

Muscle relaxation therapy for anxiety disorders: It works but how?

Ansgar Conrad^{a,b,*}, Walton T. Roth^{a,b}

^a Department of Psychiatry and Behavioral Sciences, Stanford University, Stanford, CA, USA

^b Department of Veterans Affairs Palo Alto Health Care System, Palo Alto, CA, USA

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Abstract

Muscle relaxation therapy (MRT) has continued to play an important role in the modern treatment of anxiety disorders. Abbreviations of the original progressive MRT protocol [Jacobson, E. (1938). *Progressive relaxation* (2nd ed.). Chicago: University of Chicago Press] have been found to be effective in panic disorder (PD) and generalized anxiety disorder (GAD). This review describes the most common MRT techniques, summarizes recent evidence of their effectiveness in treating anxiety, and explains their rationale and physiological basis. We conclude that although GAD and PD patients may exhibit elevated muscle tension and abnormal autonomic and respiratory measures during laboratory baseline assessments, the available evidence does not allow us to conclude that physiological activation decreases over the course of MRT in GAD and PD patients, even when patients report becoming less anxious. Better-designed studies will be required to identify the mechanisms of MRT and to advance clinical practice.

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1. Introduction

Muscle relaxation has been an important therapeutic technique in the modern treatment of anxiety disorders. Its origins lie with Edmund Jacobson (1934a, 1934b, 1938, 1964, 1967, 1970),

Abbreviations: AR, applied relaxation; BP, blood pressure; CBT, cognitive-behavior therapy; CT, cognitive therapy; EEG, electroencephalogram; ES, effect size; GAD, generalized anxiety disorder; HR, heart rate; MRT, muscle relaxation therapy; PD, panic disorder; PMR, progressive muscle relaxation; SAD, social anxiety disorder; SC, skin conductance; sEMG, surface electromyogram

* Corresponding author. Present address: VA Palo Alto Health Care System, 3801 Miranda Ave., 116F-PAD, Palo Alto, CA 94304, USA, Tel.: +1 650 493 5000x64793, fax: +1 650 493 4901.

E-mail address: aconrad@stanford.edu (A. Conrad).

who developed the method of progressive muscle relaxation (PMR). Since then many abbreviated methods of progressive muscle relaxation have been developed. These methods have been used either as complete treatments (e.g., Bernstein & Borkovec, 1973; Öst, 1987) or as one component among others in a treatment package (e.g., Wolpe, 1952a, 1952b, 1958). Many experimental studies attest to the clinical effectiveness of abbreviated muscle relaxation therapies for several medical conditions and psychiatric disorders, but surprisingly few of them have assessed muscle tension between patients and healthy subjects before treatment or have shown that muscle relaxation therapy (MRT) influenced physiologically measured muscle tension or other physiological measures of general activation. This neglect is perhaps one reason that in the last decade many researchers and practitioners have turned their attention to pharmacological or cognitive treatments. Some recent researchers have even considered muscle relaxation to be no more than a “psychological placebo” (Greist et al., 2002; Marks et al., 1993, 2000; Park et al., 2001), useful solely to calibrate the superiority of better treatments.

For the purpose of this review, we define muscle relaxation therapy as an abbreviated therapy based on Jacobson’s original PMR, which included in its training procedure first tensing a muscle and then releasing that tension. The basic therapeutic claim of MRT is that tense, stressed, and anxious people can find relief from their distress and its physiological accompaniments by learning to reduce muscle tension. A modern theoretical rationale for MRT is that an important element of psychological distress is elicitation of a generalized stress activation response, comprising multiple central and peripheral physiological systems (e.g., Öst, 1987). Learning to deactivate a single subsystem, the muscular system, will reduce activation in many other subsystems (e.g., Gellhorn & Kiely, 1972).

Is this rationale plausible? First, it assumes the existence of a generalized stress activation response with some consistency within and between individuals. Activation of the physiological component of this response should generally be linked to another system of emotional expression—the cognitive-language system. Stress activation of the muscular physiological subsystem would be expected to be generalized, resulting in a surface electromyogram (sEMG) intercorrelated at multiple sites, an assumption that often has been challenged. Second, the rationale assumes that patients who can be treated successfully with MRT will initially have either more tonic muscle tension, or exhibit increased muscle tension in response to stress than a nondistressed control group, assumptions with some support in generalized anxiety disorder (GAD) but little in panic disorder (PD). Third, a reduction in muscle tension should cause the multiple aspects of the activation response, as well as expressions of emotion in nonphysiological systems, to decrease, an assumption that has rarely been tested.

One could also question why it matters *how* muscle relaxation works, if it is already known *that* it works. One answer is that while relaxation works for many patients, it does not work for all. By understanding the mechanisms of muscle relaxation, better predictions should be possible of the effectiveness of this technique for different patients and different disorders. Furthermore, the optimal parameters for muscle relaxation therapy are unknown. For example, how many sessions of what length are required? Perhaps the customary 12 sessions of therapy in Öst’s applied relaxation (AR) treatment protocol (1987) are unnecessary because physiological improvement levels off after two sessions. In addition, if it turns out that muscle relaxation works more cognitively than physiologically, the therapist might do better by paying less attention to muscle tension and more to dysfunctional beliefs and attitudes. In fact, muscle relaxation may work entirely by changing dysfunctional beliefs, making the patient think that anxiety and the bodily changes that accompany it are understandable and controllable. Nevertheless, it is still possible that in certain clients directing attention to muscle

tension is a more effective way of changing beliefs than trying to deal with those beliefs directly.

Here we will examine the current state of psychophysiological research on muscle tension and relaxation in anxiety disorders, emphasizing findings in PD and GAD. First, we will summarize the most common muscle relaxation techniques and evidence for their clinical effectiveness. Then we will review the physiological basis of muscle activity and relaxation, and how two subgroups of anxious patients, PD and GAD patients, differ from controls physiologically. Finally, we will discuss relaxation treatment studies of anxious patients, paying special attention to whether in those trials there was credible evidence of physiological change.

2. A brief history of muscle relaxation therapy

PMR originated from the theory that a psychobiological state called *neuromuscular hypertension* is the basis for a variety of negative emotional states and psychosomatic diseases (Jacobson, 1938). Jacobson asserted that relaxation of muscles would lead to relaxation of the mind, “because an emotional state fails to exist in the presence of complete relaxation of the peripheral parts involved” (Jacobson, 1938, p. 218). In other words, relaxation inhibits the generation of thoughts and emotions, and undoes the effects of neuromuscular hypertension on the body. Briefly, in PMR clients sit in a comfortable chair and the therapist instructs them in contracting and releasing different muscle groups. Clients practice tensing a muscle group until they recognize the feeling of even the slightest contraction, and then learn to release it. The client “learns to recognize contraction in the various parts in a certain order. The large muscle groups are attended to first, because the sensation from them is most conspicuous. When clients relax a given part, they simultaneously relax all parts that have previously received practice” (Jacobson, 1938, p. 42). After they master relaxation while lying down, they are taught how to relax muscles in real-life situations, which requires “differential relaxation,” minimizing tension in the muscles needed for some activity while completely relaxing muscles not being used. Classical PMR was time-consuming. Jacobson (1938) initially suggested 30 to 60 min treatments several times a week for up to more than a year. Borkovec and Krogh Sides (1979) reported that PMR frequently involves over 50 sessions.

