family is sufficiently distressed, can result in premature termination of ECT.

Any attempts designed to ameliorate the emotional stress associated with ECT need to deal with these fantasies, concerns, and anxieties of the patient, the family, and the staff. The following simple measures can help with this task:

1. providing an adequate description of the procedure as the patient is to experience it (see Appendix I). This should be done prior to the first treatment and repeated, perhaps in an abridged form, before each treatment session.
2. providing an opportunity to ask questions and to correct misconceptions and fantasies. This, too should be done throughout the treatment period.
3. making available the services of a staff member with whom the patient has a trusting relationship and who:

   (a) can accompany her or him throughout the procedure;
   (b) can give assurance that this person, or someone equally acceptable, will remain with him or her during the treatment session—and particularly during the period when consciousness is lost—and will be present when the patient awakens;

4. informing the family about what the treatment involves procedurally and about the confusion and memory changes which are likely to occur and cause concern. Explanations about the temporary nature of these reactions can provide great reassurance to worried relatives.
5. insuring that the level of anesthesia is deep enough so that the patient is totally unconscious *throughout the procedure.* Objec-

tive criteria for making this determination are described in the section which follows.

Suggested Procedure For Administering ECT

The following is a detailed description of one of the several methods available for administering ECT in an acceptable manner. It is included because the Task Force anticipates that this report will serve an educational purpose and, therefore, that a detailed account of one type of treatment procedure will be helpful for illustrative purposes.

It must be emphasized that the procedure described below is but one of several acceptable methods which are used by experienced psychiatrists for administering ECT. Procedures which differ in one or more details from that which is described here are frequently used and are equally acceptable.

1. Preparation of patient

When ECT is to be administered in the early morning, nothing by mouth (including oral solid medications) should be taken after midnight. If the treatment is administered in the afternoon, a non-solid breakfast may be permitted. Careful supervision for the enforcement of these important restrictions is particularly necessary in the confused and unreliable patient—and doubly so if such patient is being treated on an out-patient basis.

The bladder should be emptied just prior to treatment.

2. Drugs and equipment

(a) *Drugs*

It is recommended that the following drugs and solutions be available with dose instructions for immediate administration:

(i) atropine sulfate = 0.4 mg/ml;
(ii) calcium chloride - 10% solution - 10 ml vial (emergency syringe);
(iii) dexamethasone (Decadron) - 4 mg/ml and/or 24 mg/ml;
(iv) dextrose - 5% in water - 250 ml units;
(v) diazepam 5 mg/ml - 2 and 10 ml vials;
(vi) epinephrine - 1:10,000 solution - 10 ml (emergency syringe)
(vii) lidocaine (Xylocaine): Special preparation for use in cardiac dysrhythmias - 2% solution = 5 ml = 100 mg in emergency syringe;

(viii) metaraminol (Aramine) - 1% solution - 10 ml vial;
(ix) methylprednisolone (Solu-Medrol) - 125 and 1000 mg/vial;
(x) sodium bicarbonate - 7.5% solution = 44.6 mEq. - 50 ml (emergency syringe);
(xi) L-norepinephrine (Levophed) - 2 mg/ml - 4 ml ampuls.

(b) *Equipment* to be available for immediate use:

(i) suction-tested for proper function;
(ii) needles;
(iii) infusion sets;
(iv) electrocardiograph;
(v) defibrillator. While the rare case of cardiac arrest occurring with ECT can usually be managed by a blow to the precordium, the adjunctive use of a defibrillator may occasionally be necessary. This apparatus should be reasonably accessible for this contingency.

(c) When ECT is administered in a hospital, the Emergency (Crash) Cart should be readily available. In other facilities, a comparably equipped unit should be available.

3. Patient is placed on a cart or bed which is adequately insulated so that the electrical current cannot be grounded through the patient.

4. Teeth should be examined just before starting treatment to note dental appliances (which should be removed if possible) as well as chipped and/or loose teeth.

5. Syringes should be loaded with anticholinergic agent (recommend methscopolamine), thiopental (Pentothal) or methohexital (Brevital) and succinylcholine (Anectine, Sucostrin, Quelcin). Alternatively, some therapists treating several patients in succession employ dilute solutions of anesthetic and succinylcholine. These solutions are infused at rates sufficient to produce the desired pharmacological effects.

6. If patient presents special risks (e.g. heart disease, emphysema, bronchopulmonary disease, obesity, advanced age), the anesthetic mask is gently placed over the face and moderately deep breathing of 100% oxygen is encouraged for a two to three minute period prior to injecting the anesthetic agent.

7. Scalp-vein needle (21G Butterfly Infusion Set) inserted into accessible vein. We suggest use of a vein on dorsum of hand (infiltration quickly detected) or of a *lateral* vein in the antecubital fossa (artery lies *medially*). The needle should be threaded up the vein and secured to the skin with tape. This needle is left *in situ* until the treatment is completed and the patient is responding; it serves as an IV line for injecting medications used in the treatment and for drugs

and solutions which may be required in the event of adverse responses.

8. After determining the pulse rate, increments of 0.25 mg methscopolamine or 0.5 mg of atropine are injected IV until a discernible increase in heart rate occurs (e.g. rate increases by 10%). The anticholinergic drug can be given subcutaneously 45 minutes prior to the treatment but this procedure makes it difficult to titrate the dose so that adequate cardiac cholinergic block will be produced.

Some patients receiving ECT will be receiving agents (e.g., antipsychotic drugs, tricyclic antidepressants, antiparkinson compounds) which singly, or in combination, will have significant anticholinergic actions as manifest by a tachycardia. It may be judged that additional anticholinergic medication is not required in these patients (46).

A quaternary (charged) anticholinergic agent (e.g. scopolamine methylbromide, methscopolamine, Pamine) is used. These drugs are effective peripheral anticholinergics and as they do not cross the blood-brain barrier should not contribute to the development of a Central Anticholinergic Syndrome (Toxic Psychosis) (71).

9. The anesthetic agent is administered next through the IV line. A test mini-dose (e.g. 50 mg of thiopental) is injected. This dose should be sufficient to evoke anaphylaxis in hypersensitive individuals and produce pain in the event that intra-arterial injection has been made. In the absence of these responses the anesthetic administration is resumed in 15-20 seconds.

The dose of anesthetic is individualized for each patient and fixed doses are not relied upon to produce an adequate depth of anesthesia. The following signs are useful indicators of an adequate level of anesthesia:

(a) the patient does not respond to spoken commands (e.g. "take a deep breath!");
(b) yawning occurs;
(c) airway obstruction occurs as manifested by noisy, snoring respiration;
(d) the *eyelash* (not eyelid) reflex disappears.

10. When the desired anesthetic end-point has been reached, the anesthetic mask is placed on the face and ventilation with 100% oxygen is started.

11. If the "Tourniquet Technique" is being employed (see above), the blood pressure cuff is now inflated to 175-200 mm Hg.

12. The succinylcholine is now injected in doses of 0.5-1.5 mg/kg. If the "Tourniquet Technique" is being employed, and particularly if it

is desired completely to paralyze the muscles not protected by the tourniquet, doses of 100-150 mg. of the drug can be used. In patients with "typical" plasma pseudocholinesterase, these doses will not significantly prolong the duration of paralysis. In patients with the "atypical" enzyme, marked prolongation of the duration of paralysis may occur even with very small doses (e.g., 5 mg or less) of succinylcholine.

13. Following the injection of the succinylcholine the syringe is left attached to the butterfly infusion set and taped to the arm.

14. The establishment of maximal muscle paralysis can be determined by the following methods:

(a) by cessation of muscle fasciculations in the lower extremity;

(b) when the "Tourniquet Technique" is employed in the lower extremity, a method for establishing that maximal muscle paralysis has occurred becomes available. This method is particularly useful in cases where muscle fasciculations cannot be observed. The ventral surface of each foot is stroked in the manner used to elicit the Babinski Sign. A withdrawal motion of the foot on the cuffed (i.e. unparalyzed side) should occur whereas no muscular response should take place on the uncuffed (paralyzed) side.

(c) by ascertaining that the lower jaw has become freely movable due to loss of muscle tone.

15. When it has been determined that maximal paralysis has occurred, preparations for administering the electrical current and for the seizure are undertaken in the following order.

(a) A mouth gag (made of gauze rolled into a ¾″ × 10″ cylinder and wrapped with tape) is inserted between the teeth—and especially between the premolars and molars. Alternatively, a soft rubber bite-block can be inserted. After these devices are in place—and when care has been taken to insure that the tongue and soft parts are not between the teeth—pressure is exerted at the tip of the mandible to approximate the teeth and to hold them in this position until the convulsion has ceased.

(b) Electrodes with cotton pads moistened with a solution containing 25% sodium bicarbonate plus a few drops of dishwashing detergent in water are held firmly in one of the following positions:

(i) For bilateral or bitemporal ECT each electrode is placed 1½″ above the middle of a line extending from the external auditory meatus and the lateral angle of the orbit (Fig. 2).

(ii) For unilateral ECT, one or both electrodes are placed on

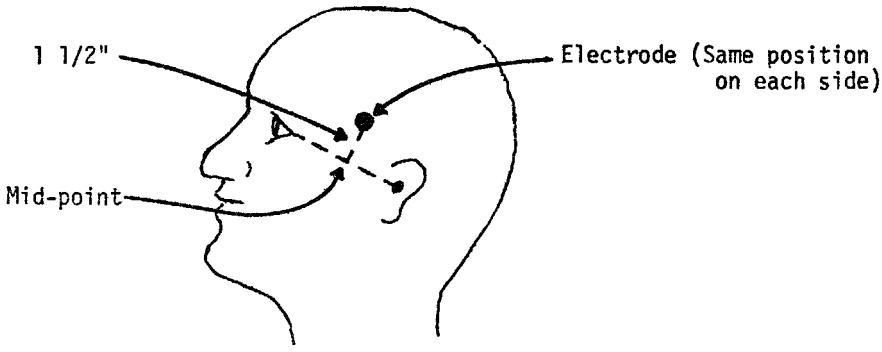

Figure 2—Bilateral or Bitemporal Electrode Placement

the same side as the dominant hand. In the classical Lancaster electrode placement, the lower electrode is positioned as described in (i), above (Fig. 3A). The upper electrode is placed 3½'' higher than the lower one and at an angle of 70° to the line. In the Muller electrode placement, one electrode is placed as described in (i), above, while the other is positioned on the forehead, anteriorly (Fig. 3B).
(There are other positions for electrode placement in unilateral ECT, but the above are commonly used.)

(c) The individual holding the electrodes in place should be protected against electrical shock by suitable insulation and/or by wearing rubber or plastic gloves.

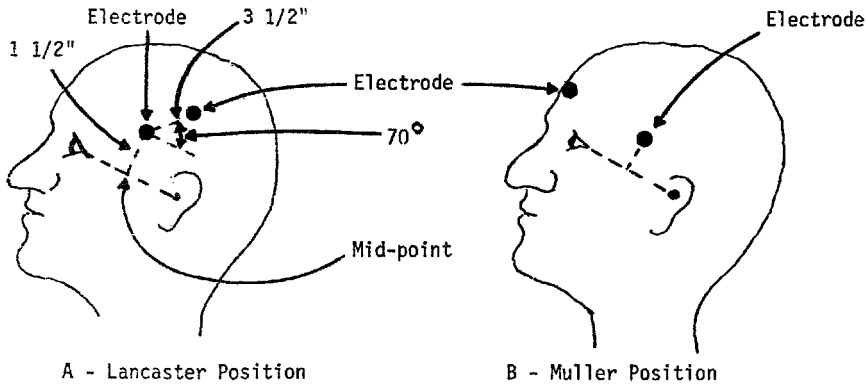

Figure 3. Unilateral Placements

(d) An attendant places one hand on each thigh near the knee. This support will prevent the patient from falling off the treatment cart in the event that a vigorous and essentially unmodified convulsion occurs unexpectedly.

16. The electrical stimulus is then administered in accordance with the directions for the machine being employed. The Task Force is not advocating the use of any particular ECT stimulus units pending the availability of the FDA's evaluation of these devices.

17. The therapeutic adequacy of the seizure is determined by one or both of the following:

(a) occurrence of a characteristic major seizure pattern on the electrocorticograph tracing or oscilloscope image if this type of monitoring is being used;

(b) occurrence of a tonic muscular contraction in an extremity. This tonic contraction persists after the electrical stimulus has ceased and it progresses into a clonic pattern.

18. In the event that a therapeutically adequate seizure has not been evoked, the electrical parameters should be increased appropriately and the stimulus reapplied. Because of the refractoriness which follows the first stimulus, an interval of 60-90 seconds should elapse before the second is applied.

19. When a therapeutically adequate seizure has occurred, the following are carried out, sequentially:

(a) the mouth gag or bite-block is removed;

(b) the teeth are quickly checked and the hypopharynx is examined with the use of a curved-blade laryngoscope. Secretions should be removed at this time—while the patient is still paralyzed. When secretions are removed by necessity after the succinylcholine's action is terminated, vomiting and laryngospasm occasionally occur.

(c) the anesthetic face mask is reapplied and artificial and assisted ventilation are instituted and continued until spontaneous respiratory muscle movements are strong enough to maintain an adequate tidal volume;

(d) if the "Tourniquet Technique" is employed, the blood pressure cuff is deflated and removed.

20. While awaiting the return of muscle activity, consciousness, and protective reflexes, the patient's abdomen should be uncovered so that impending vomiting and regurgitation can be detected promptly. Sufficient person-power should always be close at hand in order quickly to turn the patient's body over to one side in order to minimize the possibility of tracheal aspiration.

21. When spontaneous respiratory muscle activity commences, the anesthetic face mask should still be held in place until there has been enough muscle recovery from the succinylcholine to permit the establishment of an adequate tidal volume. This serves two purposes: the movements of the anesthetic bag are a measure of the tidal volume, and inadequate ventilatory movements can be supplemented by squeezing the bag.
22. With the establishment of a normal tidal volume the anesthetic face mask is removed and the patient can be moved from the cart to a bed. The positioning of the patient depends on whether or not protective pharyngeal reflexes have returned as indicated by the presence of swallowing and the capacity to phonate. When reflexes are not vigorously present it is advisable to place the patient in the semi-prone position. In moving the unconscious or semi-conscious patient, care should be taken not to twist the neck and to avoid hyperextending arms and damaging the brachial plexus. Consultation with an anesthesiologist is helpful in learning these standard positioning techniques.
23. Careful observation and monitoring of vital signs is essential during the immediate post-treatment period. Particular attention needs to be paid to the adequacy of ventilation, the absence of cyanosis, and the pulse rate, volume and rhythmicity. The awakening patient should be systematically re-oriented and reassured—preferably by someone who is familiar.
24. Most individuals receiving ECT are confused and unsteady, particularly in the hour following the treatment. Patients need to be carefully supervised, particularly during this interval, to prevent injury. Out-patients should return home in the company of a reliable individual and with clear admonitions against operating a motor vehicle or hazardous machinery.

Alternative Procedures

Although the Task Force recognizes that procedures are frequently used which differ in one or more details from that which is described above and that they may be equally acceptable and have a comparable degree of safety, we regard as essential to safe and acceptable ECT the following procedural details:

(1) the administration of an anesthetic agent;
(2) the administration of a muscle relaxant; and
(3) oxygen supplementation.

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CHAPTER VI

PHYSIOLOGICAL AND BIOCHEMICAL CONCOMITANTS OF ECT

Seizures have been used to treat the severely mentally ill for more than forty years, and yet no reasonable explanation of how they elicit improvement in a patient's condition has been formulated. Some seek an explanation in the psychological factors of fear, expiation of guilt, or memory loss; some seek a basis in electrophysiologic, pharmacologic, or biochemical changes; and others seek understanding in combinations of these factors. The mode of action is often described as "unknown" and the treatment results as inexplicable; even knowledgeable therapists and scientists proclaim their ignorance.

But we know something of the ECT process. ECT relieves severe depression and in comparative studies, the results are better for ECT than for other treatments of depression (1). The treatment is also useful in certain schizophrenic disorders and in acute mania (1-3). Psychobiologic aspects have been extensively studied and these data define the parameters of the treatment, certain changes in brain function that seem necessary to the behavioral change, and some of the biochemical and electrophysiologic events that characterize a successful therapy (2-4).

What Do we Know About the ECT Process?

