Massage and the Gate Control Model

BY

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ABSTRACT

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Purpose: Musculoskeletal pain is a significant problem in the United States, and medical interventions are not always effective in alleviating pain. Complementary therapies such as massage have been shown to have potent effects in reducing pain, stress and fatigue, as well as improving immune function and restoring function to damaged musculoskeletal tissue. However, rigorous evaluation of the mechanisms of massage is still in its infancy. The goal of the present study was to examine potential mechanisms of massage using the theoretical framework of the gate control model, which provides a framework for examining both sensory and cognitive/emotional mechanisms of massage.

Methods: The current study examined the experience of experimentally-induced pain across four study groups in a repeated measures design. Female undergraduate participants and female community participants were randomly assigned to either a no-treatment control group, guided imagery alone group, massage alone group, or massage plus guided imagery group. Pain and affect were assessed after each of three stimulation periods and two rest periods to determine if group assignment has a differential effect on the experience of pain and affect. Relaxation was assessed after the intervention period to determine if group assignment had a differential effect on self-report relaxation level. Heart-rate, respiration, and blood pressure were recorded continuously throughout the experimental period to determine if group assignment had a differential effect on sympathetic or parasympathetic outcomes.

Results: Contrary to study hypotheses there were no group differences in pain threshold, pain tolerance, pain intensity, worst pain intensity or least pain intensity. Pain unpleasantness and residual pain intensity, on-the-other hand, did show group differences. Specifically, pain unpleasantness, increased for the control group, while remaining at baseline stimulation levels for the guided imagery alone, massage alone, and massage plus guided imagery groups. Residual pain intensity remained at baseline stimulation levels for the control and guided imagery alone groups, while decreasing for the massage alone and massage plus guided imagery groups during intervention. Regarding primary affect outcomes, all three intervention groups reported a decrease in unpleasant affect during intervention and recovery, while the control group maintained baseline levels of unpleasant affect throughout the study. Pleasant affect decreased for the control and guided imagery alone groups during intervention and recovery, while the two massage groups were able to maintain baseline levels of pleasant affect throughout the study. In addition, the two massage groups reported significantly greater levels of self-report relaxation during intervention compared to the control group and guided imagery alone group. Preliminary group differences in sympathetic MHR and MIBI activity were found to be mediated by individual affect and self-report relaxation levels.

Conclusions: These findings support past research that recommends massage as an intervention for pain, and suggests that massage produces influences on ascending pain, as well as unique effects on affect and relaxation compared to guided imagery. Results provided some support for the commonly cited hypothesis that massage decreases ascending pain signals, but suggest that massage may alter affective and secondary qualities of pain versus physically interrupting pain signals via the GCM ascending pain

pathway. Overall, these results provide evidence for the value of massage in mental and physical health outcomes.

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Chapter 1

Massage and the gate control model

Background and significance

Pain is one of the leading health problems faced in the United Sates today. Chronic pain is among the most disabling and costly health conditions experienced in this country, and affects between 26% and 30% of the U. S. adult population (Eisenberg et al., 1993; U.S. Department of Health and Human Services, 2006). Pain disables more adults than cancer or heart disease and is estimated to cost upward of $100 billion annually, which is more than cancer and heart disease combined (Bonica, 1987; National Institutes of Health, 1998; U.S. Department of Health and Human Services, 2006). In particular, chronic pain conditions are diagnosed and reported more frequently in women than in men (Björck-van Dijken, Fjellman-Wiklund, & Hildingsson, 2008; Marcus, 2009).

Musculoskeletal pain is the leading cause of work disability in the U.S., with chronic musculoskeletal pain cases accounting for a disproportionately large share of disability cost (Baldwin, 2004). The most common musculoskeletal disorder, mechanical low back pain, affects approximately 53 per 1,000 working American adults and is the 4th most costly physical health condition in the U.S. (Goetzel, Hawkins, Ozminkowski, & Wang, 2003). Medical interventions are available for chronic musculoskeletal pain; however, these interventions are not always effective and complementary treatments such as massage have often been found useful (Field 2000, 2001).

Overall, complementary and alternative therapy researchers have found that massage is effective in treating various pain conditions, and their associated psychological symptoms (e.g., Moyer et al., 2004). Yet, despite evidence for the

effectiveness of massage, the mechanisms of action are poorly understood (Field, 1998). Thus, it was the purpose of this study to expand the scientific literature on the mechanisms of massage by systematically testing three of the most plausible mechanisms of action: (1) ascending touch and counter-pressure, (2) descending affect, and (3) descending relaxation (Field, 1998; Melzak & Wall, 1965; Moyer et al., 2004).

Pain interventions

The most common medical interventions used to manage pain are pharmacotherapy and surgery (Staats, 2002). Although these are effective for some individuals, many continue to experience pain (Faber, Kuiper, Burdorf. Miedema, & Verhaar, 2006; Kalso et al., 2004; McGregor & Hughes, 2002). Furthermore, many medical procedures are associated with moderate to severe side effects. Anti-inflammatory agents often used to treat pain have ceiling effects, and anti-inflammatory agents and narcotics can both lead to cardiovascular decline, gastro-intestinal complications, renal side effects, and decreased central nervous system (CNS) functioning (Carver & Foley, 2001; Field, 2000; Kalso, Edwards, Moore, & McQuay, 2004; Kean, Rainsford, & Kean, 2008). Thus, patients and clinicians often turn to complementary strategies to control and manage pain (Eisenberg et al., 1993; Furlan, Brosseau, Imamura, & Irvin, 2008).

Manual therapy techniques, such as massage, have been shown to have potent effects in reducing pain, stress and fatigue, as well as restoring function to damaged musculoskeletal tissue (Field, 2000; Field, 2001; Field, 2002). However, the mechanisms underlying the positive effects of massage are poorly understood primarily because the research suffers from a variety of methodological limitations, such as lack of theory,

primary dependence on observational data, and lack of active-treatment comparisons groups. Theoretically based, experimental research is needed to systematically examine potential mechanisms of action for the effects of massage on pain.

Massage and pain

Massage therapy is becoming increasingly accepted as an alternative or compliment to standard care in patients with chronic pain. Massage is a relatively simple therapy to teach and to learn (Field, 2000), yet it has been shown to decrease pain in cancer patients (Bardia, Barton, Prokop, Bauer, & Moynihan, 2006), patients with burns, postoperative patients, patients with fibromyalgia (Field, 2002), juvenile rheumatoid arthritis (Field, Hernendez-Reif, Seligman, Krasnegor, & Sunshine, 1997), and neonates (Jain, Kumar, & McMillian, 2006). A review of the literature on massage intervention for low back pain found that massage was more effective than inert treatment (placebo or no treatment), acupuncture, relaxation therapy, or self-education in reducing low-back pain (Furlan et al., 2008). In addition, massage was as effective as corsets and exercise in reducing low back pain and as effective as spinal manipulation and transcutaneous electrical nerve stimulation (TENS) over the course of extended treatment. Massage has also been found to be moderately effective in reducing mechanical neck pain in combination with other treatments such as heat, ice, rest, analgesics, exercise, and education (Aker, Gross, Goldsmith, & Peloso, 1996). A recent randomized, controlled trial found that deep tissue massage reduced experimentally induced pain by 25% to 50%, depending upon assessment technique (Frey Law et al., 2008).

Massage is effective in decreasing symptoms of anxiety and depression (Field et al., 1997; Field et al., 2000; Moyer et al., 2004). These effects are important because pain

patients frequently have co-morbid anxiety and/or depression (Sansone, Levengoodc, & Sellbom, 2004; Van Houdenhove & Luyten, 2006). Massage has consistently been found to reduce symptoms of anxiety and depression in patients with chronic pain, cancer and HIV (e.g., Diego et al., 2001; Post-White et al., 2009; Toro-Velasco, Amoyo-Morales, Fernández-de-las-Peňas, Cleland, & Barrero-Hernández, 2009), as well as reduce symptoms of anxiety and depression in pregnant women, women post-partum, and healthy adults (e.g., Field, 2002; Field, Diego, Hernandez-Reif, Deeds, & Figueiredo, 2009; Field et al., 2000). Thus, massage therapy appears to be effective in reducing pain, as well as effective in reducing the symptoms of psychological distress that frequently accompany chronic pain conditions.

Mechanisms of massage

Rigorous evaluation of the mechanisms of massage is still in its infancy, yet massage therapy has been used for centuries (Goats, 1994) and there is a wide range of hypotheses regarding its mechanisms of action. Notable among these hypotheses are the Gate Control Model (GCM) (see below), increased blood circulation, activation of the parasympathetic nervous system through relaxation, decreased cortisol circulation, increase in release of natural pain killers (i.e., serotonin and dopamine), increase in natural killer cells, endogenous hormone release with touch, and improved affect via social support (Deng, Cassileth, & Yeung, 2004; Field, 2002; Moyer et al., 2004).

Traditional hypotheses of action include increased circulation, increased parasympathetic activity (i.e., lower heart rate and blood pressure) and decreased stress hormone (i.e., cortisol). Research has shown that massage increases peripheral blood flow (Mori et al., 2004), induces parasympathetic changes (Delaney, Leong, Watkins, &

Brodie, 2002), and decreases cortisol levels (Field, 1998; Field Hernandez-Reif, Diego, Schanberg, & Kuhn, 2005). However, much of this literature is characterized by mixed and inconsistent findings and lacks rigor (Button, Anderson, Bradford, Cotter, & Ainslie, 2007; Field, 2001; Goodfellow, 2003; Moyer et al., 2004; Okvat, Oz, Ting, & Namerow, 2002; Shoemaker, Tiidus, & Mader, 1997). Furthermore, these hypotheses of action provide minimal explanation for reported effects of massage on the perception of pain and/or psychological distress.

More recent theories of action include neurochemical and immunological changes induced by massage. A review by Field and colleagues (2005) found that blood serotonin and dopamine levels increased after massage across various populations. In addition, Hernandez-Reif et al. (2004) found increased urine serotonin, dopamine, and natural killer cells after massage in breast cancer patients. Regarding immune function, Kuriyama et al. (2005) found that aromatherapy massage significantly increased $CD8^+$ and $CD16^+$ peripheral blood lymphocytes, indicating an increase in immune functioning. Similar immunological results have been found in HIV patients, with parameters of immune function increasing after massage (Birk, McGrady, MacArthur, & Khuder, 2000; Diego et al., 2001; Shor-Posner et al., 2006).

While documenting improvements in neurotransmitter levels and immune cell levels supports as an intervention for pain and mood disorders, this type of research fails to address how the mechanical manipulation of soft tissue leads to decreased pain, improved mood, or biochemical changes themselves. The question remains as to how massage decreases pain, improves mood, and increases neurotransmitter and immune cell levels.

Proximal mechanisms of massage. To address the question of how massage works, it is necessary to examine the immediate direct (i.e. proximal) effects of massage on the individual. Potential mechanisms include: (a) increased circulation due to the manipulation of soft tissue; (b) hormonal changes that occur with human touch; and (c) activation of the ascending and descending GCM spinal pathways.

For example, the theory that massage increases blood circulation has been tested in physical therapy research and has produced inconsistent results (Button et al., 2007; Shoemaker et al., 1997; Tiidus, 1999). Though increased blood circulation may occur at a local level, no systemic increase in blood flow has been found (Binds et al., 2004; Mori et al., 2004). Thus, it is unlikely that increased circulation is the primary mechanism of massage. Regarding touch, the findings of Grewen and colleagues give good cause to investigate this potential mechanism further. Light, Grewen, and Amico (2005) found that increased partner hugs was linked to higher levels of oxytocin, lower blood pressure, and lower heart rate. Thus, it may be the case that massage affects physiological functioning through mechanisms of touch.

GCM as a methodological model.

The remaining, and most commonly cited theory of action for massage is via the ascending pain pathway of the GCM.The GCM is composed of both an ascending pathway comprising sensory-physical components and a descending pathway comprising motivational-affective and cognitive-evaluative components. The convergence of these two pathways at the dorsal horn of the spinal cord shapes the individual's experience of and response to pain (Melzack & Wall, 1965, 1989). Given the robust framework and

empirical validation of the GCM, this model served as the theoretical foundation for the present study.

The GCM stipulates that nocioceptive signals travel from peripheral nerves to the spinal cord, where sensory messages are then reprocessed and sent to the thalamus, the primary cortical processor of tactile stimulus. Acute pain signals travel along small, myelinated, fast A-delta fibers (Caudill, 2002; Meyer, Ringkamp, Campbell, & Raja, 2006). Sustained pain signals travel along unmyelinated, slow speed, C fibers. Pain signals generated by both A-delta and C fibers can be modified by competing tactile stimuli, such as touch and pressure (counter-pressure) from massage, that travel along faster moving A-beta fibers (Mouraux & Plaghki, 2007). In this manner, the ascending GCM pathway accounts for the proximal mechanisms of sensory input (touch and pressure) for massage.

Ascending signals. The counter-pressure generated by massage on A-beta sensory fibers can act on both local and distal pain fibers due to the referral stimulation produced within a dermatome. Dermatomes are isolated transverse planes of the body that are innervated by cervical and spinal nerves projecting from the spinal vertebrae (Lee, McPhee, & Stringer, 2008). Accordingly, massage can then decrease the overall experience of pain in an individual through activation of cervical and spinal nerves.

Most researchers cite the GCM touch and counter-pressure hypothesis that massage activates ascending touch and pressure fibers, which then interrupt pain signals, as the primary explanation for massage effects (e.g., Field, 1998; Field 2001, Moyer, 2004). This hypothesis provides the most parsimonious explanation for massage effects on pain, and can be extended to those on affect and biochemistry in the presence and

subsequent reduction of pain (e.g., reduced pain may lead to improved affect and improved biochemical markers). However, this hypothesis does not account for the effects of massage on affect and biochemistry in the absence of pain, nor does it account for improvements in affect and biochemistry when no change in pain is observed after massage intervention (e.g., Diego & Field, 2009; Field, Diego, et al., 2009). Thus, it may be that indirect (i.e. distal) social support mechanisms, or mechanisms shown to influence the perception of pain, such as self-report relaxation or hedonic emotions, are involved in massage effects.

Descending, distal mechanisms of massage. Potential existing theories of action that address indirect distal mechanisms of massage include: (a) social support provided by positive patient-therapist interaction and (b) cognitive (and subsequent physiological) relaxation induced by the setting (Moyer et al, 2004). The descending pain pathway of the GCM provides an explanation as to the top-down influences on pain that can occur with changes in affect and cognition. Phenomenologically, the central nervous system and the descending pain pathway work together to interpret and respond to painful stimuli. It is at this point that psychological factors (e.g., thoughts, emotions, motivations) can modify the pain experience (Melzack & Casey, 1968).

The higher centers of the frontal cortex appear to be responsible for the interplay between cognitive-affective activities and the perception of pain (Melzack, 1986). Connections among centers in the brainstem such as the reticular formation, the hypothalamus, and the limbic system account for strong unpleasant emotions and motivations reported by those in pain. In addition, new information is processed in relation to memories of past pain experiences in areas such as the hippocampus and

amygdala (Wall & Melzack, 1983). Projections into the dorsal horns of the spinal cord can then either increase or decrease the rates of nerve firing, thereby moderating the perception and behavioral response to pain.

One psychological variable that may be related to the effects of massage on pain is social support. Multidisciplinary chronic pain interventions that provide some form of physiological, psychological, and social intervention (i.e., biopsychosocial interventions) have been found to decrease the overall experience of pain, pain-related functional disability, and emotional distress (Guzman et al., 2002; Zunin, Orenstein, Chang, & Cho, 2009). Furthermore, a lack of social support relates to increased pain and emotional distress among chronic low back pain patients, as well as the general adult population (Strine, Chapman, Balluz, & Mokdad, 2008; Waxman, Tripp, & Flamenbaum, 2008). Although there has been no research investigating social support related to massage, a well conducted meta-analysis of massage research by Moyer et al. (2004) concluded that the positive effects of massage on affect are similar to those of psychotherapy and that changes in biochemistry may be a distal result of the improved emotional state (i.e., reductions in stress, anxiety, and depression) that accompanies increased positive interpersonal interaction. Given similarities in emotional, biochemical, and immunological outcomes between massage and psychotherapy, the hypothesis that the active ingredient of massage is social support necessitates further investigation.

The GCM specifies that affect or emotion can alter the descending pain signal. Emotion research advocates the use a circumplex model of emotion due to its clear structure, testable predictions, and good reliability across measurement tools and populations (Larsen & Diener, 1992; Russell & Fehr, 1987). The circumplex model

proposes two broad affective dimensions—Pleasant Affect (e.g., happy, cheerful, pleased) and Unpleasant Affect (e.g., unhappy, sad, grouchy), and two broad emotional activation dimensions—Low Activation (e.g., quiet, tranquil, still) and High Activation (e.g., aroused, surprised, active). These four affect dimensions can then be cross-categorized into Activated Pleasant Affect (e.g., enthusiastic, lively), Activated Unpleasant Affect (e.g., annoyed, nervous), Unactivated Pleasant Affect (e.g., calm, at ease), and Unactivated Unpleasant Affect (e.g., bored, dull). Although it remains in debate as to which dimensions comprise the most basic states of affect, the broad dimensions of Pleasant and Unpleasant affect are most similar to the commonly conceptualized mood states of positive affect and negative affect (Larsen & Diener, 1992), which are related to perception of pain.

Painful events typically result in an increase of negative/unpleasant affect including tension, nervousness, and irritability (Affleck, Tennen, Urrows, & Higgins, 1991; Zautra et al., 1995). Related to this point, high rates of depression have been observed among various chronic pain populations (Dickens, McGowan, Clark-Carter, & Creed, 2002). On the other hand, positive/pleasant affect is associated with less pain among patients with cancer (Buck & Morley, 2006), fibromyalgia (Potter, Zautra, & Reich, 2000), sickle-cell disease (Gil et al., 2003, 2004), and rheumatoid arthritis (Potter et al., 2000), as well as hospital inpatients (Kvaal & Patodia, 2000) and healthy adults (Kenntner-Mabiala, Andreatta, Wieser, Mühlberger, & Pauli, 2008). Thus, affect is an important factor in the perception of pain and will be examined in this study as a primary outcome, as well as a potential pathway for the effects of massage.

It is possible that massage reduces pain and improves mood through the induction of relaxation and subsequent activation of descending GCM effects. Guided imagery is a relaxation intervention frequently used to minimize responses to acute and chronic pain, or noxious stimuli (Antall & Kresevik, 2004; Pölkki, Pietilä, Vehviläinen-Julkunen, Laukkala, & Kiviluoma, 2008; Wallace, 1997). Guided imagery involves one's imagination to create pleasant and/or healing mental images that involve the five senses (sight, smell, sound, touch, and taste) (Mobily, Herr, & Kelley, 1993; Owens & Ehrenreich, 1991). Guided imagery can be used to promote relaxation, concentration, and body awareness (Mannix, Chandurkar, Rybicki, Tusek, & Solomon, 1999). It is also used to decrease perceived pain (e.g., Daake & Gueldner, 1989; Geden, Beck, Hauge, & Pohlman, 1984; Spinhoven & Linssen, 1991). The effectiveness of imagery is related to its ability to generate and become involved in sensory or imaginative experiences.

Utilizing the GCM, relaxation strategies work via descending signals from the brain to the spinal cord that reduce the intensity or unpleasantness of perceived pain. Setting characteristics common to massage, such as dim lighting, prompted deep breathing, and relaxing music may encourage relaxation in an individual, thereby decreasing pain and improving affect (Mitchell & MacDonald, 2006; Wallace, 1997). Thus, it is important to examine relaxation as a potential primary mechanism of massage.

Lastly, the GCM specifies that cognitions such as attributions and expectancies are related to the experience of pain (Price, 2000; Price, 1988). Nisbett and Schachter (1966) demonstrated that the attribution of gastrointestinal symptoms to either a placebo pill or electrical shock can alter the pain response and increase pain tolerance. Expectancies (beliefs that a specific behavior will produce a particular outcome) also

produce changes in pain and affective state (Anderson & Pennebaker, 1980; Kirsch, 1990; Kirsch, 1999; Weiner 1985). Furthermore, treatment expectancy has been found to predict post-treatment success of cognitive behavioral therapy (Kole-Snijders et al., 1999), acupuncture, and massage (Kalauokalani, Sherman, & Cherkin, 2001) for low back pain. Past experience with relaxation and massage techniques, as well as experimental expectancies, were measured in the present study in order to assess and control for individuals attributions and expectations of guided imagery, massage, and pain.

In sum, the GCM provides a robust framework for examining and integrating potential mechanisms of massage on the experience of pain and affect. The GCM ascending pathway accounts for proximal mechanisms of sensory input (i.e., touch and pressure), while the descending pathway accounts for top-down mechanisms of affect and cognition (i.e., relaxation, social support, and expectations). In addition, GCM translates well to experimental pain models. Given the exploratory nature of this mechanistic study, along with the desire to control for potential confounds, a chronic pain model was experimentally induced in healthy participants.

Experimental pain models

Several methodologies are used to induce experimental pain: induced muscle soreness (Tang et al., 2008; Thunberg et al., 2005), cold pressor (Mitchell & MacDonald, 2006; Zachariae, Melchiorsen, Frøbert, Bjerring, Bagger, 2001), infrared heat (Kleinböhl et al., 1999), mechanical pressure (Barlas, Ting, Chesterton, Jones, & Sim, 2006), capsule injection or chemically induced pain (Koltzenburg, Torebjörk, & Wahren, 1994; Wasner, Schattschneider, Binder & Baron, 2004), and electrical current stimulation (Houle,

McGrath, Moran, & Garrett, 988; McMullen et al., 2008; Roder, Michal, Overbeck, van de Ven, Linden, 2007; Sharav & Tal, 2004; Stacher, Schuster, Bauer, Lahoda, & Schulze, 1975). While all of these are valid for inducing experimental pain, there are three primary considerations when deciding upon an experimental pain task: theoretical model fit, experimental design, and practicality.

