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## COMMENTARY

# Electroconvulsive Treatment, Neurology, and Psychiatry

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In professional and popular publications, advocates for the use of electroconvulsive treatment (ECT) take great pains to point out that ECT "has come a long way from its first unmodified use in 1938 . . ." (Kusumakar, 2000, p. 4). Indeed, anesthetics, muscle relaxants and oxygenation were introduced to the procedure by the early 1960s, in order to reduce the high risk of immediate and obvious physical adverse consequences. Nonetheless, ECT's basic and essential characteristic—what distinguishes it from any other intervention in medicine or psychiatry and what is stated in its name—has not changed since ECT was first introduced. This is, simply stated, the production of a generalized seizure (grand mal) by passing an electrical current through the brain.

In neurology, the adverse effects of seizures resulting from various forms of epilepsy are well known. Many epileptic seizures, it must be noted, are not as diffuse, prolonged, or extreme as the generalized seizures characteristic of ECT (which require muscle relaxants to prevent physical convulsions and bone fractures). In contrast to many forms of partial seizures, which involve limited areas of the brain, "generalized seizures involve both cerebral hemispheres simultaneously and produce convulsions (tonic, clonic, tonic-clonic, or atonic) or periods of absence" (Weilburg & Murray, 1991, p. 407).

How do neurologists describe, in neurological journals, the effects of various seizures on brain cells? Kendall and colleagues (1999) state that "temporal lobe gliosis and neuronal loss are pathological hallmarks of complex partial seizures" (p. 95). Tasch and colleagues (1999) write that, just like an early, fixed brain injury, "generalized seizures may also cause progressive neuronal dysfunction and loss" (p. 568). Sloviter (1999) states that "an episode of status epilepticus [any continuing type of seizure] at any age" can produce the "highly characteristic pattern of hippocampal cell loss and shrinkage" (p. s34). Mello and Covolan (1996) conclude that in rodents, whose brains are much more resistant to damage than human brains, "even single generalized spontaneous tonic-clonic seizures can induce long-lasting morphological changes" (p. 123). In rats who underwent spontaneous seizures following an episode of electrically-induced status epilepticus, Bertram (1997) demonstrated "neuronal loss" at 15 weeks in each of the four brain regions where seizures occurred.

In light of these findings from the disciplines of clinical and experimental neurology—which were located from a cursory literature search using merely three keywords—blanket assertions of the overall harmlessness of ECT found in numerous psychiatric articles take on a nearly surreal glow. For example, Kusumakar (2000) states that "there is little if any evidence of structural brain damage following ECT . . ." (p. 5). Do psychiatrists and neurologists inhabit separate scientific universes? To be sure, differences exist between electrically-induced and other types of seizures—but the evidence suggests that the former might be more damaging overall.

This leads us to point to the existence of a phenomenon that this writer has rarely seen addressed: neurologists' virtual silence about the topic of ECT. Given that neurologists are the officially recognized experts on the nervous system and on the effects of brain injuries, this silence ranks as a most remarkable omission. Every year in the United States, at least 100,000 persons receive series of electrically-induced seizures prescribed by one medical discipline, while another medical discipline—which recognizes seizures as one of the most significant traumas to the brain—does not comment on the practice. This silence is a significant topic in its own right and deserves thorough investigation.

In parallel, the silence of psychiatrists about various socioeconomic factors leading to or determining the use of ECT is just as puzzling and requires investigation, especially in light of the increased proportion in many jurisdictions of elderly persons who are given ECT, and of recent reports showing the virtual worthlessness of ECT in preventing "relapse" in depression (84% relapse rate within 6 months of receiving ECT, as found by Sackeim and colleagues, 2001). Recently, for example, it was revealed that at one major hospital in British Columbia, Riverview Hospital, the use of ECT among the elderly increased a whopping 129% in a period of only 4 years (from 678 in 1996 to 1,558 in 1999; the total number of ECT treatments increased from 6,176 to 10,028 during the same period). The increase immediately followed an administrative change in billing procedures, allowing physicians to bill each treatment individually. The British Columbia government ordered an inquiry into the practices, though its interest in the matter appears mostly pecuniary. Of particular relevance, however, Dr. Derek Eaves, the vice-president of medicine at Riverview hospital, was quoted as saying that "the only reason ECT at Riverview has increased as much as it has is because of a rising population of elderly people and the associated frequency of depression because of loneliness" (Fayerman, 2000, emphasis added).

Neurologists observe that seizures can and do cause various types of brain damage and dysfunction but are strangely silent about the potential for brain damage and dysfunction from seizures provoked by ECT, a controversial treatment with the most direct pertinence to their expertise. Some psychiatrists openly admit that loneliness causes depression, which is treated by inducing grand mal seizures in the brains of the lonely. Something is definitely wrong with this picture.

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