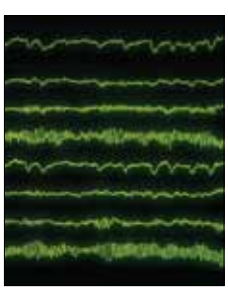
When can neurofeedback join the clinical armamentarium?

Neurofeedback appears both to improve normal brain function[1] and to treat a wide range of mental disorders, including attention deficit hyperactivity disorder (ADHD), epilepsy, depression, anxiety, insomnia, autism spectrum disorder, and alcoholism.[2] However, despite a relatively long history, the medical community continues to question the clinical utility of this technique. To earn widespread recognition as evidence-based medicine, neurofeedback must meet three challenges: first, perform at least on par with standard-of-care treatments in randomised controlled trials for each disorder that neurofeedback purports to help; second, consistently outperform highly comparable placebo control conditions (eg, sham neurofeedback); and third, establish a clear mechanism for the claimed therapeutic benefits.

In electroencephalographic (EEG) neurofeedback—the earliest and most widely practised form of neurofeedback[3]—participants attempt to modulate an ongoing feedback signal derived from real-time electrical activity of their own brain. In learning to control a particular brain signal, participants allegedly improve an associated behaviour. The underlying brain-based theory of this neurofeedback dynamic draws on research showing an association between clinical disorders and quantitative differences in EEG signal, yet rests on an unsupported tendency to reduce complex overarching behaviours to circumscribed brain processes. Moreover, relevant studies seldom show that receiving neurofeedback, let alone a precise brain signal, constitutes a necessary component for attaining the supposed benefits.[3] Alternatively, psychosocial factors (eg, expectation and motivation), rather than neurophysiological parameters, may mediate the reported clinical improvement. Typical EEG neurofeedback protocols require participants to visit a medical clinic for 20–40 sessions and interface with seemingly cutting-edge brain technology.[3] Future research should tease apart and examine these quantifiable psychosocial factors (eg, time spent at clinic and confidence in neurofeedback technology) to allow a better scientific understanding of how and to what degree such influences drive the measured outcomes.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Few consumers and practitioners appreciate that EEG neurofeedback helps patients regardless of the feedback source.[3] In other words, sham neurofeedback (eg, from irrelevant brain activity or even from a different brain) improves treatment outcomes as much as true neurofeedback.[3,4] After a thorough literature search (using the search terms "neurofeedback OR biofeedback AND electroenchapalogra* OR EEG" in Scopus, Web of Science, and Google Scholar), we could find only one sham-controlled, double-blind EEG neurofeedback study that showed clinical superiority of veridical over sham feedback.[5] Results from this study of 32 patients with chronic stroke showed that, in conjunction with physiotherapy, participants who received genuine brain-based feedback improved motor control of their affected arm to a greater extent than did those who received random feedback

(3·41 *vs* 0·35 points on the 54-point upper-limb Fugl-Meyer motor score assessment, p=0·018).[5] The other clinical EEG neurofeedback studies had either inadequate experimental design (eg, to disentangle brain-based mechanisms from psychosocial influences) or similar effects between real and sham feedback. Subsequently, placebo factors are likely to account for most research findings and clinical improvements related to EEG neurofeedback.[3] Although contemporary biomedicine often dismisses placebo outcomes as noise or non-effects, many standard treatments benefit from placebos.[6] Future research should explore the healing mechanisms common to true and sham neurofeedback, including the role of motivation, expectation, interaction with health professionals, and demand characteristics.

Despite much research into the clinical benefits of EEG neurofeedback,[7] only a few studies[8–11]—all addressing paediatric ADHD—directly compared neurofeedback with currently accepted treatments (ie, psychostimulants). Two of these experiments[8,9] showed similar improvements in attention, yet did not collect neurological measures. One study[10] showed similar changes in resting-state EEG activity, yet neglected to ascertain whether attention actually improved. Results from the other study[11] suggested superiority of medication over EEG neurofeedback in terms of both behaviour and neural activity. Notably, these studies scantily report whether participants learned to modulate the brain signal of interest and thus provide little insight into the neural underpinnings of these effects. To promote EEG neurofeedback as a brain regulation therapy, researchers will need to conduct high-quality clinical trials that confirm the alleged underlying neurological mechanisms and highlight an advantage over sham neurofeedback. To justify clinical application, EEG neurofeedback needs to perform similar to, or better than, currently accepted treatments. If research proves EEG neurofeedback effective—even if psychosocial factors rather than neurological substrates drive clinical improvement—practitioners could find ways to apply this intervention in a manner that is both scientifically judicious and ethically acceptable. Meanwhile, unlike with EEG, nascent findings from neurofeedback with functional MRI seem to pave a promising, albeit tentative, road towards the coveted holy grail of the self-regulating brain.[12]

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