Wolpe (1958), a pioneer in behavioral therapy, adapted PMR for use in his systematic desensitization therapy of phobias, which is based on the idea that cues that once elicited anxiety can be conditioned to become cues that inhibit anxiety when they are linked to a state of elevated parasympathetic tone (Wolpe, 1952a, 1952b). Wolpe chose relaxation to enhance parasympathetic tone. He first taught clients a streamlined form of muscle relaxation, generally limiting the number of sessions to fewer than seven with home practice in between. Then he exposed relaxed clients to anxiety-evoking stimuli organized in a hierarchy from least to most frightening, desensitizing them to increasing doses of anxiety as therapy progressed.

Bernstein and Borkovec (1973) wrote a detailed relaxation-training manual for therapists and researchers in order to standardize the procedure, because procedural variations would invalidate comparisons of outcome across different therapists and clinics. The manual’s approach is similar to that of Wolpe’s and Jacobson’s, in that after clients have understood and accepted the rationale of progressive relaxation training, their attention is directed to different muscles in turn. Clients learn to (a) focus their attention on and (b) tense a muscle group upon a signal from the therapist, (c) maintain contraction for 5–7 s, (d) relax the muscle upon a cue, and (e) focus on the feelings in the muscle group as it relaxes. Bernstein and Borkovec modified Jacobson’s original method in that they do not promote distinguishing between different degrees of tension, but rather instruct patients to either tense or relax a muscle group completely. In addition, the manual introduces

clients to the “pendulum effect,” whereby, in analogy to the swing of a pendulum, voluntarily increased muscle tension is said to lead to deeper relaxation after the tension is released. In one study, this assumption was confirmed for frontal but not forearm sEMG (Lehrer, Batey, Woolfolk, Remde, & Garlick, 1988).

Öst (1987) in his applied relaxation protocol teaches mainly but not exclusively muscular relaxation, focusing on recognizing anxiety early and on dealing with it quickly. AR consists of 8–12 weekly sessions lasting for 60 min and homework. Clients learn to (a) observe and record initial anxiety reactions in order to recognize these symptoms, (b) relax with a shortened version of progressive relaxation based on Wolpe’s technique (Wolpe & Lazarus, 1966), and (c) extend their skill by relaxing differentially, rapidly, under the control of cues, by release of tension alone. Patients learn to apply these skills in daily situations at the first hint of anxiety.

3. Clinical outcomes of muscle relaxation therapy

In a unique, comprehensive meta-analytic treatise Grawe, Donati, and Bernauer (2001) reported the quality and effectiveness of every psychotherapy that had been cited in electronic medical and psychological databases. In order for a study to be eligible, (a) clients had to have been subjected to some psychological influence with therapeutic intent, and (b) at least one of the following variables had to have been experimentally manipulated: method, patient sample, therapist, or setting. The meta-analysis included every study published before 1984 that fulfilled these requirements. Grawe et al. found 66 studies in which PMR or one of its successors was applied as a therapeutic intervention. As of their publication, clinical effectiveness had been primarily evaluated for hypertension, headaches, and insomnia. In 76% of studies that used standardized assessment, muscle relaxation led to significantly positive changes. PMR was superior to wait-list control group in 63% of all studies. Patients with a primary diagnosis of an anxiety disorder – the authors did not specify this any further – and/or with symptoms of tension and stress, improved significantly in 8 of 10 studies. Nine of 11 studies found MRT superior to waiting.

Hyman, Feldman, Harris, Levin, and Malloy (1989) computed effect sizes (ES s), as defined by Glass, McGaw, and Smith (1981), for studies published after 1970 that examined the effect of relaxation treatment on adult out-patients with various disorders. In 21 studies, the active treatment group was compared to no-treatment or standard-treatment control group ($M_{ES} = .63$, $S.D._{ES} = 1.02$), and in 5 to placebo treatment ($M_{ES} = .49$, $S.D._{ES} = .35$), yielding moderate ES s. In the majority of studies, muscle relaxation was used to treat anxiety or stress ($N = 6$, $M_{ES} = .50$, $S.D._{ES} = 1.72$), insomnia ($N = 4$, $M_{ES} = .69$, $S.D._{ES} = .18$), or hypertension ($N = 4$, $M_{ES} = 1.24$, $S.D._{ES} = .83$). The results of Hyman et al. together with those of Grawe et al. document that muscle relaxation is effective for a variety of clinical symptoms. However, as McGuigan (1992) pointed out, these results may not apply to Jacobson’s original PMR, because virtually every reported MRT study has deviated in length or nature from his PMR.

King (1980) assessed the clinical utility of MRT in a review that distinguished between MRT as the sole intervention and MRT as an aid to other interventions. He reported that MRT as a unimodal therapy had been employed widely and successfully, as measured by positive cognitive, performance, and behavioral changes, in the treatment of many disorders, including insomnia, hypertension, asthma, general tension and anxiety, phobia, pain, smoking, tension and migraine headache, hyperactivity, and compulsive behaviors. However, because most studies did not meet stringent scientific standards for controlling demand characteristics, reliability and validity of dependent measures, and external validity, he concluded that the value of MRT as a sole intervention remained uncertain. MRT appeared useful as an adjunct to systematic (in sensu

or in vivo) desensitization and to biofeedback. In an update, Bernstein and Carlson (1992) reviewed 30 controlled studies after 1981. They found evidence that MRT can lead to definite improvement in depression, aversion to chemotherapy, muscle headache, stress reactivity, lack of immune competence in the elderly, dysmenorrhea, low back pain, and possibly to improvement in hypertension, generalized anxiety, and chronic tinnitus. Öst (1987) reviewed controlled studies of AR. He found that AR was significantly better than control or placebo conditions for treating phobia, PD, headache, pain, epilepsy, and tinnitus. Improvement was sustained at follow-up assessments, averaging 11 months posttreatment.

Recently, Jorm et al. (2004) reviewed the clinical effectiveness of relaxation therapy for anxiety as part of a project that analyzed evidence for the effectiveness of many complementary and self-help treatments for reducing anxiety. The review included all relevant randomized controlled trials published before 2003. Based on more than 60 clinical outcome studies, Jorm et al. concluded that relaxation was as effective as pharmacologic, cognitive, or exposure-based interventions for PD with or without agoraphobia, GAD, and dental phobia. The authors found less evidence of relaxation therapy being as effective as these other therapies in specific phobia (e.g., spider or snake phobia, claustrophobia, fear of flying), social anxiety disorder (SAD), obsessive-compulsive disorder, or posttraumatic stress disorder, although for these disorders it outperformed simply waiting to be treated.

In July 2006, we conducted our own systematic, keyword-driven (*relaxation AND anxiety OR panic OR phobia*) search of the National Library of medicine's database, PubMed. To be included, studies had to be published in English in a peer-reviewed journal after 2002. Furthermore, patients had to be older than 18 years, have had a diagnosis of an anxiety disorder, and have been treated by someone with clinical training. We found five controlled studies in which the clinical outcome of PMR or MRT in anxious patients was assessed. The diagnostic groups were dental phobia (two studies), SAD, GAD, and PD. In three studies (Carlbring, Ekselius, & Andersson, 2003; Willumsen & Vassend, 2003), AR was the only treatment modality, and resulted in equivalent or superior outcomes to cognitive therapy (CT) or cognitive-behavior therapy (CBT).

