An Exploration of the Changes in Signs and Symptoms Associated with Sleep Bruxism that Follow Osteopathic Manual Therapy: A Pilot Study

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A research thesis submitted in partial fulfilment of the requirements for the degree of Master of Osteopathy.

Unitec Institute of Technology
Declaration

Name of candidate: Lorelei Messersmith

This Thesis/Dissertation/Research Project entitled “An Exploration of the Changes in Signs and Symptoms Associated with Sleep Bruxism that Follow Osteopathic Manual Therapy: A Pilot Study” is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy.

Candidate’s declaration
I confirm that:

• This Thesis/Dissertation/Research Project represents my own work;
• Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: 2011-1146

Candidate Signature: …………………………………………………… Date: 13/4/2015

Student number: 1301481
Abstract

**Background:** Sleep bruxism occurs in 8 to 20% of the population and can result in jaw and head pain, tooth destruction and diminished quality of sleep for bruxers and their partners.

**Objective:** To explore whether osteopathic manual therapy (OMT) could alter bruxing activity, pain and psychological wellbeing in self-diagnosed sleep bruxers.

**Methods:** Six sleep-bruxing participants received four weekly osteopathic treatments. Electromyographic activity of bruxing and head and jaw pain, via the Numeric Rating Scale (NRS), were monitored for a pre-treatment period, after each treatment and for a follow-up period. The 21 Question Depression Anxiety and Stress Scale (DASS21) was assessed at study onset and conclusion.

**Results:** Six participants showed clinically significant decreases in NRS score from pre-treatment to immediately following the third treatment. Group changes approached statistical significance from pre- to post-treatment despite a reduced sample size. (Effect Size = -0.71; \( P = 0.06 \)). Five participants experienced a reduction of one or more DASS21 severity ratings for stress by the end of the study. Bruxing activity showed a trend towards improvement in two participants and increased in one participant.

**Conclusions:** These results suggest that osteopathic treatment may help sleep bruxers by reducing their perceived pain and stress levels. The results are inconclusive as to the effectiveness of OMT for reducing nocturnal bruxing activity. Additional controlled studies with more detailed inclusion criteria and higher numbers of participants are suggested.

**Key Words:** osteopathy, nocturnal bruxism, manual therapy, jaw pain, complementary and alternative medicine
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<td>Osteopathic Manual Therapy</td>
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<td>NRS</td>
<td>Numeric Rating Scale</td>
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<td>DASS21</td>
<td>21 Question Depression Anxiety Stress Scales</td>
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<td>TMJ</td>
<td>Temporomandibular Joint</td>
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<td>TMD</td>
<td>Temporomandibular Disorder</td>
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<td>AASM</td>
<td>American Academy of Sleep Medicine</td>
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<td>ICSD</td>
<td>International Classification of Sleep Disorders</td>
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<td>PSG</td>
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<td>Sympathetic Nervous System</td>
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<td>PSNS</td>
<td>Parasympathetic Nervous System</td>
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<td>NREM</td>
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<td>REM</td>
<td>Rapid Eye Movement</td>
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<td>Cyclic Alternating Pattern</td>
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<td>CgA</td>
<td>Chromogranin A</td>
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<td>MAD</td>
<td>Mandibular Advancement Device</td>
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<td>ES</td>
<td>Effect Size</td>
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<td>RCT</td>
<td>Randomised Controlled Trials</td>
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<td>GABA</td>
<td>Gamma-aminobutyric acid</td>
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<td>ROM</td>
<td>Range of Motion</td>
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<tr>
<td>IMT</td>
<td>Intraoral myofascial therapy</td>
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<tr>
<td>IMTESC</td>
<td>Intraoral myofascial therapy plus self-care</td>
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<td>VAS</td>
<td>Visual Analogue Scale</td>
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Section One

Literature Review
Literature Review

Introduction
Sleep bruxism (SB) can be broadly described as the dysfunctional clenching or grinding of the teeth while asleep (Dorland, 2003). Patients often seek help from health professionals following the unpleasant grinding noise that can disturb the sleep of the patient as well as their sleeping partner and/or family (AASM, 2001). In addition to sleep disturbances, this condition can produce tremendous pressure on the teeth, which can lead to fractured and worn teeth, as well as tooth hypersensitivity, pain within the temporomandibular joint (TMJ) and headaches (G. J. Lavigne, 2008). Traditional treatments for the condition include occlusal splints, behavioural or stress therapy and medications including muscle relaxants or Botox (Huynh, Manzini, Rompre, & Lavigne, 2007). Occlusal splints, the most common treatment, have only been shown to lessen the symptoms of grinding noise, jaw pain and headaches, but do not decrease the grinding and clenching activity (Gomes, El Hage, Amaral, Politti, & Biasotto-Gonzalez, 2014; Huynh et al., 2007; Macedo, Silva, Machado, Saconato, & Prado, 2007).

The studies devoted to sleep bruxism explore most aspects of the condition, from the prevalence among various populations to the physiological and psychological associations, to the efficacy of various traditional treatments (Kalamir, Pollard, Vitiello, & Bonello, 2007b; G. J. Lavigne, 2008). However, there are still many questions regarding the aetiology and treatment of this condition that remain unanswered.

Osteopathy is a form of manual therapy that takes into account the self-regulatory mechanisms within the body and the tenet that the “body is a unit and the person is a unit of body, mind and spirit” (Ward, 2003, p. 15). Osteopathic philosophy purports to address dysfunction within the body by evaluation of the whole body and the acknowledgement that dysfunction in one area of the body can lead to pain in another area that may seem unrelated (Parsons & Marcer, 2006). There is limited published research regarding the efficacy of osteopathic treatment on temporomandibular disorders and pain and none investigating the efficacy of this modality on reducing the masticatory activity of sleep bruxism. However, published research suggests that manual therapy is as effective as drug
or splint therapy at alleviating the pain that these conditions can cause (Cuccia, Caradonna, Annunziata, & Caradonna, 2009).

This literature review is the first part of a three section thesis. It aims to provide the reader with an explanation of the phenomenon of sleep bruxism and an introduction to the complexity surrounding the research and treatment of the condition. It provides an overview of research on sleep bruxism and highlights important unanswered questions on the subject. The final part of the review explores how manual therapy and osteopathy could help patients who suffer from the effects of sleep bruxism.

Section 2 of this thesis follows this literature review and contains a manuscript formatted for submission to the *International Journal of Osteopathic Medicine*. Section 3 comprises appendices and all other pertinent information in support of this thesis.

**What is Bruxism?**

**Definition**

There have been many proposed definitions of sleep bruxism (AASM, 2001; G. J. Lavigne, 2008; Lobbezoo et al., 2013). The International Classification of Sleep Disorders, a document produced by the American Academy of Sleep Medicine (AASM), in association with European Sleep Research Society, Japanese Society of Sleep Research and the Latin American Sleep Society, defines sleep bruxism as an oral activity “characterised by grinding and clenching of the teeth during sleep, usually associated with sleep arousals” (AASM, 2001, p. 182). This definition is widely used by researchers within the field (G. J. Lavigne, 2008).

More recently, a group of international bruxism experts collaborated to develop a more relevant definition of bruxism. They defined bruxism as:

> a repetitive jaw-muscle activity characterised by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as wakeful bruxism) (Lobbezoo et al., 2013, p. 3).
This definition acknowledges both circadian manifestations of bruxism, removes negative connotations to the term and acknowledges that the edentulous population may still activate their muscles of mastication repetitively, despite their lack of teeth. They have recommended that this definition be used within clinical and research settings.

**Wakeful vs. Sleep Bruxism**

While there are clear common characteristics within various descriptions of bruxism, there is a lack of universally accepted definitions for the diurnal (wakeful) and nocturnal (sleep) forms of bruxism. (Kalamir et al., 2007b). Wakeful bruxism is characterised by an awareness of clenching type activities while conscious and is understood to be a related tic or habit that occurs as a reaction to stress or anxiety (Huynh et al., 2007). The condition is estimated to affect 20% of the general population (G. J. Lavigne, 2008). There is little known about the aetiology and pathophysiology of wakeful bruxism, as there is no gold standard for the monitoring of the phenomenon (G. J. Lavigne, 2008). SB, in contrast to the wakeful form, occurs subconsciously while asleep and includes grinding in addition to clenching (G. J. Lavigne, 2008; Manfredini, 2004). SB prevalence is reported to be similar to wakeful bruxism, however, studies have shown discrepancies between the prevalence of self-reported sleep bruxers and sleep bruxers diagnosed via polysomnographic study (Maluly et al., 2013; Raphael, 2012). Associations with stress and anxiety for SB are less clear cut than for wakeful bruxism (Abekura et al., 2011; Castelo, Barbosa, Pereira, Fonseca, & Gavião; Giraki et al., 2010) and a study has shown an association with the ‘type A’ personality type (Abe et al., 2012; Kampe, Edman, Bader, Tagdae, & Karlsson, 1997). The condition is associated with brain and cardiac activations called microarousals that occur during sleep (Huynh, Kato, et al., 2006; Kato, Rompre, Montplaisir, Sessle, & Lavigne, 2001; G.J. Lavigne, Kato, Kolta, & Sessle, 2003), hence specialist clinical and research assessment of SB entails measurement of biological variables during the sleep period, termed polysomnography (PSG). While it is useful to identify the different forms of bruxism, this review will focus specifically on SB.

**Clinical Presentation of Sleep Bruxism**

Patients with SB often present to professionals within the dentistry, orofacial pain and sleep medicine communities. They also present to manual therapists seeking help for headaches
and TMJ dysfunction and pain. Patients are often made aware of the disorder from their bed partner. They may also present with evidence of tooth destruction, temporomandibular disorders and pain as well as tension headaches (AASM, 2001; G. J. Lavigne, 2008; Shetty, Pitti, Satish Babu, Surendra Kumar, & Deepthi, 2010). If the patient lives alone, a dentist may be the first to make them aware of the condition as they would note the abnormal facet wear on the patient’s teeth.

**Prevalence of Sleep Bruxism**

SB is more prevalent in the 18–44 year-old age bracket and is not gender-biased. The prevalence has been shown to decrease significantly with age (G. J. Lavigne, 2008; Ohayon, Li, & Guilleminault, 2001) and is reported to be higher in specific populations such as military pilots and police officers (Carvalho, Cury, & Garcia, 2008; Lurie et al., 2007).

Ohayon, et al. (2001) conducted a large scale study in Europe that focused on the prevalence and risk factors associated with sleep bruxism. The study involved a sample of 13,057 subjects from the general population in three countries and was conducted by telephone interview. The study found that 4.2% of the total sample (n = 568) met the diagnostic criteria of sleep bruxism as per the ICSD. This study showed that SB was comparable between men and women (M = 4.1%, F = 4.6%) and that the prevalence of the SB diagnosis was highest in subjects between the ages of 19 and 44 years (5.8%). This number decreased in subjects 65 years and older (1.1%). A second group of 491 (3.8%) subjects identified themselves as sleep grinders but did not fit the diagnostic criteria for SB as per the study.

Limitations addressed in the study include a possible underestimate of bruxers resulting from under-reporting of SB in individuals living alone, due to lack of a witness to the condition. This may disproportionately affect estimates of prevalence in elderly people, a bias potentially compounded by the lower proportion of elderly bruxers meeting the criterion of abnormal tooth wear due to a higher proportion of individuals without teeth (Lobbezoo et al., 2013). Finally, it was noted that the diagnosis of SB was self reported as per the ICSD criteria and did not include an examination or polysomnographic findings to clinically confirm the diagnosis.
Maluly, et al., (2013) sampled 1042 individuals within the general population of Brazil in order to determine the prevalence of SB within a general population. Participants answered questionnaires and findings were confirmed via PSG examination. The prevalence of SB, indicated by questionnaires and confirmed by PSG, was 5.5%. Questionnaires alone (self-diagnosis) indicated a prevalence of 12.5% and PSG examination without the questionnaire indicated a prevalence of 7.4%. These numbers fall within the previously reported ranges for prevalence of the condition within the general population. While this study utilised PSG to confirm the presence of SB, it should be noted that SB episode severity can vary from night to night and that PSG only allows for a very short observational period due to resource limitations of this method of examination.

While it is assumed that patterns of SB in New Zealand are similar to other Western countries, there is limited research in the New Zealand context. A small study investigated the incidence of sleep disorders in Auckland high school students (n=1388) in which bruxism considered one of three parasomnias measured within the Auckland high school student population. The study found that 37.2% (n=517) of the students surveyed experienced sleep disorders lasting over one month or more. Of that percentage, 9.3% (n=48) experienced parasomnias, which include teeth grinding, sleep walking and sleep-talking. (Fernando, Samaranayake, Blank, Roberts, & Arroll, 2013). Tooth grinding/bruxism was determined according to self-diagnosis criteria set by ICSD; however, results were pooled with other parasomnias, providing little insight into the prevalence of bruxism within the population studied.