Theoretical model fit. Because chronic pain is primarily transmitted through the activation of slow firing C fibers, in experimental chronic pain studies such as this one, it is important for pain induction to largely activate C fibers in order to simulate the biological experience of chronic pain. Furthermore, it is important to note that if fast-pain A-delta fibers are activated preferentially before C fiber activation, C fiber activation will be reduced (Mouraux & Plaghki, 2007; Tran, Matre, & Casey, 2008). In the current study, there was a further need for minimal A-beta fiber pain activation due to A-beta fibers carrying the touch and pressure signals associated with massage.

Experimental design and practicality. The pain task must be compatible with the study design. If the pain stimulus is to be delivered repeatedly, pain nerves must have time to recover (deactivate) between pain tasks and must not become differentially fatigued throughout the course of the study. Measurement issues, such as the inevitable within and between person variability in pain levels and the ability to quantify pain threshold and pain tolerance, must also be taken into account given study goals and design. Practical considerations also influence study design and methodology. For experimental pain trials there are the likelihood of participant drop-out and ease of pain stimuli administration.

Pain models. Two experimental pain methods that have minimal activation of A-delta and A-beta fibers and allow for change of over time in pain outcomes are capsaicin intradermal injection and electrical current stimulation. Induced muscles soreness, infrared heat, cold-pressor, and mechanical pressure paradigms do not meet these criteria (Ervilha, Farina, Arendt-Nielsen, & Graven-Nielsen, 2005; Meyer et al., 2005; Tang et al., 2008, Thunberg et al., 2005). See Table 1. Capsaicin intradermal injection and electrical current stimulation both robustly activate C fibers while minimally activating A-delta and A-beta fibers (Gerber et al., 2007). They are both effective experimental chronic pain models; however, capsaicin injection is reported to be more painful than electrical stimulation and has longer-lasting effects (Meyer et al., 2005). Electrical stimulation, on the other hand, is reported to be moderately painful and allows nerves to deactivate quickly with minimal nerve fatigue (Houle et al., 1988; McMullen et al., 2008; Roder, Michal, Overbeck, van de Ven, Linden, 2007; Sharav & Tal, 2004; Stacher et al., 1975). Therefore, electrical stimulation was selected for this study.

There are several models for delivering electrical stimulation. Recent research has used electrical stimulation paradigms to measure objective pain threshold through the nociceptive flexion reflex (NFR) test (Emery et al., 2006; Campbell et al., 2008; Guieu, Blin, Pouget, & Serratrice, 1992). While NFR allows for more objective measurement of pain threshold than self report, it does not permit measurement of pain tolerance, which is important in chronic pain models. Pain threshold is barely perceptible pain. Pain threshold varies with age, sex, cultural background, experience, attention, fitness level, etc. (Guieu et al.1992; Melzack, 1973). Despite its importance in measuring central

nervous system reactivity to pain, pain threshold has been found to have little relationship with clinical pain (Gracely, Dubner, McGrath, & Heft, 1978).

In contrast, pain tolerance, defined as the most intense stimulation an individual can endure (Telli & Cavlak, 2006), emphasizes verbal reports of aversion or unpleasantness and is more modifiable than pain threshold by cognitive and emotional variables (Gracely, Dubner, McGrath, & Heft, 1978). The responsiveness of pain tolerance to variations in cognitive and emotional states during experimental pain studies is similar to the responsiveness of chronic musculoskeletal pain to changes in cognition and mood seen in observational studies (Hamilton et al., 2008; Hamilton, Catley, & Karlson, 2007; Leeuw et al., 2007; Van Houdenhove & Luyten, 2006). Thus, it is advantageous to examine both pain threshold and pain tolerance using electrical current stimulation as an experimental model of chronic musculoskeletal pain.

Summary

Musculoskeletal pain is a major concern in the United States due to its associated prevalence and cost of disability. The most common treatments, medication and surgery, are effective for some patients, yet they carry risks and are not effective for all. It is therefore necessary to determine the effectiveness of complementary therapies such as massage for the treatment of musculoskeletal pain. With this aim in mind, it is critical to understand the mechanisms of action of massage. Though massage has been shown to reduce pain and improve mood and immune outcomes, little research has systematically examined potential active mechanisms of massage. The GCM provides a robust framework for conceptualizing and testing hypothesized mechanisms of massage, and translates well to an experimental chronic pain model. The present study used the GCM

to test three proposed mechanisms of massage for reduction of pain: (1) GCM interruption of pain signals via ascending touch and counter-pressure; (2) changes in affect; and (3) induced relaxation, measured by both self-report relaxation and physiological sympathetic and parasympathetic activity. See Figure 1. Given that the GCM is the most commonly cited explanatory theory of action of massage, this study will be an important first step in examining the GCM framework and testing potential mechanistic pathways.

Study model

The purpose of this study was to disentangle the potential unique ascending effects of massage from the descending effects of affect and self-report relaxation. In order to test competing hypotheses about the mechanisms of massage, it was necessary to compare massage alone with several comparison groups and examine the effects of massage on a variety of outcome measures. Specifically, the unique effects of massage on the perception of pain were compared to a guided imagery alone group, massage plus guided imagery group, and no-treatment control group.

With the goal of utilizing a minimally invasive measurement protocol in humans, several assumptions were made based on previous physiological and experimental research. These assumptions are: (1) the touch and pressure produced during light to moderate massage activated ascending A beta fibers; (2) the guided imagery intervention produced activation of cognitive components of the descending pain pathway, and (3) the simultaneous administration of massage and guided imagery allowed for additive intervention effects (Field, Deeds, et al., 2009) of the ascending and descending pain

pathways. Allowing for these assumptions, the study's primary outcome and mechanistic hypotheses were:

1. Primary hypothesis: The effects of massage are delivered via ascending touch and counter-pressure interruption of pain signals. It was expected that pain would be reported as significantly higher following periods of electrical stimulation compared to baseline rest and recovery rest periods. If the primary mechanism of massage was interruption of pain signals via ascending touch and counter-pressure, then the massage alone group and massage plus guided imagery group would report increased pain threshold and tolerance and decreased pain intensity, unpleasantness, current intensity, worst intensity, and least intensity in comparison to the control group. However, the guided imagery intervention would be expected to produce pain reduction through a descending pathway. Thus, it was expected that guided imagery alone, massage alone, and massage plus guided imagery would all lead to less pain than the control condition and that massage alone would yield equivalent or greater pain relief than guided imagery alone. If ascending and descending additive effects occurred for pain relief, the massage plus guided imagery group would report the more relief than all other groups.
2. Competing hypothesis A: Massage operates on pain via descending affective pathways. It was expected that affect would become more unpleasant and less pleasant during intervention stimulation, compared to the baseline rest and recovery rest periods. It was further expected that guided imagery alone, massage alone, and massage plus guided imagery would lead to less increase in unpleasant affect and less decrease in pleasant affect than the control condition. If the primary mechanism of

massage is positive influence on affect, massage alone would yield equivalent or greater effects on affect than guided imagery alone. If additive descending effects occur for affect, then massage plus guided imagery would produce the greatest maintenance of baseline rest affect, compared to all other conditions. Lastly, if massage operates on pain via descending affect, then affect would mediate any observed group differences in pain.

3. Competing hypothesis B: Massage operates via descending relaxation pathways. a) Psychological: It was expected that guided imagery alone, massage alone, and massage plus guided imagery would yield greater self-report relaxation, measured by the relaxation visual analog scale (VAS), than the control condition. (b) Physiological: It was expected that sympathetic activity would increase during intervention stimulation, compared to baseline rest and recovery rest periods. However, it was expected that guided imagery alone, massage alone, and massage plus guided imagery would lead to less increase in sympathetic activity and more increase in parasympathetic activity during intervention than the control condition. If the primary mechanism of massage is top-down relaxation, then massage alone would yield equivalent or greater cognitive and physiological relaxation than guided imagery alone. If additive descending effects occur for relaxation, then massage plus guided imagery would lead to the greatest psychological and physiological relaxation of all other groups. Lastly, if massage operates on pain via descending relaxation, then relaxation would mediate any observed group differences in pain.

It is important to note here that the potential confounds of relaxing setting and differential influence of social support were experimentally controlled for throughout the

study. Other potential moderating variables such as experience, expectations, and sleep were assessed prior to experiment onset and controlled for in analyses.

Chapter 2

Methods

Study design overview

This study draws upon the GCM and integrates physiological and psychological approaches to better understand the influence of massage and guided imagery on an individual's pain response. Participants were randomly assigned in equal numbers to one of four experimental conditions that were delivered at the same time as three electrical stimulation pain trials: (1) no-treatment control (control), (2) guided imagery alone, (3) massage alone, or (4) massage plus guided imagery. Pain experience (pain intensity ratings, pain unpleasantness, and affect) were measured repeatedly.

Participants

Informed consent. Participants were provided an informed consent form approved by the institutional review board to read and sign prior to beginning this study (Appendix A). They were given a full briefing on study procedures and given the opportunity to ask questions and/or decline participation. Participants and experimenters were unaware of assigned experimental condition until just prior to its beginning. No deception was used in this study.

Exclusion criteria. Participants were excluded from this study if they (a) were under 18 years of age; (b) had a serious chronic medical condition (i.e., dermatological disorder, hypertension, heart disease, diabetes, neurological disorders, or respiratory illness), pain condition (i.e., rheumatoid arthritis, osteoarthritis), or injury to the arm receiving the pain stimulus; and (c) had elevated depression symptoms (>10 on the Beck Depression Inventory-II) or elevated anxiety symptoms (>14 on the Beck Anxiety

Inventory). In addition, men were excluded from the study in an effort to reduce cross-gender experimenter effects (Gijsbers & Nicholson, 2005; Kallai et al., 2004). These exclusionary criteria were enacted in order to obtain a physically and mentally healthy population and thus minimize the influence of external factors such as health condition, mood disorders, and/or experimenter effects on participant response to pain stimuli and intervention.

Study sample. A total of 403 undergraduate female students were offered the opportunity to participate in this study for course credit. Study flyers were also displayed at several campus locations in order to advertise the study to female university community members. Community members were offered up to $20 compensation for their time. A total of 118 undergraduate students and 7 community members were screened for this study. Of these 125 individuals, 21 were deemed ineligible due to elevated BDI and/or BAI scores, one was deemed ineligible due to autoimmune disease, and three withdrew from the study prior to completion. A total of 100 participants completed the study. According to plan, data from four participants were classified as pilot data and were not analyzed as part of the study. Figure 2 provides a flow diagram of participant enrollment and retention.

The final sample included 89 undergraduate students and 7 community members (84.4% White, 3.1% Black or African American, 2.1% Asian, 5.2% Hispanic, 5.2% Multi-racial). Mean participant age was 20.13 years (SD = 5.93, range = 18 to 57). This was a mentally and physically healthy sample. Mean BDI score was 3.52 (SD = 2.66); mean BAI score was 5.07 (SD = 3.33). Furthermore, participants reported, in general or typically, low unpleasant emotions (M = 0.66, SD = .36, on a 0 to 4 scale) and moderate

pleasant emotions (*M* = 2.76, *SD* = .62, on a 0 to 4 scale). Mean body mass index was in the normal range at 23.44 (*SD* = 4.47) and mean medical illness index was 7.91 (*SD* = 13.64) on a 0 to 100 scale. Regarding medication use, 4.2% (*n* = 4) reported taking a selective serotonin reuptake inhibitor (SSRI) or serotonin norepinephrine reuptake inhibitor (SNRI), 5.2% (*n* = 5) reported taking a stimulant, 2.1% (*n* = 2) reported taking a steroid, 45.8% (*n* = 44) reported taking birth control, 27.1% (*n* = 26) reported taking a vitamin, and 22.9% (*n* = 22) reported taking some other type of medication.

Randomization. Prior to the study, 160 numbers were randomly assigned to one of the four study groups using nQuery Advisor 7.0 (Statistical Solutions, Cork, Ireland). Numbered group assignments were then sealed in individual envelopes that experimenters opened just prior to the administration of the intervention or no-treatment control.

Apparatus

Physiological reactivity. Heart rate and respiration were measured continuously using the BioMedical Life System (BIOPAC Systems, Inc, Goleta, CA). Three pre-gelled electrodes were placed in a standard Eindhoven triangle formation. Respiration was measured using a respiration belt fitted just below the sternum. Blood pressure was measured every 2 minutes using an automated blood pressure cuff. Participants were fitted with the electrocardiogram electrodes, respiration belt, blood pressure cuff, and electrical stimulation electrodes prior to onset of baseline. All physiological measures were recorded automatically in real time on a laboratory computer with appropriate time stamps.

Mindware Heart Rate Variability Software version 2.6 (Mindware Technologies, LTD., Gahanna, OH) was used to calculate mean heart rate (MHR), mean R-R inter-beat interval (MIBI), respiratory sinus arrhythmia (RSA), low frequency spectra power (LF power, 0.04-0.15 Hz), high frequency spectra power (HF power, 0.15-0.4 Hz), LF/HF power ratio for every 30 to 60 second study interval. RSA and HF power represent parasympathetic nervous activity (Akselrod et al., 1981; Pagani et al., 1986), while LF power and LF/HF ratio represent sympathetic nervous activity (Koizumi, Terui, & Kollai, 1985; Yergani et al., 1993).

Experimental pain stimuli: Electrical stimulation was delivered by the STM100C Stimulator Module and STMISOE Stimulator Isolation Adapter 200 V (BIOPAC Systems, Inc, Goleta, CA) using 2" square muscle stimulation electrodes from VERMED, Inc. (Bellows Falls, VT). Electrical stimulation was delivered to the non-dominant palmar forearm peripheral nerves using a rectangular wave form of continuous direct current administered using 2 ms pulses at 2 ms intervals (Telli & Cavlak, 2006). Stimulation intensity began at 0 volts and increased by 5 volts every 20 ms (every 5 pulses) (Levitt, 1971) until pain threshold and then tolerance was reached. Participants were asked to report when they first felt pain (pain threshold; Phillips & Gatchel, 2000), by pressing a computerized marker button. Next, participants indicated when they could no longer tolerate the pain (pain tolerance; Telli & Cavlak, 2006), by pressing a second marker button. This second marker button was linked to the BIOPAC Life System and automatically terminated the electrical stimulation when pressed by the participant.

At baseline, electrical stimulation was presented in the described ascending manner up to 100 volts or until the participant terminated stimulation. No participant

allowed the stimulation to reach the maximum 100 volts, though several participants reached 90 volts. After baseline threshold and tolerance were established, the presentation waveform was modified such that subsequent stimulation presentations did not exceed baseline tolerance levels. Limiting stimulation to baseline tolerance during subsequent pain trials removed the ability to fully measure change in pain tolerance but allowed for the measurement of change in pain intensity, which is the primary outcome of interest.

Intervention conditions

Social support was standardized between the four groups by having the massage therapist remain in the room during all four experimental intervention periods. In addition, verbal interactions were standardized and kept to a minimal (i.e., instructions and one to two questions regarding management of experiment).

No-treatment control. Participants were asked to sit quietly for the duration of the experimental intervention period, while receiving three trials of electrical stimulation.

Guided imagery alone. Participants listened to a standardized recording of a licensed clinical psychologist providing instructions on deep breathing and a guided imagery scenario, while receiving three trials of electrical stimulation. See Appendix B for outline of guided imagery script.

Massage alone. Participants received massage performed by a certified massage therapist, while receiving three trials of electrical stimulation. Massage consisted of light to moderate massage of the neck, shoulders, back, and upper arms (Field, 2000). See Appendix C for massage protocol.

Massage could not be directly applied to the area receiving the pain stimuli (the right forearm) due to the manner by which stimulation was administered—topical electrodes. Therefore, massage was focused on the thoracic dermatome associated with the arms and forearms. Proximal stimulation of a dermatome plane near the spine leads to referral stimulation, at a reduced level, of the distal nerves in that dermatome (Lee et al., 2008). Thus, nerves in the forearm were mildly stimulated by massaging the cervical and thoracic areas of C6 through T1 and the upper arms. See Appendix D for dermatome body outline.

Massage plus guided imagery. Participants listened to the standardized guided imagery audio recording while simultaneously receiving the massage intervention described above. Participants also simultaneously received three trials of electrical stimulation.

Procedure

Pretest. The study timeline is depicted in Figure 3. Participants were first asked to complete a battery of questionnaires including: medical history, relaxation and massage experience, National Sleep Foundation sleep diary, Frid Scale, Profile of Mood State Short Form-Revised (POMS-R), Personal Affection and Touch Scale (PATS), Beck Anxiety Inventory (BAI), and Beck Depression Inventory (BDI) II. Immediately after completion of the questionnaire battery, the medical history was reviewed and the BAI and BDI were scored in order to determine eligibility.

After eligibility was established, participant height and weight were measured by a research assistant using a standard balance-beam scale. Participants were then seated comfortably in a reclining chair for the duration of the experiment and fitted with

physiological recording devices for heart rate, respiration, and blood pressure, along with electrical stimulation electrodes.

Baseline rest. Participants sat quietly for 10 minutes to establish a resting heart rate, respiration, and blood pressure. Pain experience (i.e., pain intensity, pain unpleasantness, McGill items, and affect) were then measured using pain intensity & unpleasantness visual analogue scales, items from the Short-Form McGill Pain Questionnaire (McGill), and the Profile of Mood State Short Form-Revised (POMS-R), respectively.

Baseline stimulation. Once baseline rest questionnaires were completed, electrical stimulation was presented twice to establish a baseline pain threshold and pain tolerance. Each stimulation trial lasted no longer than one minute and pain threshold and tolerance were assessed at each trial. It should be noted that while stimulation was presented twice at baseline, only the second stimulation trial was used as a final index of baseline threshold and tolerance. This was due to frequent participant error while marking threshold and tolerance during the first baseline stimulation trail. Pain experience was measured again immediately after the baseline pain trials.

Intervention. After baseline stimulation, the envelope containing group assignment was opened and participants were informed of intervention group assignment. They were provided instructions and then proceeded to complete three pain trials while simultaneously participating in one of the four experimental conditions described above. The intervention period lasted approximately 15.5 minutes. Pain trials were administered at 3 minutes, 8 minutes, and 13 minutes. Self-report measures of pain experience were collected immediately following termination of the intervention period.

Recovery rest. Participants were asked to sit quietly for 10 minutes after the intervention period to establish a recovery heart rate, respiration, and blood pressure. Pain experience was again measured at the end of this 10 minute recovery period.

Post-recovery stimulation. After recovery, the last electrical stimulation trial was presented in order to measure recovery pain threshold and tolerance. Pain experience was measured for the last time immediately following termination of post-recovery stimulation.

Measures

All measures and published scales cited here are provided in Appendices E through O.

Medical history. Participants reported basic demographic information such as age, gender, and ethnicity. Participants also reported on health conditions such as injury, trauma, dermatological disorders, chronic pain conditions, hypertension, heart disease, respiratory illness, and medication use. Experience with relaxation techniques and massage were measured using author-generated questions asking if the participant had ever engaged in that activity, and if so how often (1 = a little, 2 = some, 3 = a lot).

Illness severity. Chronic illness severity was coded using the Medical History Severity Index (Benyamini, Leventhal, & Leventhal, 1999). Scores ranged from 0 to 100, with higher scores indicating more severe illness. If participants had co-morbid diagnoses, the highest illness severity score was used.

Beck Anxiety Inventory. The Beck Anxiety Inventory (BAI: Beck, Epstein, Brown, & Steer, 1988; Beck & Steer, 1990) is a 21-item self-report index of anxiety symptom severity. Fourteen items target somatic symptoms and seven items assess

specific cognitive features of anxiety and panic. The BAI subscales have acceptable internal consistency (Cronbach's αs = .92 and .94) and one-week test-retest reliability (*r*s = .67 and .75) (Beck et al., 1988; Fydrich, Dowdall, & Chambless, 1992). The BAI also shows adequate concurrent validity, correlating .58 and .47 with the Trait and State scales of the State-Trait Anxiety Inventory, Form Y, respectively (Fydrich et al., 1992). In the current study, the BAI had poor to modest reliability with α = .43 for the somatic subscale and α = .59 for the cognitive subscale.

The BAI was used to screen participants for potential clinical elevations in anxiety. A cut-off score of 14 was derived from a large sample of non-patient college females (M= 13.46; *SD* = 9.39; Clark, Steer, & Beck, 1994). In the current sample, this cut-off for healthy mood was supported in variance analyses showing that BAI scores of 14 and below did not significantly influence primary pain or affect outcomes (all *p*'s > .05). Thus, the current study used a score of 14 and below to indicate that an individual was likely not suffering from an anxiety disorder. A score of 15 and above indicated that an individual may suffer from an anxiety disorder. Persons with a BAI score above 14 were deemed ineligible and referred to the university psychology clinic.

Beck Depression Inventory-II. The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item assessment of the severity of symptoms of depression. Each item is rated on a 0-3 scale with summary scores ranging between 0 and 63. The BDI-II has adequate internal consistency (α = .93 among college students, α = .92 among outpatients; Beck, Steer, Ball, & Ranieri, 1996; Beck et al., 1996). Adequate validity (e.g., content, factorial) has been demonstrated, and diagnostic discrimination has

also been established in these populations. In the current study the BDI demonstrated modest reliability with $\alpha = .60$.

This scale was used to screen participants for clinical depression. A score of 11 and below indicates that an individual is likely not clinically despressed, whereas a score of 12 and above may indicate the presence of clinically significant depression (Beck, Steer, Ball, & Ranieri, 1996; Beck et al., 1996). Persons with a BDI score above 11 were deemed ineligible and referred to the university psychology clinic.

Frid Scale. The Frid Scale (FRID; Frid, Singer & Rana, 1979) is a 10-item, 5-point scale assessing expectancies and attitudes regarding experimental pain procedures. The measure consists of three subscales including Psychological Involvement in the experiment (4 items), Negative Expectancies regarding the experiment (1 item), and Efficacy and Control beliefs (5 items), and was used to assess pain and experiment expectancies. Two items are reverse coded. Mean subscale scores were calculated by summing subscale items and dividing by the number of items, giving a range of 1 to 5 for each subscale.