Willumsen and Vassend (2003), Willumsen, Vassend, and Hoffart (2001a, 2001b) treated 62 dental phobics with nitrous oxide, CT, or AR in 10 weekly sessions and found that all of these treatments resulted in significant and equivalent posttreatment improvement. ESs (Cohen's d) ranged from .9 to 4.6. Treatment remained effective at 12 and 60 months follow-up, with 81% of participants continuing to find intervention received helpful after 5 years. Carlbring, Ekselius, and Andersson (2003) assigned 22 panic patients randomly to AR or CBT. The treatments were administered in a web-based version, with 6–9 modules. Both treatments were effective, having medium to large effect (ESs: $d = .71$ for AR, $d = .42$ for CBT). Arntz (2003) randomized 45 GAD patients to 12 weekly sessions of AR or CBT. He found both treatments to be effective (ES: $d = .53$ – 1.14). AR did better than CT at 1-month follow-up, with CT catching up at 6 months. Fifty-five percent of CT patients and 53% of AR were recovered at 6 months follow-up.

Two studies combined AR with CT or frontalis sEMG biofeedback. In Bogels' study (2005), eight sessions of AR preceded eight sessions of CT. Sixty-five SAD patients were recruited and randomized to the AR-CT condition or to a "task concentration training" followed by CT. Both conditions were effective after 8 sessions, 16 sessions, and at follow-ups (ES for SAD symptoms: $d = .3$ – 1.3), with patients in the latter showing more improvement than the AR-CT condition. Lundgren, Carlsson, and Berggren (2006) randomized 127 dental phobia patients, who were divided by fear etiology and physiological response style, to eight weekly sessions of MRT enhanced with frontalis sEMG biofeedback or CT. Both treatments were effective in reducing

physiological response and dental fear. However, there was limited evidence that fear etiology or physiological response interacted with treatment condition.

In conclusion, a considerable number of published studies have succeeded in showing that muscle relaxation is beneficial in a variety of medical conditions and in several psychiatric disorders, particularly anxiety disorders such as dental phobia, GAD, and PD.

4. Physiology of tension and relaxation

While many therapies help anxiety disorders, often little is known about how they contribute to improvement and which individuals are most responsive to them (Marks, 2002). In order to understand muscle relaxation as a treatment, we need to understand its physiological basis, if indeed it has one. The contraction of skeletal muscle fibers leads to the sensation of muscle tension, the result of a complex interaction of the central and peripheral nervous system with the muscular and the skeletal systems. In this unipolar psychobiological model of tension, relaxation is the absence of perceived muscle tension. According to Jacobson (1938), complete muscle relaxation is incompatible with having any thoughts or feelings.

Usually, however, tension and relaxation are regarded more broadly as part of a generalized activation or stress response, which in the autonomic nervous system involves firing of sympathetic or parasympathetic nerve fibers. Several organs are innervated with nerve cells of both kinds, allowing for distinct sympathetic and parasympathetic effects originating in higher centers in brain and spinal cord (Andreassi, 2000). The sympathetic and parasympathetic nervous systems often work reciprocally in that elevated activation of one leads to deactivation of the other. Sympathetic activation is part of what has been called the ergotropic or ‘fight or flight’ response (Cannon, 1929), whereby organs are activated for emergency and stress situations. Cannon observed in dogs increases in heart rate (HR), blood pressure (BP), blood flow to voluntary muscles, hyperglycemia, and papillary dilatation. Parasympathetic activation has been called trophotropic in that it promotes rest and repair. Its activation is the basis for what Benson (1975) called the relaxation response. Parasympathetic responses include reductions in HR and BP, increased activity of the digestive system, and increased blood flow to the sexual organs during sexual arousal.

Hess and colleagues (e.g., Hess, 1957; Hess & Brugger, 1943) found the diencephalon to be the critical brain location for coordinating ergotropic and trophotropic actions. Citing Hess’ work, Gellhorn and Kiely (1972) explained the relationship of muscle tension, relaxation, and the autonomic nervous system in this way: Afferent neuromuscular pathways innervate the reticular formation, which in turn projects to the posterior hypothalamus. The reduction of the skeletal muscle tone “leads to a loss in ergotropic tone of the hypothalamus, a diminution of hypothalamic-cortical discharges, and, consequently, to a dominance of the trophotropic system through reciprocal innervation” (Gellhorn & Kiely, 1972, p. 404). In other words, muscle relaxation in the periphery results in a centrally mediated shift of the bodily system towards a trophotropic response.

Although Gellhorn’s and Jacobson’s peripheralist mechanism is a popular neurophysiologic explanation of relaxation (King, 1980), Davison (1966) among others has criticized it as an oversimplification inconsistent with the fact that animals (e.g., Solomon & Turner, 1962) and humans (e.g., Smith, Brown, Toman, & Goodman, 1947) can evidence distress and fear under total curarization, where muscles are completely without tone. Furthermore, Gellhorn and Kiely’s explanation does not mention pathways by which central, cognitive events could affect relaxation, either directly or indirectly by influencing peripheral systems. It is even conceivable

that relaxation could be experienced in the cognitive realm without any somatic accompaniment or at least without muscular relaxation. That thoughts influence feelings of tension or calmness hardly needs demonstration. A popular psychological paradigm is to examine the effects of imagining fearful situations on a variety of cognitive and somatic measures (e.g., Cuthbert et al., 2003; Lang, Davis, & Ohman, 2000). Cuthbert et al. (2003) assessed psychophysiological response to imagery of fearful memories in participants with specific phobia, social anxiety disorder, posttraumatic stress disorder, PD, and healthy controls. The authors recorded HR, skin conductance (SC), and sEMG of the corrugator muscle while participants responded to tone cues signaling previously memorized descriptor sentences. Images included neutral scenes, personal or social fears, and fears of physical danger. Results indicated that participants were more reactive to fear than neutral stimuli in HR, SC, and corrugator sEMG.

Less expected are research findings that repetitive, intrusive anxious cognitions (worrying) can suppress the somatic activation expected to accompany anxious states (e.g., Borkovec & Hu, 1990; Borkovec, Lyonfields, Wiser, & Deihl, 1993). Borkovec and Hu (1990) presented phobic images to speech-anxious participants who either engaged in worried or in relaxed/neutral thinking prior to having to speak. In the relaxed/neutral thinking condition, the phobic images led to increases in HR, while there was no cardiovascular reaction in the participants that engaged in worried thinking. However, effects on both muscle tension and HR have been assessed in only one study. Peasley-Miklus and Vrana (2000) measured both muscle activity from the corrugator supercilii and HR. They replicated Borkovec and Hu's findings for HR, but facial sEMG increased in the relaxed group as well as the worried groups during the presentation of phobic images.