**Diagnosis, Evaluation and Measurement of Sleep Bruxism**

The ICSD document outlines the generally accepted diagnostic criteria for sleep bruxism. The criteria are as follows:

The patient reports or is aware of tooth-grinding sounds or tooth-clenching during sleep.

One or more of the following:

- Abnormal wear of the teeth
- Sounds associated with the bruxism
Jaw muscle discomfort

The diagnosis is made following exclusion of other medical or mental disorders (for example, sleep-related epilepsy, accounts for the abnormal movements during sleep). It is noted that other sleep disorders (for example, obstructive sleep apnoea syndrome) can present concurrently (AASM, 2001).

Lobbezoo, et al., (2013) proposed, in conjunction with other bruxism experts, to suggest a new diagnostic grading system for bruxism. The group proposed to diagnose “possible, probable and definite sleep or awake bruxism” (Lobbezoo et al., 2013). The grading were based on the evidence presented to confirm the probability of bruxism and the system has been suggested for use in clinical as well as research settings. The grading is as follows:

“Possible” sleep or wakeful bruxism - based on self-report, by means of questionnaires and/or the case history part of a clinical examination.

“Probable” sleep or wakeful bruxism - based on self-report plus the inspection part of a clinical examination.

“Definite” sleep bruxism - based on self-report, a clinical examination and a polysomnographic recording, preferably along with audio/video recordings.

The PSG cut-offs have been suggested as per the work presented by Lavigne, et al. (1996).

Polysomnography – the Gold Standard for Monitoring Sleep Disorders

Polysomnography employs a variety of monitoring methods, including electroencephalographic (EEG) for the monitoring of brain waves, electromyography (EMG) to measure muscle activity, electro-oculographic to measure eye movement as well as video and audio recordings of the subject. Respiratory and cardiac events are also recorded. The EEG is used to evaluate patients’ sleep cycles by measuring certain markers that allow for the distinction between the various stages of sleep, as well as the quality of sleep. Many studies on SB use PSG to provide insight into the various associations between brainwave activity, autonomic neural activity and muscle activation. (Greenfield, Geyer, & Carney, 2010; Guilleminault, 2005)
Audiovisual monitoring of the subject is an important aspect of PSG with regards to SB. Various non-bruxing-related oromotor activities such as swallowing, snoring and yawning account for 30% of bruxers’ facial movements during sleep. PSG allows for this non-bruxing activity to be differentiated from SB episodes (Guilleminault, 2005; G. J. Lavigne, 2008).

There are some limitations imposed by the use of PSG diagnostic criteria. These include the requirement of expensive and limited diagnostic laboratory services. PSG also imposes an artificial sleeping environment that could detrimentally affect participants’ sleep (Kalamir et al., 2007b; G. J. Lavigne, 2008; Shetty et al., 2010), although a recent study shows that this effect is negligible in moderate to severe sleep bruxers (Hasegawa et al., 2013). Most research studies utilising PSG collected data include one night in which to acclimate the subjects to the new sleep environment (Huynh, Kato, et al., 2006; Kato et al., 2001; Khoury et al., 2008; Nashed et al., 2012).

**PSG Diagnostic Criteria**

The literature suggests that there is a failure to standardise diagnostic parameters such as frequency, duration and intensity of episodes for SB when using PSG (Kalamir et al., 2007b). However, Lavigne, et al., (1996) conducted a PSG study of 18 bruxers and 18 non-bruxers in order to establish PSG diagnostic criteria and proposed the following: (1) more than 4 bruxism episodes per hour, (2) more than 6 bruxism bursts per episode and/or 25 bruxism bursts per hour of sleep and (3) at least 2 episodes with grinding sounds. It was indicated that these criteria needed to be tested over time and within a larger research population (G. J. Lavigne et al., 1996).

**Other Devices**

Ambulatory electromyography (EMG) devices such as the Bitestrip™ and Sleepguard™ can be used to measure the activity of the masseter or temporalis muscles during the sleep period. These devices allow for the monitoring of clenching and grinding activity in the home sleep environment. They are less expensive than PSG and as a result, can provide the user information over a longer period of time. This information is ideally suited for the person working towards finding relief from their bruxism but can be useful for research purposes as well (Shochat et al., 2007; Weinstein, 2013).
Despite their practicality, ambulatory EMG devices do have many limitations when used for research purposes. Orofacial movements such as swallowing are not differentiated from the clenching or grinding movements. This may potentially lead to a false diagnosis with regards to severity of the patient’s bruxism. These devices are also prone to user error if used without proper training in placement and programming. (Shochat et al., 2007; Weinstein, 2013).

**Aetiology of Sleep Bruxism**

One of the main obstacles in understanding sleep bruxism is the fact that the aetiology of the condition is not understood. However, it is thought to be multi-factorial (Lobbezoo, 2012). Earlier SB research pointed towards occlusal causes; these have since been disproven. (Behr et al., 2012; Lobbezoo, 2012; Manfredini, 2004; Ommerborn et al.). Current studies focus on more centrally-mediated causes for sleep bruxism. (Behr et al., 2012; Lobbezoo, 2001). An extensive review by Lavigne, et al., (2008) emphasised the need to integrate research with clinical knowledge in order to provide more clarity with regards to the aetiology of SB. The following diagram from the Lavigne review helps to explain the various hypotheses that have been investigated and highlights the ones to investigate further.

![Figure 1](image-url)  
*Figure 1. Evolution of the aetiology and pathophysiology of sleep bruxism (circles = older theories; arrows = new avenues). GABA, gamma-aminobutyric acid. (G. J. Lavigne, 2008, p. 482)*
Physiology of Sleep, Stress and Sleep Bruxism
The first step in understanding the potential aetiology of SB and how to treat it is to understand that pathology and physiology are inter-related. Numerous studies show an association to autonomic arousal and the stress response (Huynh, Kato, et al., 2006; Khoury et al., 2008; Gilles J. Lavigne et al., 2007; G.J. Lavigne et al., 2003; Nashed et al., 2012). This section is intended to give the reader an insight into the complexity surrounding the potential aetiology of SB.

Stress
Stress is generally defined as the response to a real or perceived threat to homeostasis (S. M. Smith & W. W. Vale, 2006). People with SB have been shown to have elevated sympathetic tone and tend to identify themselves as being stressed with life situations (Giraki et al., 2010; Ohayon et al., 2001; Winocur, 2010). The physiological stress response involves a complex interaction of the nervous, endocrine and immune systems. Three of the main components of the stress response are the sympathetic nervous system, the hypothalamic-pituitary-adrenal (HPA) axis and the parasympathetic nervous system (S. M. Smith & W. W. Vale, 2006). The following is a brief description of these systems in relation to SB.

Sympathetic Nervous System
The sympathetic nervous system (SNS) is often referred to as the “fight or flight” system. The SNS can be stimulated to produce a generalised sympathetic activation, or stress response, in times of real or perceived threat to an organism’s homeostasis (Sean M. Smith & Wylie W. Vale, 2006). The stress response causes the adrenal medulla to release high levels of noradrenalin and adrenaline. These hormones work to increase arterial blood pressure, muscle blood perfusion, breathing and heart rate while increasing energy supplies (Guyton & Hall, 2006).

This is important to note because sleep bruxers have signs of increased sympathetic activation (Huynh, Kato, et al., 2006), either preceding or during episodes of sleep bruxism (Huynh, Lavigne, Lanfranchi, Montplaisir, & de Champlain, 2006). These include raised blood pressure (Nashed et al., 2012), raised levels of urinary stress hormones (Seraidarian,
Seraidarian, das Neves Cavalcanti, Marchini, & Claro Neves, 2009) and increased breathing amplitude (Khoury et al., 2008). A more detailed discussion of these studies will follow.

**HPA Axis**
Stress also causes the release of cortisol from the adrenal cortex via the HPA axis. The HPA axis will continue to release cortisol in response to the body reacting to the stressor, maintaining the stress response. Salivary cortisol levels are routinely used as a biomarker to indicate quantitative stress levels (Benson, 1983).

**Parasympathetic Nervous System**
The parasympathetic (PSNS) division of the ANS, the “rest and digest” division deals mainly with maintaining gastrointestinal activity such as salivary and gastric secretion. (Guyton & Hall, 2006). The PSNS balances the SNS and plays a role in the relaxation response, an aspect of treatment that manual therapy is reported to engage with (Benson, 1983; King, Janig, & Patterson, 2011; S. M. Smith & W. W. Vale, 2006).

**Sleep and the Sleep Cycle**
Sleep is defined as a “state of unconsciousness from which one can be aroused by sensory stimulation” (Guyton & Hall, 2006, p.739). The human adult sleep cycle normally consists of 60 – 100 minutes per cycle, with 3-6 cycles per sleep episode (G.J. Lavigne et al., 2003). Each cycle alternates through four stages of non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. (Huynh, Kato, et al., 2006; G.J. Lavigne et al., 2003). The following is an explanation of the SB-relevant PSG markers and sleep activities followed by a brief description of each stage of sleep in the human adult as monitored via PSG.

**SB-Relevant Activity**
These activities are common observations within PSG studies:

**Rhythmic masticatory muscle activity** (RMMA) is a normal sleep activity that approximately 60% of non-bruxing people will show. It consists of repetitive masticatory muscle contractions (Kato et al., 2001; G. J. Lavigne, Rompre, et al., 2001; Macaluso et al., 1998).

**Microarousals** are noted as an abrupt change in the frequency of cortical EEG that is occasionally associated with motor activity, usually of the jaw and legs. It is normal activity in healthy non-bruxing young adults. In sleep bruxers, up to 80% of microarousals are
associated with cardiac sympathetic activation as well as increased muscle tone and activity, including masticatory activity and occur with a higher magnitude than in non-bruxers (Kato et al., 2001; G.J. Lavigne et al., 2003; Macaluso et al., 1998).

**Cyclic Alternating pattern (CAP)** is identified in PSG by repetitive clusters of intense EEG, muscle and autonomic activation and occurs in NREM sleep. Microarousals and RMMA are associated with the CAP, therefore most SB episodes occur in clusters in relation to the CAP (Carra et al., 2011; Huynh, Kato, et al., 2006).

**Stages of the Sleep Cycle**

**NREM 1** is transitional and lasts from 1–7 minutes. During this phase autonomic cardiac sympathetic activity diminishes (Kato et al., 2001; G.J. Lavigne et al., 2003). **NREM 2** lasts 10–25 minutes in the first sleep cycle then increases as the sleep episode progresses to comprise 45–55% of the total episode (G.J. Lavigne et al., 2003). CAP episodes associated with microarousals occur during this stage, particularly during transition from deep (NREM3 and 4) to REM sleep. Sixty to eighty percent of SB episodes happen during this stage (G. J. Lavigne, Rompre, et al., 2001; Macaluso et al., 1998). **NREM 3** and **NREM 4** consist of 13–23% of total sleep and are considered deep sleep. These stages fluctuate and parasympathetic-vagal cardiac activity dominates. Most deep sleep happens in the first third of the sleep episode and gradually diminishes over the episode. **REM** is referred to as “active” or “paradoxical sleep”. The REM stage lasts 1–5 minutes initially, gradually increasing with each cycle towards wakening. REM EEG activity is noted as being similar, if not more active, than in an individual who is awake. Emotional centres of the brain are active and dreaming occurs in this stage. Muscle atonia of the jaw and limbs occurring in parallel with phasic eye movements is characteristic of this stage (G. J. Lavigne, Rompre, et al., 2001). Less than 10% of SB episodes occur during REM sleep (G.J. Lavigne et al., 2003).

There are complex neural and neurochemical interactions with each stage of sleep; however, a detailed explanation of the neurochemical regulation of sleep is beyond the scope of this literature review¹.

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¹ Information can be obtained from Lavigne, et al.’s, 2003 review “Neurobiological Mechanisms Involved in Sleep Bruxism”.

