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Response: Can ECT cause 'kindling' in the human brain ?

Thank you for your interest in our report, in which we describe five patients who developed florid temporal epileptiform abnormalities on EEG, including three who developed clinical epileptic seizures, whilst receiving ECT and despite no prior history of epilepsy.[1] Dr Asadi-Pooya has queried whether a causal relationship can be inferred from these findings, as previous studies have not found an association between ECT and epilepsy, and ECT has been used to to control seizures in some patients.

Studies that have reported no association between ECT and epilepsy, including the two referenced by Dr Asadi-Pooya, differ significantly in patient cohort and methodology to our series.[2,3] In Ray's study the median number of ECT sessions was 7, and 57% of patients received between 6 and 8 sessions. EEG findings were not documented and assessment was performed retrospectively using patient files. In Blackwood et al's study the mean number of ECT sessions was 16.8 (range 1 to 75) and the mean duration between last ECT course and clinical assessment was 18 months. Conversely, the mean number of ECT sessions in our series was 174.6 (range 36 to 348) and all patients were assessed during their ECT course. Of two patients receiving under 100 sessions both had total treatment durations under 12 months. Our patients were assessed under particularly intensive and prolonged treatment regimes. Interestingly, these regimes fit more closely with animals models of kindling, as raised in Blackwood et al's introduction. Specifically, frequent and regular electrical stimulations appear most effective at inducing this phenomenon.[4]

As Dr Asadi-Pooya has pointed out there is evidence that ECT has anti-convulsant properties under certain situations. However this does not exclude the possibility of a pro-convulsant effect through other mechanisms. As raised in our discussion there is neuroimaging evidence to support a differential impact of ECT upon different brain networks.[5] Thus ECT may potentially suppress

seizures in networks sustaining generalized convulsions whilst promoting seizures threshold in mesio-temporal (limbic) networks.

Whilst re-introducing ECT and observing a recurrence of epileptiform changes on EEG would strengthen a causal link, we felt this would be unethical. The dramatic resolution of EEG abnormalities on cessation of ECT argues that it is most likely that the EEG changes are causally related. To the best of our knowledge the alternative possibilities raised by Dr Asadi-Pooya were excluded: psychiatric drugs were not reduced (in one patient the clozapine dose was increased), neuro-imaging was normal and there was no known past or family history of epilepsy.

Following these initial observations, our psychiatry colleagues now have a lower threshold for ordering EEG on patients receiving prolonged courses of ECT, and we have observed temporal lobe epileptiform discharges in several other patients. However, we agree that a larger study is required to determine the prevalence of these changes. All subjects would require an epilepsy clinical history, neuroimaging and EEG prior to commencement of the ECT course, and at regular intervals during the treatment course. If epileptiform changes or seizures developed, cessation of ECT would be recommended and follow-up EEG could be performed.

References

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We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

None of the authors has any conflict of interest to disclose.

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