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

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

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

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

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

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

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

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

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

Stimulus efficiency

The ECT electrical stimulus has two separate attributes that influence seizure intensity: dose and waveform efficiency. Returning to the gasoline analogy, stimulus dose corresponds to gallons of gasoline, stimulus efficiency to miles per gallon, and seizure induction to a particular trip. With low efficiency, a higher dose (more gasoline) is needed to reach the goal. By definition, the characteristics of a waveform that is more clinically effective at the same stimulus dose are more efficient.

Before we consider these characteristics, there is a basic question about the virtues of stimulus efficiency. Compare two stimuli that are equally effective in producing ECT seizures but the efficiencies of which differ. For equal effectiveness, the less efficient stimulus is administered at a higher dose. The question is: Does the less efficient higher dose stimulus have more side effects? A related question is: For the higher dose stimulus, does the extra electricity cause adverse effects? The lower efficiency stimulus is analogous to driving with the parking brake on. In view of this analogy (and because of the higher dose), some of the less efficient stimuli presumably interfere with seizure generation, so we should expect that less efficient stimuli have more side effects, and the most efficient stimuli are the most clinically desirable. This expectation is clearly true in the comparison between brief-pulse and sine wave stimuli, and it is presumably true in general.

Basic waveform characteristics include wave shape (e.g., rectangular or sine), continuous or pulsed train, phase width, wave frequency, and charge rate. The phase width is the width of the stimulus wave. For rectangular waves, it is pulse width. For sine waves it is half the wavelength. Mathematically, wavelength equals the number 1 divided by wave frequency, and charge rate is the average absolute value of the current. Charge rate is the amount of stimulus charge delivered per second counting intervals when the current is off. In contrast, stimulus current refers only to periods when the current is on. Specifying any two of charge rate, pulse width, and frequency at a specific current determines the third, by the following mathematical relationship: *charge rate* (mC/s) $= 2 \times$ *frequency* (Hz) $\times$ *pulse width* (ms) $\times$ *current* (A).

Sine wave versus brief-pulse stimuli

The report that brief-pulse stimuli have milder side effects and use less charge than sine wave stimuli do is well known (Weiner et al., 1986). However, the result was never proven as just stated. Although the comparison found milder side effects and less charge with brief-pulse stimuli, there were large electrical differences between stimuli outside of wave shape. There are several reasons why the clinical differences

Sickness can include delusions of being full of waste, poisoned, having organ dysfunction (especially stomach or intestines), or parasitosis. Sensory distortions or hallucinations can accompany these delusions. Middle-aged or elderly patients with psychotic melancholia often have constipation or other gastrointestinal complaints (Parker et al., 1991).

Psychotic melancholia is one of several common types of psychotic depression, most of which are treatable with ECT, but for some types ECT is not suitable (Swartz and Shorter, 2007). Some other types of psychotic depression are bipolar mixed depression, deteriorative psychotic depression, catatonic psychotic depression, psychotic-equivalent demented depression, tardive psychotic depression, drug-induced psychotic depression, and coarse brain disease psychotic depression. ECT should help patients with these types of depression but should be only a last resort in tardive and coarse brain disease psychotic depressions.

Several reports from Columbia University claim that medication-resistant major depression responds poorly to ECT (Prudic et al., 1990, 1996). Indeed, the outcome of their studies was markedly worse than expected. Response – which is a substantially lower outcome than remission – was obtained in only 35% to 65% of their patients with an average ECT course of 10 treatments, a long course. Their claim of medication resistance predisposing to poor ECT outcome contrasts markedly with other studies – some quite large – reporting that medication-resistant patients respond very well to ECT (e.g., Pluijms et al., 2002). In the DeCarolis study (Avery and Lubrano, 1979) 85% of 110 patients who failed to respond to one month of imipramine achieved remission with 8 to 10 ECT treatments. The "endogenous depression" that these patients had presumably corresponds to melancholia or simultaneous melancholia and catatonia. The inability of an acute course of ECT to treat comorbid anxiety disorders such as PTSD may explain the poor results at Columbia.

The Columbia study patients had long suffered with psychiatric conditions with averages of 15 to 20 years since illness onset. Still, the reports do not state that anxiety disorders were accounted for, disqualified, or even examined for. The simple explanation for the bad outcome is that the Columbia patients had comorbid dysphoric anxiety disorders that increased depression rating scores. Hints that this was so include the regular use of lorazepam in 84% (67/80) of their ECT study patients and the fact that higher depression scores at the end of the ECT course were strongly associated with more frequent relapse (Sackeim et al., 2000). Anxiety disorders are present in at least half of patients with DSM major depression (Zimmerman et al., 2002), and long-suffering patients should have a still higher incidence. Indeed, having concurrent anxiety symptoms predicts worse long-term outcome to antidepressant medication (Andreescu et al., 2007), and anxiety is so common in this group that the converse is surely true, that worse long-term

degree of actual memory side effects and they have possibly moderated the patients' subjective perception of memory problems. Coleman et al. (1996) used the Squire Subjective Memory Questionnaire (SSMQ; Squire et al., 1979) in depressed patients treated with either bitemporal or right unilateral placement at either low (threshold) or high (2.5 times threshold) dose. They found high correlations between SSMQ scores and depression levels at baseline. That is, more severely depressed patients rated their memory worse than did more moderately depressed patients before ECT was started. Immediately following the course of ECT, patients' assessments of memory improved dramatically, more so in responders than in nonresponders. Patients' SSMQ scores continued to improve through two months of follow-up, finally approaching the scores of the healthy control group. There was no association between SSMQ scores and objective memory test scores in the immediate post-ECT period. Still, after two months, objective testing for retrograde memory concerning autobiographical information was associated with self-rated greater memory impairment. When clinical improvement was statistically controlled there was evidence that high stimulus dose and bitemporal placement resulted in less improvement in self-ratings. The authors interpret differences between the results of their study and those of earlier researchers as attributable to "fundamental changes in ECT practice" involving more efficient electrical waveforms and electrode placement.

In a review of subjective memory complaints in ECT, Prudic et al. (2000) reported that, with modern treatment parameters, subjective memory improves with ECT. They found no clear evidence of an association between subjective and objectively measured memory but substantial evidence of a negative association between subjective memory complaints and mood state. They do concede that there may be aspects of memory impairment not measured by standard psychometric instruments. They note a lack of information about the number and characteristics of the patients who complain of severe memory impairment after ECT.

Retrograde amnesia

Retrograde amnesia refers to an inability to access memories already stored before the course of ECT begins. It does not mean an impairment in the ability to learn. Its presence can be a source of distress in some patients. Typically the loss is not permanent, and the period of amnesia shrinks with earlier memories returning first and those stored immediately before ECT recovering last, if at all. The issue of retrograde amnesia has already been touched on under the section heading of postictal disorientation and confusion.

In a major study of the effects of ECT on retrograde amnesia, Lisanby et al. (2000) examined differences between personal and public information in relation to electrode placement (bitemporal or right unilateral) and stimulus dose (barely