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Sleeping as a Sleep Bruxer
Most sleep bruxers have normal sleep macrostructure, that is, they cycle through the NREM and REM stages of sleep during a “normal” sleep episode. (Huynh, Kato, et al., 2006; G.J. Lavigne et al., 2003; G. J. Lavigne et al., 2002; Macaluso et al., 1998). Moreover, a study conducted in 2002 demonstrated that people with SB actually show via EEG the hallmarks of good sleep; that is, their EEG reading showed fewer indicators of disrupted sleep than non-SB controls (G. J. Lavigne et al., 2002). What makes sleep bruxers different from non-bruxers is the magnitude of their “normal” behaviours; that is, they exhibit activity and endogenous activations that non-SB subjects experience, however, their experience of these behaviours is of a greater frequency, duration and strength/amplitude (Kato et al., 2001; G.J. Lavigne et al., 2003). For example, Lavigne, et al., noted that up to 60% of normal subjects experience RMMA at a frequency of about 1.8 episodes per hour of sleep. Sleep bruxers, in comparison, were shown to experience RMMA at a frequency of 5.4 to 5.8 episodes per hour of sleep. It was also shown in the same study that sleep bruxers use higher muscle amplitude than non-bruxers (2001).

Associations with SB
Since there is no confirmed aetiology for SB, it is necessary to investigate the associations to the condition. Ohayon, et al., found in their large scale study involving 13, 057 subjects from the UK, Italy and Germany that caffeine consumption (OR, 1.4) smoking (OR 1.3), alcohol consumption (OR 1.8), obstructive sleep apnoea syndrome (OR, 1.8), highly stressful life (OR, 1.3) and anxiety (OR, 1.3) were all significantly associated with SB. This study was conducted via phone interviews with self diagnosis for SB (2001).

The association between SB and sleep apnoea was confirmed by Saito, et al, (2013). Their study revealed that in 10 sleep bruxing males, 80% of SB episodes happen within a 5 minute window of a sleep apnoea episode, suggesting that sleep apnoea is associated with a secondary form of SB. Wincour, et al., (2010) investigated the associations between perceived stress, motivation for control, dental anxiety and gagging in self-reported bruxers, both wakeful and sleep. The Perceived Stress Scale, Desirability of Control Scale, Dental Anxiety Scale, Gagging Assessment Scale and self diagnosis of bruxism as per the
AASM(2001) were distributed to 600 people within a general adult population with 402 respondents. The study found that 104 (25.9%) of participants reported being sleep bruxers, however; only 55 participants actually were diagnosed as sleep bruxers according to the study criteria. Awake bruxism was reported in 39 (70.9%) of the diagnosed sleep-bruxing participants. The sleep bruxers showed significantly higher levels of dental anxiety, gagging and emotional stress than the non-bruxers. It was concluded that wakeful bruxism greatly increases the odds for SB and vice versa. With those observations, the question of potential similarities in aetiology was discussed. Previous studies show an association of psychological factors to wakeful bruxism (Manfredini & Lobbezoo, 2009). That role is questionable in sleep bruxism. Considering the high percentage of sleep bruxers who also brux during the day, it was proposed that perhaps psychological factors could play a role in the pathogenesis of SB and that more investigations were warranted (Winocur, 2010).

**Psychological Stress**

The evidence of physiological stress in sleep bruxers has been well documented with PSG studies (Huynh, Lavigne, et al., 2006; Khoury et al., 2008; Gilles J. Lavigne et al., 2007; G.J. Lavigne et al., 2003; Nashed et al., 2012). There are conflicting reports from the PSG studies that investigate the physiological evidence of psychological stress. Many studies exploring the connection between psychological factors and bruxism don’t differentiate between sleep or wakeful bruxism (Manfredini, 2004; Schneider et al., 2007). They are also usually based on self-diagnosis, with no EMG or PSG confirmation of bruxing activity. This makes it difficult to establish a true picture of the association of psychological stress with SB. Previously mentioned studies show a strong association of psychological factors, including stress, to wakeful bruxism (Manfredini & Lobbezoo, 2009; Winocur, 2010). These studies also show a very strong association of SB to wakeful bruxism and vice versa. One may argue that, in fact, a strong indicator of SB is wakeful bruxism. The following section explores some of the studies that investigate the connection of SB to psychological factors such as stress.

Two studies have revealed high populations of bruxers in high-stress environments. Brazilian police officers were studied to investigate the prevalence of bruxism and emotional stress. Carvalho, et al.,(2008) found that within the 394 police officers studied, 50.25% (n=198) showed dental evidence of bruxism. Of those, only 64.6% were aware of
the presence of the condition. Significant association (P=.0004) was found between the presence of emotional stress and bruxism among the police officers. Stress was measured via the Stress Symptoms Inventory (SSI). Almost half (45.7%) of the studied officers were considered emotionally stressed and among the stressed officers, 48.3% reported grinding while either asleep or awake.

Lurie, et al.,(2007) investigated the prevalence of bruxers within a high-stress, military aviation environment. Fifty-seven male Israeli Air Force officers (17 jet pilots, 18 helicopter pilots and 22 non-pilot officers) were examined for dental evidence of bruxism. A battery of psychological questionnaires assessing magnitude of workplace stress and coping style were also administered to the group. The study revealed that 69% of the overall group of pilots had evidence of bruxism, while 27% of non-pilots displayed evidence of bruxing behaviour. Pilots and non-pilots had similar stress levels (3.84 and 3.59 respectively). Bruxing pilots had more emotionally-oriented and denial-based coping strategies, considered less effective than the non-pilots’ more task-driven methods of coping. It was concluded that the military environment, particularly the military aviation setting was a pathogenic factor associated with psychological stress and bruxism (Lurie et al., 2007). The limitations to this study include the lack of clarification between the sleep and wakeful bruxers and the lack of EMG, PSG, or self diagnosis of bruxing activity. An investigation of physiological evidence of stress would have been an interesting dimension to this study.

The study by Giraki, et al.,(2010) emphasises the observation that people with high SB activity seem to feel more stressed within their lives. This SB specific study used a Bruxcore™ occlusal plate for 5 consecutive nights to confirm and quantify SB activity. Sixty-nine subjects including 48 sleep bruxers completed 3 German questionnaires assessing various stress parameters and stress-coping strategies. Significant correlations were found for ‘daily problems' (p<0.01), ‘trouble at work’, ‘fatigue’, ‘physical problems’ and coping strategy ‘escape’ (all p<0.05). The observation was made that sleep bruxers feel more stress at work and within daily life and tend to have negative stress coping strategies, previously observed by Schneider, et al. (2007).

An association between SB and stress sensitivity was found and physiologically quantified by Abekura, et al.(2011). Seventy-six subjects, (54 non-bruxers and 22 sleep bruxers) were
recruited and subjected to a stress task. SB was self-diagnosed according to the AASM criteria. Salivary chromogranin A (CgA) levels were sampled prior to and after the stress test. Stress levels were subjectively measured before and immediately after the test using a ten-division visual analogue scale (VAS). The results demonstrated that the mean salivary CgA levels of the bruxism group were significantly increased after the stress task as compared to before. There was no significant increase of CgA before or after the stress task in the non-bruxing group. VAS scores for both groups were increased after the task. This study highlights the connection between an input of psychological stress and physiological output of stress in sleep bruxers. Although, these results differ from some published studies (Castelo et al., 2012), they are consistent with those of Seraidarian, et al., (2009) who found that sleep bruxers have higher levels of urinary catecholamines than non-bruxers. The interpretation of this study is somewhat limited by the lack of PSG observation and limited numbers or participants.

**Physiological Stress and SB**

The physiological mechanisms of activation prior to and during SB episodes have been observed and documented. Numerous studies have demonstrated an increase in sympathetic activity or tone prior to the onset of SB episodes (Huynh, Kato, et al., 2006; Huynh, Lavigne, et al., 2006; Nashed et al., 2012; Seraidarian et al., 2009). The following studies document the sympathetic nature of SB arousal. It should be noted that most of these studies have been conducted within the PSG sleep lab environment, utilising the gold standard for SB diagnosis and observation. This contributes to the internal validity of the measurements; however, most have limited participant numbers and limited observational time due to the invasive nature of PSG and the expense involved with the examination method.

**Autonomic Activity Associated with Microarousals**

Kato, et al., (2001) demonstrated that SB episodes are preceded by an increase in sympathetic activity. In this observational study, 10 sleep bruxing subjects and 10 non-sleep bruxing subjects were monitored via PSG for 2 nights in a sleep laboratory (1 night for habituation, 1 for data collection). In particular, heart rate, EEG analysis and motor activity were noted along with RMMA/SB episodes. It was shown that prior to any grinding activity, there was an increase in EEG activity in sleep bruxers four seconds prior to the episode. This
was accompanied by an increase in heart rate for the ten beats prior to the grinding. Normal subjects exhibited no significant increase in EEG activity prior to normal jaw movements during sleep and heart rate increases only happened after the onset of jaw activity. The study also observed that both groups displayed similar incidences of microarousals (an abrupt change in the frequency of cortical EEG that can be associated with motor activity (AASM, 2001)), however; SB subjects showed 8 times more RMMA episodes per hour of sleep than non-bruxers, particularly during sleep stage NREM 2. They concluded that increased brain and autonomic nervous system activity precede RMMA. Limitations to this study are the low number of subjects (Kato et al., 2001).

Huynh, et al., corroborated these findings in their 2006 study. Observations were made via PSG between 20 moderate to high sleep bruxers, 20 low sleep bruxers and 20 non-bruxing control participants. In sleep bruxers, an increase in sympathetic EEG activity occurred 8 minutes prior to the SB onset and persisted for approximately 28 minutes following the episode. A decrease in parasympathetic EEG activity started at the same time as the SB episode and continued for approximately 28 minutes after the episode. It was concluded that a clear increase in sympathetic activity and decrease in parasympathetic activity precedes SB onset in moderate to severe sleep bruxers (Huynh, Kato, et al., 2006).

This observation was further tested by Huynh, et al.,(2006). A PSG study of 25 subjects was undertaken, observing the effects of propranolol (a sympatholytic non-selective beta blocker) and clonidine (an alpha-adrenergic antagonist), on sleep bruxers. Both medications work to decrease sympathetic activity via different pathways. Results showed that propranolol didn’t have any effect on SB activity, but clonidine decreased bruxing activity by disrupting the cascade of autonomic to motor activations from 1 minute preceding a SB episode. This resulted in a 61% decrease in SB activity. This helps to support the theory that sympathetic autonomic activity has a role in SB (Huynh, Lavigne, et al., 2006).

**Catecholamine Levels in Sleep Bruxers**

Sleep bruxers have higher levels of urinary catecholamines (P=0.00). In a study by Seraidarian, et al., (2009) the entire urine volume excreted over a 24 hour period of 20 self-diagnosed sleep bruxing participants (with dental examination to confirm presence of wear of posterior occlusal teeth) and 20 non-bruxing participants was collected, analyzed and
compared via liquid chromatography for the presence of adrenaline, noradrenaline and dopamine. Participants were instructed to avoid activity that could increase catecholamine release over the 7 days prior to urine collection. Higher 24-hour production of catecholamines in subjects with SB were shown than in control subjects (adrenalin=318%, noradrenaline=175% and dopamine=238%). This information suggests that catecholamines may play a role in the aetiology and maintenance of SB. Limitations of this study include limited number of participants and lack of PSG diagnosis of SB.

In contrast to the previous study, Castelo, et al., (2012) found that children with SB were more likely to present low levels of salivary cortisol upon waking. Castelo, et al., sampled the awakening saliva for cortisol levels of a group of 100 healthy children (mean 7.23+ 0.64 years). Twenty-seven sleep-bruxing participants were diagnosed via family reporting of grinding noises at least 3 times a week as well and dental examination, leaving 77 non-bruxers as controls. The weight, height, heart rate, BMI and parafunctional habits (including enuresis and nail biting) for all participants were also recorded upon examination. Two samples of saliva were obtained from each participant; one just after waking while lying in bed and another 30 minutes after waking (fasting). The results showed that children with SB presented with low concentrations of salivary cortisol. There was also no significant correlation between cortisol levels and heart rate. Some concerns highlighted in the study include the lack of understanding surrounding the awakening cortisol response and its role in stress. SB was diagnosed only on family reporting; there was no PSG or EMG confirmation of bruxing activity.

