Aus der Klinik und Poliklinik für Zahnärztliche Prothetik

der Universität Würzburg

Direktor: Professor Dr. med. dent. Marc Schmitter

EMG activity of masticatory muscles of patients with different bruxism grades during tasks with submaximal controlled force

Inauguraldissertation

zur Erlangung der Doktorwürde der

Medizinischen Fakultät

der

Julius-Maximilians-Universität Würzburg

vorgelegt von

Nada Obid

aus Bietigheim-Bissingen

Würzburg, März 2023

Referent: Priv.-Doz. Dr. med. dent. Nikolaos Nikitas Giannakopoulos
Korreferent: Prof. Dr. med. dent. Gabriel Krastl
Dekan: Prof. Dr. Matthias Frosch

Tag der mündlichen Prüfung: 11.07.2023

Die Promovendin ist Zahnärztin

To my parents

Arbeitstitel

Elektromyographie der Kaumuskulatur unter kontrollierter Beißkraft.

Table of Contents

1. Introduction

Bruxism is often considered a masticatory muscle activity (MMA) that affects many people worldwide. To date, it partially remains an unidentified condition that may be a risk factor for other disorders but cannot be considered a disorder per se (Lobbezoo et al., 2018). Due to the multifactorial nature of its etiology, it is challenging to treat bruxism causally. Hence, several people are believed to suffer from the impact of bruxism, i.e., extensive tooth wear, wear - or technical complications of restorative materials (Johansson et al., 2011), and a higher prevalence for temporomandibular disorders (TMD) (Manfredini et al., 2010). This fact makes it crucial to identify bruxers at risk early on, in order to reduce the sequelae and to treat them prophylactically.

Developing new diagnostic tests suitable for the daily praxis may help simplify the (early) identification of bruxers and will contribute to recognize the fine line between therapy while avoiding overtreatment.

1.1. Bruxism

1.1.1. Definition of bruxism

According to the international consensus in 2013, updated in 2018, experts developed the definition of bruxism and described it as a “repetitive jaw-muscle activity characterized 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 awake bruxism)”, (Lobbezoo et al., 2013) or as a combination of both (Lobbezoo et al., 2018).
Based on further investigations, it gradually became more relevant to develop two separate definitions for sleep and awake bruxism, emphasizing the two distinct behavioral phenotypes. Therefore, experts updated the definition in 2018, as follows:

1. *“Sleep bruxism is a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals.*
2. *Awake bruxism is a masticatory muscle activity during wakefulness that is characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible and is not a movement disorder in otherwise healthy individuals*.” *(Lobbezoo et al., 2018).*

1.1.2. Anatomy of masticatory muscles

In order to understand bruxism, it is inevitable to discuss the muscles that participate in the chewing process, also called mastication. Eight masticatory muscles (MM) enable the chewing process but are also involved in most other functions of the masticatory system. Four of these major muscles are involved in jaw movements: the masseter muscle, temporal muscle, lateral pterygoid muscle, and medial pterygoid muscle. *Table 1* and *Figure 1* illustrate the anatomy of the masseter and temporalis muscle, as they are the most relevant for the subsequent clinical trial.

Table 1: Anatomy of the masticatory muscles (masseter and temporalis), their origin, insertion, innervation, and function.

Muscle	**Masseter**	**Temporalis**
Origin	Superficial part: maxillary process of zygomatic bone, zygomatic arch (anterior 2/3) Deep part: zygomatic arch (posterior 1/3)	Temporal fossa (up to inferior temporal line), temporal fascia
Insertion	Lateral surface of ramus and angle of the mandible	Coronoid process of the mandible and retromolar fossa
Innervation	Mandibular nerve (trigeminal nerve, V3)	Mandibular nerve (trigeminal nerve, V3)
Function	• Jaw elevation (jaw-closing) and • Protrusion of the mandible • Jaw clenching	• Elevation and • Retraction of the mandible • Jaw clenching • Unilateral contraction leads to laterotrusion to the ipsilateral side

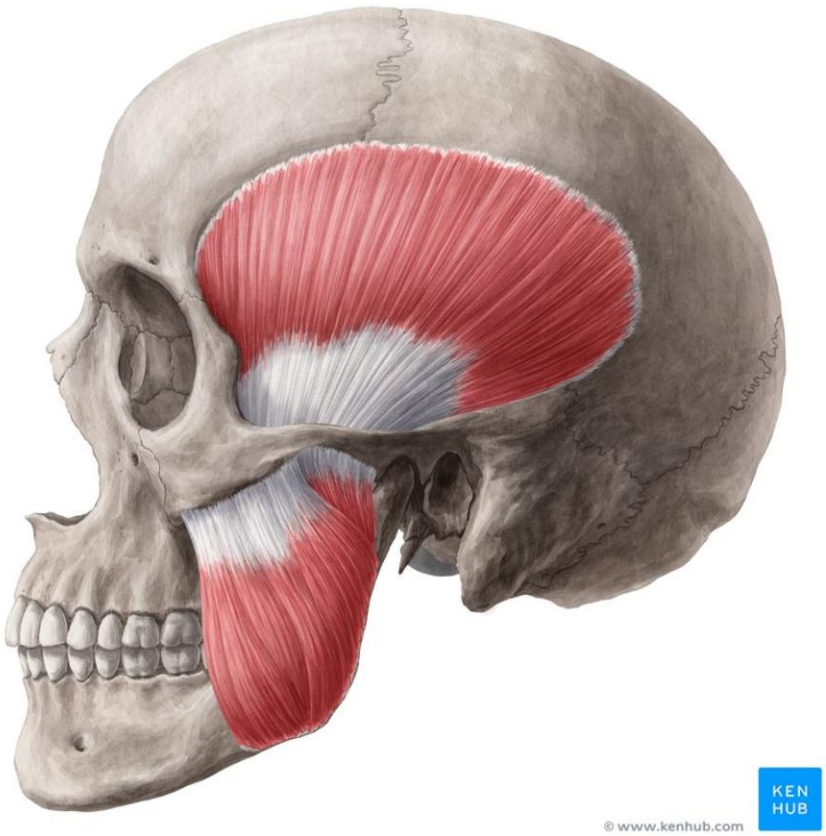


Figure 1: Musculus masseter and musculus temporalis.
(With friendly permission by © Kenhub (www.kenhub.com); Illustrator: Yousun Koh).

1.1.3. Epidemiology

The prevalence of bruxism varies tremendously in current literature due to the use of various diagnostic tools (e.g., self-report, clinical inspection, polysomnography [PSG] and electromyography [EMG]). The majority of available data focuses on sleep bruxism (SB) and report a prevalence of 12.8% ± 3.1%, and 22.1% up to 31% related to awake bruxism (AB) (Lavigne et al., 2008; Lobbezoo et al., 2012). Up to date, it is premature to suggest a significant predominance of one gender in the prevalence of bruxism, as the currently available evidence has been inconclusive. Some studies suggest a higher prevalence for men (Manfredini et al., 2012), others for women (Lavigne et al., 2008). Further data analysis indicate no correlations with sex (Manfredini et al., 2013).

The onset of bruxism is observed after the eruption of the first deciduous tooth, rather decreasing across the life span (Lavigne et al., 1994; Shetty et al., 2010), with a prevalence peak in the second until the third decade of life (Lobbezoo et al., 2012; Peroz et al., 2019). Among the few existing longitudinal studies, there are two which have proven that increased oral activity in childhood is indeed a risk factor for bruxism in adulthood (Carlsson et al., 2003; Egermark et al., 2001; Peroz et al., 2019).

1.1.4. Classification

The bruxism classification adopted by Lobbezoo et al., as mentioned above (1.1.1), is based on the circadian rhythm of bruxism, underlining the different temporal and behavioral patterns. It is most frequently used among all bruxism classifications for it distinguishes between sleep – and awake bruxism. However, bruxism can also appear as a combination of both. Another classification relies on an etiological differentiation, separating primary bruxism and secondary bruxism. Primary bruxism is the idiopathic form of bruxism, occurring spontaneously and in the absence of a medical cause. Secondary bruxism, on the other hand, results from other medical conditions associated with neurologic/ psychiatric/ sleep disorders or as a side-effect of medication and drug abuse/ withdrawal. *Table 2* displays the disorders and medications which induce iatrogenic bruxism (Murali et al., 2015; Shetty et al., 2010).

Table 2: Secondary bruxism (Murali et al., 2015; Shetty et al., 2010).

Neurologic disorder	Cranial and cervical dystonia, epilepsy, Huntington's disease, Parkinson's disease, coma, Alzheimer's disease, anoxic/traumatic brain injury, stroke (Guaita et al., 2016)
Psychiatric disorder	Depression, bipolar disorder grade II, schizophrenia, anxiety disorder (Piccoli et al., 2014)
Sleep disorders	Sleep apnea, insomnia
Medications	Antidepressants (SSRI), anticonvulsive drugs, antipsychotics, antihistamines, dopaminergic drugs, calcium antagonists
Drugs	Cigarettes, Alcohol, Amphetamines, Cocaine, Ecstasy (Milosevic et al., 1999; Peroz et al., 2019; Thie et al., 2001)

Bruxism can appear in different intensities (low, medium, high) and frequencies (tonic, phasic). Therefore, it can be further classified according to the type of muscle activity.

- Non-rhythmic (tonic) bruxism = Muscle contraction > 2s.
- Rhythmic (Phasic) bruxism = short, repetitive contractions of the masticatory muscles, with more than three muscle activations in the electromyogram (EMG) and a duration of 0.25 up to 2 seconds.
- Combined: Alternating appearance of tonic and phasic episodes.

However, 90% of sleep bruxism EMG events are of phasic and combined (tonic and phasic) nature, while the tonic form is instead associated with awake bruxism (Lavigne et al., 1996; Murali et al., 2015; Peroz et al., 2019; Rompre et al., 2007; Zucconi et al., 2014).

Lavigne et al. have suggested that the most typical EMG pattern related to SB shall be referred to as “rhythmic masticatory muscle activity (RMMA)”. These RMMAs appear in “healthy” individuals as well as in patients with SB, although much more frequently in the latter case (Lavigne et al., 1996; Lavigne et al., 2001). *Figure 2* illustrates different RMMA EMG patterns.

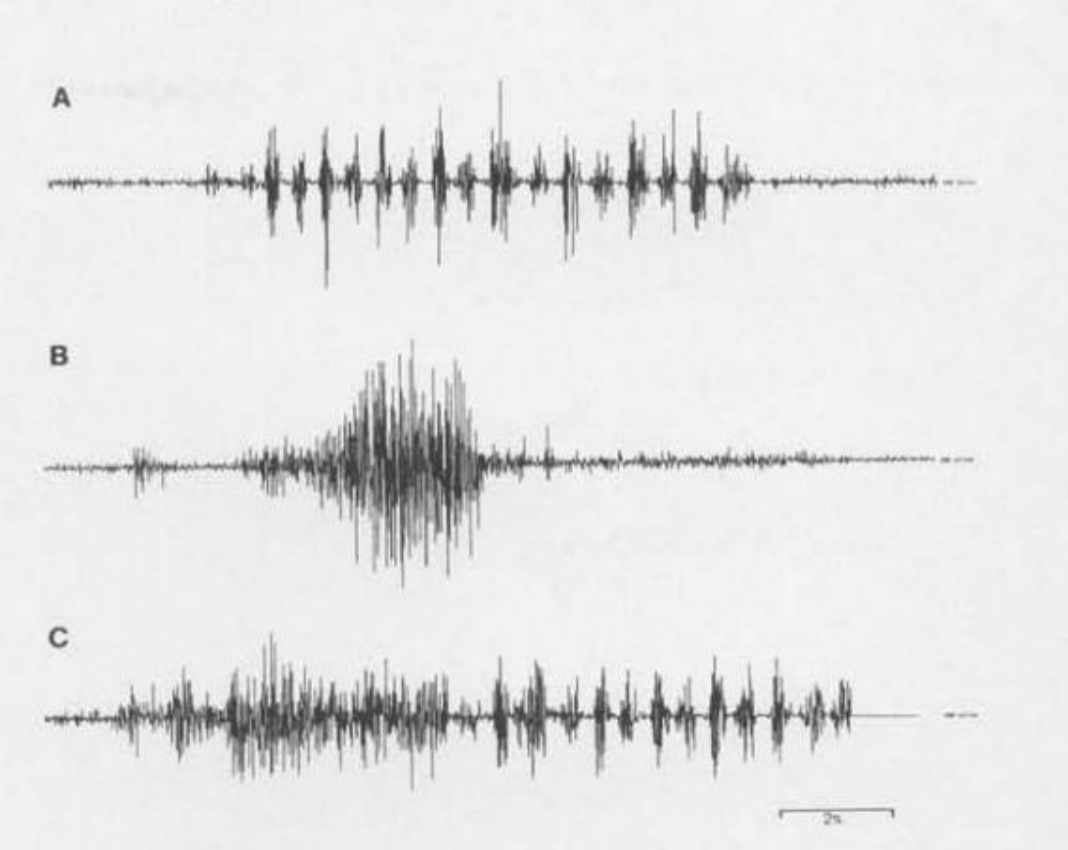


Figure 2: Examples of phasic (A), tonic (B), and mixed (C) bruxism episodes recorded from a right masseteric muscle (Lavigne et al., 1996). (With friendly permission by SAGE Publications).

1.1.5. Etiology

The complexity of bruxism lies in its multifactorial, yet, not fully understood etiology (Manfredini et al., 2009; Murali et al., 2015). Since current evidence is inconclusive, it is premature to suggest that one specific etiology is responsible for sleep or awake bruxism. It is important to emphasize that the following reviewed etiological factors are merely contributing factors to the multifactorial nature of bruxism (Lavigne et al., 2008; Lobbezoo et al., 2001b). Furthermore, AB and SB are considered different entities, leading to the assumption of distinct underlying etiologies. Nevertheless, as both phenomena are inadequately distinguished in most studies, it is impossible to evaluate the different causative factors yet and, therefore, essential to review the etiology in its entirety.

The etiology of bruxism in adulthood distinguishes between peripheral (morphological) and central (pathophysiological and psychological) factors. Previously, it was assumed that morphological factors, e.g., occlusal discrepancies and the anatomy of the bony structures of the orofacial region were the cause for bruxism. Establishing harmony between maximum intercuspation and centric relation was believed to prevent and treat bruxism. However, current research recedes from a peripheral cause for bruxism, as lack of evidence led to the assumption that neither occlusal factors nor the morphology of the bony structures of the orofacial region are consistently associated with a higher probability for bruxism, and if so, it is only a secondary factor (Lavigne et al., 2008; Lobbezoo et al., 2001b; Lobbezoo et al., 2012; Manfredini et al., 2004a; Manfredini et al., 2004b). Recent focus has shifted towards the central pathophysiological factors, although it still requires further investigation. First of all, sleep bruxism is suggested to be part of a sleep arousal response and is therefore classified among the parasomnias. “Arousal response” is a sudden change in the depth of sleep to a lighter stage, to the extent of waking up. During this phenomenon, several symptoms occur, e.g., body movements, increased heart rate, respiratory changes, and increased RMMA. Most bruxism episodes happen during this stage (Lavigne et al., 2003).

