Appl Psychophysiol Biofeedback (2014) 39:109–135
DOI 10.1007/s10484-014-9246-9

Biofeedback for Psychiatric Disorders: A Systematic Review

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Published online: 8 May 2014

Abstract Biofeedback potentially provides non-invasive, effective psychophysiological interventions for psychiatric disorders. The encompassing purpose of this review was to establish how biofeedback interventions have been used to treat select psychiatric disorders [anxiety, autistic spectrum disorders, depression, dissociation, eating disorders, schizophrenia and psychoses] to date and provide a useful reference for consultation by clinicians and researchers planning to administer a biofeedback treatment. A systematic search of EMBASE, MEDLINE, PsycINFO, and WOK databases and hand searches in Applied Psychophysiology and Biofeedback, and Journal of Neurotherapy, identified 227 articles; 63 of which are included within this review. Electroencephalographic neurofeedback constituted the most investigated modality (31.7 %). Anxiety disorders were the most commonly treated (68.3 %). Multimodal biofeedback appeared most effective in significantly ameliorating symptoms, suggesting that targeting more than one physiological modality for bio-regulation increases therapeutic efficacy. Overall, 80.9 % of articles reported some level of clinical amelioration related to biofeedback exposure, 65.0 % to a statistically significant ($p < .05$) level of symptom reduction based on reported standardized clinical parameters. Although the heterogeneity of the included studies warrants caution before explicit efficacy statements can be made. Further development of standardized controlled methodological protocols tailored for specific disorders and guidelines to generate comprehensive reports may contribute towards establishing the value of biofeedback interventions within mainstream psychiatry.

Keywords Biofeedback · Psychopathology · Psychophysiology · Anxiety · Behavior therapy

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Introduction

Despite 27 % of people in Europe suffering from mental health problems each year (Lancet Global Mental Health Group 2007), 74 % of these people receive no pharmaceutical or traditional psychological treatment from mental health care services, often due to multiple barriers in accessing such services. A call for action to introduce innovative and easily accessible cognitive and behavioral strategies for treating depressive, anxiety and other common mental disorders (CMDs), which can be implemented by general physicians and community health workers, has been proposed (Lancet Global Mental Health Group 2007). This target may be met by the adjunctive use of less traditional therapies in treatment programs for psychiatric disorders. Studies suggest alternative interventions are used more frequently by people with psychiatric disorders, particularly anxiety and depressive symptoms (Kessler et al. 2001) than people without mental health problems. A survey conducted in the US found that 34.4 and 30.2 % of alternative treatments employed for anxiety and severe depression, respectively, consisted of "cognitive feedback" approaches, defined as relaxation, imagery, self-help groups, hypnosis, and biofeedback. Of particular note, only

1.6 % of studies treating anxiety and 1.5 % of studies treating severe depression utilized biofeedback treatments (Kessler et al. 2001).

Dysregulation in autonomic nervous system (ANS) activity often provides biomarkers for various mental health problems. For example, "relaxed" ANS patterns include slow, regular heart rate, increased heart rate variability, and warm skin temperature due to increased vasodilation, low sweat gland activity (electrodermal activity) (Schwentker and Vovan 1995), and dominance of EEG frequencies in the theta to low alpha (3.5–10 Hz) bandwidth range. Hyperarousal, in contrast, 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 (15–42 Hz), often reflecting anxiety and/or panic states (Putman 2000). Thus, biofeedback which targets maladaptive physiology may help enable patients to recognize and alter problematic physical symptoms (Pal Singh and Kaur 2007) that may be facilitating and/or perpetuating the associated psychological problem.

The clinical efficacy of biofeedback has been investigated in a range of psychiatric disorders, including; anxiety (Beckham et al. 2013; Kim et al. 2012; Reiner 2008; Meuret et al. 2001; Rice et al. 1993), depression (Walker and Lawson 2013; Siepmann et al. 2008; Uhlmann and Froscher 2001; Baehr et al. 1997), to schizophrenia (Schneider et al. 1992). Schneider (1987) evaluated the cost effectiveness of biofeedback treatment in clinical settings, where reduction in physician visits and/or medication usage, decrease in medical care costs to patients, decrease in frequency and duration of hospital stays and re-hospitalization, decrease in mortality, and increase in quality of life, were considered. Biofeedback was found to be cost-effective on all dimensions reviewed, with cost/benefit ratios ranging between 1:2 and 1:5, with a median of 1:4.

The present systematic review was carried out to explore the current therapeutic use of biofeedback for a range of

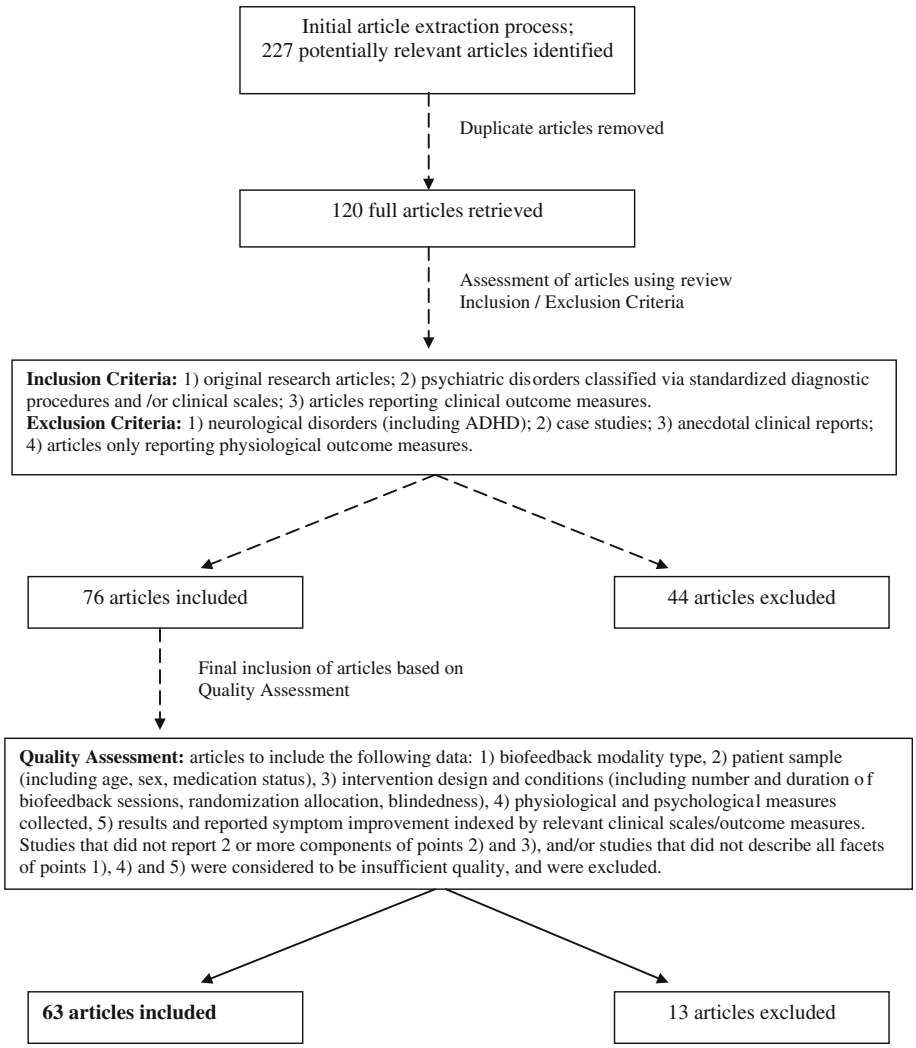

Fig. 1 Search and elimination process

psychiatric disorders, established via the following questions; (1) which psychiatric disorders have been treated using biofeedback; (2) which physiological parameters were targeted during the biofeedback; (3) what duration and intensity of biofeedback exposure was utilized; and 4) was biofeedback reported as helpful in treating these psychiatric disorders based on clinical scales/reports evaluated in these studies? From this enquiry, some suggestions as to how biofeedback treatment might be implemented more effectively into mainstream psychiatry and clinical psychology practice are considered (Fig. 1).

Method

Inclusion/Exclusion Criteria

Original articles reporting data from studies investigating the efficacy of biofeedback in the treatment of the following psychiatric disorders were initially considered: addictions, anxiety disorders, Autism Spectrum Disorders (ASDs), depressive disorders, dissociative disorders, personality disorders, and psychoses. Neurological disorders, aside from ASDs, were excluded. One study of somatoform disorder was included in this review but other conditions which may be considered somatoform such as chronic pain, headache, fibromyalgia, irritable bowel syndrome etc., and hence arguably having a significant psychiatric component, were not included. Articles where the diagnosis of psychiatric disorder was ascertained via non-standardized diagnostic procedures and/or clinical scales were excluded. Articles reporting outcome measures pertaining to symptom change indexed via relevant standardized clinical scales and/or evaluation in more than one participant per study were included. Articles reporting only changes in physiological outcome variables were excluded.

Quality Assessment

Articles that satisfied the above inclusion requirements were then assessed (designed in alignment with the Efficacy Task Force (La Vaque et al. 2002) 5-level behavioral intervention efficacy ratings), as to whether the following information was included within the article: (1) biofeedback modality type, (2) patient sample (including age, sex, medication status), (3) intervention design and conditions (including number and duration of biofeedback sessions, randomization allocation, blindedness), (4) physiological and psychological measures collected, and (5) results and reported symptom improvement indexed by relevant clinical scales/outcome measures. Studies that did not report 2 or more components of points (2) and (3), and/or studies that did not describe all facets of points (1), (4) and (5) were considered to be of insufficient quality and were excluded.

Search Strategy

Relevant studies were initially identified by searching EMBASE, MEDLINE, PsycINFO, and Web of Knowledge (WOK) databases searching all years. The key words "biofeedback" or "neurofeedback" were searched alongside the following 16 terms, using the 'AND' search function: "addiction", "anxiety", "anorexia nervosa", "Asperger Syndrome", "autism", "bipolar affective disorder", "depersonalization disorder", "depression", "derealization", "dissociation", "eating disorder", "Obsessive–Compulsive Disorder (OCD)", "panic", "phobia", "Post-Traumatic Stress Disorder (PTSD)", "psychiatric disorder", "psychological disorder", "psychopathology", "psychosis", and "schizophrenia". Reference lists of relevant articles and previous literature reviews were hand searched for articles not included in the database search. A computer search was supplemented by hand searches in Applied Psychophysiology and Biofeedback (formerly 'Biofeedback and Self-Regulation') from March 1976 to 22 February 2014, and the Journal of Neurotherapy from May 1995 to 22 February 2014.