**Breathing Amplitude in Sleep Bruxers**

Khoury, et al., (2008) conducted an observational study of 20 SB subjects. They conducted 2 nights (1 for subject habituation, 1 for data collection) of PSG readings to collect data on SB, arousals and respiratory variables. They found that 4 seconds prior to RMMA activity the amplitude of respiration increased by 8 to 23%. Breathing amplitude increased again by 108 to 206% during RMMA activity and then quickly returned to levels preceding RMMA. There was also a significant correlation found between the frequency of RMMA episodes and the amplitude of breath (P=0.02). A major limitation to this study is the lack of a control group for comparison.
Rise in Blood Pressure

Nashed, et al., (2012) conducted a PSG observational study of 10 sleep bruxing subjects and 9 controls over 3 nights to investigate if there was a rise in arterial blood pressure in association with SB events and to identify if the blood pressure fluctuation varied in conjunction with cortical arousals and/or body movement, or when SB occurred in clusters. They observed a significant increase in blood pressure for all single RMMA/SB and arousal episodes ($P=0.05$). RMMA Index, average RMMA duration and arousal index were all significantly higher (1777%, 290% and 177% respectively) in sleep bruxers than control subjects. Limitations to this study include the limited number of participants and the limited observation of the SB/RMMA episodes (only episodes during NREM2 were analysed).

The physiological and psychological association of stress to SB is well documented in the literature. This review will now focus on the conventional treatment of SB and will subsequently explore manual therapy treatments of TMD, an associated condition of SB.

Treatment of Sleep Bruxism

Conventional treatment of SB usually consists of an occlusal splint. Medications such as muscle relaxants or sympatholytic drugs have been used in limited cases and are associated with undesirable side effects (Huynh et al., 2007). Behavioural therapy, such as biofeedback and physical therapy have been mentioned; however, there were few recent studies found investigating the effects of those modalities on SB (Lobbezoo, van der Zaag, van Selms, Hamburger, & Naeije, 2008). Most of the recommendations for treatment of SB aim to reduce bruxing activity with a multi-disciplinary approach (Thompson, Blount, & Krumholz, 1994). It has been noted that most studies are of low rigour and that further research needs to be conducted (Lobbezoo et al., 2008).

Oral Devices

Oral devices, in particular occlusal splints, are the most common form of treatment for SB. They provide a shield between the biting surfaces of the teeth which reduces wear and grinding noise. They have been hypothesised to decrease bruxing activity by altering muscular patterns of the masticatory system and studies have shown a reduction in bruxism activity (Amorim, Vasconcelos Paes, de Faria Junior, de Oliveira, & Politti, 2012). In a PSG study of 13 severe sleep bruxers the mandibular advancement device (MAD), normally used
for severe snoring or sleep apnoea, showed a high effect size (ES=1.46) in reduction of
bruxing episodes (42% decrease, P < .001) (Landry et al., 2006). Despite this, pain was
reported by 8 of the 13 participants after one night of use. Dubé, et al., measured the effect
of the occlusal splint on 9 participants in a PSG study. They found a statistically significant
reduction in the number of SB episodes per hour (decrease of 41%, p = 0.05) and SB bursts
per hour (decrease of 40%, p < 0.05) with 50% fewer episodes with grinding noise (P =0.06)
(2004). While these studies demonstrate the benefit of oral devices, both of the studies had
limited participant numbers and were restricted to people exhibiting severe bruxism as per
PSG findings. A Cochrane review in 2007 investigated the efficacy of the occlusal splint in
comparison to palatal splint, mandibular advancement device, transcutaneous electric nerve
stimulation and no treatment. Of the 32 potential studies identified, only 5 RCTs were
included in the review. It was concluded that there is no sufficient evidence that occlusal
splints can treat sleep bruxism; however, they may help prevent tooth wear (Macedo et al.,
2007). A review by Klasser, et al., (2010) concurred that oral appliances are best used to
decrease tooth destruction and hopefully reduce masticatory muscle activity when
prescribed for SB.

**Medication**

Medication such as clonidine, a selective alpha2 antagonist, has been shown to be
promising for severe cases of bruxism. Clonidine reduced the SB index by 61% (P<0.05) in
one PSG study involving 25 participants. It decreased cardiac activity and sympathetic tone
during stable sleep and in the minute leading to RMMA activity, breaking the sympathetic
arousal sequence that precedes SB activity (Huynh, Kato, et al., 2006; Gilles J. Lavigne et al.,
2007). In contrast, propranolol, a non-selective beta blocker, did not affect SB. This study
supports the observations of the role of sympathetic activity in the genesis of SB and may
contribute towards understanding the aetiology of the condition. This intervention does
need additional dose dependant research as one of the side effects of the clonidine was
severe waking hypotension (Huynh, Lavigne, et al., 2006).

Botulism toxin (Botox) injected into the masseter muscles has been effective for severe
cases of SB. An 85% reduction of SB activity was observed in a study involving 18 sleep
bruxers who had prolonged histories of the condition and difficulty speaking, swallowing or
chewing. Only 1 subject reported adverse effects (dysphagia) from the Botox treatment. It
was noted that there is a risk of excessive joint laxity and dislocation with this treatment. The study concluded that Botox injections into the masseter muscles could be an effective solution for people suffering from severe cases of SB. It was emphasised that Botox should only be considered for people who did not experience any relief from other conventional medical treatments and that it must be administered by skilled practitioners (Tan & Jankovic, 2000).

The administration of clonazepam, which enhances the action of gamma-aminobutyric acid (GABA), showed a significantly decreased SB index in all 21 participants in a PSG study (mean: -42 ± 15%); however, the medication is associated with dependency issues and drowsiness (Saletu, Parapatics, Anderer, Matejka, & Saletu, 2010). Other studies by the same author produced similar results (Saletu et al., 2005).

Other medications including antidepressants, serotonergic and dopaminergic medicines have been investigated, there has been no consensus regarding their effectiveness on SB (Lobbezoo et al., 2008). While medications seem to show promise for decreasing SB activity, there is much research to be done with regards to dosage and safety. The side effects of drowsiness, hypotension and potential dependency issues currently make the use of medications restricted to only the most severe of SB cases. Again, most studies investigating the effects of medications on SB have limited participant numbers, varied risk of bias and overall low rigour. A Cochrane review points to the need for more well-designed RCTs with larger participant numbers and standardised outcome measures (Macedo, Macedo, Torloni, Silva, & Prado, 2014).

Behavioural Therapy
Biofeedback seems to show short term decreases in SB behaviour. Casas, et al., (1982) found that stress-reduction behavioural counselling in combination with nocturnal auditory biofeedback was more effective than counselling or biofeedback on their own. A more recent study using slightly noxious lip stimulation as a feedback mechanism was found to significantly decrease the duration of individual bruxism events (p=0.038) (Nishigawa, Kondo, Takeuchi, & Clark, 2003). Another study using auditory biofeedback demonstrated dramatic decreases in short term nocturnal bruxism activity but is very biased as the author invented the EMG device used in the study (Weinstein, 2013). While the results of studies utilising auditory biofeedback seem promising, there are concerns about the long-term use
of such devices. These include daytime sleepiness from the auditory arousal and the general long-term efficacy of the method (Lobbezoo et al., 2008).

**Manual Therapy**

Manual therapies for the treatment of the effects of SB have been mentioned in reviews but very few studies were found investigating the effect of any modalities on SB activity. They have, however, been shown to reduce pain and increase range of motion of the TMJ in studies on manual therapy for TMD. Since there is a high association between the two conditions and SB presents with many of the same symptoms of TMD, the following section will review the literature focused on manual therapy modalities on the symptoms of TMD.

Manual therapies include physical therapy, massage therapy, chiropractic therapy and osteopathic manual therapy (OMT). Each of these modalities share some similarities with regards to the techniques employed, but the philosophy behind examination and treatment implementation differ (King et al., 2011). This section will briefly examine the proposed mechanisms behind the effects of manual therapy and will discuss recent investigations on the effect of manual therapy for TMD and SB. Finally, OMT will be introduced.

Manual therapies generally aim to reduce pain and discomfort by utilising a variety of hands-on techniques, including but not limited to soft-tissue massage, myofascial release, manipulation, trigger point release and joint articulation. These techniques can help to reduce tissue hypertonicity, stimulate proprioception, break fibrous adhesions stimulate the production of synovial fluid (Simons & Travell, 1998). Local tissue perfusion is enhanced and any inflammatory exudates can be dispersed from the area, decreasing pain (Parsons & Marcer, 2006). Some of these techniques also engage with the autonomic nervous system via the non-noxious stimulation of cutaneous sensory nerves (Olausson et al., 2002). The stimulation activates the insular region of the brain, which has seems to have an effect on positive emotion, affiliation and coping and increases PSNS activity (Craig, 2003). Physiological stress is also reduced due to the release of oxytocin in the brain, which reduces HPA-axis activity and helps to facilitate the relaxation response (Benson, 1983; Uvanas-Moberg, Arn, & Magnusson, 2005).
**Temporomandibular Disorder**

Temporomandibular disorder (TMD) is a term used to describe a collection of conditions associated with the TMJ area, including pain, sounds and decreased range of motion (ROM) of the temporomandibular joint (Scrivani, Keith, & Kaban, 2008). Bruxism, both wakeful and sleep, is one factor that has been shown to have a strong association with the condition (Blanco Aguilera et al., 2014; Kalamir et al., 2007b; Scrivani et al., 2008). TMD has been documented to respond favourably to manual therapy techniques, including manipulative therapy, soft tissue techniques, articulation and ischemic pressure (Kalamir, Pollard, Vitiello, & Bonello, 2007a; Scrivani et al., 2008).

There are a few studies focusing on the treatment of symptoms associated with TMD with manual therapy techniques; however, most of these studies are limited in their validity due to the study design or low participant numbers. It should be noted that these studies invariably focus on the decrease in pain and headaches and the increase of ROM of the TMJ and do not address bruxing activity (Kalamir et al., 2007b; Sault, Emerson Kavchak, Courtney, & Tow, 2014). They are still important to review due to the lack of literature available on manual therapies and SB.

A review by Medlicott, et al., (2006) investigated the effectiveness of a variety of physical therapy interventions for TMD. The authors established the following recommendations from the 30 studies evaluated: active exercises and joint articulations may be effective for reducing pain and increasing range of motion within the TMJ; relaxation techniques and biofeedback, electromyography training and proprioceptive re-education seem to be more effective than placebo treatment or occlusal splints. Combinations of active exercises, manual therapy, postural correction and relaxation techniques may also be effective (M. Medlicott & S. Harris, 2006). It was noted that most of the studies were of low rigour and limited by the number of participants or research methods and emphasised the need for larger, more rigourous studies.

A literature review focused on manual therapy treatment of TMD was conducted by Kalamir, et al.,(2007a). The modalities reviewed included manipulative therapies, massage, trigger point release and articulation. The authors mentioned that many studies investigating manual therapy didn’t include the exact techniques employed. Eight RCTs of sufficient rigour were included in the review, of which three included manipulative therapy
of the TMJ. The authors concluded that manual therapy is a viable and cost-effective conservative treatment for TMD and that the use of manual therapy for the treatment of TMD will increase in the future. The authors also emphasised the need to investigate multi-disciplinary approaches for the treatment of TMD in the future.

Kalamir, et al., (2012) more recently conducted an RCT that investigated the long-term effects of intraoral myofascial therapy (IMT) and the effect of education and self-care on a group of participants with chronic TMD. Ninety-three participants were randomised into 1 of 3 groups: IMT, IMT plus self-care (IMTESC) and a wait-list control. Outcome measures were jaw ROM and pain scores using an 11-point chronic pain scale. Participants in the treatment groups received 10 treatments over a 5 week period and were assessed at baseline, 6 weeks, 6 months and 1 year. Participants in the IMTESC group also received 2 minute lectures on basic TMJ anatomy, biomechanics, disk displacement and dysfunction and the role of psychoemotional factors in TMD at the end of the first 4 treatments. They were also taught mandibular exercises to be performed twice daily at home. Both treatment groups significantly decreased their pain levels and increased their jaw ROM (p<0.05) compared to the control group during the baseline period and through the one year followup period. Interestingly, there was little difference in outcome measures between the IMT and IMTESC group during the 6-week and 6-month assessments. It wasn’t until the 1 year assessment that the IMT-only group started to regress while the IMTESC group had plateaued. The authors suggested that most of the treatment effects was from the IMT and the education and self-care sustained those effects in the long-term. This study also demonstrates the benefit of a multi-faceted approach of treatment.

