Biofeedback and Anxiety Disorders: A Critical Review of EMG, EEG, and HRV Feedback

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Anxiety disorders are characterized by ongoing and situationally disproportionate fear and anxiety, and the associated significant distress and impairment of normal functioning (American Psychiatric Association, 2013). These disorders affect nearly one third of Americans in their lifetimes, indicating that a massive group of people stand to benefit from the development of effective and feasible treatments for anxiety symptoms (Valentiner, Fergus, Behar, & Conybeare, 2014). Possibly as a result of the rise in use of pharmaceutical treatments, research on exciting alternatives such as biofeedback to treat the symptoms of anxiety slowed in the early 1990s. However, sufferers generally seem open to the use of complementary and alternative therapies for anxiety and depression, especially those that have fewer side effects than pharmaceutical treatments (Kessler et al., 2001). Fortunately, there has been a resurgence in studying these treatments as researchers learn more about the patterns of neural activity and states of physiological functioning associated with anxiety disorders. Researchers are investigating the general efficacy of biofeedback for anxiety, as well as which types of biofeedback may be most effective for which types of symptoms and disorders. For example, EEG biofeedback (also referred to as neurotherapy) may be most effective for disorders such as generalized anxiety disorder in which conscious experience is a crucial component, and HRV (heart rate variability) biofeedback may be most effective for disorders such as panic disorder in which the individual experiences frequent and intense autonomic arousal (Schoenberg & David, 2014).

In this paper, I will review the available research on biofeedback treatments for anxiety disorders, a project I restrict to those anxiety disorders within the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) categorization; I focus on those disorders that have received enough attention in the field to draw at least preliminary conclusions (panic disorder, generalized anxiety disorder, and similar historical diagnoses). I will consider each popular type of biofeedback treatment with respect to specific anxiety disorders when possible, and discuss which symptoms the treatments impact and why certain

treatments may be most effective for certain symptoms or anxiety disorders. I will consider what factors contribute to or mitigate the efficacy of biofeedback treatments, and I will delve into the limitations of the research that has been conducted thus far, such as limited samples and ethical concerns, (Hammond, 2005; Reiner, 2008). Finally, I will discuss implications of the current findings and I will make recommendations for future research to advance the field. It is critical that biofeedback research continue to support the development of promising alternatives to pharmaceutical treatments, potentially providing opportunities for more accessible treatment with fewer side effects.

Theoretical Background

Evolution of the DSM
One challenge in reviewing this body of literature is that research depends on consistent functional definitions of the disorders studied, but the literature spans multiple editions of the DSM, which define and categorize anxiety disorders differentially over time. The relevant research really began in the late 1970s and early 1980s around the advent of the DSM-III, which split anxiety neurosis into panic disorder (characterized by periods of intense anxiety or panic/anxiety attacks) and generalized anxiety disorder (Barlow et al., 1984). This split was supported by evidence that the two types of disorders responded better to different types of medication (Klein, 1964; Rickels, 1993). The DSM-III-R redefined generalized anxiety disorder from a residual category into a disorder with its own specific criteria, with extensive worry at its core (Rice, Blanchard, & Purcell, 1993). The DSM-IV anxiety category included generalized anxiety disorder, panic disorder, agoraphobia without panic disorder, specific phobia, social phobia, obsessive-compulsive disorder, post-traumatic stress disorder, and acute stress disorder.

The DSM-5 split these disorders based on response to treatment and possible etiologies, and so the anxiety category includes only separation anxiety disorder, selective mutism, specific phobia, social anxiety disorder, panic disorder, agoraphobia, generalized anxiety disorder, and residual categories (American Psychiatric Association, 2013). This review will largely focus on research regarding panic disorder and generalized anxiety disorder as these disorders have the longest legacy of research and remain central to the field today. The early research on biofeedback and anxiety disorders tends to conflate panic disorder and generalized anxiety disorders, but I have attempted to tease these results apart when possible. There is minimal, if any, research on the effects of biofeedback on the majority of the other DSM-5 anxiety disorders. It is likely that this dearth of biofeedback research is due to satisfactory existing treatments, such as exposure

therapy for specific phobias, which is very effective in a short time frame with minimal resources (Valentiner et al., 2014).

Panic Disorder and Generalized Anxiety Disorder: Why Biofeedback Might Work

Both panic disorder and generalized anxiety disorder have physiological components, which may be targeted with a variety of biofeedback techniques. Panic disorder is characterized by panic attacks, which are brief periods of massive autonomic arousal including elevated heart rate. Generalized anxiety disorder is associated with persistent worry, but this cognitive state is associated with overall muscle tension and the physiological consequences of prolonged tension such as headaches and muscle soreness (Valentiner et al., 2014). Biofeedback targeting these physiological components may be administered alone or in conjunction with psychotherapy and/or medication.

Types of Biofeedback

Strong trends in popular types of biofeedback to study run parallel to the evolution of the DSM and the development of new technologies over time. The major therapeutic techniques have been EMG, EEG, and HRV feedback training.

EMG. The first major trend in biofeedback was centered on electromyography (EMG) training, which provides feedback—usually auditory (click or tone) feedback—reflecting the electrical activity of the muscles, an indicator of muscle tension. Much of the early EMG research suggested frontalis EMG feedback, in which sensors are placed on the forehead in the hope that this location is critical to the muscle tension of anxiety and that it correlates with general bodily tension, as a promising direction for developing anxiety treatments (Hoffman, 1979). The technique was originally developed to treat tension headaches, but Hoffman (1979) hypothesized that it may be a useful treatment for the somatic symptoms of anxiety neurosis. This sparked major interest in utilizing EMG to treat anxiety for at least the following decade. There are many limitations to this technique, which may explain its disappearance from the current literature, including mixed experimental results, prohibitive costs, and the development of new technologies.

