

enzyme even when those laboratories' own publications show clearly and repeatedly that they did not. Readers can obtain these articles and judge for themselves who deserves credit for isolating and cloning the full-length *Taq* DNA polymerase.

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ECT has proved effective in treating depression ...

Sir—Peter Sterling¹ criticizes the reviewer of my book² for stating that electroconvulsive therapy (ECT) is both safe and effective in treating the severely mentally ill³. Using the example of a friend who, a year after having ECT, complained of “huge gaps in recall of major life events”, he argues that history will view ECT as another “great and desperate cure” akin to psychosurgery.

Sterling errs on many counts, the principal one being his reliance on images of ECT from half a century ago. Despite Sterling's assertion, electric currents are not inherently dangerous just because touching an open electric socket is painful. For example, they are widely used to slow a racing heart. In ECT, because of the resistance of skin, bone and galea, very little of the energy gets to brain tissue; indeed, modern technique calls for the determination of the minimum current needed to elicit a seizure.

Scientists have sought evidence of brain damage for half a century, using elaborate brain imaging methods and psychological tests in humans, and neuropathological studies in animals given high levels of currents or large numbers of seizures. No such evidence has emerged⁴.

Sterling is wrong when he argues that the cure for depression “requires this procedure to be repeated 10–20 times over a week or so”. Modern ECT resolves depression in four to eight sessions, spaced at two to three times a week, in more than 80 per cent of patients using optimal treatment methods. No study has found any other antidepressant treatment to be more effective than ECT⁵.

ECT has no semblance to stroke, and Sterling's comparison is unfounded. Again, contrary to Sterling's argument, studies of the functions of the non-dominant hemisphere fail to demonstrate persistent effects of unilateral ECT on both non-verbal and verbal testing^{5,6}.

ECT was developed in the late 1930s,

and by 1950 tests of memory function were part of our treatment schedule^{2,5}. Such tests are routine today, and the literature on ECT and memory is extensive⁶. There are defects in memory associated with severe mental illnesses, and these defects are exaggerated during treatment with medications or ECT. But when patients recover from their illness, and from the immediate effects of ECT or medicines, they are as able to recall life events, to learn anew and to work effectively as age- and education-matched controls.

The benefits of ECT are temporary, but so are the benefits of psychiatric medications. These treatments must be continued for years to prevent relapse.

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... and there's no proof of lasting brain damage

Sir—Peter Sterling¹ asserts that electroconvulsive therapy (ECT) damages the cerebral hemispheres. A comprehensive review of the relevant literature provides no objective evidence that ECT is capable of causing brain damage in human beings^{2,3}.

In support of his belief that psychiatrists would find evidence for ECT-induced brain damage if only they would test for it, Sterling cites a single study, conducted more than 50 years ago. In sharp contrast are the many studies in the modern era examining short- and long-term memory before and after bilateral or unilateral ECT, examining both left- and right-hemisphere functions and including non-ECT control groups for comparison. All but one of these studies reveals no evidence for persistent or long-term deficits².

The single positive study⁴ found detectable deficits in autobiographical memory six months after bilateral (but not unilateral) ECT. Unfortunately, this was published only in academy proceedings, which are not subjected to peer review.

Sterling's claim that ECT “releases massive quantities of glutamate”, thus causing neuronal death, is not supported by any citation. However, myelin basic protein, which is found in the cerebrospinal fluid following stroke, does not

appear there following ECT⁵, nor does the CPK brain isoenzyme⁶.

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Shrinking shrews

Sir—Wikelski and Thom, in their interesting description of marine iguanas *Amblyrhynchus cristatus* shrinking in response to food shortage¹, thought theirs was the first report of shrinkage in adult vertebrates. But in 1949, the Polish mammalogist August Dehnel drew attention to the shrinkage overwinter of braincase size in common shrews *Sorex araneus*². This phenomenon, which now bears Dehnel's name, affects other organs (especially the liver and kidneys), and occurs in all northern soricines examined so far. It is, like the changes in the marine iguanas, a reversible response of individuals, not just a phenomenon of populations³.

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Cloning shock: was Dolly a three-legged accident?

Sir—We all know Dolly was cloned from one of her dam's cells, but why does *Nature* publish a photo of her¹ with three of her own legs and one of her mother's apparently grafted on to her umbilicus? On the cover of the *Nature* issue that announced Dolly, this fourth leg had a black hoof, quite properly since—in the photo² from which the cover image was taken—it belonged to the Scottish Blackface dam standing behind her. It's time the world was told what happened to Dolly's other leg.

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