A study comparing the effects of osteopathic treatment with conventional therapy for TMD was conducted by Cuccia, et al., (2009). Fifty participants were randomised into an OMT group (n=25) and a conventional treatment group (n=25). The OMT group received 15 to 20 minute treatments using a variety of osteopathic techniques including myofascial release, balanced membranous technique, muscle energy, manipulation, articulation and cranial-sacral therapy directed at the TMJ and cervical regions. Conventional treatment consisted of an oral appliance and physical therapy consisting of gentle stretching of the masticatory muscles and relaxation exercises as well as cold or hot packs. Both groups received
treatment every 2 weeks for 36 weeks and both groups had access to non-steroidal medication (anti-inflammatories and analgesics) and/or muscle relaxants. Improvements in pain and TMJ ROM in both groups were noted at T1 (24 weeks) and T2 (36 weeks) compared to T0. The results revealed no significant difference between the improvements in both groups. The OMT group needed significantly less medication (24% (n=6) OMT vs 56% (n=14) conventional) during the course of the study. This study concurs with the findings of Licciardone, et al., (2003), in which OMT was shown to be as effective as conventional medical treatment for lower back pain. The authors concluded that OMT was recommended as a treatment for TMD and that dentists should work in collaboration with osteopaths and physical therapists to address the management of TMD.

**Sleep Bruxism**
Very few studies were found investigating manual therapy for the relief of sleep bruxism. A case study was published by Knutson (2003) which chronicles a complete cessation of pain and sleep bruxing activity in a six year-old girl following manipulative therapy to the upper cervical spine. While the results of the case presented indicate a positive outcome, this is a single case study with no PSG investigation to confirm the reported outcome. A study was found comparing massage therapy, traditional occlusal splints, massage therapy with an occlusal splint and silicone occlusal splints for the treatment of TMD and SB yielded no significant decreases in EMG activity for any group. The outcome measure in the study was flawed in that it sought only to determine if the EMG activity of the masseter and anterior temporalis muscles had changed from pre-treatment activity levels. No recording of clenching or grinding activity during sleep was undertaken (Gomes et al., 2014).

**Osteopathic Manual Therapy**
Osteopathy is a recognised system of diagnosis and treatment distinguished by the fact that it “emphasises the interrelationship between structure and function and that has an appreciation of the body’s ability to heal itself” (Parsons & Marcer, 2006, p.5). There were no published studies found that investigated the effects of osteopathic treatment on SB.

**Conclusion**
Current literature emphasises the multi-factorial nature of SB. It is also evident that conventional treatment for the condition may reduce the symptoms of SB; however, a
treatment that safely addresses the pain and disability as well as the associated grinding and clenching activity of the masticatory apparatus has yet to be discovered. Due to its holistic approach, osteopathic manual therapy inherently lends itself to the treatment of SB. There is currently a dearth of literature that has specifically explored the use of OMT for the treatment of SB. This review shows evidence for the need of further investigation of OMT for the treatment of SB. The following study aims to build upon the body of literature by examining the effect of OMT on the signs and symptoms of SB.
Bibliography


Gomes, C., El Hage, Y., Amaral, A., Politti, F., & Biasotto-Gonzalez, D. (2014). Effects of massage therapy and occlusal splint therapy on electromyographic activity and the intensity of signs and symptoms in individuals with temporomandibular disorder and


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Section 2

Manuscript

Note: The manuscript presented here is intended for submission to the International Journal of Osteopathic Medicine (IJOM) but rather than the referencing style specified in the IJOM guidelines for authors, the referencing style follows the American Psychological Association (“APA”). Elsevier’s initiative ‘Your Paper, Your Way’ (www.elsevier.com/yourpaperyourway) now permits manuscripts submitted using other referencing formats and APA was selected because it is easier to follow authors’ names in the text and it matches that required for the Literature Review.
Abstract

**Background:** Sleep bruxism occurs in 8 to 20% of the population and can result in jaw and head pain, tooth destruction and diminished quality of sleep for bruxers and their partners.

**Objective:** To explore whether osteopathic manual therapy (OMT) could alter bruxing activity, pain and psychological wellbeing in self-diagnosed sleep bruxers.

**Methods:** Six sleep-bruxing participants received four weekly osteopathic treatments. Electromyographic activity of bruxing and head and jaw pain, via the Numeric Rating Scale (NRS), were monitored for a pre-treatment period, after each treatment and for a follow-up period. The 21 Question Depression Anxiety and Stress Scale (DASS21) was assessed at study onset and conclusion.

**Results:** Six participants showed clinically significant decreases in NRS score from pre-treatment to immediately following the third treatment. Group changes approached statistical significance from pre- to post-treatment despite a reduced sample size. (Effect Size = -0.71; \( P = 0.06 \)). Five participants experienced a reduction of one or more DASS21 severity ratings for stress by the end of the study. Bruxing activity showed a trend towards improvement in two participants and increased in one participant.

**Conclusions:** These results suggest that osteopathic treatment may help sleep bruxers by reducing their perceived pain and stress levels. The results are inconclusive as to the effectiveness of OMT for reducing nocturnal bruxing activity. Additional controlled studies with more detailed inclusion criteria and higher numbers of participants are suggested.

**Key Words:** osteopathy, nocturnal bruxism, manual therapy, jaw pain, complementary and alternative medicine
Introduction

Sleep bruxism (SB) is the nocturnal, non-functional grinding of the teeth during sleep (Lavigne, 2008). It is associated with pain within the temporomandibular area upon waking, temporal headaches and tooth damage. The condition can affect individuals’ bedroom partners as well, due to the noise from the grinding activity (Giraki et al., 2010; Lavigne, 2008). Approximately 8 to 20 percent of the general population is affected by SB (Lavigne, 2008; Ohayon, Li, & Guilleminault, 2001). The condition is usually self-reported and is more prevalent between the ages of 20 to 45 years with a decline in older people (Giraki et al., 2010; Ohayon et al., 2001). SB is normally self-diagnosed and treatment tends only to be sought when individuals start to experience a decrease in their quality of life due to their bruxing activity (Lavigne, 2008).

The aetiology of SB is unknown, but is understood to be multi-factorial (Lavigne, 2008; Schneider et al., 2007). Occlusal factors were initially thought to be the main contributors to the genesis of SB; however, there is no reliable clinical evidence to support this idea despite its plausibility (Lavigne, 2008; Manfredini, 2004). Current studies tend to support a more centrally located cause of SB. These central factors may involve disruptions of input in the basal ganglion area of the brain, which are associated with muscle hyperactivity during nocturnal dyskinesia, neuroplasticity and neurotransmitter disruptions. Genetics may also play a role in SB (Abe et al., 2012).

Many variables are associated with the disorder. These associations include nicotine dependence, caffeine and heavy alcohol drinking, stressful lifestyle, anxiety and sleep disorders such as snoring, sleep apnoea, or periodic limb movement (Abekura et al., 2011; Lavigne, 2008; Ohayon et al., 2001; Rintakoski et al., 2010). SB has also been linked with neurophysiologic factors. Serotonin receptor polymorphisms have been linked to SB (Abe et al., 2012) and a number of studies show associations between SB, the central nervous system and autonomic arousal (Huang, Song, Wang, Guo, & Liu, 2014; Huynh et al., 2006; Kato, Rompre, Montplaisir, Sessle, & Lavigne, 2001; Khoury et al., 2008; Nashed et al., 2012). Furthermore, numerous studies have shown SB to have a positive association with psychological stress (Abe et al., 2012; Abekura et al., 2011; Carvalho, Cury, & Garcia, 2008;
Giraki et al., 2010; Lurie et al., 2007; Seraidarian, Seraidarian, das Neves Cavalcanti, Marchini, & Claro Neves, 2009).

Traditional treatment for sleep bruxism usually consists of an occlusal splint designed to decrease tooth wear by shielding the biting surfaces. In a review of various SB treatments the splint was shown to reduce noise associated with grinding noise and protect teeth, without major adverse effects (Huynh, Manzini, Rompre, & Lavigne, 2007). In this review, Huynh, et al., also concluded that the mandibular advancement device (MAD) and the alpha-adrenergic agonist clonidine also reduced bruxing activity but were associated with adverse effects (Huynh et al., 2007).

Treatments consisting of manual therapy, medications, counselling and behavioural approaches have also been shown to be effective for reducing the symptoms of SB (Medlicott & Harris, 2006; Scrivani, Keith, & Kaban, 2008). Manual therapy has been shown to reduce pain levels and headaches in people with temporomandibular disorders (TMD) (Cuccia, Caradonna, Annunziata, & Caradonna, 2009). Manual therapy focused on the masticatory system has the potential to promote neurologic change due to the local integration of the sensorimotor system (Yin, Lee, & Lee, 2007). Treatment affects cervical and suboccipital proprioceptors which can influence posture as well as activity in the brainstem, subcortical and cortical brain centres (Cuccia et al., 2009; Yin et al., 2007).

The psychological and endocrine response to manual therapy may play a role in the treatment of SB. Therapeutic touch has been demonstrated to reduce physiological stress by reducing activity within the hypothalamic-pituitary axis and activating parasympathetic activity via the non-noxious stimulation of the cutaneous nerves, resulting in the release of oxytocin and activation of the insular cortex of the brain, which is associated with sensations of wellbeing (Craig, 2003; Olausson et al., 2002; Uvanas-Moberg, Arn, & Magnusson, 2005). A pilot study exploring the effect of osteopathic manual therapy on symptoms of menopause suggests that osteopathic care may decrease psychological stress in some patients due to the amount of time spent with the patient and the therapeutic relationship developed between patient and practitioner (Bone, 2012).

Osteopathic practitioners claim to assist with pain and dysfunction associated with TMD and temporal headaches (Parsons & Marcer, 2006; Ward, 2003): two common presenting
complaints of individuals with SB (Cuccia et al., 2009; Kalamir, Pollard, Vitiello, & Bonello, 2007; Scrivani et al., 2008). Few studies report on the effectiveness of integrated manual therapy techniques such as osteopathy on SB and there is currently minimal research demonstrating a clear mechanism. The aim of this study is to examine the changes in bruxing activity, pain, depression, anxiety and stress occurring following osteopathic treatment and management of people with sleep bruxism.
Methods

Study Design
This study uses as a Single Research Case Design (Cohen, Feinstein, Masuda, & Vowles, 2014). This design was chosen as it allows for the participant to act as their own control and is ideal for externally valid clinical studies within osteopathy due to the personalised (non-standard) nature of each treatment (Sanders, 2003).

The Unitec Research Ethics Committee granted ethical approval on January 18, 2011 (UREC 2011-1146).

Development of the OMT Intervention
The primary researcher conducted face-to-face or Skype interviews with five experienced, registered osteopaths who routinely treat patients presenting with pain from self-diagnosed bruxism. The osteopaths were asked how they generally treat patients presenting with bruxism and what they would focus on assessing outside of the general osteopathic examination. This information was used to develop a semi-standardised assessment and treatment protocol. This allowed for the principles of osteopathic assessment and treatment to be employed while maintaining a level of consistency of treatment.

Recruitment
Intervention Study
Participants were recruited using a study recruitment website (www.researchstudies.co.nz, formerly www.getparticipants.co.nz) and via posters displayed within the Unitec Student Clinic. Men and women aged between 18 and 50 years were invited to participate if they fulfilled the self-diagnostic sleep bruxism criteria outlined by the American Academy of Sleep Disorders. This includes:

- An awareness of tooth grinding activity during sleep.
- Pain in their temporomandibular (TMJ)/temporal area upon waking.
- A partner who can verify the grinding noises during sleep.
- Abnormal tooth wear as confirmed by a dentist.
Potential participants also had to be free from any current treatment for sleep bruxism, including occlusal devices. Medication was noted and checked for the possibility of causing secondary sleep bruxism.

People who registered an interest attended an interview with the lead researcher to confirm eligibility via medical history and were provided with an information sheet outlining the aims and requirements of the study [see Appendix A]. Those eligible and willing to participate signed a consent form.

Sub-Study
A sub-study was performed using non-bruxing participants to confirm the ability of the Sleepguard™ to demonstrate a difference between bruxing and non-bruxing activity. Participants for this part of the study were recruited from the Unitec student clinic and the general public. Non-bruxing participants had to be free from pain in the jaw, head, or neck area upon waking, have no evidence of daytime or nocturnal tooth grinding and no history of tooth grinding as verified by a bedroom partner.

Outcome Measures
Measurements were recorded over a 5-night pre-intervention period, during the 4-week intervention period (for the 3 nights following the first 3 interventions) and over 5 nights following the final intervention. A full briefing was provided to participants prior to the commencement of the study.

Numeric Rating Scale for Perceived Pain (NRS)
Perceived pain levels in the TMJ, occipital and temporal areas were recorded upon waking via the Numeric Rating Scale (NRS) for pain. The NRS is an 11-point numeric scale ranging from 0 to 10. Participants quantified their waking pain level in the TMJ and temporal area with 0 indicating “no pain” and 10 indicating “the worst imaginable pain”. Mild pain is indicated by 1 – 3, moderate pain levels 4 – 6 and severe pain is considered to be 7 – 10. The Minimally Clinical Important Difference (MCID) for chronic musculoskeletal pain is 1 point (Salaffi, Stancati, Silvestri, Ciapetti, & Grassi, 2004).