EEG. Electroencephalography (EEG) feedback, also known as neurofeedback or neurotherapy, was originally developed as a relaxation technique related to meditation (Kamiya, 1969; Michael, Krishnaswamy, & Mohamed, 2005). EEG involves measuring the general patterns of activity of the brain and can be used as a diagnostic assistant (usually via quantitative EEG or qEEG) or feedback tool (Hammond, 2010, 2011). Feedback can be auditory or visual, even in the form of a computer game. In this way, participants learn to regulate specific

frequencies of cortical activity. As a feedback therapy, it has been used in many different psychological and medical disorders, including attention deficit-hyperactivity disorder, autism spectrum disorder, and substance use disorder (Myers & Young, 2012). It has also been used to treat the more cognitive components of anxiety disorders and may be more effective in those disorders in which the cognitive experience is central, such as generalized anxiety disorder (Schoenberg & David, 2014). Most practitioners claim there is little to no short- or long-term risk involved in neurofeedback, but some caution that to minimize risk it is critical to individualize treatment using qEEG because of the heterogeneity of EEG presentations of various disorders and comorbidities (Hammond, 2010; Walker, 2010).

HRV. Heart rate variability (HRV) was initially applied to panic disorder and related diagnoses because tachycardia (fast heart rate) and related autonomic activities are central to the definition of a panic attack (American Psychiatric Association, 2013). HRV is related to earlier measures of autonomic activity, such as the simple heart rate or blood pressure reading, but it allows for more sensitive interpretations that take into account contributions of both the sympathetic nervous system (SNS; activation, generally) and parasympathetic nervous system (PNS; inhibition, generally, Friedman & Thayer, 1998a, 1998b). To a lesser extent, HRV has also been studied in the context of generalized anxiety disorder, especially during periods of worry (Thayer, Friedman, & Borkovec, 1996).

Three major HRV components are typically analyzed in this body of research. The first is the quick, high frequency component (HF), which is related to respiration and is used as a vagal tone index. The mid-frequency component (MF) is associated with blood pressure regulation and is influenced by both the SNS and the PNS to varying degrees. Finally, the slow, low frequency component (LF) is influenced by temperature, vasomotor, hormonal, and metabolic regulation, and may be primarily regulated by the SNS (Friedman, 2007).

There is a large body of work arguing for various theories explaining the autonomic dysregulation in anxiety, but an in-depth analysis of this work is largely beyond the scope of this review. In brief, polyvagal theory and neurovisceral theory can be combined into an autonomic flexibility-neurovisceral integration model (Porges, 1992; Thayer & Lane, 2000). In this model, anxiety is associated with impaired homeodynamics (reactive flexibility critical to normal functioning) of the autonomic nervous system, as mediated by the central autonomic network (CAN) and a lack of normal inhibition of excessive emotional response at multiple levels (Friedman, 2007).

Two common forms of HRV biofeedback are resonant frequency HRV biofeedback, which teaches participants to optimize their breathing rate to the cardiovascular system, and HeartMath HRV biofeedback, which incorporates an

affective component to create a sort of physiological-cognitive-behavioral hybrid treatment (Hassett et al., 2007; Henriques, Keffer, Abrahamson, & Horst, 2011; Muench, 2008).

Biofeedback Evidence

EMG

EMG feedback research has been conducted with a diverse set of clinical and nonclinical populations, including those with DSM-II anxiety neurosis (Canter, Kondo, & Knott, 1975; Hoffman, 1979; Lavellée, Lamontagne, Annable, & Fontaine, 1982), psychiatric patients with chronic anxiety (Leboeuf & Lodge, 1980; Rupert, Dobbins, & Mathew, 1981; Scandrett, Bean, Breeden, & Powell, 1986), and individuals with subclinical but high anxiety (Lustman & Sowa, 1983; Reed & Saslow, 1980). Barlow et al. (1984) performed a critical comparison of the effects of EMG feedback as a component of therapy for individuals with DSM-III panic disorder or generalized anxiety disorder, but results were inconclusive and forms of therapy were gaining traction around this time. By the end of the 1980s, the field had progressed past EMG biofeedback. Research on the effects of EMG feedback is shown in Table 1.

Early evidence of EMG feedback was quite promising for anxious patients. In a pool of 28 anxious neurotics, half of which presented with panic attacks and half of which did not, EMG feedback and Jacobsenian Progressive Relaxation was associated with significant reduction in muscle tension for both groups, but the EMG-treated patients showed a greater and more rapid reduction. Further, a significantly greater proportion of the EMG group reported improvements in anxiety symptoms, a change which was corroborated by their primary therapists. Ultimately, the patients without panic attacks who received feedback training demonstrated reduced muscle tension to a lesser degree, possibly due to a lower baseline (Canter et al., 1975). Hoffman (1979) considered an extremely small sample of 4 adult patients with tension headache and 5 adult patients with anxiety neurosis. Overall, he found that EMG feedback was associated with reduced muscle tension in all patients, but only one of the patients with anxiety neurosis was denoted as "much improved" in terms of anxiety symptoms by clinical assessment – the others were either "unimproved" or "worse" following EMG treatment.