1. The behavioral changes following ECT result from repeated cerebral seizures. The motor events (convulsion), apnea, hypoxia, and other peripheral events affect behavior secondarily.

In comparisons of convulsive and subconvulsive treatments, some patients referred for ECT received currents above the convulsive threshold and convulsions were elicited. For others, the currents were below the threshold and failed to do so. Patients were assigned randomly to either regimen and were independently evaluated by clinicians who did not know the type of treatment administered. In two studies the results were the same: patients receiving convulsive currents were rated as much improved or recovered more often and sooner than patients who received subconvulsive currents (5, 6). In one study, the patients who failed to improve after 12 treatments were continued in the same course but now received convulsive currents.

Almost all who had not improved did so during this second course, indicating that the two treatments differed in efficacy (5).

With the addition of curare and later succinylcholine (Anectine) to the treatment, the effects of the convulsion—the motor effects on muscles and limbs—were separated from the cerebral seizure. Clinically ECT modified by succinylcholine and barbiturate is approximately equivalent to unmodified ECT in efficacy, although some find unmodified seizures to be slightly better. This observation was unexplained until the EEG was recorded during the seizures. Not every administration of electric current or flurothyl is accompanied by a cerebral seizure, even when motor responses resembling clonic or tonic phases are observed. It is particularly difficult to be sure of a cerebral seizure when succinylcholine masks the motor convulsion (7). These observations led Blachly to suggest ECT or flurothyl treatments should be under EEG control, i.e., with monitoring of the seizure for duration and pattern so that patients with incomplete seizures can be immediately treated again (8).

The therapeutic efficacy of ECT is not reduced when given with forced ventilation with oxygen and when the normal levels of blood gases are maintained (8). This suggests that the clinical effects of ECT are not due to oxygen lack.

Attempts to relate the therapeutic effects of seizure treatments to a biochemical or physiological measure have usually been unsuccessful (9, 10). The best correlations for behavioral improvement are with persistent EEG slow wave activity i.e. with the EEG correlates of brain seizure activity (11, 12). The faster the development of EEG slowing and the greater its amount (11), and the greater the decrease in EEG fast activity (13), the better the clinical result (11, 12, 13). Some authors suggest that the relationship is not a causal one but rather an expression of secondary effects similar to memory loss (14).

2. Improvement in behavior is independent of the mode of induction of the seizure, with seizures produced by electricity, by pentelenetetrazol (Metrazol), and by the inhalant flurothyl (Indoklon) yielding equivalent clinical results.

The number of inductions necessary for a therapeutic effect are about equal for patients treated by flurothyl, pentelenetetrazol, and ECT (4, 15, 16, 17). The principal differences among the inductions are in the number of seizures needed to elicit changes in behavior and in the degree to which the secondary effects of memory impairment, disorientation, and confusion are developed. The different inductions usually produce seizures of equivalent length although some studies report differences in duration. Behavioral improvement is related

more to the occurrence of seizures and their duration than to the mode of induction (18).

If these observations are true, we would expect that the treatment method providing the least discomfort and risk to the patient should persist in clinical practice. Metrazol often elicited fear reactions and incomplete seizures, and it was replaced by electrical inductions as these were found to be safer and easier to administer. The difficulty of a safe induction and a higher incidence of missed seizures with flurothyl eventually led to its discontinuation.

In ECT, the type of current is less important than its intensity (i.e. the relationship to seizure threshold). Studies of square wave, unidirectional, and pulsed-wave currents find little relation between current characteristics and outcome, provided the current used is sufficient to induce a cerebral seizure (18).

The location of the ECT electrodes affects memory and orientation more than clinical outcome. Comparison of the effects of bitemporal, unilateral dominant, and unilateral non-dominant electrode placements show that the degree and type of clinical change are approximately equivalent, but tests of memory and recall show significant differences. Patients treated with electrodes over the non-dominant hemisphere complain less of memory difficulties than patients treated with bitemporal electrodes (19, 20). The differences are clearest in tests of verbal memory, particularly using auditory tasks and less so with visual and non-verbal memory tasks (21, 22). These observations suggest that the electrical currents directly affect tissues that subserve the psychological processes of memory and orientation but not those that subserve the therapeutic process (23). From these data, also, it is improbable that changes in memory processes explain the therapeutic process (4, 23).

3. The number of seizures required for a clinical response varies with the psychopathology and age of the subjects, and less with factors in the induction of seizures.

The target populations for convulsive therapy are the depressive psychoses, particularly those with endogenous features, in whom therapeutic results can be achieved with six to ten inductions. In other populations, as in schizophrenic patients, more seizures need be applied with shorter time intervals to elicit a behavioral change. This differential sensitivity to seizures is reflected in clinical criteria and in physiological (as EEG) and psychological tests (4, 15, 24).

Personality type affects the type of behavioral response to ECT. The extent the subject uses explicit verbal denial as a defense

mechanism (25), the degree of authoritarianism on the California F Scale (26), and the organization of the Rorschach protocol (27) have each been related to the type of behavioral outcome and to the amount of treatment needed to achieve it.

4. Vegetative changes, as in sleep, appetite, weight, menses, libido, and mood, accompany the behavioral changes induced by repeated seizures.

The depressive psychoses are characterized by impairment of the physiological functions associated with the activity of the hypothalamus and these impairments improve early in the course of ECT (2, 4, 23). The efficacy of ECT may be predicted by the degree and persistence of the changes in appetite and sleep and their impairment seems to accompany or precede relapse. The relation between these physiologic functions and improvement seems more direct than the changes seen in memory tests or in persistent EEG changes, suggesting that hypothalamic processes may be involved in the ECT treatment process.

5. The effects of repeated seizures are modified by biochemical and pharmacologic interventions.

The behavioral effects of repeated seizures are enhanced by barbiturates (12, 25, 32). If patients are given an injection of thiopental after the first or second ECT treatment, those patients who exhibit an increase in EEG slow wave activity or who show an improvement in their depressive symptoms show the best clinical results; while patients who do not show this augmented physiologic effect show a poor therapeutic response (12). The same relations are found for changes in language (defined as explicit verbal denial) following the administration of intravenous amobarbital (25). Sedation threshold studies using the EEG response to amobarbital also are useful physiologic predictors of the therapeutic response to ECT (32).

It has been reported that the behavioral effects of ECT are reduced by anticholinergic drugs, such as Ditran, atropine, or benactyzine, and by some hallucinogens as LSD (33). High voltage EEG slow wave activity and denial language patterns after ECT are temporarily reversed by these drugs, and during these reversals the patients exhibit their pre-treatment behavioral patterns. These observations suggest that cerebral cholinergic processes are involved in the ECT treatment process (34).

6. The physiologic and psychologic effects of repeated seizures

are reversible in time, usually measured in weeks; while the clinical and psychopathologic changes may be more persistent, although these also are reversible.

Physiologic effects of seizures as increased slow wave activity of the EEG, redundancy in speech, changes in language patterns, and performance in memory and psychomotor tasks improve gradually after the last seizure and appear substantially normal after six months (4, 24, 26, 35). For some psychologic tests, performance improves over the initial measurements—an improvement ascribed to an impairment in test performance caused by the depressive illness itself (4, 24, 35).

ECT Theories

An explanation of any therapeutic process is limited by our knowledge of the etiology of the disorder which the treatment is found to resolve. In the four decades of its use, the convulsive therapy process has been explained according to the prevailing theories of the etiology of mental illness. Thus, the explanations in the 1930s reflected the strong emphasis on brain pathology, while in the 1940s the explanations focused on psychological and intrapersonal mechanisms. Since the 1960s explanations have been based on neurobehavioral, biochemical, and neuroendocrine factors. Considering the diversity of present theories of mental illness, we are not surprised that no single explanation exists for the efficacy of ECT in depressive, manic, and schizophrenic disorders; in endogenous and reactive subtypes; and among the elderly. If we seek to explain the process, we must develop different explanations for its effects in different populations.

The status of ECT today is similar to that of psychotropic drugs in 1957-1962—a time when the distinctions among the antipsychotic drugs were related poorly to chemical structure but more to clinical diagnosis and physiological responses. The separation of thymoleptics, stimulants, MAOI, and antipsychotic drugs in the treatment of depression and psychoses did not allow a unitary theory of their mode of action and a pluralistic view has developed. The ECT process, while superficially similar in different disorders, must also be viewed as different processes for at least the endogenous (bipolar and unipolar) depressive psychoses, and the acute schizophrenias—syndromes in which the efficacy of ECT has been demonstrated. It is difficult to say whether the response of manic patients can be subsumed by one of these explanations or whether a third mechanism will need to be elaborated.

There is clearly a need for research into the relationship of ECT to biogenic amines (49-53), neuroendocrine systems (54-57), electro-

lyte metabolism (59-61), and events at the neurophysiological level (63-67).

A special problem in assessing neurochemical events is to separate those with specific therapeutic effects from the non-specific changes which accompany any physiologic stress on the body's economy; stresses which may result from the seizure, convulsion, hypoxemia, or hypercapnia, or the psychosomatic consequences of anticipatory fear, panic, and anxiety which can accompany any treatment in medicine.

It appears virtually certain, however, that these non-specific factors—hypoxia, hypercapnia, hypoglycemia, anticipatory fear, memory disturbance, and the physical stress of the procedure—are not responsible for ECT's therapeutic effects in primary affective disorders and schizophrenia. The only demonstrated absolutely necessary change is a generalized electrical seizure of the central nervous system.

What biological changes produced by the seizure are responsible for ECT's therapeutic effects? The answer is unknown but several areas of research appear promising (2).

1. The seizure is accompanied by an alteration in the permeability of the blood brain barrier. It is possible that important therapeutic substances gain access to the brain in this way, but no evidence for this has been developed as yet.

2. The seizure provokes an acute neuroendocrine discharge, reflected particularly in pulses of ACTH and prolactin secretion. It remains, however, speculation as to whether hypothalamic tropic hormones released by ECT might exert therapeutic effects.

3. ECT may affect the turnover of brain neurotransmitters, the activity of their regulatory enzymes, and/or the sensitivity of their specific neuroreceptors. The animal data, however, are clouded by the effects of non-specific stress on these same neurochemical variables and the human data are quite sparse. Nevertheless, such a relationship would be of particular interest because it would tie ECT's neurochemical effects to the hypothesized mode of action of antidepressant drugs.

4. There are also some data suggesting that ECT affects electrolyte metabolism, which may also be altered in depressive illness.

5. There is a body of electrophysiologic evidence that indicates that centrencephalic structures are implicated in the mechanism of the ECT process. (12, 14, 19, 20, 23, 33, 62-67)

6. Since a different number of treatments are needed for

schizophrenia and since efficacy is less, the mode of action of ECT may be different in schizophrenia than in depression.

How ECT benefits depression is unclear, but many theories of the mode of action of ECT have been disproven because of evidence developed in the last twenty years. ECT does not produce this therapeutic benefit in depressed patients because of non-specific stress or anticipatory fear, since sham ECT is ineffective. Also unmodified ECT, which is much more stressful, is no more effective than modified ECT. In addition, ECT does not produce its effect through non-specific events such as anoxia, hypercapnia, etc., since well-oxygenated ECT is equally effective to unmodified ECT. Furthermore, ECT does not produce its effects through memory disturbance since unilateral treatment with minimal memory changes is often equally effective to bilateral treatment with a greater degree of memory change. The physical concomitants of the convulsion are not essential since unmodified treatment is equally effective to modified treatment.

The convulsion, as a central electrical event, is critical to ECT's therapeutic action. Since ECT given with excess electrical current is no more effective than ECT given with minimal current to produce a convulsion and since subconvulsive electrical current is not beneficial to depressed patients, the dose of electrical current is not essential to ECT's therapeutic effect. There is a growing literature exploring the possibility that the cause of depression may lie in alterations of central neurotransmitters. It is of interest that ECT has effects on neurotransmitters in man and its mode of action may parallel that of active antidepressant (thymoleptic) drugs.

On the other hand, experience in psychoneuropharmacology provides grounds for optimism: if the specific neurochemical effects of ECT can be unraveled, these data will provide important clues to the psychobiologic etiology of affective and schizophrenic illnesses.

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CHAPTER VII

SOCIAL, ETHICAL, AND LEGAL ASPECTS OF ECT

Introduction

In recent years the status and the treatment of the psychiatrically ill have been critically judged in the light of strongly championed legal principles and social reform. The issues of civil rights, consumer rights, and civil liberties have been cogently introduced and have by now permeated the field producing many unanswered questions, admissions of guilt, declarations of innocence, and legal requirements some of which at best complicate good clinical practice and at worst prevent it from taking place.

History reminds us that in keeping with the attitudes of the nineteenth century, the mentally ill were treated by the courts as patients rather than as criminals. Negligible legal review and poor attention to the procedural safeguards normally available in the criminal courts led to increasingly larger numbers of people being confined in state hospitals and related institutions where they were held involuntarily and sometimes indeterminately. They were deprived of their personal freedom and in recent decades not infrequently treated unwillingly. When the highly unsatisfactory levels of care in many of the public institutions became public knowledge, public guilt at having sanctioned the incarceration of thousands without due process became a target for the demands of those who transformed the subject of the mentally ill into an issue of civil rights and civil liberties. Criticism of the involuntary system rapidly drew attention to criteria considered by many to be essential to a free society, namely the right to treatment when involuntarily detained and the right to refuse treatment. Ultimately, with regard to ECT and other therapies, the nature of the treatment itself has come under scrutiny.

Stone, in *Mental Health and Law: A System in Transition* (1), provides a useful analysis of relevant judicial decisions.

> These decisions are almost unanimous in concluding that loss of freedom in civil commitment is at least as grievous as criminal confinement . . . The courts thus increasingly reject

> the long unexamined proposition that loss of freedom for purposes of treatment, rehabilitation, or custodial care is qualitatively different in law from loss of freedom for purposes of punishment . . . Simply put, the distinction is between those who attack involuntary civil confinement as a dangerous and potentially repressive force in a free society and those who endorse involuntary confinement and proper treatment of the mentally ill as a moral responsibility of the state.

Traditionally physicians see themselves as having a duty and a moral responsibility to their patients, and many psychiatrists who have continued through the years to treat the mentally ill in the absence of fully informed consent have probably done so in good faith. Their actions have been reinforced by the expressions of appreciation and gratitude from the close relatives and then from the patients themselves who have benefited from the treatment. It should come as no surprise that members of the profession were largely unprepared to meet the criticisms of lawyers and civil libertarians who now sometimes cast them in the role of incarcerators and enemies of individual freedom.

Stone (2), in a critical review of recent mental health litigation and so-called reforms, points to the error of comparing psychiatrists with agents of the criminal justice system and patients with criminal defendants. Legal demands for evidence of danger to society, for the right of the patient to remain silent during questioning lest he/she incriminate himself/herself, and for the alleged patient to be provided with a lawyer to invoke constitutional safeguards and to advocate his/her freedom, all force the psychiatrist into the role of the prosecutor—a position for which he/she has neither the taste nor the training.

The recent emphasis on litigation and legislative action to ensure the civil rights of patients has loaded the balance against good clinical management of challenging problems. The traditional principles of good medical management are as foreign to lawyers with no clinical background as are the criminal court procedures to psychiatrists. If we correctly suppose that no professional discipline has a monopoly on the ultimate truth, the system that eventually emerges must realistically balance rights against clinical needs. In the event that it fails, the patient will enjoy good legal care but poor medical attention.

This Task Force is committed to a balanced view, believing that these antagonisms can be minimized by compromise; we endorse the principle of involuntary confinement when indicated, and the appro-

priate treatment of the mentally ill including ECT as a moral responsibility of the State. We recognize the necessity for introducing explicit constraints on the procedures used but can support such controls only if they are practical and if they do not interfere with good medical care.

Social Aspects

Considering the data that are available to us regarding ECT, the members of this Task Force are especially sensitive to the need for a rational perspective and the prescription and furtherance of such a position. This is made necessary by the polemics surrounding the use of ECT and by the zeal of several of its protagonists as well as its detractors. Before examining the main ethical and legal principles that must command our attention, we wish to note some of the beliefs and attitudes in society that influence the ways in which mental illness, ECT, and the treatment of the mentally ill generally are perceived. These factors are not infrequently drowned in the moral indignation of some of the critics.