Results

Overview of Included Studies

An initial search yielded 227 citations; 160 from EMBASE, MEDLINE, PsycINFO, and WOK databases, 38 from bibliographies, and 29 from journal searches (Applied Psychophysiology and Biofeedback, Journal of Neurotherapy). Duplicate articles (i.e. the same articles sourced from different search engines) were eliminated, leaving 120 relevant articles. Of these retrieved articles, 76 met initial inclusion criteria. Forty-four articles initially identified were subsequently excluded: case studies (n = 9); omission of clinical outcome measures (n = 4); neurological disorders, such as attention deficit hyperactivity disorder (ADHD), epilepsy, brain injury or learning disability studies (n = 21). Readers are referred to articles not included within this review; Moriyama et al. (2012), Holtman and Stadler (2006), and Monastra et al. (2005); for extensive reviews of biofeedback interventions in the treatment of ADHD. Furthermore articles pertaining to the use of biofeedback for the addictions (n = 10) were also dropped. Readers are referred to an extant review carried out by Sokhadze et al. (2008), providing a thorough overview of the clinical applications of neurotherapy for substance use disorder (SUD) over the last three decades. We considered it unnecessary to duplicate their findings.

Table 1 Electroencephalographic (EEG) biofeedback (BF) studies

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design		Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
		(a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	(d) No. of sessions (e) Duration of BF (per session) (f) Follow-up			
Sarkar et al. (1999)	(a) **GAD**[a] (b) 50 (c) 20–55 (d) No	(a) 2 conditions; α BF (N = 25), versus pharmacological treatment (N = 25) (b) Yes (c) N/A	(d) Missing data (e) Missing data (f) No	(1) Hamilton anxiety rating scale (objective rating) (2) Somatic inkblot series-I (projective rating)	Both BF and pharmacological treatments sig. ↓ GAD symptoms	⇑ [although symptom reduction was not specific to BF only]
Vanathy et al. (1998)	(a) **GAD** (b) 18 (14 M, 4 F) (c) Mean age = 32.72 (d) No	(a) 3 conditions; α BF, θ BF, wait-list (b) No (c) Blind experimenter	(d) 15 (e) 30 min (f) No	Pre and post: (1) EEG spectral analysis (2) Hamilton anxiety rating (3) STAI[a], (4) GQL[a]	Both α-BF and θ-BF sig. ↓ Hamilton anxiety (objective) ratings Only θ-BF sig. ↓ GQL scores Only α-BF sig. ↓ STAI-T scores	⇑
Plotkin and Rice (1981)	(a) **High trait/chronic anxiety** (b) 10 (c) 18–29 (d) No; those in psychotherapy were excluded	(a) 3 groups; α ↑ or ↓, wait-list controls (b) Yes (c) No	(d) 5–7 (over 3 weeks) (e) 40 min (f) No	(1) Pre: entire MMPI[a], Welsh-A and Taylor manifest anxiety scores (2) STAI (trait) (3) Completed STAI (state) each session (4) Post: same as 1	Pre-post ANOVAS on Welsh-A, Taylor manifest and STAI trait scales were sig., indicating both groups were successful in ↓ trait anxiety. No change in anxiety in WL control group	⇑
Watson and Herder (1980)	(a) **Anxiety in psychiatric inpatients** (b) 66 (c) Mean age = 36.1 (d) Yes	(a) α – BF, placebo (sham) BF, no-treatment control (b) No (c) Yes (single)	(d) 10 (e) 60 min (f) No	Pre and post: (1) STAI, (2) MAACL[a], (3) BPRS[a], (4) MMPI, (5) blood pressure, (6) pulse rate	No sig. changes in any clinical ratings/scales evident	□
Hardt and Kamiya (1978)	(a) **High versus low trait anxiety** (b) 16 (c) Not specified (d) No	(a) 2 groups; high (n = 8) versus low (n = 8) anxiety; ↑ and ↓ α in both groups (b) No (c) N/A	(d) 7 consecutively (e) 32 min α ↑, 16 min α ↓ (f) No	Pre-post: MMPI, α baseline During: each session completed mood scale (MAACL[a]) before baseline, after α ↑, and after α ↓. Frontalis EMG and respiration	Low trait anxiety Ss sig. better at both α ↑and ↓ High trait anxiety Ss showed α change was related to ↓ in anxiety intensity. Anxiety ↓ unrelated to resting physiology or change	⇑
Hammond (2003)	(a) **OCD**[a] (b) 2 (1 F, 1 M) (c) Both 25 yrs (d) No med: 2 wks (F) 3 days (M) before	(a) QEEG[a]; photic stim. (b) N/A (c) N/A	(d) 50 and 40 (e) 30–35 min (f) 15 and 13 months	(1) Yale-Brown obsessive Compulsive (Y-B OC) Scale (2) Padua inventory (3) MMPI in 1st case	Normalization of Y-B OC and Pauda scale scores. MMPI scores ↓ in OCD, depression, anxiety, som and ↑ in extroversion	⇑

Table 1 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Glucek and Stroebel (1975)	(a) **OCD** (b) N = 26 BF[a], N = 12 AT[a], N = 187 TM[a] (c) College students (d) Yes	(a) α ↑ BF (b) Yes (c) N/A	(d) 20 (e) 60 min (f) 4 weeks	Pre and post: subjective reports of OCD symptoms and relaxation	Most patients reported relaxation as a result of the BF, not maintained in follow-up. Patients gained α control after 15 sessions on average	□
Mills and Solyom (1974)	(a) **OCD** (b) 5 (3 F, 2 M) (c) Mean age = 32.10 (d) Med suspended 2 weeks prior to BF	(a) α ↑ (b) N/A; no controls (c) N/A	(d) 7–20 (e) 60 min (f) No	(1) GSR[a], (2) heart rate, (3) EMG (4) respiration, (5) digital pulse volume, (6) EEG, (7) subjective reports about rumination patterns	All Ss reported sig. ↓ (in 4 Ss, cessation) of ruminations during the BF correlating with ↑ α state	⇑
Kouijzer et al. (2013)	(a) **ASD**[a] (b) 38 (30 M, 8 F) c) 12–17 d) Yes (n = 8)	(a) EEG BF (n = 13) based on Neuroguide assessment versus SC ↓ (n = 12) versus WL b) Yes c) Single (re: BF group)	(d) 23–40 (majority 40) (e) 21 min f) 6 months	Pre and post: (1) SCQ[a], (2) cognitive flexibility (3) inhibition, (4) planning, (4) attention, (5) working memory (WM)	No sig. improvement in the clinical measure (SCQ) was evident. Although sig. ↑ in cognitive flexibility pre-to-post EEG BF	□
Coben and Padolsky (2007)	(a) **ASD** (b) 49 (41 M, 8 F) (c) 3.92–14.66 Mean age = 8.56 (d) Yes	a) EEG BF (n = 37) based on QEEG assessments versus wait-list control group (n = 12) (b) Yes (c) N/A	(d) 20 (e) Not specified (f) No	Pre-assessment: QE Pre and post (1) ATEC[a], (2) GADS[a], (3) GARS[a], (4) PIC-2[a], (5) BRIEF[a], (6) Infrared (IR) imaging	EEG BF sig. ↓ ASD symptoms versus the wait-list control group. Specifically, improvements in attention, executive, visual perceptual and language functions	⇑
Scolnick (2005)	(a) **Asperger's syndrome** (b) 5 (M) (c) 12–16 (d) Yes	(a) Mu rhythm ↑ slow wave (4–10 Hz) suppression (b) N/A (c) N/A	(d) 24 (2 per week) (e) 30 min (f) No	Pre and post: (1) quantified EEG analysis, (2) parent and teacher behavioral checklist; social skills, empathy, inflexibility anxiety	Behavioral checklists showed improvement via ↓ anxiety, mood change, and tantrums, although not to statistically sig. levels 50 % drop out rate (5/10)	□

Table 1 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Jarusiewicz (2002)	(a) **ASD** (b) 24 (22 M, 2 F) (c) 4–13 Mean age = 7 (d)) No	a) EEG BF (N = 12) protocol depending on personal EEG activity versus wait-list controls (N = 12) b) Yes c) N/A	(d) Mean = 36 Range = 20–69 (e) 30 min (f) No	Pre and post: (1) ATEC, (2) 15 min assessment based on 'free play', (3) FEAS[a]	Neurofeedback group showed sig. improvements in autism symptoms and behaviours compared to wait-list controls. Specifically, sig. ↑ ATEC: sociability, speech/language/communication, and sensory/cognitive awareness	⇑
Walker and Lawson (2013)	(a) **Medication-resistant depression** (b) 183 (110 F, 73 M) (c) 12–70 (d) No	(a) θ ↓ + β ↑ (at electrode: FPO2) (b) N/A (c) N/A	(d) 6 (e) 20 min (f) 1 year	Pre and post: depressive symptoms assessed by the rush quick self-rated inventory	β-BF sig. ↓ ($p < .001$) in average depression scores. 84 % sample achieved > 50 % ↓ in depression scores. Least effective in 'very severe' patients; where 18/44 = no improvement	⇑
Choi et al. (2011)	(a) **Depression** (b) 23 (17 F, 6 M) (c) Mean age = 28.5 (d) No	(a) α asymmetry BF versus placebo psychotherapy (b) Yes (c) No	(d) 10 (twice per week) (e) 24 min (f) 1 month	Pre and post: (1) BDI, (2) Hamilton depression inventory (HAM-D)	Sig. ↓ in BDI and HAM-D scale scores in BF group only. No such clinical improvement via placebo psychotherapy	⇑
Baehr et al. (1997)	(a) **Depression** (b) 2 (F) (c) 65, 40	(a) α-θ and α asymmetry (b) N/A (c) N/A	(d) Ss 1 = 66 Ss 2 = 36 (1–2 per week) (e) 30 min (f) 5 month for Ss 1	Pre and post: MMPI-2	Depression ↓ in both Ss. MMPI-2 scores = sig. ↑ in general and social functioning, affect, and ↓ rumination	⇑
Saxby and Peniston (1995)	(a) **Depression in alcohol addiction** (b) 14 (8 M, 6 F) (c) Mean age = 48.38 (d) No	(a) Temperature BF pre-training α-θ BF (b) N/A (c) N/A	(d) 20 (e) 40 min (f) 21 months	Pre and post: (1) BDI[a] (2) MCMI[a] personality scale	BDI scores sig. ↓ after BF. Sig. ↓ in pathological personality dynamics (MCMI) 1/14 relapsed (alcohol consumption) during 21 month follow up	⇑

