Review

Muscle tension in generalized anxiety disorder: A critical review of the literature

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ABSTRACT

Background: Generalized anxiety disorder (GAD) is a prevalent, disabling, and often chronic disorder. With a typical recovery rate of only about 40% with current psychological treatments a better understanding of potential psychophysiological mechanisms is vital.
Methods: Since the most discriminative somatic symptom of GAD compared to other anxiety disorders is muscle tension this review qualitatively examines the literature on muscle tension as it relates to GAD and muscle relaxation therapy for GAD patients.
Results: Muscle tension in GAD is poorly understood. Experimental studies refute the often-assumed direct relationship between anxiety and muscle tension. However, muscle relaxation therapies have been as effective as cognitive interventions directly addressing the defining symptom worry.
Conclusions: Muscle tension in its objective and subjective representations may play a role in GAD through various pathways that are testable. Future research needs to better examine the different aspects and functions of muscle tension in GAD.

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First defined in 1980 with publication of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1980) generalized anxiety disorder (GAD) is a rather new diagnosis, yet may be today's most common anxiety disorder (Hoehn-Saric, 1998). According to a review of Wittchen and Hoyer (2001) lifetime prevalence estimates for GAD in the general population (5% lifetime and 3% 12-month prevalence) are remarkably stable, despite the many changes of the diagnostic criteria over time. The latest diagnostic criteria define excessive anxiety and uncontrollable worry as the essential feature of GAD (American Psychiatric Association, 2000) accompanied by at least three additional symptoms from a list that includes restlessness, being easily fatigued, difficulty concentrating, irritability, muscle tension, and disturbed sleep. Joormann and Stober (1999) investigated in a nonclinical sample the correlation between these six somatic symptoms and pathological worry and found that only muscle tension showed a unique and substantial correlation with pathological worry across all their analyses. This suggests that muscle tension plays a prominent role among the somatic symptoms of GAD and may be related to worry, the cardinal feature of GAD.

Given prominence of muscle tension in GAD efficacy of various muscle relaxation interventions for GAD has been evaluated and shown to be effective. In their analysis of outcome studies Fisher and Durham (1999) found the most effective treatments for GAD to be both individual cognitive-behavioral therapy (CBT) and applied relaxation (AR). Even though these two treatments target different symptoms of GAD they have comparable efficacy (Arntz, 2003). However, unlike other anxiety disorders whose psychological treatments have been found to be highly effective, for example CBT programs for panic disorder, posttraumatic stress disorder, and social phobia (Butler, Chapman, Forman, & Beck, 2006), GAD remains a difficult disorder to treat. At a 6-month follow-up about 60% of GAD patients will have shown significant improvement but only 40% will have recovered (Fisher & Durham, 1999).

Hence, there is an evident need for a better understanding of the relationship between the somatic symptom of muscle tension and anxiety or worry in GAD. This review explores the various psychological and physiological constituents in this relationship, pointing out apparent methodological difficulties in their accurate assessment in most studies. Since recent systematic reviews are lacking, the efficacy of psychological treatments for GAD incorporating muscle relaxation will be compared. Although evidence from treatment studies is circumstantial, results may hint at possible mechanisms. Finally, the discussion attempts to integrate findings and expand the current view by suggesting complementary pathways contributing to muscle tension in GAD.

1. Assessment of muscle tension

Muscle tension is a complex psychophysiological phenomenon that has both subjective and objective components. Thus, the question arises how muscle tension has been assessed in studies on anxiety and how valid and reliable assessments have been. Most studies of muscle tension and anxiety rely either solely on self-report or physiological measurement in various forms. Studies that have assessed both can provide information on the concurrent validity of each assessment modality and guide a critical evaluation of studies on muscle tension in GAD.

1.1. Self-report measurement

Measuring patients' symptomatic experience by questionnaires or standardized interviews is the gold standard method for evaluating the presence and severity of a mental disorder. Thus, most outcome studies of GAD treatments have used questionnaires to assess treatment efficacy. Often questionnaires included the item "feeling tense." An apparent problem with this term is that it can be interpreted as tension in the muscles or as a psychic or emotional state of tension. In fact, in a study of Sainsbury and Gibson (1954) on symptoms reported by anxious patients, "feeling of tension" was mostly described as a "feeling of tightness, as if muscles taut", "stiffness of the muscles", "being cramped", and "unable to relax my body". However, besides the majority of patients describing tension as something experienced exclusively in their muscles, 25% of patients experienced it solely as mental state, using phrases such as "on edge," "keyed up," "over-alert all the time," "jittery and unable to settle." Hence, the term muscle tension may be interpreted quite differently by different individuals. Furthermore, anxiety patients' interoception of muscle tension may be more sensitive to subtle changes in muscular activity compared to healthy subjects, resulting in biased ratings on items relating to tension—similarly to what has been observed with self reported heart rate in patients suffering from panic disorder (Ehlers & Breuer, 1996). In summary, self-reports of muscle tension exhibit a restricted validity and therefore need to be interpreted with caution.