Furthermore, bruxism appears to be modulated by various neurotransmitters in the central nervous system. More specifically, disturbances in the central dopaminergic system have been associated with bruxism (e.g., Parkinson’s disease, pharmacological therapy) (Lobbezoo et al., 2001b). According to the available literature, it is controversially discussed whether further central factors, such as psychosocial factors and several psychopathological symptoms (viz., emotional distress, anxiety, depression, and nervous tics), could be risk factors for bruxism. If so, these risk factors could be mainly associated with awake bruxism (Lavigne et al., 2008; Lobbezoo et al., 2018; Manfredini et al., 2009). In contrast to AB, there seems to be no evidence relating SB to psychological disorders. Hence, SB is regarded as a result of a disorder in the central nervous system (Manfredini et al., 2016b; Peroz et al., 2019). However, research on these psychological factors comes to equivocal results and needs further attention (Lobbezoo et al., 2001b).

1.1.5.1. Risk factors and Comorbidities of bruxism

Identifying possible risk factors may help understand the etiology and find the proper management for bruxism. An important term in this context is the introduction of the term odds ratio (OR), which is a measure for risk assessment and indicates how much greater the chance is to encounter a disease in a group with a certain risk factor compared to a group without this factor. When the OR is greater than 2 it is considered “harmful”, while an OR smaller than 0.5 is rather “protective” (Gesch et al., 2005). Another important term is the confidence interval, a range of values that is likely to include the estimated value with a certain degree of confidence. These two measures are used to quantify the correlation between the actual disease and the risk factors, which increase the probability of developing it.

Risk factors can have a causal influence (causal risk factors) or lead to an elevated chance of developing the disease (risk indicator). *Table 3* summarizes risk factors for bruxism in general and differentiates grades of severity (A - D) depending on the size of the odds ratio as well as the lower limit of the 95% confidence interval (CI LL), according to Kuhn et al.’s systematic review (Kuhn et al., 2018). The risk factors were graduated by severity as follows: A= very high (OR>2; CI LL>2); B= high (OR>2; 1<CI LL≤2); C= probable (1<OR≤ 2; CI LL>1), D= possible (1<OR≤2; CI LL≤ 2) (Kuhn et al., 2018).

Table 3: Examples of risk factors for bruxism (risk indicator and causal risk factor for bruxism in adults) *(Kuhn et al., 2018), graduated by severity (A=very high, B=high, C=probable, D=possible).*

Grade of severity	**Adults**
A	Social anxiety disorder, emotional stress, material status (married), regular smoking, awake bruxism, sleep bruxism
B	Reflux esophagitis, nocturnal frontal lobe epilepsy, sleep apnea syndrome, anxiety
C	Consumption of stimulants (alcohol, coffee), depression, female gender, depression
D	Chewing of khat (qat)

Furthermore, there have been several medical conditions that have been associated with bruxism. *Table 4* shows some of the comorbidities, which are frequently and simultaneously present along with bruxism.

Table 4: Comorbidities in pediatric and adult populations (Carra et al., 2012).

Parasomnias	Enuresis, sleep talking, sleep walking
Other sleep disorders	Sleep disordered breathing (e.g., snoring, obstructive sleep apnea), insomnia (e.g., frequent awakenings, longer sleep latency)
Medical and psychological conditions	Hypertrophic tonsils and adenoids, allergies, ADHD, headaches, orofacial pain and TMD, anxiety, separation anxiety at bedtime, neurologic disorders
Medications	Methylphenidate (Ritalin), SSRIs (Paroxetine, Fluoxetine, Fluvoxamine, Sertraline), antipsychotics (Haloperidol)
Concomitant oral habits	Nail biting, pen biting, wake-time tooth clenching
Further Comorbidities	Restless legs syndrome, Rett-Syndrome, Parkinson's disease, Huntington's Disease, Autistic spectrum disorders, Down Syndrome (Ella et al., 2017), Ecstasy (Milosevic et al., 1999)

To date, bruxism remains a behavior with possible sequelae, which may be a risk factor for some disorders but may also have a protective impact (Lobbezoo et al., 2013; Lobbezoo et al., 2018). Remarkably, in some individuals' positive effects were observed. First, bruxism is believed to reduce abnormal chemical tooth wear, resulting from gastroesophageal reflux, by increasing salivation during sleep (Ohmure et al., 2011). Second, after sleep apnea episodes, there have been observations of restoring airway patency by a sudden activation of the jaw muscles, which are commonly associated with SB (Hollowell et al., 1989; Lavigne et al., 2003). The stabilization of the mandible prevents the upper airway from collapsing and is ensured by a coactivation of agonists (genioglossus and lateral pterygoid muscle) and antagonist (masseter muscle) masticatory muscles (Yoshida, 1998).

It is commonly known that bruxism may also have adverse sequelae. Patients report masticatory muscle pain (myalgia) or temporomandibular joint pain (arthralgia) (Ahlberg et al., 2004; Fernandes et al., 2012; Manfredini et al., 2010), extreme mechanical tooth wear (Abe et al., 2009; Zucconi et al., 2014), jaw muscle hypertrophy, tooth destruction, excessive tooth mobility (Holmgren et al., 1993), fracture of dental restoration or rehabilitation (such as chipping or fracture of dental ceramics) (Johansson et al., 2011; Raphael et al., 2016), and social impairment (Lavigne et al., 2008; Lobbezoo et al., 2012; Ohmure et al., 2011).

1.1.6. Diagnosis

Bruxism diagnosis can be based on subjective as well as objective clinical findings. Not all diagnostic instruments are practical for clinical practice and scientific research; hence, they should be distinguished. Due to the behavioral differences between sleep and awake bruxism, it is crucial to separate these two phenotypes' diagnoses further.

Generally, the four Sleep Bruxism Research Diagnostic Criteria (SB-RDC) have been postulated for scientific research. All four criteria must be positive for a valid SB diagnosis (Lavigne et al., 1996), as outlined in *Table 5*.

Table 5: Sleep Bruxism Research Diagnostic Criteria (SB-RDC).

Criterion 1	Grinding or Clenching sounds during sleep, on 3 or more nights/month during the last 3-6 months, which are confirmed by the bed partner
Criterion 2	Tooth wear (with dentin exposure) in one or more sextants
Criterion 3	Masseteric muscle hypertrophy (twice/thrice the volume during contraction, as compared to resting activity)
Criterion 4	PSG findings according to Lavigne et al. (Lavigne et al., 1996) (Table 7)

In 2014, the third edition of the International Classification of Sleep Disorders (ICSD-3) was published by the American Academy of Sleep Medicine. This manual outlines the key findings for a clinical sleep bruxism diagnosis, which is classified under the sleep-movement disorders as presented in *Table 6* (Zucconi et al., 2014).

Table 6: Criteria for a sleep-related bruxism diagnosis by the American Academy of Sleep Medicine (AASM) (adapted from ICSD-3) (Zucconi et al., 2014).

	Criteria A and B must be met
A	The presence of regular or frequent tooth grinding sounds occurring during sleep
B	The presence of one or more of the following clinical signs: 1. Abnormal tooth wear consistent with above reports of tooth grinding during sleep 2. Transient morning jaw muscle pain or fatigue; and/or temporal headache; and/or jaw locking upon awakening consistent with above reports of tooth grinding during sleep

A bruxism diagnosis can be performed with instrumental (polysomnography = PSG, electromyography = EMG, ecological momentary assessment = EMA) and non-instrumental approaches (self-report: questionnaires, oral history; clinical inspection), which differ in their validity and objectivity. A general objective for a valid diagnosis is high sensitivity and specificity of the tests when compared with the gold-standard examinations. Moreover, the tests should be reliable (consistent and repeatable at any time and under any condition) and valid (representing the actual state). *Figure 3* illustrates the criteria for diagnosis**:**

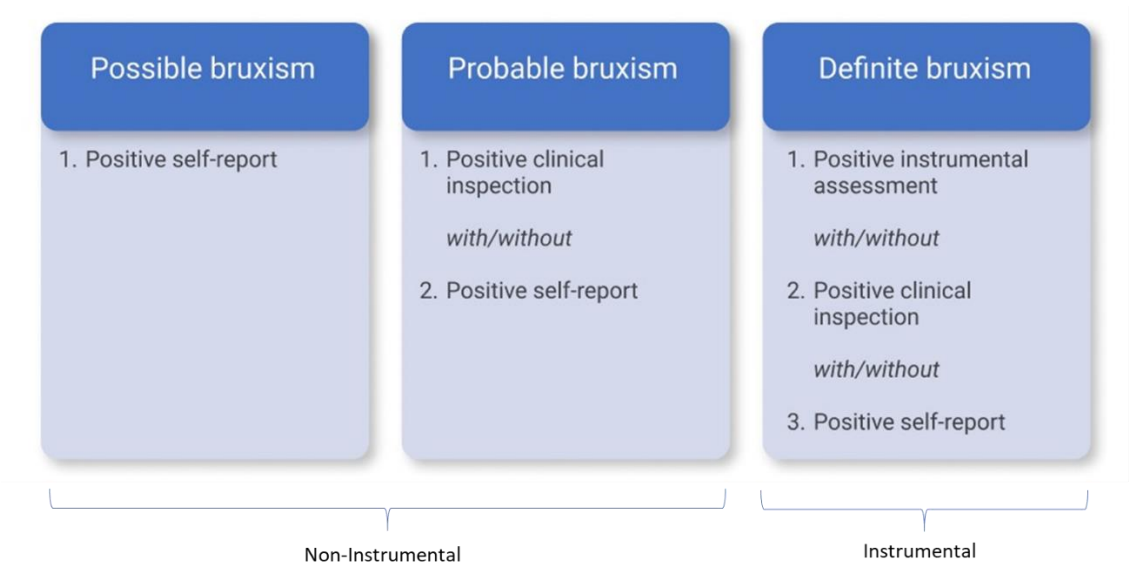


Figure 3: Criteria for a "possible", "probable" and "definite" bruxism diagnosis (Lobbezoo et al., 2018).

1.1.6.1. Non-Instrumental assessment (possible and probable bruxism)

Self-report (possible bruxism)

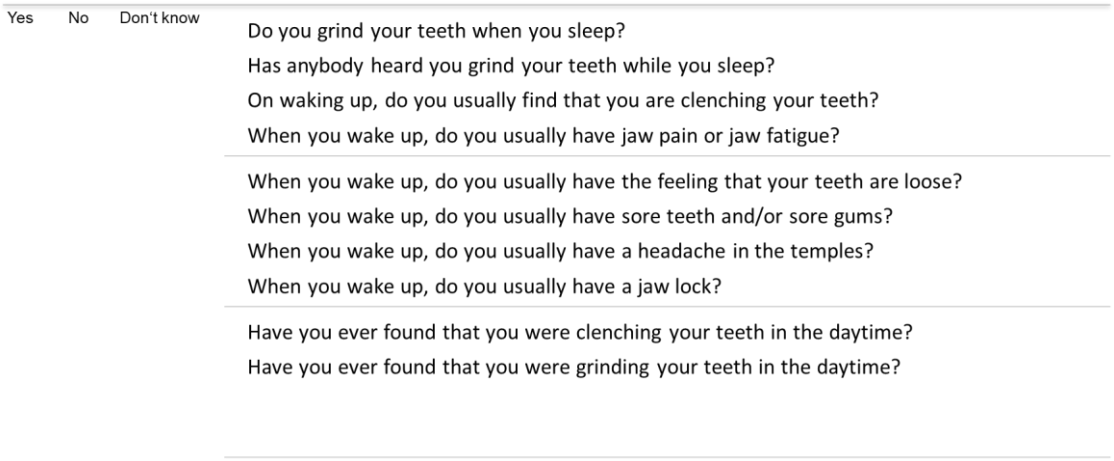
Self-report of either current or anamnestic awake or sleep bruxism via questionnaires and interviews (the anamnestic part of a clinical examination) is still a ubiquitous diagnostic tool in bruxism research and clinical practice. It includes a self-report of current bruxism status, a history report of bruxism status, and a report of complaints possibly related to bruxism (Lobbezoo et al., 2018; Manfredini et al., 2020). Furthermore, it enables a distinction between the presence of possible awake and sleep bruxism (Lobbezoo et al., 2018). These instruments entail questions about the patients' (direct self-report), their bed partner's or parents' (indirect self-report) awareness of SB or AB behavior, sleep habits, anxiety, stress, fatigue, nervousness, teeth soreness, grinding sounds, dental history (e.g., history of broken teeth or restorations), intermittent locking, current facial pain intensity, painful jaw/ jaw stiffness upon awakening, as well as fatigue of masticatory muscles (Manfredini et al., 2020). One example for a questionnaire is the one developed by Pintado et al. (Pintado et al., 1997) with questions illustrated in *Figure 4*.

Yes	No	
		1. Has anyone heard you grinding your teeth at night?
		2. Is your jaw ever fatigued or sore on awakening in the morning?
		3. Are your teeth or gums ever sore on awakening in the morning?
		4. Do you ever experience temporal headaches on awakening in the morning?
		5. Are you ever aware of grinding your teeth during the day?
		6. Are you ever aware of clenching your teeth during the day?

Figure 4: Questionnaire for detection of bruxism (Pintado et al., 1997).

At least two of the six items have to be positive in order for the patient to be classified as bruxer, according to the clinical diagnosis based on anamnesis and clinical inspection (Pintado et al., 1997).

Paesani et al. have developed a similar questionnaire, adding the field "Don't know" to avoid false positive or false negative answers (Paesani et al., 2010). The questionnaire by Paesani et al. is shown in *Figure 5*.



Yes	No	Don't know	
			Do you grind your teeth when you sleep?
			Has anybody heard you grind your teeth while you sleep?
			On waking up, do you usually find that you are clenching your teeth?
			When you wake up, do you usually have jaw pain or jaw fatigue?
			When you wake up, do you usually have the feeling that your teeth are loose?
			When you wake up, do you usually have sore teeth and/or sore gums?
			When you wake up, do you usually have a headache in the temples?
			When you wake up, do you usually have a jaw lock?
			Have you ever found that you were clenching your teeth in the daytime?
			Have you ever found that you were grinding your teeth in the daytime?

Figure 5: Questionnaire for detection of bruxism (Paesani et al., 2010).

Furthermore, there is the 12-item oral parafunctions questionnaire by Van der Meulen et al. which is a questionnaire with good statistical features, introduced to score bruxism in clinical practice and scientific research. The answers generated by this questionnaire can be categorized into three scales: the bruxism scale (BRUX), including clenching and grinding activities, the “bite” scale (BITE), including biting and chewing items, and the “soft tissues” scale (SOFT), including tongue, lip and cheek movements (Van der Meulen et al., 2006). These are a few examples of questionnaires meant to diagnose bruxism. However, there are many further such as the Oral Behavior Checklist (OBC), which will be discussed in detail in chapter 2.5.1, as it is an instrument of this study. Apart from bruxism questionnaires, there are questionnaires regarding the detection of temporomandibular joint disorders, e.g., the TMD Pain Screener, presented in chapter 2.3, also used in this study.

The information obtained is retrospective at a single observation point, making it difficult to judge the frequency of bruxism behavior over the selected timespan. The resulting generic answers can also be biased due to reporting errors (Manfredini et al., 2013; Stone et al., 1994). Studies have shown a statistically significant association between self-reported bruxism and psychological states (i.e., anxiety and stress). These studies conclude that self-report reflects distress rather than representing a valid scale for bruxism (Ahlberg et al., 2004; Lobbezoo et al., 2013; Lobbezoo et al., 2018; Pintado et al., 1997; Raphael et al., 2015; Winocur et al., 2011).