Table 1 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Schneider et al. (1992)	(a) **Depression** (b) 8 (M) and 8 (M) controls (c) 38–56 (d) Yes	(a) SCP regulation (b) N/A (c) N/A	(d) 20 (e) 14.66 min (f) No	Pre and post: (1) GAF[a], (2) Hamilton depression scale (HAM-D), (3) BPRS[a]	Patients could consciously regulate SCP ↑ and ↓. No associated change in clinical symptoms reported. Minimal correlation between SCP-BF and psychopathology	□
Manchester et al. (1998)	(a) **Dissociative identity disorder** (b) 11 (F) (c) 26–50 (mean = 41.1) (d) No	(a) α-θ BF (b) N/A (c) N/A	(d) 30 (e) 30 min (f) 7–25 months	Pre and Post: (1) MCMI-II[a] (2) GAF[a] Follow-up: (1) and (2), (3) DES[a]	All met Kluft's criterion for unification after BF. Mean GAF scores sig. ↑. 'Normal' range DES scores at follow-up	⇑
Peniston and Kulkosky (1991)	(a) **PTSD**[a] (b) 29 (M) (c) War veterans (d) Yes	(a) α-θ BF versus traditional treatment (b) Yes (c) N/A	(d) 30 (e) 30 min (f) 30 months	Pre and post: MMPI[a]	All patients in BF group sig. improved in all 10 clinical MMPI scales. Traditional treatment group only improved in one. All BF patients required ↓ medication after trial	⇑
Schneider, Heimann et al. (1992)	(a) **Schizophrenia** (b) 12 (M) and 12 (M) healthy controls (c) 23–32, 20–32 controls (d) Yes	(a) SCP regulation (b) N/A (c) N/A	(d) 20 (e) 14.6 min (f) No	Pre and post: (1) GAF, (2) BPRS, (3) Scale for assessment of negative symptoms (SANS)	Patients required 17 sessions of BF to gain conscious control of SCP; controls required only 5 sessions. No clinical changes reported	□

[a] See Table 8

Of the remaining 76 articles, 63 fulfilled the quality assessment and are considered in this review (see Tables 1, 2, 3, 4, 5, 6, 7). Thirteen of the 76 articles initially considered were subsequently excluded due to: vague or missing information pertaining to methods/outcome measures (n = 11); vague and unclear/non-clinical reportage of results (n = 1); no description of biofeedback modality used (n = 1). Due to the heterogeneity of article content and outcome measures, it was not feasible to carry out meta-analyses; rather, information from the quality assessment is summarized in Tables 1, 2, 3, 4, 5, 6, 7. Electroencephalographic (EEG) biofeedback was employed in 31.7 % (n = 20) of all reviewed studies, a further 28.6 % (n = 18) incorporated electromyographic (EMG), 15.9 % (n = 10) heart rate variability (HRV) and/or sole respiration, 6.3 % (n = 4) heart rate (HR), 4.8 % (n = 3) electrodermal (EDA), and 3.2 % (n = 2) thermal biofeedback methodologies. A further six articles (9.5 %) reported using a multi-modal biofeedback methodology; three combining EEG + EMG biofeedback, two EMG + thermal feedback, and one EEG + respiration. Overall, 68.3 % (n = 43) of articles reported testing the efficacy of

Table 2 Electromyographic (EMG) biofeedback studies

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	 (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Scandrett et al. (1986)	(a) **Anxiety disorder** (b) 88 (47 F, 41 M) (c) 18–65 (d) Yes	(a) Frontalis-EMG BF versus PMR or wait-list control (b) Yes (c) N/A	(d) 10–12 (e) 20 min (f) 1 month	Pre, post and follow-up: (1) McReynold's anxiety checklist (2) Verbal review of anxiety symptoms	No significant symptom changes were found. Somatic symptoms related to anxiety, were in some cases rated as more pronounced after BF	□
Barlow et al. (1984)	(a) **GAD and panic disorder** (b) N = 20, 9 = GAD, 11 = Panic disorder (13 M, 7F) c) 20–54 Mean age = 38 d) Not specified	(a) EMG BF treatment or 'no treatment' group (b) Yes (c) N/A	(d) 8 (over 14 weeks) (e) 20 min (f) 3–12 months	Pre and post: (1) anxiety disorders interview schedule (ADIS), (2) STAI, (3) BDI, (4) Psychosomatic symptom checklist (5) Daily anxiety self-rated scales During: EMG	Treatment group sig. improved on clinical ratings, physiological measures and self-reported measures of symptom improvement. Both GAD and PD patients responded equally well; 'no treatment' group did not improve clinically. BF group continued clinical improvement at follow-up	⇑
Lustman and Sowa (1983)	(a) **Anxiety and stress** (b) 24 (23 F, 1 M) (c) 20–24, Mean = 21.5 (d) No	(a) 3 groups; EMG BF, stress inoculation, or 'no treatment' control (b) Yes (c) N/A	(d) 10 (2 sessions per week for 5 weeks) (e) treatment sessions: 50 min (f) No	Pre and post: (1) Taylor manifest anxiety scale (2) teaching anxiety scale (3) Systolic and diastolic blood pressures	Both EMG BF and stress inoculation ↓ blood pressure levels. Limited ↓ in anxiety post BF, and not sig. when compared to controls. Sig. ↓ anxiety in PMR versus controls	□

Table 2 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? ☐ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Weinman et al. (1983)	(a) **GAD**[a] (b) 20 (F) (c) Over 18 (d) No	(a) EMG BF relaxation allocated to either high or low stress group (b) No (c) N/A	(d) 10 (2 per week) (e) 25 min (f) 6 weeks	Pre and post: (1) STAI, (2) BDI, (3) biological symptoms of anxiety During: frontalis EMG	70 % of high stress and 56 % of low stress Ss able to achieve maximal EMG relaxation. 70 % of high stress said the BF enabled them to feel more in control of their bodies. High stress group sig. changed assessment scores, whereas low stress group only biological symptoms of anxiety	⇑
Lavellee et al. (1982)	(a) **Chronic anxiety** (b) 40 (29 F, 11 M) (c) 21–50 (d) Medication free for this study unless symptoms became unbearable	(a) EMG frontalis (b) N/A (c) N/A	(d) 8 (1 per week) (e) 45 min (f) 6 months	Pre, post and follow-up: (1) hamilton anxiety scale (2) Zung self-rating anxiety scale, (3) Wechsler intelligence scale, (4) Eysenck personality inventory	32 Ss completed study. All Ss sig. ↓ EMG activity 25 % of Ss sig. ↓ anxiety according to clinical scales. 43.75 % 'mildly improved' (not sig.). 31.25 % showed no change in anxiety post trial. 'Responders' tended to have ↓ depression scores pre-trial	☐ [only 25 % sig. improved: BF seemed to have limited effect overall]
Rupert et al. (1981)	(a) **Chronic anxiety** (b) 20 (15 F, 5 M) (c) 20–55 (d) Medication free for this study	(a) 4 groups; EMG BF, relaxation, combined EMG BF and relaxation, 'no treatment' control (b) Yes (c) N/A	(d) 9 (e) 25 min (f) Not specified	Pre and post: (1) STAI state and trait (2) TMAS[a] scale During: EMG Post: progress evaluation form	No group showed sig. ↓ in muscle tension to adaptation level. EMG BF groups showed sig. ↓ trait anxiety scores Thus, BF proved most consistently effective treatment	⇑

Table 2 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? ☐ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Leboeuf A. (1980)	(a) **Chronic anxiety** (b) 26 (17 F, 9 M) (c) Mean age 38 (d) Yes	(a) 2 groups; frontalis EMG BF, progressive relaxation (b) No (c) N/A	(d) 16 (over 12 weeks) (e) 20 min (f) 3 months	Pre and post: (1) TMAS, (2) STAI –T, (3) EMG, (4) HR During: EMG	BF more successful at ↓ EMG activity. Both BF and PMR sig. ↓ anxiety scale scores, thus, no specificity	⇑
Raskin et al. (1980)	(a) **Anxiety** (b) 31 (c) Mean age 33.3 (d) Yes	(a) 3 groups; EMG BF, transcendental meditation, relaxation therapy (b) Yes (c) N/A	(d) 18 (3 per week for 6 weeks) (e) 25 min (f) 3–18 months	Pre and post: (1) TMAS, (2) current mood checklist, (3) sleep disturbance measures, (4) structured and social interview to assess maladjustment During: EMG	No differences between groups regarding treatment efficacy. 40 % Ss sig. ↓ anxiety levels to clinical significance	☐ [40 % of Ss showed sig. ↓ in anxiety scale scores: not sig. overall and not specific to BF]
Reed and Saslow (1980)	a) **GAD + test anxiety** (b) 27 (21 F, 6 M) (c) Mean age = 19 (d) No	(a) 3 groups; EMG BF, relaxation training alone no BF, no treatment control (b) Yes (c) N/A	(d) 8 (2 per week) (e) 20 min (f) Not specified	Pre and post: (1) AAT[a], (2) STAI, 3) Rotter locus of control scale During: forehead EMG	Both groups yielded sig. ↓ in anxiety scores, test-taking anxiety and general anxiety. No change was found in controls	⇑
Hurley (1980)	(a) **Chronic anxiety** (b) 60 (37 F, 23 M) (c) 18–29 Mean age = 19 (d) No	(a) 4 groups; EMG BF, hypnosis, trophotrophic treatment and control (b) Yes (c) N/A	(d) 8 (1 per week) (e) 20 min (f) Not specified	Pre and post: (1) IPAT[a] (2) anxiety scale, (3) ego strength scale, (4) I-E rotter scale During: frontalis EMG	Hypnosis group lowered anxiety levels more compared to EMG BF. Both hypnosis and EMG BF equally effective in ↑ ego strength	☐
Hoffman (1979)	(a) **Anxiety disorder** (b) N = 9, 4 = tension headache, 5 = anxiety disorder (c) 21–50 (d) Not specified	(a) Auditory EMG frontalis BF (b) N/A (c) N/A	(d) 10–35 (2 per week for 6 weeks, then 2–4 times a month over 2–8 months) (e) 30 min (f) 6 months	Pre and post: (1) psychiatric assessment (2) TMAS During: EMG, EEG (α, β, θ) HR, SC, BSR	All (bar 1) able to relax frontalis muscle. 3 tension and 1 anxiety patient clinically improved, also at follow-up. EMG BF found to be more beneficial for sig. ↓ tension headache versus anxiety	☐ [clinical improvement in 1 anxiety patient]