1.2. Physiological measurement

Electromyography (EMG) measured on the skin provides a noninvasive way of directly assessing physiological muscle activity. It is a technology that has been known for many years and has been used, amongst others, as a biofeedback method to teach patients to relax their muscles. EMG is based on well-understood electrophysiological properties of muscles. When a muscle contracts an electrical signal is generated along the muscle fiber. This so-called action potential spreads from the muscle to the skin where it can be recorded with electrodes. The total amount of recorded voltage is proportional to the number of muscle fibers contracting simultaneously. Unfortunately, as pointed out by Fridlund, Cottam and Fowler (1982), the voltage of the integrated EMG signal depends on several factors in addition to the aggregated action potentials. These include tissue noise, amplifier noise, and electrical conductance of the skin surrounding the electrode site. Furthermore, the EMG differs between individuals depending on neural density, size of muscle fibers, and thickness of electrically insulating fatty deposits

beneath the skin. Hence, it is difficult to compare EMG measurements between individuals within a study and even more between studies, especially if the measuring procedure is inadequately described. Fridlund and Cacioppo (1986) developed helpful guidelines for human electromyographic research, but these standards have not always been kept. For example, the inference that relaxed muscles show electrical silence at times can only be made if amplifier system noise is taken into account (DeVries, 1965; Sagberg & Kveim, 1981). Without this correction system noise can be misinterpreted as muscle activity.

Electrode placement and number of sites measured are other important issues for a meaningful and reliable EMG measurement. Assuming that a single muscle can represent general body tension (Budzynski & Stoyva, 1969) many studies have measured muscle tension in only one muscle, usually the *musculus frontalis* (forehead) or the forearm extensor muscle. However, this can obviously be flawed and may result in unrepresentative and unreliable estimates of general body tension. The current consensus is that EMG can be a valid physiological measure of muscle tension if recorded carefully according to approved guidelines (Fridlund & Cacioppo, 1986), yet inferences about general muscle tension always need to be supported by simultaneous recording from more than one site. Studies on anxiety only seldomly assessed multi-site EMG, likely weakening the reliability and validity of these measurements as objective indicators of overall muscle tension.

1.3. Concurrent assessment by self-report and physiological measurement

Only moderate concordance between experience and physiology has been observed in basic emotion research even with elaborate measurement designs (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). On the other hand, by focusing on specific bodily symptoms and their putative physiological correlates rather than on global constructs such as anxiety or other emotions much higher concordance rates have been demonstrated in psychophysiological research (Wilhelm & Roth, 2001). Relatively high concordance might thus be expected especially in the case of muscle tension since it can be perceived if attention is put towards it, which is in stark contrast to autonomic changes like heart rate or skin conductance. However, if each assessment method carries substantial error variance as we pointed out above one cannot expect a close relationship. Thus, a lack of correlation between self-reported muscle tension and EMG measurement does not necessarily mean that subjects are not able to accurately report the physiological tension of their muscles but may reflect inadequate assessment methods.

In the 1970s and 1980s EMG biofeedback was thought to be an effective treatment for decreasing muscle tension and producing general relaxation in anxious patients, and this literature is especially interesting in this context. For example, Sime and DeGood (1977) investigated the effect of frontalis EMG biofeedback on subjective awareness of frontalis muscle tension in anxious women. Even though they found a relationship between subjective awareness of

muscle tension and the reduction of muscle tension (frontalis EMG) after biofeedback training, their results suggest that self-reported estimates of tension are unrelated to EMG in subjects with minimal or no training. Shedivy and Kleinman (1977) found in non-anxious subjects that decreases in frontalis EMG during biofeedback training were accompanied by neither decreases in neck muscle activity nor increases in subjective relaxation. Lehrer, Batey, Woolfolk, Remde, and Garlick (1988) tested Edmund Jacobson's assumption that the repetition of tense-release cycles increases the accuracy of self-perception of muscle tension (Jacobson, 1970). Although correlations between self-report of tension and EMG were moderate during relaxation, they did not increase across successive tense-release trials, indicating no increase in self-awareness of muscle tension, at least in the non-anxious subjects tested.

Importantly, McLeod, Hoehn-Saric, and Stefan (1986) investigated the extent to which complaints of muscular tension in GAD patients correspond to actual EMG activity. Subjects diagnosed with GAD according to DSM-III filled out the Somatic Symptoms Scale (Hoehn-Saric, 1981) and the State-Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) questionnaires. During a mild stressor they rated how they felt at a given moment using the Body Feeling Analog Scale (Tyrer, Lee, & Alexander, 1980) while frontalis and gastrocnemius (lower leg) muscle EMG were continuously measured. Under a mild stress condition gastrocnemius EMG activity increased but no significant changes were seen in frontalis EMG activity or in ratings of muscle tension. Correlations between physiological recordings and structured interview scales concerning muscle tension were absent. While patient reports of sweating and heart rate appeared to be related to the direction of actual changes in skin conductance and heart rate, self-reports of muscle tension were not reliably reflected by the EMG measures.

In summary, there is only little support for concordance between physiological measures and subjective estimates of muscle tension, which is at least to some degree due to limitations of assessment methods used in studies (e.g., a limited sample of muscles assessed, unspecific language), and to some degree a reflection of inaccurate interoception.