In these studies, EMG feedback training was seemingly effective in lowering frontalis muscle tension, but this was associated to varying degrees with the central symptoms of anxiety. When considering who may respond to such treatment, Lavellée et al. (1982) demonstrated that only 25% of participants reported decreased anxiety as a result of successful EMG training and that these

"responders" were more extraverted and less depressed. Further, individuals who respond to such treatment may be more susceptible to hypnosis, implying a critical cognitive component to EMG biofeedback (Rupert et al., 1981). Hoffman (1979) had posited that EMG feedback may not be an appropriate treatment for anxiety, but others attempted to dig deeper. EMG feedback seemed to improve some of the somatic symptoms associated with anxiety such as blood pressure in subclinical populations (Lustman & Sowa, 1983), as well as reducing heart rate, frequency of anxiety attacks, sleeping difficulties, and locomotor agitation in clinical populations (Barlow et al., 1984; Scandrett et al., 1986). It was unclear, however, to what degree this treatment exceeded the efficacy of more traditional treatments such as pharmaceuticals and relaxation instructions. In a sample of 40 adult outpatients suffering from chronic anxiety (likely conflating panic disorder and generalized anxiety disorder), EMG feedback resulted in reduction in frontalis muscle tension as well as anxiety symptoms and this effect was maintained at a 3 month (but not a 6 month) follow-up, whereas patients taking diazepam showed an initial reduction in tension, but this was not maintained at follow-up and was associated with negative side effects (Lavallée, Lamontagne, Pinard, Annable, & Tétreault, 1977). Some studies showed that both EMG feedback and relaxation instructions resulted in reduced trait anxiety in chronic anxiety patients (Rupert et al., 1981), even when panic disorder and generalized anxiety disorder were considered separately (Barlow et al., 1984). EMG feedback and relaxation also reduced test anxiety and general anxiety in highly anxious undergraduate students (Reed & Saslow, 1980), but another study showed that neither EMG feedback nor progressive relaxation had a significant effect on reducing anxiety in chronically anxious patients (Leboeuf & Lodge, 1980).

The theoretical foundation of EMG feedback seems especially sound for chronic anxiety and generalized anxiety disorder, as it could target the somatic manifestations of worry. However, the evidence is mixed at best regarding the relative efficacy of EMG biofeedback. In addition, this body of research is rife with experimental design limitations, including small sample sizes, lack of control groups, and inconsistencies in the EMG feedback itself (length of treatment, combination with other forms of therapy). On the whole, the research tended to exclude anxious participants who presented with potentially confounding comorbidities such as phobias, psychosis, depression, and obsessions, but many studies did not isolate anxious individuals who presented with persistent panic attacks, and when they did, they suffered from other design limitations such as small sample size. Anxiety is understood to be a complex disorder, so it is likely that biofeedback in conjunction with traditional cognitive-behavioral therapy would have a different effect than biofeedback alone, but in this research, EMG biofeedback was generally assessed as an isolated treatment, and if combined, it

was usually combined with relaxation techniques. Perhaps as a result of these limitations as well as the financial means and technological expertise required to utilize EMG feedback (Reed & Saslow, 1980), this particular realm of biofeedback research has since fallen out of fashion.

Table 1

Electromyography (EMG) Biofeedback (BF)

References	Sample	Design	Results
Barlow et al. (1984)	GAD (n=9) and PD (n=11)	Treatment (fEMG, progressive relaxation, + CBT over 14 weeks) vs. no treatment	Improvement in muscle tension and anxiety symptoms, both groups
Canter et al. (1975)	Anxiety neurosis (n=28)	EMG vs. progressive relaxation with no feedback	EMG and PMR → reduced muscle tension; EMG → reduced anxiety
Hoffman (1979)	Anxiety neurosis (n=9)	EMG, no control	Improvement in one anxiety patient
Lavallee et al. (1977)	Chronic anxiety (n=40)	EMG and Diazepam, EMG and placebo, EMG w/o feedback (control), EMG control and placebo	All groups reduced anxiety, Diazepam alone least effective; EMG improvements maintained 3 months
Lavallee et al. (1982)	Chronic anxiety (n=40)	fEMG, no control	Only 25% improved; Responders initially less depressed and more extraverted
Leboeuf and Lodge (1980)	Chronic anxiety (n=26)	fEMG vs. progressive relaxation	Both improved anxiety and muscle relaxation
Lustman and Sowa (1983)	Anxiety and stress (n=24)	EMG BF vs. stress inoculation vs. no treatment	Both treatments lowered blood pressure, reduced anxiety
Reed and Saslow (1980)	Test anxiety and general anxiety (n=27)	EMG vs. relaxation training vs. no treatment	Both treatment groups improved in test and general anxiety
Rupert et al. (1981)	Chronic anxiety (n=20)	EMG vs. relaxation vs. combination vs. no treatment	EMG improved trait anxiety
Scandrett et al. (1986)	Anxiety disorder (n=88)	fEMG vs. PMR vs. wait-list control	No sig. changes

EEG

Though EEG technology has been utilized in human psychology since 1924 (Haas, 2003), EEG feedback training has a relatively short history. Research has burgeoned in the last decade or so, with approximately 250 studies utilizing neurofeedback from 2008-2012 with promising but not definitive results (Myers & Young, 2012). This line of research corresponds to the NIMH Research Domain Criteria, encouraging investigation of the biological underpinnings of psychological processes and dysfunctions that result in pathology (Insel et al., 2010). While EEG is a relatively cheap research technique (compared with massive equipment such as fMRI), its cost has been rather prohibitive for use in therapy or counseling without evidence significant clinical results. The estimated cost is $10,000, including extensive training and equipment (Myers & Young, 2012). To examine and establish the effectiveness of EEG training in reducing anxiety, many researchers have focused on high trait-anxiety in non-clinical populations as this is a risk factor for developing anxiety disorders, but working with this population is more ethically acceptable as the research is not impeding patient access to treatment (Hardt & Kamiya, 1978; Logemann, Lansbergen, Van Os, Böcker, & Kenemans, 2010; Plotkin & Rice, 1981; Vendemia & Rodriguez, 2010; Wang et al., 2013). A survey of research on the effects of EEG biofeedback is shown in Table 2.