The theoretical formulations and empirical findings reviewed above do not give us satisfactory answers about the extent to which self-reported tension and inability to relax are related to muscle tension, or the extent to which learning to relax muscles is a reasonable and efficient way to overcome this self-reported tension. Clearly, the tension and distress of anxiety involve more than the muscular system. Generalized stress activation involves cognitions, and to some extent the cognitive, physiological, and action tendencies associated with this activation can vary independently. Patterns of physiological activation may depend on the action tendencies associated with specific emotional reactions.

5. The surface EMG and the general tension factor

The sEMG is an accurate and safe noninvasive way to measure muscle activity. Active motor units are registered from two active electrodes over the muscle. Because it avoids percutaneous placement of electrodes in the muscle itself, sEMG has been used in many studies investigating the effects of muscle biofeedback and relaxation on psychiatric patients and healthy controls. Before discussing psychophysiological studies that assessed muscle activity in anxious patients, it is important to mention two common assumptions about sEMG.

The first is that the baseline resting tone in muscles is normally above zero, making therapeutic reduction of that tone possible. This is contradicted by reports of electrical silence at rest (DeVries, 1965; Fridlund, Cottam, & Fowler, 1982; Ralston & Libet, 1953). Unfortunately, if there is a resting tone, sEMG magnitude may not be able to faithfully quantify it since sEMG amplitude varies with electrode placement, tissue noise, noise voltage, and dermal resistance (Fridlund et al., 1982; Mercer, Bezodis, Delion, Zachry, & Rubley, 2006). For example, Mercer et al. (2006) found that contraction conditions were influenced so much by sensor locations that different conclusions could have been reached from the same experiment depending on those locations. Furthermore, experimental subjects, particularly anxious ones, may generate sEMG

artifacts by blinking or swallowing, which can be picked up as muscle tension in integrated records of the frontalis sEMG. Upon repeated testing, the same subjects may show less of such artifact, which could be falsely interpreted as a reduction in muscle tension.

The second assumption is that psychic tension is reflected by general muscle tension, in multiple muscles in different parts of the body. Hess' theory of trophotropic and ergotropic response (Hess, 1957) seems to imply that the activity of the different muscles will be highly correlated (Fridlund et al., 1982). Consequently, sEMG biofeedback relaxation therapies often train one muscle and assume its relaxation will result in relaxation of multiple muscles. Some earlier studies (e.g., Balshan, 1962; Malmo & Smith, 1955) supported the assumption that muscle tension is general in finding that sEMG amplitudes were correlated with each other. Malmo and Smith (1955) conducted a factor analysis on data collected from six different sEMG sites in "psychoneurotic" patients and healthy controls and found that five of the six sites loaded highly on one factor. Balshan (1962) found high correlations of sEMG levels between 14 and 16 sites in women. However, these results are problematic, because Balshan performed principal component analyses on intercorrelation matrices of muscle groups by subjects. In such a design, tension is confounded with EMG responsivity of the subject (Fridlund, Fowler, & Pritchard, 1980). Furthermore, Balshan's method of integrating sEMG levels over 30-s periods may have artifactually produced a general tension factor by integrating temporally EMG activity that would not have been correlated in shorter time windows. In a carefully conducted replication, Fridlund et al. (1982) found little evidence for a general tension factor in individuals. Additional studies have reported that sEMG recordings of one muscle do not generalize to other muscles (Alexander, 1975; Shedivy & Kleinman, 1977).

Furthermore, Lacey and colleagues (e.g., Lacey, Bateman, & Vanlehn, 1953; Lacey & Van, 1952) found in a series of experiments that the autonomic nervous system does not work as a single unit. Rather, subjects reacted to stressors with increases in some measures and decreases in others. He coined the term *relative response specificity*, meaning that some individuals have a stereotyped response pattern to stress while others respond to stress with random patterns. If response patterns appeared repeatedly, the degree of activation of the different physiological measures varied significantly but the rank order often remained the same. Another argument against the idea that muscle relaxation produces an overall decrease in generalized stress activation is that sEMG biofeedback training has failed to affect autonomic parameters such as HR, SC, respiration, and skin temperature (e.g., Burish, Hendrix, & Frost, 1981; Carlson, Basilio, & Heaukulani, 1983; Jones & Evans, 1981). Learning to relax one or more muscles does not necessarily lead to generally reduced physiological arousal.

Because sEMG data collection, reduction, and analysis requires special amplifiers and recording and analysis programs, researchers and therapists have been tempted to rely on self-report to assess muscle tension. However, a number of studies using sEMG have found no relationship between self-reported and physiologically measured tension (e.g., Katkin, Morell, & Goldbond, 1982; Pennebaker, 1981, 1982; Shedivy & Kleinman, 1977; Tyrer, Lee, & Alexander, 1980). For example, Shedivy and Kleinman (1977) assessed whether sEMG recorded activity of somatic muscles correlated with verbal reports of tension and relaxation in controls, and found no significant relationship. McLeod, Hoehn-Saric, and Stefan (1986) found no relationship between the two in GAD patients. Thus, to understand the basis of complaints of tension in general and muscle tension in particular, one must record muscle activity. Furthermore, recording from multiple muscles is indispensable, since one cannot assume that any one muscle is representative of other muscles or the tensional state of the body as a whole. Recording also must meet technical standards that assure data quality (Fridlund & Cacioppo, 1986).

6. Physiological activation in GAD and PD

Tension of some kind and difficulty relaxing are implicit in the DSM-IV (American Psychiatric Association, 1994) definitions for GAD and PD. The criteria for GAD state that the patient may be *restlessness or feeling keyed up or on edge* and may experience *muscle tension* and *sleep disturbance*. Although one would expect that these criteria would entail heightened sympathetic activation, Hoehn-Saric and colleagues did not find tonically elevated levels HR or SC in GAD patients (Hoehn-Saric, 1982; Hoehn-Saric, Hazlett, & McLeod, 1993), and observed diminished responsiveness of these variables during stressful situations such as a divided-attention task and a risk-taking task (Hoehn-Saric & McLeod, 1988; Hoehn-Saric, McLeod, & Zimmerli, 1989). Consistent with this, Wilhelm, Trabert, and Roth (2001) found no differences between GAD patients and controls who were sitting quietly for 30 min, in an array of electrodermal and cardiovascular measures, including SC level and nonspecific fluctuations, HR, BP, stroke volume, and cardiac output. However, Thayer, Friedman, and Borkovec (1996) recorded the cardiopulmonary responses of 34 GAD patients and 32 healthy controls during three 5–12 min periods in which the participants were either resting (baseline), were asked to relax, or were worrying, and found that in all these conditions, GAD patients tended to have shorter interbeat intervals, and more low frequency and less high frequency heart rate variability. These findings were interpreted as indicating lower parasympathetic tone in GAD. In an ambulatory monitoring study, Hoehn-Saric, McLeod, Funderburk, and Kowalski (2004) recorded HR, SC, respiration, motion, and ratings of subjective somatic symptoms, tension, and anxiety in four 6 h recording sessions in 26 patients with PD, 40 with GAD, and 24 controls. GAD patients showed less HR variance and less SC variance than controls, accompanied by higher psychic and somatic anxiety symptoms.

