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Letters to the Editor

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To the Editor:

For some years, clinicians working with borderline personality disorder (BPD) patients have noticed that these patients appear to have problems with their capacity to concentrate, remember, integrate disparate percepts, and learn. Until very recently, the presence in these patients of organically-based cognitive impairment—as distinct from, but not necessarily etiologically different from, psychostructural "deficits"—has been hypothesized (1) but not definitively demonstrated by objective measures.

The recent study by Burgess in this journal (2) is the third published study of BPD patients that uses psychological testing measures to attempt to identify cognitive dysfunction in these often very difficult, "treatment-resistant" patients. Burgess' purported finding of subtle organic impairment is consistent with the one other study with positive findings (3), but is inconsistent with the one previous negative study (4). However, certain aspects of Burgess' study suggest that its findings must be viewed with caution.

First, previous surveys of BPD patients' demographics indicate a preponderance of females to males in ratios between 2:1 and 4:1. Since Burgess' sample has twice as many males as females, it is likely to be unrepresentative of the larger BPD population. In addition, Andrulonis has shown using neurodevelopmental and other markers of organicity that approximately two-and-one-half times as many male BPD patients as females probably have organic impairment (1). Thus, Burgess' results may be skewed toward organicity with respect to the total BPD population.

Second, the use of a 10-minute screening battery with only one item assessing major cognitive features is highly susceptible to a Type I (false positive) statistical error which would lead to a false impression of organicity due to confounding state variables such as poor motivation, anxiety, uncooperativeness, and so forth.

Third, Burgess relied solely on clinically-applied DSM-III-R criteria to diagnose BPD, rather than the much more preferable use of a semi-structured interview or, better yet, a combination of these and other concordant measures, such as self-report inventories, collateral interviews, and psychological tests. Thus, whether Burgess' data ultimately are concerned with BPD patients is not at all certain.

Despite these criticisms, Burgess' results appear consistent not only with the O'Leary study, but also with our own research (unpublished data), though all sample sizes are small, and diagnostic and methodologic congruence between these studies is mostly lacking. These findings of organicity may be quite nonspecific, however, or may reflect general difficulties that personality disorder patients have either with test-taking, with undefined state variables, or, perhaps, with global cognitive function. Until they are replicated and extended by other larger studies, these results remain tantalizingly suggestive but extremely tentative.

Would that a simple, bedside screening battery might eventually assist clinicians in identifying BPD patients with subtle organic impairment so that treatment planning might take this into account and patients' ultimate outcomes might be improved! Even if these findings are confirmed, however, clinicians may have to wait for revised sets of testing norms that include more precise measures of subtle impairment if they are routinely to be able to
detect these patients’ patterns of impairment without having to use clinical control groups for comparison.

Gregg E. Gorton, M.D.
Steven Samuel, Ph.D
Thomas Swirsky-Sacchetti, Ph.D
Richard Sobel, M.D.

REFERENCES


To the Editor:

Dr. Wright is to be commended on his succinct history of the introduction of electroconvulsive therapy. He describes the contributions of Wagner-Jauregg, Sakel, Meduna, and Cerletti in an exciting period in modern psychiatry. He errs, however, in claiming that the origin of electroconvulsive therapy is related to Roman interest in electric eels and to Eighteenth Century interest in the novelty of ‘electricity’.

The development of convulsive therapy is documented in Meduna’s first reports, in a posthumous autobiography published in 1985, and in Cerletti’s report of the origins of ECT (1). Wagner-Jauregg’s treatment of neurosyphilis by malaria encouraged scientists to consider biological antagonisms as a model for therapy, and it was this concept that led Meduna on his scientific trail. Reports that epileptic patients rarely showed the delusions or hallucinations of dementia praecox; and that patients with dementia praecox either did not have epilepsy, or if they developed seizures, as after a head injury, would have a remission of their psychosis, led Meduna to conclude that there was an antagonism between epilepsy and dementia praecox. He first induced seizures with intramuscular camphor, later with intravenous pentyleenetetrazol (Metrazol). His first report was published in 1935, and by 1937, his success was heralded throughout Europe. Electricity played no role in these developments (2).