In this study the Efficacy and Control beliefs subscale was found to have poor internal consistency ($\alpha = .26$), but was found to inversely correlate with the BAI ($r = -.21$, $p < .05$) and unpleasant affect subscale of the pretest POMS-R ($r = -.26$, $p < .05$) as expected. Reliability and validity of the other two subscales was also poor (i.e., Psychological Involvement $\alpha = .16$; no significant correlations between the Psychological Involvement subscale and other measures of emotion; no significant correlations between the Negative Expectancies item and other measures of emotion). Thus, this measure was primarily used for descriptive purposes and cautiously evaluated for covariance.

National Sleep Foundation sleep diary. Participants recorded their sleep habits for the previous day and night in order to asses and control for the influence of sleep on primary pain and affect outcomes. The National Sleep Foundation 7-day sleep diary (National Sleep Foundation, 1999) was shortened to include only the previous day and night and was used to record sleep duration (time in bed - number of minutes till fell asleep), subjective sleep quality ("When I woke up for the day, I felt: 0 = refreshed, 1 = somewhat refreshed, 2 = fatigued"), and number of awake bouts ("I woke up during the night ___ times"). Subjective sleep quality items were reverse scored so that higher scores indicated better sleep quality.

Personal Affection and Touch Scale. The Personal Affection and Touch Scale (PATS) is a 20-item measure newly constructed by S. Pressman and C. Karlson assessing physical contact frequency (e.g. hugging, pat on the back, massage) via a 5-point Likert scale (1 = very rarely or never, 5 = at least once daily). In addition, one item assesses satisfaction with physical contact (PATS-SAT; min = I want much less contact, max = I want much more contact) and one item assesses discomfort with physical contact (PATS-D; min = always uncomfortable with touch, max = not at all uncomfortable with touch). The two primary subscales assess non-romantic (i.e. friends, family, and acquaintances; PATS-GEN) and romantic (PATS-ROM) physical contact. Because there was little psychometric data available for this measure, reliability and validity were examined as part of the current study. As expected, more frequent physical contact reported on the PATS-ROM subscale was associated with increased experience with massage ($r = .31$, $p < .05$). In addition, satisfaction with physical contact level was associated with decreased hostility ($r = .29$, $p < .01$) and anxiety ($r = .25$, $p < .05$) subscale scores on the POMS-R.

The PATS also demonstrated good internal reliability with α = .86 for the total PATS, α = .83 for the PATS-GEN subscale, and α = .91 for the PATS-ROM subscale.

The Cognitive and Affective Mindfulness Scale - Revised (CAMS-R; Feldman, Hayes, Greeson, & Laurenceau, 2007) measures four aspects of mindfulness, including mindful attention, present focus, awareness, and acceptance. Higher scores on the CAMS-R reflect higher levels of mindfulness. The CAMS-R has demonstrated good overall internal reliability (α = .74 to .77) and convergent validity with other mindfulness measures such as the Mindful Awareness Attention Scale (*r* = .51) and Freiburg Mindfulness Inventory (*r* = .66). The CAMS-R also correlated positively with measures of emotional clarity and wellbeing, and correlated negatively with measures of avoidance, rumination and worry. In the current study, the CAMS-R was used to measure attention to and engagement in the intervention period, and demonstrated adequate reliability with α = .70.

Pain intensity scale. Pain intensity was measured on a 100 mm visual analogue scale with 0 = no pain, 50 = moderate pain, and 100 = worst possible pain (Campbell et al., 2008; Mitchell & MacDonald, 2006). The VAS is a standardized rating scale sensitive to both pharmacological and non-pharmacological pain interventions (Price, Harkins, Rafii, & Price, 1986). In the current study, pain intensity had an overall mean of 52.36 (*SD* = 19.35) and range of 0 to 91. Thus, this item appeared to have adequate variability for analyses.

Pain unpleasantness scale. Pain can be characterized by both intensity and unpleasantness (Melzack, 1975). Pain unpleasantness was measured on a 100 mm visual analogue scale with 0 = none, 50 = moderate, and 100 = most possible unpleasantness.

During the present study, pain unpleasantness had an overall mean of 51.17 (*SD* = 21.40) and range of 0 to 95. Thus, this item appeared to have adequate variability for analyses.

Short-Form McGill Pain Questionnaire (McGill). The Short-Form McGill Pain Questionnaire (SFMPQ; Melzack, 1987) is designed to assess the experience of pain. It consists of 15 descriptors (11 sensory and four affective) that are rated on a four-point intensity scale of 0 = none to 3 = severe. In the current study, three individual items from the Short-Form McGill were used to describe residual pain intensity (i.e., present pain intensity after intervention), worst pain intensity, and least pain intensity on a 5-point Likert scale with 1 = mild, 3 = distressing, and 5 = excruciating. Residual pain intensity, worst pain intensity and least pain intensity items were evaluated individually across participants and were considered face valid.

Profile of Mood State Short From-Revised. The Profile of Mood State Short Form (POMS-SF; McNair, Lorr, & Droppleman, 1992) was revised in the current study to measure five identifiable mood states: tension–anxiety, depression–dejection, anger–hostility, vigor–activity, and fatigue–inertia. Seven items from the Positive and Negative Affect Scale-X (Watson & Clark, 1994) were added to 25 items from the POMS-SF in order to better asses the circumplex dimensions described above: unactivated unpleasant affect, low activation affect, high activation affect, unpleasant affect, and pleasant affect. Participants respond to the items using the following scale: 0 = not at all, 1 = a little, 2 = moderately, 3 = quite a bit, and 4 = extremely. In the current study, internal reliability of the unpleasant affect scale (n = 18) and pleasant affect scale (n = 9) was good with α = .86 and α = .81, respectively.

Relaxation visual analogue scale. Self-report relaxation levels were measured after the intervention period using a 10 cm visual analogue scale (0 = not relaxed at all, 5 = somewhat relaxed, 10 = completely relaxed). This item had an overall mean of 7.25 (*SD* = 1.97) and range of 1 to 10. Although this item was slightly skewed to the relaxed end of the VAS, given the 3:1 intervention to control group participant ratio, this item appeared to have adequate variability for analyses.

Statistical analysis

Sample size determination. It was proposed that 28 participants be recruited for each arm of this study (total *N* = 112) based on a priori power analysis that utilized extant experimental data with electrical stimulation (Emery et al., 2006; Houle et al., 1988; McMullen et al., 2008; Sharav & Tal, 2004) to determine the minimal sample size necessary to achieve power of .80. The primary outcome variable for power analysis was *pain intensity VAS*. Estimates of effect size and variance were based on predicted between group differences. Baseline values for VAS pain intensity (mean ± SD = 60 ± 20.3) are based on a broad literature search of baseline experimental pain values reported in adults on a 100 point VAS. Participants were measured at multiple time points during the experiment; however, the sample-size determination was based on the immediate post-intervention pain assessment. The smallest important difference was hypothesized to be the guided imagery alone treatment group at a 25% relative reduction from baseline stimulation (mean ± SD = 60 ± 20.3) to post-intervention (mean ± SD = 45 ± 20.3).

Missing data: Physiological data. Based on a previous study conducted by this researcher using a similar physiological measurement protocol (Hamilton, Karlson, Luxton, & Nelson, in preparation), missing heart rate and blood pressure data were

expected to occur on an infrequent basis due to computer and/or investigator error. Missing heart beats (missed beats) are uncommon in the proposed young-adult sample, but may increase under stress (van Well, Kolk, & Klugkist, 2008). Missed beats were imputed using Mindware Heart Rate Variability Software version 2.6. Missing continuous cardiovascular data (1.95%) and blood pressure data (4.15%) were imputed using LISREL 8.71 (Scientific Software International, Inc., Lincolnwood, IL) mean estimate multiple imputation strategy with 5 iterations (Tabachnick & Fidell, 2007).

Missing data: All other measures. Missing continuous pre-test data (0.13%), pain threshold and tolerance data (4.41%), and repeated measures self-report data (1.88%) were imputed following recommended procedures using LISREL 8.71 mean estimate multiple imputation strategy with 5 iterations. There were no missing categorical data.

Analyses of results. Data were entered into SPSS 17.0 and analyzed using LISREL 8.71 for repeated measures analyses and SPSS 17.0 for all other analyses. Data were screened for univariate and multivariate outliers and tested for satisfaction of distributional assumptions where required for univariate and multivariate analyses.

Preliminary analyses. Descriptive statistics were analyzed for group differences between completers and non-completers and between intervention groups using Chi-squared for categorical variables and Univariate Analysis of Variance (ANOVA), Mann-Whitney tests for continuous variables, as appropriate. Yates's correction was applied to Chi-square when $df = 1$. Bivariate zero-order correlations were used to test for potential covariates. Any baseline demographic or psychosocial variable that showed group differences at $\alpha = 0.05$ were entered as a covariate in subsequent analyses of the independent effects of the interventions.

Primary analyses. Analyses of within-subject and between-subject changes over time were conducted using multilevel modeling (MLM). MLM facilitates the analysis of data that have a hierarchical (nested) structure, such as repeated measures data (Tabachnick & Fidell, 2007). In this case, 3 measurement periods (baseline, intervention, and recovery) were nested within 96 participants. There were two principle sources of variance: variability between measurement periods and variability between persons. *Level 1* variables were those measured on a repeated basis throughout the course of the study (i.e., pain, affect, physiological outcomes). Variables that were measured once, such as group, medication use, and experimental expectations, contained only between person variance and were modeled as *Level 2* variables. Cross level interaction effects were examined for group x time variables.

Multilevel analyses were conducted according to the following data analytic strategy. First, an autoregressive covariance matrix was used to control for serial dependency in repeated measures (Affleck, Tennen, Urrows, & Higgins, 1994). Second, the change from baseline periods to intervention and the change from intervention to recovery periods were examined separately by using a fixed continuous piecewise model (Cudeck & Klebe, 2002). This piecewise model fixed the end of intervention as the change point in slope function (i.e., node), thereby allowing for independent examination of the two resulting slopes. This also fixed the examination of main group effects at the node. See Figure 4. A piecewise model was utilized due to the expectation that the slope from baseline to intervention would be different from the slope from intervention to recovery. For example, it was expected that unpleasant affect would increase from baseline to intervention and would decrease from intervention to recovery.

In pain and affect MLM equations, outcome (y) was predicted by an intercept (B_0), change from baseline to intervention (TimeRegime1), change from intervention to recovery (TimeRegime2), group assignment (Group), interaction between group and time regime 1 (Group)(TimeRegime1), interaction between group and time regime 2 (Group)(TimeRegime2), potential covariate (Covariate), and random error terms (*e* and *u*). The Level 1, Level 2, and reduced-form equations are as follows:

Level 1: $y_{ij} = \beta_{0j} + \beta_{1j}(\text{TimeRegime1})_{ij} + \beta_{2j}(\text{TimeRegime2})_{ij} + e_{ij}$

Level 2: $\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Group})_j + \gamma_{02}(\text{Covariate})_j + u_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Group})j$

$\beta_{2j} = \gamma_{20} + \gamma_{21}(\text{Group})_j$

Reduced form: $y_{ij} = \gamma_{00} + \gamma_{10}(\text{TimeRegime1})_{ij} + \gamma_{20}(\text{TimeRegime2})_{ij} + \gamma_{01}(\text{Group})_j + \gamma_{11}(\text{Group})_j(\text{TimeRegime1})_{ij} + \gamma_{21}(\text{Group})_j(\text{TimeRegime2})_{ij} + \gamma_{02}(\text{Covariate})_j + u_{0j} + e_{ij}$

Lastly, physiological data measurement provided enough degrees of freedom to allow for consideration of a random slopes model. Treating slopes as random effects allows analysis at the individual level, as well as the group level. Chi-squared analysis of difference between deviance statistics for the fixed slopes and random slopes models was used to determine final model fit. Fixed slopes models included a fixed intercept and fixed slopes, while alterative models included a fixed intercept and random slopes.

In physiological MLM equations, outcome (y) was predicted by an intercept (B_0), change from baseline to intervention (TimeRegime1), change from intervention to recovery (TimeRegime2), group assignment (Group), interaction between group and time regime 1 (Group)(TimeRegime1), interaction between group and time regime 2

(Group)(TimeRegime2), potential covariate (Covariate), and random error terms (*e* and *u*). The Level 1, Level 2, and reduced-form equations are as follows:

Level 1: $y_{ij} = \beta_{0j} + \beta_{1j}(\text{TimeRegime1})_{ij} + \beta_{2j}(\text{TimeRegime2})_{ij} + e_{ij}$

Level 2: $\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Group})_j + \gamma_{02}(\text{Covariate})_j + u_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Group})j + u_{1j}$

$\beta_{2j} = \gamma_{20} + \gamma_{21}(\text{Group})_j + u_{2j}$

Reduced form: $y_{ij} \quad \gamma_{00} + \gamma_{10}(\text{TimeRegime1})_{ij} + \gamma_{20}(\text{TimeRegime2})_{ij} + \gamma_{01}(\text{Group})_j + \gamma_{11}(\text{Group})_j(\text{TimeRegime1})_{ij} + \gamma_{21}(\text{Group})_j(\text{TimeRegime2})_{ij} + \gamma_{02}(\text{Covariate})_j + u_{0j} + u_{1j}(\text{TimeRegime1})_{ij} + u_{2j}(\text{TimeRegime2})_{ij} + e_{ij}$

All other analyses. Analyses of between-subject variance at a single time point (e.g., relaxation VAS, CAMS-R) was conducted using Analysis of Variance (ANOVA). Post-hoc analyses were conduced using simple comparison paired *t*-tests. Estimates of effect size were determined using mean difference scores and η^2. Hierarchical linear regression was used to test for mediating and moderating effects of affect and relaxation on the relationship between group assignment and pain outcomes (Barron and Kenny, 1986). Potentially significant mediating effects were further evaluated using the Aroian version of the Sobel test (Preacher & Hayes, 2004). All tests were two-tailed at $\alpha = 0.05$.

Chapter 3

Results

Descriptive statistics

Two participants withdrew from the current study after completing baseline rest, and one withdrew after being randomized to the massage alone group (see Figure 2). Participants who withdrew (n = 3) prior to completion had significantly higher BDI scores ($M_{withdrew}$ = 8.67, SD = 1.15 vs. $M_{completed}$ = 3.39, SD = 2.65), $F(1,97) = 11.26$, $p < .01$, and marginally higher BAI scores ($M_{withdrew}$ = 8.67, SD = 4.93 vs. $M_{completed}$ = 5.04, SD = 3.34), $F(1,97) = 3.35$, $p = .07$, than those who completed the study (n = 96). In addition, participants who withdrew from the study prior to completion reported significantly higher negative experimental expectations ($M_{withdrew}$ = 3.33, SD = 1.15 vs. $M_{completed}$ = 2.10, SD = .93), $F(1,97) = 4.98$, $p < .05$, than those who completed the study.

For those participants who completed the study, there was no difference between intervention group participants for age, $F(3,92) = 1.13$, $p > .05$, ethnicity, $F(3,92) = 1.70$, $p > .05$, BMI, $F(3,92) = .94$, $p > .05$, illness severity score, $F(3,92) = .76$, $p > .05$, or medication use, $F(3,92) = 1.38$, $p > .05$. By chance, a greater number of women who reported wanting more physical contact were randomized to the massage only intervention group compared to the control group and other intervention groups, $F(3,92) = 2.66$, $p = .05$. No group differences were found for any other psychosocial variable.

For past experience with relaxation, frequency of and comfort with touch, sleep, and experiment expectancies there were no differences between groups (all p's > .05).

Regarding past experience with relaxation techniques, 46.9% (N = 45) reported at least some experience with relaxation massage (*n* = 41), deep tissue massage (*n* = 21), shiatsu massage (*n* = 1), or reiki massage (*n* = 1). 22.9% (N = 22) of participants reported experience with meditation (*n* = 12), guided imagery (*n* = 7), or deep breathing (*n* = 21) relaxation techniques. In addition, 62.5% (N = 60) reported at least some practice of yoga (*n* = 59), tai chi (*n* = 2), or martial arts (*n* = 6), all of which utilize controlling one's breath during practice and may aid in relaxation.

Related to experience with massage and comfort level with touch from a massage therapist, participants reported a moderate frequency of giving and receiving non-sexual touch with a romantic partner (*M* = 38.90, *SD* = 9.19) and a moderate frequency of giving and receiving non-sexual touch with friends, family, and acquaintances (*M* = 25.72, *SD* = 7.31). 82.3% of participants reported being satisfied with their levels of physical contact, whereas 17.7% reported wanting a little more (*n* = 15) or much more physical contact (*n* = 2). The majority of participants reported being comfortable with massage, with most endorsing "not at all uncomfortable" (*n* = 32, 33.3%), or "a little uncomfortable" (*n* = 42; 43.8%). Only 22.9% reported being moderately (*n* = 17), a lot (*n* = 4), or always (*n* = 1) uncomfortable with physical contact.

Participant sleep and experimental attitudes were also assessed because of previous research findings that these variables may influence the experience of pain. Overall, participants reported normal sleep with mean sleep duration of 448 minutes (7 hrs, 28 min; *SD* = 88 min.), mean number of awake bouts of .94 (*SD* = 1.13), and mean sleep quality of 1.04 ("somewhat refreshed"; *SD* = .63). On the Frid experimental expectancies scale, participants reported moderate psychological involvement (*M* = 3.20,

SD = 0.51), moderate experimental efficacy and control beliefs (*M* = 3.86, *SD* = 0.45), and moderate negative experimental expectancies (*M* = 2.10, *SD* = 0.93). In sum, overall background characteristics are consistent with norms reported for healthy educated women and showed no group differences.

Covariates

Previous experience with massage and/or meditation was hypothesized to influence participant response to intervention. Thus, these variables were evaluated as potential covariates of affective response, relaxation response, and sympathetic activity response to intervention. Zero-order correlations revealed that past experience with meditation, guided imagery, deep breathing, yoga, tai chi, or a martial art was associated with decreased high activation affect (e.g., alert, aroused, $r = -.21$, $p < .05$) and decreased mean heart rate ($r = -.21$, $p < .05$) during the intervention period. Past experience with massage was not related to any affective response, relaxation response, or sympathetic activity response during the intervention period (all *p*'s > .05). Past experience with any type of meditation was further evaluated for covariance with high activation affect and MHR in regression analyses.

Experimental expectations may be related to perceptions of pain. The Frid subscale variables were evaluated as potential covariates of pain outcomes in a zero-order correlation matrix. The baseline stimulation task was used for evaluation. The Frid Efficacy and Control subscale was associated with baseline threshold ($r = .20$, $p < .05$), baseline tolerance ($r = .20$, $p < .05$), and McGill residual pain intensity ($r = -.26$, $p < .05$), with increased efficacy and beliefs of control being associated with increased pain threshold, increased pain tolerance, and decreased residual pain intensity,. The Frid

Negative Expectancies item was associated with baseline threshold (r = -.30, p < .01) and baseline tolerance (r = -.33, p < .01), with increased negative expectancies being associated with decreased pain threshold and tolerance. Therefore expectancies were treated as a potential covariate in analyses of pain threshold, pain tolerance and residual pain intensity outcomes.

Sleep variables were examined as possible covariates. Sleep duration (r = .23, p < .05) and number of awake bouts (r = .27, p < .01) were associated with McGill residual pain intensity: increased sleep duration and increased number of awake bouts were associated with greater residual pain intensity. Sleep duration, and number of awake bouts were further evaluated as potential covariates of residual pain intensity outcomes in regression analyses.

Although a greater number of women who reported wanting more physical contact were randomized to the massage only group, satisfaction with physical contact was not significantly correlated with the primary variables of pain intensity VAS (r = .04, p > .05), relaxation (r = -.10, p > .05), affect ($r_{unpleasant}$ = .10, p > .05; $r_{pleasant}$ = .05, p > .05), or sympathetic and parasympathetic activity (r_{MHR} = -.14, p > .05; r_{MIBI} = .15, p > .05; r_{RSA} = .01, p > .05; $r_{HFpower}$ = -.06, p > .05). Satisfaction with physical contact was not treated as a covariate in subsequent group analyses.

Lastly, although there were no group differences in age, BMI, illness severity score, or medication use, these variables were evaluated as potential covariates of sympathetic activity based on research findings that increased age, BMI, illness, antidepressant use, and stimulant medication use influence cardiac functioning (Adler, Weisler, Goodman, Hamdani, & Niebler, 2009; Bild et al., 1993; Chambers, Guo,

Siervogel, Hall, & Chumlea, 2002; Daviglus, 2003; Licht et al., 2009; McTigue, Hess, & Ziouras, 2006). Increased age was associated with decreased RSA (r = -.29, p < .01) during the baseline rest period. In addition, stimulant medication use was associated with increased MHR (r = .46, p < .05) and decreased MIBI (r = -.38, p < .05) during the baseline rest period. No other demographic variables were associated with baseline sympathetic activity. Therefore, age and stimulant medication use were further evaluated for covariance with RSA and MHR and MIBI outcomes, respectively, in regression analyses.

Primary analyzes

All MLM analyses follow the pattern depicted in Figure 4. There were two different analysis protocols that each included three sections of data. Analysis of pain outcomes utilized: (1a) baseline stimulation (2 electrical stimulation pain trials); (2) intervention (one of four experimental conditions + 3 electrical stimulation pain trials); and (3a) post-recovery stimulation (1 electrical stimulation pain trial). Analysis of affect and physiological outcomes utilized: (1b) baseline rest (no electrical stimulation pain trials); (2) intervention (one of four experimental conditions + 3 electrical stimulation pain trials); and (3b) recovery rest (no electrical stimulation pain trials).

Manipulation checks. To confirm that the pain-manipulation (electrical stimulation) produced significant pain compared to baseline rest, and did not have long lasting effects that carried over to recovery rest, a manipulation check of pain intensity VAS was performed from baseline rest to intervention to recovery rest. Pain intensity outcome means are provided in Table 3. As expected, there was a Level 1, main effect of time. Pain intensity significantly increased from baseline rest to intervention (β = 55.92, z

= 14.75, $p < .001$) and significantly decreased from intervention to recovery rest (β = -11.49, $z = -3.03$, $p < .01$). A simple comparison showed that recovery rest pain intensity was not significantly different from baseline rest pain intensity, $t(1,95) = -.49$, $p > .05$. Thus, pain intensity was significantly higher during the intervention stimulation trials than during the baseline rest period, and returned to baseline rest levels during the recovery rest period. This indicates that the pain manipulation was successful and short-acting.

