

When Can Neurofeedback Join the Clinical Armamentarium?

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Neurofeedback appears to both improve normal brain function¹ and treat a wide range of mental disorders, including attention deficit hyperactivity disorder (ADHD), epilepsy, depression, anxiety, insomnia, autism spectrum disorder, and alcoholism.² Despite a relatively long history, however, the medical community continues to question the clinical utility of this technique. To earn widespread appellation as evidence-based medicine, neurofeedback must meet three challenges: 1) perform at least on par with standard-of-care treatments in randomized controlled trials for each disorder where neurofeedback purports to help; 2) consistently outperform highly comparable placebo control conditions (e.g., sham neurofeedback); and 3) establish a clear mechanism for the claimed therapeutic benefits.

In electroencephalographic (EEG) neurofeedback – the earliest and most widely practised form of neurofeedback³ – participants attempt to modulate an on-going 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 correlating clinical disorders with quantitative differences in EEG signal, yet rests on an unsupported tendency to reduce complex overarching behaviours to circumscribed brain processes. Relevant studies, moreover, seldom demonstrate that receiving neurofeedback, let alone a precise brain signal, constitutes a necessary component to attain the supposed benefits.³ Alternatively, psychosocial factors (e.g., 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 (e.g., time spent at clinic,

confidence in neurofeedback technology) to allow a better scientific understanding of how and to what degree such influences drive the measured outcomes.

Few consumers and practitioners appreciate that EEG-neurofeedback helps patients regardless of the feedback source.³ In other words, sham neurofeedback (e.g., 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 (query “*neurofeedback OR biofeedback AND electroencephalography* OR EEG*” in Scopus®, Web of Science™, and Google Scholar) we could find only one sham-controlled, double-blind EEG neurofeedback study that demonstrated clinical superiority of veridical over sham feedback.⁵ This study engaged 32 chronic stroke patients and found that, in conjunction with physiotherapy, participants who received genuine brain-based feedback, compared to random feedback, better improved motor control of their affected arm (3.41 versus 0.35 points on the 54-point upper limb Fugl-Meyer motor score assessment, $p = 0.018$).⁵ The other clinical EEG-neurofeedback studies featured either inadequate experimental design (e.g., to disentangle brain-based mechanisms from psychosocial influences) or comparable effects between real and sham feedback. Subsequently, placebo factors likely account for most research findings and clinical improvements related to EEG-neurofeedback.³ While 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 on the clinical benefits of EEG-neurofeedback⁷, only a few studies – all addressing paediatric ADHD – tested a direct comparison of neurofeedback to currently accepted treatments.^{8–11} Two of these experiments reported comparable improvements in attention between the neurofeedback and medication groups yet shied away from collecting neurological measures;^{8,9} one showed similar changes in resting-state EEG activity yet neglected to ascertain whether attention actually improved;¹⁰ the other suggested superiority of medication over EEG-neurofeedback in terms of both behaviour and neural activity.¹¹ 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 fashion that is both scientifically judicious and ethically acceptable. Meanwhile, unlike with EEG, nascent findings from neurofeedback with functional magnetic resonance imaging seem to pave a promising, albeit tentative, road towards the coveted holy grail of the self-regulating brain.¹²

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CONTRIBUTORS

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DECLARATION OF INTEREST

We declare no competing interests.

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