Table 2 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Lavallee et al. (1977)	(a) **Chronic anxiety** (b) 40 (c) 25–49 (d) No	(a) 4 groups; EMG and Diazepam, EMG and Diazepam placebo, EMG control (no feedback) and Diazepam, EMG control and Diazepam placebo (b) Yes (c) Double (relevant to Diazepam placebo)	(d) 8 (e) 30 min (f) 6 months	Pre and post:Anxiety measures: (1) Hamilton anxiety scale (2) IPAT[a] anxiety scale	All active treatment groups ↓ anxiety post treatment Diazepam (with or without BF) least effective in ↓ anxiety when comparing treatment groups. Sole BF group maintained sig. ↓ in anxiety at a 3 months follow-up, not evident in other treatment groups, although this was not maintained at 6 months follow up	⇑
Canter et al. (1975)	(a) **Anxiety disorder** (b) 28 (15 M, 13 F) (c) 19–48 Mean age = 34.6 (d) Medication free for study	(a) 2 groups; EMG BF, progressive relaxation with no feedback (b) Yes (c) N/A	(d) 10–25, 3–4 per wk (e) 20 min (f) Not specified	Pre and post: therapist anxiety rating/assessment, self-rating anxiety measures During: EMG, skin temp	Both groups yielded sig. ↓ in muscle tension. EMG BF showed to be more effective in ↓ anxiety based on therapist pre-to-post assessments and self-reports. No statistical analyses reported	□ [reduced symptoms were reported]
Hickling et al. (1986)	(a) **PTSD**[a] (b) 6 (M) (c) 33–60 (d) 3/6	(a) Frontalis EMG[a] relaxation (b) N/A (c) N/A (a) Desensitization	(d) 7–14 (over 8–16 weeks) (e) Not specified (f) 12–25 months (d) 48	Pre and post: (1) MMPI (2) STAI, (3) BDI (4) multidimensional health locus of control	Sig. ↓ in EMG with ↓ in subjective tension ratings. All 5 who completed STAI and BDI sig. ↓ in scores. MMPI scores ↓ in all Ss	⇑
Peniston (1986)	(a) **PTSD** (b) 16 (M) (c) 29–42 (d) 11/16	EMG BF and no BF (b) Yes (c) Single	(e) 30 min (f) 24 months	Pre and post: PTSD evaluative measures	Sig. ↓ in forehead muscle tension in BF group, little change in control group. Sig. less reports of recurring nightmaresand flash-backs from BF group at follow-up	⇑

Table 2 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Pharr and Coursey (1989)	(a) **Schizophrenia** (b) 30 (c) Under 65 (d) Yes	(a) 3 conditions: EMG ↓ BF, progressive relaxation, control (b) Yes (c) N/A	(d) 7 (e) 20 min (f) No	Pre: (1) Nurses observation scale for inpatient evaluation (NOSIE[a]) Post: (2) Finger Tapping Test (FTT) (3) Tension-Anxiety factor of the Profile of Mood States (POMS)[a]	BF group had sig.↓ EMG recordings. Sig. ↑ in FTT scores in BF group only. BF group sig. ↑ in social competence and interest scores on the POMS	⇑
Nigl and Jackson (1979)	(a) **Schizophrenia/ anxiety disorder** (b) 20 patients, 10 healthy controls (c) Not specified (d) Yes	(a) Muscle relaxation; frontalis and extensor muscle training (b) Yes (c) N/A	(d) 6 (e) treatment sessions: 90 min (f) No	Pre and post (patients only): (1) MMPI, (2) Ward Behavior Inventory, (3) BPRS	All 3 groups sig. ↓ muscle tension; schizophrenic and controls sig. greater ↓ than anxiety disorder group. Both patient groups sig. ↓ symptom scores and maladaptive behaviors	⇑
Acosta and Yamamoto (1978)	(a) **Schizophrenia/ anxiety disorde**r (b) N = 15; 6 Schizophrenia, 6 Anxiety, 3 Tension Headache patients (11 F, 4 M) (c) Mean age = 39 (d) Not specified	(a) Muscle relaxation; frontalis muscle training (b) N/A (c) N/A	(d) At least 10 (1 per week) (e) 15 min (f) No	Pre and post: (1) Kent intelligence scale (2) Clinical reports	All patient groups showed sig. ↓ in muscle tension. No sig. differences found between groups. No sig. clinical improvements reported in either patient group	□

[a] See Table 8

biofeedback as an intervention for anxiety disorders (including GAD, OCD, panic, phobia and PTSD), 14.3 % (n = 9) depression, 6.3 % (n = 4) symptoms and general functioning in schizophrenia patients, 6.3 % (n = 4) ASDs, 3.2 % (n = 2) dissociative disorders, and 1.6 % (n = 1) eating disorders. The mean number of patients per study was 33.3 (range 2–183). Mean biofeedback duration of each session per study was 27.4 min (range 12–60 min), with 15.6 (range 1–69) sessions of biofeedback carried out on average. In terms of medication, nine articles (14.3 %) did not specify this information, 29 (46.0 %) studies reported no medication, and 25 (39.7 %) reported patients were already receiving medication upon commencement, and during, the biofeedback intervention. Controlling for medication status in statistical analyses was rarely carried out and/or reported.

Clinical 'improvement' (as reported in Tables 1, 2, 3, 4, 5, 6, 7) required articles to report statistically significant ($p < .05$) symptom reduction in patients participating in an active biofeedback (BF) condition, where the following analyses were considered valid: (1) pre versus post BF comparisons, (2) post measure comparison of active BF versus control condition/group, (3) correlation between BF-regulated physiological change and reduction in

Table 3 Heart rate variability (HRV) and/or respiration biofeedback studies

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Beckham et al. (2013)	(a) **Anxiety in perinatal depression** (b) 15 (F) (c) 19–42 (d) Yes-various	(a) HRV ↑ (b) N/A (c) N/A	(d) 1 demo + individual practice across 2.2 days (e) varied-dependent on individual practice (f) 6 weeks	Pre, post, follow up: (1) STAI, (2) quality of life scale, (3) well- being scale,	STAI sig. ↓ pre-to-post. However, patients also received other treatments (medication, psychotherapy) not statistically disentangled. Results should be approached with caution	⇑
Kim et al.(2012)	(a) **Panic disorder** (b) 74 (51 F, 23 M) versus 30 HCs (c) Mean age = 41.9 (d) Not specified	(a) respiratory CO2 ↑ versus respiratory CO2 ↓ versus wait-list/WL control (b) Yes (c) N/A	(d) 5 (e) 10 min (f) 1 and 6 months	Pre and post: (1) PDSS[a], (2) end-tidal PCO_2 (partial pressure of CO2), (3) respiration rate Pre, 1/6 months: (1)–(3), (4) anxiety, (5) depression, (6) agoraphobia	Sig. ↓ in PDSS scores, also at 1 month follow up in both CO2 ↑ and CO2 ↓ BF types, compared to WL. Both BF-types sig. anxiety ↓ at 1 month follow up	⇑
Wollburg et al. (2011)	(a) **Chronic anxiety and panic disorder** (b) 45 PD, 39 chronic anxiety patients (c) Mean age = 44.25 (d) Yes, stabilized	(a) respiratory ↑ versus respiratory ↓ versus wait-list control (b) Yes (c) N/A	(d) 5 (e) 12 min (f) Pre-Post assessments, no further follow up	Pre and post: (1) BDI, (2) Anxiety Sensitivity Index (3) Anxiety symptom checklist	Chronic anxiety patients unable to ↑ CO2 in resp. ↓ BF. No sig. change in anxiety responses for either BF-type in either clinical group	□
Pop-Jordanova (2009)	(a) **Anxiety, OCD, somatoform problems, ADHD, and CD**[a] (b) 59 (c) Mean age = 11.98 (d) Not specified	(a) HRV-increase BF + healthy control group (N = 15) (b) N/A (c) N/A	(d) 15 (e) 16 min (f) No	(1) HRV HF + LF[a] spectra (2) Eysenck Personality Questionnaire (3) Clinical measures relevant to each disorder	BF was reported to have positive influences on clinical outcomes in anxiety + CD children, partially for OCD + somatoform disorders, and least effective for ADHD	□ ["positive influences" were reported; but no sig. changes in clinical scale data]
Reiner (2008)	(a) **Anxiety disorders**, e.g. GAD, phobia OCD, insomnia (b) 24 (12 F, 12 M) (c) 18–65	(a) RSA BF to ↑ HRV (adjunct to CBT[a]) (b) N/A (c) N/A (d) Yes	(d) 21 (e) 20 min (f) No	Pre and post: (1) STAI, (2) STAEI, (3) PSQI (sleep inventory), (4) HRV	Sig. ↓ in STAI and STAEI scores post intervention. ↑ in sleep quality (PSQI). 75 % reported ↓ in stress, 80 % ↑ relaxation, 46 % ↑ positive emotions. Some side effects (dizziness, drowsiness)	⇑
Meuret et al. (2001)	(a) **Panic disorder (PD)** (b) 4 (2 M, 2 F) (c) 40–44 (d) No	(a) Respiratory ↓ (b) N/A (c) N/A	(d) 5 over 4 weeks (e) treatment sessions: 80 min (f) 8 weeks	Pre, post and Follow up: (1) PDSS, (2) ASI[a],(3) STAIT-T, (4) BDI, (5) respiratory rate, (6) PCO2	All scores on PDSS, ASI BDI and STAIT-T ↓ in all 4 Ss. Resting levels ofPCO_2 ↑ and respiratory rates ↓	□ [no stats reported, although clinical improvements]

Table 3 continued

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	(d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Lande et al. (2010)	(a) **PTSD** (b) 39 (33 M, 6 F) (c) 18–41 (d) No	(a) 2 conditions; HRV↑ BF with TAU[a], or solely TAU (b) No (c) N/A	(d) 6 (2 per week) (e) 20 min (f) No	(1) PCL[a] military version (2) Zung Self-Rating Depression Scale	Sig. pre-to-post ↓ in PTSD (PCL) and depression (Zung) scores evident in both groups suggesting BF had similar therapeutic effects to TAU	⇑
Zucker et al. (2009)	(a) **PTSD** (b) 38 (21 M, 17 F) (c) 18–60 (d) No	(a) 2 conditions; N = 19 RSA[a] BF (↑ HRV), N = 19 PMR[a] (b) Yes (c) N/A	(d) 28 (e) 20 min (f) No	Pre and post: (1) PTSD checklist, (2) BDI, (3) ISI[a], (4) HRV amplitude (SDNN)	HRV/RSA BF sig. ↓ BDI scores compared to PMR. Both groups sig. ↓ PTSD symptoms post intervention	⇑
Siepmann et al. (2008)	(a) **Depression** (b) N = 38, 14 (13 F, 1 M) patients, 24 (12 M, 12 F) healthy controls (c) 18–47 Mean age = 28 (d) Yes	(a) All patients received RSA BF to ↑ HRV. Controls were randomly assigned to either RSA BF or an active control (no BF) (b) Yes – applicable to controls (c) N/A	(d) 6 (e) 25 min (f) 2 weeks	Pre and post: (1) BDI, (2) STAI-T, (3) VLF[a], LF[a], HF[a], LF/HF ratio of HRV spectra	Sig. ↓ BDI, STAI-T, HR, and ↑ in HRV in patient group post intervention and follow-up. No change in control group	⇑
Karavidas et al. (2007)	(a) **Depression** (b) 11 (7 F, 4 M) (c) 25–58 (d) Yes	(a) HRV ↑ BF (b) N/A (c) N/A	(d) 10 (e) 30 min (f) No	HAM-D[a] and BDI collected sessions 1, 4, 7 and 10	Patients were able to ↑ HRV. Sig. ↓ in HAM-D and BDI scores from session 4 onwards	⇑

[a] See Table 8

symptomatology. Forty-one (65.0 %) articles reported statistically significant reduction in targeted/specific symptomatology related to the biofeedback. A further 10 articles reported slight to moderate clinical amelioration that did not reach statistically significant levels, thus overall, 80.9 % (n = 51) of articles reported positive clinical effects from biofeedback treatment.

