Book Reviews

A Somatic Therapy in Psychiatry

Psychobiology of Convulsive Therapy. Papers from a conference, San Juan, Puerto Rico, Apr. 1972. MAX FINK, SEYMOUR KETY, JAMES MCGAUGH, and THOMAS A. WILLIAMS, Eds. Winston, Washington, D.C., 1974 (distributor, Halsted [Wiley], New York). xii, 312 pp., illus. $11.95.

Convulsive therapy is one of the oldest somatic therapies still used in clinical psychiatry. Electroconvulsive therapy (ECT) is one of the most effective treatments known for severe depressive illness. It is also acknowledged to be effective treatment for acute mania and some acute variants of schizophrenia, particularly catatonia. Though most psychiatric clinicians are capable of providing safe and effective ECT, there has been little understanding of the mechanisms involved. This book, a comprehensive review of the current investigational and theoretical aspects of convulsive therapy, goes far toward providing a rational basis for its use.

The clinical application of convulsive therapy in reasonably standardized form is well accepted in practice and in the literature. The indications for its use mentioned above are widely followed. In most centers, patients are treated approximately 6 to 12 times at 48-hour intervals with electrical stimuli delivered through bilaterally placed electrodes. When ECT is used in conjunction with rapidly acting barbiturate anesthesia, muscle relaxant, and atropine administered by an anesthesiologist, major complications are rare. There are very few contraindications to treatment, mortality risk is probably on the order of 0.1 percent, and efficacy is well documented.

Amnesia, the most commonly studied concomitant of treatment, is addressed in the book both as an undesired side effect of therapy and as an experimental key to the understanding of mechanisms of memory. Clinically, memory impairment correlates with intensity of stimulus (or concentration of chemical or inhalant convulsant) but not with the cerebral seizure per se, which is the primary correlate to therapeutic efficacy. In an effort to minimize the side effect, unilateral electrical stimulation of the nondominant hemisphere has been used to induce seizures. D'Elia's paper reviews this work and documents that anterograde and retrograde amnesia and posttreatment confusion are less with unilateral treatment. A paper by I. Small discusses the use of the inhalant convulsant flurothyl, another alternative means of induction, which also seems to produce less amnestic effect than bilateral ECT. Cherkin capitalizes on the high lipid solubility of flurothyl to study and document factors in amnesia that are independent of specific neuroanatomic pathways of electrical conduction.

McGaugh's and Zornetzer's discussions of amnesia in infrahuman species suggest that electroconvulsive shock (ECS) interferes with consolidation or storage of memory. Zornetzer provides descriptive neurophysiologic data on electroencephalographic correlates of memory disruption in rats. These data indicate that brain seizures are neither necessary nor sufficient for the production of amnesia, reinforcing the belief that memory disturbance is a side effect rather than a primary therapeutic factor.

Dunn *et al.* discuss amnesia in relation to impaired protein synthesis at synaptic sites. Most important clinically is their suggestion that ECS may produce small amounts of protein degradation and membrane damage, one of the rare indications that minute brain damage may result from treatment. Essman, in studies of ECS effects on mouse forebrain, shows that decreased synaptic RNA is associated with an increase in turnover time and in concentration of 5-hydroxytryptamine (5-HT). Interestingly, exogenous 5-HT not only seems to reduce the rate of amino acid incorporation into proteins at nerve endings, but also, when administered intracerebrally in mice, can produce a retrograde amnesia similar to that produced by ECS.

Possible mechanisms of the therapeutic action of ECT are clarified in this book. For example, Kety's summary of the effects of ECS on biogenic amines indicates that its mode of action is similar to that of antidepressant drugs. A decrease in norepinephrine and an increase in its metabolites after an acute ECS have been reported. There is further evidence of an increase in norepinephrine after a series of ECS, suggesting that there is a cumulative overall increase in norepinephrine synthesis and turnover during a course of treatment.

Some of these changes in the metabolism of indoleamines and catecholamines persist for some time after the last induced seizure. Kety suggests, therefore, that repeated ECS may induce a metabolic change (increased amine turnover) that contributes to the clinical efficacy of the therapy. This conclusion is indeed tentative, but it represents a conceptual leap from the old attempts at psychologic explanation of ECT to the level of psychobiologic integration of pathophysiology, neurophysiology, and biochemistry.

This book will be read with interest by anyone involved in the study of memory function and it should be read by all clinicians who administer convulsive therapy. It is regrettable that two years must pass between a symposium and its publication in book form. We must hope that the authors will soon present an updated account of the evolution of their ideas.

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Neurotransmitter

Serotonin—New Vistas. E. COSTA, G. L. GESSA, and MERTON SANDLER, Eds. In two volumes. Histochemistry and Pharmacology. xvi, 330 pp., illus. $19.75. Biochemistry and Behavioral and Clinical Studies. xviii, 428 pp., illus. $19.75. Raven, New York, 1974. Advances in Biochemical Psychopharmacology, vols. 10 and 11.

One of the rewards attending a novel scientific discovery is the opportunity to watch the progress of the research it stimulates. It has been just 25 years since pure serotonin, 5-hy-