TECHNICAL COMMENTARY

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Biofeedback

Introduction

Biofeedback is a technique in which information about the person's body is fed back to the person so that they may be trained to alter the body's conditions. Physical therapists use biofeedback to help stroke victims regain movement in paralyzed muscles. Other specialists use biofeedback to help their patients cope with pain. It is also commonly used to reduce stress and anxiety, and to encourage relaxation.

Electromyographic biofeedback is used by psychologists to help anxious patients learn to relax. The electromyograph picks up electrical signals in the muscles and translates these signals into a flashing light or a beep every time muscles grow tense. If patients relax their tense muscles, they can slow down the flashing or beeping. Electroencephalographic biofeedback is used to teach self-regulation of brain function. It is usually provided using video or sound, with positive feedback for desirable brain activity and negative feedback for undesirable brain activity. Thermal biofeedback uses a temperature sensor to allow the patient to track his or her body temperature. During times of stress, the body will divert blood from the surface area of the body to the muscles and organs, allowing us to better respond to a nearby threat. When a patient is stressed, this will show as a drop-in temperature in the body's surface areas. When a patient's surface temperature is high, it typically means they are in a relaxed or sleepy state.

Dysregulation in autonomic nervous system activity is common in a variety of mental health disorders and presents targets for biofeedback. Hypoarousal patterns include slow, regular heart rate, increased heart rate variability, warm skin temperature, low sweat gland activity, and dominance of EEG frequencies in the theta to low alpha range (3.5–10 Hz). In contrast, hyperarousal is reflected by increased heart rate and decreased heart rate variability, high electrodermal activity, and higher frequency EEG bandwidth ranges in high-alpha or beta range (15–42 Hz).

Method

We have included only systematic reviews (systematic literature search, detailed methodology with inclusion/exclusion criteria) published in full text, in English, from the year 2000 that report results separately for people with a diagnosis of schizophrenia, schizoaffective disorder, schizophreniform disorder or first episode schizophrenia. Reviews were identified by searching the databases MEDLINE, EMBASE, CINAHL, Current Contents, PsycINFO and the Cochrane library. Hand searching reference lists of identified reviews was also conducted. When multiple copies of reviews were found, only the most recent version was included. Reviews with pooled data are prioritised for inclusion.

Review reporting assessment was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist that describes a preferred way to present a meta-analysis[1]. Reviews reporting less than 50% of items have been excluded from the library. The PRISMA flow diagram is a suggested way of providing information about studies included and excluded with reasons for exclusion. Where no flow diagram has been presented by individual reviews, but identified studies have been described in the text, reviews have been checked for this item. Note that early reviews may have been guided by less stringent reporting checklists than the PRISMA, and that some reviews may have been limited by journal guidelines.

Evidence was graded using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) Working Group approach where high quality evidence such as that gained from randomised controlled trials

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(RCTs) may be downgraded to moderate or low if review and study quality is limited, if there is inconsistency in results, indirect comparisons, imprecise or sparse data and high probability of reporting bias. It may also be downgraded if risks associated with the intervention or other matter under review are high. Conversely, low quality evidence such as that gained from observational studies may be upgraded if effect sizes are large or if there is a dose dependent response. We have also taken into account sample size and whether results are consistent, precise and direct with low associated risks (see end of table for an explanation of these terms)[2]. The resulting table represents an objective summary of the available evidence, although the conclusions are solely the opinion of staff of NeuRA (Neuroscience Research Australia).

Results

We found two systematic reviews that met our inclusion criteria[3, 4].

- Low quality evidence from few small studies is unable to determine the benefits of biofeedback for patients with schizophrenia.

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Schoenberg PLA, David AS

Biofeedback for Psychiatric Disorders: A Systematic Review

Applied Psychophysiological Biofeedback 2014; 39: 109-135

View review abstract online

Comparison	**Biofeedback for medicated patients with schizophrenia.**
Summary of evidence	**Low quality evidence (very small samples, unable to assess consistency or precision, direct) is unable to determine the benefits of biofeedback for patients with schizophrenia.**
Electroencephalographic biofeedback	
1 pre-post study (N = 24) 20 x 15-minute sessions reported that patients required 17sessions to gain conscious control of slow cortical potentials regulation vs. 5 sessions for healthy controls. No differences in symptoms were reported.	
Electromyographic biofeedback	
1 randomised pre-post study (N = 30) with 7 x 20-minute sessions reported significant improvement post-biofeedback in motor speed and lateralized coordination (finger tapping test), with no change in the patient control group. The biofeedback group also showed significant improvement in social competence and interest (Tension-Anxiety factor of the Profile of Mood States scale). 1 randomised pre-post study (N = 30) with 6 x 90-minute sessions reported significant improvement post-biofeedback on symptom scores and maladaptive behaviours (no control comparison). All patients reported reduced muscle tension. 1 pre-post study (N = 15) with 10 x 15-minute sessions reported no significant differences in clinical improvements between patients with schizophrenia, patients with anxiety or patients with tension headache. All patients reported reduced muscle tension.	
Thermal biofeedback	
1 randomised pre-post study (N = 40) with 10 x 20-minute sessions reported no significant differences between groups post-biofeedback treatment for anxiety between those receiving minimal treatment (control), relaxation, thermal biofeedback and thermal biofeedback + relaxation.	
Consistency in results‡	Unable to assess; no measure of consistency is reported.
Precision in results§	Unable to assess; no measure of precision is reported.