Non-randomized studies were included if specified categories relating to study design and methodology were fulfilled. The quality of the randomized controlled interventions was not always of a high standard. Of the 63 studies reviewed, 50 (79.4 %) included more than one experimental group; of these 32 (50.8 % of whole sample) were randomized. In articles where patients were randomly allocated to experimental conditions, the randomization procedure was rarely described. Six studies compared the effects of biofeedback treatment against traditional treatments, such as cognitive behaviour therapy (CBT), systematic desensitization (SysD), anxiety management training, or pharmacological medication, e.g. diazepam. Four studies compared differing clinical groups, four compared differing BF conditions; a sham/placebo biofeedback comparison was also classified within this category; nine utilized a no-treatment (or 'wait-list') control, and nine compared biofeedback with different complementary/alternative therapies; i.e. progressive muscle relaxation, meditation, and hypnosis. Another five articles utilized a healthy control comparison group. Finally, 13 interventions had several conditions, where biofeedback was compared to another treatment (or biofeedback condition), and a wait-list/no-treatment control/healthy control group.

EEG Biofeedback (Neurotherapy)

Twenty reviewed articles investigated EEG BF (neurotherapy), presented in Table 1. Fourteen of these (70.0 %)

Table 4 Heart rate (HR) biofeedback studies

References	Sample (a) Patient group (b) N (sex) (c) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Chernigovskaya et al. (1991)	(a) **Anxiety** (b) 30 versus 10 controls (c) 18–32 (d) Not specified	(a) 2 conditions; HR BF, 'no treatment' control (b) Yes (c) N/A	(d) 8–10 (alternate days) (e) 40 min (1 min rest period every 5 min) (f) No	Pre and post: (1) Slovak Academy of Sciences and Spielberger-Khanin Tests During: (2) HR, respiration, blood pressure	Anxiety BF group sig. ↓ HR, 'normalising' autonomic activity. Post psychological scales showed sig. ↓ in reactive anxiety	⇑
Rupert and Schroeder (1983)	(a) **Anxiety inpatients** (b) 24 (M) (c) 18–55 (d) Either not medicated or very low, stable dosage	(a) 3 groups; BF, no BF, adaptation group; (resting whilst HR recorded). In BF and no BF conditions, sessions 1and3 ↑ HR, Sessions 2and4 ↓ HR (b) Yes (c) N/A	(d) 4 (4–7 days) (e) 25 min (f) No	Pre: STAI During: Heart rate Post: STAI	BF effective for aiding HR ↑, but not HR ↓, in comparison to adaptation group. During final session HR changes + correlated to anxiety ↓ in BF group only. Suggesting more BF sessions may have been optimal	⇑
Nunes and Marks (1976) (replication study)	(a) **Phobia** (b) 10 (F) (c) 17–48 (d) No	(a) Heart Rate ↓ BF (b) N/A (c) N/A	(d) 1–4 (e) 30 min (f) No	Pre and post: (1) Subjective anxiety reports (2) Skin Conductance (3) Heart Rate	Replication of study below (Nunes, 1975). Similarly HR was better ↓ when given feedback, but no sig. anxiety ↓ from pre-to-post trial	□
Nunes and Marks (1975)	(a) **Phobia** (b) 10 (F) (c) 19–52 (d) No	(a) Heart Rate ↓ BF (b) N/A (c) N/A	(d) 2–4 (e) 30 min (f) No	Pre and post: (1) Subjective anxiety reports (2) Skin Conductance (3) Heart Rate	All Ss had sig. ↓ in anxiety from pre-to-post trial. All able to lower HR	⇑

[a] See Table 8

included a comparison treatment; either sham (placebo) biofeedback, a differing EEG parameter for feedback, another clinical intervention, or no treatment/wait-list control. Seven interventions (35.0 %) were randomized, four (20.0 %) non-randomized, and for the remaining 9 (45.0 %) randomization was not feasible. Mean number of sessions per study was 23.7 (range 5–69), with BF exposure lasting 28.7 min (range 14.6–60 min) on average per session. Five studies utilized α regulation BF for OCD (Glucek and Stroebel 1975; Mills and Solyom 1974), and anxiety (Sarkar et al. 1999; Plotkin and Rice 1981; Hardt and Kamiya 1978). Four of these five studies reported significant improvements in specific anxiety/OCD symptoms post exposure. Four studies investigated the therapeutic effects of α-θ regulation BF, for dissociative identity disorder (DID) (Manchester et al. 1998), PTSD (Peniston and Kulkosky 1991), depression in alcohol addicts (Saxby and

Table 5 Electrodermal (EDA) skin conductance biofeedback studies

References	Sample (a) Patient group (b) N (sex) (C) Age range (years) (d) Medicated (Y/N)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	(d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change in clinical indexes used
Schoenberg et al. (2012)	(a) **Depersonalization disorder** (b) 32 (24 M, 8 F) + 16 healthy controls (c) 19–59 (d) Yes (stabilized)	(a) SCL ↑: Real versus Sham BF (b) Yes (c) Single (patient)	(d) 8 (e) 20 min (f) 3 months	Pre, post and Follow-up: (1) CDS[a] (trait version) (2) DES (3) BAI (4) BDI After each BF session: CDS (state version)	Unlike healthy controls, patients could not ↑ SCL Instead patients sig. ↓ SCL leading to sig. ↓ in 'state' depersonalization symptoms, in the real-time group only, suggesting transient clinical change	□ [although sig. ↓ in symptoms via 'state' CDS, 'trait' CDS scores pre-to post were not sig]
Khanna et al. (2007)	(a) **Anxiety and stress** (b) 30 (F) (c) Not specified (d) Not specified	(a) 3 conditions: (1) GSR[a] BF, (2) PMR[a], (3) No treatment control (b) Yes (c) N/A	(d) 10 (e) 20 min (f) No	Pre and post; (1) Pulse rate (2) Comprehensive Anxiety Test Questionnaire	Both GSR BF and PMR elicited ↓ in pulse rates. However, only PMR sig. ↓ anxiety scores (not BF)	□
Pop-Jordanova (2000)	(a) **Anorexia nervosa and bulimia** (b) N = 27 (F) anorexia N = 76 (F) bulimia N = 35 healthy controls (c) Mean age = 14.25 (d) Yes	(a) EDA BF[a], along with nutritional menu and supportive therapy (b) N/A (c) N/A	(d) Not specified (e) Not specified (f) No	Pre and post; (1) MMPI (2) CMI[a] Neuroticism scale (3) General anxiety scale	Biofeedback was concluded to be an effective adjunctive treatment in eating disorders. Better receptivity to the intervention from girls with anorexia nervosa	□ [clinical improvement was reported, but no statistical analyses for clinical changes]

[a] See Table 8

Peniston 1995), and alongside α-asymmetry regulation, for depression (Baehr et al. 1997). All four articles reported significant clinical improvement from the BF intervention. An additional study implemented α-BF or θ-BF, reporting significant decreases in subjective anxiety from α-BF, and significant increases in perceptions of quality of life post θ-BF, with both conditions yielding significant clinical improvement in objectively rated anxiety (Vanathy et al. 1998). Sole α-asymmetry feedback was investigated in depressed patients (Choi et al. 2011), yielding significant reduction in symptoms according to standardized clinical inventories, compared to placebo psychotherapy. A further study alternating θ-decrease/β-increase neurofeedback showed significant symptom reduction in medication-resistant depressed patients, also generally maintained at 1-year follow-up. To note, the neurofeedback was least effective in the most severely depressed patients, with a 41 % failure rate within this group, compared to 7–14 % in less severely depressed patients (Walker and Lawson 2013). Two studies described using slow cortical potential (SCP) biofeedback, assessing specifically psychosocial and negative symptomatology in patients with schizophrenia (Schneider et al. 1992), and another study with depressed patients (Schneider et al. 1992). Neither study reported SCP BF to be effective in alleviating any clinical symptoms for either disorder. An advanced neurotherapy technique, quantified EEG (qEEG), successfully reduced OCD symptoms in sufferers (Hammond 2003). Finally, four articles utilized neurotherapy for autistic spectrum disorders (ASD). Significant improvements in autistic symptoms were emitted when using BF protocols based on individual qEEG (Coben and Padolsky 2007) and EEG (Jarusiewicz 2002) assessments. A third also used individual EEG

Table 6 Thermal biofeedback studies

References	Sample (a) Patient group (b) N (sex) (c) Age range (years)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	(d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change
Hawkins et al. (1980)	(a) **Reduction of anxiety in schizophrenics** (b) 40 (23 F, 17 M) (c) M = mean age 31, F = mean age 38 years (d) Yes; Inpatients	(a) 4 treatment groups; minimal treatment control, relaxation, Thermal BF, Thermal BF + relaxation (b) Yes (c) N/A	(d) 10 (5 per week) (e) 20 min (f) 12 months	(1) Finger temp. in BF groups Pre: (2) Hamilton Anxiety Scales Test (3) Brief Psychiatric Rating Scale (4) State-Trait Anxiety Inventory During: BF group; 20 min baseline, 20 min BF treatment Post: (2), (3) and (4) repeated	No sig. differences between groups post treatment for anxiety. Pre-post analysis showed sig. ↓ STAI and Hamilton anxiety scores in 10 Ss; although, not specific to the BF treatment group BF not necessarily more effective in ↓ tension compared to other treatments	□
Klee and Meyer (1981)	(a) **Depression** (b) 30 (c) Not specified (d) No	(a) 3 groups; non-depressed and 2 depressed groups; either depressed control or depressed BF[a] condition BF = skin temp. increase (b) No (c) N/A	(d) 1 (e) 45 min (f) No	Pre and post: BDI Pre-test BF: for depressed BF group only. Skin temperature During: 'Learned Helplessness' Task; measure of clinical severity	Depressed BF group did not show performance deficits evident in depressed controls (no BF) after biofeedback training. Alleviation of deficits indirect measure of clinical improvement	⇑

[a] See Table 8

profiles as the regulation signal (via 'Neuroguide'); despite yielding improvements in cognitive flexibility and executive functioning, no significant alleviation in specific ASD symptoms (SCQ) were evident (Kouijzer et al. 2013). Furthermore, increasing sensory motor ("mu") rhythm (SMR; 8–13 Hz) (Scolnick 2005), in patients with Asperger's Syndrome was also not statistically effective, although behavioral improvements were reported.