Furthermore, many patients are unconscious about their bruxism habits when their dentist reports extensive tooth wear. Implicating the presence of bruxism during a clinical inspection, in turn, induces self-report due to the dentist's report, resulting in the tendency to overestimate the true extent of bruxism activity (Lobbezoo et al., 2018; Peroz et al., 2019). The bruxism episodes, particularly the clenching type, of approximately 80% of patients are not accompanied by noise. This fact is not concordant with the patients' knowledge of bruxism, resulting in underestimating the bruxism activity (Koyano et al., 2008).

The diagnosis generated through self-report has shown a lack of concordance with validated instrumental diagnosis (Raphael et al., 2015). Considering all the aspects and difficulties mentioned above in assessing bruxism solely via self-report, it becomes clearer why this assessment tool only indicates a "possible" diagnosis (Lobbezoo et al., 2018; Prasad et al., 2021).

Clinical examination (intraoral – extraoral) (probable bruxism):

In 2020 Manfredini et al. have accomplished releasing the first draft of a bruxism evaluation system, which will consequently lead to the definition of a Standardized Tool for the Assessment of Bruxism (STAB). It will ultimately allow clinicians and researchers to modulate the assessment of bruxism patients concerning the clinical impact of the different bruxism activities and etiologies (Manfredini et al., 2020). Axis A of STAB deals with assessing bruxism/ masticatory muscle activity, including self-reports, clinical evaluation (signs/symptoms/consequences), and instrumental assessment. Clinical indicators of both awake and sleep bruxism are outlined in *Figure 6*.

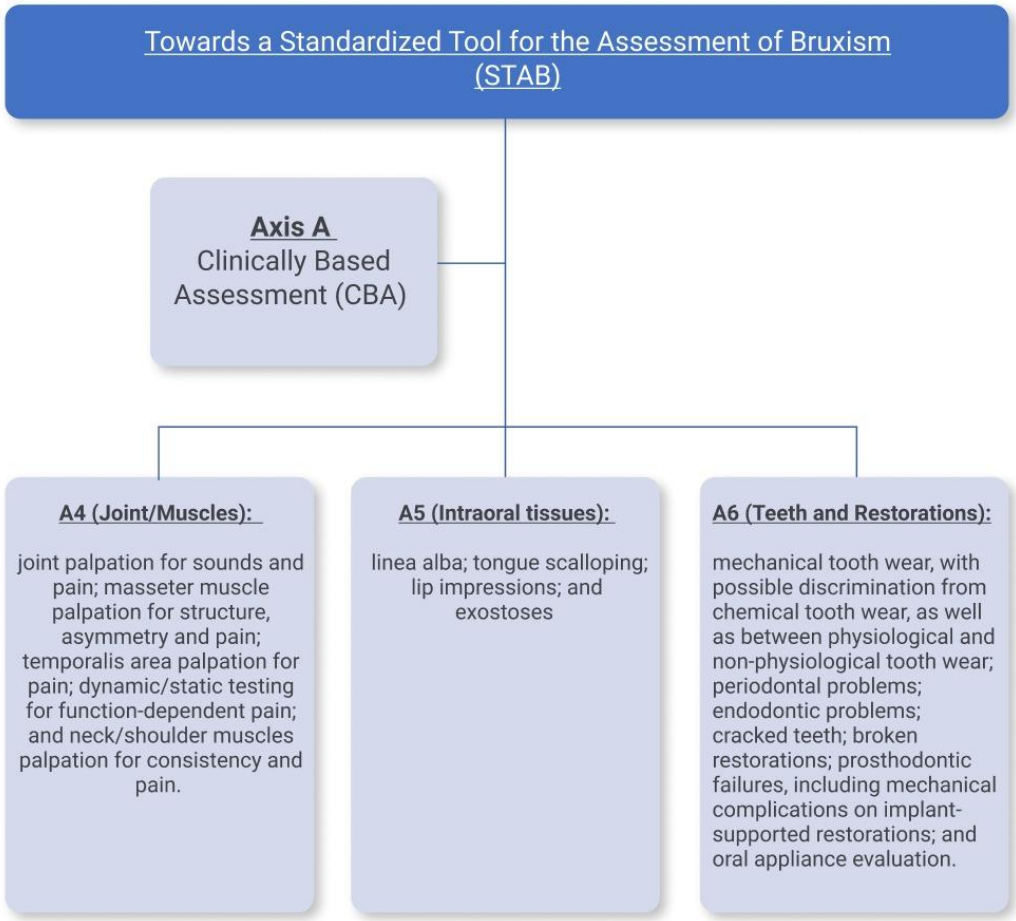


Figure 6: STAB (Manfredini et al., 2020).

Although the use of clinical symptoms for a bruxism diagnosis is still common, they shall be interpreted with caution. These symptoms are not only specific symptoms of bruxism but can also indicate multiple differential diagnoses or even be attributable to mere physiological activities. For example, the linea alba along the biting plane can further result from physiological swallowing (Castroflorio et al., 2015; Yap et al., 2016). Furthermore, a study by Rompre et al. even suggests that individuals with low frequencies of orofacial activities are more at risk of reporting pain than higher-frequency sleep bruxers (Rompre et al., 2007). The clinical symptom “extensive tooth wear (attrition)” may be mainly a marker for sleep bruxism. Nevertheless, it is diagnostically less informative as it does not exclude former bruxism without current activity and is less specific due to its cumulative nature and multiple differential diagnoses (Lobbezoo et al., 2018).

Interindividual differences in enamel density, saliva quantity and quality, and lubricating efficacy affect the magnitude of attrition, making standardization difficult (Gupta et al., 2017). Over the years, there have been several approaches to quantify and objectify occlusal and incisal tooth wear, for example, the all-clinical indices developed by Smith and Knight and by Hugoson et al., which are both imprecise in terms of unequivocal and standardized use. The Smith and Knight Index, for instance, includes the need to determine whether the exposition of dentin is secondary to bruxism, while the Hugoson Index uses terms like “negligible wear” and “obvious wear,” which makes a precise estimation difficult (Lobbezoo et al., 2001a). A further method is an indirect approach by grading dental casts. Studies have shown that grading occlusal/incisal wear on dental casts is more reliable than grading nonocclusal/ nonincisal wear. However, the indirect method is still inferior to the clinical assessment of tooth wear, as it has proven to be less reliable. The difficulty of assessing dental casts lies in the identification of dentin exposure (Lobbezoo et al., 2001a; Wetselaar et al., 2009).

All in all, the field of self-report and clinical inspection is still lacking standardized quantification methods. Therefore, clinical symptoms should not be considered separately for a “probable” bruxism diagnosis but rather by evaluating the overall clinical picture.

1.1.6.2. Instrumental assessment (definite bruxism)

Since the definition of bruxism describes it as a masticatory muscle activity (Lobbezoo et al., 2018), the best way to diagnose it would be to measure this muscle activity. The acquisition of reliable data of muscle activity requires the use of electromyography (EMG) in terms of a neuromuscular functional analysis (Hugger et al., 2008; Lobbezoo et al., 2018). Surface EMG in the dental field is a suitable tool for the graphic recording of the accessible masticatory neuromuscular function analysis (Preston, 1987). The temporalis and masseter muscles are relatively close to the skin surface and are therefore preferably utilized (Hugger et al., 2008). It can provide information about resting activity, maximum muscle activation, frequency spectrum under various applications of force, and comparison of bilateral symmetry during contraction, which consequently can be visualized on a screen (Hugger et al., 2008).

The origin of a neurological stimulation lies in the cortex (premotor cortex, the supplementary motor area, and others), which excites or inhibits various neurons of the primary motor cortex. The signal (as action potentials) is consequently forwarded to the spinal cord to directly influence alpha motoneurons (Merletti et al., 2004). The muscle fibers innervated by a single alpha-motoneuron form one motor unit, the smallest functional unit of a muscle (*Figure 7*) (Konrad, 2005). When muscle cells are neurologically stimulated, measurable electrical activity results. EMG is the recording of this electrical activity, generated by many muscle fibers in a muscle.

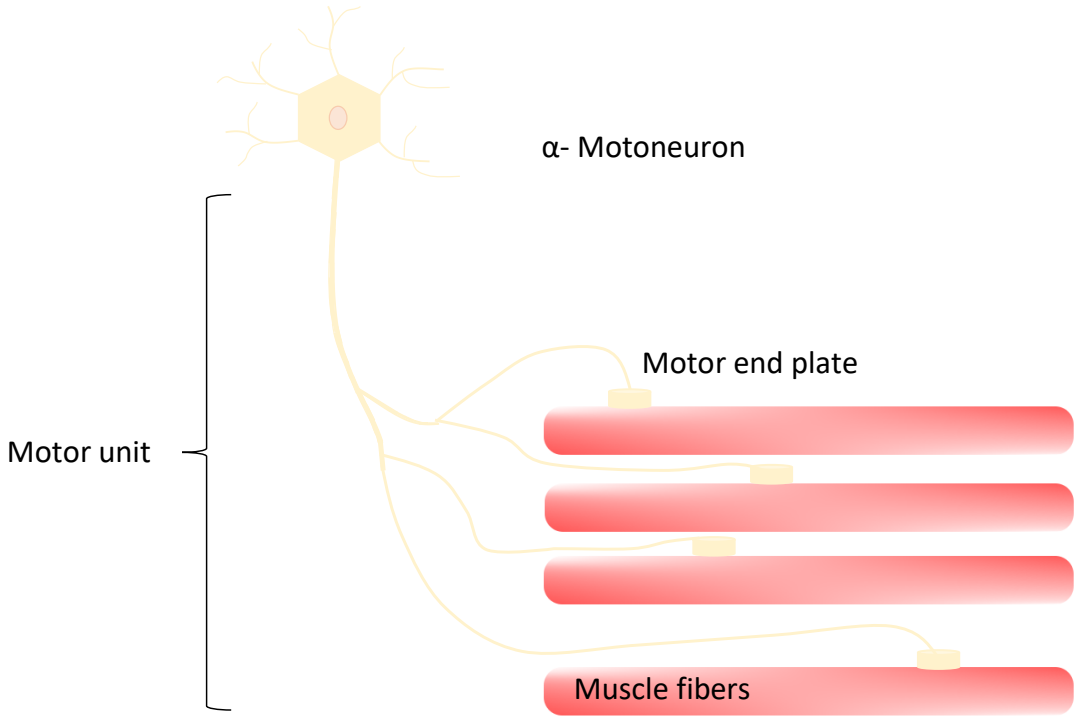


Figure 7: The composition of a motor unit (own illustration).

The depolarization – repolarization cycle of an action potential induces a depolarization wave, which travels along the surface of a muscle fiber. Transmitted action potentials innervate the motor units with a conduction velocity between 1 and 100 m/s (Hugger et al., 2008).

This transmitted signal can be registered through the use of two electrodes due to the underlying potential difference between them, induced by the depolarization wave (Konrad, 2005). The amplitude and form of the electrical activity depend on several variables, such as the conductivity of the tissue, the distance between the electrodes, as well as their distance from the source. External electromagnetic noise from various sources can modify the required signal. A differential amplifier can remove these alterations by forming the difference between the two signals since both electrodes simultaneously register noise. The signal acquisition process involves intramuscular needles, wires, or external surface electrodes. Furthermore, a distinction is made between unipolar and bipolar recordings. During the potential measurement in a unipolar recording, the actual measuring electrode is placed in or near the excited muscle, while the reference electrode lies in a region not influenced by the signal. In bipolar recordings, both electrodes are located with distance on the excited muscle; hence, the potential difference between these two electrodes is measured.

Figure 8 illustrates the depolarized membrane of a muscle fiber (1) and the EMG electrodes adhered to the skin, which measure the potential difference between them (2).

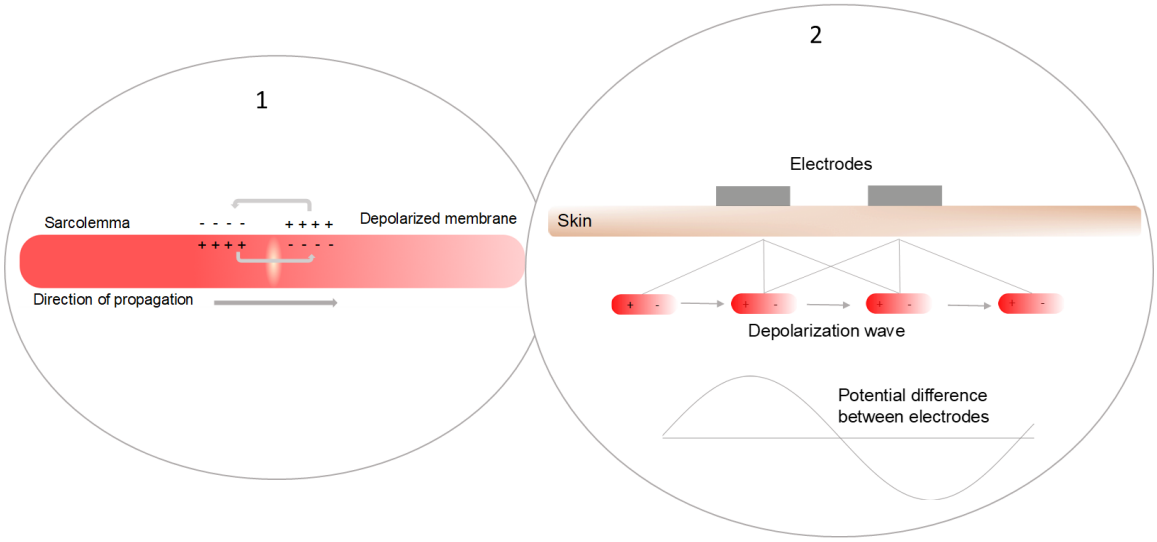


Figure 8: (1) Depolarization wave traveling along the surface of a muscle fiber, (2) EMG signal acquisition with two electrodes, the depolarization induces a potential difference between the electrodes. Modified from (Konrad, 2005).

As mentioned above, the muscle consists of motor units with numerous muscle fibers at different distances to the sensor location. The resulting signal from each muscle fiber reaches the sensor with low runtime differences, according to their distance from the sensor location. Furthermore, the part of the muscle closest to the measuring electrode influences the signal the most. Each muscle contraction is characterized by the activation of many motor units innervated at different times, in different frequencies, and different geometrical fiber orientations in ratio to the electrode site. Typically, all motor unit action potentials (MUAP) of all active motor units detectable are electrically superposed, with equal positive and negative amplitudes distribution, as paradigmatically shown in *Figure 9*. These physiological aspects during the excitation propagation are the reason for the so-called interference patterns generated in the surface EMG signal. The electrical activity and contraction strength are directly proportional to the number of activated motor units (Hugger et al., 2008; Konrad, 2005).