To assess whether participants were engaged in the experiment during the intervention period, a manipulation check of attention and focus was performed using the CAMS-R. There were no group differences on CAMS-R scores immediately following the intervention period, $F(3,92) = .20$, $p > .05$. This indicates that there was no significant difference across the four experimental conditions for reported attention to, focus on, awareness of, and acceptance of the present (i.e., mindfulness of the present). Thus, participants across the four groups reported similar levels of engagement during the intervention period. See Table 5.

Hypothesis 1: Pain. MLM regression analyses were utilized to examine whether time, experimental condition, and/or their interaction had a significant effect on pain outcomes. It was expected that the massage groups (i.e., massage alone and massage plus guided imagery) would report higher pain threshold and pain tolerance, and lower pain intensity, pain unpleasantness, residual pain intensity, worst pain intensity, and least pain intensity than the control group.

Pain threshold. The Frid Negative Expectancies item was a significant predictor of pain threshold across time and groups (β = -4.31, $z = -3.15$, $p < .01$), and was thus

included as a covariate in pain threshold analyses. Frid Efficacy and Control, however, was not a significant predictor and was thus removed from the model ($p > .05$). MLM analyses revealed no significant Level 1 main effects for time from baseline stimulation to intervention ($z = 1.50$, $p > .05$) or from intervention to post-recovery stimulation ($z = -1.19$, $p > .05$). There was also no Level 2 main effect for group ($z = .51$, $p > .05$), nor were there significant group x time interactions for trajectory of pain threshold (all p's >.05), indicating that interventions had no effect on pain threshold. Means and standard deviations can be found in Table 2.

Pain tolerance. Similar to pain threshold, the Frid Negative Expectancies item significantly predicted pain tolerance ($\beta = -4.53$, $z = -3.39$, $p < .01$), and was therefore included as a covariate in pain tolerance analyses. Frid Efficacy and Control, however, was not a significant predictor and was thus removed from the model ($p > .05$). MLM analyses revealed no significant Level 1 main effect for time from baseline stimulation to intervention ($z = -.34$, $p > .05$). However, a Level 1 trend was observed for tolerance, decreasing from intervention to post-recovery stimulation ($z = -1.81$, $p = .07$). There was no Level 2 main effect for group ($z = .19$, $p > .05$), nor any significant group x time interactions for trajectory of pain tolerance (all p's > .05), indicating that interventions had no effect on pain tolerance. Means and standard deviations can be found in Table 2.

Pain intensity. There was a Level 1 main effect for time. Pain intensity significantly increased from baseline stimulation to intervention ($\beta = 9.01$, $z = 2.86$, $p < .01$) and significantly decreased from intervention to post-recovery stimulation ($\beta = -11.49$, $z = -3.65$, $p < .001$). There was no Level 2 main effect of group ($z = .67$, $p > .05$), meaning that there was no difference between groups on pain intensity during

intervention stimulation. Moreover, there were no group x time differences in slopes. Groups did not differ in pain intensity from baseline stimulation to intervention (z = -.69, p > .05) or from intervention to post-recovery stimulation (z = 1.34, p > .05). Means and standard deviations can be found in Table 3.

Pain unpleasantness. There was a Level 1 main-effect for time. Pain unpleasantness marginally increased from baseline stimulation to intervention (β = 5.77, z = 1.74, p = .08) and significantly decreased from intervention to post-recovery stimulation (β = -9.01, z = -2.72, p < .01). There was no Level 2 main group effect for pain unpleasantness during intervention stimulation (z = -.79, p > .05). Consistent with predictions, there were significant group x time interactions. There was a marginal group difference from baseline stimulation to intervention (β = -3.12, z = -1.77, p = .08) and a significant group difference from intervention to post-recovery stimulation (β = 4.51, z = 2.56, p < .05). See Figure 5.

Post-hoc simple comparisons showed that, for the control group, pain unpleasantness increased significantly from baseline stimulation to intervention, $t(1,21)$ = -3.20, p < .01, and decreased significantly from intervention to post-recovery stimulation, $t(1,21)$ = 2.97, p < .01, returning to baseline stimulation levels at post-recovery stimulation, $t(1,21)$ = .91, p > .05. In contrast, pain unpleasantness did not change for the guided imagery alone group from baseline stimulation to intervention, $t(1,25)$ = -.41, p > .05, η^2 = .05, or from intervention to post-recovery stimulation, $t(1,25)$ = .93, p > .05, η^2 = .07. There was also no significant change in pain unpleasantness for the massage alone group, $t(1,23)$ = .83, p > .05, η^2 = .14, and, $t(1,23)$ = -.76, p > .05, η^2 = .17, or for the massage plus guided imagery group, $t(1,23)$ = .26, p > .05, η^2 = .04, and, $t(1,23)$ = -.56, p

> .05, η^2 = .10, from baseline stimulation to intervention and from intervention to post-recovery stimulation, respectively. See Table 3. Thus, all interventions reduced the unpleasantness of pain in comparison to the control group, with the largest effects observed for the massage groups.

Residual pain intensity. Frid Efficacy and Control was a significant predictor of residual pain intensity in MLM analyses and was thus retained in the model (β = -.37, *z* = -3.58, *p* < .001). Sleep duration and number of awake bouts were not significant predictors and were removed from the model (all *p*'s > .05). There was no Level 1 main effect for time: residual pain intensity did not change from baseline stimulation to intervention (*z* = .43, *p* > .05) or from intervention to post-recovery stimulation (*z* = -1.00, *p* > .05). There was also no Level 2 main effect for group on residual pain intensity after intervention stimulation (*z* = -1.22, *p* > .05). There was, however, a significant group x time interaction effect. Groups differed in their slopes from baseline stimulation to intervention (β = -.13, *z* = -2.44, *p* < .05). See Figure 5.

Post-hoc analyses revealed that residual pain intensity did not change from baseline stimulation to intervention for the control group, *t*(1,21) = -.62, *p* > .05, or the guided imagery alone group, *t*(1,25) = 1.14, *p* > .05. However, there was a significant decrease in residual pain intensity from baseline stimulation to intervention for the massage alone group, *t*(1,23) = 2.29, *p* < .05, η^2 = .08, and a marginal decrease for the massage plus guided imagery group, *t*(1,23) = 1.90, *p* = .07, η^2 = .07. Furthermore, residual pain intensity remained marginally lower than baseline stimulation after post-recovery stimulation for the massage alone group, *t*(1,23) = 2.02, *p* = .056, η^2 = .04. See Table 3.

There were no Level 1 main effects of time, no Level 2 main effects of group, and no group x time interactions for worst or least pain intensity (all *p*'s > .05). See Table 3 for means and standard deviations.

Summary of pain outcomes: There were no group differences on immediate measures of pain sensation (pain threshold, pain tolerance, and pain intensity). However, group x time interactions were observed for pain unpleasantness and residual pain intensity. All three intervention groups reported decreased pain unpleasantness compared to the control group, while the two massage groups reported additional decreases in residual pain intensity. There was no evidence of additive effects for massage and guided imagery given that massage plus guided imagery did not produce superior effects to massage alone.

Hypothesis 2: Affect. MLM regression analyses were utilized to examine whether time, experimental condition, and/or their interaction had a significant effect on affect outcomes. It was expected that affect would become more unpleasant and less pleasant during intervention stimulation, compared to the baseline rest and recovery rest periods. It was further expected that the guided imagery alone group, massage alone group, and massage plus guided imagery group would report less increase in unpleasant affect and less decrease in pleasant affect, compared to the control group. All affect outcome means and standard deviations are provided in Table 4.

Unactivated unpleasant affect. There was a significant Level 1 main effect for time. Unactivated unpleasant affect decreased from baseline rest to intervention ($\beta = -.29$, $z = -2.32$, $p < .05$) and marginally increased from intervention to recovery rest ($\beta = .24$, $z = 1.88$, $p = .06$). There was also a significant Level 2 main effect for group ($\beta = -.17$, $z =$

-2.18, $p < .05$) and significant group differences in change from baseline rest to intervention ($\beta = -.19$, $z = -2.74$, $p < .01$) and in change from intervention to recovery rest ($\beta = .13$, $z = 1.92$, $p = .05$). See Figure 6.

Post-hoc analyses revealed no change in unactivated unpleasant affect from baseline rest to recovery rest for the control group, $t(1,21) = -.18$, $p > .05$. In contrast, the guided imagery alone group, $t(1,25) = 4.47$, $p < .05$, massage alone group, $t(1,23) = 5.13$, $p < .01$, and massage plus guided imagery group, $t(1,23) = 3.98$, $p < .01$, all reported significantly decreased unactivated unpleasant affect levels from baseline rest to intervention and returns to baseline rest levels during recovery rest (*t's* > 1.97, *p's* > .05).

Low activation affect. There was a Level 1 main effect for time: Low activation affect (i.e., quiet, serene) significantly decreased across groups from baseline rest to intervention ($\beta = -.58$, $z = -4.35$, $p < .01$) and increased from intervention to recovery rest ($\beta = .32$, $z = 2.42$, $p < .05$). There was no Level 2 main effect for group during intervention ($z = -.03$, $p > .05$). However, a group x time interaction qualified these results: There was a significant group difference in low activation affect change from baseline rest to intervention ($\beta = .18$, $z = 2.49$, $p < .05$) and change from intervention to recovery rest ($\beta = -.19$, $z = -2.61$, $p < .01$). See Figure 7.

Post-hoc analyses revealed that, for the control group, low activation affect decreased significantly from baseline rest to intervention, $t(1,21) = 4.72$, $p < .01$, and increased significantly from intervention to recovery rest, $t(1,21) = -2.40$, $p < .05$, to approach baseline rest levels at recovery rest, $t(1,21) = 2.02$, $p = .056$. Low activation affect did not change for the guided imagery alone group, $t(1,25) = 1.28$ $p > .05$, massage

alone group, $t(1,23) = 1.63$, $p > .05$, or massage plus guided imagery group, $t(1,23) = 1.96$, $p > .05$, from baseline rest to recovery rest.

High activation affect. Past experience with meditation was not a significant predictor of high activation affect (i.e., alert, aroused) and was thus removed from the model ($p > .05$). There was a Level 1 main effect for time. High activation affect significantly increased from baseline rest to intervention ($\beta = .29$, $z = 3.32$, $p < .01$) and decreased from intervention to recovery rest ($\beta = -.48$, $z = -5.57$, $p < .01$). There was no Level 2 main effect for group during intervention ($z = -.74$, $p > .05$). However, these effects were qualified by group x time interactions. There was a marginal group difference in high activation affect change from baseline rest to intervention ($\beta = -.08$, $z = -1.74$, $p = .08$) and a significant group difference in high activation affect change from intervention to recovery rest ($\beta = .11$, $z = 2.37$, $p < .05$). See Figure 7.

Post-hoc analyses revealed that, for the control group, high activation affect increased significantly from baseline rest to intervention, $t(1,21) = -2.99$, $p < .05$, and decreased significantly from intervention to recovery rest, $t(1,21) = 3.81$, $p < .05$, to return to baseline rest levels at recovery rest, $t(1,21) = 1.89$, $p > .05$. High activation affect did not change for the guided imagery alone group from baseline rest to intervention, $t(1,25) = -.63$, $p > .05$, but decreased significantly from intervention to recovery rest, $t(1,25) = 4.21$, $p < .05$, to end lower than baseline rest levels, $t(1,25) = 3.84$, $p < .05$. There was no change in high activation affect for the massage alone group, $t(1,23) = 1.47$, $p > .05$, or the massage plus guided imagery group from baseline rest to recovery rest, $t(1,23) = -.19$, $p > .05$.

Unpleasant affect. There was no Level 1 main effect for time on unpleasant affect (e.g., fatigued, hostile, depressed, anxious) from baseline rest to intervention (z = -.70, p > .05) or from intervention to recovery rest (z = -.72, p > .05). However, there was a significant Level 2 main effect for group on unpleasant affect (β = -.09, z = -3.36, p < .05) and significant group x time interactions: Groups differed in their change from baseline rest to intervention (β = -.11, z = -4.44, p < .01) and from intervention to recovery rest (β = .07, z = 2.90, p = .05). See Figure 8.

Post-hoc analyses revealed no change for the control group in unpleasant affect from baseline rest to intervention, $t(1,21)$ = -.41, p > .05, or from intervention to recovery rest $t(1,21)$ = .88, p > .05. In contrast, the guided imagery alone group reported significantly decreased unpleasant affect from baseline rest to intervention, $t(1,25)$ = 3.28, p < .01, η^2 = .13, with unpleasant affect remaining significantly lower than baseline rest at recovery rest, $t(1,25)$ = 2.91, p < .01. The massage alone group also reported significantly decreased unpleasant affect from baseline rest to intervention $t(1,23)$ = 6.00, p < .01, η^2 = .30. For the massage alone group, unpleasant affect significantly increased from intervention to recovery rest, $t(1,23)$ = -3.11, p < .01, η^2 = .15, but remained significantly lower than baseline rest at recovery rest $t(1,23)$ = 2.68, p < .05. The massage plus guided imagery group had a similar pattern, with unpleasant affect significantly decreasing from baseline rest to intervention, $t(1,23)$ = 5.68, p < .01, η^2 = .28, and increasing from intervention to recovery rest, $t(1,23)$ = -3.78, p < .01, η^2 = .18. However, unpleasant affect did not remain significantly lower than baseline rest for the massage plus guided imagery group, $t(1,23)$ = 1.90, p = .07.

Pleasant affect. There was a Level 1 main effect for time. Pleasant affect (e.g., happy, calm, energetic) decreased from baseline rest to intervention ($\beta = -.76$, $z = -7.19$, $p < .01$) and increased from intervention to recovery rest ($\beta = .30$, $z = 2.83$, $p < .01$). There was also a Level 2 main effect for group during intervention ($\beta = .26$, $z = 3.75$, $p < .01$), and significant group x time interactions. Specifically, there was a significant group difference in change from baseline rest to intervention ($\beta = .34$, $z = 5.98$, $p < .01$), as well as a group difference in change from intervention to recovery rest ($\beta = -.19$, $z = -3.31$, $p < .01$). See Figure 8.

Post-hoc analyses revealed a significant decrease in pleasant affect for the control group from baseline rest to intervention, $t(1,21) = 6.05$, $p < .01$, and a significant increase from intervention to recovery rest, $t(1,21) = -3.01$, $p < .01$. However, pleasant affect remained significantly lower than baseline rest for the control group at recovery rest, $t(1,21) = 4.15$, $p < .01$. The guided imagery alone group also reported a significant decrease in pleasant affect from baseline rest to intervention, $t(1,25) = 3.12$, $p < .01$, $\eta^2 = .19$, and an increase from intervention to recovery rest, $t(1,25) = -.74$, $p > .05$, $\eta^2 = .06$. However, pleasant affect also remained significantly lower than baseline rest for the guided imagery alone group at recovery rest, $t(1,25) = 2.20$, $p < .05$. In contrast, the massage alone group maintained baseline rest levels of pleasant affect from baseline rest to intervention, $t(1,23) = -.43$, $p > .05$, $\eta^2 = .32$, and from intervention to recovery rest, $t(1,23) = 1.29$, $p > .05$, $\eta^2 = .19$, with no significant difference from baseline rest to recovery rest, $t(1,23) = .33$, $p > .05$. The massage plus guided imagery group also maintained baseline rest levels of pleasant affect from baseline rest to intervention, $t(1,23) = -1.11$, $p > .05$, $\eta^2 = .40$. The massage plus guided imagery group reported a

significant decrease in pleasant affect from intervention to recovery rest, $t(1,23) = 2.97$, $p < .01$, $\eta^2 = .29$; however, there was no difference in pleasant affect from baseline rest to recovery rest, $t(1,23) = 1.19$, $p > .05$.

Affect mediation of pain. Affect during intervention was further evaluated as a potential mediator of the group x time interactions for pain unpleasantness and residual pain intensity. Group assignment was a significant predictor of change in pain unpleasantness from intervention to recovery rest ($\beta = -4.52$, $z = -2.68$, $p < .01$). Low activation, high activation, unpleasant, and pleasant affect were not significantly related to the change in pain unpleasantness from intervention to recovery rest (all p's $> .05$), and thus did not meet criteria for mediation. Unactivated unpleasant affect was significantly related to the change in pain unpleasantness from intervention to recovery rest ($\beta = 5.61$, $z = -2.68$, $p < .01$). However, the Aroian version of the Sobel test revealed that unactivated unpleasant affect was not a significant mediator of the relationship between group assignment and change in pain unpleasantness ($t = -1.64$, $p > .05$).

Group assignment was a significant predictor of change in residual pain intensity from baseline rest to intervention ($\beta = .13$, $z = 2.22$, $p < .05$). Thus, change in residual pain intensity from baseline rest to intervention was examined further for mediation effects. Unactivated unpleasant affect, low activation affect, high activation affect, unpleasant affect, and pleasant affect were not significantly related to change in residual pain intensity from baseline rest to intervention (all p's $> .05$), and were not further evaluated as potential mediators. These results suggest that the effects of guided imagery and massage intervention on pain were independent of reported affect during intervention.

Summary of affect outcomes. The results of affect analyses suggest that guided imagery and massage interventions have a robust effect on emotional responses during experimental pain, with massage intervention showing the largest effect sizes. Contrary to expectations, there was little evidence of additive effects for massage plus guided imagery, given that massage plus guided imagery did not produce consistently superior effects compared to massage alone. Furthermore, affect-related changes during experimental pain do not appear to be related to group differences in pain perception.

Hypothesis 3: (a) Psychological relaxation. ANOVA was used to test the between-group differences in relaxation during intervention. Consistent with the hypothesis that massage works via descending relaxation, it was expected that the guided imagery alone, massage alone, and massage plus guided imagery would report greater self-report relaxation than the control condition.

Relaxation VAS. Significant group differences were observed on the relaxation VAS for the intervention period, $F(3,92) = 6.24$, $p < .01$. There was no difference in relaxation level reported by the guided imagery alone group compared to the control group, $t(1,46) = -1.59$, $p > .05$. However, both the massage alone group, $t(1,44) = -3.84$, $p < .01$, $\eta^2 = .25$, and massage plus guided imagery group, $t(1,44) = -3.09$, $p < .01$, $\eta^2 = .18$, reported significantly greater relaxation than the control group. There was no significant difference in relaxation levels reported between the massage alone group and the massage plus guided imagery group, $t(1,46) = .82$, $p > .05$. See Table 5.

Relaxation VAS mediation of pain. Self-report relaxation was evaluated as a potential mediator of the relationship between group assignment and pain unpleasantness and residual pain intensity. Relaxation was not significantly related to the changes in pain

unpleasantness (z = -1.19, p > .05) or residual pain intensity (z = -.78, p > .05). These results suggest that the effects of guided imagery and massage intervention on pain were independent of reported relaxation levels.

Hypothesis 3: (b) Physiological relaxation. Measures of sympathetic and parasympathetic activity were calculated from raw heart rate and respiration using Mindware physiology analysis software (MHR, MIBI, LF power, LF/HF ratio, RSA, and HF power). Blood pressure was also measured and considered a measurement of sympathetic activity. MLM regression analyses were utilized to examine whether time, experimental condition, and/or their interaction had a significant effect on physiological outcomes. It was expected that sympathetic activity would increase during intervention stimulation, compared to baseline rest and recovery rest periods. However, consistent with the hypothesis that massage effects work via relaxation, it was expected that the guided imagery alone group, massage alone group, and massage plus guided imagery group would experience less of an increase in sympathetic activity, as well as a greater increase in parasympathetic activity, compared to the control group. Physiological outcome means and standard deviations are provided in Table 6.

Sympathetic activity: Mean heart rate (MHR). Stimulant medication use (β = 19.06, z = 4.56, p < .01) and past experience with meditation (β = -4.52, z = -2.08, p < .05) were significant predictors of MHR and were thus retained as covariates in regression analyses. Chi-squared analysis of the difference between deviance statistics for the fixed slopes model, $\chi^2(10) = 16389.73$, and random slopes model, $\chi^2(15) = 16096.31$, indicated that change over time should be allowed to vary randomly across participants, $\Delta\chi^2(5) = 293.42$, $p < .001$.

MLM analyses revealed no Level 1 main effect for time on MHR from baseline rest to intervention ($z = 1.37$, $p > .05$). However, there was a marginal decrease in MHR from intervention to recovery rest ($\beta = -.17$, $z = -1.67$, $p = .09$). There was no Level 2 main effect for group on MHR during intervention ($z = -.82$, $p > .05$) and no group x time interaction for baseline rest to intervention ($z = -1.46$, $p > .05$). There was, however, a marginal group x time interaction in MHR change from intervention to recovery rest ($\beta = .10$, $z = 1.76$, $p = .08$). MLM post-hoc analyses revealed that this was due to the massage plus guided imagery group's experiencing a slower return to baseline rest during recovery rest, compared to the control group ($\beta = .44$, $z = 2.06$, $p < .05$). These results indicate that there were no clear between group differences in MHR across the study period.

Mean inter-beat interval (MIBI). Stimulant medication use ($\beta = -167.08$, $z = -3.26$, $p < .01$) was a significant predictor of MIBI and was thus retained as a covariate in regression analyses. Chi-squared analysis of the difference between deviance statistics for the fixed slopes model, $\chi^2(9) = 29697.56$, and random slopes model, $\chi^2(14) = 29498.10$, indicated that change over time should be allowed to vary randomly across participants, $\Delta\chi^2(5) = 199.46$, $p < .001$.