Sleepguard™
Bruxing activity was measured using the Sleepguard™ EMG [Holistic Technologies, Arlington, MA, USA]. This audible biofeedback headband device was used for it’s monitoring
capabilities. The Sleepguard™ is composed of two EMG sensors placed over the temporalis muscles and a mini-computer that counts the number of sustained clenches and the total time spent clenching through the night. Participants received instruction in the use of this device and demonstrated the ability to properly programme the device, to position sensors over the temporal muscles and activate the unit and to retrieve information from and reset the device. Participants were offered the opportunity to use the biofeedback capabilities of the Sleepguard™ after the conclusion of the study. The information was recorded upon waking by each participant in a journal provided by the lead researcher.

The 21-item Depression Anxiety Stress Scale (DASS21)
Participants filled out a DASS21 (www.psy.unsw.edu.au/dass/) prior to starting their initial pre-intervention data collection and on conclusion of the study. The DASS21 comprises three self-reported scales that measure the emotions of depression, anxiety and stress experienced by the participant over the past week. The DASS21 assigns severity ratings of normal, mild, moderate, severe, or extremely severe to these three emotional states, taking into account the correlation among them and is considered to be a relevant tool for both research and clinical purposes (Lovibond & Lovibond, 1995).

Bruxing Activity Journal
All participants were instructed to record their bruxing activity (from the Sleepguard™) and their NRS scores upon waking after each measurement period. Bruxing participants had the opportunity to make note of any worsening or improvements of their condition that they may have noted through the intervention period [Appendix D].

Intervention Implementation
Four one-hour treatments were provided over four weeks and were administered by two Unitec postgraduate student osteopaths at the Unitec Osteopathic Clinic under the supervision of registered osteopaths. Treatments utilised included cross fibre, harmonic, muscle energy, articulation, high-velocity, low-amplitude thrusts, balanced ligamentous tension and cranial techniques to areas of dysfunction as indicated from the semi-standardised assessment of the individual participant.

Data Analysis
Outcome variables for each individual were inspected visually for changes that occurred from before to after the intervention as suggested by Cohen et al. (2014). Visual analysis
entailed assessment for changes in magnitude, trend or variability coinciding within the intervention and for an abrupt change in magnitude with the onset of the intervention (Cohen et al., 2014). To aid the evaluation of these changes, the conservative dual criteria method was applied (Cohen et al., 2014). Mean level encompassed in a 2 standard deviation band and a linear regression line from the pre-intervention phase were calculated and plotted allowing visual comparison of data points in later phases relative to these parameters. The sizes of effects of treatment for each individual were calculated by dividing the mean score from pre- to post-intervention by the standard deviation of the change scores, thus the size of the effects were scaled according to the variability in response between participants (Wu, Chuang, Lin, Lee, & Hong, 2011).

Group inferential statistical analyses were also undertaken. For NRS and bruxism outcomes, measurements taken pre-intervention (5 test points) and following the final treatment (post-intervention; 5 test points) were averaged. Treatment measurements (total 9 test points) were also averaged in groups of three, after the first, second and third treatments. Nonetheless, analyses of variance (ANOVAs) with Fisher’s LSD post-hoc pairwise comparisons were applied for bruxism and NRS outcome measures and paired t-tests for changes in DASS scores.
Results

Participants
Twelve people from the general public responded to an advertisement on the recruitment website. Ten responders were eligible for the study; two were unable to commit due to time constraints. Eight bruxing participants started the intervention study. One dropped out after the first treatment due to a treatment reaction, the second (D) after the third treatment due to unexpected adenoid surgery. Six participants went on to complete the four interventions and the post-intervention measurements. The participants’ characteristics are described in Table 1.

One participant (E) had noted a history of abuse including physical trauma to the face and head. Her bruxing activity was notably higher than the other participants.

The sub-study group consisted of 9 self-diagnosed non-bruxers recruited from the public via word of mouth. Pain levels, number of clenches and seconds spent clenching were recorded over a 5-night measurement period. One non-bruxing participant was actually determined to be a bruxer via use of the Sleepguard™. This diagnosis was later confirmed by her dentist and her data was removed from the results. The non-bruxing group completed all outcome measures apart from DASS21.

Effects of Treatment
Numeric Rating Scale
Visual inspection of individual trends throughout the pre-intervention, intervention and post-intervention periods showed evidence of improvements for four out of seven participants (Figure 1, left-hand column). For Participants A, B and D, between 71 and 100% of intervention and post-intervention test points were below two standard deviations of the mean NRS score, as calculated from the five baseline measurements. While measurements remained within two standard deviations of the baseline measurements for Participant F, there was a very clear change in trend from the decrease in her NRS score prior to the intervention to consistently no pain during and following it.
Individual effect sizes (ES) for NRS from mean pre-intervention to post-intervention ranged from 0 to 2.05 decrease for all participants except Participant E, who showed a moderate increased effect size in score (0.64; Table 2). Three participants (A, B and F) experienced clinically significant decrease as defined by Salaffi et al. (2004) for chronic pain from baseline to follow-up. All participants, except E, showed a clinically significant decrease from pre-intervention to immediately following the third treatment.

The overall group showed a moderate sized reduction in NRS which marginally failed to attain statistical significance (ES from baseline to follow-up = -0.71; P = 0.06 for ANOVA), with significance reached when Participant E was removed from analysis (ES = -1.10; P = 0.02). Post hoc analyses showed that the greatest change was from pre-intervention to immediately following the third intervention (P = 0.01 when Participant E was excluded).

**Bruxing Activity**
Individual results of the number of clenches and duration of clenching (Figure 1, middle and right hand columns) show that two participants (A and F) experienced a trend towards decrease in their bruxing activity during the treatment phase. Participant A had 35% of intervention and post-intervention test points below 2 standard deviations of the mean clenching duration score calculated from the five baseline measurements while Participant F stayed within the 2 standard deviations. Participant G’s bruxing activity stabilised from the pre-intervention period but stayed within the 2 standard deviations. Three participants (B, C and D) showed little to no change to their bruxing activity during the treatment phase. Participant E showed an increase in bruxing activity with 50% of intervention and post-intervention test points for number of clenches and 29% of test points for clenching duration above the 2 standard deviations. Individual effect sizes for number of clenches from mean pre-intervention to post-intervention ranged from 0.085 to -1.89 for all participants except Participant E, who showed a large increase (ES = 9.23; Table 2). Effect sizes for duration clenching ranged from 0.96 to -1.42 for all participants except Participant E (ES = 6.23).

Changes in neither index of bruxing activity attained statistical significance, even when Participant E was removed from analysis (ES number of clenches = -0.47 and ES duration of clenching = -0.26 with Participant E removed).
DASS21
Three participants (A, B and C) experienced a decrease of at least one severity rating within the depression and anxiety subscales, while two participants (E and F) did not change from their baseline severity ratings and one (G) increased in severity. All participants except G decreased their stress severity rating by one or more levels (Table 2).

Individual effect sizes for the depression subscale ranged from 0.94 to -1.89, anxiety subscale ranged from 0.71 to -1.41 and stress subscale ranged from 0.68 to -1.70.

Changes in DASS21 measures failed to attain statistical significance, even with Participant E removed from analysis (ES ranging from -0.47 to -0.75 with Participant E removed).

Sub-Study Results
Non-bruxers reported almost no pain and recorded a lower number of clenches and duration of clenches per night than bruxers (Figure 2). In addition, their responses were less variable, coefficients of variability (CV) ranged from 0.2–0.5 (mean 0.31) for number of clenches and 0.1–0.6 (mean 0.27) for duration of clenching compared to 0.2–1.1 (mean 0.63) and 0.3–1.2 (mean 0.69) respectively in sleep bruxers.
## Figures and tables

### Table 1. Participant Characteristics

<table>
<thead>
<tr>
<th>Participant</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Clenches baseline median (range)</th>
<th>NRS baseline median (range)</th>
<th>Clenches (no./night) *Mean (SD)</th>
<th>Clenches (no./night) *Median (range)</th>
<th>Hx of bruxism</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>32</td>
<td>F</td>
<td>4 (3-6)</td>
<td>66.2 (27.1)</td>
<td>62 (38-104)</td>
<td>Day and night for 8-10 years.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Dx by dentist, Hx indicated yellow</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>flags** and trauma to jaw area.</td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>23</td>
<td>F</td>
<td>3 (2-4)</td>
<td>52.0 (42.4)</td>
<td>53 (9-112)</td>
<td>unknown</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>26</td>
<td>M</td>
<td>6 (6-6)</td>
<td>50.4 (10.1)</td>
<td>54 (33-59)</td>
<td>Dx 10 years prior by dentist</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>from enamel loss.</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>27</td>
<td>F</td>
<td>6 (5-6)</td>
<td>46.4 (21.5)</td>
<td>40 (31-80)</td>
<td>Dx 6 years prior by dentist.</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>22</td>
<td>M</td>
<td>5 (4-7)</td>
<td>39.8 (43.1)</td>
<td>26 (3-104)</td>
<td>Day and night since “quite young”.</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>39</td>
<td>F</td>
<td>2 (1-2)</td>
<td>35.4(24.1)</td>
<td>31 (4-65)</td>
<td>Bruxing since orthodontic treatment</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>at age 15.</td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>50</td>
<td>F</td>
<td>3 (0-8)</td>
<td>5.2 (3.9)</td>
<td>8 (0-8)</td>
<td>Started age 5 cracks teeth regularly</td>
<td></td>
</tr>
</tbody>
</table>

Participants arranged in order of severity of clenching

*Descriptive statistics include mean, standard deviation (SD), median and range for 5 nights baseline measurements.

**Yellow flags indicative of psychosocial barriers to recovery.

Abbreviations: Dx, diagnosis; Hx, history
Table 2. Effect Size of Changes from Pre-Treatment to Post-Treatment

<table>
<thead>
<tr>
<th>Participant</th>
<th>NRS</th>
<th>Depression</th>
<th>DASS21</th>
<th>Stress</th>
<th>No. Clenches/night</th>
<th>Seconds clenched/night</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pre</td>
<td>post</td>
<td>ES</td>
<td>pre</td>
<td>post</td>
<td>ES</td>
</tr>
<tr>
<td>E</td>
<td>4.4</td>
<td>5.4</td>
<td>0.64</td>
<td>2</td>
<td>4</td>
<td>0.47</td>
</tr>
<tr>
<td>G</td>
<td>3.0</td>
<td>2.8</td>
<td>-0.13</td>
<td>6</td>
<td>10</td>
<td>0.94</td>
</tr>
<tr>
<td>B</td>
<td>6.0</td>
<td>4.0</td>
<td>-1.28</td>
<td>8</td>
<td>6</td>
<td>-0.47</td>
</tr>
<tr>
<td>A</td>
<td>5.6</td>
<td>2.4</td>
<td>-2.05</td>
<td>8</td>
<td>0*</td>
<td>-1.89</td>
</tr>
<tr>
<td>D</td>
<td>5.4</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>C</td>
<td>2.0</td>
<td>2.0</td>
<td>0.00</td>
<td>6</td>
<td>4</td>
<td>-0.47</td>
</tr>
<tr>
<td>F</td>
<td>3.2</td>
<td>0</td>
<td>-2.05</td>
<td>2</td>
<td>0</td>
<td>-0.47</td>
</tr>
</tbody>
</table>

Participants arranged in order of severity of clenching at baseline
ES (Effect Size) calculated as the change from pre- to post-intervention for each participant divided by the standard deviation of changes between participants (excluding Participant E).
DASS21 subscale results indicative of clinically significant change of DASS21 severity rating.
* indicates 2 or more rating change.
Figure 1. Individual Analysis for Numerical Rating Scale (NRS), number and duration (s/night) of clenches
Trendline is indicative of trend during pre-intervention measurements (first five data points). Points outside of the ±2 standard deviation band indicate change from variability of baseline measurements.
**Figure 2.** Sub-Study: Non-Bruxers vs. Bruxers

Data shows mean with error bars indicating standard deviation.
Discussion

The intention of this study was to investigate whether osteopathic treatment preceded noticeable decreases in nocturnal bruxing activity, pain levels and indices of depression, anxiety and stress in sleep bruxers. While bruxing activity, depression and anxiety changes were variable, results here demonstrate a reduction in pain in the TMJ area in most cases that coincided with the onset of treatment. A reduced level of stress in most participants was also observed. The study also demonstrated that the Sleepguard™ device was reliable enough to detect a clear difference between the nocturnal masticatory muscle activity of sleep bruxers and non-bruxers and could be used to evaluate outcomes in a larger controlled trial.