Reducing these limitations requires carefully conducted multi-site EMG measurements (e.g., eight sites: forehead, neck, two shoulders, two lower legs and two forearms), ideally derived from both experimental (laboratory) as well as real-life observational (ambulatory) settings. In addition, self-report items on muscle tension need to be phrased very specific, clearly identifying the muscles patients describe as being physically tensed, thereby avoiding ambiguous interpretation.

2. Muscle tension and anxiety

Most treatment studies incorporating muscle relaxation utilize self-reported changes in anxiety – not muscle tension – as outcome measure. This raises the question whether self-reported anxiety and physiologically measured muscle tension correlate across repeated measurement times. In a study with healthy participants, Paul

(1969) found that brief relaxation training compared to a self-relaxation control condition produced self-reported and physiological (EMG) changes in the direction expected for anxiety reduction, suggesting that there may be a significant within-individual relationship. However, Raskin, Bali, and Peeke (1980), who evaluated this directly, did not find statistically significant correlations between muscle tension changes (EMG) and anxiety changes in their study of treatment of clinically anxious subjects. Individuals who had the most substantial reductions in self-reported anxiety were not different from those with the smallest reductions in terms of their EMG scores or treatment-related changes in these scores. Burish, Hendrix, and Frost (1981) confirmed these findings with non-anxious subjects and found that while frontalis EMG levels were significantly reduced, this did not relate to self-reported anxiety or autonomic indices of physiological arousal (pulse rate, skin temperature, finger pulse volume).

Rice, Blanchard, and Purcell (1993) randomly allocated 45 subjects suffering from generalized anxiety, of which 38 met DSM-III criteria for GAD, to four treatment conditions or a waitlist. The four conditions were eight sessions of frontalis EMG biofeedback, biofeedback to increase EEG alpha, biofeedback to decrease EEG alpha, and a pseudo-meditation control condition. All subjects participated in a psychophysiological assessment at pre and post treatment during which they were confronted with two mental stress conditions (mental arithmetic and imagination of a personally stressful scene). While all treated subjects showed significant reductions in self-reported anxiety, there was no differential pre–post change in frontalis EMG. Although the EMG biofeedback group showed an almost threefold within-session decrease in EMG over the course of the training compared to the other groups, the difference between groups was not statistically significant.

Several studies have found elevated muscle tension (measured by EMG) between anxious and healthy subjects only during or directly after a mild stress situation (e.g., auditory noise) but not during a relaxed state (Balshan, 1962; Goldstein, 1964; Malmo, 1970). In contrast, several other studies have found reliable differences between anxious and nonanxious groups even during instructed relaxation (Fridlund, Hatfield, Cottam, & Fowler 1986; Hazlett, McLeod, & Hoehn-Saric, 1994; Hoehn-Saric, Hazlett, Pourmotabbed, & McLeod, 1997; Hoehn-Saric, McLeod, & Zimmerli 1989; Sainsbury & Gibson, 1954; Smith, 1973). Based on these studies, elevated muscle tension appears to be a consistent physiological finding related to anxiety.

At first sight one may assume that there is a close relationship between neuromuscular activation and the fight-or-flight response related to anxiety (Hess, 1957). Consideration of the neuronal pathways does not support this, however. In brief, the physiological reactions associated with anxiety are for the most part controlled by the autonomic nervous system (ANS) which is divided into the sympathetic (SNS) and the parasympathetic nervous system (PNS). The SNS controls those activities that are mobilizing during stress and anxiety. This reaction is also called the ergotropic reaction, corresponding to the fight-or-flight response (Hess, 1957) and includes acceleration of heart rate, dilation of pupil, increased blood pressure, increased blood sugar, enhanced blood flow to the skeletal muscles, decreased blood flow to the internal organs and extremities, and increased sweating. However, the SNS does not control muscle tension. Physiologically opposing activities under PNS control serve its basic functions of rest, repair, and relaxation of the body and restoration of energy stores: decreases in heart rate and blood pressure, stimulation of the digestive system, pupillary constriction, sexual arousal, resting, and sleep, but again there is no direct link to muscle tension (Andreassi, 2000).

One would expect that people experiencing chronic anxiety, as is the case with GAD, displayed all the physiological reactions controlled by the SNS as mentioned above. Interestingly, Hoehn-Saric and Masek (1981) found that GAD patients reporting anxiety respond physiologically predominantly with disturbed sleep and increased muscle tension rather than with autonomic sympathetic signs. These findings were confirmed in a further study (Hoehn-Saric et al., 1989) in which GAD patients differed from healthy controls during baseline rest periods in physiological muscle tension but not in autonomic activity (heart rate, blood pressure, skin conductance, and respiration rate). Hoehn-Saric et al. consequently concluded that GAD is not characterized by autonomic hyperactivity but by muscle tension as the unique, distinguishing physiological characteristic.