Cerletti’s doctoral thesis was a description of the neuropathology of experimental seizures induced by electricity. After observing the clinical success and difficulties of seizure inductions with Metrazol, he suggested that electricity be used. I see no evidence in his writings, nor in the history of the use of electricity in medicine in Europe early in this century, to justify the conclusion that convulsive therapy was derived from Roman or Eighteenth Century dalliance with electricity.

Dr. Wright notes that many consider ECT a ‘controversial’ treatment. But the contro-
versy is not about its efficacy (which is very well documented), nor about its safety (which is undisputed when done properly), but in its place in psychiatric practice (3). Dr. Wright suggests that the anti-psychiatry movement is the basis for the controversy.

The anti-psychiatry movement has been relatively ineffective in restricting the use of ECT. There are few challenges in malpractice suits, but most are abandoned or settled with small awards. The legislative interdiction of ECT (as in California in 1974 and 1982) immediately fell to court challenges, and never was actively operational. The hostile media presentations are infrequent and while they affect the willingness of patients and their families to undergo a recommended course of ECT, the effects are generally transient.

The principal reason for the decline in the use of ECT is its poor image among psychiatrists. While some will argue that this decline results from the antipsychiatry movement, I believe that it stems mainly from psychiatrists’ lack of training in ECT and their reluctance to ‘lay on one’s hands’ in a medical intervention (3). For more than 20 years, medical schools and psychiatric residency training programs provided little more than a historical introduction to ECT with minimal to no clinical experience for their medical students and psychiatric residents. A whole generation of psychiatrists are unaware of the advantages of ECT, are unskilled in its use, and are ambivalent about the treatment.

But there is an influence of the anti-psychiatry movement on our practice. Because society is most concerned about the direct effects of ECT on brain functions, the research supported by NIMH has been to assess the effects of currents and electrode placement on memory, rather than on clinical issues or hypotheses of the mode of action. It has led to our greater dependence on anesthesiologists in ECT, despite the lack of complaints that the anesthesia induced by psychiatrists is of greater risk than that induced by anesthesiologists. One of the main effects of the 1978 APA Task Force report was its encouragement of the use of anesthesiologists in ECT, a mission that neither psychiatrists nor anesthesiologists requested, and that was motivated by a desire to minimize a potential criticism of ECT practice.

There is a maldistribution of ECT, which is mainly available in major academic medical centers but generally unavailable in Federal, state, and municipal mental hospitals. The large mental hospital systems are under direct political control and public scrutiny, and vociferous complaints by lobbyists are important considerations to political leaders who are not protected by the independence of the academic or private medical practice systems.

There are, however, signs of a resurgence of professional interest in ECT. The 1990 report of the American Psychiatric Association, and the 1989 report of the Royal College of Psychiatrists, are important contributions to standards of professional care. Publication of the quarterly scientific journal *Convulsive Therapy* since 1985 provides a venue for clinical and research issues. Annual meetings of the Association for Convulsive Therapy provides opportunities for scientific and educational discussions. The balance seems to be shifting to a greater acceptance of ECT by psychiatrists, and if sustained, should do much to change the ‘controversial’ image of ECT.

Dr. Wright seems to represent a new generation of psychiatrists better trained in ECT, who may look upon the treatment for its contribution to the welfare of his patients, and not as a political ‘hot potato’ to be dropped as soon as possible. I hope he continues his interest in ECT and trust that when he writes a review of the changes in ECT practice in the 1990s, he will be able to document that ECT is available in all the major mental hospitals of the nation and taught in almost all our medical schools and post-graduate residency programs with the
enthusiasm and energy that are now applied to the teaching of psychopharmacology and psychotherapy.

Max Fink, M.D.
Professor of Psychiatry
State University of NY at Stony Brook
P.O. Box 457
St. James, New York 11780

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