Directness of results‖	Direct

Fielenbach S, Donkers FCL, Spreen M, Visser HA, Bogaerts S

Neurofeedback training for psychiatric disorders associated with criminal offending: A review

Frontiers in Psychiatry 2018; 8

View review abstract online

Comparison	**Neurofeedback for criminal offenders with schizophrenia.**
Summary of evidence	**Low quality evidence (very small samples, unable to assess consistency or precision, direct) is unable to determine the benefits of neurofeedback.**
Electroencephalographic biofeedback	
2 studies employed slow-cortical potential neurofeedback at central electrode positions over 10-20 sessions. 1 study (N = 25) found patients able to learn to control interhemispheric asymmetry, while the other study (N = 24) found patients achieved differentiation of feedback trials comparable to controls in the last three sessions of training. 1 case study trained the individual alpha peak frequency over 12.5 hours within four consecutive days and reported short-term memory improvements.	
Consistency in results	Unable to assess; no measure of consistency is reported.
Precision in results	Unable to assess; no measure of precision is reported.
Directness of results	Direct

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Explanation of technical terms

* Bias has the potential to affect reviews of both RCT and observational studies. Forms of bias include; reporting bias – selective reporting of results; publication bias - trials that are not formally published tend to show less effect than published trials, further if there are statistically significant differences between groups in a trial, these trial results tend to get published before those of trials without significant differences; language bias – only including English language reports; funding bias - source of funding for the primary research with selective reporting of results within primary studies; outcome variable selection bias; database bias - including reports from some databases and not others; citation bias - preferential citation of authors. Trials can also be subject to bias when evaluators are not blind to treatment condition and selection bias of participants if trial samples are small[5].

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† Different effect measures are reported by different reviews.

Prevalence refers to how many existing cases there are at a particular point in time. Incidence refers to how many new cases there are per population in a specified time period. Incidence is usually reported as the number of new cases per 100,000 people per year. Alternatively some studies present the number of new cases that have accumulated over several years against a person-years denominator. This denominator is the sum of individual units of time that the persons in the population are at risk of becoming a case. It takes into account the size of the underlying population sample and its age structure over the duration of observation.

Reliability and validity refers to how accurate the instrument is. Sensitivity is the proportion of actual positives that are correctly identified (100% sensitivity = correct identification of all actual positives) and specificity is the proportion of negatives that are correctly identified (100% specificity = not identifying anyone as positive if they are truly not).

Mean difference scores refer to mean differences between treatment and comparison groups after treatment (or occasionally pre to post treatment) and in a randomised trial there is an assumption that both groups are comparable on this measure prior to treatment. Standardised mean differences are divided by the pooled standard deviation (or the standard deviation of one group when groups are homogenous) which allows results from different scales to be combined and compared. Each study's mean difference is then given a weighting depending on the size of the sample and the variability in the data. Less than 0.4 represents a small effect, around 0.5 a medium effect, and over 0.8 represents a large effect[5].

Relative risk (RR) refers to the probability of a reduction (< 1) or an increase (> 1) in a particular outcome in a treatment group, or a group exposed to a risk factor, relative to the comparison group. For example, a RR of 0.75 translates to a reduction in risk of an outcome of 25% relative to those not receiving the treatment or not exposed to the risk factor. Conversely, a RR of 1.25 translates to an increased risk of 25% relative to those not receiving treatment or not having been exposed to a risk factor. A RR of 1.00 means there is no difference between groups. A medium effect is considered if RR > 2 or < 0.5 and a large effect if RR > 5 or < 0.2[6]. Odds ratios (ORs) are similar to RRs, but they are based on the probability of an event occurring divided by the probability of that event not

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occurring. ORs and RRs are similar in size when the event is rare, such as with schizophrenia. lnOR stands for logarithmic OR where a lnOR of 0 shows no difference between groups. Hazard ratios (HRs) measure the effect of an explanatory variable on the hazard or risk of an event.

Correlation coefficients (eg, *r*) indicate the strength of association or relationship between variables. They can provide an indirect indication of prediction, but do not confirm causality due to possible and often unforseen confounding variables. An *r* of 0.10 represents a weak association, 0.25 a medium association and 0.40 and over represents a strong association. Unstandardised (*b*) regression coefficients indicate the average change in the dependent variable associated with a 1 unit change in the independent variable, statistically controlling for the other independent variables. Standardised regression coefficients represent the change being in units of standard deviations to allow comparison across different scales.

‡ Inconsistency refers to differing estimates of effect across studies that is not explained by subgroup analyses and therefore reduces confidence in the effect estimate. I^2 is the percentage of the variability in effect estimates that is due to heterogeneity rather than sampling error (chance) - 0% to 40%: heterogeneity might not be important, 30% to 60%: may represent moderate heterogeneity, 50% to 90%: may represent considerable heterogeneity and over this is considerable heterogeneity. I^2 can be calculated from Q (chi-square) for the test of heterogeneity with the following formula[5];

$$I^2 = \left(\frac{Q - df}{Q}\right) \times 100\%$$

§ Imprecision refers to wide confidence intervals indicating a lack of confidence in the effect estimate. Based on GRADE recommendations, a result for continuous data (standardised mean differences, not weighted mean differences) is considered imprecise if the upper or lower confidence limit crosses an effect size of 0.5 in either direction, and for binary and correlation data, an effect size of 0.25. GRADE also recommends downgrading the evidence when sample size is smaller than 300 (for binary data) and 400 (for continuous data), although for some topics, these criteria should be relaxed[7].

‖ Indirectness of comparison occurs when a comparison of intervention A versus B is not available but A was compared with C and B was compared with C that allows indirect comparisons of the magnitude of effect of A versus B. Indirectness of population, comparator and/or outcome can also occur when the available evidence regarding a particular population, intervention, comparator, or outcome is not available and is therefore inferred from available evidence. These inferred treatment effect sizes are of lower quality than those gained from head-to-head comparisons of A and B.

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