Muscle tone appears more sensitive than electrodermal or cardiovascular variables in distinguishing GAD patients from controls. Several studies have reported that GAD patients show elevated tone in the frontalis and gastrocnemius muscles at rest and during tasks (Hazlett, McLeod, & Hoehn-Saric, 1994; Hoehn-Saric & Masek, 1981; Hoehn-Saric et al., 1989). (In Hoehn-Saric and Masek (1981), the patients were characterized as “chronically anxious,” but later retrospectively diagnosed as having GAD (Hoehn-Saric & McLeod, 1988).) For example, Hazlett et al. (1994), assessed 18 women with GAD and 19 controls during baseline, laboratory stressor, and recovery. SEMG levels in the frontalis and gastrocnemius muscles were greater in the GAD group in each condition. Hoehn-Saric, Hazlett, Pourmotabbed, and McLeod (1997) interpreted this rather idiosyncratically as meaning that GAD patients exhibit central rather than autonomic hyperarousal, because muscle tension had been correlated with the power of the electroencephalogram (EEG) in the beta range.

The literature on physiological activation is more extensive for PD than for GAD. The DSM-IV definition of panic attacks lists many autonomic and respiratory symptoms. Sometimes panic patients have been reported to have elevated HR and low frequency heart rate variability at rest (Cohen et al., 2000; Holden & Barlow, 1986) and reduced HR variance and SC variance during the day (Hoehn-Saric et al., 2004), but Wilhelm et al. (2001) found differences only in respiratory measures. In the last study, PD patients showed lower end-tidal $p\text{CO}_2$ combined with higher tidal volumes and larger numbers of sighs. PD and GAD patients both showed greater instability in regards to $p\text{CO}_2$, respiratory rate, and minute volume. Carr, Lehrer, Hochron, and Jackson (1996) found lower airway impedance in PD, suggesting a chronic state of preparedness that may promote hyperventilation. Several laboratory studies have found that anxious patients exhibit a slower decline in SC than do controls (Roth, Ehlers, Taylor, Margraf, & Agras, 1990; Roth et al.,

1986), presumably indicating difficulty relaxing. Roth, Wilhelm, and Trabert (1998) gave a standardized activating task before a quiet sitting period in PD patients and controls. The results were inconsistent in that shallower SC slopes suggested that panic patients relaxed slower than controls, while steeper finger temperature slopes indicated the opposite. In one of the few studies assessing sEMG in PD patients, Hoehn-Saric, McLeod, and Zimmerli (1991) measured frontalis and gastrocnemius sEMG as part of a battery of physiological measures during a baseline and during a psychological stressor. During baseline, panic patients exhibited higher forehead muscle tension as well as higher systolic BP and HR, but electromyographic reactivity to a stressor did not distinguish patients from controls. HR and systolic BP rose more in panic patients, while SC rose more in controls.

Thus, the literature seems to indicate greater generalized physiological activation at rest in GAD and PD in some systems but not in others. SEMG seems to be more sensitive than electrodermal or cardiovascular measures. Tidal volume variability is often elevated in PD but the relation of this to generalized activation is unclear. A slower decline of SC in PD patients while quietly sitting suggests an inability to relax, although subjects were not explicitly asked to relax in the studies above. However, in an EEG study in which panic patients and control subjects were given audiotapes with neutral or relaxation instructions (Knott, Bakish, Lusk, & Barkely, 1997), the EEGs of patients tended not to change with the relaxation tape as much as the EEGs of controls, which contained more alpha and theta than in the neutral condition. Increases in alpha and theta are associated with reduced generalized activation.

7. Physiological assessment of muscle relaxation therapy in anxiety patients

The literature on muscle relaxation is a large one, in which its psychological and physiological effects in a great variety of individuals have been investigated. Many studies have tested mentally and physically healthy volunteers, and yielded interesting results. However, in this review we restrict ourselves to studies that have both treated anxious patients and taken physiological measurements. Our main questions relate to the validity of the basic rationale of muscle relaxation therapies in treating anxiety. We examined treatment studies for evidence pertaining to five basic assumptions that form the rationale for treating anxiety with muscle relaxation:

1. *Single or multiple muscles are tenser in anxious patients than in healthy volunteers before the therapy begins.* This requires recruiting and testing a normal control group, since unlike with psychological or clinical tests, the methods and procedures for muscle and ANS tests are not well enough standardized for comparisons between studies. The tension should be greater at the times when patients are having symptoms. For GAD this could be most of the time, as GAD is characterized by excessive worrying, which is more chronic than episodic. For PD the measurements ideally need to be made during attacks, which for natural attacks is technically difficult since ambulatory recording is necessary to capture them.
2. *Muscle tension is correlated with other kinds of physiological activation such as autonomic signs of increased sympathetic and decreased parasympathetic tone or hormonal signs of HPA-axis activation.* Without this correlation, reduction in muscle tension is less likely to be a sign of better emotional regulation.
3. *Changes in muscle tension and self-reported anxiety symptoms before and after therapy are positively correlated.* Anxiety, symptoms, and muscle tension should be all high before therapy and in successfully treated patients, all low afterwards. Although correlation is not causation, a lack of correlation would indicate that muscle tension is neither a cause nor an

effect of anxiety. At what times and under what circumstances these measurements should be made depends on how the therapy is conceptualized. If muscle relaxation is a skill under voluntary control, measurements should be made when the patients report that they are deliberately exercising that skill. The skill may be demonstrable when the patient is not having symptoms, but that does not guarantee that it can be applied when the person is frightened or worried, and whether application at that time would reduce symptoms. On the other hand, it is possible that muscle relaxation can become a persistent habit, which is constantly present or appears automatically when needed, requiring intentional effort to suppress. In any case, if anxiety is intermittent, observations restricted to nonanxious periods are inadequate to test this assumption.

4. *Muscle relaxation becomes faster or deeper with practice.* Otherwise, muscle relaxation is less a specific skill that is learned in therapy, than an innate or previously learned voluntary response that can be evoked fully with proper motivation and attention.
5. *Muscle relaxation treatment can produce a substantial reduction in the distress and functional impairment associated with at least some kinds of anxiety.* In other words, the treatment results in not just a statistically significant change in some psychological test of anxiety, but in at least a moderate overall improvement in the person's life.

As will become apparent below, existing studies by their design or methodology often have not tested these assumptions adequately.

Physiological research on intentional muscle relaxation began with its founder, [Jacobson \(e.g., 1938\)](#), who asserted that training in his procedure led to decreases in sEMG, BP, and HR. In one study, Jacobson assessed forearm muscle activity in 16 healthy college students and 50 individuals with “neuromuscular hypertension” before and after relaxation training ([Jacobson, 1938](#)). Jacobson observed that even healthy subjects contracted their arm muscles at least occasionally while being tested, and thus were not completely relaxed, but less so than the neuromuscular hypertension group, who contracted their arm muscles much more. After four to 9 months of PMR, sEMGs in the neuromuscular hypertension group showed considerable improvement. Yet from the standpoint of modern research methods, Jacobson's studies are deeply flawed (e.g., [Mathews, 1971](#)). PMR training was not standardized and varied in length from weeks to years. Results were not analyzed statistically. There was no waiting list or placebo group to control for time effects and expectancy. Neuromuscular hypertension is a poorly defined diagnosis, whose relationship to today's anxiety disorders is indeterminate.