When considering clinical populations, researchers have looked at either diverse diagnoses accompanied by varying degrees of anxiety (Bhat, 2010; Michael et al., 2005; Saldanha, Chaudhury, Pawar, Ryali, & Srivastava, 2007) or generalized anxiety disorder (Rice et al., 1993). It is likely that researchers rarely considered panic disorder because the theoretical foundation of EEG feedback training is that it targets the cognitive components of a disorder. This is well-suited to generalized anxiety disorder and subclinical high trait-anxiety because this type of anxiety is characterized by cognitive worry and attentional bias, whereas panic disorder is less associated with such cognitions and is more associated with misattribution of and overreaction to physical experience (Chen et al., 2013).

In general, this research has focused on EEG alpha wave enhancement – a low frequency band of brain activity associated with a relaxed awake state, and associated with meditation and pleasant feelings through research in the 1960s (Kamiya, 1969). Higher frequency beta waves are associated with cognitive processing, and the lowest frequency theta waves are associated with sleep (Myers & Young, 2012). Hardt and Kamiya (1978) were among the first to demonstrate that when applied to a group of high trait-anxiety individuals, EEG alpha enhancement effectively reduces state anxiety whereas alpha suppression increases state anxiety. No effect was seen for low trait-anxiety individuals, which may account for the inconclusive results in previous studies. This research set a precedent for considering individual differences to understand the impact of EEG

feedback training. In another seminal study, Plotkin and Rice (1981) exposed how the placebo effect influences EEG feedback results in a nonclinical sample, albeit with an extremely small sample size (n=10). Both EEG alpha enhancement and EEG alpha suppression feedback produced a significant reduction in trait anxiety when participant expectation was manipulated to induce perceived success. In a follow-up to this study, Rice et al. (1993) demonstrated that perceived success plays a role in treatment, but does not account for the entire effect of EEG feedback. In adults meeting the DSM-III-R criteria for GAD or high subclinical levels of generalized anxiety, EEG alpha enhancement resulted in significant reduction in heart rate responsivity and EEG alpha suppression resulted in the opposite pattern. This study also demonstrated that fEMG and EEG alpha training all reduce trait anxiety and anxiety symptoms, implying that GAD may be treatable with a variety of biofeedback techniques (Rice et al., 1993).

In current EEG feedback research with clinical populations, it seems that studies have mostly, if not entirely, been conducted outside of the United States, potentially due to stringent ethical guidelines. Currently, there are generally effective treatments for anxiety, and EEG may be associated with risk, but some argue that it is unnecessary to conduct research on a potentially harmful new treatment. Perhaps for similar reasons, the international literature tends to consider the anxiety levels of psychiatric and medical patients with diverse diagnoses. Michael et al. (2005) considered cardiac patients at a Malaysian hospital with three different levels of anxiety: normal, mild/moderate, and severe. Many patients who completed initial screenings in the study dropped out, but that population agreed to participate in the follow-up testing and were considered a pseudo-control group. Overall, the participants demonstrated reduced anxiety symptoms over controls after EEG beta and sensory motor rhythms (another type of EEG wave) feedback training, but little change in actual EEG activity, implying a placebo effect (Michael et al., 2005).

Interestingly, two Indian studies investigated the differential effects of neurofeedback, pharmaceutical treatment, and a combination of the two in military veterans. Bhat (2010) compared the efficacy of EEG alpha enhancement relative to anxiolytics alone in a sample of 100 veterans with mild to severe anxiety and found that anxiety symptoms improved for both groups and that EEG feedback was particularly effective for generalized anxiety disorder and for females (possibly due to a greater motivational "buy-in" or compliance). In a similar population of individuals with neurotic and/or psychosomatic disorders, Saldanha et al. (2007) utilized multiple combined methods of biofeedback, psychoactive medication, and a combination of biofeedback and medication. The combination of biofeedback and medication seemed to be most effective in reducing anxiety symptoms, and as the medication was gradually withdrawn and effects maintained

over the course of treatment, this strategy was the most promising in certain cases (Saldanha et al., 2007). In a general sample of 40 Canadian aboriginal adults, a population in which psychopathology tends to be overrepresented, Hardt (2013) found that EEG alpha enhancement resulted in significant decreases in anxiety symptoms and negative affect as measured in four separate and validated personality tests. While these results seem promising, the general lack of controlled research regarding EEG feedback in treating anxiety is a major limitation in this field.

One of the newest directions of EEG feedback research is focused on training to reduce or normalize attentional biases that may be pathological in anxiety disorders in particular. The attentional bias theory states that high trait-anxious individuals tend to experience an exaggerated interpretation of threat-related stimuli (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Eysenck & Derakshan, 1997). While Logemann et al. (2010) failed to show an effect of EEG feedback on attention in non-clinical inattentive and impulsive undergrads, this may have been due to design limitations such as a small sample size and ceasing the study early, which was due to ethical considerations and the lack of progress mid-way through the planned timeline. Vendemia and Rodriguez (2010) discovered differential EEG patterns correlated to coping style (high-anxious vs. low-anxious vs. repressors), particularly that the high-anxious group showed more overall activity throughout alpha and beta bands in response to the emotional Stroop task. Taking this research one step further, Wang et al. (2013) considered EEG feedback and attentional biases in two groups of Chinese female undergraduates, a high trait-anxiety group (HTA) and a nonanxious group (NA). First, in an emotional Stroop task, the HTA group demonstrated a significant attentional bias toward negative words manifested in a slower reaction time, associated with a longer P300 latency (measured with event-related potential [ERP] recording). In a second experiment, EEG alpha enhancement was compared with sham-biofeedback in the HTA group. Critically, EEG feedback significantly reduced trait anxiety relative to sham-biofeedback, alpha amplitudes were significantly enhanced, and P300 latencies for negative words were shortened (Wang et al., 2013). This is strong evidence that EEG alpha enhancement can reduce the negative attentional biases commonly found in anxiety patients, and that the mechanism by which neurofeedback affects anxiety may work at the level of attentional bias. This promising result is consistent with the theory that neurofeedback is dependent on cognitive mechanisms and affects the cognitive aspect of psychopathology, and may have major implications for the development of a neurofeedback treatment for anxiety targeting attentional bias.