(1) *Reactions to mental illness*

Society must share the responsibility for the deplorable state in many of the nation's mental hospitals used for decades as warehouses for those who, in numerous instances, were avoided by their families. Observing mental illness close at hand can be an anguished and frustrating reality for relatives.

While considerable emphasis has been placed by society on schemes to eliminate mental illness, very little effort has been made in adapting realistically to its continued presence in our midst. Euphemistic titles such as *mental health programs* are used as blandishments in an effort to obscure the refractoriness of the *mental diseases* they are designed to treat (3); and some critics work hard at trying to persuade us that mental illness is but a myth (4).

Despite initial enthusiastic support, when the cost of change has to be faced we find considerable reluctance in providing for fiscal allocations, and attempts to settle discharged patients in comfortable suburban environments meet with strong opposition from local residents often very liberal in their views who espouse the idea of halfway houses but preferably in neighborhoods other than their own.

It is evident that society copes poorly with mental illness.

(2) *The influence of films and fiction*

The fiction of novels, plays, television programs, and films (5, 6) is readily adopted as a true reflection of fact. The undue emphasis on hyperbole and the use of dramatic license in these works designed for theatrical impact on the audience, leads to an exaggerated and often distorted public image of hospitals, routine psychiatric treatment, and ECT.

(3) *Speculation and theory*

According to Zilboorg (7) history reminds us that "for numberless centuries these branches of thought (jurisprudence, theology, and philosophy) and not medicine, dealt with mental disease. They claimed it theirs by right of eminent domain and they refused to admit the medical man or permitted him only a limited right of way."

> Further on, Zilboorg states:
> After Hippocrates . . . philosophies diverted medicine from the observational and experimental path which Hippocrates would have wished it to follow. This is a point not to be passed over lightly, for it denotes an almost permanent characteristic of the history of psychiatry—a tendency in medicine to make an earnest beginning in psychiatry and then to shy away as it were, and to recede in favor of the speculative philosopher— . . . the human mind soon became again less of a medical problem than a philosophical one (8).

We are once again in a phase where some of the jurists, philosophers, and critics are prescribing for the mentally ill. They are for the most part singularly out of touch with the practical realities of clinical experience. Neither the complex nature of mental illness itself nor the attitudes of society permit simple and rapid remedies. In attempting to dissolve mental illness in behavioral and socio-cultural theories (4, 9, 10), the modern critic has paid little attention to the pervasive and stubborn nature of severe intrapersonal psychopathology and the suggestive evidence that at least some aspects of mental disorder spring from biological factors as yet only poorly understood. Clinical observation persuades us that these aspects are not likely to disappear without active intervention of a medical nature.

(4) *Attitudes regarding the application of electric current to the brain*

The image of an electric current passing through an organ as sensitive as the brain evokes anxiety in most people. Many are

captives of their own imaginative belief that permanent damage to the brain *must* result if an electric current is passed through it despite the absence thus far, of incontrovertible scientific evidence to support it. The following quote by a neurology resident captures the sentiment (11).

> But for me, studying the central nervous system was its own reward. The brain was a magic mushroom, two plus pounds of electrical jelly, the circuitry of consciousness. . . I was teased by the notion that the brain is more quintessentially 'self' than any other part of the human anatomy . . . The illusion of the self as thing, a thing within the brain, is perennially seductive . . . And I came to conceive of the neurologist as a protector of the brain, not only from bacteria, viruses and trauma, but from overly ambitious fellow creatures—especially those who would harm the brain to help mankind, that is the practitioners and proponents of electroconvulsive therapy.

(5) *A possible explanation for some of the complaints*

There might be many reasons, as yet not fully understood, to account for complaints by former patients about ECT. However, as psychiatrists sensitive to psychodynamic issues, we must draw attention to the frequency with which involuntary hospitalization has been associated with the administration of ECT among those patients who have been most vocal in their criticisms of the treatment (12). Angry complaints appear to be rare among the many thousands of patients who voluntarily seek treatment and agree to ECT. The question must be raised regarding the likelihood of global anger and resentment against involuntary commitment and the family members responsible for initiating it being focused on the ECT which is then viewed as the essential evil in the whole scheme of things. It is also important to acknowledge the possible role of the subsequent attention and the likelihood of its reinforcing the anger and sustaining the complaints of memory impairment. The frustration and anger associated with the often lengthy nature of the illness can readily be shifted on to ECT despite the possibility that in some instances unremitting depression and even subclinical dementia could account for the memory problems. While these kinds of assertions still await good scientific investigation, they mesh readily with our current understanding of human behavior.

We are also cognizant of the fact that the successful use of ECT for persons who subsequently achieve distinction in various fields is rarely if ever reported, because of the public attitude to psychiatric

illness in general and to ECT in particular. The sacrifice of a vice-presidential nominee in 1972 (13) is a case in point.

Relevant Ethical and Legal Issues

Ethical and legal issues have developed primarily in recent years from the enunciation of major principles in the courts. One is the Right to Treatment, justified legally under several different constitutional and statutory theories and associated with the Right to Refuse Treatment. Informed Consent is a somewhat broader principle and the underpinning of the Right to Refuse Treatment. We will consider all of these developments in relation to ECT, drawing attention to their major impact on its use.

(1) *The Right to Treatment*

Although the courts had recognized that the promise of treatment to juveniles within a system established to divert young people from the criminal system into rehabilitative and reformative programs was illusory (14) and that it was illegal to lock a man up in a building for the violently insane in which no treatment was provided (15), the major legal decision usually cited as the first suggestion by a court that the right to treatment has constitutional dimensions is *Rouse v. Cameron* (16) in 1966. The Rouse decision, in addition to suggesting constitutional implications, extended the significance of the right to treatment by stating that continued failure to provide treatment cannot be justified by an insufficiency of resources. The court neither established standards for acceptable treatment nor did it demand improvement or the best possible treatment. It asked only for a bona fide effort to provide an individualized treatment program with periodic evaluation.

The case of *Wyatt v. Stickney* (17) in 1973 pushed further and directed attention to the plight of the large numbers of mentally ill patients involuntarily confined in less than adequate state hospitals. Judge Johnson clearly stated:

> "To deprive any citizen of his or her liberty upon the altruistic theory that the confinement is for humane and therapeutic reasons and then fail to provide adequate treatment violates the very fundamentals of due process."

Judge Johnson went much further than his predecessors in spelling out criteria of adequate treatment, indicating a willingness in his court to set the standards for institutional practice in the state

hospitals of Alabama. His decree was formulated from a study of testimony by experts and representatives of national mental health organizations and included specifics regarding staffing, floor space, record keeping, and review. The successive steps in the development of the principle of the right to treatment are well covered by Stone (18).

In his decree Judge Johnson dealt in some detail with the use of ECT describing it as an extraordinary and potentially hazardous mode of treatment (19). He ruled that before it can be administered to patients in the state hospitals of Alabama, its use must be recommended by a qualified mental health professional trained and experienced in the use of ECT; that the recommendation must be accompanied by supportive documents recording the clinical justifications and rejected alternatives; that the recommendation must be agreed to by another qualified mental health professional trained and experienced in the use of ECT; and that the use of the treatment must be approved by the superintendent or medical director of the hospital. In addition to the patient's clearly fully informed consent if he/she is deemed competent to give it, additional approval of an Extraordinary Treatment Committee is to be obtained before the ECT may be administered. This Extraordinary Treatment Committee is to be nominated by the Human Rights Committee of the hospital, appointed by the court, and is to consist of five members to include at least one psychiatrist, one neurologist, and one attorney. None of these is to be an officer, employee, or agent of the Department of Mental Health or involved otherwise in the proposed treatment. The patient is to be represented throughout all proceedings including the signing of consent and the deliberations of the Extraordinary Treatment Committee, by legal counsel.

In essence, before the treatment can be given to a consenting patient in the state hospitals of Alabama, its indications are to be adjudged by at least four psychiatrists and one neurologist, with at least two attorneys monitoring the proceedings. This in the presence of competence to provide informed consent! In the event that the patient is deemed incompetent to supply informed consent, the procedure prescribed becomes even more complicated to involve the patient's relatives and review in a court of competent jurisdiction.

It is important to bear in mind that Judge Johnson's criteria for the regulation of ECT arose in the context of the right to adequate treatment. His assumption was that unless specified minimum standards were available in Alabama state hospitals, adequate and effective treatment was not possible. While we object to the description of ECT as an extraordinary and potentially hazardous mode of treatment,

consider the layers of professionals required by the judge to approve the use of ECT extraordinarily cumbersome and unnecessary under routine circumstances, and question the advisability of the courts regulating medical practice, we recognize the judge's concern with the provision of adequate treatment in the state hospitals in Alabama. He had been struck by the unacceptable standards of treatment that existed, the inadequate medical care, and the fact that only unmodified ECT was available. There was scant likelihood that treatment decisions would be made by adequately trained psychiatrists. His rigid regulations must be evaluated within the context of that sorry situation. He was apparently not regulating ECT in private facilities in Alabama nor was he regulating it outside the woefully inadequate state hospitals. It seems clear that he was placing as many obstacles as he could in the path of the use of ECT in the state facilities. There is little doubt that he succeeded because as of December 1976 no ECT had been given in the state hospitals of Alabama since the decree (20).

It cannot be assumed that Judge Johnson would have imposed the same restraints in a different and more therapeutic situation. Unfortunately, some legal advocates ignore the differences in context in an attempt to apply the regulations to ECT *per se* rather than to the circumstances prevailing in the Alabama state hospitals at the time.

It is clear that consistent with the right to adequate treatment, the court, in Alabama at least, has sought to regulate the circumstances under which ECT may be given in its state hospitals. Even acknowledging that those regulations are unduly restrictive and that the description of ECT as extraordinary and potentially hazardous is unfortunate, this Task Force must recognize the implications of the judgment. Viewed from the standpoint of the law, ethical considerations, and good medical care, the incarceration of patients in facilities incapable of providing adequate treatment cannot be condoned. Even though it is likely that some state and local jurisdictions will attempt to evade the cost of providing adequate care by prematurely sending patients back into the community or by refusing to admit others, we must unequivocally recommend that all public and private hospitals that care for the mentally ill should either possess adequate facilities for the administration of ECT (see Recommendations), or be affiliated with or have ready access to a hospital or clinical center that does.

(2) *The Right to Informed Consent,* and *The Right to Refuse Treatment*

These two principles are intricately interwoven. Concern about a mental patient's right to consent to treatment leads directly to the

recognition of his right to refuse such treatment (21). As the legal doctrine of informed consent took hold and began to evolve over the past two decades, it reinforced and nurtured the right to refuse.

The law of consent to medical care has undergone important changes, for the most part effected by judicial decisions in particular cases rather than through legislative promulgation of general rules (22). In recent centuries, physicians were expected to adhere to the customary professional standards of practice in informing a patient about what was to be done to him/her, that he/she may take courage to enable him/her to undergo the operation. Judgments appearing in the early part of this century were based on the dictum that "every human being of adult years and sound mind has a right to determine what shall be done with his body" (23). A basis for imposing liability for unauthorized treatment is thus found in the law of battery.

During the late 1950s, cases holding physicians liable for failing to disclose information about treatment to the patients before administering it began to appear with some frequency. In a brief period of a few days in 1960, the Supreme Courts of Kansas and Missouri issued opinions that were to change the law of consent to medical treatment drastically. In a case in which a woman suffered injuries from radiation therapy after a mastectomy (24) and in a case where ECT treatments caused the fracture of several vertebrae (25), the courts held that the consent of each patient was invalid because the physicians had not informed the patients of the possible serious hazards of the treatments. In each case the court held that the patient's consent was insufficient to protect the doctors from liability for untoward results of treatment even though the physicians may not have been negligent in performing the procedures.

The doctrine of informed consent both reflects and enforces the concern of Anglo-American law with the individual's right to be free from the conduct of others that affronts bodily integrity, privacy, and individual autonomy. Furthermore, the doctrine seeks to promote intelligent decisions by medical patients by assuring that relevant information is available to them (26). The traditional physician-patient relationship in which the physician is the predominant agent and the patient entirely passive or trusting has been pushed in the direction of the model of mutual participation in decision making (27).

For a valid decision, the patient must be competent to understand the information provided, the precondition of voluntariness, and the consequence of his/her consent or refusal (22). Patients must be able to act voluntarily without coercion. They must be provided with: adequate information concerning the nature of the therapy, with its risks, discomforts and side-effects; the anticipated benefits; the avail-

able alternative treatments with their attendant risks, discomforts, and side effects; and the likely consequences of a failure to be treated at all.

Despite the apparently straightforward nature of the formula outlined above, in practice it encompasses many unanswered questions and ambiguities.

(a) How voluntary can the decision about the recommendation for ECT be when the alternatives are likely to be longer hospitalization, prohibitive costs, or even transfer to a less attractive facility that can better handle the problems of a serious preoccupation with suicide?
(b) What amount of information is adequate for an informed decision; how detailed should it be; and how should the risks and side effects be described without causing the patient unnecessary distress or frightening him/her away from consenting? Should all risks be described, even highly improbable ones?
(c) Even though a patient may be given adequate information, how certain can one be that he/she understands? There is no single acceptable test for competency to provide consent. In practice, competency to consent seems to be based on the overall impression of the patient's mental state, his/her seeming ability to respond to the information provided as a reasonable person would be expected to, and his/her understanding of the risks, benefits, and alternatives. The legal presumption is one of competency until found otherwise. "In practice, judgments of competency go beyond semantics or straightforward applications of legal rules; such judgments reflect social considerations and societal biases as much as they reflect matters of law and medicine" (28). Societal biases are readily apparent with regard to ECT.
(d) Slovenko (29) has drawn attention to the influence of the physician's status and prestige when his/her recommendation for treatment is to be considered. This is a well-recognized principle, amply documented by many, including Anna Freud (30). Slovenko's commentary deals with the issue of psychosurgery but his allusion to the nature of the relationship between physician and patient is relevant to any treatment decision. Not only *what* is told the patient is of importance but *who* tells it, and how the patient relates to him/her will influence the ultimate decision to consent or refuse.
(e) A study (31) of the informed consent given by cardiac surgery patients preoperatively revealed that the large majority of them, postoperatively, had faulty memories of multiple significant items

that had been discussed with them previously. How much more might this apply to mental patients suffering from mood or thought disorders who have to make reasoned decisions about ECT?

It is apparent that the current status of the law of consent is incomplete, ambiguous, and evolving. Meisel, Roth and Lidz (22) aptly describe the situation in these words:

> What consitutes a valid consent today may not remain so tomorrow; what constitutes a valid consent in one jurisdiction may not be an accurate representation of the law in a neighboring jurisdiction; and what constitutes a valid consent in one branch of medicine may be a less than wholly accurate guide to a valid consent in another branch of medicine. Although the applicability of informed consent to psychiatric practice is still in its incipient stages, it is clear that the doctrine does apply. . . . The trends are unmistakeably clear: the emphasis is on more information, and the consequence may well be an increase in patient participation in decision making.

Assuming that we can avoid the extreme positions that the physician always knows best or that every patient no matter how disturbed or disoriented has an absolute right to refuse therapy, we must develop a system which reflects the principle of competent informed consent. The important goal of such a system should be the achievement of overall good clinical management. We believe that the considered opinion of this Task Force on the efficacy of ECT, and our review of its risks and side effects including memory dysfunction, fairly reflect the current status of the accumulated knowledge. We have included, therefore, in Appendix I to this report an example of how we believe the benefits and risks of ECT might be presented to patients and their relatives. This example is based on the contents of the consent form of the Department of Psychiatry, Sibley Memorial Hospital, Washington, D.C.; it has been modified and amplified to reflect the views of the members of the Task Force and the outcome of studies on the use of unilateral and bilateral electrode placement.

(3) *Recommended Procedures*

In considering these, we recognize the need to think in only the most general terms. Different local and state jurisdictions across the nation have already adopted varying laws and regulations and the recommendations that follow can be applied only if they are molded to articulate with local requirements.

For instance, in some jurisdictions the issue of mental incompetence is separated from the voluntariness of hospitalization. In other

words, patients who are admitted to hospital involuntarily are not necessarily considered mentally incompetent; the court decides the issue of competence regardless of whether such patients have recognized their own need for treatment or whether they are admitted to hospital against their will. In other jurisdictions involuntary admission to hospital is taken to imply incompetence.