[Mathews and Gelder \(1969\)](#) studied 10 patients who were suffering from generalized anxiety, agoraphobia, social phobia, or specific phobia. They were randomly assigned to either a control condition, in which the patients' histories were taken and their practical problems were discussed, or to MRT, in which they received six weekly sessions of muscle relaxation training based on an abbreviated method suggested by [Jacobson \(1967\)](#). Forearm sEMG, SC, and forearm blood flow were recorded for 20 min while patients were asked to attempt to relax before and after the six sessions. The authors found no significant before–after by condition effects in the physiological data, indicating that relaxation did not decrease activation. In a second investigation, the authors provided 14 phobic patients with two weekly sessions of brief relaxation training. The training was followed by a test relaxation session and a test control session (during which the patients were instructed not to attempt to relax) during which frontalis sEMG, HR, SC, and respiration were recorded. The order of the two test sessions was randomly assigned. The investigators found that frontalis sEMG and nonspecific SC fluctuations were significantly lower in the test relaxation condition than in the test control condition, indicating

less activation in the relaxed group. They concluded, by comparing their data to other studies, that relaxation training in phobic patients reduces autonomic activity to the levels found in normal subjects at rest. However, since subjects were not tested before training, they may have been able to relax even before receiving the relaxation instructions.

Lehrer (1978) randomized 20 “anxiety neurotic” patients and 20 nonpatient controls to a muscle relaxation or waiting list group. Relaxation training consisted of four or five sessions over the period of 3 weeks. Ten additional nonpatient controls were given four sessions of EEG alpha activity feedback. Before and after the therapies, each group took part in a testing procedure, in which SC, HR, and EEG alpha activity were assessed in three conditions: relaxation for 5 min, the presentation of five 1 s 100 dB tones, and a reaction time task. There was no significant relaxation training effect for tonic HR or tonic SC, but training did reduce cardiac acceleration to the tones, especially in anxious patients. Unfortunately, the authors did not measure sEMG activity.

Canter, Kondo, and Knott (1975) studied a group of 28 psychiatric patients diagnosed with anxiety neurosis and having complaints of muscle tension and insomnia. About half had panic episodes, and about half were in-patients. Fourteen were assigned to frontalis sEMG biofeedback and 14 to muscle relaxation treatment. Relaxation was taught by tapes with instructions only to relax muscles, not to tense them first as in Jacobson’s method. Number of training sessions varied between 10 and 25. Muscle recording was done at the first and last 5 min of each training session. Both therapies produced significant reductions in frontalis tension levels between the first and last session, except for a subgroup without panic attacks assigned to feedback. Global self-ratings and observer ratings of change showed improvement that did not significantly differ between treatments. It was not clear from the article how clinically significant that improvement was. Results for sessions between the first and last were not reported, nor were correlations between improvement and amount of relaxation.

The Canter et al. study was replicated and extended by Leboeuf and Lodge (1980) who randomized 26 outpatients with a main complaint of pervasive anxiety for at least 2 years to frontalis sEMG feedback training or muscle relaxation. The relaxation training was individualized and involved tensing followed by relaxation, unlike the procedure used by Canter et al. Frontalis sEMG activity and HR were measured before and after three and a half months of weekly therapy, and prior to session 4, 7, 10, and 13. Subjects were told to relax the best they could during the assessment periods. The two treatments proved equally effective in reducing symptoms of chronic anxiety, but this improvement only reached the “moderate” level in 10% of the patients in each group. An analysis of variance and post hoc comparisons showed that that biofeedback group alone had a marked decrease in frontalis sEMG activity during assessment without feedback at session 13 and 16. Correlations between reduction in muscle tension and clinical improvement were low and insignificant. The authors concluded that greater relaxation of the frontalis muscle did not translate into substantial clinical improvement, and that without a delayed treatment group, they could not be sure that muscle relaxation was more than a placebo.

Miller et al. (1978) recruited patients identified by a dentist in his practice as having dental anxiety for a comparison of the effects of frontalis EMG biofeedback, progressive relaxation, and a control therapy in which patients simply closed their eyes and tried to relax. Frontalis sEMG measurements were made just before dental work at an initial appointment, during each of the ten therapy sessions, and just before dental work at a second appointment. Results indicated linear trends of decreasing sEMG activity with biofeedback and relaxation, but not for the control therapy, which was consistent with reductions of psychological measures of state anxiety and

dental fear. The design of this study is superior to many in employing a control therapy and measuring sEMG at multiple training sessions so that the rate of progress could be observed. A limitation of the study is that sEMG was only measured in one site.

Lehrer, Woolfolk, Rooney, McCann, and Carrington (1983) compared progressive relaxation to meditation and a wait list control group in 61 anxious subjects recruited by newspaper advertisements who had anxiety scores more than one standard deviation above the mean of a standardization group. SC, HR, EEG alpha activity, and forearm extensor and frontalis sEMG were recorded in test sessions before and after therapy. During these two sessions, subjects were asked to sit quietly and relax, to listen to 100 dB tones, and to watch a disturbing film sequence. After therapy, forearm sEMG response to the loud tones and film was significantly lower in the relaxation group than in the meditation group. However, frontalis sEMG reactivity was reduced as much by meditation as by MRT, arguing against a specific effect of instructions directed towards muscles. SC and alpha did not decrease over the course of therapy, which is evidence against reduction of a generalized activation response. Whether the subjects recruited by Lehrer et al. would have met current criteria for anxiety disorders is uncertain, and if they did, it is uncertain whether their improvement would have been clinically significant.

Barlow et al. (1984) were the first to use DSM-III criteria in the kind of studies under consideration here, giving a more precise definition to patient groups and kinds of anxiety disorders than previously. Nine GAD and 11 PD patients were randomly assigned to treatment package comprised of MRT, frontalis sEMG feedback, and cognitive restructuring or to a wait list control group. Before and after therapy, the subject underwent a physiological assessment comprised of quiet sitting, relaxing, and stressor tasks. Physiological measures were frontalis sEMG and HR. In contrast to most findings regarding muscle activity while sitting quietly or being stressed, PD patients showed higher sEMG levels before treatment than did GAD patients. Compared to the wait list group, treated patients improved not only on clinical ratings of improvement, daily self-monitored measures and self-reports, but also had lower HRs and frontalis sEMG during the assessment. Since a treatment package with multiple components was used, which therapy elements were responsible for the reduction in sEMG and clinical improvement, are uncertain.

Michelson, Mavissakalian, and Marchione (1985) and Michelson et al. (1990) were interested in the differential psychophysiological effects of paradoxical intention (a cognitively based intervention), MRT, or graduated exposure in the treatment of agoraphobia. The authors recruited 88 agoraphobics, and provided 12 weekly 2 h long group therapy sessions. Continuous HR was recorded before and after treatment and at 3 months follow-up during resting, walking, and as part of the standardized behavioral avoidance course (S-BAC). The three treatments were effective in reducing agoraphobic cognitions with exposure and MRT outperforming paradoxical intention. This pattern was upheld at follow-up. The HR analysis indicated that HR declined in exposure and MRT conditions from pretreatment to postassessment and follow-up, while there was no change in the paradoxical intention condition. There was limited evidence that the HR changes were larger in the MRT than exposure condition.