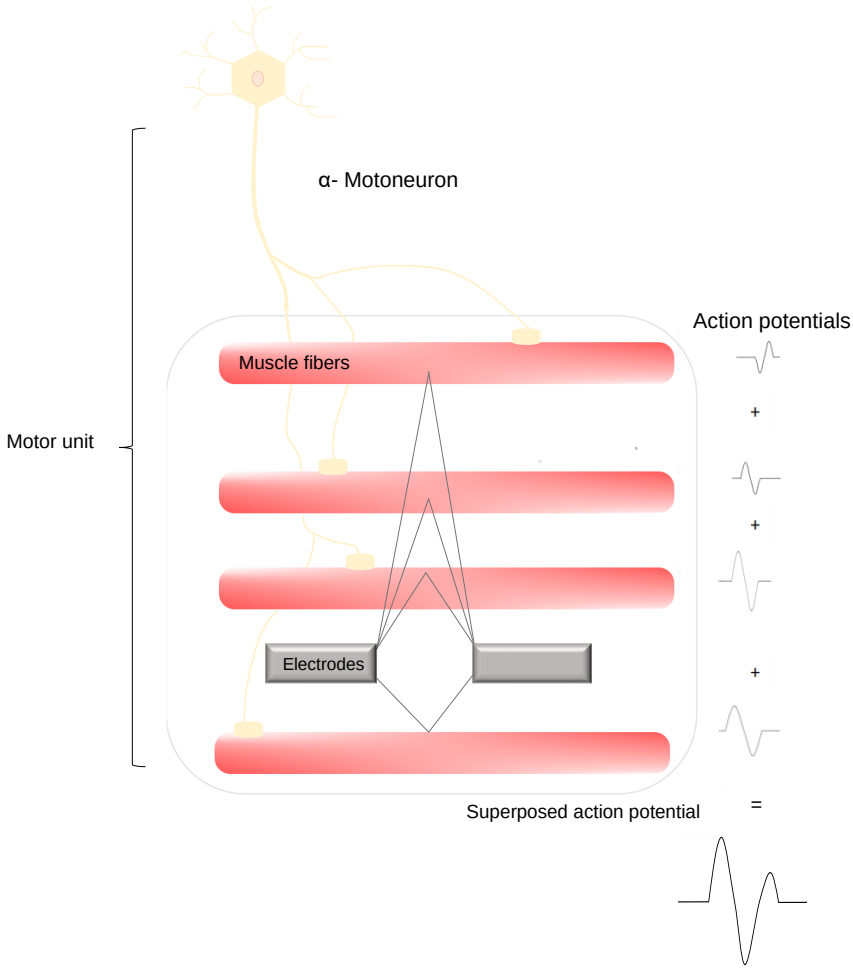


Figure 9: Interference pattern and superposition of action potentials of a few muscle fibers. Modified from (Konrad, 2005).

Sleep bruxism – Polysomnography (PSG):

The instrumental SB assessment approach, i.e., polysomnography (PSG), is the scientific gold standard for a definite and unequivocal sleep bruxism diagnosis. During a PSG in a sleep laboratory or ambulatory different variables (i.e., EMG of the masticatory muscles, electroencephalography [EEG], electrocardiography [ECG], electrooculography [EOG], and saturation of oxygen) can be measured simultaneously. Combining these parameters in a sleep clinic with audio and video signals enables a differentiation between SB and other oral activities during sleep (such as yawning, sleep talking, and chewing), which potentially falsify the results. By use of this method, it is possible to record RMMA episodes, which are representative of bruxism (Peroz et al., 2019; Raphael et al., 2012; Zucconi et al., 2014). PSG is the method highest in accuracy and most valid when it comes to scoring RMMA episodes (Lavigne et al., 1996). Nevertheless, it is rather difficult to conduct, expensive, time-consuming and might influence results because of foreign sleep environment. Due to these complexities it is currently rather used for research purposes, examining small sample sizes (Ommerborn et al., 2015; Raphael et al., 2012), and not routinely applicable in everyday practice.

Awake bruxism - Ecological momentary assessment (EMA)*:*

The measurement of MMA during wakefulness through long-term EMG faces challenges considering the technical application and depends on good patient compliance. Therefore, the introduction of ecological momentary assessment (EMA) emerged as an interesting assessment option aiming to overcome the difficulties of EMG implementation while gaining a “definite” awake bruxism diagnosis (Zani et al., 2019). EMA requires patients to report about the variable under investigation (viz. bruxism behavior) close in time to experience by answering questions several times a day, thus offering the advantage of minimizing bias due to the natural environment (Manfredini et al., 2013; Stone et al., 1994). The usefulness of EMA can be increased by adding a dedicated smartphone application, such as BruxApp® by Manfredini et al. (BruxApp®, BruxApp Team, Pontedera, Italy). The App provides preprogrammed and randomly generated auditory signals. Upon alert receipt, the user must answer in real-time by tapping on the combo box, which refers to the current condition of the jaw muscles or teeth position “relaxed jaw muscles, tooth contact, tooth clenching, tooth grinding or jaw clenching (without tooth contact [i.e., mandible bracing]).

These conditions were selected as they are part of the AB spectrum (Bracci et al., 2018; Manfredini et al., 2016a). The EMA increases the representativeness of collected data and enables a generalization of the real-life of an individual. Nonetheless, current data cannot completely rule out possible self-awareness effects through reminding the patient multiple times a day to report jaw habits, leading to higher reported average frequencies (Shiffman et al., 2008; Zani et al., 2019). Although EMA gathers data multiple times a day, it depends on the precise timing of assessments and participants' compliance (Prasad et al., 2021).

AB and SB; Ambulatory EMG:

To date, the importance of EMG for scoring RMMA remains incontestable as it provides objective and reproducible data on the frequency, duration, and intensity of muscle contractions (Prasad et al., 2021). The introduction of portable EMG devices has enabled an objective, valid and handier method for bruxism activity measurement, while overcoming the difficulties in assessing AB and SB at home. Several EMG devices have been fabricated with the ability to examine uni- or bilateral MMA during sleep and wakefulness. Some are even combinable with measures of heart rate and additional parameters. Examples of portable EMG devices are the device of Gallo, BruxOff® (Bioelettronica, Italy), GrindCare® (BUTLER®, Sunstar Suisse S.A., Etoy, Switzerland) and BiteStrip® (Alldent, Australia), which solely detect SB, and the device from Prasad which detects AB. Prasad's device is a smartphone-assisted wireless EMG device, recording up to eight hours of muscle activity with a single surface electrode. The smartphone-assisted device has shown good results when compared to the fixed-wired EMG, although it needs further investigation since the sample size was small which resulted in a low power for the different tests (Prasad et al., 2019). Currently, only BiteStrip®, BruxOff®, and GrindCare® affirm high validity compared with PSG recordings while not disrupting the sleep quality (Jadidi et al., 2011; Manfredini et al., 2014; Stuginski-Barbosa et al., 2016). BruxOff® unites the EMG, with two disposable electrodes, and ECG, with three ECG electrodes. It is a bilateral measurement of masseter activity, which also makes it unique. BiteStrip® is a disposable EMG with the capacity of monitoring the single-night, unilateral masseteric activity for 5 hours which can be a major disadvantage due to the fluctuating nature of bruxism.

GrindCare® (GC) is superior in that respect, as it operates as a long-term EMG, measuring the temporalis muscle activity unilaterally, while also offering a stimulation mode to inhibit bruxism activity (Giannakopoulos et al., 2013). GrindCare® is a single-channel self-contained EMG device able to make a definite bruxism diagnosis (Dreyer et al., 2015), as it provides the same EMG information as an EMG of the masseter muscle during sleep (Koyano et al., 2008; Stuginski-Barbosa et al., 2016). GC enables the recording of EMG activity, their quantification, and computing. The following *Figure 10* displays the adhesion of the GrindCare® on the temporalis muscle (1). When an action potential depolarizes the muscle fiber, a potential difference is induced (2). The electrodes of the GC acquire the potential difference between them (3).

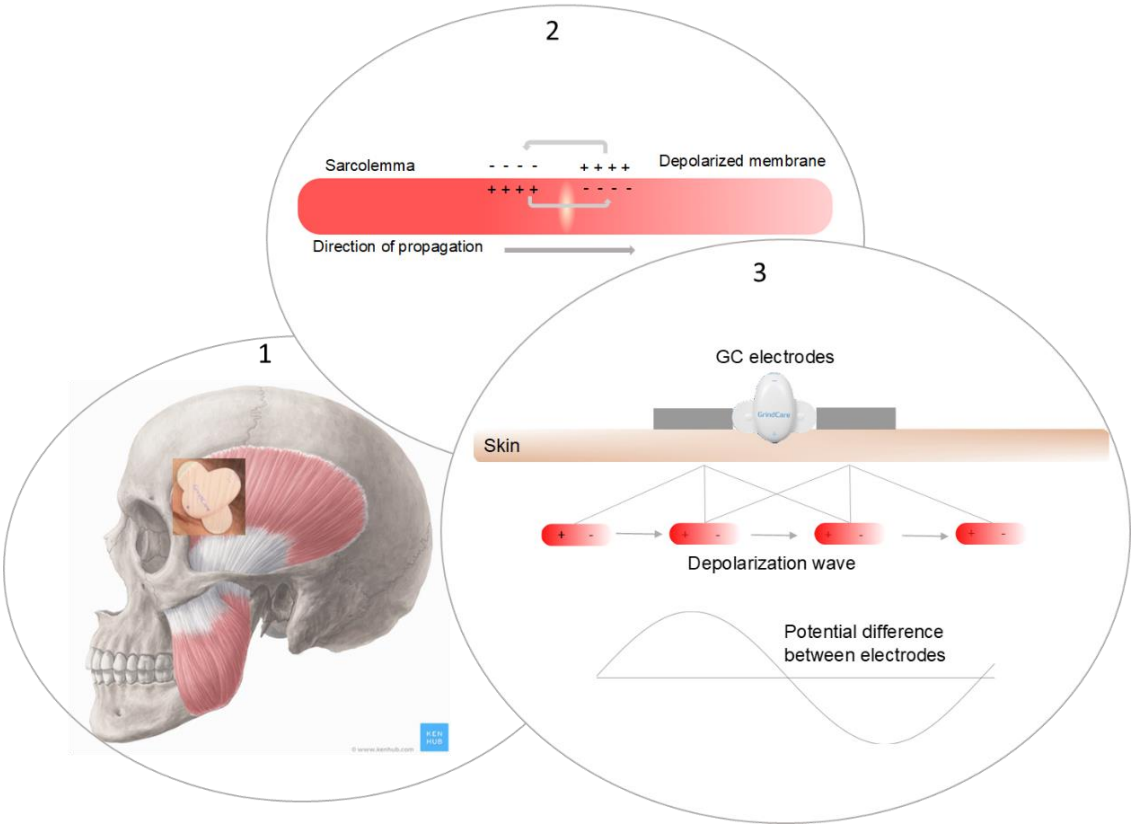


Figure 10: Adhesion of the GrindCare® on the temporalis muscle (1), depolarization wave traveling along the surface of a muscle fiber (2), EMG signal acquisition with the GrindCare® device inducing a potential difference between the electrodes (3).
1: Adopted and modified from © Kenhub (www.kenhub.com); Illustrator: Yousun Koh; 2 & 3: modified from (Konrad, 2005).

Each of these devices has an algorithm that recognizes the EMG amplitude and decides whether the activity is physiological, or bruxism related, either according to a predefined or an adapting (moving average) threshold. The most popular cut-off quantification criteria of RMMA for SB were defined by Lavigne et al. 1996 (Dreyer et al., 2015; Peroz et al., 2019) and are presented in *Table 7.*

Table 7: Cut-off criteria for a definite bruxism diagnosis by Lavigne et al. and their appearance in EMG (Murali et al., 2015), (Lavigne et al., 1996; Murali et al., 2015).

Cut-off criteria	
1.	**RMMA/h – Diagnosis via PSG by Lavigne et al. (Lavigne et al., 1996):**
	• > 4 bruxism episodes / h • > 6 bruxism bursts / episode • And/or ≥ 25 bruxism bursts / h of sleep • And > 2 episodes with grinding sounds
2.	**RMMA in EMG can occur as following patterns (Murali et al., 2015):**
	• Non rhythmic (tonic) = muscle contraction > 2 sec. • Rhythmic (phasic) = short, repetitive contractions of the masticatory muscles, with more than three muscle activities in the EMG with a duration of > 0.25 < 2 sec. • Combination of both

The term “bursts” signifies amplitude spikes in the EMG, usually defined as having at least twice the amplitude of baseline. Intermission in-between these spikes should be less than 2 seconds for the activities to count as a spike, while intermission of more than 3 seconds differentiates two amplitude spikes.
Bruxism episodes are defined as more than six amplitude spikes, which usually occur in a rhythmic pattern and are therefore called rhythmic masticatory muscle activity (Peroz et al., 2019). These cut-off criteria enable a bruxism diagnosis according to the collected EMG data. A reevaluation of the RDC-SB in 2007 graded the severity of bruxism into three categories, light bruxism (> 1 < 2 bruxism episodes/hour), moderate bruxism (> 2 < 4 bruxism episodes/hour) and severe bruxism (> 4 bruxism episodes/hour) (Rompre et al., 2007). However, there has been a lot of criticism over those criteria in the last years, due to the large variability in the thresholds defined to grade sleep bruxism events.

This circumstance results in challenges when comparing studies as it lacks in standardization, while it may also lead to different sleep bruxism diagnosis (Thymi et al., 2021). To date, the sleep bruxism outcome variables include RMMA, frequency, duration, and intensity of MMA, and a combination of two or more parameters. Thymi et al. created an overview of the thresholds applied to score sleep bruxism events in their scoping review from 2021, which is displayed in *Table 8* (Thymi et al., 2021).

Table 8 : "Cut-off values and grading criteria for defining sleep bruxers" (Thymi et al., 2021). Reprinted from "Signal acquisition and analysis of ambulatory electromyographic recordings for the assessment of sleep bruxism: A scoping review", Vol: 48, Peter Svensson, Gilles Lavigne, Daniele Manfredini et al., Copyright (2021), with friendly permission by John Wiley & Sons Inc.

	Outcome	*n*	First author & year
Cut-off	>2 episodes/h	1	Camara-Souza 2018
	≥2 episodes/h	2	Murakami 2014, Schmitter 2015
	>4 episodes/h	3	Castroflorio 2015, Manfredini 2016, Mude 2017
	>25 events/h	1	Takaoka 2017
	SB/research criteria	2	Ono 2008, Suganuma 2007
	5.5 EMG-episode/h, 32.2 EMG-burst-all/h and 26.4 EMG-burst-5%/h	1	Maeda 2019
	18 EMG/h or higher in three consecutive nights and 19 EMG/h or higher in five consecutive nights	1	Stuginski-Barbosa 2015
Cut-off and grading	>2 episodes/h for moderate and >4 episodes/h for intense/severe sleep bruxism	2	Ohlman 2018 & 2020
	0 = <40 events; 1 = 40–74 events; 2 = 75–124 events; and 3 = ≥125 events (0–2: non-severe SB, score 3: severe SB)	1	Nagamatsu-Sakaguchi 2017
	0 = <40 events; 1 = 40–74 events; 2 = 75–124 events; and 3 = ≥125 events	2	Saueressig 2010
	0 = <30 events, 1 = 31–60 events, 2 = 61–100 events and 3 = ≥100 events	3	Carvalho Bortoletto 2016, Karakoulaki 205, Minakuchi 2012
	0 = <30 events, 1 = 31–60 events, 2 = 61–100 events and 3 = ≥100 events (0–1 normal controls, 2–3 severe SB)	1	Minakuchi 2014
	0 = no bruxism (≤39 episodes), 1 = mild bruxism (40–74 episodes), 2 = moderate bruxism (75–124 episodes) and 3 = severe bruxism (≥125 episodes)	3	Ahlberg 2008, Mainieri 2012, Palinkas 2019
	SB frequency score in four grades (0, 1, 2 and 3)	1	Minakuchi 2016

Abbreviations: EMG, electromyographic, h, hour, *n*, number of studies, SB, sleep bruxism.