Another aspect of the competence issue that seems not to have been generally resolved relates to who should make the decision regarding incompetence. The judgment must ultimately be based on clinical data but in view of the deprivation of civil rights attached to such a judgment, the courts become involved, at times to consider the psychiatric testimony or merely to ensure the appointment of some person or persons to oversee the affairs of the patient. In practice, there is probably little uniformity across the nation.

It is not our intention here in the context of ECT to struggle with problems that the courts and the legislatures have yet to resolve at a much more general level regarding psychiatric treatment as a whole. In our recommendations, however, we cannot ignore them. Therefore what follows is a list of general categories with, when appropriate, a series of options that are available. Whichever option is chosen must mesh with the practices of the local jurisdiction; the goal in each instance should be the best possible clinical care for the patient.

(a) In the case of competent patients who consent to ECT, procedures should enable them to have the treatment within the context of the doctor-patient relationship and without the interference of others however well meaning they might be.

(b) Competent patients who, with good understanding of the situation, refuse ECT, also fall into a clear-cut category. They should not be treated. However, the psychiatrist should be able to withdraw from the case, and within the ethics practiced by the profession, be replaced by a colleague if he/she feels incapable of treating the patient without ECT.

(c) Incompetent patients who are incapable of providing informed consent but who do not protest the use of ECT in their treatment, cannot be described as giving informed consent. Good faith on the part of the patient's relatives and psychiatrist should be sufficient to ensure that what is being done, and done expeditiously, is in the best interests of the patient. Informed consent by concerned relatives fully meets the spirit of the doctrine of informed consent and should be sufficient to enable the psychiatrist to proceed with the treatment.

Doubts about the motivation or good faith of those involved in the decision-making can be dispelled by insisting on a consultation with an independent psychiatrist who does not practice in the same facility,

who understands the use of ECT, and who has no conflicting interests to influence his/her opinion. If he/she concurs, in writing, the treatment should be administered.

On the other hand, where local regulations involve the participation of the court either in the judgment regarding incompetence or in the appointment of a person or persons to oversee the affairs of the patient, such representatives must participate in the decision regarding the treatment.

(d) The category of the incompetent or involuntary patient who protests the use of ECT is complicated. If we assume that well-meaning relatives and the psychiatrist all in good faith agree that ECT is a necessary treatment, and if we assume too that an independent psychiatric consultant without a conflict of interest concurs, coercing the patient to the treatment room would hardly persuade a neutral observer that the doctrine of informed consent had been observed.

A series of options provides answers to the special demands of these circumstances; they range from extensive involvement of the court on the one hand, to minimal legal representation on the other with considerable dependence on the sensitivity and professional concern of the clinician and the good faith of the patient's relatives. Clearly, here again, whichever choice is made must mesh with the current laws and regulations of the local jurisdiction.

Extensive involvement of the court at times, including the appointment of legal counsel for the patient, is advocated by some authorities to consider psychiatric testimony regarding competency, to decide whether a reasonable person would object to the treatment, or to designate who may give informed consent on behalf of a patient declared incompetent. We share the concerns of Stone (32) and others that involving the courts in not infrequently complicated judicial hearings will involve cost, and perhaps more importantly, delays in the rendering of treatment.

Alternatively, and more in keeping with the views of this Task Force, there is place for greater emphasis on acceptable standards for the use of ECT, consultation with colleagues, and periodic retrospective peer review with minimal legal involvement confined largely to the appointment of a person or persons to represent the patients when this is considered advisable.

Other possible checks and balances, midway between these two approaches, might include involvement of the local mental health authority as a supervisory body or a court of special jurisdiction to expedite the resolution of treatment issues.

As indicated above, the problems must ultimately be resolved according to the regulations and practices of the various local jurisdic-

tions; and however relatives, guardians, independent professional consultants, legal counsel, and the courts finally have to articulate on behalf of a patient incapable of making judgments for himself/herself, the guiding principle should be good overall medical management with a minimum of delay and no unnecessary restrictions on the exercise of good clinical judgment.

(4) *Regulating ECT Use*

(a) *By Statute e.g. California Assembly Bill No. 1032, September 1976*

This bill illustrates what could occur in other states. It regulates medical practice by statute and contains inaccuracies and unnecessarily cumbersome and restrictive requirements. It is difficult to implement and is a poor way to achieve quality care. As such it must be addressed.

It regards ECT in the same light as it does psychosurgery which is an experimental procedure; ECT is not.

The Bill casts the psychiatrist in the role of a person intent on depriving patients of their civil rights. This prejudice is poorly substantiated and has been fostered by citizens' pressure groups and civil libertarians whose credentials and motives have not been subject to scrutiny.

The Bill specifically states that there exists a division of opinion as to the efficacy of the proposed treatment, why and how it works, and its commonly known risks and side effects. This Task Force finds no division of *informed* opinion about the efficacy of the proposed treatment in appropriately selected cases and goes to great lengths in this report to indicate that when ECT is well administered, the risks and side effects can be minimized. We cannot accept that legislators are able to evaluate clinical data and believe that in California HB 1032 they reached decisions beyond their competence.

The Bill allows the physician to urge the proposed treatment as the best one but does not allow him/her to "use, in an effort to gain consent, any reward or threat, express or implied, nor any form of inducement or coercion, including but not limited to, placing the patient in a more restricted setting, transfer of the patient to another facility, or loss of the patient's hospital privileges." These requirements are unrealistic. While no threats should be used to induce patients to consent to the treatment, it is highly unlikely that patients receiving either no treatment or slow acting treatment such as medication, can be granted the privileges and less limiting restrictions intended for patients who are recovering. Furthermore, restrictive

facilities might be essential for untreated patients continuously preoccupied with serious thoughts of suicide.

The unnecessarily large number of psychiatrists and concurring consultants needed to approve the use of ECT are costly and will encourage psychiatrists to abandon the treatment in favor of less effective methods. There is value in a consultant's opinion but one independent opinion is usually adequate under most circumstances. Among some who supported the Bill might well be those who hoped that by complicating the procedure they would dissuade both psychiatrists and patients from using it and they might yet succeed. Furthermore, provision for the financial cost of all the additional professionals is not included in the Bill and presumably funding must be found either through third party payers or the taxpayers.

(b) *By Regulations*

If the use of ECT is to be regulated in some manner, the regulations should be flexible to keep pace with the developing knowledge and should be in the hands of psychiatrists and other mental health professionals who are better able than legislators to evaluate clinical criteria. Furthermore, such professionals are not influenced by the give-and-take and political compromise so much a part of legislating. For example, state commissioners of mental health, with the assistance of their staffs and adequate consultation with psychiatrists who are trained or knowledgeable about ECT (should such individuals not ordinarily be on the staff of that department of mental health), are in a better position to formulate *regulations* about the use of ECT and supervise their application. This has already been illustrated by the regulations governing the use of ECT in the state of Massachusetts (33). These regulations were based on the Task Force Report on ECT in Massachusetts (34) and require that all cases receiving ECT in the state are to be reported periodically to the Department of Mental Health and are subject to review. A subsequent study (35) revealed that the regulations had been effective in considerably reducing the number of patients receiving more than 35 treatments per year. It is clear that the regulations can call for as much or as little review and exercise of authority as the local commissioner believes is needed at any time. This can vary according to the analysis of the data reported to the commissioner's office.

(5) *Patients for Whom ECT is Considered as an Experimental Procedure or as an Unusual Use*

As indicated in the previous section of this report, the weight of the research data leaves little room for doubt that ECT is an effective

and valuable treatment in some types of affective disorders and probably effective in the treatment of some cases of schizophrenia. The consensus of clinicians who use ECT, reflected in the answers to our questionnaire, supports this view.

However there exists among a minority of psychiatrists the opinion that ECT is an appropriate treatment for conditions like anxiety state, tension state, mild neurosis, mild depressive reaction, obsessive compulsive neurosis, and feelings of grief. This apparent difference of opinion is not unknown in medical practice.

These less seriously ill patients (quite capable of comprehending the information necessary for informed consent) are suffering with conditions considered by most psychiatrists to be responsive to the diligent use of other treatment methods such as medication or psychotherapy. Good clinical studies effectively demonstrating the superiority of ECT over more conservative treatments in such cases are lacking. The use of ECT for relatively minor conditions that appear to respond in the hands of others to less active and less costly treatments does, therefore, raise the question of overtreatment.

It seems to us that if such use of ECT is contemplated, care should be taken to study its effectiveness in a research setting or to consult first with colleagues who use a different type of therapy. As indicated elsewhere in this report, no individual is compelled to comply with these suggestions but if we aim to increase knowlege or enrich psychiatric practice generally by means of an exchange of opinions among the various schools of thought, the measures suggested here are highly desirable.

Psychiatrists involved in the study of the use of ECT in an experimental way are urged to recognize that their roles as researchers might make it difficult for them to evaluate the clinical needs of their patients without the consultative advice of peers who have no personal interest in the research (36). Those engaged in research should consult the DHEW guide on the protection of human subjects (37).

Before leaving this section on the experimental and unusual uses of ECT, the Task Force wishes to draw attention to the fact that there is little evidence to support the use of ECT in children; however, its use has been acceptable to a minority of psychiatrists on rare and exceptional occasions.

ECT should not be used with any patient to alter symptoms or to control behavior in the absence of a diagnosis.

(6) *Records*

(a) All deliberations and decisions relating to the use of ECT

should be recorded in the patient's medical record and be available to appropriate medical reviewing agencies.

(b) The numbers of ECT treatments administered to individual patients in any facility, the names of the psychiatrists administering them, and the details of the treatment procedure (see Chapter X, Recommendations) should be recorded and available to the appropriate medical reviewing agencies.

(7) *Professional Competence*

The qualifications and appropriate training of physicians recommending and administering ECT should be the concern of the licensing or registering agency of the local or state jurisdiction.

(8) *Facilities*

Only facilities adequately equipped for the administration of ECT (see Chapter X, Recommendations) should be used for such treatment.

(9) *Public Education*

The Task Force recommends the development and dissemination of relevant material to the public in order to keep it informed about ECT and to promote its confidence in the value of the treatment as well as in the capacity of our profession to review its own activities.

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CHAPTER VIII

TRAINING AND EDUCATION

Training and education in the theory and practice of ECT have been anything but standardized and have varied greatly in effectiveness in medical schools and residency training programs through the years. The crucial ingredients determining the level of instruction and on-the-job training have been the orientation and experience of the professional staff, their readiness to consider and incorporate growing knowledge into the treatment planning, and the degree to which ECT was used in the given institutions. An overview of psychiatric facilities throughout the country quickly reveals that there are centers where ECT has never been used, institutions where it has been extensively used, and all gradations in between.

After the introduction of ECT for the treatment of mental disorders in 1938, many years passed before anything resembling formalized training in ECT developed in psychiatric centers. Initially training was gained informally by apprenticeship to a senior therapist whose commanding position set the pace for the practice of ECT in that locale. Even today, because of the paucity of good training programs in ECT, this method of learning persists.

In the early years instruction was not prolonged, for there appeared not much to learn beyond the simple technique of delivering an electrical current capable of producing a grand mal seizure while standing by until the convulsion was safely over. With increasing use, knowledge accumulated. Technical developments were incorporated into new types of equipment with variations in electrical current, wave form, and monitoring devices (EEG and EKG readings simultaneous with the administration of the treatment). The use of premedication, anesthesia, oxygen and resuscitation equipment, experimentation in electrode placement, and varying numbers and frequency of seizures, were introduced in the service of making treatment safer, freer of complications, and therapeutically more effective.

Clinical developments have largely taken the form of sharpening and clarifying the disorders for which the treatment is more or less appropriate. We have a constantly growing body of data about physical, chemical, mental, and emotional changes that occur as a consequence of ECT, and many indications for investigative studies. More recently

ethical and legal issues have arisen, influencing the practice of psychiatry in general and the discipline of ECT in particular.

In sum, the body of information about ECT is now so large that a simple apprenticeship is of doubtful value, while thorough training courses tend to be few and far between.

This Task Force put together a series of questions on training in ECT which was included in a recent computerized questionnaire survey done by the American Association of Directors of Psychiatric Residency Training (AADPRT) of all aspects of psychiatric residency training programs.* About 100 responses were received but not all questions were answered. The following tabulation reveals as complete a picture as we have at present, with some mathematical discrepancies due to a lack of response to certain questions:

(1) What percentage of the total time devoted to the subject of Convulsive Treatment consists of:
   (a) lectures 19.6%
   (b) seminars 14.8%
   (c) practical, on-the-job, supervised participation administering ECT 51.7%

(2) Is education and training in CT the responsibility of a single staff member?
   Yes -32
   No -67

(3) Is an anesthesiologist involved in your CT education and training program?
   Yes -52
   No -47

(4) How many instructional hours on each of the following topics will an individual get during the course of an internship and residency?
   (a) 7.3 hours - clinical aspects of CT, i.e. indications, pre-CT workup, contraindications
   (b) 2.3 hours - legal and contractual issues involved in the use of CT
   (c) 4.5 hours - technical and procedural aspects of the treatment
   (d) 2.1 hours - complications of CT
   (e) 2.5 hours - airway management, cardio-pulmonary resuscitation
   TOTAL: 18.7 hours

* Taintor, Z., M.D., Director MSIS, Rockland Research Institute, Orangeburg, New York.

(5) Is CT administered at your hospital
   (a) by *ANY* staff psychiatrist?
      Yes -38
      No -62
   (b) by a treatment team which administers all treatments?
      Yes -60
      No -40
   (c) solely by an intern or resident physician?
      Yes -10
      No -90
      (Several respondents mentioned supervision by a senior staff psychiatrist and an anesthesiologist.)

(6) How many CTs will on the average be administered by an intern/resident during the course of his/her training?
   Mean Average 21.2

(7) Is competency of residents to administer CT assessed?
   Yes -58
   No -42

(8) Is competency of residents to administer CT reassessed?
   Yes -21
   No -79

(9) At what intervals is reassessment made?
   (a) Twice yearly 50%
   (b) Once yearly 50% (16 respondents)

(10) What specific text or literature do you use for instruction?
   The replies to this question revealed a paucity of useful texts and manuals which were specifically of value in ECT training. The most frequently mentioned texts were:
   Freedman and Kaplan - "Comprehensive Textbook of Psychiatry"
   Kalinowsky L., "Shock Treatments"
   Also mentioned were locally developed articles and manuals by individual practitioners or committees, journal articles and various textbooks.

(11) Called for "Comments"; answers most often given:
   7 - no longer administered because of legal issues
   5 - very little interest or enthusiasm in a program for ECT
   4 - ECT not used in a child psychiatry program
   3 - training ECT is closely supervised by senior staff
   2 - it was too small a part of the residency program to assign any members to it
   2 - questionnaire stimulated interest in re-formulating program
   2 - we believe ECT is one of the best treatments in psychiatry.

It is often under used and on occasion abused. We instruct residents to use ECT in the proper medical framework

1 - we need a consensus from the field on apppropriateness of ECT

1 - interest in CT in recent years has been extremely low but the new residents are very interested in it which would probably result in a more formalized program

1 - our facility no longer uses ECT. The small amount of training is done via affiliate hospitals

1 - ECT is used more in private hospitals so we use volunteer instructors on private hospital staffs to instruct residents in ECT

1 - we are currently re-evaluating ECT and hope to formulate a more specific program for teaching ECT

1 - ECT is an elective taken by 40% of the residents in the third year

1 - insurance rates are so excessive that treatment is no longer considered. The rare cases needing these treatments are referred or transferred elsewhere.

Several other surveys of training in ECT are of interest(*). Of 435 residents finishing their third year of training in 1974 only:

1.6% thought that giving ECT was undesirable;
11% reported no preparation to give ECT;
36% reported "some" preparation to give ECT;
22% reported "moderate" preparation;
23% reported "extensive" preparation.

In a survey of practice during 1975-1976 in Metropolitan (New York) hospitals on the use of ECT (**), of 30 departments giving ECT, 25 had resident physicians on their staff. They were allowed to give ECT in 22 units. Nineteen required a supervisor (staff psychiatrist with ECT experience) to be present. In three, a supervisor did not have to be present after an instruction period but had to be at the facility so that he/she could be reached if problems arose. In three departments residents were not permitted to give ECT but observed treatments. Lectures on ECT were limited to one to two hours during the three year residency. One department had a required seminar on ECT.

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* Taintor Z, Seiden A, Val E, et al: 1974 Residency Survey, unpublished data.