Pain
The reduction in perceived pain levels from osteopathic treatment supports the findings of other studies focusing on TMD pain and dysfunction. Cucci, et al., (2009) conducted an RCT (n = 50) comparing OMT with conventional therapy including an oral appliance, physical therapy consisting of gentle muscular stretching and relaxing exercises, hot and cold packs and transcutaneous electrical nerve stimulation in patients with chronic TMD. The authors demonstrated decreases in the signs and symptoms of pain and dysfunction, including a 3.1mm (OMT) and 2.0mm (conventional treatment) reduction in VAS compared to the 2.2 point average reduction in NRS (excluding Participant E) observed here. While there were no differences between groups, the group receiving OMT needed 60% less pain medication (p = 0.0001) than the conventional treatment group. The focus of the OMT in Cucci, et al., (2009) was on the cervical and TMJ areas to decrease dysfunction within the ligaments and to retrain neuromuscular control of posture and balance. Although sleep bruxism activity was not evaluated, it was noted that bruxism (both wakeful and sleep) may contribute to the development of TMD.

One potential explanation for the reduction in pain levels noted in the present and Cucci (2009) studies is that a combination of manual techniques tailored to each participant’s unique presentation reduces biomechanical dysfunction, leading to a decrease in physiological stress and possible psychological stress. Studies have demonstrated that manual therapy techniques can reduce cortisol levels and increase serotonin levels, with a review by
Field et al. showing average 30% reductions in cortisol following a course of massage therapy treatment (Field, Hernandez-Reif, Diego, Schanberg, & Kuhn, 2005).

The general increase in pain after the third intervention may be due to the knowledge that involvement in the study would be finishing soon. The Hawthorne effect, or clinical trial effect, demonstrates the benefit of being in a trial or study for the participant due to the careful observation of the participant by the researcher. There is the potential that the participants unconsciously reverted back towards their previous pain levels because treatment was finishing (Braunholtz, Edwards, & Lilford, 2001; McCarney et al., 2007).

**Bruxing Activity**

Bruxing activity was variable in most participants. The aetiology of nocturnal bruxism is not fully understood and it is possible that biomechanical or neurological factors determining motor activity in the masticatory musculature wasn’t adequately addressed during the intervention.

The lack of consistent change in bruxing activity reported here concurs with findings of Gomes, et al., in their 2014 RCT investigating the effects of massage therapy and conventional treatment on the EMG activity of the muscles of mastication of people with severe sleep bruxism and TMD. The authors noted no significant decrease in EMG activity of the masseter and anterior temporalis muscles in groups of TMD sleep bruxers treated with either massage therapy (n=15), a conventional occlusal splint (n=15), or a combination of the two (n=15) from pre- to post-treatment.

Similarly to the present study, Gomes, et al., (2014) also demonstrated a decrease in intensity of the signs and symptoms of TMD and sleep bruxism in participants. The largest decrease in TMD intensity (25 points) was in the massage therapy and occlusal splint group (p = >.0001). The manual therapy intervention in this study was massage therapy, which focused on reducing hypertonicity therefore increasing tissue health and muscle function and decreasing local pain of the masseter and anterior temporalis muscles (Gomes et al., 2014).

**Sub-Study**

The sub-study found that the Sleepguard™ was reliable for the non-bruxers and that it was sensitive enough to discriminate the nocturnal masticatory activity between bruxers and non-bruxers. This was shown during the course of the sub-study when one outlier was diagnosed.
as a sleep bruxer by her dentist. She was unaware of her bruxing activity until her bruxing activity (mean number of clenches was 71.2/night compared to non-bruxing group mean of 13.1/night) was recorded by the Sleepguard™. This implies that it could be a portable, more cost-efficient measure of sleep bruxism useful for further studies. The Sleepguard™ is not without limitations. While it is possible to set a threshold for activation of the computer, it does not discriminate bruxing activity from other activations of the temporalis muscles. It is also prone to user error. The results for Participant F show her nocturnal masticatory activity level to be below the average for a sleep-bruxer. This may have been due to faulty placement of the EMG sensors, or a very high threshold setting on the device. However, since her teeth were breaking and she had pain, partner confirmation of the grinding sounds and dental confirmation of her bruxing activity, her results were included in this study. Due to the nature of the study design and hence, individual analysis of each participant, it was concluded that her results could still contribute towards the study.

**Psychological Stress**
The decrease in the psychological stress levels as noted on the stress subscale of DASS21 may be connected to the overall treatment, an effect which has been demonstrated by other manual therapy studies (King, Janig, & Patterson, 2011, p.147). Manual therapy has been shown to activate the insular area of the brain which is connected to a feeling of wellbeing. This is achieved via the release of oxytocin and the non-noxious stimulation of cutaneous nerves which engages the relaxation response (Benson, 1983; Uvnas-Moberg, Handlin, & Petersson, 2014). The review by Field reported post-massage therapy increases of both dopamine and serotonin, neurotransmitters acknowledged for their activating effect on the central nervous system that can lead to a reduction in cortisol release (2005).

**Study Design**
This study used a Single Research Case Design. These study designs, also termed SRCD, are acknowledged to be useful to help generate hypotheses for more resource-intensive controlled trials when evaluating new interventions, when the individual responses to the intervention vary widely and confounding variables are unclear (Riddoch, 1991), or when there is a relatively low prevalence of the condition and large randomised samples are unethical or impractical (Cohen et al., 2014; Kooistra, Dijkman, Einhorn, & Bhandari, 2009). One limitation of this design is the lack of a control group and a consequent inability to
determine the mechanism behind the decreased pain and stress levels most of the participants experienced. However, for this study, the effects for perceived pain and stress were large and coincided with the treatment. This may provide reasonable evidence to suggest that there is a link between the treatment and its effects. Most importantly, the evidence presented suggests that there is a need to conduct further investigations with larger, more rigorous group-based designs (Kooistra et al., 2009).

**Limitations**
Other limitations to this study include the small number of participants. There is reduced statistical power to verify changes noted following the intervention. The reduction in NRS, in particular, might have attained statistical significance in a larger sample, and was statistically significant when one individual’s data were removed. Studies with small sample size are also more vulnerable to outlier effects, thus it is difficult to determine whether it is justifiable to remove individual participants with exceptional clinical presentations from group analyses. Here, we report findings including all recruited participants in addition to results following removal of an exception case.

A potential source of bias in participant selection for this study may have resulted from the recruitment methods employed. Recruitment was limited to convenience sampling around a university setting and people interested in participating in studies enough to join a participant-finding website, potentially increasing the clinical trial effect (Braunholtz et al., 2001). Student practitioners were used, which potentially decreases the effect of treatment due to limited experience with particular techniques. The author was one of the practitioners, which would also contribute to potential bias.

This study’s treatment emphasised an individualised approach that follows osteopathic principles. It was not about investigating one particular technique; rather, a semi-standardised protocol was implemented. This allowed for the practitioner to adapt the treatment to address each participant’s individual needs. This treatment approach may compromise internal validity of the study in favour of external validity, as it replicates more closely current clinical osteopathic practice. With all participants, the practitioner strived to decrease dysfunction in the tissues supporting the upper cervical and TMJ area as well as areas of the body that were highlighted through examination. Each treatment was long enough to allow the participant time to experience a decrease in physiological and
psychological stress and provoke the physiological relaxation response. The practitioner also sought to teach the participant tools they could employ at other times to reduce tension in the TMJ and upper cervical area and promote an overall sense of relaxation. Essentially, the intervention sought to address many of the variables often associated with SB, which are treatable within a New Zealand osteopathic scope of practice. Nonetheless, this study did not reveal that the treatment implemented was able to address the underlying causes of sleep bruxism.

Conclusion

This is the first known study investigating the effects of osteopathic manual therapy on sleep bruxism. The results of this study suggest that osteopathic therapy may decrease pain and stress in people who suffer from sleep bruxism. The results were inconclusive as to the effect of osteopathic treatment on bruxism activity. The study also revealed the reliability of the Sleepguard™ for the measurement of nocturnal masticatory activity in bruxers. This study may contribute to the body of knowledge surrounding manual therapy, particularly osteopathic therapy, for reduction in symptoms of sleep bruxism and provides evidence for larger, controlled studies to be conducted.
Bibliography


Section 3

Appendices
Appendix A: Participant Information

RESEARCH INFORMATION FOR PARTICIPANTS

Can Osteopathic Manipulative Therapy Help to Alleviate Bruxism: A Pilot Study

You are invited to participate in our research investigation. Please read carefully through this information sheet before you make a decision about volunteering.

Principal Researcher

Lorelei Messersmith (Bachelor of Applied Science (Human Biology)) – Lorelei is currently in her 2nd year of the Masters of Osteopathy programme at Unitec New Zealand.

Our Purpose

This study aims to determine if osteopathic treatment is effective for the reduction of sleep bruxism. Sleep bruxism is the unconscious or involuntary grinding or clenching of one’s teeth during sleep. It is usually self diagnosed: sufferers may wake with pain in their jaw and/or teeth, experience jaw pain when eating and/or report complaints from their partner. It may also be determined by abnormal tooth wear.

By taking part in this study you will help us discover if osteopathic treatment helps people who suffer from sleep bruxism. You are also helping us provide initial data about the subject and the treatment of this condition which may contribute to further osteopathic research in the area.

Your voluntary participation

Your participation in this study is entirely voluntary and you may withdraw at any time up to 2 weeks prior to the conclusion of the study. Data collected from your involvement in the study may be withdrawn up until 1 week following your final assessment.
Who may participate?

We are looking for adults between the ages of 18-50 who suffer from sleep bruxism. Participants must have pain within the jaw region that they attribute to sleep bruxism and that rates ≥3 on the numeric (NRS). They must also have evidence from a family member, roommate or partner as to their audible nocturnal grinding habits.

Unfortunately, you will not be included in the study if you have:

- A history of adverse effects with osteopathic treatment
- Are currently under orthodontic treatment or treatment for TMD
- Use a dental prosthesis
- Have evidence of any oro-facial pain condition, neurological or psychiatric disorders or systemic inflammatory disorders.
- Are using any medication that could affect nocturnal bruxing activity.

Please feel free to contact the lead researcher if you are unsure about your eligibility.

What will happen in the study?

Should you agree to participate in the study, you will be required to establish a baseline measurement of your bruxing by wearing a Sleepguard EMG device for 5 nights in a row. This device is worn on your head and has two rubber contacts that sense activity within the temporalis muscle on the sides of your head. It measures the number of times you clench or grind during the night as well as the duration of the clenching or grinding episodes. Instruction will be given as to the placement and use of the Sleepguard. Along with recording the nightly number of episodes and duration of clenching/grinding, you will be asked to rate any pain you associate with bruxing each morning following the measurement. You will be given a diary in which to record this information. You will also be asked to fill in a Depression, Anxiety Scale questionnaire (DASS) to determine the level of stress you are experiencing prior to the beginning of the interventions.

Once the baseline measurement is established, you will receive weekly osteopathic treatment at the Unitec Osteopathic Clinic at the Carrington campus for 4 weeks, aimed at reducing pain in your jaw and reducing the number of times your clench or grind at night by addressing any somatic dysfunction the student osteopath finds when s/he examines you. Treatment will consist of a semi-standardised treatment protocol, determined by a group of qualified osteopaths who have had success in treating this condition. You will be informed of any diagnoses that have been made and the proposed treatment will be explained to you prior to its implementation. The initial session will take 60 minutes, with subsequent sessions lasting 30 minutes. For effective osteopathic diagnosis you will be required to undress to your underwear (shorts and bra or bra-top for females are acceptable). Osteopathic techniques applied as part of this intervention will be restricted to those that are regularly used in the Student Osteopathic Clinic. Some techniques may require that the student osteopath touch your head or jaw. If this is a concern in any way, you can let the student osteopath know and an alternative method may be able to be implemented. The osteopathic treatment will be carried out by a student osteopath currently completing a Master of Osteopathy at Unitec New Zealand, who will be supervised by a registered Osteopath.
Following each treatment, you will be requested to wear the Sleepguard for three nights and record the number and duration of episodes as well as a NRS score each morning upon waking on a sheet provided to you. Once the four treatments have finished, you will be asked to wear the Sleepguard for five consecutive nights and again, record the requested information upon waking. You will be asked to fill in a DASS again to observe any change in the stress levels you are currently experiencing compared to at the start of the study.