MLM analyses revealed no Level 1 effect for time on MIBI from baseline rest to intervention ($z = -1.03$, $p > .05$). There was a marginal increase in MIBI from intervention to recovery rest ($\beta = 1.96$, $z = 1.83$, $p = .07$). There was no Level 2 main effect for group on MIBI during intervention ($z = .49$, $p > .05$), and no group x time interaction from baseline rest to intervention ($z = 1.07$, $p > .05$). There was, however, a marginal group x time interaction from intervention to recovery rest ($\beta = -1.05$, $z = -1.85$, $p = .06$). Post-hoc MLM analyses revealed that this marginal group x time interaction

was due to the massage plus guided imagery group experiencing a slower return to baseline rest levels of MIBI during recovery rest, compared to the control group (β = -4.27, z = -1.90, p = .057). These results indicate that there were no clear between group differences in MIBI across the study period.

MHR and MIBI follow-up analysis of random slope effects. Given the variation in MHR and MIBI fixed slopes versus random slopes models, follow-up analyses were conducted to determine if individual reports of pain, affect, and/or relaxation accounted for significant group x time interactions observed in the fixed slopes MHR and MIBI models. First, a difference score for mean MHR and MIBI was calculated to represent mean change from baseline rest to intervention and from intervention to recovery rest. Next, linear regression (LISREL 8.7) was done to determine if pain, affect or relaxation mediated relationships between group assignment and changes in heart rate.

Group assignment was a significant predictor of change from baseline rest to intervention for MHR, controlling for stimulant medication use and experience with meditation (β = 1.25, z = 2.90, p < .01), and for MIBI, controlling for stimulant medication use (β = -15.10, z = -3.07, p < .01), High activation affect, unpleasant affect, and self-report relaxation were significantly related to change in MHR and MIBI from baseline rest to intervention. Therefore, these three variables were examined as potential mediators of the relationship between group assignment and change in MHR and MIBI.

High activation affect was significantly related to change in MHR from baseline rest to intervention (β = -1.99, z = -2.13, p < .05). High activation affect was also significantly related to change in MIBI from baseline rest to intervention (β = 22.20, z = 2.08, p < .05). However, high activation affect did not significantly influence the

relationship between group assignment and change in MHR ($\Delta\beta = .06$, $p > .05$) or change in MIBI ($\Delta\beta = .69$, $p > .05$). Thus, high activation affect did not meet criteria for mediation of the relationship between group assignment and change in MHR or change in MIBI.

Unpleasant affect was also related to change in MHR from baseline rest to intervention ($\beta = -4.82$, $z = -2.73$, $p < .01$), as well as change in MIBI from baseline rest to intervention ($\beta = 31.25$, $z = 2.53$, $p < .05$). Contrary to high activation affect, unpleasant affect significantly influenced the relationship between group assignment and change in MHR ($\Delta\beta = .44$, $p < .05$) and change in MIBI ($\Delta\beta = 4.62$, $p < .05$). Using the Aroian version of the Sobel test, unpleasant affect was further determined to be a significant mediator of the relationship between group assignment and change in MHR ($t = 2.38$, $p < .05$) and change in MIBI ($t = -2.31$, $p < .05$). Thus, unpleasant affect mediated the relationship between group assignment and change in MHR and MIBI from baseline rest to intervention.

Self-reported relaxation was also significantly related to change in MHR from baseline rest to intervention ($\beta = .51$, $z = 2.01$, $p < .05$) and change in MIBI from baseline rest to intervention ($\beta = -6.34$, $z = -2.17$, $p < .05$). In addition, self-reported relaxation significantly influenced the relationship between group assignment and change in MHR ($\Delta\beta = .38$, $p < .05$) and change in MIBI ($\Delta\beta = 4.69$, $p < .05$). Using the Aroian version of the Sobel test, relaxation was further determined to be a significant mediator of the relationship between group assignment and change in MHR ($t = 1.93$, $p = .05$) and change in MIBI ($t = -2.01$, $p < .05$). Thus, relaxation and unpleasant affect appeared to at

least partially mediate the fixed slope effects of group on change in MHR and MIBI from baseline rest to intervention.

Low frequency spectra power (LF power). No convergence could be reached in MLM analyses regarding LF power indicating that the specified model parameters could not be fit to the measured data. This suggests that there was no pattern of change over time or observable group difference in LF power. Individual paired *t*-tests confirmed this, showing no change in LF power across time or as a function of group assignment (all *p*'s > .05).

LF/HF power ratio (LF/HF ratio) and blood pressure. Chi-squared analysis of the difference between deviance statistics for the fixed slopes model, $\chi^2(8) = 15655.61$, and random slopes model, $\chi^2(13) = 15272.47$, indicated that change over time should be allowed to vary randomly across participants for LF/HF ratio, $\Delta\chi^2(5) = 383.14$, $p < .001$. No main effects or interactions were significant for LF/HF power, systolic and diastolic blood pressure (all *p*'s > .05).

Parasympathetic activity; Respiratory sinus arrhythmia (RSA). Age ($\beta = -.05$, $z = -2.62$, $p < .01$) was a significant predictor of RSA and was thus retained as a covariate in regression analyses. Chi-squared analysis of the difference between deviance statistics for the fixed slopes model, $\chi^2(9) = 6642.08$, and random slopes model, $\chi^2(14) = 6539.41$, indicated that change over time should be allowed to vary randomly across participants, $\Delta\chi^2(5) = 102.67$, $p < .001$. No main effects or interactions were significant (all *p*'s > .05).

High frequency spectra power (HF power), No convergence could be reached in MLM analyses regarding HF power, indicating that the specified model parameters could not be fit to the measured data. This suggests that there was no pattern of change over

time and no observable group difference in HF power. Individual paired *t*-tests confirmed this, showing no change in HF power across time or as a function of group assignment (all *p*'s > .05).

Summary of relaxation outcomes. (a) The results of these analyses suggest that massage has a robust effect on self-report relaxation during experimental pain. However, there was no evidence of additive effects for massage plus guided imagery, given that massage plus guided imagery did not produce superior effects compared to massage alone. In addition, experienced relaxation levels do not appear to be related to group differences in pain perception. (b) No clear effects of group assignment were found on physiological outcomes. Follow-up analyses indicate that physiological response to massage may be related to an individual's experience of affect and relaxation.

Chapter 4

Discussion

Results of this study provided some support for the commonly cited hypothesis that massage directly influences the experience of pain via the interruption of ascending pain signals. Contrary to hypotheses, no effects of massage were observed on measures of immediate pain (i.e., pain threshold, pain tolerance, and pain intensity) or measures of worst and least pain intensity. Massage intervention effects were, however, observed on quality of the pain experience (i.e., pain unpleasantness) and residual pain intensity. Results also showed that massage has potent effects on affective and relaxation pathways. However, the affect and relaxation effects of massage do not appear to act in accordance with the GCM descending pain pathway, given that neither affect-related changes nor relaxation-related changes mediated group effects on pain perception. See Figure 9 for diagram. Furthermore, physiological effects of massage may be mediated via mechanisms of unpleasant affect and self-report relaxation.

Hypothesis 1: Pain. The GCM suggests that the most likely mechanism for the effects of massage on pain is the ascending touch and counter-pressure signal, which blocks pain signals traveling to the brain. If massage works via this pathway we would expect the massage groups to show less pain than the control group during the intervention. However, the data showed limited evidence for this hypothesis.

Contrary to study hypotheses there were no group differences in change in pain threshold, pain tolerance, pain intensity, worst pain intensity, or least pain intensity during intervention. Pain unpleasantness and residual pain intensity, on the other hand, did show group differences in change from baseline stimulation to intervention: Pain

unpleasantness increased from baseline stimulation to intervention for the control group, but remained at baseline level for all three interventions. In addition, residual pain intensity did not change from baseline stimulation to intervention for the control group or the guided imagery alone group. However, residual pain intensity was marginally lower for the massage plus guided imagery group and significantly lower for the massage alone group after intervention. Moreover, the massage alone group maintained marginally lower residual pain intensity at post-recovery stimulation, compared to baseline stimulation.

Thus, no effects on measures of immediate pain were observed for any of the three interventions. All three were moderately effective in reducing pain unpleasantness during intervention, with the two massage interventions exhibiting the largest effect sizes. In addition, the two massage conditions decreased residual pain intensity after intervention, compared to baseline stimulation. Interestingly, massage alone lead to the largest effects on pain unpleasantness and residual pain intensity. These results support the hypothesis that massage works via ascending touch and counter-pressure to lessen the perception of pain; however, these results do not support the primary GCM hypothesis that massage works via immediate interruption of pain signals.

Hypothesis 2: Affect. The GCM also suggests that pain can be reduced via descending pathways. Thus, if the effects of massage on pain worked via changes in affect, we would expect that the massage groups would experience equivalent or less increase in unpleasant affect and equivalent or less decrease in pleasant affect compared to the guided imagery alone group. Furthermore, if the effects of massage on pain worked via changes in affect, we would expect affect levels to mediate group differences in pain

perception. It was also expected that all three intervention groups would show less increase in unpleasant affect and less decrease in pleasant affect compared to the control group.

There was strong evidence for the effects of massage on affective pathways. Specifically, the control group maintained their baseline rests levels of boredom during the intervention period, while decreasing in feelings of quiet and serene and increasing in feelings of alertness and arousal. In addition, although the control group was able to maintain their baseline rest levels of unpleasant affect; they reported significantly decreased feelings of pleasant affect during both intervention and recovery rest.

In contrast to the control group, all three intervention groups reported decreased feelings of boredom during the intervention period, while maintaining their baseline rest feelings of quiet, serene, alertness and arousal. All three intervention groups also reported decreases in unpleasant affect; however, the two massage groups reported noticeably greater effects on unpleasant affect than the guided imagery alone group. In addition, the two massage groups were able to maintain their baseline rest levels of pleasant affect, while the guided imagery alone group reported decreased pleasant affect during both the intervention and recovery rest periods. Contrary to expectations and GCM descending pain pathway predictions, observed changes in affect did not mediate group differences in pain perception. Thus, while massage exhibited robust effects on affect, these effects do not appear to act in accordance with the descending GCM pain pathway prediction.

Hypothesis 3: (a) Psychological relaxation. The GCM also suggests that pain can be reduced via descending relaxation. Thus, if the effects of massage on pain worked via induced self-report relaxation, we would expect that the massage groups would

experience equivalent or greater self-report relaxation than the guided imagery alone group. Furthermore, if the effects of massage on pain worked via induced relaxation, we would expect self-report relaxation levels to mediate group differences in pain perception. It was further expected that all three intervention groups would experience greater cognitive and physiological relaxation than the control group.

Good support was found for the effects of massage on self-report relaxation. While the guided imagery alone group reported no difference in self-report relaxation levels during intervention compared to the control group, both the massage alone group and the massage plus guided imagery group reported significantly greater self-report relaxation than the control group. There was no statistical difference between massage groups in relaxation levels. Mediation analyses showed that self-report relaxation level was not related to group differences in pain unpleasantness or residual pain intensity. Thus, while massage induced greater self-report relaxation than the control or guided imagery conditions, the self-report relaxation effect of massage does not appear to act in accordance with the GCM descending pain pathway predictions.

Hypothesis 3: (b) Physiological relaxation. If the effects of massage on pain worked via induced physiological relaxation, we would expect that the massage groups would experience equivalent or less increase in sympathetic activity and equivalent or greater increase in parasympathetic activity compared to the guided imagery alone group. Furthermore, if the effects of massage on pain worked via induced physiological relaxation, we would expect physiological relaxation levels to mediate group differences in pain perception.

Contrary to expectations, no evidence was found for the unique effects of massage on sympathetic or parasympathetic activity. Changes in MHR and MIBI sympathetic activity were better explained by individual differences in experienced unpleasant affect and self-report relaxation than group assignment. There was also no evidence of group differences in RSA or HF power parasympathetic relaxation levels. Thus, physiological mediation of pain perception was not possible.

Mechanisms of massage

Ascending pathway. The commonly cited theory that massage works via the ascending pathway of the gate control model by physically interrupting the pain signal through touch and counter-pressure (e.g., Field, 1998; Field, 2001) received some support. There were no group differences on measures of immediate pain (i.e., pain threshold, pain tolerance, and pain intensity VAS), or measures of worst and least pain intensity, during intervention or post-recovery stimulation. However, there were significant effects on pain unpleasantness for all three intervention groups, as well as effects on residual pain intensity for the two massage groups. A potential explanation for these results is that massage may influence affective qualities of pain as well as residual/secondary pain signals via mechanisms of touch and counter-pressure, versus physically interrupting or decreasing the ascending nerve signal of pain.

For example, research has found that experimental pain which simulates chronic pain C fiber activation is more related to affective indexes of pain such as pain unpleasantness, as well as more evaluative indexes of pain such as that used to measure residual pain intensity (i.e., 5-point Likert scale, 1 = mild and 5 = excruciating). This is in contrast to indexes such as pain intensity VAS, which is more related to immediate

sensory activation (Chen & Treede, 1985; Price et al., 1986; Price, Harkins, & Baker, 1987). Findings that all three interventions decreased pain unpleasantness is consistent with research showing that affective dimensions of pain are associated with the anterior cingulate cortex (ACC) brain region, which is more malleable to top-down processes (Rainville, Duncan, Price, Carrier, & Bushnell, 1997; Tolle, et al., 1999) such as a guided imagery intervention. However, it is important to note that the massage interventions produced greater effect sizes than the guided imagery alone intervention and that observed changes in pain unpleasantness were independent of changes in affect. Thus, massage intervention appears to have a unique ascending influence on affective pain unpleasantness that is independent of top-down affective influences.

The unique effects of massage intervention on residual pain intensity, as well as the independence of changes in residual pain intensity from affect and relaxation, further supports the theory that massage has ascending somatosensory effects on pain. Evidence suggests that the somatosensory cortices SI and SII contain neurons that code spatial, temporal, and intensive aspects of innocuous and noxious sensory stimuli (Chudler, Anton, Dubner, & Kenshalo, 1990; Kenshalo & Isensee, 1983; Kenshalo, Chudler, Anton, & Dubner, 1988). While first pain signals (A-delta and A-beta fibers) are primarily related to activation of SI and SII brain regions, secondary pain signals (C fibers) are most closely related to activation of SII and ACC brain regions (Ploner, Gross, Timmermann, & Schnitzler, 2002). Thus, it may be that massage alters the experience of chronic C fiber pain through ascending activation of the SII and ACC brain regions, versus physically interrupting ascending pain signals, by which more immediate

measures of pain such as pain threshold, pain tolerance and pain intensity VAS would be expected to show effects.

If it was the case that ascending effects of massage differentially activated SII and ACC brain regions versus SI brain regions, it might help to explain observed variations in the effects of massage on the experience of pain in other experimental and endogenous pain studies (e.g., Ekici, Baker, Akbayrak, & Yuksei, 2009; Jane, Wilkie, Gallucci, Beaton, & Huang, 2009; Post-White et al., 2009). That is, different experimental pain models activate A-delta, A-beta, and C fibers differentially, as discussed in Chapter 1. Variations in pain models, as well as differences in pain measurement across studies (e.g., immediate pain intensity VAS versus affective pain unpleasantness versus evaluative pain intensity) may explain why some studies find positive effects of massage on pain; where as other studies show no effects of massage on pain.

Descending pathways: Affect. An alternative mechanism of massage to the GCM ascending pain pathway is the GCM descending pain affect. Large effect sizes of massage intervention on affect were observed in the current study. Both massage groups showed superior effects to the control group and guided imagery alone group on measures of unpleasant affect and pleasant affect. These findings are consistent with other experimental (Hatayama, Kitamura, Tamura, Nagano, & Ohnuki, 2008; Wentworth et al., 2009) and endogenous pain (Jane et al., 2009; Post-White et al., 2009; Walton, 2009) studies showing large effect sizes of massage intervention on mood. However, contrary to expectations, the effects of massage on affect did not appear to act in accordance with the descending GCM prediction that changes in affect would mediate changes in pain. Thus, massage appears to have a unique influence on affect that is

independent of pain outcomes. This finding may further support the notion that massage activates the affective ACC brain region through ascending pathways.

Relaxation. Another potential theory to explain the effects of massage on the experience of pain is the descending relaxation pathway of the GCM. Self-report relaxation techniques such as guided imagery have been found to induce relaxation via top-down processing (e.g., Daake & Gueldner, 1989; Geden, Beck, Hauge, & Pohlman, 1984; Spinhoven & Linssen, 1991), thus it was purposed that massage may work through a similar mechanism to influence the experience of pain (Field, 2000; Field, 2001). It was also purposed that massage may induce equivalent or greater physiological relaxation than guided imagery alone via descending relaxation effects.

Study results suggest that massage may induce greater self-report relaxation than guided imagery alone or a control condition. Furthermore, self-report relaxation, along with affect, may mediate the effects of massage on physiological cardiovascular outcomes. However, relaxation levels showed no mediational effects on perceptions of pain. Thus, the relaxation effect of massage appears to be independent of the GCM descending pain pathway.

Current study findings that physiological cardiovascular outcomes may be mediated by experiences of affect and relaxation may help explain the inconsistent physiological research findings observed across the massage literature. Recent research studies continue to report a wide variety of positive physiological effects of massage in both experimental and clinical settings. For example, studies have found that participants receiving as few as 15 minutes of massage had increases in RSA (Toro-Velasco et al., 2009), HF power, and LF/HF ratio (Diego & Field, 2009), as well as decreases in heart

rate and blood pressure (Billhult, Lindholm, Gunnarsson, & Stener-Victorin, 2009; Post-White et al., 2009; Walton, 2009). However, massage related changes in physiological parameters are not consistent across studies, and some research studies have reported no positive physiological effects of massage (e.g., Albert, Gillinov, Lytle, Feng, Cwynar, & Blackstone, 2009). Current study results suggest that physiological effects of massage may be mediated via unpleasant affect and self-report relaxation. Therefore, individual differences in the response to massage (e.g., affect and relaxation) may influence differences in physiological outcomes.

Pathway group comparisons. A multiple group study design allowed for further mechanistic pathway comparisons. The guided imagery alone intervention showed positive effects on pain unpleasantness and affect compared to the control condition. Therefore, the guided imagery alone group provided a comparison group for top-down influences on pain-related variables. Overall, the massage alone intervention and massage plus guided imagery intervention showed greater effects on pain experience, relaxation, and affect than the guided imagery alone group. Contrary to expectations, there was no consistent difference between the massage alone and massage plus guided imagery group on any of these outcomes. In fact, the massage alone group showed a general trend for larger effect sizes than the massage plus guided imagery group and a trend for increased maintenance of effects during the recovery periods; however, these differences were not statistically significant.

Given these group comparisons, the hypothesis that the massage plus guided imagery intervention would produce additive effects of both ascending and descending processes was not supported. Results suggest that the two massage interventions utilized

both ascending and descending processes to positively impact pain experience, affects, and relaxation. As a potential explanation for the slight differences observed between massage groups, given the robust affective and self-report relaxation effects of massage, it may be that the guided imagery intervention served as a cognitive distracter from the massage intervention, thereby slightly reducing the effects of massage plus guided imagery on outcome variables.

Other mechanisms. It remains unclear as to what specific aspects of soft tissue manipulation of the back, neck, shoulders, and upper arms positively influence pain experience, affect, and relaxation level. However, study methodology ruled out several external components of the typical massage experience (Smith, Sullivan, & Baxter, 2009), and strongly suggests that effects of massage on pain experience, relaxation, and affect are not due merely to expectations, setting, or social support. For instance, the current study eliminated patient seeking behavior and positive expectations of massage therapy by having undergraduate participants unaware of study purpose and protocol prior to informed consent. The only procedural aspect undergraduate participants were aware of prior to informed consent was study time requirement. Therefore, undergraduate participants were likely not seeking therapeutic massage from this experiment and likely did not arrive to their study appointment with high expectations of receiving massage for pain relief, as might happen with a patient seeking massage for endogenous pain relief.

In addition, this study eliminated relaxing setting characteristics that are typically confounded with massage. Specifically the study was conducted in a controlled laboratory setting with bright florescent lighting and a single high watt lamp, no wall decoration, no music during intervention, and no aromatherapy. Participants were fitted

with physiological recording equipment and seated upright throughout the study. Furthermore, all researchers, including the massage therapist, wore a white laboratory coat throughout the study. This setting is atypical of massage therapy settings were dim lighting, soft music, supine/prone body positioning, aromatherapy and non-intimidating clothing are utilized (Smith, Sullivan, & Baxter, 2009).

Along with setting, social support has been suggested as a primary mechanism by which massage influences pain, particularly given the consistent positive effects of massage on mood (Moyer et al., 2004). The current study, although not testing this hypothesis directly, controlled for social support across groups and kept participant-researcher/therapist interaction to a minimum. Specifically, researchers engaged in little to no small talk with participants prior to study completion. Researchers were also not informed of group assignment until immediately prior to the intervention period so as to reduce researcher bias. Moreover, the massage therapist did not interact with participants prior to or after the intervention period unless physiological recording equipment required adjustment that could not be completed by the other researcher. The same massage therapist was present in the room with each participant, regardless of group assignment, and engaged in similar scripted verbal interactions with each participant during the intervention period. Thus, researcher bias toward group assignment, prior-relationship with the massage therapist, and differential social support were all minimized in the current study.

Despite minimizing a priori participant expectations, utilizing a clinical laboratory setting, and providing minimal social support during the study, sizable effects of massage were observed on pain experience, affect, and relaxation. It is, therefore reasonable to

conclude that while setting and social support may enhance the effects of massage, they do not wholly account for observed effects on pain experience, affect, and relaxation. It is important for researchers to be cognizant of setting and social support variables in future studies and to either control or manipulate these variables as theory and study purpose dictate.

Methodological considerations

Although there are several methodological strengths of this study there are also several limitations that warrant further discussion. In particular, the brevity of the intervention period raises concerns regarding effectiveness. Research has shown that effects of massage can be measured around 15 minutes (e.g., Diego & Field, 2009), thus the intervention period for this study was established to be 15.5 minutes. However, dosing effects of massage have also been documented, with greater effects observed at 30 minutes and one hour (Moyer et al., 2004; Beider & Moyer, 2007). Furthermore, repeated experience with massage has also been found to positively impact pain, mood and function response. Similar dose effects have been reported for guided imagery intervention, with more frequent relaxation practices being associated with improved mood, health, quality of life, and immune response (Eremin et al., 2009; Watanabe, Fukuda, & Shirakawa, 2005). Thus, the brief intervention period of 15.5 minutes may not have been long enough to see the full effects of either massage or guided imagery on pain experience, affect, relaxation, or physiological outcomes.