Even long before GAD had been added to the DSM-III, Horvath and Fenz (1971) were able to differentiate anxiety patients showing primarily symptoms of elevated autonomic arousal (e.g., elevated heart rate) from patients showing primarily heightened muscle tension on the basis of EMG measurements, suggesting the existence of a subgroup of anxiety patients who react predominantly with muscle tension rather than with activation of the SNS as later confirmed by Hoehn-Saric et al. (1989). Thayer, Friedman, and Borkovec (1996) criticized the conclusion of Hoehn-Saric et al., in that their studies of autonomic activity in GAD tended to focus on the SNS, neglecting the potential role of the PNS. Thayer et al. (1996) found in their study that persons with GAD were characterized by lower cardiac vagal activity as evidenced by shorter cardiac interbeat interval and lower high-frequency (HF) spectral power, even across the baseline conditions of their study. They suggested that GAD patients have tonically weak vagal and strong sympathetic cardiac autonomic control and that GAD symptoms like irritability, restlessness, difficulty concentrating and disturbed sleep may be associated with low vagal activity. Further, they concluded that worry and ANS activity are linked, with abnormalities possibly being caused by the PNS.

However, findings in GAD of self-reported anxiety with muscle tension but without cardiovascular or respiratory activation (Hoehn-Saric et al., 1989) are difficult to reconcile with the neurophysiological framework mentioned earlier. The typical ergotropic or fight-or-flight reactions associated with anxiety should include SNS activation across different bodily systems. Given that the skeletal muscle system is governed by other mechanisms than the SNS and PNS and is in large part under voluntary control, muscle tension in GAD cannot be understood as

part of the ANS's reaction to anxiety invoking stimuli or situations.

3. Efficacy of treatment for anxiety and GAD focusing on muscle tension

Despite contradictions that largely emerged in the more recent literature, based on a layperson's understanding of a link between muscle tension and anxiety, various anxiety treatments focusing on muscle relaxation have been put forward for a long time. According to Jacobson (1928), anxious feelings and thoughts are impossible when muscles are deeply relaxed, a premise adopted by Wolpe (1958) in his theory of systematic desensitization. However, experiments in which total paralysis was induced by drugs (curare) have shown that intense anxiety can be experienced even when muscles are completely relaxed (Smith, Brown, Toman, & Goodman, 1947), refuting the incompatibility theory. LeBoeuf and Lodge (1980) compared progressive relaxation and frontalis EMG biofeedback training as treatments for chronic anxiety. Even though EMG feedback was much superior in reducing muscle tension (frontalis EMG), it was not more effective than progressive relaxation in reducing self-reported symptoms of anxiety.

Schandler and Dana (1983) compared guided cognitive imagery relaxation, frontalis EMG biofeedback relaxation, and a self-rest control procedure in which subjects were told to relax to as great an extent as they could achieve and to maintain that level of relaxation for as long as possible. Subjects were females with heightened but not pathological subjective anxiety. Physiological measures consisted of frontalis EMG activity, heart rate, and skin temperature. While all procedures were associated with reductions in physiological tension, the EMG biofeedback group displayed little self-reported change in anxiety, while imagery relaxation resulted in significant, consistent reductions in state anxiety. Interestingly, although EMG feedback was associated with consistently larger reductions in physiological tension levels than the other procedures, this condition produced the lowest frequency and magnitude of subjective changes whereas the self-rest control group expressed a positive attitude toward their treatment even though specific tension reductions were not shown.

Some findings suggest that voluntary relaxation may even have paradoxical effects in exactly the situations where it is most indicated. Wegner, Broome, and Blumberg (1997) found that healthy subjects instructed to relax under high mental load or stress exhibited higher skin conductance levels (SCL) than those instructed to relax under low load, and tended to have higher SCL than those under high load not instructed to relax. Their findings suggest that intentional relaxation in a stressful situation may actually enhance stress symptoms and imply that mental load and/or stress are exactly the wrong conditions for the practice of relaxation therapy. Heide and Borkovec (1983) compared two relaxation methods (progressive relaxation and mantra meditation) in patients suffering from general tension. Several subjects displayed clinical evidence of an anxiety reaction during a preliminary practice period, and 30.8% of the total group under progressive relaxation versus 53.8% under mantra meditation reported increased tension due to the relaxation session. This phenomenon, the so-called relaxation-induced anxiety, has been observed and confirmed in further studies (Borkovec, Mathews, Chambers, Ebrahimi, Lytle, & Nelson, 1987; Braith, McCullough, & Bush, 1988; McKeachie, 1985; Norton, Rhodes, Hauch, & Kaprowy, 1985). Braith et al. (1988) investigated the personality characteristics of the chronically anxious subjects that exhibited relaxation-induced anxiety during their study and found that it may be associated with an internal locus of control, a generalized fear of becoming anxious, and a fear of losing control. In subjects displaying these personality traits tensing muscles seems to suppress anxiety while relaxation induces anxiety.

Surprisingly, the total number of controlled outcome studies for GAD therapy is rather low considering the prevalence of the disorder. Overall, both psychological treatments and pharmacotherapy have been shown to be equally effective for GAD (Gould, Otto, Pollack, & Yap, 1997; Mitte, 2005). According to a multidimensional meta-analysis by Westen and Morrison (2001) based on five controlled studies, of which three included muscle relaxation therapy, the average effect size (Cohen's d) of psychological GAD treatments at post-therapy was 0.9, pre-post effect size 2.09 (SD = 0.76), and 52% of the patients were considered significantly improved. Borkovec and Ruscio (2001) evaluated the mean effect size based on 13 controlled studies, of which 9 included muscle relaxation therapy, and found a mean within-group post treatment effect size of 2.48 for cognitive-behavioral therapy (CBT), 1.72 for behavioral (BT) or cognitive therapy (CT), and 2.09 for placebo therapy, while the waiting-list condition (WL) produced an effect size of only 0.01. Mean between-group post therapy effect sizes were 0.26 for CBT versus BT and CT alone, 0.71 for CBT versus placebo therapy, and 1.09 for CBT versus WL. In addition, effective treatment for GAD was found to produce a large reduction in the number of comorbid diagnoses.