Although it is widely accepted that RMMA represent sleep bruxism events in the EMG (derived from the polysomnographic audio-video recordings), current research shifts from scoring RMMA to scoring the wide spectrum of jaw motor behaviors (MMA) instead, which also include physiological oro-facial activities (yawning, swallowing, coughing) and oro-motor activities (MMAs resulting from major movements, such as head, neck and body movements), as this technique seems more representative (Thymi et al., 2021).

This is due to the shift towards the importance of clinical usefulness (Manfredini et al., 2019; Manfredini et al., 2020). Negative clinical health outcomes can be related to RMMA but are not limited to them (Thymi et al., 2021). Although this topic still requires further investigation it can already be concluded that aside from RMMA, further MMA variables can be important, such as background EMG activity, intensity and timing, amplitude of activity, and variability of activity over time. These variables seem to alter musculoskeletal signs and symptoms, although the extent remains unclear (Baad-Hansen et al., 2019). Current research assumes that different sleep bruxism variables are associated with different clinical outcomes, as they are different expressions of muscle work (Manfredini et al., 2019; Manfredini et al., 2020). The authors speculatively suggest that the clinical outcome “masticatory muscle pain” may be related to frequency and duration of MMA while duration and intensity are more relevant when investigating tooth wear and failures of dental restorations (Thymi et al., 2021). Hence, the scoping review has postulated two recommendations.

Firstly, it is not sufficient to define cut-off values which theoretically confirm the presence (or absence) of bruxism, as for some individuals bruxism has no clinical impact (Raphael et al., 2016). It is recommended to define a suitable threshold for the broad spectrum of MMA in association to the increased (or reduced) probability of any health outcome variables and diagnose clinically relevant bruxism (Manfredini et al., 2019; Thymi et al., 2021).

Secondly, the devices should be classified according to the type of MMA outcomes they are able to assess. It is further recommended to choose the MMA variables based on the assessed health outcomes (Thymi et al., 2021) . Furthermore, experimental bruxism studies are encouraged to also include individuals with musculoskeletal symptoms, somatization, depression, fear of movement since they might react differently to mechanical loading or might be influenced in the way they experience symptoms (Baad-Hansen et al., 2019).

Particularly in dental research there has been a trend towards the use of ambulatory EMGs, since they are more convenient in large sample sizes while maintaining high validity. However, as discussed above there is yet a lack of information regarding cut-off criteria of these ambulatory EMG devices. Therefore, there is still a large demand on more research regarding EMG parameters and thresholds.

Sleep bruxism - Splints:

Finally, a further tool to diagnose sleep bruxism is by using occlusal splints (such as the Bruxcore Bruxism Monitoring Device (BBMD) with differently colored surface layers and computer analysis of the results. Through abrasion caused by tooth contacts different colors are exposed, depending on the degree of abrasion. The two variables “abraded area” and “abraded depth” are used to quantify the severity of bruxism activity, thus, affirming the diagnosis of bruxism (Ommerborn et al., 2015). The studies on these devices emphasize their objectivity and their practical use in clinical practice. Another form of a diagnostic occlusal splint is the BruxChecker from Sato et al., which has a similar visualization scheme by abrasion of the material through MMA. BruxChecker even suggests that qualitatively classifying and differentiating the dynamic occlusal schemes on the splints enables the development of a precise and personalized treatment plan for each patient based on current bruxism patterns. This hypothesis, however, still requires further investigation (Greven et al., 2015). Despite the thinness of these devices, they still interfere with the vertical dimension of occlusion; hence, they might change neuromuscular mechanisms. Additionally, it registers only the grinding type of bruxism, excluding the clenching form (Peroz et al., 2019; Terebesi et al., 2016).

1.1.7. Management of bruxism

Given the multifactorial etiology of bruxism, there is no definite treatment for bruxers at risk. The current approach rather aims to manage the symptoms in acute phases, especially acute pain, mechanical tooth wear, and dental restoration wear (Macedo et al., 2007; Yap et al., 2016). The management methods entail behavioral approaches (i.e., condition counseling, cognitive behavioral therapy, biofeedback, the Radboud tooth wear project), physical therapy, an occlusal approach, a pharmacological approach, and contingent electrical stimulation (CES).

1.1.7.1. Behavioral approach

Prior to complex treatment approaches, the essential strategy in “treating” bruxism is patient’s counseling. It is significant to start with a comprehensive explanation of bruxism being a centrally regulated behavior and not a harmful parafunction that requires treatment. Treatment modalities shall consequently be discussed with the patient if there is still a need for an intervention. While discussing with the patient, it is crucial to ensure the information transferred is comprehensible, not using medical language with abstract terms.

The first step in management starts with an adequate self-perception of the frequency and the timing of the clenching/ grinding or thrusting/ bracing behavior during wakefulness to deliberately counteract by conscious interruption of the behavior (Peroz et al., 2019). The second step recommends counseling the patient regarding sleep hygiene measures (quitting coffee, alcohol, and smoking, especially before sleeping). Even though there is no evidence in the efficiency of this method nor a benefit for sleep quality, authors suggest that it is important to recommend good sleep hygiene for patients, considering the fact that coffee, alcohol, and tobacco are still risk factors for bruxism (Valiente Lopez et al., 2015). While coffee is a CNS stimulant which induces a more superficial sleep (Victor et al., 2017), the other two psychoactive substances alter glutamate synaptic transmission and consequently increase dopamine release, which could be strongly related to bruxism (De la Hoz-Aizpurua et al., 2011; Guaita et al., 2016; Murali et al., 2015; Rintakoski et al., 2010; Valiente Lopez et al., 2015).

Other behavioral approaches comprise hypnotherapy, cognitive behavioral therapy, progressive relaxation management, and meditation. All behavioral treatment techniques mentioned above lack a solid scientific basis and are therefore questionable. More evidence is needed in order to properly assess these approaches (De la Hoz-Aizpurua et al., 2011; Guaita et al., 2016; Lobbezoo et al., 2008; Valiente Lopez et al., 2015; Yap et al., 2016).

EMG-Biofeedback:

According to the principle of "aversive conditioning", biofeedback is based on the concept that bruxers can abandon their detrimental jaw muscle activity when a stimulus makes them aware of it. This technique can be used for both awake and sleep bruxism. During wakefulness, patients' consciousness can be addressed via visual and auditory signals.

For sleep bruxism, auditory, electrical, vibratory, and even taste stimuli can be used for feedback to address the patient in a subconscious manner (Lobbezoo et al., 2008; Murali et al., 2015; Shetty et al., 2010).

- Biofeedback on patients with awake bruxism: Although findings support the efficacy of biofeedback in terms of management of awake bruxism, by reducing masticatory muscle activity, authors of literature reviews remain reserved as there are no long-term results and the sample size was small (Jokubauskas et al., 2018; Lobbezoo et al., 2008; Manfredini et al., 2015b; Shetty et al., 2010; Treacy, 1999).
- Biofeedback on patients with sleep bruxism: In most cases, the intervention with diverse stimuli resulted in a positive sleep bruxism reduction. Nevertheless, all effects were transient, and some resulted in frequent arousals, which may lead to severe side effects (i.e., excessive daytime sleepiness). Biofeedback is therefore not generally applicable as a management modality (Jokubauskas et al., 2018; Lobbezoo et al., 2008; Manfredini et al., 2015b; Sato et al., 2015; Shetty et al., 2010; Treacy, 1999).

The Radboud tooth wear project:

The Radboud tooth wear project was designed in the Netherlands for management of severe tooth wear patients. It recommends that even patients with non-progressive severe tooth wear without any complaints are not in need of restorative treatment, but rather require counselling and monitoring. However, if the tooth wear is classified as progressive there are two further options. Firstly, counselling and monitoring while establishing the causing factors and starting the prevention plan, while dental casts (stone or digital) should be used for reference. Secondly, when the patient has complaints or wants to improve his/her appearance minimally invasive and adhesive restorations are preferred especially for young people when increasing the vertical dimension.

Direct composites are superior to “definitive” treatment modalities as the definitive method may have a limited durability. However, a prevention of the wear process cannot be achieved regardless of the type of restorative material. It can merely modify the rate, location, and nature of the tooth wear (Loomans et al., 2018).

1.1.7.2. Physical therapy approach

A further intervention method is physical therapy, aiming to alleviate the sequelae of bruxism, i.e., reducing pain-related secondary symptoms or addressing muscle tension. Even though evidence in this field is deficient, few studies suggest that training for the jaw opening muscles, muscular awareness relaxation training (MART), and transcutaneous electrical nerve stimulation (TENS) might positively affect the reduction of sleep bruxism. While not all of these methods show effects in EMG (Gomes et al., 2014; Lobbezoo et al., 2008) they still provide significant benefits in terms of muscle function and pain relief (De la Hoz-Aizpurua et al., 2011; Gomes et al., 2014; Treacy, 1999).

1.1.7.3. Occlusal approach

Occlusal therapy can be divided into two categories:

1) "invasive" occlusal intervention (irreversible treatment) and
2) occlusal appliances (reversible treatment).

Invasive occlusal intervention employing selective occlusal adjustment, occlusal rehabilitation, or orthodontic treatment aims for a harmonic occlusal equilibrium. Various materials like dental composite resin, ceramic, and metal or orthodontic appliances can be used to achieve selective adjustments of occlusal tooth contacts. This technique is highly questionable, not only because it is irreversible but also because it presumes a direct correlation between occlusal factors and the occurrence of bruxism. One case report using buccal separators to evaluate its effectiveness in reducing bruxism activity showed no significant difference between pre- and post-treatment in EMG evaluation (Abraham et al., 1992). Current research does not support these treatment methods due to the lack (or subordinate) etiological connection between dental morphological factors and bruxism (De la Hoz-Aizpurua et al., 2011).

Occlusal appliances (OA), e.g., occlusal splint therapy, is a reversible intervention covering the dental arch of only one or both jaws and have been the most commonly applied tools for the management of bruxism. Their effect was anticipated to reduce sleep bruxism activity, consequently reducing the bruxism-associated symptoms, particularly pain in the face, head and neck, abnormal tooth wear, and excessive tooth mobility.

Notwithstanding the ineffectiveness of OA to stop bruxism altogether (Holmgren et al., 1993), a significant decrease of the masseter and anterior temporalis muscles EMG activity, frequency, and intensity of RMMA was observed in short-term observation periods (Dube et al., 2004; Madani et al., 2013; Stapelmann et al., 2008). Therefore, OA have proven to be highly efficient in protecting teeth and restorations from wear (Macedo et al., 2007), reducing headache, tooth mobility, myofascial, joint, and neck pain (Amorim et al., 2012; Dube et al., 2004; Holmgren et al., 1993; Stapelmann et al., 2008).

Furthermore, the literature review in 2015 by Manfredini et al. reports that every type of splint therapy has shown a positive effect in reducing sleep bruxism (Manfredini et al., 2015b). One possible explanation for sleep bruxism reduction is due to a change of the vertical dimension of occlusion when inserting an occlusal splint. Minor changes in vertical jaw relations consequently change the recruitment of masseter muscle motor units (Terebesi et al., 2016). They hypothesize a “novelty effect” due to a transient reorganization of motor unit recruitment. This thesis is supported by a clinical trial with an intermittent use of splints, which has shown to be more efficient than a consequent use and resulted in a significant reduction of muscle activity (Matsumoto et al., 2015). A further observation is that OA, constructed with a high mandible advancement, were highly effective in reducing SB (Huynh et al., 2007). Such observations could be explained with reduced contractile units when the mandible is advanced (Woda et al., 2001) or due to the elimination of the masticatory muscle activity, that is part of apnea-induced arousal (Manfredini et al., 2015a; Manfredini et al., 2015b). Nevertheless, some studies report an adaption process after a certain period, as decreased EMG parameters were found to be transient (Peroz et al., 2019; van der Zaag et al., 2005).

Therefore, it seems most likely that the therapeutic mechanism of splints is related to factors that modify and reduce parafunctional activity and redistribute its overload on the dental arches instead of eliminating bruxism entirely (Holmgren et al., 1993). As the effect of occlusal splints remains partially controversial, bruxism experts do not generally recommend OA for all bruxism patients (van der Zaag et al., 2005). In combination with other treatment methods, however, OA remain an excellent tool for bruxism management.

1.1.7.4. Pharmacological approach

While many drugs have been tested regarding the efficacy in diminishing MMA, only clonidine, L-dopa, and clonazepam have shown a reduction of SB in clinical trials (Huynh et al., 2007; Lobbezoo et al., 1997; Saletu et al., 2010). Nevertheless, the regular and long-term use of these medications (Benzodiazepines, antidepressants) is restricted since most of them cause also fatigue and entail the risk of addiction (benzodiazepine) and hypotension (clonidine) (De la Hoz-Aizpurua et al., 2011; Huynh et al., 2007; Manfredini et al., 2015b; Winocur et al., 2003; Yap et al., 2016).

Another pharmacological approach is the local administration of botulinum toxin (BTX Type A) as a peripheral cholinergic synapse-blocking agent, which induces motor weakness up to paralysis. Clinicians affirm the safety and efficacy of the off-label use, nevertheless, recommend it only for patients with severe bruxism, associated pain, masseter muscle hypertrophy, and for those refractory to conventional management methods (Al-Wayli, 2017; De la Hoz-Aizpurua et al., 2011; Lee et al., 2010; Shim et al., 2014). Possible side effects were reported, such as speech disturbance, masticatory difficulty, muscle ache, prominent zygoma, and facial asymmetry due to masseter atrophy (Park et al., 2003; Yap et al., 2016).

1.1.7.5. Contingent electrical stimulation

The concept of contingent electrical stimulation was to find a diagnostic and intervention technique, high in efficiency and more convenient in its practicability than the gold-standard PSG. The purpose was to simplify and improve the diagnosis of bruxism for large populations in research and in clinical practice, while also introducing the simultaneous intervention technique for prevention.

One example of a CES device is GrindCare®, which provides a definite bruxism diagnosis and intervenes when masticatory muscle activity is high. Hence, GrindCare® has two modes of operation. The first mode is to measure the EMG activity of the anterior temporalis muscle (diagnostic mode, or inactive GC), and the second is to modulate the activity during sleep by use of CES (active GC) (Raphael et al., 2013; Stuginski-Barbosa et al., 2016). Led by high temporal muscle activity, these CES impulses are emitted, aiming for an inhibitory effect.

The principle of the devices is based on a direct reduction of RMMA, consequently reducing the sequelae of bruxism.

It is questionable whether these devices belong to the biofeedback approach per se, as they are not aiming for a conscious change of the activity but rather by reducing RMMA through reflex activation (Peroz et al., 2019). Conditioning this natural reflex when repeated frequently leads to a learning effect and thus manages bruxism causally (Desmedt et al., 1976; Godaux et al., 1975; Jadidi et al., 2008). Due to a reduction of SB-related motor activities, a beneficial effect was reported (Jadidi et al., 2008; Jokubauskas et al., 2018; Manfredini et al., 2015b; Needham et al., 2013), even though there is still a lack of information in terms of clinical evidence and long term efficiency. Several studies have shown that high intensities of CES result in a reduction of jaw-muscle soreness, tiredness, and unpleasantness, while jaw-muscle pain was not affected (Conti et al., 2014; Jadidi et al., 2013; Needham et al., 2013; Shimada et al., 2019).