Table 2

Electroencephalography (EEG) Biofeedback (BF)

References	Sample	Design	Results
Bhat (2010)	Indian veterans, mild to severe anxiety (n=100)	EEG alpha enhancement vs. anxiolytics	Anxiety symptoms improved in both groups; EEG-BF better for GAD and females
Chen et al. (2013)	GAD (n=42), PD (n=34), and controls (n=46)	GAD vs. PD vs. Controls in emotional Stroop task with GD-related and PD-related words	GAD and PD slower to respond; Differential pattern of attentional bias, no evidence to suggest unique PD bias
Hardt and Kamiya (1978)	Non-clinical male undergrads, low vs. high anxiety (n=16)	EEG alpha enhancement vs. EEG alpha suppression, no control	High anxiety group – alpha enhancement reduced state anxiety, alpha suppression increased state anxiety
Hardt (2013)	Canadian aboriginal adults (n=40)	EEG alpha enhancement, no control; Personality measures	Statistically significant decreases in anxiety and negative affect with 4 personality tests
Logemann et al. (2010)	Non-clinical high impulsivity/inattention (n=27)	EEG vs. sham feedback	No effect, ended early due to ethical guidelines
Michael et al. (2005)	Malaysian cardiac patients, normal to severe anxiety (n=38); many dropped out	EEG beta/sensory motor rhythms training vs. dropouts (pseudocontrol)	Reduced anxiety vs. dropouts; Minimal EEG changes, implies placebo effect
Plotkin and Rice (1981)	Non-clinical male undergrads, high trait anxiety (n=10); many dropped out	EEG alpha enhancement vs. suppression; expectation of success consistent	Both groups – significant reduction in trait anxiety; perceived success most important
Rice et al. (1993)	GAD or high subclinical anxiety (n=45)	fEMG vs. EEG alpha enhancement vs. EEG alpha suppression vs. pseudomeditation control	All treatments → sig. reduction in trait anxiety and symptoms; only EEG alpha increase → reduction in HR

			reactivity
Saldanha et al. (2007)	Indian, neurotic and psychosomatic disorders (n=78)	Psychoactive drugs vs. Biofeedback (multiple method, including EEG) vs. combination	All groups – reduced anxiety symptoms; Combination worked best, pharmaceuticals gradually withdrawn
Vendemia and Rodriguez (2010)	Non-clinical female undergrads, repressive, low-anxious, and high-anxious coping styles (n=49)	Correlation – EEG differences related to coping styles in emotional Stroop task	High-anxious participants – more alpha and beta power than low-anxious; More attentional bias to negative stimuli
Wang et al. (2013)	Chinese non-clinical female undergrads, high trait-anxiety (HTA) and nonanxious (n=45)	EEG alpha enhancement vs. sham biofeedback in emotional Stroop task	HTA attentional bias confirmed, associated with longer P300 latency; Feedback → reduced attentional bias, anxiety scores, and P300 latency

HRV

As mentioned above, the use of heart rate variability (HRV) feedback for anxiety disorders is rooted in complex theory regarding the typical functioning of the autonomic nervous system and how it becomes dysfunctional. Generally, the models predict that anxiety is associated with a reduced range of HRV and a low vagal tone (Friedman, 2007). Low vagal tone is also associated with low responsivity to the environment as well as anxiety and antisocial behavior in adolescents (Beauchaine, 2001; Mezzacappa et al., 1997). Research investigating the effect of HRV biofeedback is shown in Table 3.

HRV biomarkers. Evidence suggests that panic disorder is associated with a low vagal tone and high SNS cardiac control, which fits with the diagnostic picture that individuals with panic disorder have less ability to inhibit the fight-or-flight interaction which leads to a panic attack (Valentiner et al., 2014). HRV has also been used to differentiate the autonomic profiles of individuals with panic disorder from individuals with blood phobia and nonanxious controls – "panickers" presented with the highest heart rate and the lowest heart rate variability, whereas blood phobics' HRV was relatively more vagally mediated. This particular study utilized a stress task and a fear task to produce autonomic reactions (Friedman & Thayer, 1998a).

There is some evidence to suggest that HRV markers can also differentiate anxiety disorders without panic. In a seminal study by Thayer et al. (1996), individuals with generalized anxiety disorder presented with lower vagal activity

(shorter cardiac interbeat intervals [IBIs] and lower HF spectral power) than controls at rest. When individuals with GAD were compared with controls in a laboratory-induced worry task, both groups demonstrated lower vagal activity, implying that this effect may be representative of the physiological correlate of persistent worry associated with anxiety (Thayer et al., 1996). In a study by Alvares et al. (2013), social anxiety disorder was found to be associated with reduced HRV compared to controls with a relatively large sample size (n=106). These results imply that low resting HRV may be a useful biomarker for identifying individuals with low approach-related motivation and a potentially overactive behavioral inhibition system (Alvares et al., 2013).

HRV biofeedback. Despite the significant body of evidence to support the role of HRV in pathological anxiety, very little research examining HRV biofeedback with anxious populations exists. There is some preliminary HRV biofeedback research with medical populations (e.g. cardiac diseases), and some with clinically anxious populations, but most anxiety-related HRV biofeedback training has been conducted with nonclinical populations (Wheat & Larkin, 2010).

