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Letter

ECT, Sleep, and Biogenic Amines in Affective Illness

To the Editors:

With great interest, we read the study of Hoffmann et al. (1985) on the effects of electroconvulsive therapy (ECT) on sleep and biogenic amines. This study is a nice confirmation and generalization of the results of a case report we recently published (Grunhaus et al., 1985). In that study we presented serial sleep recordings and dexamethasone suppression test (DST) results of a psychotically depressed patient undergoing ECT. Essentially, we found normalization of the latency of rapid eye movement (REM) sleep, decrease in REM density, and normalization of the DST with the treatment, similar to the results reported by Hoffmann et al.

Several methodological questions are unclear from the report of Hoffmann et al. They state that patients did not receive any antidepressant treatment during the ECT course; did they receive any other form of psychotropic medication, e.g., benzodiazepines for sleep, or neuroleptics? A similar question arises with regard to the followup sleep recordings which occurred 9 to 11 days after the ending of the ECT course; did they receive any psychotropic medication, such as starting a maintenance tricyclic antidepressant, during this interval between end of ECT and sleep recordings? The presence of psychotropic medications may significantly modify the sleep electroencephalogram (EEG) results and, thus, it is very important to clarify this point.

An interesting point not addressed by the authors in their article is the stability of the sleep recordings across nights. We are currently analyzing the results of serial sleep EEG recordings in our own sample of patients treated with ECT. One of our interesting observations is that sleep recordings vary, on occasions greatly, from night to night. Some patients, following the full course of ECT treatment or advanced in the course, may show approximately normal REM latency one night and on the second night drop to 20-30 minutes of REM latency. It is unclear to us the reason for such marked fluctuations. We do not have the advantage that Hoffmann et al. apparently had (see above) of waiting 9-14 days to institute the maintenance treatment after ECT; thus, it would be very interesting to note whether these changes in REM recording from one night to the other also occurred in their sample. The article reports means of 3 nights, but it would be of great interest if the authors could comment on the internight variability.

The use of EEG recordings in the monitoring of ECT response is a novel and very interesting idea. In general, the use of sleep EEG as a device to monitor treatment has been limited by the impact that psychotropic medications have on its parameters. Its use during ECT avoids some of these limitations. In general, patients receiving ECT are more severely ill or have been resistant to medications; an instrumental device which will help us monitor the course of treatment and possibly identify parameters associated with response or lack of response will most certainly be welcomed and put to good use. Serial EEG sleep recording may well be one such device.

References

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