Another relative limitation of the current study was that the massage intervention and guided imagery intervention did not provide direct intervention to the pain stimulus. As discussed previously, electrical stimulation methodology did not permit massage to be

directly applied to the area of pain stimulation. Therefore, in order to maintain equivalency of intervention, the guided imagery alone intervention did not instruct participants to focus on reducing their experienced pain levels.

This limitation was partially addressed for the massage groups, however, by focusing the massage intervention on the dermatome area of C5 and T1, which innervates the forearm nerves (Lee et al., 2008). Furthermore, the right upper arm was being massaged during the third intervention stimulation trial, which should have provided proximal touch and counter-pressure nerve stimulation to the forearm area. Although this lack of focus on the pain stimulus was uniform across groups, and partially addressed in the massage intervention, greater intervention effects on pain may have been observed if the massage and guided imagery interventions directly addressed the pain stimulus. Future experimental studies may find methods of applying massage closer to the area of pain, and/or have a guided imagery intervention focus on reducing pain levels in order to provide a comparison to these results.

Other limitations include the reliance on a relatively homogenous and young undergraduate population. Though this population allowed for minimization of participant expectations, minimization of cross-gender participant-experimenter biases, and provided a physically and mentally healthy study population, it is possible that pain, affect, and relaxation experiences common to this study population do not generalize to older or more ethnically diverse female adults. Furthermore, these findings may not generalize to male populations. Future studies using a more demographically diverse sample are needed.

Despite these methodological limitations, there are several strengths to this study. For instance, this study was a repeated measures randomized trial with multiple comparison groups that allowed for the examination of three potential mechanistic pathways: Ascending interruption of pain signals, descending affective influences, and both cognitive and physiological relaxation influences. In addition, this study utilized the well validated multifaceted gate control model (Melzack, 1986; Melzack & Casey, 1968; Melzack & Wall, 1965; Melzack & Wall, 1989) for study design and protocol development. This theoretical model allowed for specific outcome hypotheses to be developed and tested. Moreover, a highly controlled laboratory setting was utilized in order to control the pain stimulus, minimize researcher bias, minimize the influence of setting, and minimize the influence of social support. Despite these methodological controls, effects of guided imagery and massage intervention were observed on pain experience, affect, and relaxation, with the massage interventions having a greater effect on outcomes than the guided imagery alone intervention.

Future Research

This study is a first step toward addressing the mechanistic pathways of massage. There are many additional mechanistic questions that need to be addressed, such as whether relaxation effects utilize the descending only pathway or both the ascending and descending pathways. Direct manipulation of social support should also be examined in order to further evaluate social support as a mechanism of massage (Moyer et al., 2004). Further evaluation of the physiological effects of massage is needed during longer intervention periods, as well as further examination of unpleasant affect and relaxation as mediators of the effects of massage on physiological outcomes. Studies examining the

most proximal potential mechanisms of massage (i.e., endogenous hormone release with touch versus the mechanical pressure of massage) are also needed. Lastly, new theories are needed that piece together proximal mechanisms with biophysiological outcome studies, such as the potential for the dopamine pleasure pathway to mediate the relationship between soft tissue manipulation and the experience of decreased pain, increased relaxation, and improved affect. Regardless of which mechanisms or pathways future researchers choose to evaluate, it is clear from the massage literature that more randomized controlled trials, with theory-based methodology, are needed to address these questions of mechanism and, thereby, improve the creditability of massage with health care professionals.

Conclusions

The commonly cited hypothesis that massage influences the experience of pain via the reduction of ascending pain signals received some support in this study. There was no effect of massage on immediate pain measures of pain threshold, pain tolerance, or pain intensity VAS over time or in comparison to a control group and a guided imagery alone group. However, effects of massage intervention were observed on pain unpleasantness and residual pain intensity. These findings support past research that recommends massage as an intervention for pain but suggests that massage may alter the affective and residual/secondary qualities of pain versus physically interrupting pain signals via the GCM ascending pain pathway. Results also support hypotheses that massage has unique effects on affective and relaxation processes compared to guided imagery, though these processes appear to be independent of the GCM descending pain pathway. Furthermore, physiological sympathetic activity effects of massage may be

mediated via mechanisms of unpleasant affect and self-report relaxation. Overall, these results provide evidence for the value of massage in mental and physical health outcomes.

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Table 1. Characteristics of experimental pain models

Pain model	Theoretical Model			Experimental Model			Practical
	Activates C fibers	Minimal A-delta fibers	Minimal A-beta fibers	Quick nerve deactivation	Low nerve fatigue	Measurement precision	Low participant and administration burden.
Saline muscle soreness	●						
Cold-pressor	●				●	●	●
Infrared heat	●			●			●
Mechanical Pressure	●			●		●	●
Capsaicin intradermal injection	●	●	●				
Electrical current stimulation	●	●	●	●	●	●	●

Table 2. Pain threshold and tolerance means

	Baseline stimulation *M(SD)*	Intervention stimulation 1 *M(SD)*	Intervention stimulation 2 *M(SD)*	Intervention stimulation 3 *M(SD)*	Recovery stimulation *M(SD)*
Pain threshold					
Control	27.59 (11.87)	26.41(10.60)	27.23(10.90)	28.25(12.03)	26.34(11.09)
Guided imagery alone	23.66(13.95)	23.14(16.18)	26.29(15.25)	25.85(16.12)	24.79(13.68)
Massage alone	26.31(15.05)	28.24(14.16)	27.82(14.77)	28.78(15.04)	26.09(14.83)
Massage plus guided imagery	29.92(14.64)	28.68(14.57)	31.49(12.25)	31.01(14.42)	28.95(11.79)
Pain tolerance					
Control	42.36(13.28)	41.69(12.31)	42.02(12.35)	42.16(12.75)	41.26(12.86)
Guided imagery alone	37.31(13.23)	36.54(13.19)	36.98(13.39)	37.23(13.32)	36.34(12.77)
Massage alone	41.30(13.73)	41.19(13.37)	41.79(12.55)	41.77(12.62)	40.73(13.63)
Massage plus guided imagery	41.66(14.29)	40.57(12.81)	41.37(13.23)	41.58(12.97)	40.09(12.71)

Table 3. Pain outcomes means

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
Pain intensity					
Control	.70(2.57)	51.07(20.02)	59.45(21.42)	2.05(5.28)	48.41(28.57)
Guided imagery alone	3.04(6.04)	47.67(15.17)	55.90(20.43)	3.31(8.47)	45.23(22.77)
Massage alone	1.25(3.19)	56.25(15.25)	61.04(16.04)	2.46(5.13)	55.58(23.49)
Massage plus guided imagery	3.23(11.38)	54.73(25.48)	62.31(23.77)	2.29(7.37)	57.21(27.85)
Pain unpleasantness					
Control	2.64(4.91)	48.16(22.41)	55.48(23.78)	3.43(6.99)	43.96(31.25)
Guided imagery alone	4.23(6.91)	50.98(15.18)	52.21(14.71)	6.35(13.62)	49.46(23.64)
Massage alone	4.60(7.42)	53.52(19.54)	51.23(20.03)	3.13(4.25)	53.06(17.93)
Massage plus guided imagery	5.06(7.62)	51.77(28.05)	50.15(27.84)	1.99(3.66)	53.29(31.21)
Current pain descriptive					
Control	.95(.38)	1.09(.53)	1.18(.85)	.95(.38)	1.05(.58)
Guided imagery alone	.96(.20)	1.23(.71)	1.12(.52)	1.04(.34)	1.08(.48)

Table 3. Pain outcomes means continued

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
Massage alone	.96(.36)	1.29(.75)	1.00(.42)	.92(.28)	1.04(.36)
Massage plus guided imagery	.96(.36)	1.29(.81)	1.00(.42)	1.08(.78)	1.17(.48)
Worst pain descriptive					
Control	---	2.55(.74)	2.59(.91)	---	2.50(.86)
Guided imagery alone	---	2.73(.60)	2.54(.71)	---	2.31(.84)
Massage alone	---	2.79(.66)	2.79(.88)	---	2.88(.90)
Massage plus guided imagery	---	2.58(1.06)	2.67(.87)	---	2.54(1.02)
Least pain descriptive					
Control	---	1.27(.70)	1.23(.43)	---	1.14(.35)
Guided imagery alone	---	1.23(.43)	1.15(.37)	---	1.15(.37)
Massage alone	---	1.25(.44)	1.21(.51)	---	1.33(.48)
Massage plus guided imagery	---	1.29(.46)	1.17(.48)	---	1.21(.51)

Table 4. Affect outcome means

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
Unactivated unpleasant affect					
Control	1.02(.76)	.57(.78)	.89(1.02)	1.05(.84)	.77(.81)
Guided imagery alone	1.27(1.04)	.60(.75)	.62(.73)	1.00(1.05)	.85(.86)
Massage alone	1.13(.86)	.52(.63)	.38(.52)	.88(.59)	.71(.83)
Massage plus guided imagery	1.10(1.02)	.73(.93)	.38(.68)	1.06(.92)	.83(.89)
Low activation affect					
Control	2.64(.99)	1.50(.96)	1.86(1.10)	2.34(1.08)	1.70(1.00)
Guided imagery alone	2.23(.86)	.98(.75)	2.06(.94)	2.06(.86)	1.38(.91)
Massage alone	2.02(1.09)	1.27(.86)	1.88(.98)	1.77(1.04)	1.33(.96)
Massage plus guided imagery	2.25(.96)	1.40(.91)	2.04(.88)	1.88(1.01)	1.77(.93)
High activation affect					
Control	.26(.29)	.83(.48)	.64(.58)	.14(.20)	.50(.41)
Guided imagery alone	.60(.53)	1.32(.72)	.67(.51)	.27(.30)	.73(.63)

Table 4. Affect outcome means continued

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
Massage alone	.53(.45)	1.10(.69)	.58(.43)	.38(.33)	.65(.51)
Massage plus guided imagery	.33(.48)	1.18(.75)	.53(.52)	.35(.46)	.60(.66)
Unpleasant affect					
Control	.57(.25)	.55(.32)	.59(.32)	.55(.24)	.50(.30)
Guided imagery alone	.64(.32)	.58(.35)	.46(.26)	.47(.27)	.51(.38)
Massage alone	.64(.27)	.59(.32)	.34(.23)	.47(.23)	.43(.29)
Massage plus guided imagery	.65(.33)	.59(.30)	.36(.25)	.54(.35)	.47(.33)
Pleasant affect					
Control	1.85(.60)	.94(.73)	.93(.59)	1.33(.57)	.90(.67)
Guided imagery alone	1.71(.86)	.74(.67)	1.38(.86)	1.47(.80)	.95(.74)
Massage alone	1.58(.72)	.81(.70)	1.64(.68)	1.53(.73)	1.26(.82)
Massage plus guided imagery	1.76(.82)	.98(.92)	1.90(.79)	1.63(.87)	1.21(.86)

Table 5. Relaxation and mindfulness scores

	Intervention *M(SD)*
Relax VAS	
Control	6.01(2.24)
Guided imagery alone	6.96(1.89)
Massage alone	8.15(1.51)
Massage plus guided imagery	7.78(1.63)
CAMS-R	
Control	35.86(4.49)
Guided imagery alone	36.31(4.59)
Massage alone	35.50(3.55)
Massage plus guided imagery	35.50(4.54)

Table 6. Physiological outcome means

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
MHR					
Control	73.70(12.69)	73.45(12.63)	72.88(11.98)	71.73(12.53)	72.83(11.81)
Guided imagery alone	77.29(13.64)	78.22(12.43)	75.74(11.95)	75.68(12.47)	75.28(12.24)
Massage alone	75.18(9.77)	74.00(9.17)	71.64(9.83)	71.55(8.97)	72.24(8.63)
Massage plus guided imagery	73.54(8.67)	73.18(8.43)	68.85(7.12)	70.09(7.52)	70.58(8.39)
MIBI					
Control	838.89(148.13)	840.32(137.56)	849.22(134.40)	862.67(146.69)	848.21(145.20)
Guided imagery alone	800.56(142.98)	791.39(129.73)	813.56(135.10)	816.18(143.63)	821.10(149.35)
Massage alone	811.37(100.65)	823.63(944.81)	855.63(121.10)	853.15(102.89)	844.75(103.23)
Massage plus guided imagery	827.23(91.03)	832.56(89.20)	882.32(85.41)	869.27(91.25)	864.89(101.36)
LF power					
Control	911.69(916.05)	806.17(694.15)	129.58E2(563.56E2)	162.97E1(163.97E1)	120.73E1(115.35E1)
Guided imagery alone	935.62(675.75)	124.97E1(220.05E1)	104.98E3(527.41E3)	148.88E1(215.34E1)	103.92E1(219.29E1)

Table 6. Physiological outcome means continued

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
Massage alone	106.65E1(144.11E1)	774.54(625.13)	137.59E1(228.63E1)	170.81E1(247.75E1)	132.57E1(229.43E1)
Massage plus guided imagery	901.63(494.29)	995.89(640.48)	169.36E1(225.20E1)	505.27E1(160.68E2)	132.86E1(221.01E1)
LF/HF power ratio					
Control	.80(.69)	.78(.88)	.93(.68)	1.37(1.30)	.83(.74)
Guided imagery alone	1.22(1.24)	1.34(1.09)	1.25(.94)	1.48(1.15)	.65(.62)
Massage alone	1.13(1.12)	.93(1.15)	1.18(1.20)	1.39(1.34)	.80(.69)
Massage plus guided imagery	1.03(.66)	1.07(.68)	.95(.42)	2.28(3.85)	1.76(4.45
Systolic blood pressure					
Control	107.09(8.82)	108.61(9.23)	105.19(9.00)	104.70(9.62)	101.64(10.84)
Guided imagery alone	107.96(7.94)	110.35(9.65)	107.32(10.39)	105.54(9.71)	106.58(9.72)
Massage alone	107.25(8.87)	112.17(10.26)	105.33(8.06)	106.81(7.34)	107.25(6.87)
Massage plus guided imagery	108.61(7.83)	110.52(7.87)	104.87(8.54)	107.13(8.07)	109.08(12.53)

Table 6. Physiological outcome means continued

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
Diastolic blood pressure					
Control	77.25(8.63)	79.84(8.91)	81.50(7.62)	81.05(9.25)	78.82(8.88)
Guided imagery alone	77.60(7.64)	78.73(10.21)	78.44(8.66)	79.20(8.37)	79.54(9.95)
Massage alone	77.28(10.46)	82.33(9.62)	80.32(7.67)	82.16(5.81)	83.63(8.06)
Massage plus guided imagery	75.46(10.14)	81.31(10.04)	80.99(8.90)	81.86(7.91)	82.33(14.93)
RSA					
Control	6.94(1.23)	7.08(1.37)	7.13(1.45)	7.16(1.24)	7.09(1.34)
Guided imagery alone	6.71(1.15)	6.64(.97)	6.77(.95)	6.59(1.02)	6.81(1.00)
Massage alone	6.79(1.13)	6.77(1.15)	6.72(1.26)	6.82(1.17)	6.91(1.18)
Massage plus guided imagery	6.83(.74)	6.87(.85)	7.08(.85)	6.83(.94)	6.93(1.03)
HF power					
Control	249.60E1(418.23E1)	338.83E1(657.84E1)	521.79E3(243.70E4)	311.48E1(473.26E1)	728.15E1(142.90E2)
Guided imagery alone	153.47E1(129.66E1)	151.94E1(230.65E1)	114.72E4(581.52E4)	658.12E1(265.49E2)	841.52E1(273.95E2)

Table 6. Physiological outcome means continued

	Baseline *M(SD)*	Baseline stimulation *M(SD)*	Intervention *M(SD)*	Recovery *M(SD)*	Recovery stimulation *M(SD)*
Massage alone	168.44E1(170.43E1)	164.36E1(139.70E1)	753.06E1(275.78E2)	763.46E1(276.00E2)	919.75E1(283.77E2)
Massage plus guided imagery	133.96E1(768.24)	158.94E1(144.34E1)	755.53E1(275.16E2)	738.54E1(275.63E2)	733.65E1(275.76E2)