As a result of the insufficient data, clinicians do not suggest GrindCare® as a single management option but rather recommend combining and gradually increasing the intervention methods starting with counseling. As other symptoms appear, i.e., extensive mechanical tooth wear, the use of occlusal splints can contribute to a positive outcome. When these attempts fail to reduce jaw muscle activity and the associated sequelae, GrindCare® is recommended to be added to the management protocol. In case all attempts fail, pharmacological strategies should be added as a last resort (Lobbezoo et al., 2019). However, the diagnostic function of GrindCare® seems to be a promising alternative to the gold standard PSG and comes in handy in clinical practice (Stuginski-Barbosa et al., 2016).

1.2. Aim and hypothesis

The following study sets out an approach to find electromyographical parameters relevant for a reliable and valid bruxism diagnosis. This basic idea will help improve current diagnostic techniques for primary care.

The main objective of this experimental clinical trial was to test whether subjects with different diagnoses and degrees of bruxism differ regarding various surface EMG parameters of the masticatory muscles, and whether changes in bruxism behavior, induced by CES intervention, affect those parameters. The hypothesis was that CES intervention influences EMG parameters, and after its' cessation all EMG parameters return to the initial status (exposure- response relationship).

2. Materials and methods

2.1. Ethics vote

The trial protocol was approved by the Ethics Committee of the Medical Faculty of the Würzburg University Hospital (ref. no: 226/17-sc) and was in conformity with the World Medical Association Declaration of Helsinki ("World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects," 2013). All subjects were precisely informed about the study protocol before the first session and individual questions were clarified. Participation in the study was voluntary and could be canceled anytime if a termination was required by the subject. The consent for participation, for photo-documentation, as well as for publication was additionally obtained in a written, and signed agreement. In order to maintain data security all subjects were given numbers by a randomization list and maintained these numbers throughout the entire study.

2.2. Study design

This study is a single-blind, prospective, experimental cohort study for basic research on sleep bruxism. In order to identify parameters which differ between different diagnoses and degrees of bruxism the study participants were divided into two groups, the control – and intervention group, according to a combined randomization and matching process. In accordance with the study protocol, these two groups were built and matched according to gender and age while aiming for an equal distribution of male to female participants. The groups were built by randomly assigning one study participant for the control group or to the intervention group and his/her matched counterpart to the other group, respectively. By using *https://www.randomizer.org/* two randomization lists were created. The first list randomized the distribution of the participants into the intervention – and control group, as described above. The second list was used to determine on which side of the face the EMG device should be applied, for bruxism diagnosis and control for each pair of participants. Each group was designed by convenience to have 20 participants who met the predefined eligibility criteria. The eligibility criteria were defined before the beginning of the study.

2.3. Study participants

The study sample was recruited via social media platforms (Facebook), friends, lists of former clinical trials of the Prosthodontic Department of the University of Würzburg, and by word-of-mouth recommendation. All subjects were selected randomly and by the time of the study they fulfilled the preset eligibility criteria, which are presented below.

Inclusion criteria:

Inclusion criteria for this study were: generally healthy male and female adult (18 years or older) participants, regardless of whether they assume to grind their teeth or not.

Exclusion criteria:

Exclusion criteria were pregnancy, breastfeeding, the report of any orofacial – head- or neck pain within the last 30 days, refusal of shaving off the beard for male participants, incomplete dental arch (except for the 3rd molars), age below 18 years, acute bruxism-related impairments, for which the subject would consult a dentist, and plaster allergies. Further exclusion criteria were the need for dental/ orthodontic treatment, and the intake of psychotropic drugs/ muscle relaxants.

For this, the following questions were additionally inquired in a general questionnaire created by the University of Würzburg, aiming to identify possibly unsuitable subjects: "Have you already completed, or are you currently undergoing orthodontic treatment? Do you take medications regularly?".

The absence of painful TMD was controlled with the TMD-pain screener, entailing the following questions: In the last 30 days, how long did any pain last in your jaw or temple area on either side? In the last 30 days, have you had pain or stiffness in your jaw on awakening? In the last 30 days, did the following activities change any pain (that is, make it better or make it worse) in your jaw or temple area on either side?". A positive outcome on the TMD pain screener were > 3 out of 7 possible scores. The figure below (*Figure 11)* shows the TMD-Pain Screener by Gonzalez et al. (Gonzalez et al., 2011), while the German version is attached in the Appendix *(Figure 46).*

TMD-PAIN SCREENER

1. In the last 30 days, how long did any pain last in your jaw or temple area on either side?
   a. No pain
   b. Pain comes and goes
   c. Pain is always present

2. In the last 30 days, have you had pain or stiffness in your jaw on awakening?
   a. No
   b. Yes

3. In the last 30 days, did the following activities change any pain (that is, make it better or make it worse) in your jaw or temple area on either side?

   A. Chewing hard or tough food
      a. No
      b. Yes

   B. Opening your mouth or moving your jaw forward or to the side
      a. No
      b. Yes

   C. Jaw habits such as holding teeth together, clenching, grinding, or chewing gum
      a. No
      b. Yes

   D. Other jaw activities such as talking, kissing, or yawning
      a. No
      b. Yes

Figure 11: TMD-Pain Screener (Gonzalez et al., 2011).

2.4. Outcome measures

2.4.1. Normalized EMG of the masticatory muscles

One disadvantage of EMG recordings is the influence of the data by the given detection condition, i.e., electrode positions, subjects, and day-to-day measures of the same muscle sites (Konrad, 2005). In order to quantitively compare several EMG measurements over several experiments or several participants, a constant and reproducible variable is needed. This variable is the EMG activity signal at maximal voluntary contraction (MVC), which enables normalizing the integrated EMG recording (Hosman et al., 1979).

The idea is to benefit from calibrating the arbitrary force to a reference which results in “percent of maximum innervation capacity”. This principle allows a quantitative comparison by rescaling the values from microvolt to percent of the reference value. The process does not change the shape of the EMG curve, but the scaling of the Y-axis and eliminates the influence of the given detection condition. Furthermore, MVC normalized data provide an understanding at which capacity level the muscles works and how much ergonomic demand a specific task is asking from the subject (Konrad, 2005). The maximal contraction capacity changes with the center of force. In our experimental setting (examining jaw adductors) the maximal contraction refers to maximal clenching and it can be concluded that the capacity is the highest in the molar region of the dental arch and is, therefore, a function of periodontal quantity. The limiting factor during the EMG is the pain threshold of the periodontium, yet the threshold is a constant variable given that the maximal voluntary contraction is constant too.

The amount of maximal contraction capacity is limited due to three factors: First, maximal clenching may induce pain in the masseter muscles, which are perceived by nociceptors in the masseter region. Second, the Golgi tendon organs of the masticatory muscles and the joint receptors in the temporomandibular joints determine the amount of maximal clenching (Hosman et al., 1979). Third, the proprioceptive feedback limits the muscle activity and modulates the muscle force output (Eberhard et al., 2014). Studies suggest that additional muscle activity of the jaw-closing muscles are under control of reflex mechanisms of sensory origins (periodontal proprioceptors but also muscle spindles), with a maximum of reflex output, enabling an automatic functioning while maintaining protective control (Abbink et al., 1999; Ottenhoff et al., 1992). It was investigated that the proprioceptors provide a positive feedback to the jaw-closing muscles (Lavigne et al., 1987), inhibit the positive feedback (Dessem et al., 1988), or even evoke the jaw-opening reflex (Lund et al., 1983). This is also referred to as peripheral feedback. Following conditions should be fulfilled if a comparison in-between various EMG-sessions is required:

(1) “For any individual the maximal clenching force should be a constant and reproducible force.

(2) During each session a particular static muscle force should always be accompanied by the same normalized integrated EMG activity” (Hosman et al., 1979).

One way of fulfilling the first condition is by exerting the utmost in intercuspal position. The other option is to exert the utmost on dental cotton rolls (located between the second mandibular premolar and the first molar). For the latter, maintaining the location of the cotton between the molars for each subject is essential. It enables the same conditions for everyone and guarantees the maintenance of the maximal clenching force, as it changes with the location. Biting on dental cotton rolls can result in larger activity of masticatory muscles than MVC in maximal intercuspal position. Patients contract their MM more efficiently, particularly the masseter and temporalis muscles (Hugger et al., 2008). Tartaglia et al. describe this phenomenon as a result of reducing proprioceptive inputs from unstable occlusion through biting on dental cotton rolls (Tartaglia et al., 2008).

Furthermore, many studies have investigated the effect of verbal encouragement on EMG activity and MVC, and confirmed that verbal motivation has statistically significant effects on achieving maximal performance (Binboğa et al., 2013; McNair et al., 1996).

2.4.2. Bite force measurement devices

In order to achieve the second condition defined by Hosman et al., a simultaneous recording of the bite force (amount and direction of bite force) and EMG is required (Schindler et al., 2005). This combination provides reliable information about masticatory muscle activity while performing several tasks, e.g., bilateral clenching. One type of controlling the exerted bite force is by visual feedback. Various bite force transducers are available for force feedback, such as the gnathometer, a deformation-sensitive piezoelectric film, a quartz force transducer, a strain-gage bite force transducer, and exposed pressure-sensitive foils (PSF). Floystrand developed the first miniature bite force recorder in 1982. It was an electronic semiconductor which transforms the bite force exerted on the sensory unit proportionally into electric alterations in the circuit. A similar technique was used by Fernandes et al., the conductive polymer pressure-sensing resistors, also referred to as Force Sensing Resistor (FSR) (Fernandes et al., 2003; Koç et al., 2010).

The mechanisms of force feedbacks are comparable. However, most register the vertical direction rather than registering the results of both the horizontal and vertical components of bite force (Van Eijden et al., 1988).

Furthermore, there are three-component (horizontal: antero-posterior, transverse and vertical) piezoelectric force transducers which are commercially available (e.g., Kistler Instruments AG, Winterthur, Switzerland).

The transducer mainly contains a contact plate, the sensor, which is placed parallel to the occlusal plane in the second premolar and first molar region, as the bite force is larger at the posterior end of the dental arch (Van Eijden et al., 1988). A small occlusal impression for the upper and lower dental arch allows for a simple and reproducible placement of the transducer, and direction of the bite force relative to the dental arch (Van Eijden et al., 1988). Subjects are asked to bite on the device, consequently, the intraorally measured force vector is displayed on a monitor, e.g., as an additional vertical bar. The predefined target values are marked on the display and subjects aim to reach that value through various contraction forces. Meanwhile, an EMG device records the masticatory muscle activity exerted during force feedback (Schindler et al., 2005). The gained data are normalized accordingly using the maximal clenching values and the percentage value of MVC results, enabling a comparison between sessions and subjects.

2.5. Measuring instruments

As described above, current literature recedes from the concept of bruxism being a disorder per se. Researchers and clinicians, such as Raphael et al. and Lobbezoo et al. point out that higher levels of masticatory muscle activity might increase the risk of adverse oral health outcomes. While for other individuals it might “just” be a motor behavior without any value, or even have advantages. Thus, higher levels of jaw muscle activity may be considered a risk factor rather than a disorder in otherwise healthy individuals. This knowledge makes it more evident why bruxism itself cannot be considered an abnormality unless clearly associated with negative impact (Lobbezoo et al., 2018; Raphael et al., 2016).

According to this concept, it is not the intent of this clinical trial to differentiate between “healthy” and “diseased” subjects, but rather to differentiate between subjects with different bruxism degrees and diagnoses. In order to measure bruxism degree, following questionnaires and the single-channel EMG device, viz. GrindCare® was used.

2.5.1. Questionnaire

The first bruxism assessment in our clinical trial was the self-report of bruxism by using two questionnaires. First, the general questionnaire which gathers demographic data, including date of birth, and gender, and the self-perception of SB and AB. The latter questions are displayed in *Table 9* (Giannakopoulos et al., 2013).

Table 9: General bruxism questionnaire (own translation) (Giannakopoulos et al., 2013).

Yes	No	
		Have you been told, or do you notice that you grind your teeth or clench your jaw while sleeping at night?
		Are you aware or has anyone heard you grind your teeth or clench your jaw during stressful situations, situations in which you're concentrated or when carrying heavy load?

The second questionnaire is the Oral Behavior Checklist (*Figure 12*) developed by the expert panel for Diagnostic Criteria for TMD (DC/TMD) as a convenient and handy tool for bruxism-diagnostics, entailing 21 questions about oral behaviors between dormancy and waking-state. The questionnaire includes two items about bruxism behaviors during sleep and 19 items about behaviors during the day, in the last 30 days. These questions were not only employed to analyze possible sleep -, but predominantly awake bruxism behaviors. The modality of the answer possibilities extends from "none of the time" (= 0 scores) to "all of the time" (= 4 scores) and grades the frequency of the oral behaviors. The total score allows a quantified decision of whether an increased oral behavior exists (= score 24) or not.

The DC/TMD regards bruxism as a risk factor for painful TMD which is the reason why most of the available literature investigates TMD patients. However, an Italian study proved in pain-free subjects, by filling out the OBC twice in a two weeks interval, that the OBC is indeed a reliable tool for a possible bruxism diagnosis (Donnarumma et al., 2018). Ohrbach et al. compared the EMG data with the OBC results and found that different EMG patterns were in concordance with the inquired oral behaviors. They have shown that TMD patients understand the various listed behaviors well and can therefore answer the questionnaire correctly (Ohrbach et al., 2008). However, this thesis could not be confirmed by all studies as the examination between self-report and a portable EMG device showed a lack of association between these two parameters (Prasad et al., 2021).

The Oral Behavior Checklist

How often do you do each of the following activities, based on **the last month**? If the frequency of the activity varies, choose the higher option. Please place a (√) response for each item and do not skip any items.

	Activities During Sleep	None of the time	< 1 Night /Month	1-3 Nights /Month	1-3 Nights /Week	4-7 Nights/ Week
1	Clench or grind teeth **when asleep**, based on any information you may have	☐	☐	☐	☐	☐
2	Sleep in a position that puts pressure on the jaw (for example, on stomach, on the side)	☐	☐	☐	☐	☐
	Activities During Waking Hours	**None of the time**	**A little of the time**	**Some of the time**	**Most of the time**	**All of the time**
3	Grind teeth together **during waking hours**	☐	☐	☐	☐	☐
4	Clench teeth together **during waking hours**	☐	☐	☐	☐	☐
5	Press, touch, or hold teeth together other than while eating (that is, contact between upper and lower teeth)	☐	☐	☐	☐	☐
6	Hold, tighten, or tense muscles without clenching or bringing teeth together	☐	☐	☐	☐	☐
7	Hold or jut jaw forward or to the side	☐	☐	☐	☐	☐
8	Press tongue forcibly against teeth	☐	☐	☐	☐	☐
9	Place tongue between teeth	☐	☐	☐	☐	☐
10	Bite, chew, or play with your tongue, cheeks or lips	☐	☐	☐	☐	☐
11	Hold jaw in rigid or tense position, such as to brace or protect the jaw	☐	☐	☐	☐	☐
12	Hold between the teeth or bite objects such as hair, pipe, pencil, pens, fingers, fingernails, etc	☐	☐	☐	☐	☐
13	Use chewing gum	☐	☐	☐	☐	☐
14	Play musical instrument that involves use of mouth or jaw (for example, woodwind, brass, string instruments)	☐	☐	☐	☐	☐
15	Lean with your hand on the jaw, such as cupping or resting the chin in the hand	☐	☐	☐	☐	☐
16	Chew food on one side only	☐	☐	☐	☐	☐
17	Eating between meals (that is, food that requires chewing)	☐	☐	☐	☐	☐
18	Sustained talking (for example, teaching, sales, customer service)	☐	☐	☐	☐	☐
19	Singing	☐	☐	☐	☐	☐
20	Yawning	☐	☐	☐	☐	☐
21	Hold telephone between your head and shoulders	☐	☐	☐	☐	☐

Figure 12: The Oral Behavior Checklist (Ohrbach et al., 2008).