** Asnis G, Saferstein S, Fink M: Dept. Psychiatry & Behavioral Science, School of Medicine, Stony Brook.

Comments

In general the Task Force was surprised to find as much instruction as the most recent questionnaire indicated even though we advocate substantially more. It appears that there are centers where interest and instruction in ECT are of a very low order or the subject is practically avoided; other centers have well-developed, highly skilled departments of somatic therapy with good programs of training and research. An unfortunate aspect of the growth of legal restrictions on the use of ECT is that some departments of psychiatry have discontinued its use.

In an informal telephone survey it appeared that ECT is used more in private hospitals than in university training centers and hospitals. This is especially evident in California since the passage of the restrictive bill (AB 1032) governing its use. Several private hospitals continue to provide instruction in the use of ECT, varying from relatively informed on-the-job training to highly structured, well-organized courses given as electives to residents in nearby residency training programs.

A few well-organized courses of instruction have been inaugurated for practitioners by specialists in the field. Examples are the course offered by the Department of Psychiatry—Research, University of Oregon Health Sciences Center at Portland, Oregon in November 1976 and the workshop given at the Toronto APA Annual Meeting in May 1977. A similar one-day course entitled "Electroseizure Therapy: Problems and Solutions" was given at the Veterans Administration Hospital, Northport, New York in March 1977 by experts from various medical schools and hospitals in the area. Sample subjects covered in these seminars are: (1) neurophysiology of convulsions and theories on how seizures may improve psychic functioning; (2) indications and contraindications for ECT; (3) controlled studies of ECT; (4) treatment procedures; (5) nursing management; (6) management of complicated cases; (7) complications and their prevention; (8) newer techniques and equipment; (9) anesthesia in ECT; (10) cardiovascular and neurological problems; (11) the place of psychotherapy and pharmacotherapy in ECT; (12) legal aspects; (13) political and legislative considerations.

This Task Force, having reviewed the available teaching methods at undergraduate, graduate, and continuing education centers; having found textbooks and manuals on electroconvulsive treatment limited in scope; and having found the teaching curricula brought to its attention largely unsatisfactory, recommends the following:

(a) The APA recommend to the American Board of Psychiatry and

Neurology that it include questions regarding ECT in both written and oral Board examinations.

(b) A program for basic training in ECT be included in all psychiatric training programs. It should encompass:
  (i) indications and criteria for the use of ECT;
  (ii) clinical management of patients receiving ECT;
  (iii) mechanisms of therapeutic action in ECT;
  (iv) effects of ECT—including complications;
  (v) methods of administration and discussion of equipment and conditions requiring extraordinary care;
  (vi) informed consent and consent procedures;
  (vii) evaluation of outcome of ECT;
  (viii) new developments;
  (Elective training programs should be available to residents intending to use ECT in practice. A minimum of 30 hours should be devoted to the subject including the management of at least two patients through their courses of ECT.)

(c) Medical students should be exposed to a modified version of (b) above which should include observation of treatment by demonstration or videotape.

(d) Efforts should be made to provide continuing education in ECT. To this end, it is suggested that:
  (i) APA include continuing education training sessions on ECT at annual meetings, at divisional meetings, and Hospital and Community Psychiatry Institutes;
  (ii) APA includes a section on ECT in the annual program;
  (iii) the Rules and Regulations of each hospital using ECT require that psychiatrists giving ECT be qualified by training and experience to give the treatment. This should be determined by the Peer Review Committee and/or the Medical Quality Assurance Committee or its equivalent in each hospital.
  (iv) medical schools and hospitals be urged to establish divisions of somatic therapy and to provide courses in various aspects of ECT for the practicing psychiatrist;
  (v) the report of this Task Force be made available to those planning training programs.

CHAPTER IX

FUTURE RESEARCH

This chapter will briefly review which of the several unexplained aspects of ECT should be investigated to provide the data that are urgently needed. Before proceeding to an outline of the topics to be studied, we will refer briefly to funding and outline the criteria essential for systematic research and the observance of ethical requirements.

The Research Task Force of the National Institute of Mental Health reported in 1974 (1) that less than one percent of its total somatic therapy research grants from 1968 to 1973 were devoted to convulsive therapy. Of 9.9 million dollars granted for somatic therapy research in 1972 and 1973, less than five thousand dollars was provided for the study of ECT. Considerably greater sums of money will have to be spent before we resolve the challenging questions referred to in this report.

Many of the earlier publications referred to in these pages lack the rigorous scientific methods of modern clinical research; the data in those instances are unsatisfactory. The very nature of the ECT procedure renders blind clinical studies difficult to achieve. Nevertheless, attention should still be paid to the methodological principles of homogeneous diagnostic groups (when feasible), random samples, prospective methods, appropriate and simultaneous controls, significant sample numbers, and blind outside raters where possible. The study should attempt to specify the treatment task; the nature of the intervention; details of the procedure; the action mechanisms; and the nature, degree, and durability of the effected change.

It is important not only to consider the diagnosis and personality differences in categorizing patients but also to consider the cultural or class differences that might affect response. Standardized report forms should be utilized, permitting increased comparability of data. These measures should distinguish between "change" and "outcome," i.e. what criteria of change were used in arriving at a judgment regarding degree of improvement. Computer analysis of derived data, dissection of confounding variables, and multiple hospital involvement should be considered. Methods should be well-designed to include number

and frequency of ECT, parameters of electrical stimulation, electrode placement, type of equipment, and evidence of a cerebral seizure as well as adequate drug levels (serum measurements), quantitative neurophysiologic measures, and detailed behavioral assessments.

Clinical research involving ECT should be conducted in a research setting that meets community standards and the requirements of patients' rights. Prior approval should be obtained from the human rights and research committees or their equivalent. Special attention should be paid to adequate record-keeping including: details of previous history, rationale for treatment, careful observations of the patient, documentation of all deliberations relating to the use of ECT, the number of treatments, complications, etc. It is essential that informed consent be given by the patient and/or the court or the relatives. (See Chapter VII)

The areas reviewed below have been abstracted from the preceding chapters and represent the more important questions that require attention.

(a) *Clinical Efficacy in Depression*

(i) How does ECT compare with high-dose tricyclics, and tricyclics plus antipsychotic drugs, in the treatment of delusionally depressed patients?

(ii) Is there a pure ECT-response, non-drug-responsive group of patients and what are their clinical characteristics?

(iii) When in the course of antidepressant and antipsychotic treatment should ECT be employed, and when should ECT be the preferred treatment? Does the simultaneous use of drugs and ECT enhance outcome? What are the risks?

(iv) Can a dose-response curve be determined for ECT for various clinical entities?

(v) What is the long term follow-up of matched groups of patients treated with ECT and long-acting antipsychotic medication respectively?

(vi) What is the long term effect of ECT on the natural history of the illness?

(vii) How do the results of maintenance ECT compare with antidepressant drug maintenance and lithium?

(viii) What is the comparative efficacy of unilateral and bilateral electrode placement in severe depressive illness?

(b) *Clinical Efficacy in Schizophrenia*

(i) What is the efficacy of ECT in schizophrenia?

(ii) What specific diagnostic categories are most responsive?

(iii) Does the presence of a marked affective component improve the response to ECT?
(iv) What is the value of unilateral electrode placement in schizophrenia?
(v) How effective is ECT in the treatment of chronic schizophrenia?
(vi) Do schizophrenic patients require larger numbers of treatments for a response to occur?
(vii) What is usually the minimum number of treatments needed for a response?
(viii) Are multiple treatments more effective than single treatments?
(ix) Does the simultaneous use of drugs and ECT enhance the outcome? What are the risks?

(c) *Clinical Efficacy in Mania*

(i) How do lithium, ECT, and phenothiazines compare?

(d) *Clinical Efficacy in Conditions other than Schizophrenia and Affective Disorders*

The role of ECT in organic brain syndromes, hypochondriasis, intractable pain, and severe neurotic conditions is very poorly defined.

(e) *Adverse Effects of ECT*

(i) What is the incidence of spontaneous seizures after ECT and are barbiturates prophylactic?
(ii) What is the present incidence of death with ECT and what is the cause of death?
(iii) a. How long after ECT do memory complaints persist?
b. Is there any permanent loss of memories acquired during the period of one month to three years prior to ECT?
c. Are persisting memory complaints related to ECT or to other factors?
d. How important to the patient is memory loss?
e. How do patients view the ECT experience six months or more after treatment?
(iv) Is multiple-monitored-ECT less damaging to memory than single ECT?
(v) What are the comparative effects on memory of different kinds of current parameters?

(vi) What is the effect of the cerebral seizure and, in general, how can the therapeutic effect of ECT be dissociated from its adverse effects?
(vii) Can suitable animal models of depression be found for ECT research?

(f) *Theoretical Questions*

The challenge is to find more specific ways of evoking the central process by which the therapeutic effect of ECT is accomplished.

(i) Are subcortical structures primarily or exclusively involved in the therapeutic effect?
(ii) What is the role of acetylcholine in the ECT process?
(iii) Would stimulation of the brain stem directly produce behavioral improvement in depressed patients?
(iv) Do personality variables affect the outcome of ECT?
(v) Are there differences in response between schizophrenic and depressed patients (measured in number and frequency of seizures for a behavioral change) which may be related to CSF chemistry, cerebral electrophysiology, or to other factors? Continued efforts are needed to formulate an integrated biochemical and electrophysiologic hypothesis for the action of ECT in schizophrenia.

(g) *Advisability of a Committee on ECT*

Because of the developing nature of our understanding of ECT, it is important that this report allow for the integration of new knowledge. We are therefore recommending the establishment of a Committee on ECT which would act as a clearinghouse for data accumulated from further studies. It would act in an advisory capacity to provide guidance on the use of ECT; and it will retain the flexibility which will allow for the modification and updating of the recommendations of this report.

Reference

1. Research in the Service of Mental Health: Report of Research Task Force of National Institute of Mental Health. DHEW Publication (ADM) 75-236, 1975, chapter 12

CHAPTER X

RECOMMENDATIONS REGARDING THE USE OF ELECTROCONVULSIVE THERAPY

In formulating the following we emphasize that:
(i) Clinical diagnosis of psychiatric disorders is not yet precise and absolute but is developing.
(ii) There is insufficient evidence to support exclusively any one preferred sequence of treatments in psychiatry.
(iii) The techniques of ECT are evolving and differ today from what they were during the first two decades after its introduction, e.g. the routine use now of anesthesia, oxygenation, and muscle relaxation. Further, the placement of both electrodes over the right hemisphere in right-handed patients (non-dominant unilateral ECT) has been demonstrated as an effective treatment with considerably less memory impairment compared with bilateral electrode placement.
(iv) Indications for the use of ECT have been more sharply defined.
(v) The dangers of alternative therapies can be considerable.

I. *Indications*

A. ECT is an effective treatment in cases of:
   (1) severe depression where the risk of suicide is high and/or where the patient is not taking adequate food or fluids and/or where the use of drug or other therapy entails high risks and/or will take an unacceptably long period to manifest a therapeutic response;
   (2) severe psychoses characterized by behavior which is a threat to the safety and well-being of the patient and/or others and which cannot be controlled by drugs or other means, or for which drugs cannot be employed because of adverse reactions or because of the risks which their use entails;
   (3) severe catatonia which has not responded to drugs and/or where the patient is not taking food or fluids and/or where drug therapy or other means entail unacceptable risks;
   (4) severe mania where the use of drug therapy entails unacceptable risks and/or where coexisting medical problems (e.g.

recent myocardial infarction) either require prompt resolution of the mania and/or make the use of drug therapy unacceptable.

B. ECT is probably effective in:
   (1) depressions, particularly those characterized by vegetative or endogenous symptoms, which have not responded satisfactorily to an adequate course of therapy with antidepressant drugs;
   (2) depressions, particularly those with vegetative or endogenous elements, in which the use of drug therapy is contraindicated;
   (3) psychoses, and particularly those with an endogenous affective component, which have not responded to an adequate trial of anti-psychotic drugs or where drugs cannot be used because of adverse reactions.

C. There exists among some psychiatrists the opinion that ECT is an appropriate treatment for conditions other than the affective disorders and schizophrenia. Although there are few published studies reporting the efficacy of ECT in the treatment of behavior disorders, character disorders, anxiety neurosis, or feelings of depression related to recently saddening or disappointing events in life, such use has been acceptable in the past to a minority of psychiatrists. Even in children and adolescents, the use of ECT has been acceptable to a small group of psychiatrists on rare and exceptional occasions. If ECT is used in conditions other than the affective disorders and schizophrenia, it is advisable that care be taken to study its effectiveness in a research setting or to seek consultation with colleagues who though understanding the use of ECT generally use a different type of treatment method. No individual is compelled to comply with these suggestions but if we aim to increase knowledge or enrich psychiatric practice by means of an exchange of ideas among the various schools of thought, the measures suggested here are highly desirable.

D. In the absence of the clearly defined conditions listed above in sections A and B, ECT should not be used solely to control symptoms or violent behavior.

E. The idea of selecting ECT as a treatment method for reasons that are politically discriminatory, or for punishment, is abhorrent.

F. Conditions requiring extraordinary care and experience:
   In the presence of serious physical conditions such as space-occupying intracranial lesions and recent myocardial infarction, as well as advanced pregnancy, the administration of ECT should be in the hands of a team of physicians who, collectively, have had considerable experience both with the use of ECT and with these conditions.

II. *Civil Liberties and Informed Consent.*

The Task Force, recognizing that psychiatric illness can and does at times uniquely modify a patient's capacity to give informed consent, is attempting in these recommendations to balance the individual's clinical needs with his or her civil rights. Particular attention has been paid to this balance because a rigid emphasis on either legal rights or clinical demands leads to poor clinical management. In whichever manner the recommendations regarding consent procedures will be implemented, to be effective they must ultimately mesh with or be influenced by local and state regulations and rulings. The following four general categories of patients have been considered in Chapter VII, pages 143-145. For details, the reader should refer to that section of the report. The categories are: (a) competent patients who consent to ECT, (b) competent patients who refuse ECT, (c) incompetent patients incapable of providing informed consent who do not protest the use of ECT in their treatment, and (d) incompetent or involuntary patients who protest the use of ECT.

We recommend meticulous record keeping by all psychiatrists and facilities involved in the use of ECT. The records should include:

(a) the nature and history of the clinical condition leading to the consideration of ECT;
(b) the details of previous treatments including therapeutic response and adverse reactions;
(c) the reasons for selecting ECT;
(d) the details of all discussions relevant to consent to treatment;
(e) the signed consent form, with the signatures of the patient and/or the relatives, or guardian when appropriate;
(f) the signed concurring and contradictory professional opinions where they exist; and
(g) specifics of the treatment, e.g. unilateral or bilateral electrode placement, dates of treatment, characteristics of the current, drugs administered, etc.

Informed Consent

To provide informed consent, the patient and/or his/her close relative or guardian must be competent to understand the following in simple language and, in agreeing, should sign a Consent Form indicating consent to the procedure:

(a) the nature and seriousness of the disorder;
(b) the probable course that is likely with or without ECT (without providing guarantees);

(c) a description of the procedure;
(d) the nature, degree, duration, and probability of significant risks and/or side effects and/or adverse effects. Special attention should be paid to post-treatment confusion and memory dysfunction;
(e) a description of reasonable treatment alternatives, and why ECT is being recommended;
(f) the right of the patient to accept or refuse ECT, the right to revoke his/her consent at any time, and acknowledgement that the consent is for a specified maximum period of time. Additional treatments shall require a renewed written informed consent; and
(g) the cost of the proposed treatment.

Furthermore, the following information should be entered in the patient's official record in a standard form:

that the information outlined from (a)-(g) above has been presented to the patient, and/or his/her relative or guardian;
that the patient, relative, or guardian is competent to understand and intelligently act upon this information; and
that the consent for a course of treatment is being provided voluntarily, without coercion.

The above statement should appear before the signature of the attending physician.

(Those interested in an example of how the information necessary for informed consent can be presented to patients, relatives or guardians should see Appendix I.)

III. *The Administration of ECT*

Here we wish to emphasize what we have already repeated in the pages of this document, namely, that what follows is a detailed description of but one of the several methods available for administering ECT in an acceptable manner. Procedures which differ in one or more details from that which is described here are frequently used and may be equally acceptable.