The majority of neurotherapy studies treated anxiety disorders. Differing cortical activity may reflect a biomarker for OCD, where patients yield significantly lower power in θ (2–4 Hz), β 1 (13–18 Hz), and β 2 (19–25 Hz) bandwidths (Kuskowski et al. 1993). Both interventions utilizing α regulation neurotherapy for the treatment of OCD (Glucek and Stroebel 1975; Mills and Solyom 1974) suggested that increasing α rhythm reduced OCD symptoms, specifically rumination and anxiety. Of all the biofeedback approaches, neurotherapy seems particularly promising for disorders where inducing particular states of conscious experience (through the alteration or regulation of cortical oscillatory activity) is a driving mechanism in alleviating symptomatology. Fourteen (70.0 %) studies reported statistically significant clinical amelioration following EEG BF exposure.

EMG Biofeedback

Eighteen articles outlined an EMG biofeedback protocol (see Table 2). Twelve studies (66.7 %) were randomized, two (11.1 %) non-randomized, and for the remaining four studies (22.2 %) randomization was immaterial due to clinical design. Mean number of biofeedback sessions conducted per intervention was 14.3 (range 6–48), lasting for 29.7 min (range 15–90 min) per session on average. One article omitted information pertaining to session duration of biofeedback. The majority of articles reported using an EMG biofeedback intervention for anxiety disorders (n = 15), with the remaining four EMG BF

Table 7 Multi-modal biofeedback studies

References	Sample (a) Patient group (b) N (Sex) (c) Age range (years)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	(d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? □ = no change, ⇑ = improvement sig. [$p < .05$] change
D'Amato (1996)	(a) **Anxiety** (b) 150 (c) School children (d) No	(a) 2 groups; BF, 'no treatment' control BF utilised skin temperature and EMG (b) Yes (c) N/A	(d) 12; 6 thermal, 6 EMG (over 12 weeks) (e) Not specified (f) No	Pre: IPAT Anxiety Scale Post: STAI state and trait	BF group yielded sig. ↓ in state and trait anxiety scores post-BF Concluded 2 types of BF more beneficial than just one modality	⇑
Sargunaraj et al. (1987)	(a) **Anxiety** (b) 21; n = 8 in each expt group, n = 5 in control group (c) Not specified (d) Not medicated (c) Not specified	(a) 2 expt conditions 1 relaxing with EMG, 1 relaxing with α. Control group: no contact with clinic (b) No	(d) 20 (e) 30 min (f) Assessment prior to and after 20 day activity period	(1) Pre and post: 3 consecutive day baseline measures of frontal EMG, SCL, % time α. Hamilton's Anxiety scale, Behaviour Disorder Checklist (BDC)	Both EMG and α relaxing groups changed physiology EMG BF yielded sig. ↓ in anxiety, unlike α BF. EMG and α BF showed greater ↓ in BDC than controls	⇑
Kappes (1983)	(a) **Anxiety disorder** (b) 37 (29 F, 8 M) (c) 18–66 Mean age = 32 (d) Not specified	(a) 4 groups; (1) relaxation training, temp and EMG BF, (2) temp and EMG BF, (3) temp followed by EMG BF (4) EMG BF followed by temp BF (b) Yes (c) N/A	(d) 16 (over 11 weeks) (e) 20 min (f) Not specified	Pre and post: (1) STAI 2) Symptom check list for anxiety During: finger skin temp and frontalis EMG	Sig. ↓ in state and trait anxiety, symptom checklist for anxiety and self-concept across the trial. Such improvement was sig, greater in Relaxation, temp and EMG BF, and EMG + temp BF groups, compared to remaining two treatment groups	⇑
Agnihotri et al. (2007)	(a) **GAD** (b) 45 (24 F, 21 M) (c) 18–30 (d) No	(a) 3 conditions; N = 15 per group; (1) Frontalis ↓ EMG BF, (2) α↑ BF, (3) No BF control group (b) Yes (c) N/A	(d) 12 (e) 25 min (f) 2 weeks	Pre and post: (1) GSR (2) State and Trait Anxiety Inventory	Both BF groups sig. ↓ state/trait anxiety scores and ↑ GSR (indication of relaxation), compared to control group (no BF). EMG BF showed to be sig. more effective at ↑ GSR and ↓ trait and state anxiety scores compared to EEG BF	⇑

Table 7 continued

References	Sample (a) Patient group (b) N (Sex) (c) Age range (years)	Design (a) Conditions (b) Randomized (Y/N) (c) Blind (single/double)	Design (d) No. of sessions (e) Duration of BF (per session) (f) Follow-up	Physiological and psychological measures used	Results	Symptom change? ☐ = no change, ⇑ = improvement sig. [$p < .05$] change
Rice et al. (1993)	(a) **GAD**[a] (b) 45 (23 F, 22 M) (c) Mean age = 27.4 (d) Not medicated	(a) 4 expt. groups; frontal EMG, EEG α ↑, α ↓, pseudo-meditation. 1 waiting list group (b) Yes (c) Single	(d) 8 (2 per week) (e) 20 min (f) 6 weeks	(1) Heart rate, forehead EMG, SCL, fingertip temp pre-post (2) Forehead EMG, HR, Occipital alpha measured during each session (3) Spielberger State-Trait Anxiety scale: Trait (4) Dahlstrom Welsh A scale (5) Attanasio Psychosomatic	All 4 expt. conditions ↓ STAI trait anxiety scores, and ↓ psychophysiological symptoms on psycho somatic scale. Sig. ↓ in HR α ↓ more responsive post-treatment. Sig. ↑ in EMG and ↓ on Welsh-A scale	⇑
Uhlmann and Froscher (2001)	(a) **Depression** (in refractory epilepsy) (b) 20 (c) Mean age = 38.5 (d) 70 % medicated	(a) 2 conditions; N = 10 per group Respiration feedback and SCP[a] feedback (b) Not specified (c) N/A	(d) 35 (e) SCP BF – 19.33 min (f) 6 months	Pre and post: (1) BDI (2) German version of Levenson's IPC[a] scale	Mean BDI scores sig. ↓ in all 20 patients in 6 month follow-up. Self-control scores in the IPC sig. ↑ after BF and further increased after 6 month follow-up	⇑

[a] See Table 8

interventions investigating either the treatment of anxiety in schizophrenic patients, or global functioning in schizophrenia. Sixteen articles describe training patients to lower frontalis muscle activity, including chronic anxiety (Lustman and Sowa 1983; Rupert et al. 1981; LeBoeuf and Lodge 1980; Raskin et al. 1980; Hurley 1980; Lavallee et al. 1977), GAD (Weinman et al. 1983; Lavellee et al. 1982; Reed and Saslow 1980; Hoffman 1979; Canter et al. 1975), panic disorder (Barlow et al. 1984), PTSD (Hickling et al. 1986), and schizophrenia (Pharr and Coursey 1989; Nigl and Jackson 1979; Acosta and Yamamoto 1978). One study trained patients with PTSD to increase and decrease muscle activity which significantly reduced reports of recurring nightmares and flashbacks (Peniston 1986). Overall, although it was the consensus for patients to significantly alter their muscle activity, this was not necessarily a reliable indicator that symptomatology would improve. That is, if muscle activity was not the key contributing factor to the disorder's primary symptoms, a reduction in muscle activity was beneficial for lowering general stress levels, but this alone would not necessarily target specific psychiatric symptoms. An above chance proportion of studies investigating anxiety disorders proposed EMG biofeedback to be valid therapeutic technique based on significant improvements in symptoms (11 out of 15 articles, 73.3 %). The three interventions implemented with schizophrenia patients reported significant change in anxiety (Nigl and Jackson 1979), and social functioning (Pharr and Coursey 1989), whilst the remaining study reported no significant change in symptoms and/or functioning. Overall, 12 studies compared EMG biofeedback to other treatments, including progressive relaxation (Pharr and Coursey 1989; Rupert et al. 1981; Leboeuf and Lodge 1980; Reed and Saslow 1980; Canter et al. 1975), stress inoculation (Lustman and Sowa 1983), meditation (Raskin et al. 1980), hypnosis (Hurley 1980), and diazepam medication (Lavallee et al. 1977). A further four comprised comparisons with another clinical group (Hoffman 1979), a wait-list control group (Barlow et al. 1984; Peniston 1986), or healthy controls (Nigl and Jackson 1979). The remaining two studies did not include a control. With the exception of one study, EMG biofeedback was shown to be more