In order to directly compare the outcome data of six controlled studies on GAD treatment, of which four included muscle relaxation therapy, Fisher and Durham (1999) gathered the raw data of the Spielberger State-Trait Anxiety Inventory (Spielberger et al., 1983) which has been incorporated as outcome measure in all included studies. The Jacobson method for defining clinically significant change was used (Jacobson, Roberts, Berns, & McGlinchey, 1999) allocating each patient, according to their STAI measures, to one of four outcomes: worse, unchanged, improved and recovered. Based on this, a recovery rate of 40% was found for the sample as a whole with 12 of the 20 treatment conditions achieving only modest recovery rates of 30% or less, while CT and applied relaxation (AR) did relatively well with overall recovery rates of 50–60% at 6-month follow-up. Fisher and Durham (1999) suggested the general conclusion of previous reviews that CBT is the most effective therapy, to be oversimplified and potentially misleading, since AR produced the highest overall recovery rates with 60% compared to 51% with CBT.

We conducted a systematic, keyword-driven (generalized anxiety disorder, psychotherapy) search of the National Library of medicine's database PubMed (March 2008) for an evaluation of the clinical effectiveness of muscle relaxation therapy for generalized anxiety disorder. In order for studies to be included, they had to be published in peer-reviewed, English-language journals. Furthermore, patients had to be older than 18 years with a diagnosis of generalized anxiety disorder and treatment had to include some sort of relaxation therapy. Table 1 displays the 13 controlled studies we found according to these selection criteria. Overall, the results of existing controlled outcome studies demonstrate that muscle relaxation therapy and CBT are the most effective treatments for GAD. These therapies target chronic muscle tension and excessive worry, which are both cardinal features of the disorder. Even though dealing with quite different symptoms of GAD the two kinds of treatments show similar efficacy, with CT/CBT exhibiting a better long-term efficacy than relaxation therapy in several studies. Interestingly, the combination of muscle relaxation therapy and cognitive therapy was not more efficacious than the single treatments alone (Barlow, Rapee, & Brown, 1992; Borkovec & Costello, 1993). In a component analysis of CBT, Borkovec, Newman, Pincus, and Lytle (2002) compared progressive relaxation training (PRT) and self-control desensitization (SCD) with CT or a combination of all. All treatment conditions resulted in significant improvement in anxiety and depression measures which were maintained for 2 years, but no differences in outcome were found between conditions.

The only controlled study incorporating physiological measures of muscle tension is the one by Barlow et al. (1984), which included both subjects diagnosed with GAD (DSM-III) as well as panic disorder (PD) patients. The treatment conditions were CBT (PRT, frontalis EMG feedback and CT) and WL. Physiological outcome measures were frontalis EMG and heart rate. PD patients showed a higher pre-treatment EMG than GAD patients. Compared to WL, treated patients improved not only on clinical ratings of improvement, daily self-monitored measures and self-reports, but also on psychophysiological measures, with a significant pre–post reduction in EMG. Interestingly, there were no significant differences in outcome across diagnoses since GAD and PD patients responded equally well to treatment, despite the PD patients displaying significantly higher somatic responding on both psychological questionnaires and physiological assessment before treatment.

Except for the study by Barlow et al. (1984), we have no evidence that physiological muscle tension is less after successful treatment of GAD. Hence it is not clear whether the efficacy of muscle relaxation therapy, almost always assessed by self-report measures of anxiety, is due to physiological or cognitive mechanisms. In summary, treatment findings refute the assumption that muscle relaxation directly decreases anxiety, corroborating doubts concerning the long assumed reciprocal relationship between muscle tension and anxiety. In addition, these findings raise questions concerning the effective mechanisms underlying muscle relaxation therapy in GAD.

4. Discussion

Current empirical evidence supports the assertion that muscle tension is the most distinctive somatic symptom characterizing GAD patients compared to patients with other anxiety disorders and that GAD can be treated effectively with muscle relaxation therapy. Yet many questions regarding the relationship between muscle tension and anxiety remain unanswered. Findings of several studies suggest that physiological decrease in muscle tension does not necessarily result in diminished experienced anxiety. Although these results need to be viewed with caution because of the often suboptimal assessment methods used, we think that there is in fact substantial discordance beyond that caused by measurement error between the experienced and physiological facets of muscular tension as they relate to anxiety. Thus, the question remains how exactly muscle tension in GAD may be related to anxiety.

4.1. Propositions for muscle tension in GAD

Since muscle tension does not appear to be part of the physiological response complex of anxiety, as discussed earlier, other alternative explanations of the apparent relationship between muscle tension and generalized anxiety need to be considered.