Carlson, Collins, Nitz, Sturgis, and Rogers (1990) explored the use of muscle stretching procedures in relaxation training. In their first study, 7 men and 17 women responders to an advertisement for people “experiencing moderate levels of muscle tension or anxiety” were randomized to four weekly stretching exercises for four muscle groups (obicularis oculi, sternocleidomastoid/trapezius, triceps/pectoralis major, and forearm/wrist flexors), to tense-release relaxation (Bernstein & Borkovec, 1973), or to a control condition. The authors recorded frontalis, trapezius, masseter, and brachioradialis sEMG before and after therapy in a “stress

profiling” procedure (2 min baseline, 2 min relaxation, 2 min stressful imagery, 2 min pleasant imagery, 2 min relaxation, 2 min baseline). A 2-min resting segment followed each period. In addition, sEMG was assessed before, during and after each session. A group by session MANOVA indicated that patients in the stretch relaxation condition rated several muscles as more relaxed than in the tense-release group, but analyses of the sEMG recordings revealed greater relaxation of the stretch relaxation group only in the right masseter. The groups did not differ on pretreatment adjusted sEMG measures assessing recovery from the stressor after treatment, although both treatment groups rated the right obicularis oculi region as more relaxed than in the control condition.

In their second study, 3 men and 17 women were recruited in the same way as in the first study. An expanded stretch relaxation protocol was given to all subjects. In addition to monitoring frontalis, right trapezius, submandible, and right brachioradialis sEMGs during therapy, the authors measured HR, skin temperature, respiration, and BP. For pre- and postassessments the authors used a modified stress test, in which participants participated in quiet sitting (5 min), mental mathematics (5 min), recovery (5 min), cold pressor stressor (1 min), recovery (5 min), and baseline (5 min). Based on the baseline stress testing, participants were classified as muscle tension responders or cardiovascular responders or both. While self-reported muscle tension was reduced in all subjects after treatment, muscle tension responders showed reduced trapezius sEMG and respiration rates. Diastolic BP was reduced in cardiovascular responders.

Seven studies of Öst’s (1987) applied relaxation treatment protocol in anxiety patients recorded physiological measures: five recorded only HR, while two recorded in addition BP and electrodermal activity (Jerremalm, Jansson, & Öst, 1986a, 1986b; Öst, Jerremalm, & Jansson, 1984; Öst, Jerremalm, & Johansson, 1981; Öst, Johansson, & Jerremalm, 1982; Öst, Lindahl, Sterner, & Jerremalm, 1984; Öst, Sterner, & Fellenius, 1989). The participants were patients with agoraphobia, claustrophobia, social phobia, dental phobia, and blood phobia. In three studies (Öst, Jerremalm, et al., 1984; Öst et al., 1981, 1982), patients were divided into physiological reactors and behavioral reactors. Physiological reactors showed large differences in HR between a baseline recording and a recording in a stressful situation, but only small overt behavior reactions during the stressor. Behavioral reactors showed large behavioral and small HR reactions. Physiological and behavioral reactors were randomized to a physiologically focused method (AR) or to a behaviorally focused method that fit their classification. In the social phobia study (Öst et al., 1981), the authors offered social skills training, and in the claustrophobia (Öst et al., 1982) and agoraphobia (Öst, Jerremalm, et al., 1984) studies, in vivo exposure. In each study, physiological reactors showed significant reductions in HR reactivity after AR therapy compared to their baseline values. However, social skills training and exposure therapy also led to significant decreases in HR reactivity. Only in claustrophobia patients did exposure lead to significantly less reduction in HR than did AR. None of behavioral reactors showed significant differences in HR between pre- and posttreatment, probably since by definition they did not react much at pretreatment.

Jerremalm et al. (1986a, 1986b) divided patients with dental phobia and social phobia into physiological reactors and cognitive reactors by HR reactivity and answers to a self-report scale assessing thoughts during the stressor. Participants were randomized to AR (a physiologically focused method) or to self-instructional training (a cognitively focused method). Again, both treatments led to significant reductions in HR reactivity in the physiological reactors, while cognitive reactors, as expected, showed no difference.

Öst, Lindahl, et al. (1984) compared AR to exposure therapy in blood phobic patients. Patients were treated individually for nine sessions. Before therapy, after therapy and at 6 months

follow-up, the authors recorded HR and BP during a 10 min baseline, during a 30 min film clip presenting four thoracic operations with large amounts of bleeding, and during a 10 min postbaseline. The data were analyzed as the difference between baseline mean and mean of the subsequent test period. ANOVAs and follow-up *t*-tests indicated significant pre–post changes in diastolic BP reactivity in both groups. Systolic BP reactivity significantly decreased in the exposure group, while there were no significant effects for HR. Using a similar design, Öst et al. (1989) compared applied tension, AR, and applied tension/AR combined in the treatment of blood phobia. The authors used a stress testing procedure similar to Öst, Lindahl, et al. (1984) but assessed SC in addition to HR and BP. HR changed significantly from pre- to posttreatment in all groups. There was a significant difference between pretreatment and follow-up reactivity in both the AR and the applied tension/AR groups. Diastolic BP reactivity did not change over time, while there was an interaction effect for SC reactivity. SC decreased over time in the applied tension/AR group, but remained relatively stable in the other groups. Thus, six of the seven studies of Öst's AR treatment protocol failed to show that muscle relaxation led to larger reductions in physiological activation than other therapies. In addition, none of them included sEMG measurements of muscle tension, the target of this therapy.

Beck, Stanley, Baldwin, Deagle, and Averill (1994) compared CT and relaxation training (based on Bernstein and Borkovec's manual) in PD patients, while controlling for time effects with a waiting list group. Sixty-four patients underwent psychological assessment before and after the 10 weekly therapy sessions, and at follow-up. At the same time, SC, HR and upper trapezius sEMG were assessed in each subject during a 3 min resting baseline. Although psychological measures confirmed that the treatment was successful, no significant effects were found for sEMG or HR. There was a significant group by time effect for SC, which was attributed to elevation in SC at pretreatment in patients in the relaxation condition. Baselines, however, are an inadequate test for either cognitive or relaxation therapy changes, since baselines will not reflect cognitive changes if the sensations and thoughts that trigger panic do not happen to be present during the baseline, in which case the patient will not feel threatened and will not be motivated to exercise those skills.

Physiological measurements have been common in investigations of the effectiveness of exposure for specific phobia, where relaxation apparently can be a helpful adjunct. For example, in two studies McGlynn and colleagues (McGlynn, Moore, Lawyer, & Karg, 1999; McGlynn, Moore, Rose, & Lazarte, 1995) repeatedly exposed snake phobics to caged snakes. Half of the subjects had received between four and six sessions of consecutive muscle relaxation training based on Bernstein and Borkovec's manual. After completion of treatment or waiting, HR and SC were recorded during a baseline procedure and during snake exposure. Both studies indicated that the baseline to exposure change in cardiac or electrodermal activity was lower in participants who had received MRT. In addition, treated participants reported less fear during the exposure sessions.