Table 8 Acronym list for Tables 1, 2, 3, 4, 5, 6, 7

Acronym	
AAT	Alpert-haber achievement anxiety test
AMT	Anxiety management training
ASD	Autism spectrum disorder
ASI	Anxiety status inventory
AT	Alternative therapy
ATEC	Autism treatment evaluation checklist
BAI	Beck anxiety inventory
BDI	Beck depression inventory
BF	Biofeedback
BPRS	Brief Psychiatric Rating Scale
BRIEF	Behavior rating inventory of executive function
CD	Conduct disorder
CDS	Cambridge depersonalization scale
CMI	Cornell medical index
DBP	Diastolic blood pressure
DES	Dissociative Experiences Scale
FEAS	Functional Emotional Assessment Scale
GAD	General anxiety disorder
GADS	Gilliam Asperger's Disorder Scale
GAF	Global Assessment Scale
GARS	Gilliam Autism Rating Scale
GSR	Galvanic skin response
GQL	Global Quality of Life questionnaire
HF	High frequency—0.15–0.4 Hz (measure of HRV)
HRV	Heart rate variability
IPAT	Institute of personality and ability testing (Anxiety Scale)
IPC	(Levenson's) Internal—External Control Scale
ISI	Insomnia Severity Index
LF	Low frequency—0.04–0.15 Hz (measure of HRV)
MAACL	Multiple affect adjective check list
MCMI-II	Millon clinical multiaxial inventory
MMPI	Minnesota multiphasic personality inventory
MR	Muscle relaxation
NOSIE	Nurses Observation Scale for Inpatient Evaluation
OCD	Obsessive–compulsive disorder
PCL	Post-traumatic stress disorder checklist
PDSS	Panic Disorder Severity Scale
PIC-2	Personality inventory for children
PMR	Progressive muscle relaxation
POMS	Profile of Mood States questionnaire
PTSD	Post-traumatic stress disorder
RSA	Respiratory sinus arrhythmia
QEEG	Quantitative EEG
SBP	Systolic blood pressure
SCL	Skin conductance level
SCP	Slow cortical potentials
SCQ	Social communication questionnaire
SysD	Systematic desensitization
STABS	Suinn Test Anxiety Behavior Scale
STAI (S or T)	Spielberger state-trait anxiety inventory (State or Trait)
TAU	Treatment as usual
TAS	Test Anxiety Scale
TOVA	Test of variables of attention
TM	Transcendental meditation
TMAS	Taylor Manifest Anxiety Scale
VLF	Very Low Frequency—0.01–0.04 Hz (measure of HRV)
Symbol	Corresponding EEG bandwidth (approx)
θ	Theta (4–7.5 Hz)
α	Alpha (8–13 Hz)
β	Beta (13–40 Hz)
μ (SMR)	Mu (Sensory Motor Rhythm—SMR) (12–15 Hz)

effective in altering muscle tension levels compared to comparison conditions. Overall, 55.6 % (n = 10) of articles reported significant reduction in symptoms related to EMG biofeedback.

Heart Rate Variability (HRV) and/or Respiration Biofeedback

Ten studies utilized HRV/RSA or sole respiration biofeedback (see Table 3), for the treatment of panic disorder (Kim et al. 2012; Wollburg et al. 2011; Meuret et al. 2001), depression (Siepmann et al. 2008; Karavidas et al. 2007), anxiety in perinatal depression (Beckham et al. 2013), PTSD (Lande et al. 2010; Zucker et al. 2009), and a mixed anxiety sample including OCD, GAD, phobia and insomnia patients (Pop-Jordanova 2009; Reiner 2008). Seven studies (Beckham et al. 2013; Lande et al. 2010; Pop-Jordanova 2009; Zucker et al. 2009; Reiner 2008; Siepmann et al. 2008; Karavidas et al. 2007) used Respiratory Sinus Arrhythmia (RSA) biofeedback to alter HRV. HRV/RSA-BF protocols train slow paced breathing in order to increase the amplitude of RSA, a component of HRV. RSA refers to cyclical fluctuations in heart rate coincident with the respiratory cycle, whereby increases and decreases in HR occur during inhalation and exhalation, respectively (Song and Lehrer 2003). Of clinical relevance, HRV provides a measurement of autonomic and psychological homeostasis (Porges 2001).

Four studies reported a randomized design. On average, patients received 10.2 (range 1–28) sessions of biofeedback, lasting a mean of 25.8 min (range 10–80 min) per

session. Five (out of the 7) HRV/RSA biofeedback studies reported significant change in clinical indexes (Beckham et al. 2013; Zucker et al. 2009; Reiner 2008; Siepmann et al. 2008; Karavidas et al. 2007), although in one case biofeedback was administered within a perinatal inpatient unit whereby other treatments were also available and not controlled for (Beckham et al. 2013). Additionally, the mixed anxiety group (anxiety, OCD, somatoform disorder) study (Pop-Jordanova 2009) did report "positive influences" from the biofeedback, but no statistically significant results were reported. The sole respiration biofeedback study for chronic anxiety and panic disorder (PD) (Wollburg et al. 2011) compared respiration increase versus decrease, with no significant change in anxiety response in either clinical group. Moreover, patients with chronic anxiety were unable to increase CO_2 levels in the respiration decrease protocol, impeding investigation into the efficacy of the technique with these patients. The same authors replicated their 2011 study with a larger sample of PD patients, whereby both respiratory CO_2 increase and decrease significantly ameliorated panic disorder symptoms (PDSS scores), in addition to anxiety sensitivity scores, pre-to-post BF and at 1-month follow up (Kim et al. 2012). Meuret et al. (2001) study required PD patients to decrease their respiration rates, which proved effective in reducing experiences of panic. Overall, respiration/RSA-HRV biofeedback significantly improved clinical symptoms in seven (70.0 %) studies reviewed. The Wollburg et al. (2011) study suggests further investigation into sole respiration biofeedback for chronic anxiety is warranted, based on the fact patients could not consciously decrease their respiration rates. A further note, the quality of the HRV/RSA articles was particularly high; in general, study methodologies were reported in detail compared to other articles in the review.

Heart Rate (HR) Biofeedback

Four studies investigated heart rate (HR) biofeedback (see Table 4), for various anxiety disorders, including chronic anxiety (Chernigovskaya et al. 1991), anxiety in psychiatric inpatients (Rupert and Schroeder 1983), and phobia (Nunes and Marks 1975, 1976). Two studies used a randomized design, the remaining two exempt from randomization due to the intervention set-up. Mean number of sessions administered per study was 6.3 (range 4–10), with a mean biofeedback duration of 76.3 min (range 25–120 min) per session. Three studies aimed to decrease heart rate (HR) with significant symptom improvement in two of these studies (Chernigovskaya et al. 1991; Nunes and Marks 1975), and interestingly one study (Chernigovskaya et al. 1991) reported anxiety patients performed better than healthy controls in controlling their HR. Although, significant clinical improvements reported in Nunes and Marks (1975) phobia intervention were not replicated a year later (Nunes and Marks 1976) using the same protocol. The remaining study tested both increases and decreases in HR for anxiety in psychiatric inpatients, with success (Rupert and Schroeder 1983). Overall, three of the four studies reported significant symptom amelioration, whereby patients were able to consciously alter their HR, in turn, lowering experienced anxiety. Although, based on these few heterogeneous studies, no statements regarding efficacy can be made.

Electrodermal (EDA) Biofeedback

Along with thermal biofeedback, EDA biofeedback training was the least reported (n = 3) (see Table 5) in the reviewed articles. Randomization was not applicable for one study due to the intervention design. Data pertaining to number of biofeedback sessions and duration of biofeedback per session was omitted in one article.

Schoenberg et al. (2012) investigated the effects of eight sessions of skin conductance level (SCL) enhancement BF in patients with Depersonalization Disorder (DPD) randomly allocated to either a real-time or sham (placebo) group. Unexpectedly, the patients' baseline SCLs were significantly high, thus, marshalling further increase appeared difficult, suggesting the inclusion of an SCL-decrease protocol would have been apt from the outset. As such, SCL reduction was evident across the BF-trial, which coincided with significant reduction in 'state' depersonalization symptoms (recorded after each session of biofeedback) in the real-time BF group only, not the sham/placebo. Thus, a transient ameliorating effect on dissociative symptoms was evident, but not necessarily linked to the investigated SCL-increase protocol. Pop-Jordanova (2000) compared the efficacy of EDA biofeedback with other treatments, such as self-control desensitization, psychotherapy or a selected nutritional menu, and combinations of treatments, for eating disorders (anorexia nervosa and bulimia). EDA BF was reported to alleviate symptoms related to stress, anxiety and coping skills, intrinsically linked to the maladaptive eating behaviours, to a greater extent when used adjunct to another treatment. The article also reported the application of biofeedback treatment had a significantly greater positive effect on such symptomatology in the anorexia group compared to bulimics. Khanna et al. (2007) compared 10 sessions of 20 min of BF with progressive muscle relaxation (PMR) and a no-treatment group, for chronic anxiety and stress patients. Although significant changes in physiology were reported, only PMR yielded significant improvement in anxiety symptoms, not present post-BF. In sum, EDA biofeedback may be more effective for clinical symptoms if used in

conjunction with an additional treatment, or if the biofeedback is 'tailored' to physiological profiles due to the wide physiological variability within electrodermal measures. However, further studies are warranted to draw any conclusions regarding efficacy.

Thermal (temperature) Biofeedback

Two studies are included in this review, of which one was randomized (see Table 6). Mean number of biofeedback sessions per study was 5.5 (range 1–10), with sessions lasting on average 32.5 min (range 20–45 min). One study compared finger skin temperature enhancement BF against usual pharmacological treatment in schizophrenia inpatients for reducing anxiety (Hawkins et al. 1980), with no significant clinical change following BF exposure. The second study (Klee and Meyer 1981), trained depressed patients to increase skin temperature (it was not specified exactly where on the body), with positive outcomes in clinical measures compared to a wait-list control group. Due to the few studies utilizing thermal biofeedback, it is difficult to make any statements concerning its efficacy for psychiatric disorders at present.

Multi-Modal Biofeedback Interventions

Six articles reported using a multi-modal biofeedback approach; three combining EEG + EMG for anxiety disorders (Agnihotri et al. 2007; Rice et al. 1993; Sargunaraj et al. 1987), two incorporating EMG + thermal BF (finger temperature) for anxiety disorders (D'Amato 1996; Kappes 1983), and a fifth utilizing EEG + respiration BF for depression (Uhlmann and Froscher 2001). Four studies (66.7 %) were randomized, one non-randomized, and the sixth exempt due to study design. On average, 17.2 sessions (range 8–35) of biofeedback were administered, for an average duration of 22.9 min each session (range 19.3–30). All studies reported significant reduction in symptomatology, suggesting multi-modal biofeedback exposure increases the likelihood of a successful clinical outcome compared to one physiological biofeedback modality.

Discussion

This review was undertaken to establish how biofeedback interventions have been used to treat psychiatric disorders and gain preliminary insights into clinical utility. Specifically, (1) how many studies cited in the current literature have used a biofeedback paradigm; (2) which disorders have been treated; (3) what duration and intensity of biofeedback exposure has been utilized; and (4) was biofeedback reported as helpful in treating these psychiatric disorders?