A *first* explanation for self-reported symptoms of muscle tension in individuals suffering from GAD is that the muscle tension reported by GAD patients may be an exaggerated representation of actual physiological changes due to several sources of bias inherent to self-report, such as focused attention, context dependence, and expectancy effects (Wilhelm & Roth, 2001). Consistent with this, several studies have found low concordance between changes in EMG activity and self-reported tension. On the one hand, as we pointed out previously, this may to some degree be due to patients' limited ability to accurately report physiological muscle tension and also reflect poor reliability of an unrepresentative EMG measurement. On the other hand, and to a sizeable degree, anxiety patients' self-reported muscle tension may be more a reflection of central nervous system changes than actual peripheral physiological muscle tension. Damasio and colleagues recently proposed the existence of an "As-If-Loop" as part of their somatic marker hypothesis (Damasio, 1996), supporting the possibility of decoupling between subjective experience of somatic symptoms and somatic reality with a series of neuroscience experiments (Bechara, Damasio, & Damasio, 2000). However, even though self-report measures are questionable regarding their validity for indexing true muscular tension, several studies clearly found elevated physiological muscle tension (EMG) in GAD patients compared to other clinical and nonclinical groups. In order to shed more light on the body of inconsistent findings concerning the correlation between physiologically assessed and self-reported muscle tension, specific somatic questionnaires regarding physiological muscle tension need to be developed and compared to carefully conducted multi-site EMG measurements, as mentioned earlier in more detail.

Table 1
Controlled studies comparing psychological treatments for GAD

Study	Sample size	Treatment conditions	Treatment duration	Measures	Post-treatment outcome	Follow-up outcome
Barlow et al. (1984)	11 PD 9 GAD	CBT + PRT + BF WL	14 weeks	PP CR SR	CBT + PRT + BF > WL	Gains maintained
Blowers, Cobb, and Mathews (1987)	66 GAD	AMT (CT + PRT) NDT WL	10 weeks	CR SR	AMT > NDT + WL	Gains maintained
Durham and Turvey (1987)	41 GAD	CT BT (=RT)	6 months	SR	CT = BT	CT maintained BT declined
Borkovec et al. (1987)	30 GAD	CT + PRT NDT + PRT	6 weeks	CR SR D	CT + PRT > NDT + PRT	Both groups only slight anxiety
Butler, Cullington, Hibbert, Klimes, and Gelder (1987)	45 GAD	AMT (+RT) WL	4–12 session	CR SR	AMT > WL	Gains maintained
Borkovec and Mathews (1988)	18 GAD 12 PD	CT + PRT SCD + PRT NDT + PRT	6 weeks	PP CR SR D	No difference between groups, all groups improve significantly	Gains maintained
Butler et al. (1991)	57 GAD	CBT (no PRT) BT (=PRT) WL	4–12 sessions	CR SR	CBT > BT > WL	Gains maintained
Barlow et al. (1992)	65 GAD	PRT CT CT + PRT WL	15 sessions	CR SR D	PRT = CT = CT + PRT > WL	Gains maintained
Borkovec and Costello (1993)	55 GAD	PRT CBT (+PRT) NDT	6 weeks	CR SR	CBT = PRT > NDT	Gains maintained
Stanley et al. (1996)	48 GAD	CBT (+PRT) SP	14 weeks	CR SR	CBT (+PRT) = SP	Gains maintained
Öst and Breitholtz (2000)	36 GAD	AR CT	12 weeks	CR SR	AR = CT	Gains maintained
Borkovec et al. (2002)	69 GAD	PRT + SCD CT PRT + SCD + CT	14 weeks	CR SR	No difference between groups, all groups improve significantly	Gains maintained
Arntz (2003)	45 GAD	AR CT	12 sessions	SR D	AR = CT	Gains maintained

Note: AMT = anxiety management therapy, AR = applied relaxation (Öst, 1987), BF = biofeedback (EMG), BT = behavior therapy, CBT = cognitive-behavioral therapy, CR = clinicians' ratings, CT = cognitive therapy, D = diary, GAD = generalized anxiety disorder, NDT = non-directive therapy, PD = panic disorder, PP = psychophysiological, PRT = progressive relaxation training (Bernstein & Borkovec, 1973), RT = relaxation training (unspecified), SCD = self-control desensitization, SP = supportive psychotherapy (group discussion), SR = self-reports and WL = wait list.

A *second* explanation of muscle tension in GAD patients is that preoccupation with excessive worry may keep their attention from sensing prolonged static tension in their muscles. In contrast, healthy people may more easily sense that their muscles are getting tense during prolonged postures or repetitive motions and consequently relax muscles that have been overworked by slight changes in posture and movement, which can avoid muscle tension, pain and exhaustion. In the case of GAD patients the uncontrollable worry may interfere with this somatosensory regulation process. Unaware of exhausted muscles GAD patients may develop painful muscle tension more frequently. The experience of tensed muscles after a cognitively challenging situation that requires full attention (e.g., an exam) is probably known to most people. Since this *worry-as-distraction* explanation for muscle

tension has not been brought up in the literature, data confirming or refuting this explanation of muscle tension in anxiety or GAD is not available at this point. To test this proposition we suggest to place subjects on a chair and measure their muscle tension by multi-site EMG and their movement by accelerometers and video ratings while worrying is induced by the presentation of a documentary with worrisome content. Based on our proposition we would expect GAD patients to show higher muscle tension, fewer movements and fewer adjustments in posture than healthy controls.