**What we do with the data and results and how we protect your privacy.**

Personal information is collected and stored under the guidelines provided by the Privacy Act 1993 and the Health Information Privacy Code 1994. Your name will be recorded on a case history form as per usual clinical policy. However, in all other instances of information collection your identity will remain anonymous and you will simply have an identification number. If the information you provide is reported or published, this will be done in a way that does not identify you as its source. All the data recorded will be stored in a password-locked computer and archived in a locked file room in the Unitec Student Osteopathic Clinic and will be stored for a minimum of 5 years. Access to this data will be limited to the principal researcher, the research supervisor, the osteopathic tutors at the Student Osteopathic Clinic and yourself.

**Discomforts/risks and benefits**

If at any time you feel uncomfortable with a technique, you may inform the student osteopath and the technique will be ceased. An alternate technique may be implemented to achieve the same goal, provided you allow it to be implemented.

Osteopathic treatment is gentle and consent is obtained before any manipulative techniques are implemented. There is the possibility of some discomfort following some techniques. Usually any aggravation lasts for less than 24 hours. Should it persist, assistance will be given to relieve the discomfort.

Your participation in this study may help many people besides yourself who suffer from sleep bruxism by providing some evidence towards the efficacy of osteopathic treatment for this condition.

Please contact us if you need further information about the study.

**Contact Details**

Lorelei Messersmith  
Phone: 021 345 816  
Email: lmaymess@yahoo.com

Jamie Mannion
UREC REGISTRATION NUMBER: 1146

This study has been approved by the UNITEC Research Ethics Committee from January 26, 2011 to January 25, 2012. If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the UREC Secretary (ph: 09 815-4321 ext 6162). Any issues you raise will be treated in confidence and investigated fully and you will be informed of the outcome.
Appendix B: Participant Consent

Can Osteopathic Manipulative Therapy Help to Alleviate Bruxism: A Pilot Study

This form is to ensure that you understand the requirements of your participation and that you are aware of your rights. Please read carefully through the points below. If you are happy and agree with the points then please sign at the bottom of the page. If you have any questions at all please ask the researcher before signing this form.

• I have had the research project explained to me and I have read and understood the information sheet given to me.

• I understand that everything I say and the information I provide will be collected in accordance with the Health Information Privacy Code 1994 and kept confidential and in accordance with the Privacy Act 1993. I understand that the only persons who will have access to my information will be the researchers and relevant clinical staff.

• I understand that all the information I give will be stored securely on a computer at Unitec for a period of 5 years.

• I understand that my discussion with the researcher will be recorded on a case history form as per usual clinical policy.

• I understand that I can see the finished research document.

• I have had time to consider the information provided, to ask questions and to seek any guidance.

• I give my consent to be a part of this project

Participant Signature: ………………………… Date: ……………………………

Principal Researcher: ………………………… Date: ……………………………
# Appendix C: Patient Consent to Treatment

## Unitec

### CLINIC 41

**Osteopathy Services**

Cnr Entrance Three and Carrington Road

Mt Albert

AUCKLAND

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**PATIENT CONSENT TO TREATMENT BY CLINICAL STUDENT FORM**

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**REFERRED BY:** (please circle one)

- Advertising e.g. Flyer Mail-drop, Existing Patient, Healthcare Provider e.g. Doctor, Green Prescription Programme, Clinic WebPage, Osteopathic Staff Member, Osteopathic Student, Self-Referral, Signage, UNITEC Staff Member, UNITEC Student, Word of Mouth.
- Other: ...........................................

**FEE CHARGE:** (please circle one)

- Baby (0 – 4 years), Child (5-12 years), College Student (13 years+), Member of the Public, Osteopathic Student, Pensioner, Student (at another Institute), UNITEC Staff Member, UNITEC Student, Green Prescription Programme Patient (NOT WORKING), Green Prescription Programme (WORKING), ACC Referred.

---

I have received the information booklet about the consultation and consent to, *examination* at the Unitec Osteopathic Clinic by a Clinical Student.

......................................................

*(Signature of Patient, Parent or Guardian)*

......................................................

*(Today’s Date)*

I understand that, before every treatment commences, the diagnosis and treatment will be fully explained to me and verbal consent will be requested. I understand that I will be treated by a Clinical Student under the supervision of an Osteopath (Clinical Tutor).
Appendix D: Participant Data Journal

THANK YOU for taking the time to be a participant in my study! Below you will find a schedule and directions for use of the SleepGuard.

**Week 1**
Intro to SleepGuard/DASS/recording information – please wear the Sleepguard for at least 5 consecutive nights to establish a baseline for your grinding before we commence treatment. Each morning, please be sure to fill in the provided journal detailing the number of clenches, the duration of the clenching and a VAS score upon waking.

**Week 2**
treatment 1  Please wear the SleepGuard for 3 nights following the treatment, and record the data and a VAS score each morning upon waking.

**Week 3**
treatment 2  as above

**Week 4**
treatment 3  As above

**Week 5**
treatment 4  Please wear the SleepGuard for 5 nights, recording the requested information each morning upon waking

**Week 6**
final evaluation, DASS and followup  Please bring the data journals and the SleepGuard to this meeting.

PLEASE!! Ensure that the volume is on 0 before going to bed each night. Feel free to expand on any changes you are experiencing on this journal. I will email or text you reminders for appointments and recording information. Please feel free to contact me at 021 345 816 or unitecbruxingstudy@gmail.com with any questions or concerns.

Sincerely,

Lorelei
**Baseline Measurements**

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<td></td>
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<tr>
<td>NRS</td>
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<td>1</td>
</tr>
<tr>
<td></td>
<td>No pain</td>
<td>worst possible pain</td>
</tr>
</tbody>
</table>

Number of clenches
Number of seconds clenching
<table>
<thead>
<tr>
<th>Date -</th>
<th>Number of clenches</th>
<th>Number of seconds clenching</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRS</td>
<td>0  1  2  3  4  5  6  7  8  9  10</td>
<td>worst possible pain</td>
</tr>
<tr>
<td>No pain</td>
<td>worst possible pain</td>
<td></td>
</tr>
</tbody>
</table>

Date -

Number of clenches
Number of seconds clenching
Appendix E: DASS21

<table>
<thead>
<tr>
<th>DASS21</th>
<th>Name:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The rating scale is as follows:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 Did not apply to me at all</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Applied to me to some degree, or some of the time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Applied to me to a considerable degree, or a good part of time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Applied to me very much, or most of the time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>I found it hard to wind down</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>I was aware of dryness of my mouth</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>I couldn't seem to experience any positive feeling at all</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>I found it difficult to work up the initiative to do things</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>I tended to over-react to situations</td>
<td>0</td>
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<tr>
<td>7</td>
<td>I experienced trembling (eg, in the hands)</td>
<td>0</td>
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<tr>
<td>8</td>
<td>I felt that I was using a lot of nervous energy</td>
<td>0</td>
</tr>
<tr>
<td>9</td>
<td>I was worried about situations in which I might panic and make a fool of myself</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>I felt that I had nothing to look forward to</td>
<td>0</td>
</tr>
<tr>
<td>11</td>
<td>I found myself getting agitated</td>
<td>0</td>
</tr>
<tr>
<td>12</td>
<td>I found it difficult to relax</td>
<td>0</td>
</tr>
<tr>
<td>13</td>
<td>I felt down-hearted and blue</td>
<td>0</td>
</tr>
<tr>
<td>14</td>
<td>I was intolerant of anything that kept me from getting on with what I was doing</td>
<td>0</td>
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<tr>
<td>15</td>
<td>I felt I was close to panic</td>
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<tr>
<td>16</td>
<td>I was unable to become enthusiastic about anything</td>
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<tr>
<td>17</td>
<td>I felt I wasn't worth much as a person</td>
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</tr>
<tr>
<td>18</td>
<td>I felt that I was rather touchy</td>
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<td>Description</td>
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<tr>
<td>19</td>
<td>I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)</td>
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</tr>
<tr>
<td>20</td>
<td>I felt scared without any good reason</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>I felt that life was meaningless</td>
<td></td>
</tr>
</tbody>
</table>
Appendix F: DASS Scoring

DASS 21  NAME ___________________________ DATE

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:
0 Did not apply to me at all - NEVER
1 Applied to me to some degree, or some of the time - SOMETIMES
2 Applied to me to a considerable degree, or a good part of the time - OFTEN
3 Applied to me very much, or most of the time - ALMOST ALWAYS

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<thead>
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<th>AA</th>
<th>FOR OFFICE USE</th>
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TOTALS

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DASS Severity Ratings

The DASS is a quantitative measure of distress along the 3 axes of depression, anxiety1 and stress2. It is not a categorical measure of clinical diagnoses.

Emotional syndromes like depression and anxiety are intrinsically dimensional - they vary along a continuum of severity (independent of the specific diagnosis). Hence the selection of a single cut-off score to represent clinical severity is necessarily arbitrary. A scale such as the DASS can lead to a useful assessment of disturbance, for example individuals who may fall short of a clinical cut-off for a specific diagnosis can be correctly recognised as experiencing considerable symptoms and as being at high risk of further problems.

However, for clinical purposes it can be helpful to have ‘labels’ to characterise degree of severity relative to the population. Thus the following cut-off scores have been developed for defining mild/moderate/severe/ extremely severe scores for each DASS scale.

Note: the severity labels are used to describe the full range of scores in the population, so 'mild' for example means that the person is above the population mean but probably still way below the typical severity of someone seeking help (i.e. it does not mean a mild level of disorder).

The individual DASS scores do not define appropriate interventions. They should be used in conjunction with all clinical information available to you in determining appropriate treatment for any individual.

1Symptoms of psychological arousal
2The more cognitive, subjective symptoms of anxiety

DASS 21 Score

<table>
<thead>
<tr>
<th>DEPRESSION SCORE</th>
<th>ANXIETY SCORE</th>
<th>STRESS SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>5 - 6</td>
<td>4 - 5</td>
</tr>
<tr>
<td>Moderate</td>
<td>7 - 10</td>
<td>6 - 7</td>
</tr>
<tr>
<td>Severe</td>
<td>11 - 13</td>
<td>8 - 9</td>
</tr>
<tr>
<td>Extremely Severe</td>
<td>14+</td>
<td>10+</td>
</tr>
</tbody>
</table>

N.B. Please note that there are differences in scoring between this measure and the DASS 21 as published on the DASS website http://www2.psy.unsw.edu.au/groups/dass/
Appendix G: Semi-Standardised Exam

Semi standardised examination/treatment protocol for Bruxism project

Examine General Osteopathic Examination with special attention to: (w treatment of positive findings as necessary)

- Sacrum/Si joints
- Sacroiliac Joints
- Lumbar spine
- Diaphragm (inhibit)
- Thoracic Spine (T1-T7 in particular/ribs/respiration)
- Shoulder girdle
- Anterior cervical tissues, mediastinum pectoral mms
- Clavicle, First Rib, Thoracic outlet, Hyoid
- Csp (OA, AA, C2-T1 ROM, active, passive)
- TMJ as per Hoppenfeld

  *inspection of swing and stance* phases looking for malocclusion or asymmetrical mandibular motion

  *bony palation of TMJ via external auditory canal* – feel for assymetrical motion, crepitation or clicking. Pt to open as wide as possible – feel for dislocation of the jnt

  *soft tissue palpation* – external pterygoid mm, masseter, temporalis,TMJ capsule

Suboccipitals

Advice – stress relief, breathing, awareness of tension in and release of mm of mastication, self trigger point release, self met to suboccipitals, masticatory muscles and as necessary, educate on proposed causes of bruxism.

Appendix H: Ethical Approval

Lorelei Messersmith  
26 Rossgrove Terrace  
Mt Albert  
Auckland 1025  

26 January 2011  

Dear Lorelei,  

Your file number for this application: **2011-1146**  
Title: **Can Osteopathic Manipulative Therapy (OMT) Help to Alleviate Sleep Bruxism: A pilot study.**  

Your application for ethics approval has been reviewed by the Unitec Research Ethics Committee (UREC) and has been approved for the following period:  

Start date: **26 January 2011**  
Finish date: **25 January 2012**  

Please note that:  

1. The above dates must be referred to on the information AND consent forms given to all participants.  
2. You must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.  

You may now commence your research according to the protocols approved by UREC. We wish you every success with your project.  

Yours sincerely  

Lyndon Walker  
Deputy Chair, UREC  

cc: Craig Hilton  
Cynthia Almeida
Appendix I: Instructions for Authors

Please see:

http://www.journalofosteopathicmedicine.com/content/authorinfo