Muhlberger, Herrmann, Wiedemann, Ellgring, and Pauli (2001) investigated the effects of virtual reality exposure versus MRT in flight phobics by randomizing them to one of the two conditions. The interventions were given in 60 min sessions. Participants completed a virtual reality flight before and after treatment during which HR and SC response were measured. Psychometric data were assessed before and after treatment, and at 3 months follow-up. Both treatments were effective, and clinical improvement was maintained at follow-up. Questionnaire data indicated that virtual reality therapy led to more fear reduction than MRT. HR and SC were lower during the posttreatment than the pretreatment assessment. An interaction effect for SC indicated that the SC decline from pre- to postassessment was greater for the virtual reality group.

This experiment shows some physiological evidence for relaxation. Unfortunately, the authors did not use a waiting-list control group, which would have told us whether the changes in the groups over time were because they simply adapted to the laboratory setting or because their specific phobic anxiety decreased. The relaxation training only lasted for 1 h, and may not be comparable to studies with PD or GAD patients that typically employ MRT with six sessions or more.

Strangely, publication of well-designed studies that clearly document the clinical efficacy of muscle relaxation in many DSM-IV anxiety disorders has yet to spark an interest in what goes on physiologically during the treatment, in spite of its explicit physiological rationale. Only one recent study of relaxation therapy in anxious patients, whose outcome was described above, conducted any kind of physiological assessment (Lundgren et al., 2006). Lundgren and colleagues measured HR, SC, and frontalis sEMG at baseline and during threatening situations (fearful video segments) in 127 dental phobics before and after treatment. Phobic patients were divided into subgroups based on the etiology of their fear and their psychophysiological response style, and randomized to CT or MRT with frontalis sEMG biofeedback. The treatments reduced dental fear, and there were overall time effects for the three physiological measures. Unfortunately, the authors did not report if the type of treatment (CT vs. MRT-biofeedback) moderated physiological improvement, but focused on the treatment by etiology and treatment by response style interactions. Neither of these interactions was significant.

As is apparent from this brief review of physiological assessment of muscle relaxation therapy in anxiety patients, evidence for the basic assumptions justifying the treatment is scanty. (1) We did find evidence in cross-sectional studies that certain muscles are physiologically tenser in GAD patients than in normal controls (e.g., Hazlett et al., 1994; Hoehn-Saric & Masek, 1981; Hoehn-Saric et al., 1989) and in one study, that muscles are tenser in PD patients (Hoehn-Saric et al., 1991). However, in only one study was greater tension observed at pretreatment, which then declined with treatment (Miller et al., 1978). Whether patients were experiencing their disorder-specific symptoms during physiological measurements was not assessed in these studies. Nor was a distinction made between sitting quietly and relaxing: Either participants were asked to do both at the same time, or it was assumed that sitting quietly would automatically lead to relaxation. (2) Few MRT studies examined muscle tension at all, and even fewer recorded both sEMG and autonomic measures. In some cross-sectional studies muscle tension seemed to be abnormal in GAD patients while autonomic measures were normal (e.g., Hoehn-Saric et al., 1989). None of the treatment studies showed significant correlations between EMG and autonomic parameter reduction and over the course of therapy. (3) None of the treatment studies showed that reductions in muscle tension and measures of anxiety before and after therapy were positively correlated. (4) Only the Miller et al. (1978) study of dental phobics documented a progressive decline in muscle tension across sessions. Besides this study, there is no evidence that muscle relaxation is a skill that patients can learn. (5) Only a few studies have gone beyond reporting statistical improvement with MRT by documenting clinically significant reductions in distress and functional impairment due to anxiety. For example, Öst and Westling (1995) found 82% of PD patients to be panic-free 12 months after completion of AR therapy. In another study, 67% of GAD patients who had received AR therapy showed clinically significant improvement at 12 months follow-up (Öst & Breitholtz, 2000).

8. General conclusions

Recent well-designed treatment studies have shown that MRT, a now rather old nonpharmacological treatment method, is clinically effective in treating two recently defined

anxiety disorders, PD and GAD. These findings come at a time when clinical psychology not only has developed at least equally effective treatments for these two disorders, cognitive restructuring and exposure, but also has elaborated theories of treatment that can explain in nonphysiological terms whatever good MRT does. To this theoretical skepticism can be added doubts about the validity of the assumptions of muscle relaxation therapy, the most basic of which is that patients who will benefit from it, enter therapy with elevated muscle tension, and if therapy is successful, leave it with a newly learned ability to relax their muscles at will. Relaxing muscles will lead to less tension and anxiety in the psychic, physiological, and behavior realms, not just to less tension in a single muscle or group of muscles.

We have reviewed literature relevant to these assumptions and found that evidence for them is mixed at best. For example, some PD studies suggest that distressed patients have elevated muscle activity and activation of the sympathetic nervous system compared to controls, while others do not. In GAD, elevated muscle tension seems not to be accompanied by autonomic activation. Some of the clinically best studies did not attempt to measure muscle tension. Those that did, assumed that results from the frontalis and forearm flexor sEMG could be generalized to the rest of the body, but the low correlations reported between different sEMG sites, argue against a general tension factor.

The more recent alternate explanations of why muscle relaxation works are theoretically appealing and imply that MRT is a therapeutic detour. Any improvement by MRT may be brought about entirely cognitively, namely that patients learn an increased sense of control and new ways of thinking. During the relaxation procedure, the patient is exposed to frightening thoughts and somatic sensations, which gradually abate. Having weekly contact with a trained, licensed health care provider raises the patients' confidence that their difficulties are surmountable. However, these propositions are unproven. PMR may work exactly as its rationale asserts: when people are taught to relax their muscles deeply, they learn to banish thoughts, feelings, and bodily sensations of anxiety.

Can research help here? Although it may be impossible to prove that changes in cognition are causes rather than effects of changes in anxiety (Roth, Wilhelm, & Pettit, 2005), we can imagine research results that would rule out a causal effect of muscle relaxation. For example, MRT does not work as advertised if sEMG are completely uncorrelated with improvement in subjective distress or if muscle relaxation does not really cause muscles to relax. Better tests of these assumptions are possible with proper attention to experimental methodology and design. Muscle tension and autonomic indicators of arousal need to be assessed before, during, and after MRT. Relaxation treatment protocols should be similar to those used successfully in recent clinical studies. Studies should use a randomized controlled design, in which the treatment group is compared both to healthy controls and to a delayed treatment or alternate therapy group. Technically adequate recording of multiple muscles is essential. Temporally parallel self-report and physiological measurements are necessary to answer questions about therapy mechanisms.

We believe that despite justified doubts about their assumptions, physiologically rationalized methods for treating stress and anxiety such as MRT, breathing training (Meuret, Wilhelm, Ritz, & Roth, 2003; Meuret, Wilhelm, & Roth, 2001, 2004), and various kinds of biofeedback, are here to stay. Competing therapy methods are not always successful: Some patients are resistant to cognitive therapy rationales and procedures, and some find exposure protocols frightening to undertake. In our experience, the rationale of MRT is convincing to most anxious patients, who readily accept its procedures. Most patients believe in the therapy initially, and if the therapy helps them, their belief, as well as that of the therapist, is strengthened. The cost of MRT should be less than that of cognitive or emotion-oriented methods because MRT can be applied in

standardized ways requiring less therapist training. Yet, simply knowing that MRT therapies are clinically effective is not enough. To be able improve them and to apply them optimally, we must have a better idea of how and for whom they work.

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