A *third* explanation may be that GAD patients experience a high degree of stress during daily life and their muscle tension may primarily reflect their stress levels. An association between daily stress and muscle tension is plausible and has in fact been demonstrated. Lundberg

et al. (1999) investigated this relationship among female supermarket cashiers and found a correlation between work stress and muscle tension (EMG). This association has been confirmed in further studies (Lundberg et al., 1994). Hence muscle tension could be understood as a reaction to stress rather than to anxiety. According to Rissen, Melin, Sandsjo, Dohns, and Lundberg (2000), perceived stress may have a specific influence on heightened muscle activity. Furthermore, research by McNulty, Gevirtz, Hubbard, and Berkoff (1994) on chronic myofascial pain has shown that the so called trigger points responsible for muscle pain display higher activity (EMG) during a mentally stressful condition (mental arithmetic) than during a control condition (forward counting). These findings confirm that mental stress can have a specific influence on muscle activity and even muscle pain. In the case of GAD patients we suggest that the uncontrollable and excessive worry represents a cognitive load which may intensify negative affective appraisals of daily hassles and cause stress, consequently leading to tensed muscles and muscle pain.

A more general approach to investigate this proposition would be to measure physiological muscle tension by multi-site EMG in healthy subjects while they are performing a cognitively challenging task (e.g., solving math problems under time pressure) in the controlled environment of a laboratory and compare it to baseline measurements. According to our hypothesis we would expect significantly higher scores of muscle tension during the challenging task condition. In order to test our proposition on GAD patients we suggest assessing physiological muscle tension continually by means of a portable multi-site EMG recording device in an ambulatory setting (e.g., by the Vitaport 3 system, Temec Inc., The Netherlands) and letting subjects rate their preoccupation with worry and the perceived stress thereof in equal intervals on an electronic diary. If our proposition is correct we would expect to record heightened muscle tension particularly when GAD patients are engaged in intense and stressful worrying. As with all ambulatory studies, physiological data need to be adjusted or stratified for physical activity levels to avoid confusing exercise activation with emotional activation (see e.g., Wilhelm, Pfaltz, Grossman, & Roth, 2006; Wilhelm & Roth, 1998a, 1998b).

As a *fourth* and last explanation, muscle tension may be a way of coping with excess arousal caused by anxiety. Grim (1971) investigated the effects of self-induced muscle tension and found that voluntary masseter (jaw muscle) tension increased anxiety in subjects who initially reported low anxiety, while it decreased anxiety in those who initially reported high anxiety. The shift in mood was quite dramatic: initially low-anxious subjects became noticeably irritable and withdrawn whereas initially high-anxious subjects tolerated the procedure quite well and even enjoyed it. Thus, Grim suggested that tensing be explored as an anxiety-reducing technique for highly anxious subjects. In a study by Knost, Flor, Birbaumer, and Schugens (1999), elevated muscle tension was found to reduce pain perception on the level of the central nervous system (evoked potentials) in chronic pain patients. Though the subjective pain ratings were unaffected by the level of muscle tension that was induced, the data still

suggest that tensing muscles has a physiological effect of reducing the perception of pain. The findings of Knost et al. (1999) imply that the anxiety reduction caused by muscle tension as described by Grim (1971) is also based on activities in the central nervous system. The findings that self-induced muscle tension decreases subjective anxiety, while relaxation under high mental load increases physiological stress and in some cases induces anxiety, indicate that muscle tension in GAD may not primarily be a physiological component of experienced anxiety but an anxiety-reducing coping strategy. Thus, anxious individuals may tense their muscles – probably unintentionally and outside of awareness – in order to alleviate the emotional distress of anxiety. If this is true, then the reported muscle tension of GAD patients is not so much a primary symptom of generalized anxiety or excessive worry but mostly a secondary symptom caused by prolonged tension of the muscles in order to decrease emotional symptoms of anxiety and stress. To investigate this hypothesis we propose to induce worrying in GAD patients by presenting a worrisome video and have some subjects tense and others relax their muscles during the procedure. In addition to multi-site EMG measurements subjects shall be asked to rate the degree of worrying, anxiety, and perceived stress before, during, and after the video presentation. Based on our proposition we would expect GAD patients to report less stress and anxiety during and after the muscle tensing condition compared to the relaxation condition.

4.2. Muscle tension and psychological treatments for GAD

The complex and ambiguous relationships between muscle tension and anxiety discussed in this article raise questions about mechanisms underlying psychological treatments for GAD. Cognitively oriented therapies are based on the understanding that many somatic symptoms are consequences of cognitions and therefore aim at changing dysfunctional thinking patterns. In contrast, behaviorally oriented therapies maintain that somatic symptoms induced by the ANS are in reciprocal action with the mind and that therefore emotional change can be evoked through behavior change. Even though excessive worry is the cardinal feature of GAD cognitive treatment directly addressing worry and teaching coping strategies does not yield superior results to relaxation therapy. The fact that diverse treatments for GAD such as CBT and muscle relaxation show more or less comparable results gives rise to several alternative speculations about underlying mechanisms: (1) each of these treatments specifically affects different characteristics of GAD patients (e.g., worry vs. tension), with similar efficacy on overall anxiety reduction. (2) All treatments affect indirectly the same central feature underlying GAD, e.g., distress caused by uncontrollable worries. In this case successful CBT does equip patients with skills to intentionally control the worry while relaxation therapy causes patients to focus on muscles instead of their worries, indirectly giving them a break from worries. Both approaches, though very different, result in patients being less impaired by the distress of worry. Or finally, (3) neither of these treatments

directly target the central feature of the disorder. In the latter case the positive therapy outcome is merely a result of different non-specific features of psychological treatments that impart positive effects on GAD patients, e.g., social support by the therapist (including feeling of taken care of and being understood), self-monitoring of symptoms, expectancy of recovery, effort justification, positive thinking, redirection of focus on problem solving, and behavioral activation.