Review Limitations

All articles were extracted by a sole researcher and their search methodology was not cross-checked by a second examiner, although searches were performed according to a strict procedure. The results were highly heterogeneous pertaining to the range of biofeedback types, disorder groups treated, and outcome measures used to quantify clinical change, i.e. more than one standardized clinical index exists per psychiatric disorder. Thus, it was not possible to quantify precisely the effectiveness of the intervention within the current literature, via meta-analyses for example. However, including only studies that yielded data suitable for meta-analyses would have greatly constrained the review, impeding the initial aims. Non-randomized and randomized controlled single and double blind treatment studies were all considered relevant, where other specified criteria (outlined) were met. Why condition allocation was assigned in place of randomization was not explained in the relevant articles, although this applied to just nine studies (14.3 %). Additionally, for 19 (30.2 %) included articles, randomization was not applicable because patients received the same treatment, and a comparison group included healthy controls, or less frequently, another clinical group. Victoria et al. (2004) argue that it is often impractical, and in some cases unethical, to use a randomized design for evaluating treatment interventions, although advocating treatments without an evidence base could also be considered unethical. Furthermore, pertinent information is frequently omitted in clinical trial reports despite the expectation that all relevant material is reported in such articles. For example, Hotopf et al. (1994) systematic review of clinical trials for depression demonstrated that only 1 out of 122 randomized interventions for anti-depressant medication specified the randomization procedure (Jüni et al. 2001). Thus, the rationale for carrying out the review in this way was to provide a useful reference for consultation by clinicians and researchers planning the design and implementation of forthcoming biofeedback interventions for psychiatric disorders, and for those who wish to improve the evidence base.

We did not assess the quality of included studies with a general evaluative scale for clinical trials, such as the *Cochrane* statement, *JADAD scale* (Jadad et al. 1996), *Quality of Reporting Meta-analyses* (QUOROM) (Moher et al. 1999), or *Consolidated Standards of Reporting Trials* (CONSORT) (Moher et al. 2001). Rather, we included studies which met specific criteria (outlined in the Methods), designed in alignment with a 5-level system for behavioral interventions (La Vaque et al. 2002) which classifies treatment procedures along a spectrum in ascending order; 'not empirically supported' (level 1), 'possibly efficacious' (2), 'probably efficacious' (3),

'efficacious' (4) and 'efficacious and specific' (5). Efficacious treatments (levels 4 + 5) must include a comparison group, randomization, clearly defined and specified inclusion criteria and outcome measures, comprehensive statistical analysis, and (for level 5) to show statistical superiority to an existing accepted treatment in at least two independent research settings. Whether studies reported positive or negative results indexed by clinical symptom change was not a factor considered for study quality.

Summary of Study Quality Assessment and Inclusion

1. The search strategy was comprehensive and bias-free but limited to articles published in English. Studies reporting the absence of therapeutic effects from biofeedback were included in review.
2. Study heterogeneity was considered and discussed but no statistical tests for this were applied.
3. A quality checklist, in alignment with an extant efficacy evaluation for behavioral interventions (La Vaque et al., 2002), was devised based on the review's objectives.
4. Effect sizes and sensitivity analyses were not applied because the data were too heterogeneous to carry out meta-analyses.

Limitations of the Use of Biofeedback in the Treatment of Psychiatric Disorders

Of the 63 studies reviewed; 50 (79.4 %) included a control group and 32 (50.8 %) were randomized and controlled. The randomization issue is perhaps less of a priority within the field because guidelines, such as the *Transparent Reporting of Evaluations with Non-Randomized Design (TREND)* statement (Des Jarlais, Lyle, Crepaz, and the TREND group 2004), have been developed to assess study quality where non-randomized designs may be necessary. Such as, cases where it may be ethically questionable to prolong access to treatment if patients are assigned to a wait-list or no-treatment control group, or where practicalities render a randomization procedure difficult to execute. An issue of greater pertinence relates to the proportion of studies (20.6 % in this review) failing to include a control group, consisting of either non-contingent sham (placebo) or an alternative treatment. Flaws of this nature in methodological design ultimately render such studies empirically weak, and do little to help biofeedback develop clinical prestige within psychiatric/psychological therapy practice.

Further limitations extend to the presence of biofeedback protocols for (1) differing physiological modalities, and (2) specific psychiatric disorders. The general lack in methodological benchmarks for standardized biofeedback applications are in part due to continual shifts in clinical procedures. For example, recent studies have started to investigate the therapeutic implications of real-time neuroimaging (rt-fMRI) before any precedents in methodological standards and protocols have been established for the existing physiological biofeedback techniques investigated, such as EMG, heart rate, electrodermal, temperature and respiration measures. Of all modalities, EEG biofeedback has addressed this issue to a greater extent, where some replicated biofeedback procedures are available. Monastra et al. (2005) have developed a qEEG protocol specifically for the treatment of Attention-Deficit/Hyperactivity Disorder (ADHD), widely associated with cortical under arousal (Lubar 1991) and distinct dominant slow-wave tonic EEG activity. As such, neurotherapy (EEG biofeedback) promises to be an effective and robust treatment pathway for ADHD. Furthermore, Peniston and Kulkosky's (1989, 1991; Saxby and Peniston 1995) α-θ protocol has shown to be beneficial in ameliorating symptom severity in a range of disorders, including Post-Traumatic Stress Disorder (PTSD), depression and the addictions. The α-θ protocol is considered particularly helpful for treating disorders characterized by negative perceptual affect, whereby the training aids conscious increases in α and θ alternately, inducing states of relaxation and contentment. Further large scale, robust controlled trials are awaited with interest.

Clinical Implications

Biofeedback may not be useful for disorders characterized by limited or low physiological responsivity, difficulties in recognizing physiological/affective states, or where physiological mechanisms are not centrally involved in the onset and perpetuation of symptoms (e.g. personality disorders). Albeit, whilst it does not appear logical to administer biofeedback treatments to the aforementioned disorder typologies, the potential efficacy of biofeedback upon 'opening' introspective mind–body channels within such patients which could then enhance patient-therapist interaction and/or personal insights, thus enacting nonlinear psychological benefits, has not been explored.

An important clinical consideration pertains to intervention dosage for psychiatric disorders. Referring to biofeedback modality, the review highlights EEG studies administered the most sessions of biofeedback ($\overline{X} = 21.0$, $\sigma = 12.5$), and heart rate biofeedback the least number ($\overline{X} = 5.3$, $\sigma = 2.5$), to yield clinical improvement. Whereas, temperature studies tended to administer the longest durations of biofeedback during treatment sessions ($\overline{X} = 32.5$ min, $\sigma = 17.7$); HRV/respiration ($\overline{X} = 19.1$ min, $\sigma = 6.5$), and

electrodermal ($\overline{X} = 20.0$ min, $\sigma = 0.0$), the briefest BF sessions. Consideration of the psychiatric disorder being targeted for treatment may also guide intervention dosage. On average, ASD interventions administered the most number of biofeedback sessions ($\overline{X} = 30.0$, $\sigma = 9.5$), perhaps suggesting this clinical group needed greater exposure to the intervention for significant improvements in symptomatology and functioning. The large anxiety sample (n = 43, 68.3 % of all reviewed articles) required fewer biofeedback sessions ($\overline{X} = 13.3$, $\sigma = 9.9$), suggesting biofeedback offers a relatively accessible and efficient treatment for anxiety-based disorders. Related to this point; biofeedback is an active treatment, where in order to gain optimal benefit patients must be willing to genuinely engage and interact with the technique. Further specific investigation as to whether certain psychiatric disorders have greater motivation to engage with the biofeedback process, and further train outside treatment sessions, would aid the optimal clinical development for the intervention within psychiatric contexts.

Looking at trends in the clinical use of biofeedback for psychiatric disorders: the review highlights contemporary feedback modalities include EEG, EDA and HRV, whilst all EMG, temperature, and HR biofeedback studies spanned the 1970s–1990s. This may be explained by continued advances in the mechanistic understanding in multi-levelled interplays of subcomponents of the central (CNS) and autonomic (ANS) nervous systems regulating electrocortical, electrodermal and HRV activity, alongside technical advances in biofeedback machinery and signal processing techniques to record and feedback such parameters. It could be postulated that the physiological correlates, or 'profiles', of many psychiatric disorders are complex, perhaps explaining why poly component, and decomposable, psychophysiological parameters such as EEG, EDA, and HRV have greater scope for development in the effective treatment of psychiatric disorders. For example, as a psychophysiological index HRV is mediated by a complex interplay of the CNS and ANS subsystems, reflecting physiological functioning (or dysfunction) in a range of psychiatric disorders (Yang et al. 2010), with implications in emotion and social regulation and adaptability (Porges 2001).

Synthesis

This review illustrates patients with psychiatric disorders can learn to consciously regulate their physiology modifying maladaptive physiological response associated with the disorder, enabling patients to experience positive states, such as relaxation and physiological stability via self-regulation. This can provide a strong facilitating factor in the efficacy of the technique whereby biofeedback may enhance a sense of achievement and self-control over one's physiology. This is particularly relevant for disorders where clinical symptoms may be maintained by maladaptive physiological mechanisms, i.e. heightened ANS activity can accentuate anxiety and stress experiences perpetuating clinical symptoms further; alternatively, depressed patients can train to elevate hypoactive autonomic basal states and/or response. Overall, training general medical practitioners and other health care professionals in biofeedback techniques could contribute towards achieving the aim set by the Lancet Global Mental Health Group (2007); to administer innovative and accessible cognitive and behavioral strategies for treating depressive, anxiety and other common mental disorders (CMDs).

Importantly, the review highlights the lack of standardization amongst biofeedback studies for psychiatric disorders. Templates and protocols exist, although not all studies are endeavouring to replicate previous studies or follow such guidelines. Additionally, the review emphasizes the lack of systematic communication of such studies; explanation of procedures pertaining to randomization or controlling for medication were predominantly omitted. These are pertinent issues within the biofeedback research community; without comprehensively explained methodologies a lack of replication of findings is inevitable. Furthermore, methods/results sections were inconsistent in structure and lacking empirical detail, resulting in the exclusion of several studies from the review. Within the parameters of the Efficacy Task Force system (La Vaque et al. 2002), our review findings suggest at present that only 50.8 % of the included biofeedback paradigms for psychiatric treatments met level 4 criteria based on the information reported within these articles. The remaining studies falling in the level 2/3 range; such studies were not randomized or necessarily even included a comparison control group. It must also be noted that level 1 studies were not included in this review because of exclusion criteria, potentially skewing our overall evaluation of biofeedback treatments used in psychiatric domains. It is difficult to disentangle whether this reflects the reporting of sub-par study designs, or the sub-par reporting of methodologically sound designs. An encompassing approach would be to propose guidelines for reporting the findings, in addition to standardized designs, for future biofeedback trials, in order for the technique to be comprehensively evaluated within psychiatric and psychological vocations as an accessible and valid therapeutic strategy.

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