The nature of ECT is such that it should be given with general anesthesia and muscle relaxation. In view of the safety and efficacy of these modifications of the procedure and in view of the discomfort and serious complications that can be associated with an unmodified procedure, the use of unmodified ECT is no longer advised. The setting for the treatment should provide facilities for the modified

treatment and for the immediate management of complications that may arise. The treatment team should be competent to deal with routine and special problems and should have facilities for immediate management of complications that may arise.

A. *Pre-Treatment Evaluation*

This includes an adequate medical and anesthetic history, a thorough physical examination, appropriate laboratory tests, and specialized consultations as indicated. The subject is complicated by the varying pre-anesthetic routines that are customary in the different parts of the country. The Task Force subscribes to the view that the pre-ECT evaluation should embody the principles of good medical practice supplemented by those special procedures, consultations, and investigations relevant to the use of ECT in a particular patient.

The following applies:

(a) The history includes items pertinent to the existence of pulmonary, coronary, vascular, neurological, and orthopedic disease as well as untoward responses to medications, particularly those related to previous anesthetic procedures.

(b) The physical examination includes special attention to the possible existence of pulmonary, coronary, cardiovascular, neurological, and orthopedic disorders. Special attention is paid to the presence of fractured or loose teeth and dental appliances.

(c) Laboratory and special investigations and procedures are considered within the context of the findings of the history and physical examination. Attention is given to a determination of the plasma pseudocholinesterase activity level in selected patients (see Chapter V, page 107) prior to the use of succinylcholine. Spinal radiographic examination is considered in the light of the individual's history and physical examination. X-ray procedures are contraindicated when pregnancy exists, or is possible; it is valuable as a means of demonstrating that radiographic abnormalities have existed prior to the proposed treatment (e.g. as a result of a fall, automobile accident, etc.).

The basis for these recommendations and a fuller discussion of the rationale for these and other procedures will be found in Chapter V.

B. *Treatment Considerations*

It is recommended that:

(a) Precautions be taken so that patients receiving ECT do not

have solid materials (e.g., pills, solid food particles) in the stomach because of the danger of regurgitation, vomiting, and possible respiratory tract obstruction.

(b) ECT only be administered in a location, and under circumstances, where there is immediate (i.e., within 2-3 minutes) availability of and access to the professional skills, equipment, and drugs necessary to manage complications which can occur unpredictably whenever the treatment is administered to *any* individual. In general, this will necessitate that the treatment be administered in a hospital or in some appropriately equipped facility.

(c) Whenever possible, the treatment team should include a nurse and aides who have had training and experience in carrying out the procedure of ECT and in managing the complications associated with this form of therapy.

(d) ECT be administered only to patients who are adequately anesthetized, as determined by objective criteria, so that they are unconscious of events during the period prior to the seizure e.g., discomfort from the succinylcholine muscle contractions, being paralyzed and unable to breathe, and conversations.

(e) An intravenous line (e.g., scalp-vein "butterfly" needle) be maintained, until the patient is responding following the treatment, for the administration of appropriate medications in the event of adverse reactions.

(f) An anticholinergic agent, preferably of the quaternary type (e.g., methscopolamine), be used at the discretion of the physician administering the treatment in doses sufficient to prevent severe bradycardia or asystole during the seizure (see Chapter V).

(g) All patients receive assisted or artificial ventilation with oxygen-enriched gas mixtures from the time that consciousness is lost until a normal tidal volume has been attained at the end of the procedure, except during the convulsion.

In patients who are guarded anesthetic-medical risks, a one or two minute period of deep breathing of an enriched oxygen mixture before administering the anesthetic agent is recommended as an additional safeguard against hypoxemia and hypercarboxemia.

(h) The individual psychiatrist in each case should decide whether unilateral or bilateral treatment is the best mode of ECT administration for any particular patient. It should be noted that the members of the Task Force have been persuaded by their joint experience and by the available data to

favor the use of unilateral ECT because several studies report it to be as effective as bilateral ECT and because memory disability following the unilateral method is considerably less than that following bilateral treatment. Nevertheless, some experienced clinicians in the United States currently view unilateral ECT as less effective than bilateral treatment and a few regard it as ineffective. A few of the possible explanations for this disparity between published studies and individual experience are considered in Chapter II, page 21. Further studies of the comparative value of these two methods of administering ECT are strongly recommended.

(i) A technique should be routinely employed to ascertain that a therapeutically-adequate seizure has, in fact, been induced. A seizure lasting less than 25 seconds may not be adequate. Three practical methods are available:
   (1) observing that a bilateral tonic → clonic progression of muscle convulsive activity has occurred *after* the electrical stimulus has ceased. The tonic contraction occurring *during* the application of the electrical stimulus is due to the transmotor-cortical passage of the current and does not, in itself, indicate that a therapeutically-adequate seizure has been induced;
   (2) the "tourniquet" or "controlled convulsion" technique in which the muscle paralysant is excluded from one extremity by means of an inflated blood pressure cuff, is useful for preserving a mass of unparalyzed muscle which can reliably show the post-stimulus tonic → clonic convulsive muscle sequence; or
   (3) EEG monitoring of the cerebral seizure.

(j) Psychiatrists recognize that a difference of opinion exists regarding the advisability of glissando and subconvulsive electrical current applications. It is our understanding that memory disability is worsened by increased amounts of electrical current. As both techniques unnecessarily increase the total amount of applied current, we cannot recommend their use although we recognize that they are acceptable techniques to a small minority of psychiatrists. The objective in using them to modify and "soften" or eliminate the convulsion can be more satisfactorily attained by proper use of anesthetic and muscle paralysant drugs.

(k) The teeth should be examined when the convulsion has ceased; appropriate steps should be taken if dental fractures or dislocations have been produced.

(l) Recognition should be accorded the danger associated with the occurrence of regurgitation and/or vomiting at the time skeletal muscle action is reacquired in the immediate post-convulsive period. The danger of pulmonary aspiration is the greatest in the period before pharyngeal and laryngeal reflexes have completely returned. Three precautions will minimize the possibility of pulmonary aspiration:
(1) immediate availability of working suction;
(2) attendants available for immediately turning patient on side; and
(3) particularly in the event that someone is not close by, patient should be placed in prone position until pharyngeal reflexes have definitely returned and until response to verbal statements occurs.

C. *Post Treatment Considerations*

It is recommended that:

(a) Adequate supervision should be provided, especially during the period when the patient is drowsy, confused, and less-than-normally alert. This supervision should be directed particularly toward prevention of injury to the patient or to others as a result of:
(1) falling;
(2) operation of motor vehicle or machinery. Outpatients should be sent home in the company of a responsible adult.

(b) During the post-treatment periods, protection should be provided against difficulties in professional, business, work, or personal affairs, as a result of memory dysfunction.

D. *General Considerations*

It is recommended that:

(a) Scheduling of treatments should be dictated by the nature, severity, and seriousness of the patient's psychiatric disorder, by the presence of associated medical problems, and by the intensity of memory and confusional side-effects.
Generally, three treatments are given per week. Allowing one or two days between ECTs makes possible a better assessment of the patient's response on a day free of the effects of the treatment.

It may be advisable to administer ECT more frequently, e.g. in severe manic reactions not responsive to drugs or where

psychopharmacotherapy entails unacceptable risks. In elderly patients who develop marked memory dysfunction and confusion, once-weekly or twice-weekly treatments may be advisable.

Regarding techniques involving multiple treatments given under a single anesthetic session (e.g. multiple ECT), the data are inadequate and further study is imperative.

(b) The number of treatments administered to an individual patient should be guided by the following:

affective disorders—Studies of affective disorders suggest that a course of ECT consisting of six to 10 treatments is usually sufficient to give satisfactory response in the average case. It is recommended that if an affective disorder has not adequately responded to a course of 15 ECTs, additional treatments should not be administered except under unusual circumstances and after there has been a careful review of the patient's diagnosis, treatment program, and possible alternative forms of therapy. The Task Force recommends that care, judgment, and restraint be exercised before prescribing a second course of ECT within a 12-month period.

psychotic disorders—In the absence of adequate published data regarding the use of ECT in the psychoses (especially the schizophrenias), the Task Force considers it inadvisable to make precise recommendations. Although in many instances a smaller number might suffice, we suggest that the maximum number of ECTs per course not exceed 25-30 treatments. In those exceptional cases where the attending physician believes that a second course is indicated, it is advisable that he/she consult with colleagues before proceeding.

maintenance ECT—Here again the published data regarding this use of ECT are inadequate. Further study and the documentation of clinical data are imperative.

(c) Psychiatrists recognize the lack of consensus regarding the administration of ECT to patients who have, within the previous seven to ten days, been receiving:

reserpine and reserpine-containing preparations;
monoamine oxidase inhibitors;
anticholinesterases;
tricyclic antidepressants;
lithicum; or
antipsychotic drugs.

While some authorities believe that psychotropic drugs used in conjunction with ECT may enhance outcome in selected

patients, others indicate that since psychotropic drugs have potent venous pooling effects and can cause hypotension, the possible advantage of using them simultaneously with ECT may not be uncomplicated. This issue needs further study.

(d) The so-called reactivation technique should not be employed as a therapeutic measure. This technique requires that the patient be conscious and able to evoke certain thoughts (e.g. depressive ideation) immediately prior to the induction of the seizure. The thesis that this evoked material is particularly susceptible to ECT-induced retrograde amnesia is based on *animal* work which is itself inconclusive and controversial. This fact notwithstanding, the Task Force feels that the use of this technique is incompatible with our recommendation that patients receiving ECT should be rendered unconscious by the use of adequate depths of anesthesia. The reactivation technique is an experimental procedure which should be used only under appropriate investigational conditions (see Section V on Research, this Chapter).

IV. *Training and Education*

The Task Force, having reviewed the available teaching methods at undergraduate, graduate and continuing education centers; having found textbooks and manuals on ECT limited in scope; and having found the teaching curricula brought to its attention largely unsatisfactory, recommends the following:

(a) APA recommend to the American Board of Psychiatry and Neurology that questions regarding ECT be included in both written and oral Board examinations.

(b) A program for basic training in ECT should be included in psychiatric residency training programs. It should encompass:
   (1) indications and criteria for the use of ECT;
   (2) clinical management of patients receiving ECT;
   (3) mechanisms of therapeutic action in ECT;
   (4) effects of ECT—including complications;
   (5) conditions requiring extraordinary care, methods of administration, and discussion of equipment;
   (6) informed consent and consent procedures;
   (7) evaluation of outcome of ECT; and

(8) new developments;
(Elective training programs should be available to residents intending to use ECT in practice, including the management of at least two patients through their courses of ECT.)

(c) Medical students should be exposed to a modified version of (b) above, which should include observation of treatment by demonstration or videotape.

(d) Efforts should be made to provide continuing education in ECT. To this end, it is suggested that:
(1) APA include continuing education training sessions on ECT at annual meetings, at divisional meetings, and Hospital and Community Psychiatry Institutes;
(2) APA include a section on ECT in the annual program;
(3) the rules and regulations of each hospital using ECT require that psychiatrists giving ECT be qualified by training and experience to give the treatment. This should be determined by the Peer Review Committee and/or the Medical Quality Assurance Committee or its equivalent in each hospital.
(4) medical schools and hospitals be urged to establish divisions of somatic therapy and to provide courses in various aspects of ECT for the practicing psychiatrist; and
(5) the report of this Task Force be made available to those planning training programs.

V. *Research*

There remain many unanswered questions regarding the efficacy of ECT in certain clinical situations and its mode of action. Answers to these and other questions will only be found if well-designed research is undertaken.

(a) It is strongly recommended that funds be made available at all levels to stimulate further studies in the area of ECT.

(b) Areas awaiting investigation include neuro-pathological studies, long-term follow up studies, efficacy of maintenance ECT and multiple-monitored ECT, neuro-endocrine changes with ECT, and the effectiveness of ECT in non-affective and non-schizophrenic disorders. (A more extensive review can be found in Chapter IX.)

(c) Any research undertaken should be carried out in a systematic

manner which respects the patient's rights and meets community standards. Special attention should be paid to adequate record keeping, problems of diagnosis, comparison groups whenever feasible, controls, and double blind trials. Selection should be random, samples should be homogeneous, and treatment methods should be well-designed to include, for example, adequate drug levels and evidence of cerebral seizures. Peer review and informed consent are essential.

(d) A special APA Committee on ECT should be established (see Section VI below) to monitor and supervise the recommendations of this report, and act as a clearinghouse for the data accumulating from further studies on ECT.

(e) The development of criteria for the effectiveness and safety of ECT instruments and equipment should be the responsibility of the APA Committee on ECT.

VI. *Continuity*

To permit the ideas and recommendations emanating from the work of this Task Force to develop, it is recommended that APA establish a *Committee on ECT*. The charge to such a committee would be:

(a) to implement, where possible, and/or oversee the recommendations of this report;

(b) to provide guidance on the use of ECT, the research on ECT, and the education and qualifications of those who administer it;

(c) to ensure access to data on developments in the field of ECT which will enable them to advise APA on matters of policy regarding ECT; and

(d) to retain the flexibility which will allow them to modify and update the recommendations of this Task Force, and their own recommendations, in the light of new knowledge.

VII. *Confidence and Trust*

Although confident of our findings and recommendations, we are cognizant that some may view this report as self-serving because it has been assembled by an APA committee which comprises mostly APA

members. We recommend, therefore, that APA invite the National Commission for Protection of Human Subjects of Biomedical and Behavioral Research to examine this report and to take or suggest whatever additional steps it considers necessary to confirm the effectiveness and safety of electroconvulsive therapy when administered competently to suitably selected patients.

APPENDICES

APPENDIX I. DESCRIPTION OF ECT FOR INFORMED CONSENT

The following might be helpful to those interested in an example of how the information necessary for informed consent can be presented to patients, relatives, or guardians. It is not offered as a rigid prescription and should be modified, when necessary, to meet the needs of those who must understand its contents.

Procedures of Electroconvulsive Therapy

Electrotherapy, also known as electroconvulsive therapy (ECT) or electroshock therapy (EST), is an accepted form of treatment for certain types of psychiatric disorders. It has been used successfully in thousands of cases in this country and abroad since its introduction in 1938. It is one of the most effective ways of treating depression and certain other conditions in patients who might otherwise require lengthy hospitalization as well as the prolonged use of high doses of medication.

The psychiatrist and his/her skilled and experienced team give the treatment in a specially equipped treatment room. Treatments are usually given in the morning before breakfast. The treatment consists of passing a small, carefully controlled electric current between two electrodes applied to the head. In bilateral treatment, one electrode is applied to the right side of the head, the other to the left. In unilateral treatment, both electrodes are applied to the same side of the head, usually on the right side. Prior to each treatment the patient receives an injection to reduce secretions in the mouth. No special dress or gown is required.

Once in the treatment room, the patient is given an anesthetic in the vein which leads to sleep within a matter of a minute. He/she is then given a second medication in the vein which produces muscular relaxation. The patient should experience no discomfort or pain during the treatment; he/she does not feel the electric current and most individuals have no memory of the treatment. When the treatment is

given, the patient, who is already asleep, has generalized muscular contractions of a convulsive nature. These contractions, which have been modified and "softened" by the second medication in the vein, last approximately 60 seconds. Minutes later, the patient slowly awakens and may experience temporary confusion similar to that seen in patients emerging from any type of brief anesthesia. When the patient is ready, he/she is then returned to the room. Following this, he/she is given breakfast and is permitted to be up and about. Headache, mild muscle soreness, or nausea sometimes occur but these are infrequent and usually respond to simple treatment.

The number of treatments in any given case will vary with the condition being treated, the individual response to treatment, and the medical judgment of the psychiatrist giving the treatments. A typical course of therapy may consist of four to ten treatments. In some cases more treatments may be required. Although the treatments are usually given every other day or three times a week, the frequency of treatment will also vary with each case. As the treatments progress (usually after the fourth or fifth treatment), haziness of memory may develop. This memory impairment is usually temporary and will be described in detail in the section below titled "risks, side-effects, or adverse effects."

Convalescence

After the last scheduled treatment the patient begins a "convalescent period" the duration of which varies with each individual. During this period (usually one to three weeks) he/she must either remain in the hospital or be discharged under the supervision of a family member or some responsible person selected by the family. This precaution is necessary because of the impairment of memory, largely temporary, which is an expected side-effect of the treatment. During the convalescent period, the patient should not drive an automobile, transact business or carry on usual employment until *so advised* by his/her doctor. Alcoholic beverages are prohibited. A responsible person should remain with the patient until such time as convalescence is completed. The duration of the convalescent period is determined by the patient's progress.