Figure 1. Examined potential mechanisms of massage

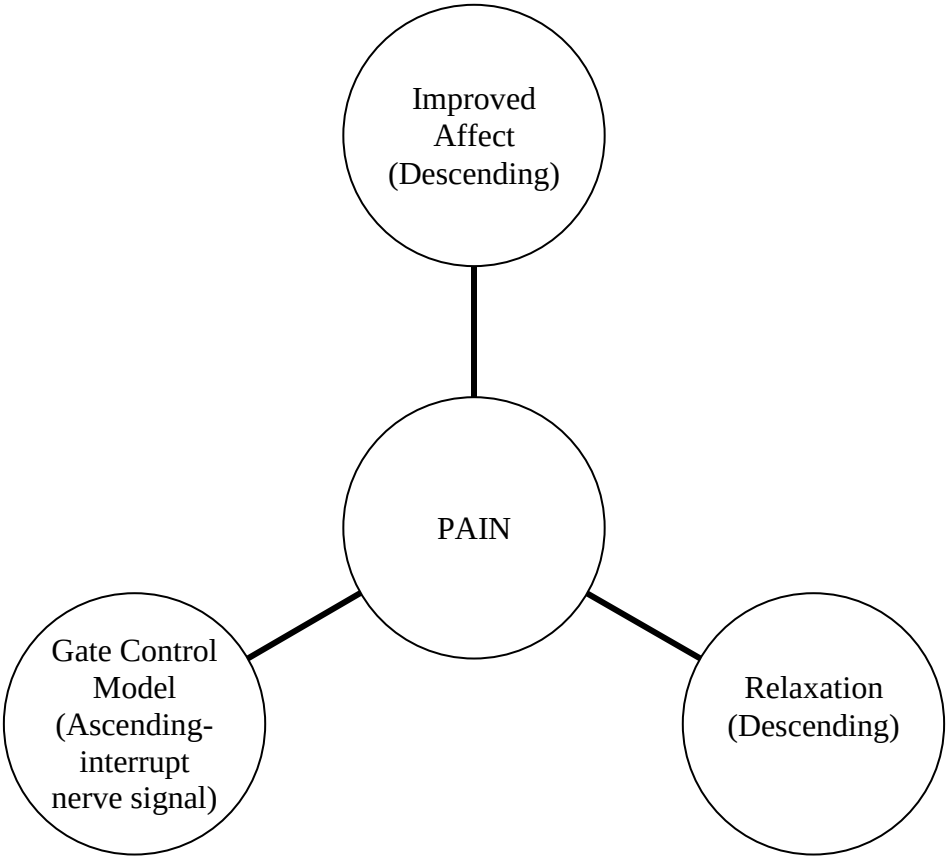


Figure 2. Flow diagram of participant recruitment and retention

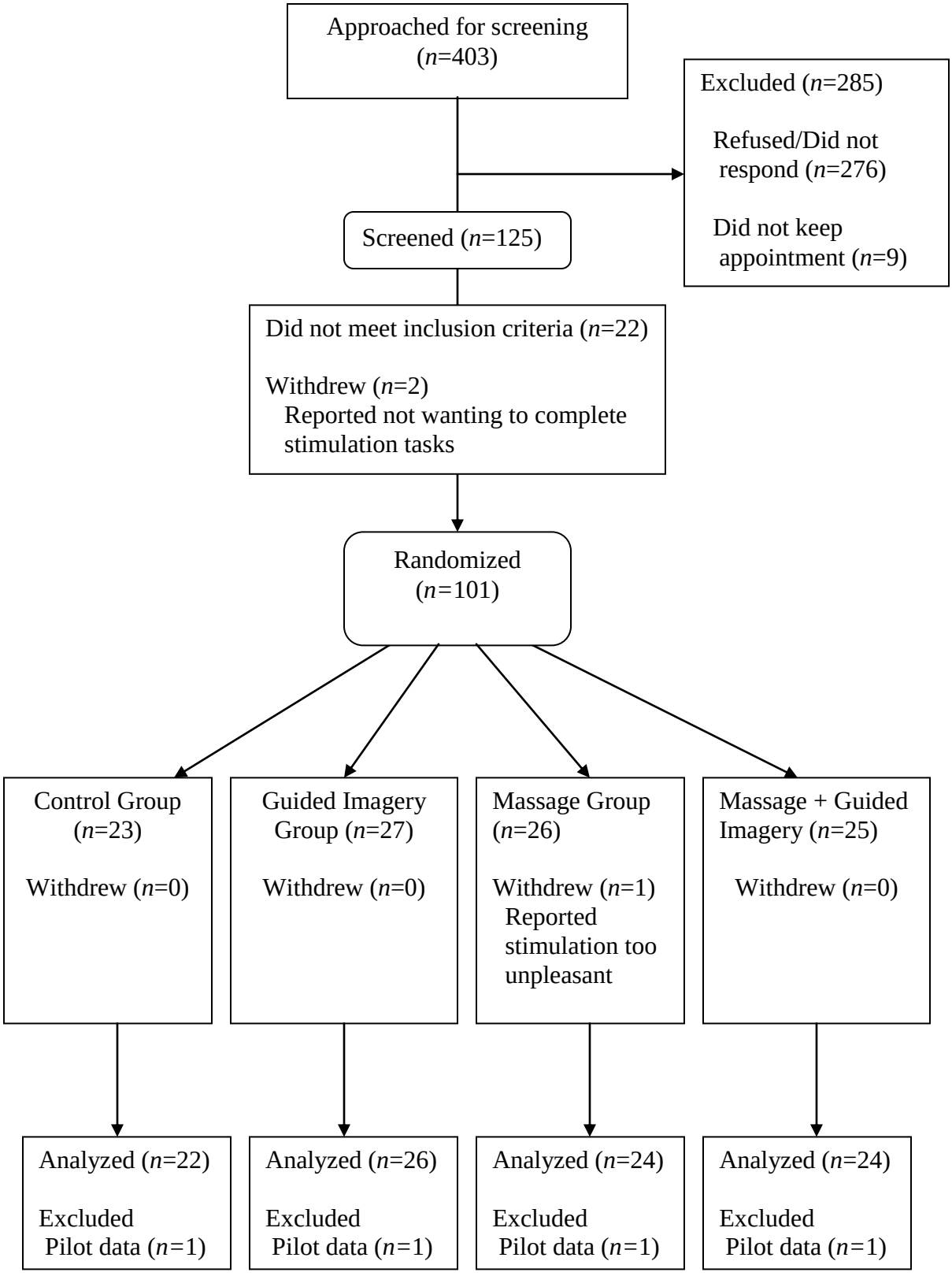
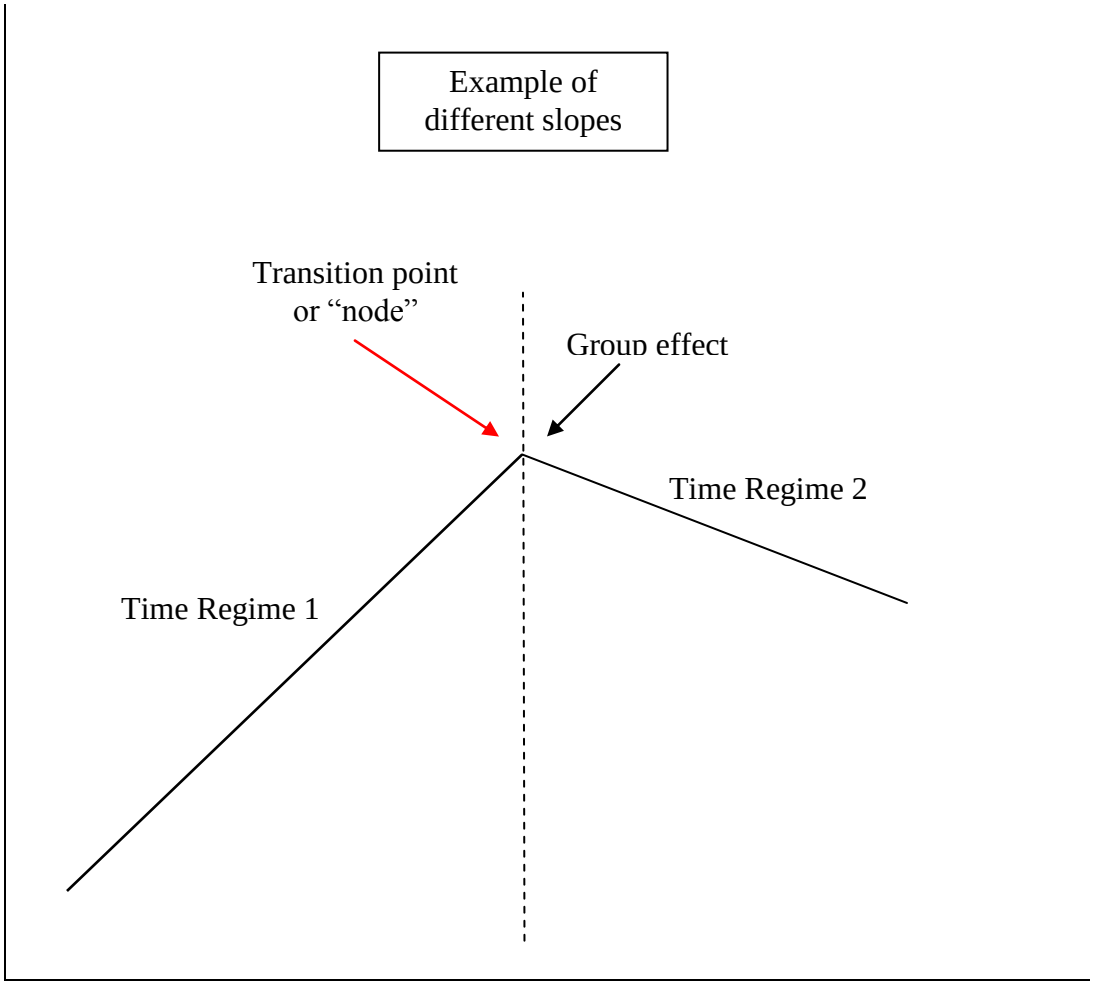


Figure 3. Study timeline

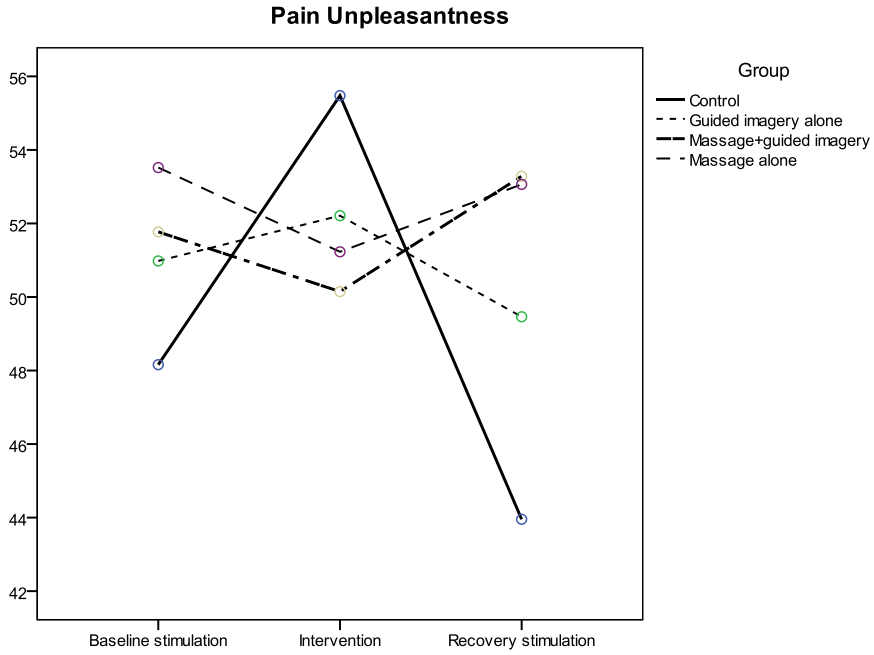
Pretest→	Baseline rest→	Baseline stimulation→	Intervention (experimental groups →)	Recovery rest→	Post-recovery stimulation
	(10 min.)	(2 pain trials)	(3 pain trials, 15.5 min.)	(10 min.)	(1 pain trial)

Figure 4. Piecewise MLM analysis model



(1a) Baseline stimulation (2) Intervention (3a) Post-recovery stimulation
(1b) Baseline rest (3b) Recovery rest

Figure 5. Group differences in pain unpleasantness and residual pain intensity across study



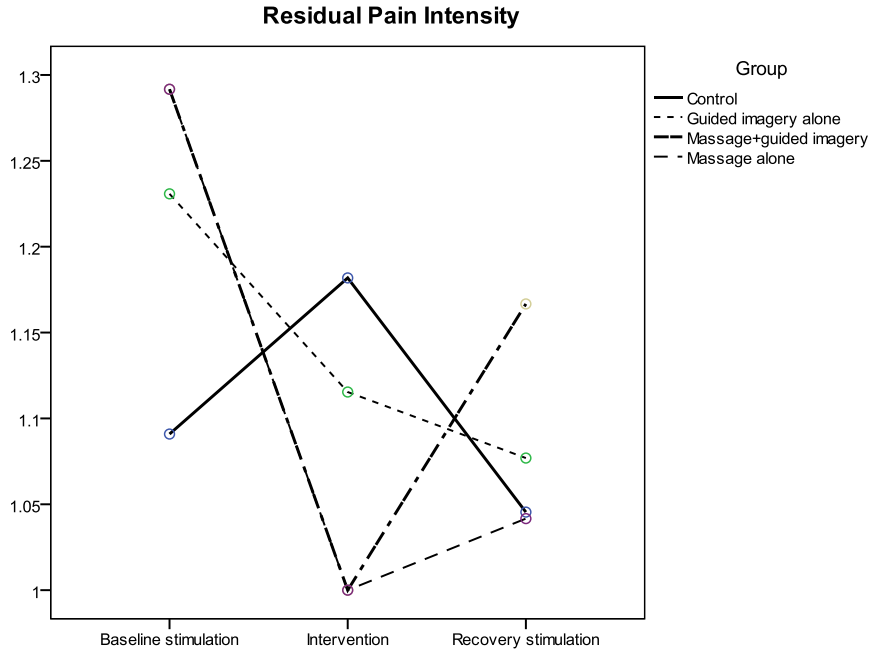


Figure 6. Group differences in unactivated unpleasant affect across study

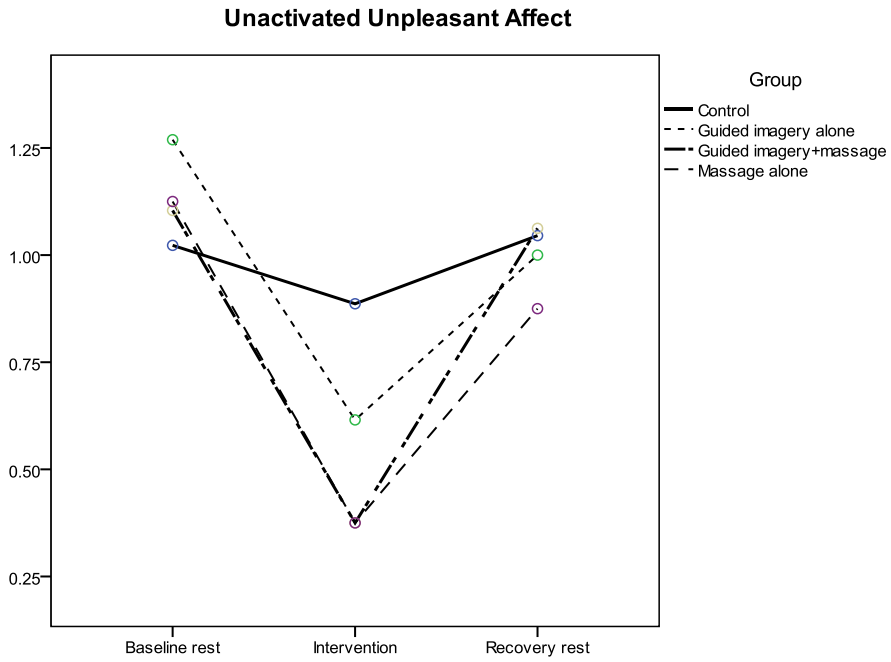


Figure 7. Group differences in low and high activation affect across study

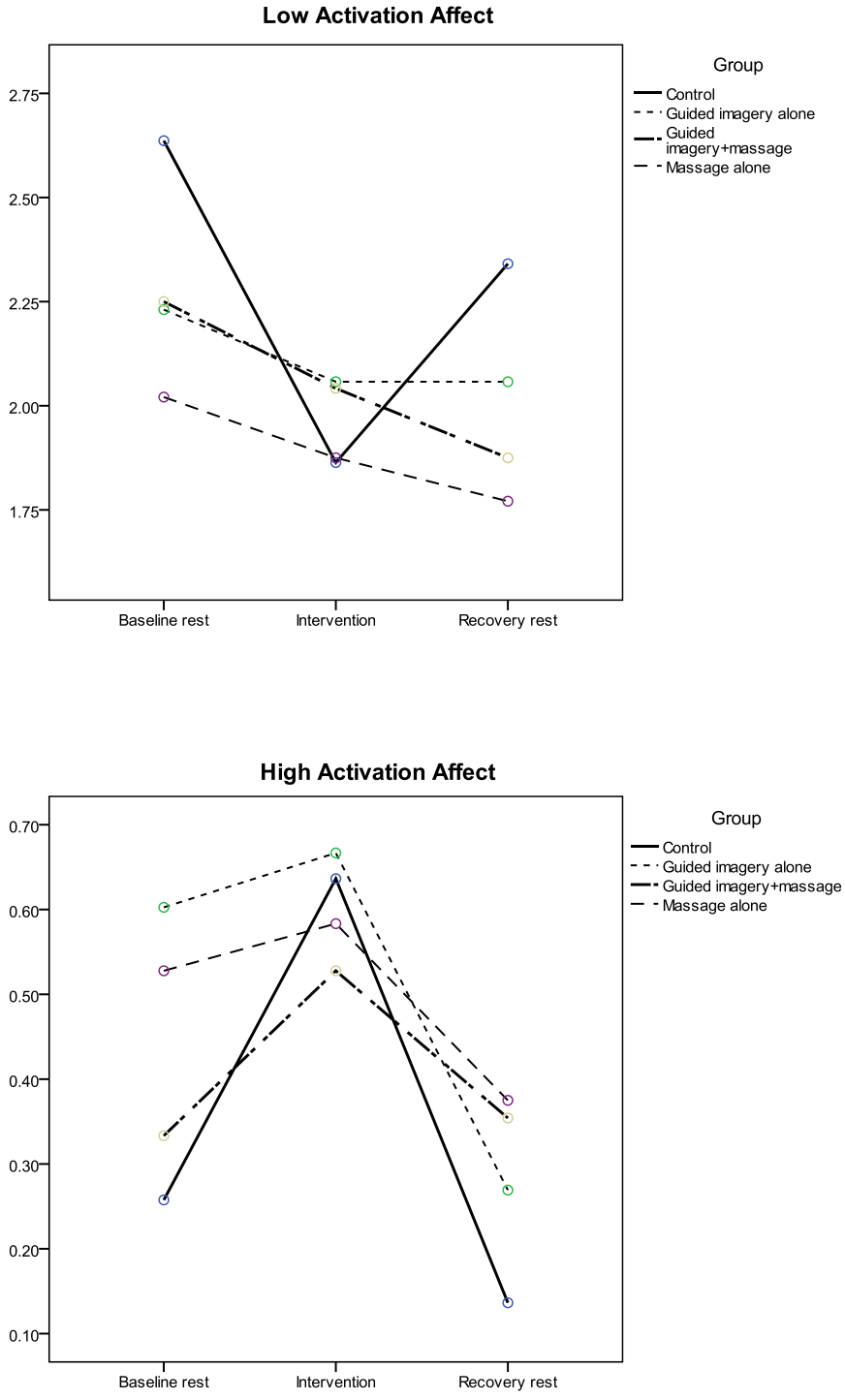


Figure 8. Group differences in unpleasant and pleasant affect across study

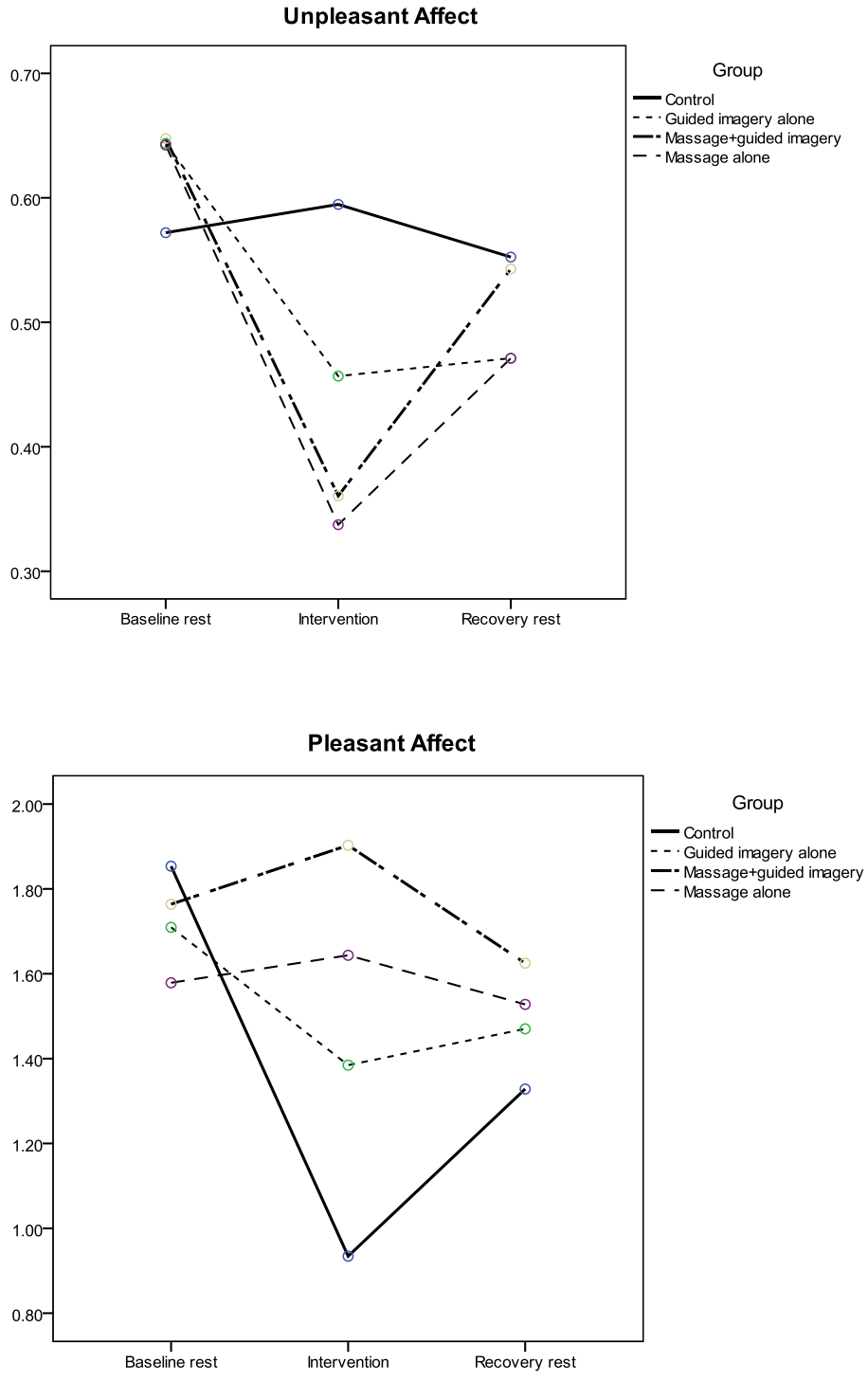
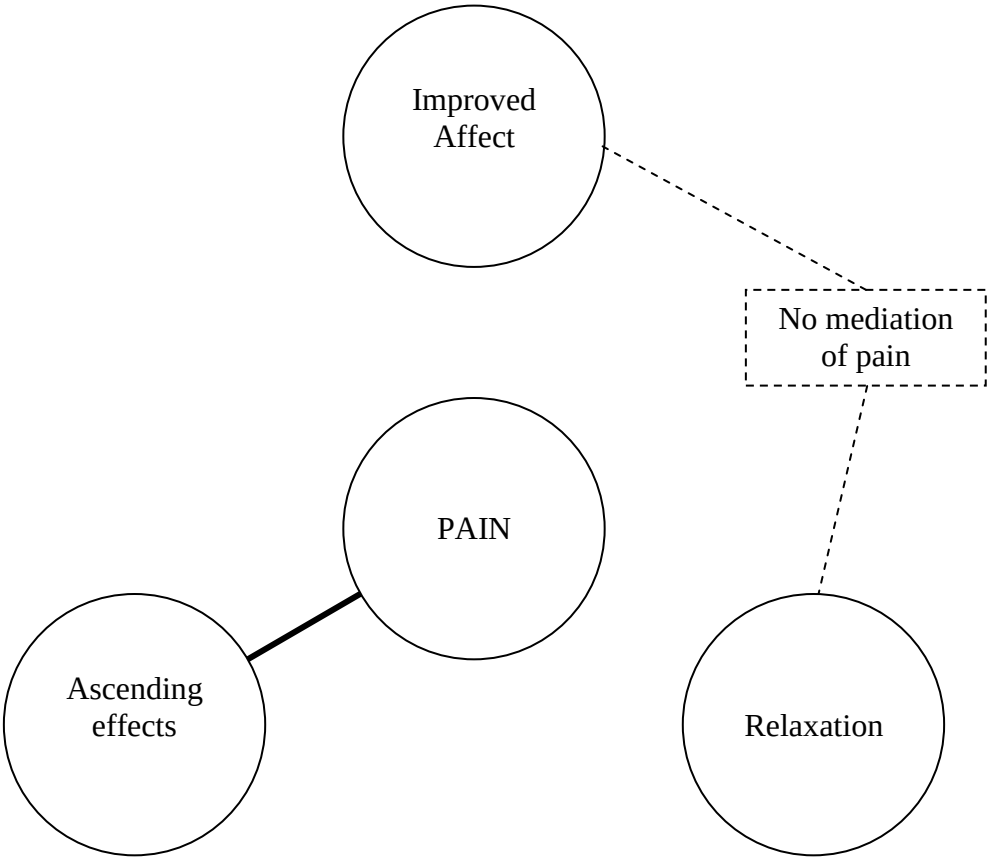


Figure 9. Supported mechanisms of massage



Appendices

Appendix A: Informed Consent Statement

Massage and the Gate Control Model

INTRODUCTION

The Department of Psychology at the University of Kansas supports the practice of protection for human subjects participating in research. The following information is provided for you to decide whether you wish to participate in the present study. You may refuse to sign this form and not participate in this study. You should be aware that even if you agree to participate, you are free to withdraw at any time. If you do withdraw from this study, it will not affect your relationship with this unit, the services it may provide to you, or the University of Kansas.

PURPOSE OF THE STUDY

The purpose of this study is to examine how guided imagery and massage interventions impact the perception of and reaction to pain. The study will also examine how various thoughts, attitudes, and emotions may be related to pain reactivity.

PROCEDURES

This study consists of two phases. During the first phase you will first be asked to complete several questionnaires that assess general health status, perceptions of pain, sleep history, and emotions. We will then assess psychological and physiological reactions to an electrical stimulation pain procedure using questionnaires and physiological recording equipment. In order to assess psychological reactivity, a same sex research assistant will place an electrocardiogram (ECG) electrode under each clavicle and just below your left floating rib. You will also be fitted with a respiration belt, and a blood pressure cuff.