2.5.2. GrindCare®

For the purpose of diagnosing definite sleep bruxism, electromyographic monitoring of the masticatory muscle activity is required (Lobbezoo et al., 2018). In this study, the portable, ambulatory EMG measurement system GrindCare® (© Sunstar in the version REF: GC401) was used. This device is very handy as it weighs just 10g and is easy to wear because of its' relatively small dimension (4cm from top to bottom) and provides a reliable bruxism diagnosis in the daily life environment.

GrindCare® consists of an EMG and a stimulation electrode (stimulator), a microprocessor, a memory for data storage (Grinddock), a display for user interface, light-emitting diodes, a rechargeable battery, Bluetooth connector for data connection to a smart phone with dedicated application "BUTLER®GrindCare® application" (© Sunstar in the version 1.3) and a battery charger. Furthermore, the stimulator has two buttons, which regulate the intensity of the biofeedback. These components generally enable the recording of EMG activity, their quantification, computing, as well as the consequent reduction of masticatory muscle activity (Koyano et al., 2008). The first GC device, investigated by Jadidi et al. in 2008 (Jadidi et al., 2008), has been updated several times over the years.

The GC401 used in this study provided the following information: number of grinds per hour, total number of grinds, number of measurement hours, date, as well as the time of the measurement. *Figure 13* shows the App Display of the GC device, presenting the activity of one night (left) and one week (right). The left picture provides a visualized tracking of the grinding activity and gives information about the date, the time during which the subject was asleep, the total measurement hours, the number of grinds/hour and the total number of grinds in one night. The overview displays the dates during which the data was acquired and the total number of grinds per night.

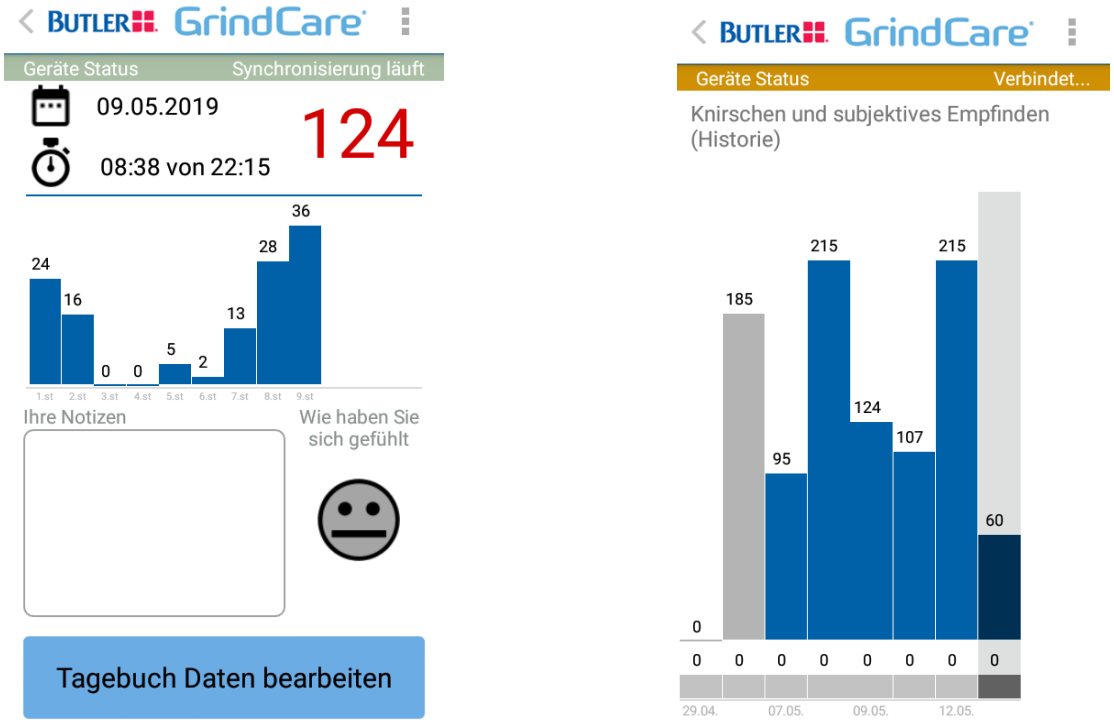


Figure 13: GrindCare© application display: one night (left), one week (right). Left: each bar corresponds to the number of grinds per hour (the x-axis reports the hour of measurement). Right: each bar corresponds to the number of grinds per night (the date appears on the x-axis). With friendly permission by Vivien Frommer.

GrindCare® has two electrode contacts (bipolar electrode) which are placed on the skin overlying the anterior temporalis area. The temporalis is used as it is active during any jaw closing muscle activity, while also providing a large surface area for sufficient skin-electrode contact. Furthermore, the temporalis is less covered by skin and fat tissue among all masticatory muscles (Koyano et al., 2008; Stuginski-Barbosa et al., 2016), does not require the male subjects to remove the beard, and is less influenced by the cross-talk of neighboring muscles (i.e., the orbicularis oris) (Rantanen et al., 2016). These aspects make it more convenient to use the temporalis muscle for signal acquisition. The GC devices have become easy for subjects to operate and are also precise in terms of the number, duration, and magnitude of bruxism episodes per hour (Needham et al., 2013).

The calibration of the device is ensued automatically and individually by considering the following aspects of essential importance. The first aspect is the automatic determination of the threshold level derived from baseline activity. This signal recognition algorithm is a moving average algorithm used for the GrindCare® 4 devices. It uses a dynamic estimation of the background EMG noise and applies the rules for the detection of RMMA (*Table 7*), which is the fundamental difference to the previous GrindCare® versions. The second aspect is the constant modification of the threshold throughout the measurement by comparing the EMG amplitude to the background level, while bursts of EMG are detected when they exceed the background noise by triple the background amplitude (Dreyer et al., 2015).

GrindCare® 4 can reach a 90% accuracy in terms of specificity when used for three- or five- consecutive nights but is not as high in sensitivity (50%), compared with polysomnography (PSG), which is considered the gold-standard for sleep-bruxism diagnosis (Stuginski-Barbosa et al., 2016). Cut-off values which differentiate SB subjects from non-SB subjects are 18/19 EMG events/h for three/five consecutive nights (Stuginski-Barbosa et al., 2016). GrindCare® operates as a long-term EMG while being able to save the data of several nights. The major advantage of this function is the ability to measure the long-term fluctuation of bruxism. Moreover, its' size and use at home could probably help avoid the "first night effect" observed in a sleep laboratory due to the unfamiliar environment and the multitude of foreign devices (mainly electrodes and cables) on different parts of the body. The first night effect is characterized by a deviation from the usual sleep pattern, including longer stage two and rapid eye movement (REM) sleep latencies, and lower sleep efficiencies, which is the reason why in most experimental trials it has become a common practice to exclude the first night of sleep in sleep recordings (Toussaint et al., 1995).

A study on the reproducibility of EMG signals acquired from the masseter muscles during mastication has confirmed a correlation between the resulting EMG signal and various variables, for instance, the relocation of the electrode, levels of skin resistance, and location of the head and body (Garnick, 1975). The accidental detachment of GrindCare® from the skin surface during sleep results in critical artifacts due to the loss of contact and movement of the detection system. This is challenging to control because of the home environment. A further issue is the recording of orofacial activities beyond bruxism, such as talking, yawning, etc., which cannot be distinguished from sleep bruxism when sleep bruxism is tracked in a sleep laboratory.

For that purpose, it is possible to combine PSG with audio – and video recordings, which is a major benefit for the precise detection of non-bruxism muscle activity (Gallo et al., 1997; Kato et al., 2003; Koyano et al., 2008; Lavigne et al., 2008).

The biofeedback option of the GrindCare® device, mentioned above as contingent electrical stimulation (CES), can be activated to modulate inhibitory reflex mechanisms in jaw-closing muscles. When the detected EMG activity exceeds the threshold level for more than 0.25 seconds, an innocuous electrical pulse of predefined intensity is emitted (Murali et al., 2015). This cutaneous CES of trigeminal nerve fibers evokes reflex suppression of voluntary contraction of both masseter and temporalis muscles for a minimum of 10 seconds, also referred to as “exteroceptive suppression”. Consequently, the major masticatory muscles relax. This interruption is believed to condition the natural reflex when repeated frequently, leading to a presumably long-lasting learning effect and thus manages bruxism causally (Desmedt et al., 1976; Godaux et al., 1975; Jadidi et al., 2008). The stimulation should be perceived clearly, yet, in a non-painful intensity (range 1-7 mA, 230Hz) (Jadidi et al., 2008). The stimulation intensity can be modulated individually by pushing the minus/plus button, adjusting the nine different stimulation levels (0= inactive, nine= highest stimulation). The number of EMG episodes/hour of sleep is significantly reduced through CES. Therefore, CES shall lead to relief of jaw musculature and oral structures (Jadidi et al., 2008; Needham et al., 2013).

Before adhering the GrindCare® to the temple through gel pads (dedicated Sunstar pads), the skin surface was cleaned using 70% isopropyl alcohol pads (B.Braun Melsungen AG, Melsungen, Germany). The gel pads ensure a good connection between the electrode contacts and the skin. When putting the Stimulator back to the docking station, both units communicate wirelessly via Bluetooth and data transfer starts automatically. The GC equipment is displayed in *Figure 14*.

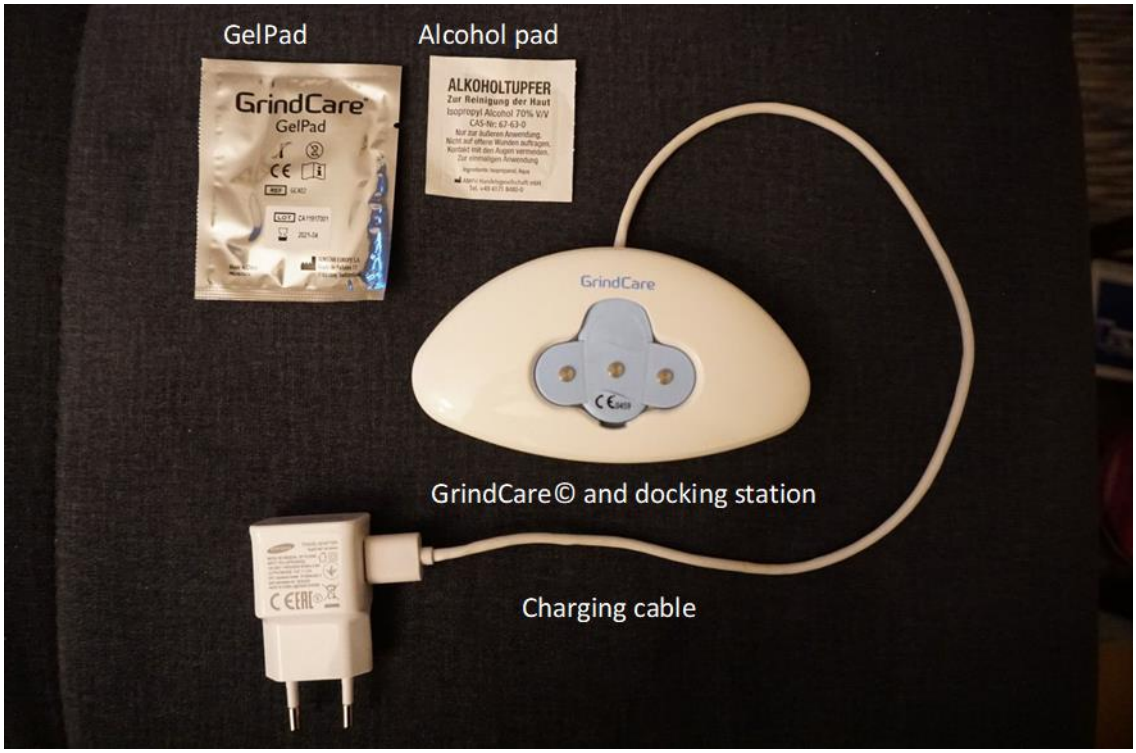


Figure 14: GrindCare© equipment, GelPad, alcohol pad, Grinddock and charging cable.

Furthermore, an individual vinyl foil template was fabricated to facilitate locating the precise position of the electrode on the skin overlying the temporalis, in the effort to ensure reproducible measurements. This method helped minimize errors due to incorrect GrindCare® positioning by the subjects (Burdette et al., 1990; Castroflorio et al., 2005; Frame et al., 1973; Im et al., 2017). The reference structures for that purpose were the ear and eyebrow. The position of the GrindCare® was, consequently, cut off from that part of the foil, so that the subject could hold the template on ear and eyebrow while adhering the device through the template's recess, as shown in *Figure 15*.

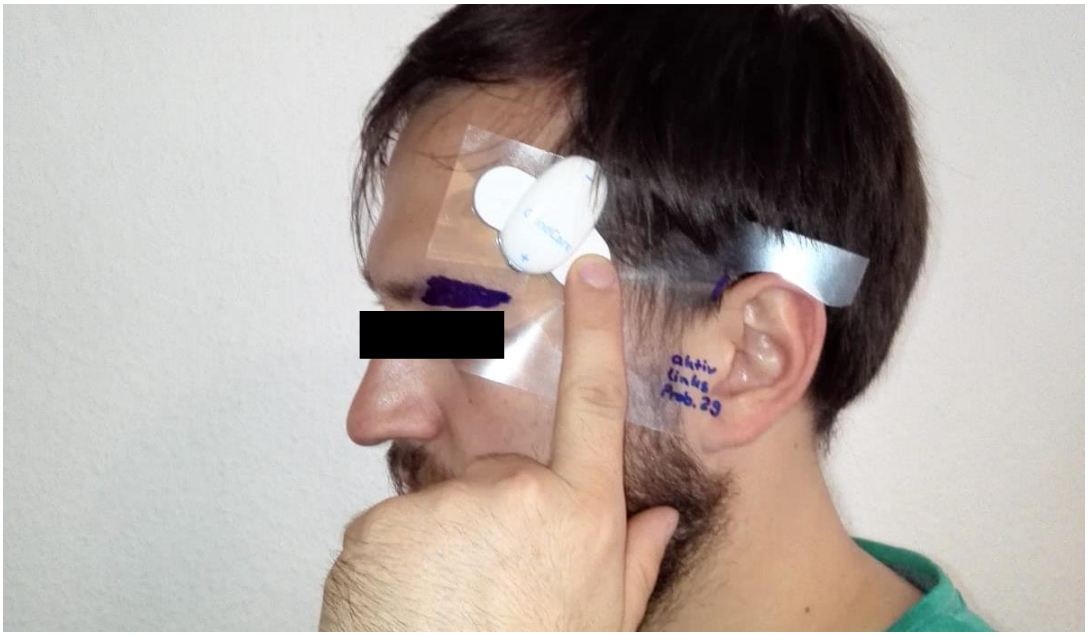


Figure 15: Template for reproducible positioning of the GrindCare® on the temporalis muscle, by using the eyebrow and ear as reference structures.
With friendly permission by Vivien Frommer.