Outpatient Treatments

In some instances outpatient treatment may be recommended; this generally consists of a full course of treatment over a two to four week

period without the patient having to enter the hospital as an inpatient. A member of the family or a designated responsible person accepts the responsibility for:

(1) seeing that the patient does not take any food or drink after midnight preceding each treatment;
(2) escorting the patient to the hospital for the appointed treatment; and
(3) escorting him/her home after the treatment has been completed.

During the period of treatment, and for at least two to four weeks following termination of treatment, the patient should be under the close supervision and constant companionship of the family.

Results

Now a word about the results of treatment. Although the results in most cases are gratifying, not all cases will respond equally well. As in all forms of medical treatment, some patients will recover promptly; others will recover only to relapse again and require further treatment; still others may fail to respond at all.

Risks, Side Effects, or Adverse Effects

ECT, like any other medical or surgical procedure, involves a certain element of risk. Careful medical evaluation is carried out in each case to insure that there are no over-riding medical contraindications to the treatment.

Fatalities are very rare.

Complications, although infrequent, may include fractures and/or dislocations or adverse reactions to the medication. These may sometimes occur in spite of all precautions and must be looked upon as a recognized hazard of the treatment. The patient should be assured, however, that every effort will be made by his/her doctors to minimize the possibility of such complications; should they occur, appropriate treatment will be instituted immediately and the family notified.

Memory Changes

The haziness of memory, or poor memory function referred to above, is a frequent side effect of the treatment and one that has

received wide publicity in recent years. From careful studies we now believe that small numbers of treatments are less likely to produce marked memory changes than large numbers. We now also believe that right unilateral treatments (both electrodes applied to the right side of the head in right-handed persons) lead to milder and shorter lived memory changes than those following the use of bilateral treatment (one electrode on each side of the head) or left unilateral treatment (both electrodes on the left side of the head). However, the doctor makes the decision about electrode placement in each case.

This section is intended primarily for patients, relatives, and guardians who are interested in the details.

Bilateral ECT or left unilateral ECT might be associated with:

(a) memory loss for some *past events* lasting from a few hours after one treatment to a few months or, possibly though rarely, even for years after a series of treatments. This memory loss is largely reversible though permanent loss can occur for the days and weeks close to the time the treatments are given. Spotty loss may remain for events that occurred during the months prior to treatment and some spotty memory loss may possibly remain for events that occurred during one or two years prior to treatment; and

(b) memory loss for *ongoing events* lasting from a few hours after one treatment to several months after a series of 5-15 treatments.

Right unilateral ECT might be associated with:

(a) spotty memory loss for *past events* lasting from an hour after one treatment to several months after a series of treatments. The memory loss is largely reversible though permanent memory loss may occur for the days and weeks close to the time the treatments are given; and

(b) spotty memory loss for *ongoing events* lasting from a few hours after one treatment to several months after a series of treatments.

The great majority of patients treated with ECT do not find these memory changes of major importance and countless individuals in the professions, in high academic positions, and in responsible executive jobs in commerce and industry, have returned to fill their former occupations effectively. Their colleagues and co-workers, unless informed of the memory changes, have paid little attention to them. Other patients, for reasons that are not yet fully understood, have continued to complain about their poor memory function for prolonged periods of time. Further studies are needed to explain the differences.

If you have any other questions about the treatment, we will do our best to answer them.

AMERICAN PSYCHIATRIC ASSOCIATION
TASK FORCE ON ELECTROCONVULSIVE THERAPY (ECT)

INSTRUCTIONS: You will help us keep the cost of processing these forms as low as possible if you will carefully record your answers according to the following rules:

Please

- Use a soft black pencil
- Erase completely any answer you wish to change
- Answer each question by writing the code number of the correct response or an actual number (such as age) in the box or boxes provided.
- For some questions your answer may be a number with fewer digits than the number of spaces provided. *In this case enter it to the extreme right in the space provided and fill the empty spaces with zeros.* To use an impossible but convenient example, if your age were 9 years, you would complete item 1 as 0 9 . *It is extremely important that you follow this instruction.*

SECTION 1			Cols.
1. YOUR AGE AT LAST BIRTHDAY?		☐☐	1–2
2. YOUR SEX?	1-Male	2-Female ☐	3
3. NAME AND LOCATION OF MEDICAL SCHOOL FROM WHICH YOU GRADUATED? (Leave boxes blank)		☐☐☐☐☐	4–8

OVER, PLEASE

4. YEAR OF GRADUATION FROM MEDICAL SCHOOL? (last 2 digits) ☐☐ 9–10

5. NUMBER OF YEARS OF APPROVED PSYCHIATRIC RESIDENCY TRAINING? ☐ 11

6. NAME OF PSYCHIATRIC RESIDENCY TRAINING PROGRAM?
Indicate last attended if more than one; indicate none if applicable.
(Leave boxes blank) ☐☐☐☐☐ 12–16

7. YEAR YOU COMPLETED PSYCHIATRIC RESIDENCY TRAINING (last 2 digits)?
Enter 99 if you did not complete a psychiatric residency. ☐☐ 17–18

8. PLEASE ENTER THE LAST TWO DIGITS OF THE YEAR IN WHICH YOU WERE CERTIFIED IN ANY OF THE FOLLOWING (Enter 99 if not certified in any given category):

ABPN in general psychiatry	☐☐	19–20
ABPN in child psychiatry	☐☐	21–22
RCPS in general psychiatry	☐☐	23–24
ABPN or RCPS in neurology	☐☐	25–26
Boarded in a medical specialty other than psychiatry or neurology	☐☐	27–28

9. HOW WOULD YOU CHARACTERIZE YOUR PREDOMINANT THEORETICAL ORIENTATION TO PSYCHOPATHOLOGY? Please code only one.

1-Organic, biochemical
2-Organic, neurological
3-Psychological, psychoanalytic
4-Psychological, other than psychoanalytic
5-Social/Community
6-Behaviorist
7-Eclectic
8-Other; please specify: ☐ 29

USING THE FOLLOWING RATING SCALE, PLEASE INDICATE YOUR DEGREE OF AGREEMENT OR DISAGREEMENT WITH EACH OF THE STATEMENTS BELOW:

1-Strongly agree
2-Agree
3-No opinion; ambivalent; undecided
4-Disagree
5-Strongly disagree

10. ECT SHOULD BE USED ONLY WHEN ALL ELSE HAS FAILED. ☐ 30

11. THERE IS A NEED FOR MORE EXPLICIT GUIDELINES (perhaps from the APA) FOR THE PROPER USE OF ECT. ☐ 31

12. THE INTRODUCTION OF ANTIDEPRESSANTS AND PHENOTHIAZINES HAS MADE THE USE OF ECT OBSOLETE. ☐ 32

13. THERE ARE MANY PATIENTS FOR WHOM ECT, EITHER ALONE OR IN COMBINATION WITH OTHER MEASURES, IS STILL THE SAFEST, LEAST EXPENSIVE, AND MOST EFFECTIVE FORM OF TREATMENT. ☐ 33

14. IT IS LIKELY THAT ECT PRODUCES SLIGHT OR SUBTLE BRAIN DAMAGE. ☐ 34

OVER, PLEASE

15. THE ISSUANCE OF GUIDELINES FROM ANY SOURCE FOR THE USE OF ECT IS LIKELY TO INTERFERE WITH GOOD PATIENT CARE. ☐ 35

16. THE USE OF ECT SHOULD BE DISCONTINUED OR AT LEAST SHOULD BE CURTAILED. ☐ 36

17. ANY PSYCHIATRIC INSTITUTION CLAIMING TO OFFER COMPREHENSIVE CARE SHOULD BE EQUIPPED TO PROVIDE ECT. ☐ 37

18. ECT SHOULD NOT BE ADMINISTERED TO CHILDREN 16 OR UNDER. ☐ 38

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19. GRANTING THAT THE QUESTION IS A GROSS OVERSIMPLIFICATION, WHICH OF THE FOLLOWING *BEST* CHARACTERIZES YOUR ATTITUDE TOWARD THE USE OF ECT.

1-Totally opposed to its use
2-Generally opposed, but O.K. as a last resort in a few selected instances
3-No really strong feeling, but tend to be more opposed than favorable
4-Ambivalent; undecided
5-No really strong feeling, but tend to be more favorable than opposed
6-Generally favorable for appropriate patients
7-Decidedly favorable to its use ☐ 39

FOR EACH OF THE DIAGNOSES OR BEHAVIORS LISTED BELOW, USE THE FOLLOWING RATING SCALE TO INDICATE YOUR OPINIONS OF THE APPROPRIATENESS OF ECT AT SOME TIME DURING THE TREATMENT PROGRAM: ASSUME THAT THERE ARE NO PHYSICAL CONTRAINDICATIONS:

1-Totally appropriate
2-Probably appropriate
3-Ambivalent; undecided
4-Probably not appropriate
5-Totally inappropriate for this condition
6-I am opposed to the use of ECT for all patients

20. Minor (non-psychotic) depressive illness ☐ 40

21. Major depressive illness (defined as including at least 2 of the following 6 ICDA criteria: dysphoric mood, sleep disturbance, anorexia and weight loss, agitation and delusions, self-deprecatory statements or serious suicidal potential, retardation of motor/thought processes) ☐ 41

22. Schizophrenia (acute or chronic) ☐ 42

23. Manic excitement ☐ 43

24. Drug or alcohol abuse ☐ 44

25. Personality disorders ☐ 45

26. Sexual dysfunction ☐ 46

27. Anorexia nervosa ☐ 47

28. Intractable pain ☐ 48

29. Unremitting hypochondriasis ☐ 49

30. Toxic dementias ☐ 50

31. Other (please specify) _____ ☐ 51

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32. DO YOU SPEND AT LEAST 50% OF A USUAL WORKING WEEK (35 or more hours per week) IN ACTIVITIES RELATED TO PSYCHIATRY AND/OR NEUROLOGY (clinical practice, teaching, administration, research)? 1-No 2-Yes ☐ 52

IF YOU ANSWERED NO, PLEASE STOP HERE AND RETURN THE QUESTIONNAIRE. THANK YOU VERY MUCH.

IF YOU ANSWERED YES, PLEASE CONTINUE WITH THE QUESTIONS BELOW.

OVER, PLEASE

33. WHICH OF THE FOLLOWING *BEST* DESCRIBES YOUR PRIMARY WORK?

1-Clinical practice: private office practice
2-Clinical practice: mental hospital, public or private
3-Clinical practice: other institutional setting (HMO, clinic or mental health center, college, industry, etc.)
4-Teaching/training (of others)
5-Research/program evaluation
6-Consultation
7-Administration
8-Writing/editing
9-Other; please specify: _____

☐ 53

34. SITE OF CURRENT FULL-TIME OR PRINCIPAL PART-TIME EMPLOYMENT?
Please code only one.

01-Solo private office practice
02-Group private office practice (incl. multispecialty groups)
03-Military (U.S. or Canadian)
04-U.S. or Canadian government, non-military (incl. V.A.)
05-Public, non-federal psychiatric hospital (i.e., state/provincial/local)
06-Private psychiatric hospital
07-Medical school (incl. parent university and associated facilities)
08-Prison or correctional facility
09-Retardation facility
10-Publicly sponsored clinic (alcohol, drug, mental health)
11-Other private, for profit, non-hospital organization/institution (incl. Industry)
12-Other private, non-profit, non-hospital organization/institution
13-Other; please specify: _____

☐☐ 54–55

35. WHICH OF THE FOLLOWING *BEST* CHARACTERIZES THE LOCATION OF YOUR PRACTICE (administration, teaching and research are considered practice)?

1-Very large urban area (500,000 population and above)
2-Large urban area (250,000 to 500,000 population)
3-Medium size urban area (100,000 to 250,000 population)
4-Small urban area (25,000 to 100,000)
5-Small city (10,000 to 25,000 population)
6-Rural (less than 10,000 population) ☐ 56

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36. IN THE LAST 6 MONTHS HOW MANY SEPARATE PATIENTS (APPROXIMATELY) DID YOU SEE FOR ANY PURPOSE: DIAGNOSIS, CONSULTATION, THERAPY, MEDICATION REVIEW, ETC. (but not for research)? Enter 000 if none. ☐☐☐ 57–59

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37. IN THE LAST 6 MONTHS, HOW MANY PATIENTS HAVE YOU PERSONALLY TREATED WITH ECT? Enter 000 if none. ☐☐☐ 60–62

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38. IN THE LAST 6 MONTHS, FOR HOW MANY PATIENTS HAVE YOU RECOMMENDED TO RESIDENTS WHOM YOU SUPERVISED THAT ECT SHOULD BE USED (Enter 000 if none)? ☐☐☐ 63–65

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39. OF THE TOTAL NUMBER OF PATIENTS REFERRED TO IN ITEMS 37 AND 38, HOW MANY WERE TREATED AS OUTPATIENTS (Enter 000 if none)? ☐☐☐ 66–68

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40. IN THE LAST 6 MONTHS, HOW MANY PATIENTS HAVE YOU REFERRED TO OTHER PSYCHIATRISTS FOR ECT (Enter 000 if none)? ☐☐☐ 69–71

OVER, PLEASE

41. HAVE YOU EVER BEEN INVOLVED IN ANY LITIGATION OVER THE USE OF ECT?
1-No 2-Yes
IF YES, WHAT YEAR (last 2 digits); IF NO, ENTER 99. ☐☐ 72 73–74

IF YOU HAVE ENTERED 000 IN ITEMS 37 AND 38 (i.e., YOU HAVE NOT USED ECT OR SUPERVISED RESIDENTS IN ITS USE IN THE LAST 6 MONTHS) STOP HERE AND RETURN THIS QUESTIONNAIRE. THANK YOU VERY MUCH FOR YOUR HELP.

IF THE ANSWERS TO ITEMS 37 OR 38 ARE OTHER THAN 000, PLEASE ANSWER THE REMAINING QUESTIONS IN SECTION II.