In terms of the resonant frequency HRV biofeedback, the StressEraser device is popularly used to teach participants to breathe at the optimal rate (5-6 breaths per minute) in the face of a stressor (Muench, 2008; Prinsloo, Derman, Lambert, & Rauch, 2013; Reiner, 2008; Sherlin, Gevirtz, Wyckoff, & Muench, 2009). These results are difficult to tease apart because of the small sample sizes and general financial support from the company producing the device, which is motivated to support research that promotes device sales. However, the device seems to show some promise, and a small independent pilot has shown that breathing at a rate of 4-6 breaths per minute may increase HRV (Song & Lehrer, 2003). As a researcher for the StressEraser company, Reiner (2008) demonstrated that the StressEraser generally decreases perceived stress and anxiety in a group of participants with anxiety disorders. Decrements in anxiety related to degree of compliance in the use of the device at home in conjunction with psychotherapy (Reiner, 2008). In early independent research with the device, Sherlin et al. (2009) demonstrated that in only one short session of biofeedback, non-clinical participants with perceived stress demonstrated a decrease in heart rate over controls who did not experience the biofeedback. However, while both groups experienced a reduction in perceived stress, the decrease in the biofeedback group was slightly stronger (Sherlin et al., 2009). In a follow-up study, Prinsloo et al. (2013) further demonstrated that a short intervention with the StressEraser in a small sample of male adults with work-related stress reduced state anxiety and increased mindfulness, energized positive feelings, and basic relaxation over a control device. Overall, the StressEraser seems to be a promising device, but larger independent and controlled studies must be conducted to determine the validity of

effects observed. It is likely that the optimal use of this device is in conjunction with psychotherapy to address the cognitive components of anxiety.

HRV biofeedback has also been investigated in the context of non-clinical performance anxiety. A well-designed and controlled study by Wells, Outhred, Heathers, Quintana, and Kemp (2012) induced performance anxiety in adult musicians and compared the effects of a slow breathing intervention, a slow breathing with biofeedback intervention, and a no-intervention control. Groups who received either intervention appeared to control physiological arousal, but there was no significantly different effect on arousal or self-reported anxiety. However, individuals with baseline higher anxiety in the interventions did report lower levels of state anxiety when compared with control (Wells et al., 2012). These results imply that general relaxation techniques may be sufficient to reduce autonomic arousal, but this should not be generalized to clinical anxiety or even high trait-anxiety individuals, who may benefit further from biofeedback as there is more room for improvement. Gruzelier, Thompson, Redding, Brandt, and Steffert (2013) compared EEG alpha/theta biofeedback and HRV Freeze-Framer biofeedback along with two controls to investigate the impact of these interventions on state anxiety and creativity in dance students. Freeze-Framer biofeedback plots the heart rate curve on a screen, and provides visual feedback to encourage optimal breathing. Only the HRV biofeedback reduced self-reported anxiety among the dancers, and HRV was increased through training (Gruzelier et al., 2013). Utilizing HRV Freeze-Framer biofeedback with the addition of a cognitive affect-shifting component, Henriques, Keffer, Abrahamson, and Horst (2011) were able to detect decreases in anxiety, but this was not associated with an increase in "psychophysiological coherence." HeartMath LLC produces Freeze-Framer, and while coherence is the goal in HeartMath terminology, it may not truly capture the effect of this HRV biofeedback. Perhaps if Henriques et al. had access to actual HRV measures, a similar effect to that in Gruzelier et al. (2013) would have been observed and a reduction in anxiety would have been correlated with an increase in HRV.

Related biofeedback. There has also been research utilizing biofeedback with components of HRV such as heart rate and breathing. These studies generally do not refer to HRV explicitly, but may have important implications for HRV feedback research.

Heart rate. Heart rate is clearly an important part of HRV, but it is not often targeted for HRV biofeedback in favor of slow breathing training. However, heart rate may be an important avenue of investigation of biofeedback for anxiety disorders. Rupert and Schroeder (1983) posited that if anxious veterans could learn to control their heart rates, they might be able to reduce their anxiety levels. Those in the heart rate biofeedback group as opposed to the controls were able to non-

significantly reduce anxiety levels, a trend which was correlated with a reduction in heart rate (Rupert & Schroeder, 1983). While this was a clinical group, individual diagnoses were not analyzed, and it is likely that many of these individuals presented with post-traumatic stress disorder, as they were a veteran population.

Thirty years later, utilizing heart rate feedback as an intervention has regained interest in the field. Houser et al. (2013) demonstrated in a rural United States population of clinically anxious adults that utilizing a heart rate monitor can improve state anxiety and self-efficacy. This is a critical pilot study which justifies further research on heart rate biofeedback because it is extremely cost effective, and may be ideal for populations that are less able to travel to psychotherapy and would benefit from a self-directed intervention (Houser et al., 2013). Again, however, the study did not differentially analyze individual diagnoses, so it is unclear which anxiety disorders for which this treatment may be optimal. In a non-clinical population, Peira, Fredrickson, and Pourtois (2013) demonstrated that heart rate biofeedback could temper an individual's physiological reactivity in response to negative valence images, regardless of individual differences in baseline anxiety level or cognitive self-regulation methods. This biofeedback technique may be a helpful addition to cognitive approaches to reduce the attentional bias to negative stimuli seen in anxious individuals (Peira, Fredrikson, & Pourtois, 2013). In a critical follow-up study, Peira, Pourtois, and Fredrickson (2013) investigated whether heart rate regulation learned in biofeedback training can transfer to other situations without the biofeedback support. Non-clinical participants responded well to heart rate biofeedback – they were able to decrease heart rate in training – and maintained regulatory improvements in response to negative valence images – decreased heart rate reactivity after training compared with pre-training assessment (Peira, Pourtois, & Fredrikson, 2013). This research should be extended to clinical populations to assess efficacy and additional research should consider the mechanism of this type of intervention effect.