You will be randomly assigned to a no-treatment group, guided imagery group or guided imagery plus massage group. You will be asked to sit quietly for ten minutes before and after the experimental group procedure. Electrical stimulation will be presented through electrodes placed on your right forearm at varying time intervals throughout the experiment. You will be asked to indicate when you first feel pain and when you can no longer tolerate the pain, at which point stimulation will be terminated. The total time that it will take to complete the questionnaires and the experiment should be no more than 90 minutes.

RISKS

There are no anticipated risks associated with the physiological recording equipment you will be fitted to, although wearing this throughout the study may feel somewhat inconvenient. The electrical stimulation will present a mild to moderate pain stimulus; however, we do not expect any long-term aversive reactions to this commonly used pain

procedure. Again, you are free to stop at any time during the study. Also, if you are currently experiencing emotional distress, we recommend that you contact the KU Psychological Clinic, 340 Fraser Hall, 785-864-4121 or the Watkins Memorial Health Center, Counseling and Psychological Services, 785-864-2277.

BENEFITS

There are unlikely to be any direct benefits to you other than receiving course credit. Because the study will increase our understanding of how massage, relaxation, cognition, and emotion are associated with psychological and physiological reactions to pain, the study may help identify the mechanisms of massage on reducing pain and therefore be beneficial to society.

PAYMENT TO PARTICIPANTS

You will not be paid for participating but you will receive up to 3 credits toward Psychology 104 requirements.

INFORMATION TO BE COLLECTED

To perform this study, we will collect information about you. This information will be obtained from questionnaires and measures of physiology. The questionnaires will assess your experience with pain and relaxation techniques, sleep quality, thoughts, attitudes, and emotions, as well as information about your general health status. We will use an electrocardiogram (ECG) to measure heart rate, a blood pressure cuff to measure blood pressure and a respiration belt to measure respiration.

The information we collect will be kept confidential. Your name will not be associated in any way with the information collected about you or with the research findings from this study. The only time we would break confidentiality would be to ensure your safety or the safety of others. You will be assigned a unique identification number and will be identified by that number in all data files. A master file with participant contact information and identification numbers will be password protected and stored separately.

The information collected about you will be used by: Dr. Nancy Hamilton, Cynthia Karlson M.A., and the members of her research team. The researchers will not share information about you with anyone not specified above unless required by law or unless you give written permission.

Permission granted on this date to use and disclose your information remains in effect indefinitely. By signing this form you give permission for the use and disclosure of your information for purposes of this study at any time in the future.

INSTITUTIONAL DISCLAIMER STATEMENT

In the event of injury, the Kansas Tort Claims Act provides for compensation if it can be demonstrated that the injury was caused by the negligent or wrongful act or omission of a state employee acting within the scope of his/her employment.

REFUSAL TO SIGN CONSENT AND AUTHORIZATION

You are not required to sign this Consent and Authorization form and you may refuse to do so without affecting your right to any services you are receiving or may receive from the University of Kansas or to participate in any programs or events of the University of Kansas. However, if you refuse to sign, you cannot participate in this study.

CANCELLING THIS CONSENT AND AUTHORIZATION

You may withdraw your consent to participate in this study at any time. You also have the right to cancel your permission to use and disclose information collected about you, in writing, at any time, by sending your written request to: Dr. Nancy Hamilton or Cynthia Karlson, M.A., Dept. of Psychology, 1415 Jayhawk Blvd, University of Kansas, Lawrence, KS 66045. If you cancel permission to use your information, the researchers will stop collecting additional information about you. However, the research team may use and disclose information that was gathered before they received your cancellation, as described above.

QUESTIONS ABOUT PARTICIPATION

Questions about procedures should be directed to the researcher(s) listed at the end of this consent form.

PARTICIPANT CERTIFICATION:

I have read this Consent and Authorization form. I have had the opportunity to ask, and I have received answers to, any questions I had regarding the study and the use and disclosure of information about me for the study. I understand that if I have any additional questions about my rights as a research participant, I may call (785) 864-7429 or write the Human Subjects Committee Lawrence Campus (HSCL), University of Kansas, 2385 Irving Hill Road, Lawrence, Kansas 66045-7563, email dhann@ku.edu.

I agree to take part in this study as a research participant. I further agree to the uses and disclosures of my information as described above. By my signature I affirm that I am at least 18 years old and that I have received a copy of this Consent and Authorization form.

_____ _____

Type/Print Participant's Name Date

_____

Participant's Signature

Researcher Contact Information:

Dr. Nancy Hamilton, Principal Investigator
Department of Psychology

Appendix B: Guided imagery script outline

Begin by getting yourself into as comfortable a position as possible. When you're ready, begin by taking some nice deep diaphragmatic breaths.

Notice as do that all the sensations that you feel, in you nose, throat, and chest, as you breath in and as you breath out.

When you are ready, I'd like for you to imagine a scene in your mind. Imagine yourself standing in front of a very beautiful garden. There are all sorts of flowers and plants in this garden and a path that travels through the garden.

When you are ready, start to walk along the path in the garden. Notice what the path feels like under you feet. Notice the sights of the garden. Notice the sounds and smells of the garden, and notice how you feel in the garden.

As you come around a corner, you find a small waterfall in the center of the garden. Notice all the sensations that are associated with the waterfall. Notice the ripples and sounds of the water. As you drink the water, you notice that it calms and soothes you inside. If you like, you can get in the water. You notice that the water is the perfect temperature and lifts you up. The water carries away all the bad feelings and leaves nothing but calm and peace behind.

It is now time to leave the water. You now notice the warmth of the sunlight. Your body soaks up the warm, relaxing sunlight and your body parts (feet, ankles, legs, abdomen, organs, chest, shoulders, upper arms, forearms, neck, and face) begin to feel warmer, heavier and relaxed. All the feelings of discomfort and tension are replaced by warmth, heaviness and relaxation. Your body is now completely bathed in sunlight and feels very pleasant and calm.

It is now time to leave the garden. Again, notice the sights, sounds, and smells of the waterfall. Notice how the water tastes and feels. Now once again, notice yourself on the path. Notice again, the sights, sounds, and smells or the garden. Now once again notice yourself at the gate of the garden, knowing that you can come back anytime you want. Now once again, focus on your breathing. Notice again the sensations in your nose, throat, and chest as you breath in and as you breath out. When you are ready, slowly open your eyes, becoming fully aware of what's around you but remaining relaxed and refreshed.

SID _____
Date _____

Appendix C: Massage protocol

1. *Back:*
   (a) downward strokes along the back
   (b) hand-over-hand movements from the upper back to the hips
   (c) hands from side to side across the back, including the sides
   (d) circular motion from head to hips along, but not touching, the spine
   (e) simultaneous strokes over the sides of the back from the middle to the sides
   (f) rubbing and kneading shoulder muscles
   (g) rubbing the neck
   (h) stroking the length of the back
   (i) stroking from head to feet.

2. *Shoulders:*
   (a) alternate squeezing of the upper shoulders
   (b) stroke from spine to shoulder blade
   (c) stripping with thumb from spine to shoulder blade
   (d) kneading with finger tips and heal of hand

3. *Neck:*
   (a) cup neck and alternate squeezing from shoulders to head
   (b) strip sides of neck with thumb and tips of fingers
   (c) stroke occiput and base of scalp with finger tips
   (d) knead back and sides of neck with finger tips

4. *Upper Arms:*
   (a) stroking from shoulders to the hands
   (b) stokes from shoulder to elbow
   (c) squeezing and twisting in a wringing motion from shoulder to elbow
   (d) stroking the arms upwards toward the heart

5. *Hands:*
   (a) opening palms in heart shaped motion
   (b) stoking inside of palm with thumbs
   (c) stripping back of hand with thumbs
   (d) squeezing and twisting in a wringing motion each finger
   (e) wringing palms from center to sides

SID _____
Date _____

Appendix D: Dermatome body outline

(Published in Lee et al., 2008)

SID _____
Date _____

Appendix E: Medical History

This booklet contains questionnaires about your general health, thoughts, attitudes, and emotions. All of your responses will be kept strictly confidential. Please respond to the following items.

What is today's date? _____

What is your name (First and Last)? _____

What is your KU student ID? _____

What is your GENDER? (circle one) *FEMALE* *MALE*

What is your AGE in years? _____

What is your ETHNICITY? ____ White/Caucasian American
____ Black/African American
____ Asian/Asian American
____ Latino(a)/Hispanic American
____ Native American
____ Multi-Racial

Staff Use:

Weight _____ (kg)

Height _____ (cm)

Group _____

SID _____
Date _____

Participant Health Information

As with all information that we collect during this study, we will keep this information strictly confidential.

Do you have, or have you ever had any of the following chronic illnesses? *Please circle yes or no*

1. Serious lung / breathing troubles?	YES NO
2. High blood pressure / hypertension?	YES NO
3. Diabetes?	YES NO
4. Hypoglycemia?	YES NO
5. Have you ever had heart disease, angina, or a heart attack?	YES NO
6. Hardening of the arteries?	YES NO
9. Ulcers of other intestinal or stomach disorders?	YES NO
10. Spinal injury or disease?	YES NO
11. Have you ever had circulation trouble in your arms or legs?	YES NO
12. Anemia?	YES NO
13. Chronic, severe headaches?	YES NO
14. Chronic, serious back or neck pain?	YES NO
15. Arthritis?	YES NO
16. Chronic pain condition (regional pain syndrome, etc)?	YES NO
17. Neurological disorders?	YES NO
18. Dermatologic disorders?	YES NO
19. Major trauma or surgery to arms or legs?	YES NO
20. Do you have any other chronic illness of any type? (if yes, please describe:_____	YES NO

SID _____
Date _____

Medications List

Please list all of the **prescription *and* non-prescription (over the counter) medication** you have taken during the *last month*

Medication name	**dosage (mg)**	**doses x day**	**currently taking?**	
1_____	_____	_____	YES	NO
2_____	_____	_____	YES	NO
3_____	_____	_____	YES	NO
4_____	_____	_____	YES	NO
5_____	_____	_____	YES	NO
6_____	_____	_____	YES	NO
7_____	_____	_____	YES	NO
8_____	_____	_____	YES	NO
9_____	_____	_____	YES	NO
10_____	_____	_____	YES	NO

Please list all of the **vitamins, food-supplements, diet-aids, herbs, natural remedies, homeopathic medicines, and alternative medicines** you have taken during the *last month*.

Medication name	**dosage (mg)**	**doses x day**	**currently taking?**	
1_____	_____	_____	YES	NO
2_____	_____	_____	YES	NO
3_____	_____	_____	YES	NO
4_____	_____	_____	YES	NO
5_____	_____	_____	YES	NO
6_____	_____	_____	YES	NO
7_____	_____	_____	YES	NO
8_____	_____	_____	YES	NO
9_____	_____	_____	YES	NO
10_____	_____	_____	YES	NO

SID _____
Date _____

Relaxation Experience

Have you ever practiced: (circle all that apply)

Yoga	If yes, how often?	A little	Some	A lot
Tai chi	If yes, how often?	A little	Some	A lot
Martial arts	If yes, how often?	A little	Some	A lot
Qi gong	If yes, how often?	A little	Some	A lot
Meditation	If yes, how often?	A little	Some	A lot
Guided imagery	If yes, how often?	A little	Some	A lot
Deep breathing	If yes, how often?	A little	Some	A lot
Other: _____	If yes, how often?	A little	Some	A lot

Have you ever received: (circle all that apply)

Relaxation massage	If yes, how often?	A little	Some	A lot
Deep tissue massage	If yes, how often?	A little	Some	A lot
Tai chi massage	If yes, how often?	A little	Some	A lot
Shiatsu massage	If yes, how often?	A little	Some	A lot
Reiki massage	If yes, how often?	A little	Some	A lot
Other: _____	If yes, how often?	A little	Some	A lot

Types of regular exercise (circle all that apply):

None			
Gym	< 1 day/week	1-3 days/week	>3 days/week
Walking	< 1 day/week	1-3 days/week	>3 days/week
Jogging/Running	< 1 day/week	1-3 days/week	>3 days/week
Swimming	< 1 day/week	1-3 days/week	>3 days/week
Sports	< 1 day/week	1-3 days/week	>3 days/week
Dance	< 1 day/week	1-3 days/week	>3 days/week
Other: _____	< 1 day/week	1-3 days/week	>3 days/week

Average # days/week exercise: 1 2 3 4 5 6 7

SID _____
Date _____

Appendix F: Beck Anxiety Inventory

(Omitted due to copy write)

SID _____
Date _____

Appendix G: Beck Depression Inventory-II

(Omitted due to copy write)

SID _____
Date _____

Appendix H: Frid Scale

Please indicate in the alternatives given below how you *think right now*

1. I want to avoid the situation	1 not at all	2	3	4	5 very much so
2. I believe I can tolerate the pain	1 not at all	2	3	4	5 very much so
3. I see the experience as a challenge	1 not at all	2	3	4	5 very much so
4. I think the procedure will be painful	1 not at all	2	3	4	5 very much so
5. I know that nothing concerning this experiment 'can really hurt me'	1 not at all	2	3	4	5 very much so
6. 1 care that other people may notice my weaknesses	1 not at all	2	3	4	5 very much so
7. I think this experiment will be interesting	1 not at all	2	3	4	5 very much so
8. I expect to suffer unpleasant after-effects	1 not at all	2	3	4	5 very much so
9. I believe that my participation is important	1 not at all	2	3	4	5 very much so
10. l expect to be in control of the situation	1 not at all	2	3	4	5 very much so

SID _____
Date _____

Appendix I: National Sleep Foundation Sleep Diary

	COMPLETE IN MORNING						
Fill out days 1-7 below	I went to bed last night at:	I got out of bed this morning at:	Last night I fell asleep in:	I woke up during the night: (Record number of times)	When I woke up for the day, I felt: (Check one)	Last night I slept for a total of: (Record number of hours)	My sleep was disturbed by: (List any mental, emotional, physical, or environmental factors that affected your sleep, e.g. stress, snoring, physical discomfort, temperature)
DAY 1 day of wk _____ date _____ mm/dd/yy	_____ PM/AM	_____ AM/PM	_____ Minutes	_____ Times	___ Refreshed ___ Somewhat Refreshed ___ Fatigued	_____ Hours	_____ _____ _____ _____ _____

	COMPLETE AT END OF DAY				
Fill out day 1 below	I consumed caffeinated drinks (e.g. coffee, tea, cola) in the:	I exercised at least 20 minutes in the:	Approximately 2-3 hours before going to bed, I consumed:	Medication(s) I took during the day: [List name of medication/drug(s)]	About 1 hour before going to sleep, I did the following activity: (List activity; e.g. watch TV, work, read)
DAY 1 day of wk _____ date _____ mmddyy	___ Morning ___ Afternoon ___ About 2-3 hours before going to bed ___ Not applicable	___ Morning ___ Afternoon ___ About 2-3 hours before going to bed ___ Not applicable	___ Alcohol ___ Nicotine (cigarettes, etc.) ___ Street drugs ___ A heavy meal ___ Not applicable	_____ _____ _____ _____ _____	_____ _____ _____ _____ _____

SID _____
Date _____

Appendix J: Personal Affection and Touch Scale

INSTRUCTIONS: Please read over the following list of types of physical contact. For each type, please indicate HOW OFTEN you typically engage in that particular type of interaction by putting an **X** in the appropriate box.

(1) For this group of questions, please indicate how often you engage in these activities with your **ROMANTIC PARTNER** (if you have one).

Type of Contact	At Least Once Daily (1)	At Least Once a Week (2)	At Least Once a Month (3)	Once Every Few Months (4)	Very Rarely or Never (5)
1. Hugging					
2. Shaking hands, slapping hands (e.g., high fives)					
3. Pat on the back					
4. Arm around shoulder					
5. Gentle touching (e.g., during conversation)					
6. Holding hands					
7. Massage					
8. Kissing hello/goodbye					
9. Intimate contact (e.g., kissing, cuddling)					
10. Tickling					

(2) For this group of questions, please indicate how often you engage in these activities **IN GENERAL** with friends, family & acquaintances (EXCLUDING your romantic partner).

Type of Contact	At Least Once Daily (1)	At Least Once a Week (2)	At Least Once a Month (3)	Once Every Few Months (4)	Very Rarely or Never (5)
1. Hugging					
2. Shaking hands, slapping hands (e.g., high fives)					
3. Pat on the back					
4. Arm around shoulder					
5. Gentle touching (e.g., during conversation)					
6. Holding hands					
7. Massage					
8. Kissing hello/goodbye					
9. Intimate contact (e.g., kissing, cuddling)					
10. Tickling					

SID _____
Date _____

(3) When considering your current, **average** level of physical contact with others, please indicate on the following scale where you would place yourself. (circle the appropriate number)

1	**2**	**3**	**4**	**5**
I want much less contact	I want a little less contact	I am satisfied with my current level of contact	I want a little more contact	I want much more contact

(4) On **average,** how much you do like physical contact with others? (circle the appropriate number)

1	**2**	**3**	**4**	**5**
Not at all	A little	Moderately	A lot	Always

(5) On **average,** how much does physical contact with others make you uncomfortable? (circle the appropriate number)

1	**2**	**3**	**4**	**5**
Not at all	A little	Moderately	A lot	Always

SID _____
Date _____

Appendix K: Cognitive and Affective Mindfulness Scale Revised

People have a variety of ways of relating to their thoughts and feelings. For each of the items below, rate how much each of these ways applies to *yo*u.

1 Rarely/Not at all	2 Sometimes	3 Often	4 Almost Always

_____ 1. It is easy for me to concentrate on what I am doing.

_____ 2. I am preoccupied by the future.

_____ 3. I can tolerate emotional pain.

_____ 4. I can accept things I cannot change.

_____ 5. I can usually describe how I feel at the moment in considerable detail.

_____ 6. I am easily distracted.

_____ 7. I am preoccupied by the past.

_____ 8. It's easy for me to keep track of my thoughts and feelings.

_____ 9. I try to notice my thoughts without judging them.

_____ 10. I am able to accept the thoughts and feelings I have.

_____ 11. I am able to focus on the present moment.

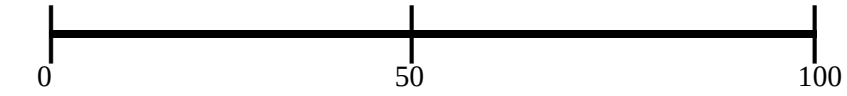
_____ 12. I am able to pay close attention to one thing for a long period of time.

SID _____
Date _____

Appendix L: Pain Intensity and Unpleasantness Scales

1) How much pain do you feel right now?

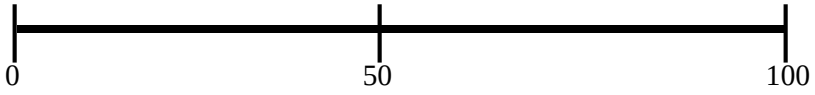
Make a mark on the line below to show your pain level



No Pain | Moderate Pain | Worst Possible Pain

2) How much unpleasantness do you feel right now?

Make a mark on the line below to show your level of unpleasantness.



None | Moderate | Most Possible

SID _____
Date _____

Appendix M: Short-Form McGill Pain Questionnaire Items

Write the number of the most appropriate word in the space beside the questions below

1	2	3	4	5
Mild	Discomforting	Distressing	Horrible	Excruciating

To answer each question below, write the number of the most appropriate word in the space beside the question.

1. Which word describes your pain right now? _____

2. Which word describes it at its worst? _____

3. Which word describes it when it is at its least? _____

SID _____
Date _____

Appendix N: Profile of Mood States-Revised

Instructions: Below is a list of common human emotions. For each emotion, circle the response that best indicates how accurately that emotion describes you. Describe yourself as you see yourself at the present time, not as you wish to be in the future. Describe yourself as you are **Generally or Typically,** as compared with other persons you know of the same sex and roughly the same age.

0= NOT AT ALL ACCURATE
1= A LITTLE ACCURATE
2= MODERATELY ACCURATE
3= QUITE A BIT ACCURATE
4= EXTREMELY ACCURATE

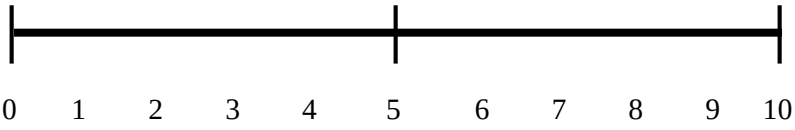
1. sluggish	0	1	2	3	4	17. sad	0	1	2	3	4
2. happy	0	1	2	3	4	18. frightened	0	1	2	3	4
3. hostile	0	1	2	3	4	19. sleepy	0	1	2	3	4
4. at ease	0	1	2	3	4	20. calm	0	1	2	3	4
5. unhappy	0	1	2	3	4	21. afraid	0	1	2	3	4
6. full of pep	0	1	2	3	4	22. angry	0	1	2	3	4
7. fearful	0	1	2	3	4	23. lively	0	1	2	3	4
8. tired	0	1	2	3	4	24. tense	0	1	2	3	4
9. on edge	0	1	2	3	4	25. cheerful	0	1	2	3	4
10. energetic	0	1	2	3	4	26. fatigued	0	1	2	3	4
11. depressed	0	1	2	3	4	27. relaxed	0	1	2	3	4
12. nervous	0	1	2	3	4	28. resentful	0	1	2	3	4
13. pleased	0	1	2	3	4	29. dull	0	1	2	3	4
14. quiet	0	1	2	3	4	30. serene	0	1	2	3	4
15. bored	0	1	2	3	4	31. alert	0	1	2	3	4
16. aroused	0	1	2	3	4	32. surprised	0	1	2	3	4

SID _____
Date _____

Appendix O: Relaxation Visual Analogue Scale

1) How relaxed do you feel right now?

Make a mark on the line below to show your level of relaxation.



0 1 2 3 4 5 6 7 8 9 10

Not Relaxed At All — Moderately Relaxed — Completely Relaxed