ID + card # Blank ☐☐☐☐1 75 76–80

SECTION II: FOR PSYCHIATRISTS WHO "USE" ECT

FOR THIS SECTION, "TO USE OR ADMINISTER ECT" MEANS
a. to administer ECT yourself
b. to recommend to residents whom you supervise that they administer ECT

A. TECHNIQUE OF ADMINISTRATION

42. DO YOU GENERALLY USE A SHORT-ACTING ANESTHETIC DRUG BEFORE ADMINISTERING ECT?
1-No 2-Yes ☐ 1

43. IF YES (to item 42) HAVE YOU HAD FORMAL TRAINING IN ADMINISTERING THE ANESTHESIA REQUIRED IN ECT WHERE MUSCLE PARALYSANTS ARE USED?
1-No 2-Yes 8-Not applicable, Item 42 answered *No* ☐ 2

44. DO YOU GENERALLY USE THE SERVICES OF AN ANESTHESIOLOGIST OR NURSE ANESTHETIST?
1-Never
2-Sometimes
3-Always
8-Not applicable, Item 42 answered *No* ☐ 3

45. OF THE TOTAL NUMBER OF PATIENTS YOU TREATED WITH ECT IN THE LAST 6 MONTHS (FROM ITEMS 37-38), WHAT PERCENTAGE RECEIVED A MUSCLE-RELAXANT DRUG BEFORE ECT WAS ADMINISTERED?
(percent, 000 to 100) ☐☐☐ 4–6

46. WHAT TYPE OF MACHINE DO YOU GENERALLY USE?

1-Medcraft
2-Reiter
3-Mecta
4-Other; specify _____ ☐ 7

OVER, PLEASE

47. WHAT TYPE OF CURRENT DO YOU GENERALLY ADMINISTER?
1-Bipolar sine wave
2-Unidirectional
3-Pulsed unidirectional
4-Pulsed bipolar
5-Other; specify _____ ☐ 8

OF THE TOTAL NUMBER OF PATIENTS YOU HAVE TREATED WITH ECT IN THE LAST 6 MONTHS (FROM ITEMS 37-38 ABOVE), WHAT PERCENT RECEIVED

48. Bilateral ECT? ☐☐☐ 9–11

49. Left unilateral ECT? ☐☐☐ 12–14

50. Right unilateral ECT? ☐☐☐ 15–17

51. Combination of bilateral and unilateral ECT? ☐☐☐ 18–20

(Percentages for items 48-51 should sum to 100)

52. DO YOU ADMINISTER MORE THAN 1 SEIZURE DURING 1 TREATMENT SESSION?
1-No
2-Yes, occasionally (25% or less of treatment sessions)
3-Yes, often (25% to 75% of treatment sessions)
4-Yes, almost always (more than 75% of treatment sessions) ☐ 21

53. IF YES TO PRECEDING, WHAT IS THE AVERAGE NUMBER?
(Enter 9 if item 52 was answered *No*) ☐ 22

54. GRANTING THAT INDIVIDUAL PATIENTS DIFFER, WHAT IS YOUR MOST COMMON SCHEDULE FOR ADMINISTERING ECT?

1-Twice a month
2-Once a week
3-Twice a week
4-Three times a week
5-Four to five times a week
6-No usual schedule, PRN
7-Other; specify _____ ☐ 23

GRANTING AGAIN THAT PATIENTS VARY, ON THE AVERAGE WHAT NUMBER OF ECT TREATMENTS DO YOU CONSIDER OPTIMAL AS A COURSE OF TREATMENT FOR

55. Schizophrenic patients ☐☐ 24–25

56. Patients with major depressive illness ☐☐ 26–27

57. Patients with minor (non-psychotic) depressive illness ☐☐ 28–29

58. IN WHAT PERCENTAGE OF YOUR ECT PATIENTS TREATED IN THE PAST 6 MONTHS HAVE YOU USED MAINTENANCE ECT?

(percent, 000 to 100) ☐☐☐ 30–32

59. IF ITEM 58 WAS ANSWERED OTHER THAN 000, APPROXIMATELY HOW OFTEN IS ECT ADMINISTERED ON A MAINTENANCE SCHEDULE?

OVER, PLEASE

1-Once a week
2-Once a month
3-Less often
4-No fixed schedule; PRN
8-Item 58 answered 000 ☐ 33

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60. OPINIONS VARY ABOUT THE COMBINED USE OF ECT AND PSYCHOACTIVE DRUGS (antipsychotic agents, tricyclic antidepressant drugs). WHAT IS YOUR PERSONAL OPINION IN THIS MATTER? IN ANSWERING, ASSUME THAT THE PATIENT HAS BEEN ON PSYCHOACTIVE MEDICATION WHEN THE DECISION IS MADE TO START ECT.

1-Psychoactive medication should almost always be stopped.
2-Psychoactive medication should usually be stopped.
3-It probably makes little difference whether or not medication is stopped.
4-Psychoactive medication should probably be continued.
5-Psychoactive medication should almost always be continued. ☐ 34

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61. REGARDLESS OF YOUR ANSWER TO ITEM 60, SHOULD PSYCHOACTIVE MEDICATION BE ADMINISTERED FOR AT LEAST A FEW MONTHS AFTER THE COMPLETION OF ECT? ☐ 35

1-No 2-Yes

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62. THE MAJORITY OF ECT TREATMENTS ADMINISTERED BY YOU ARE IN

1-a private office not in an institutional setting
2-an institutional setting accredited by the Joint Commission on Accreditation of Hospitals
3-an institutional setting not accredited by the Joint Commission on Accreditation of Hospitals ☐ 36

63. APPROXIMATELY WHAT PERCENTAGE OF YOUR ECT TREATMENTS ARE PAID FOR BY INSURANCE COVERAGE?

(percent, 000 to 100) ☐☐☐ 37–39

B. PATIENT SELECTION

IN APPROXIMATELY WHAT PERCENTAGE OF THE *SCHIZOPHRENIC* PATIENTS YOU TREATED WITH ECT IN THE PAST 6 MONTHS (as indicated in items 37-38) WAS ECT SELECTED FOR EACH OF THE FOLLOWING REASONS (skip to next series if you did not use ECT with schizophrenics in the last 6 months):

64. It was the treatment of first choice (given either with or without psychoactive drugs). ☐☐☐ 40–42

65. ECT was used in combination with other treatment methods with the expectation that its addition would yield a better quality of improvement. ☐☐☐ 43–45

66. Medication in large daily dosages (at least 1000 mg. of chlorpromazine or its equivalent) was tried and found ineffective. ☐☐☐ 46–48

67. Medication in smaller dosages was tried and found ineffective. ☐☐☐ 49–51

68. Patient was allergic to medication and/or medication led to side effects. ☐☐☐ 52–54

69. Psychotherapy (individual and/or group and/or family) was ineffective. Consider as psychotherapy for purposes of this item a minimum of two 30 to 60 minute sessions per week for three months. ☐☐☐ 55–57

70. Large dosages of medication *and* psychotherapy were ineffective. ☐☐☐ 58-60

(Percentages for items 64-70 should sum to 100)

OVER, PLEASE

ID + card # [ ][ ][ ][ ][2] 76–80

IN APPROXIMATELY WHAT PERCENTAGE OF PATIENTS WITH *MAJOR DEPRESSIVE ILLNESS* THAT YOU TREATED WITH ECT IN THE PAST 6 MONTHS (as indicated in Items 37-38) WAS ECT SELECTED FOR EACH OF THE FOLLOWING REASONS (skip to next series if you did not use ECT in major depressive illnesses in last 6 months):

71. It was the treatment of first choice (given either with or without psychoactive drugs). [ ][ ][ ] 1–3

72. ECT was used in combination with other treatment methods with the expectation that its addition would yield a better quality of improvement. [ ][ ][ ] 4–6

73. Medication in large daily dosages (at least 300 mg. of imipramine or equivalent dosage of other tricyclic antidepressant) was tried for at least 3 to 4 weeks and found ineffective. [ ][ ][ ] 7–9

74. Medication in smaller dosages was tried and found ineffective. [ ][ ][ ] 10–12

75. Patient was allergic to medication and/or medication led to side effects. [ ][ ][ ] 13–15

76. Psychotherapy (individual and/or group and/or family) was ineffective. Consider as psychotherapy for purposes of this item a minimum of two 30 to 60 minute-sessions per week for three months. [ ][ ][ ] 16–18

77. Large dosages of medication *and* psychotherapy were ineffective. [ ][ ][ ] 19–21

78. Smaller dosages of medication *and* psychotherapy were ineffective? [ ][ ][ ] 22–24

(Percentages for Items 71-78 should sum to 100)

IN APPROXIMATELY WHAT PERCENTAGE OF PATIENTS WITH *MINOR (NON-PSYCHOTIC) DEPRESSIVE ILLNESS* THAT YOU TREATED WITH ECT IN THE PAST 6 MONTHS (as indicated in Items 37-38) WAS ECT SELECTED FOR THE FOLLOWING REASONS (skip to next item if you did not use ECT with minor depressive illnesses in last 6 months):

79. It was the treatment of first choice (given either with or without psychoactive drugs). ☐☐☐ 25–27

80. ECT was used in combination with other treatment methods with the expectation that its addition would yield a better quality of improvement. ☐☐☐ 28–30

81. Medication in large daily dosages (at least 300 mg. of imipramine or equivalent dosage of other tricyclic antidepressant) was tried for at least 3 to 4 weeks and found ineffective. ☐☐☐ 31–33

82. Medication in smaller dosages was tried and found ineffective. ☐☐☐ 34–36

83. Patient was allergic to medication and/or medication led to side effects. ☐☐☐ 37–39

84. Psychotherapy (individual and/or group and/or family) was ineffective. Consider as psychotherapy for purposes of this item a minimum of once weekly 30 to 60 minute-sessions for three months. ☐☐☐ 40–42

85. Large dosages of medication *and* psychotherapy were ineffective. ☐☐☐ 43–45

86. Smaller dosages of medication *and* psychotherapy were ineffective. ☐☐☐ 46–48

(Percentages for Items 79-86 should sum to 100.) ID + card # ☐☐☐☐3 76–80

OF THE PATIENTS YOU TREATED DURING THE PAST 6 MONTHS WITH ECT (or supervised, as per items 37-38), APPROXIMATELY WHAT PERCENTAGE FELL INTO EACH OF THE FOLLOWING CATEGORIES.

87. Minor (non-psychotic) depressive illness. ☐☐☐ 1–3

OVER, PLEASE

88. Major depressive illness to include at least 2 of the following:
    Dysphoric mood
    Sleep disturbance
    Anorexia and weight loss
    Retardation (motor and thought processes)
    Agitation, delusions
    Self-deprecatory thoughts and statements, serious suicidal potential ☐☐☐ 4-6

89. Schizophrenia (acute or chronic)? ☐☐☐ 7–9

90. Manic excitement? ☐☐☐ 10–12

91. Drug or alcohol abuse? ☐☐☐ 13–15

92. Personality disorders? ☐☐☐ 16–18

93. Sexual dysfunctions? ☐☐☐ 19–21

94. Anorexia nervosa? ☐☐☐ 22–24

95. Intractable pain? ☐☐☐ 25–27

96. Unremitting hypochondriasis? ☐☐☐ 28–30

97. Toxic dementias? ☐☐☐ 31–33

98. Others; specify? _____ ☐☐☐ 34–36

(Percentages for items 87-98 should sum to 100.)

DO YOU SEE ANY INCOMPATIBILITY IN ADMINISTERING ECT TO PATIENTS ON EACH OF THE FOLLOWING MEDICATIONS? RATE AS 1 FOR *NO* AND 2 FOR *YES*.

99. Lithium ☐ 37

100. Reserpine ☐ 38

101. Phenothiazines ☐ 39

102. Antidepressants ☐ 40

103. Thiazides ☐ 41

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104. DO YOU CONSIDER ECT TO BE CONTRAINDICATED FOR THE ELDERLY, DEPRESSED PATIENT WITH A CARDIOPULMONARY PROBLEM WHO IS RECEIVING ANTIHYPERTENSIVE MEDICATION?

1-Definitely not contraindicated
2-Probably not contraindicated
3-Probably contraindicated
4-Definitely contraindicated ☐ 42

C. *TREATMENT OUTCOME*

105. OF THE PATIENTS YOU HAVE TREATED WITH ECT IN THE LAST 6 MONTHS, APPROXIMATELY WHAT PERCENTAGE OF THEM WERE AT LEAST MODERATELY SATISFIED WITH THE RESULTS OF THEIR TREATMENT ONCE ECT HAD BEEN COMPLETED?

(Percent, 000 to 100) ☐☐☐ 43–45

OVER, PLEASE

106. FOR WHAT PERCENTAGE WERE *YOU* AT LEAST MODERATELY SATISFIED WITH THE RESULTS OF ECT TREATMENT? ☐☐☐ 46–48

IN WHAT PERCENTAGE OF *SCHIZOPHRENIC PATIENTS* WHOM YOU HAVE TREATED WITH ECT IN THE LAST 6 MONTHS HAVE YOU OBSERVED EACH OF THE FOLLOWING RESPONSES (skip to the next series if you did not use ECT with schizophrenics in the last 6 months):

107. *Excellent response, e.g., complete remission of symptoms?* ☐☐☐ 49–51

108. Good response; considerable improvement? ☐☐☐ 52-54

109. Fair response; moderate improvement? ☐☐☐ 55–57

110. Poor response; little if any improvement? ☐☐☐ 58–60

111. Very poor response; worsening of patient's condition? ☐☐☐ 61–63

(Percentages for items 107-111 should sum to 100)

ID + card # ☐☐☐☐ 4 76–80

IN WHAT PERCENTAGE OF PATIENTS WITH *MAJOR DEPRESSIVE ILLNESS* WHOM YOU HAVE TREATED WITH ECT IN THE LAST 6 MONTHS HAVE YOU OBSERVED EACH OF THE FOLLOWING RESPONSES (skip to the next series if you did not use ECT in major depressive illness in last 6 months):

112. Excellent response, e.g., complete remission of symptoms? ☐☐☐ 1–3

113. Good response; considerable improvement? ☐☐☐ 4–6

114. Fair response; moderate improvement? ☐☐☐ 7-9

115. Poor response; little if any improvement? ☐☐☐ 10–12

116. Very poor response; worsening of patient's condition? ☐☐☐ 13–15

(Percentages for items 112-116 should sum to 100)

IN WHAT PERCENTAGE OF PATIENTS WITH *MINOR (NON-PSYCHOTIC) DEPRESSIVE ILLNESS* WHOM YOU HAVE TREATED WITH ECT IN THE LAST 6 MONTHS HAVE YOU OBSERVED EACH OF THE FOLLOWING RESPONSES (skip to next item if you did not use ECT in minor depressive illness in last 6 months):

117. Excellent response, e.g., complete remission of symptoms? ☐☐☐ 16–18

118. Good response; considerable improvement? ☐☐☐ 19–21

119. Fair response; moderate improvement? ☐☐☐ 22–24

120. Poor response; little if any improvement? ☐☐☐ 25–27

121. Very poor response; worsening of patient's condition? ☐☐☐ 28–30

(Percentages for items 117-121 should sum to 100)

BY ABOUT 3 MONTHS AFTER A COURSE OF ECT, APPROXIMATELY WHAT PERCENTAGE OF PATIENTS COMPLAIN OF EACH OF THE FOLLOWING ADVERSE EFFECTS:

122. Permanent loss of memory for period of ECT course. ☐☐☐ 31–33

OVER, PLEASE

123. Temporary loss of memory for period of ECT course. ☐☐☐ 34–36

124. Permanent loss of memory for a period immediately prior to ECT, i.e., recent memory generally. ☐☐☐ 37–39

125. Temporary loss of memory for period immediately prior to ECT, i.e., recent memory generally. ☐☐☐ 40–42

126. Permanent loss of distant memories. ☐☐☐ 43–45

127. Temporary loss of distant memories. ☐☐☐ 46–48

128. Very specific, spotty memory loss. ☐☐☐ 49–51

129. Marked confusion. ☐☐☐ 52–54

130. Slight confusion. ☐☐☐ 55–57

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132. WHAT PERCENTAGE HAVE TOLD YOU OR OTHERS THAT THEY HAVE BEEN PERMANENTLY PREVENTED FROM PERFORMING SPECIFIC VOCATIONAL TASKS AFTER RECEIVING ECT?

(percent, 000 to 100) ☐☐☐ 61–63

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133. IN WHAT PERCENTAGE OF PATIENTS YOU HAVE TREATED DURING THE PAST 6 MONTHS DID SPONTANEOUS SEIZURES OCCUR SHORTLY AFTER ECT (THERE WERE NO SEIZURES PRIOR TO ECT)?

(percent, 000 to 100) ☐☐☐ 64–66

134. DURING THE LAST 5 YEARS, HOW MANY DEATHS HAVE OCCURRED AMONG YOUR PATIENTS DURING, OR WITHIN 24 HOURS OF, ECT? ☐ 67

IF YOU OBSERVED 1 OR MORE DEATHS, WAS ANY OF THE FOLLOWING CONSIDERED A CAUSE OF THESE DEATHS, ACCORDING TO THE DEATH CERTIFICATE:

135. Cardiac?
1-No
2-Yes
8-Inapplicable, no deaths observed
9-Don't know ☐ 68

136. Cerebral?
1-No
2-Yes
8-Inapplicable, no deaths observed
9-Don't know ☐ 69

137. Other?
1-No
2-Yes
8-Inapplicable, no deaths observed
9-Don't know ☐ 70

DURING THE LAST 5 YEARS, HOW MANY DEATHS HAVE OCCURRED AMONG YOUR PATIENTS WHO DID NOT RECEIVE ECT TREATMENT THAT YOU THINK MIGHT POSSIBLY HAVE BEEN PRECLUDED HAD ECT BEEN ADMINISTERED?

138. In acutely suicidal patients ☐ 71

139. In acutely homicidal patients ☐ 72

140. In acutely fulminating catatonia ☐ 73

141. In other patients ☐ 74

BLANK

ID + card # ☐☐☐☐5 76–80

THANK YOU AGAIN FOR HAVING COMPLETED THIS LONG QUESTIONNAIRE AND PARTICIPATING IN THIS IMPORTANT STUDY

PLEASE DO NOT REMOVE OR DEFACE THIS LABEL; IT WILL BE REMOVED ONCE YOUR QUESTIONNAIRE RETURN IS RECORDED. THERE WILL BE NO BREACH OF CONFIDENTIALITY.