Breathing. Finally, select research on breathing training and respiratory biofeedback may be related to HRV biofeedback (which focuses on respiration), although these studies do not discuss HRV explicitly. These studies tend to focus on panic disorder (PD) because panic attacks may be produced and maintained by reduced carbon dioxide in the blood as a result of hyperventilation. In a small sample of 4 individuals with panic disorder, Meuret, Wilhelm, and Roth (2001) utilized a respiratory biofeedback device to increase carbon dioxide and reduce respiratory rate with breathing exercises. Though this study is not controlled, and respiratory biofeedback is confounded with respiratory training, patients tended to exhibit reduced panic symptoms and increased blood carbon dioxide after intervention (Meuret et al., 2001). Wolburg, Roth, and Kim (2011) provided new

evidence to support the use of breathing training to lower blood carbon dioxide, as participants with anxiety disorders were able to effectively lower or elevate blood carbon dioxide in response to a tailored treatment. This study found that PD may have specific respiratory abnormalities as previously observed, but results failed to confirm that individuals with PD have greater reactivity and slower recovery from hypo- or hyperventilation (Wollburg et al., 2011). Thus, research on respiratory biofeedback for anxiety, in particular panic disorder, has resulted in mixed evidence at best.

Table 3

Heart Rate Variability (HRV) and related biofeedback (BF)

References	Sample	Design	Results
Gruzelier et al. (2013)	Non-clinical dance students (n=64)	EEG BF alpha/theta vs. HRV BF vs. Choreology (dance) class vs. no-training control	No impact on dance performance; HRV BF → reduced anxiety
Henriques et al. (2011)	Non-clinical undergrads, high anxiety, interested in biofeedback (n=9, n=35)	a) Study 1 – computer-based HRV, no control b) Study 2 – computer-based HRV, delayed treatment control	Overall decreases in anxiety, no sig. change to mood, general well-being, or coherence scores
Houser et al. (2013)	Anxiety disorder, rural U.S. (n=53)	Heart rate monitor feedback (HRM) vs. control	HRM group improved sig. more in state anxiety and self-efficacy than control
Meuret et al. (2001)	Panic disorder (n=4)	Respiratory biofeedback, initial results so no control	Blood carbon dioxide increased during therapy and was maintained; Reduced panic symptoms
Peira et al. (2013a)	Non-clinical undergrads (n=23)	True heart rate (HR) BF (screen color changes) vs. false HR BF; HR regulation task vs. HR monitoring task; Viewing images of differential valence	In regulation task, true and false HR BF → improved regulation in response to neutral images; ONLY true HR BF → improved regulation in response to negative images
Peira et al. (2013b)	Non-clinical undergrads (n=20)	Viewing images of differential valence without feedback pre- and post-intervention; Heart rate (HR) BF (screen color	HR BF improves HR regulation and this improvement extends to image viewing situation (novel

		changes) w/o images	emotional context)
Prinsloo et al. (2013)	Non-clinical adult males, work-related stress (n=18)	StressEraser HRV BF vs. Control device; modified Stroop to induce stress	BF → large decrease in state anxiety; Control → moderate decrease in state anxiety
Reiner (2008)	Anxiety disorder, receiving concurrent psychotherapy (n=24)	StressEraser HRV BF, no control, pilot; Use as adjunct to therapy	Majority report reduced stress, increased relaxation and positive affect; Significant increase in effect with increased compliance
Rupert and Schroeder (1983)	Anxiety disorder, veterans hospital (n=24)	Heart rate (HR) training + BF vs. HR training control group vs. adaptation (resting) control group	HR BF did not significantly reduce HR or anxiety levels, but changes in HR and anxiety were positively related in the BF group
Sherlin et al. (2009)	Non-clinical adults, perceived stress (n=43)	StressEraser HRV BF vs. Control device	BF → significantly reduced heart rate; Non-sig. reduction in anxiety
Wells et al. (2012)	Non-clinical, adult musicians (n=46)	Computer-based HRV BF as an intervention for induced musical performance anxiety vs. slow breathing control vs. no-intervention control	Both slow breathing and HRV BF improved HRV in induced-anxiety situation; Significant reduction in anxiety only for high trait-anxiety individuals for both interventions
Wollburg et al. (2011)	Panic disorder (n=45), episodic anxiety (n=39), and nonanxious controls (n=20)	BF-assisted breathing training treatment to raise blood carbon dioxide vs. BF-assisted breathing training treatment to lower blood carbon dioxide vs. wait-list control; Hyperventilation and hypoventilation test	PD – higher baseline respiration; Lowering therapy → all groups lowered carbon dioxide, but did not effect ventilation test performance; Did not see differences in PD reactivity and recovery from respiratory challenges

Limitations of Biofeedback Research

There are significant limitations throughout the biofeedback literature reviewed here, many of which have already been mentioned. Far too many studies used moderate to very small sample sizes, greatly limiting their sensitivity to the phenomena of interest. This is especially true in the EMG literature, although perhaps it is because the EMG literature mostly features clinical populations that may be more difficult to access. Other studies that were able to achieve larger sample sizes were in turn limited in the scope of their conclusions because they often used non-clinical or mixed-diagnosis populations, such as many of the EEG studies. Research has shown that anxiety disorders are often comorbid with other mental disorders such as depression, and while a few studies took pains to exclude anxious patients with certain other conditions, it is likely that comorbidity limited the strength of the results observed. Even when the literature attempted to use a clearly diagnosed clinical population, the criteria and even the categorization of anxiety disorders changed over time such that it is difficult to compare research on DSM-II, DSM-III, DSM-IV and DSM-5 disorders in the event that participants may be re-classified across DSM editions. Researchers with long legacies in the field are essentially forced to use different terminology and diagnostic criteria across time, hindering their ability to draw strong conclusions.