As reviewed, some studies have shown that significant decrease in physiologically measured muscle tension does not necessarily lead to substantial reductions in self-reported anxiety (LeBoeuf & Lodge, 1980; Schandler & Dana, 1983). However, most muscle relaxation therapy studies repeatedly exhibited strong evidence of an anxiety reducing effect (Arntz, 2003). One reason for a lack of reduction in self-reported anxiety could be inadequate compliance in home practice. Hoelscher, Lichstein, and Rosenthal (1984) investigated the compliance in progressive relaxation home practice among anxious individuals (DSM-III). The time patients spent practicing was measured by a hidden stop-watch in the tape player used exclusively for the relaxation home practice. Comparisons of self-reported and measured relaxation practice revealed a strong tendency for anxious subjects to exaggerate their self-reported practice time. Correlational analyses revealed a significant relationship between anxiety reductions and amount of actual relaxation practice, while in contrast, self-reported relaxation practice was uncorrelated with anxiety reductions. While this finding supports the relationship between relaxation therapy and decrease in self-reported anxiety, it suggests that low correlations in other studies may be due to noncompliance of the subjects. However, it does not provide any proof that decreased muscle tension per se leads to decreased anxiety.

Considering the solid association between muscle tension and worry, an exclusively cognitive process, in conjunction with missing SNS activation in GAD patients (Hoehn-Saric et al., 1989; Joormann & Stober, 1999) it can be assumed that the effective mechanism underlying muscle relaxation therapy may well be more of cognitive than of psychophysiological nature. Borkovec, Robinson, Pruzinsky, and DePree (1983) found that uncontrollable cognitive activity is a salient feature of the worrier and that such activity leads to disruption in attention-focusing ability. With uncontrollable excessive worry being the cognitive core feature of GAD, CBT aims at equipping patients with cognitive strategies to control the worry and shift their attention away from worry. We suggest muscle relaxation therapy might do just the same by shifting the focus from worry to muscle tension/relaxation, resulting in both more relaxed muscles and lessened anxiety. If this is true, then shifting the focus off of worrying might be one effective cognitive component of muscle relaxation therapy and might explain the comparable efficacy of two very different treatments.

4.3. Conclusions and future directions

Based on the many inconsistent findings reviewed here we conclude that muscle tension and its relationship with anxiety is poorly understood. Some, but likely not all inconsistencies are due to unreliable measurement. While muscle tension appears to be associated with anxiety, specifically with GAD, its origin and its relationship with anxiety and worry remain unclear. Based on the presented data and its implications it seems reasonable to suggest that the excessive worry in GAD is causing stress which in turn may be responsible for the observed muscle tension in GAD patients. Thus, muscle tension would merely be a consequence of experienced stress and/or a strategy to alleviate that stress and not directly associated with anxiety. It can be further assumed that there is no direct physiological association between anxiety and muscle tension, contrary to the understanding of Jacobson (1928) and Wolpe (1958). However, a direct relationship can be assumed between excessive worry and mental distress, and between mental distress/cognitive load and muscle tension. Based on this understanding, the efficacy of muscle relaxation therapy may primarily lie in its function of stress-reduction and in helping to distract from the excessive worry by focusing on the muscles.

Further clinical research may help to better understand the nature and function of muscle tension observed in GAD patients. For this purpose, carefully conducted multi-site EMG measurements and specific somatic self-reports, clearly identifying the muscles patients describe as being tensed, may be gathered before, during and after different treatment conditions. If indeed a reduction of physiological muscle tension were found to have no direct influence on self-reported anxiety future enhancements in GAD treatment would more likely be achieved by improving cognitive-behavioral and pharmacological treatments for this disorder. In addition, nonclinical experiments could aim at a better understanding of the causal relationships between anxiety, worry, distress, and muscle tension by manipulating these factors independently. This would help clarify in which combination and to what degree they may interact to produce symptoms of GAD. If muscle tension is primarily caused by the overwhelming cognitive demand and distress associated with worrying, as proposed in this article, then the experimental induction of such distress should also lead to an increase of physiologically measurable muscle tension. If the efficacy of muscle relaxation therapy for the treatment of GAD lies primarily in its function of distracting from excessive worrying, as we assume, then other possible forms of cognitive distraction apart from muscle relaxation therapy (e.g., physical exercise, attentively listening to music, etc.) should yield the same efficacy in reducing symptoms of anxiety as muscle relaxation therapy does, provided that a convincing rationale can be offered to patients that these activities can be therapeutic.

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