In addition, many studies did not include a control group. This can be explained by ethical concerns to some degree, but other studies utilized the wait list control to strengthen their validity. One of the most blatant limitations was the inconsistency in biofeedback application (frequency, total time, modality of feedback) and existence or latency of follow-up assessment. To truly contribute to the biofeedback literature, researchers should determine a standard application of each biofeedback technique. With the variable follow-up assessment, it is unclear if any improvements seen are sustained or what kind of maintenance might be necessary. On the whole, there are severe limitations to the biofeedback research reviewed here.

Summary and Conclusions

Implications

There are certainly many reasons why pharmaceutical treatments for anxiety disorders are so popular with patients and clinicians today, but there are significant drawbacks that should motivate researchers to develop better treatments. Medication alone may seem like an easy solution to a complex disorder, but

generally has limited efficacy and many side effects. Medication is only a treatment as long as the patient is taking the medication; the patient is not learning new skills. Medication should be used in conjunction with skill-building therapies so that the patient can be weaned off of the drug treatment. It is especially important to promote such combined treatment because patients who stand to benefit the most from combined or alternative treatment (those with mild symptoms) tend to be treated with medication alone (Heinen, 2014). Further, individual differences and heterogeneity in presentation of disorders may imply that treatments can be tailored to the individual's particular symptom complex with cognitive, behavioral, and physiological symptoms. Therapies with multiple components would most likely work in this way, and biofeedback may serve as an important component for individuals with somatic or physiological symptoms. Overall, further research on biofeedback will contribute to the transition away from the disease model of psychopathology with purely pharmaceutical treatment to the complex systems learning model in which the individual patient may receive unique skill-building therapies targeted to his or her particular needs.

Recommendations for Future Research

EMG. As EMG research seems to be largely ineffective and inconvenient, I do not believe research should continue on EMG as a treatment for anxiety disorders.

EEG. Researchers may continue to study the use of EEG feedback to increase alpha wave activity and reduce anxiety symptoms, but it is critical that this research be conducted with representative populations and with stringent control. A failure of the previous research to show consistent results may be due to weak experimental design, and so this research should not be continued unless researchers are able to implement a rigorous protocol. The future of EEG research seems to be grounded in attentional bias theory, and as such, research should be conducted to further investigate the capacity of EEG feedback to alter attentional biases of those with clinical anxiety. In particular, the Wang et al. (2013) paradigm, previously used to treat Chinese undergraduate students with subclinical anxiety, should be replicated with a clinical population to determine if such a treatment could benefit individuals with disorder-level anxiety and help treat the negative attentional bias of anxiety disorders.

Additional research is also needed to determine the relative efficacy of complementary EEG feedback over cognitive behavioral therapies alone in terms of cost-effectiveness, time required to implement, and maintenance of effects over time. It is possible that EEG feedback effects, even if strong in the short term, will decay over time and individuals may need to regularly visit a trained professional

for "touch ups." This possibility should also be considered in studying the feasibility of EEG feedback as a treatment for anxiety.

        HRV. HRV feedback seems to be an extremely promising direction for developing physiological complementary treatment to traditional therapy. HRV feedback is a noninvasive treatment with minimal risk, and HRV is supported by research and theory as a measure of autonomic regulation which is central to anxiety disorders (Appelhans & Luecken, 2006). Personal HRV feedback monitors are affordable and may be a useful addition to traditional psychotherapy as therapists often teach anxious patients relaxation techniques and instruct them to practice at home, but compliance is mixed. With a personal HRV feedback monitor, patients may be more motivated to practice, and at the very least the monitors can provide information to the therapist about how much the patient is actually practicing so that they can discuss ways to increase compliance (Reiner, 2008).

        As very few studies on HRV feedback have utilized a clinical population, a critical next step for this line of research is to investigate the efficacy of various forms of HRV feedback to improve disorder-level anxiety. A large, independent, and controlled study of a personal HRV monitor like the StressEraser with a clinical population would not only advance understanding of this particular device but also help researchers ask more specific questions like which individuals and disorders may benefit the most from HRV feedback. Further, heart rate biofeedback like that demonstrated in Peira et al. (2013) should also be applied to a clinical population to understand how it may help individuals learn anxiety management skills that can generalize to daily life outside the therapist's office.

General recommendations

Researchers should strive to use stringent methodology utilizing large clinical samples with controls to produce informative research that may demonstrate the utility of biofeedback in treating anxiety disorders to a satisfactory degree. It is important to consider feasibility when developing new biofeedback technology, so research should focus on techniques that could easily transfer to a clinical setting. Additionally, to compete with the monumental funding of the pharmaceutical companies, foundations and other sources of research funding should grant financial support to research groups seeking to provide a lower-risk alternative.

Conclusions

Biofeedback treatments for anxiety disorders are generally promising, especially if future research can redress previous methodological limitations. While EMG feedback seems to be less effective than other treatments for anxiety, both EEG feedback and HRV feedback are potential avenues for impactful and cost-effective

treatment. Biofeedback therapies may be useful in conjunction with traditional psychotherapy for anxiety disorders, as many patients seem open to utilizing alternative treatments if they are covered by health insurance (Kessler et al., 2001). Further, cost-effective biofeedback may be a beneficial addition to traditional school counseling for children as children often experience school-related anxiety and may respond particularly well to computer-based interventions which feel like games (Matuszek & Rycraft, 2003; Wenck, Leu, & D'Amato, 1996).

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