2.5.3. Electromyographic (EMG) recordings (laboratory setting)

An eight-channel surface EMG device (MP 100 Biopac, Biopac® Systems, Inc., Santa Barbara/CA, USA) including the compatible Software (AcqKnowledge® 3.9.1., Biopac Systems, Inc., Santa Barbara, CA, USA.) was used to record the activity of both Mm. masseteres and anterior temporales bilaterally. The differential EMG-amplifier (input noise: 0.28 µV pp, impedance 146 kΩ, common-mode rejection ratio [CMRR]: 110 dB) was utilized to digitize analog raw EMG signals and to reduce signal artifacts. The band-width was 7.5 – 1000 Hz and signals were recorded at a sampling rate of 1000 Hz with a resolution of 12 bit (voltage amplification: 1000). These signals were transferred to the Acquire software and saved on a computer at the University of Würzburg. Surface elec-trodes described below were placed on the temporalis region, pointing toward the orbital cavity. The repeatability was ensured by using an individual template fabricated in T0, as demonstrated above. Before placing the electrodes, the subject's skin was cleaned with 70% isopropyl alcohol pad (B.Braun Melsungen AG, Melsungen, Germany), to re-duce the impedance between skin and electrodes. The impedance was further reduced through the hypoallergenic gel and adhesive under the electrodes.

Disposable bipolar Ag/AgCl surface electrodes were used, with a conducting surface diameter of 14 mm and a 20 mm distance from both centers of the two electrodes (FIAB Spa, Vicchio, Firenze/ Italy). As mentioned above, the major aspects for a reproductive EMG signal are the relocation of the electrode, levels of skin resistance, and location of the head and body (Garnick, 1975). *Table 10* presents possible influencing factors for an EMG signal and how these were minimized in this clinical trial.

Table 10: Factors influencing the EMG signal and how these were minimized in this clinical trial (Im et al., 2017; Konrad, 2005).

Factors	**Solution**
Cross talk of facial musculature	Same electrode position, so cross talk will be the same in each session
Electrode impedance	Clean skin (70% isopropyl alcohol), dry surface, and electrolytic gel
Electrode position	Coordinate system (Burdette et al., 1990; Frame et al., 1973; Im et al., 2017)
External electrical influence	No electrical devices (i.e., mobile phones) were close to the EMG device
Head/ Body movement	Subject sits in same comfortable adjustable chair in each session
Interelectrode distance	Fixed electrode distances (Castroflorio et al., 2005)
Muscular performance	Fluctuations minimalized by maintaining relatively consistent time for measurements (evening)
Pain conditions	Exclusion criteria
Variability/ Inconsistency in impedance	Reduction through use of the variable maximum voluntary isometric contraction as a reference value, (although impedance varies in active muscle fibers, electrodes, and inter-& intraparticipant measurements)

2.5.4. Bite force measurement – BiteFork®

To guarantee the reproducibility through similar experimental conditions between all three experimental sessions (T0, T1, T2), the same submaximal bite forces were achieved by using a force-feedback device (BiteFork®; ViMeS, Igel, Germany). BiteFork® enables not only bilateral bite force measurement, but also a visual illustration and analysis of bite forces, thus, reducing variability in each measurement session.

The system consists of two sensor foils, i.e., FSR, with 0.2mm thickness (FlexiForce A201-1 sensors, Tekscan Inc., Boston, MA, USA), the main device body, one pair of sensor holders (for each subject) attached to the main device, and a USB-B computer connection cable (Rauer et al., 2019; Weisskircher, 2013). For calibration, a custom-made metal box was constructed by the precision mechanic Willy Wendler in KIT (*Figure 16*).

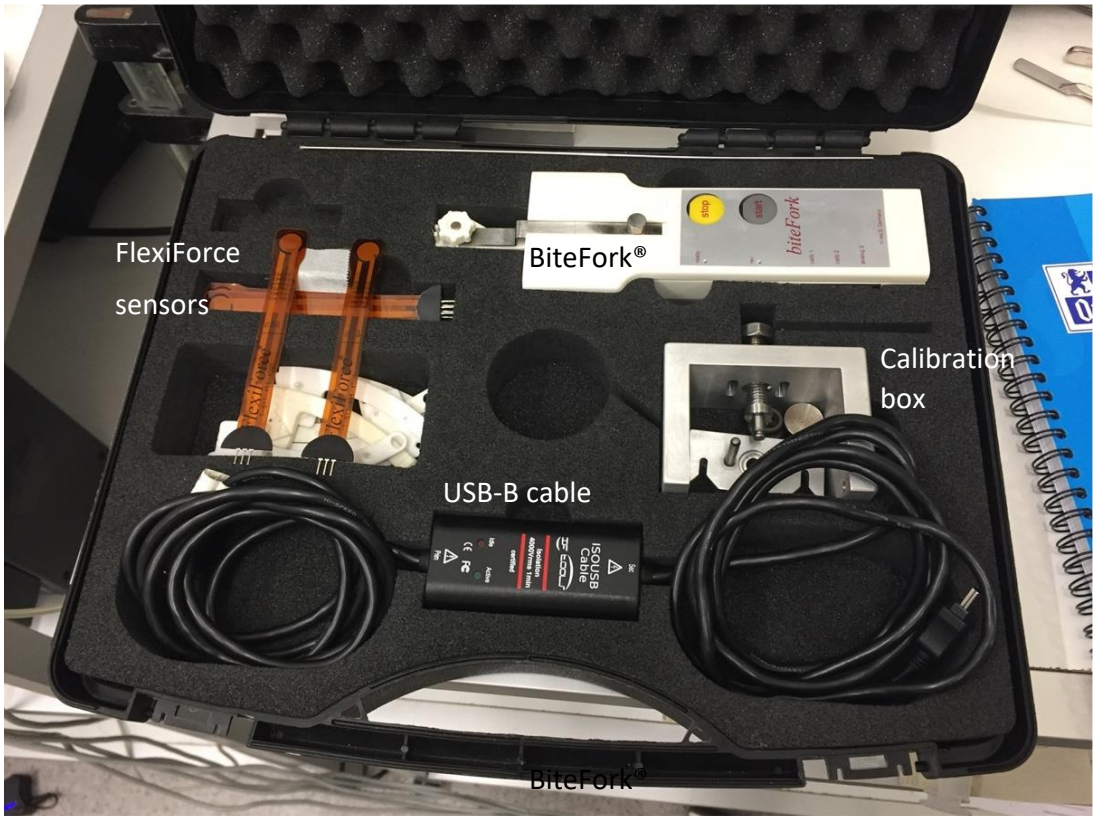


Figure 16: BiteFork® equipment, entailing the BiteFork® main device body, FlexiForce sensors, the USB-B cable, and the calibration device.

The FlexiForce sensors are Force Sensing Resistors (FSR). In terms of construction *Figure 17* the FSRs can be assigned to the piezoresistive sensing technologies, exhibiting a decrease of electrical resistance the more bite force is applied onto them. The “Thru Mode FSR” (such as FlexiForce) are flexible printed circuits containing silver circles coating the pressure-sensitive layer, followed by a conductive polymer. This compound in its entirety is also referred to as the Sensor Sensitive Area. The Sensor Sensitive Area is located between two metal electrodes, consisting of a semi-conductive material. The two parts of the FSR are adhered with an adhesive layer and framed with two polyester films. This layering technique is referred to as the “Traditional Sandwich Element“ (TSE) (Paredes-Madrid et al., 2017). The preferable polymers used are elastomers, rubbers and polydimethylsilicone (PDMS) (Stassi et al., 2014), while the conductive particles can be from nickel or copper (Bloor et al., 2005), carbon black, and carbon nanotubes (Wang et al., 2013; Wang et al., 2014), *Figure 17*.

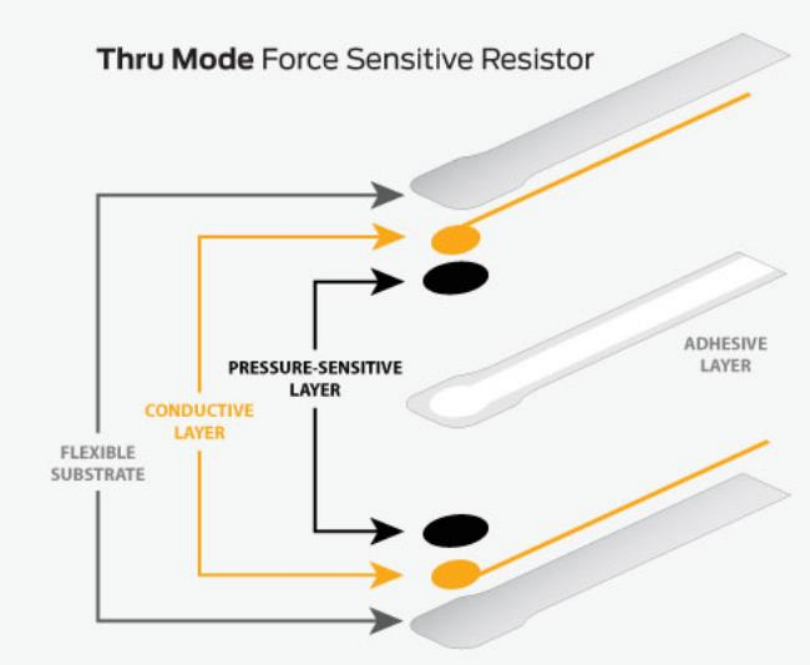


Figure 17: FlexiForce sensor characteristics, consisting of the pressure sensitive layer, the conductive layer, the flexible substrate, and the adhesive layer.

The sensor holder is a clip housing, consisting of an upper and a lower sensor holder. The sensors are fixed by using a minimal amount of denture fixative cream (blend-a-dent super adhesive cream, Procter & Gamble GmbH, Schwalbach am Taunus, Germany) in the sensor holder, as shown in *Figure 18*. Consequently, the sensors are connected to the BiteFork® main device body and screwed on tightly (*Figure 19*). The BiteFork® main device body is connected via USB-B computer connection cable to the computer, which eventually visualizes the exerted bite force on the computer screen.

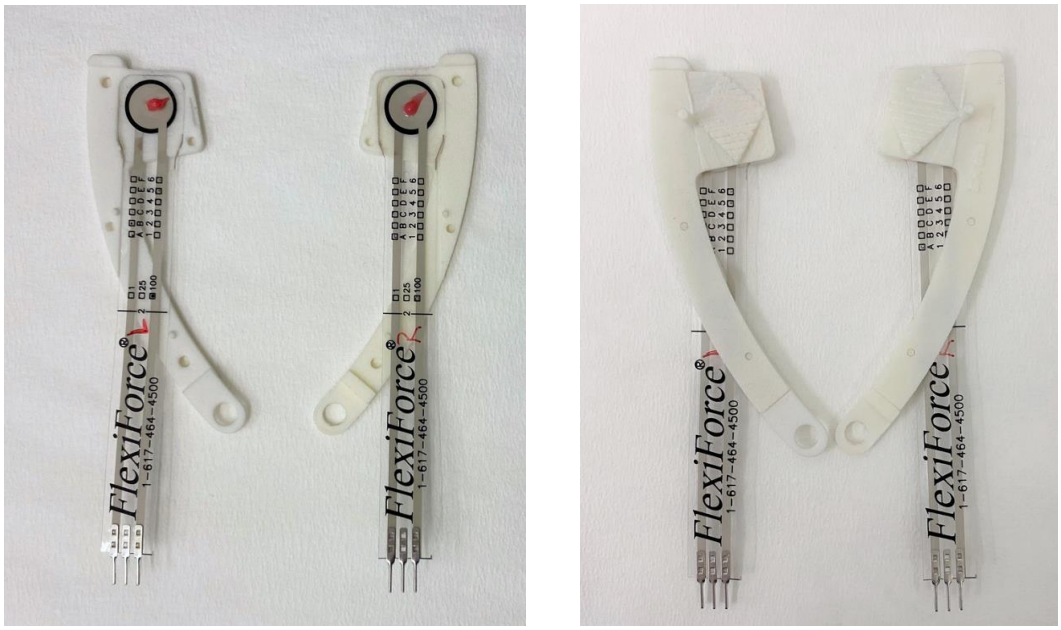


Figure 18: The FlexiForce® placement in the lower part of the sensor clip holders of the BiteFork® device, with a little amount of fixative cream for adhesion (left), sensor holder & sensor (right).

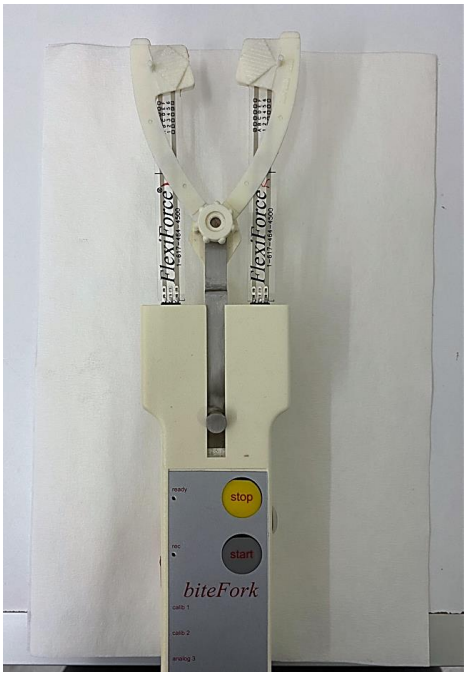


Figure 19: The whole BiteFork® device.

For ensuring the correct and reproducible interproximal position in each measurement session a C-silicone impression material (silicone modeling clay, Polysiloxane, Omni-dent Dental-Handelsgesellschaft mbH, Rodgau, Germany) was placed on both sides of the bite blocks (*Figure 20*).

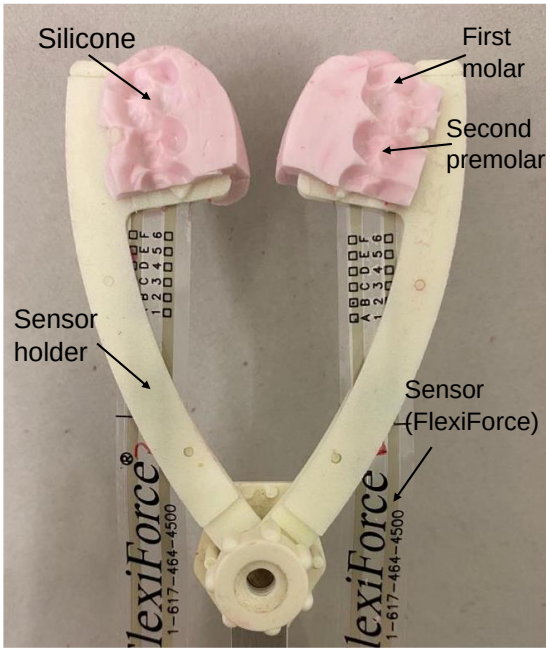


Figure 20: Left and right sensor holders, FlexiForce® sensors and silicone bite blocks.

A pin on the right and left bite block is meant to help the correct placement between the second premolar and the first molar, as shown in *Figure 21*.

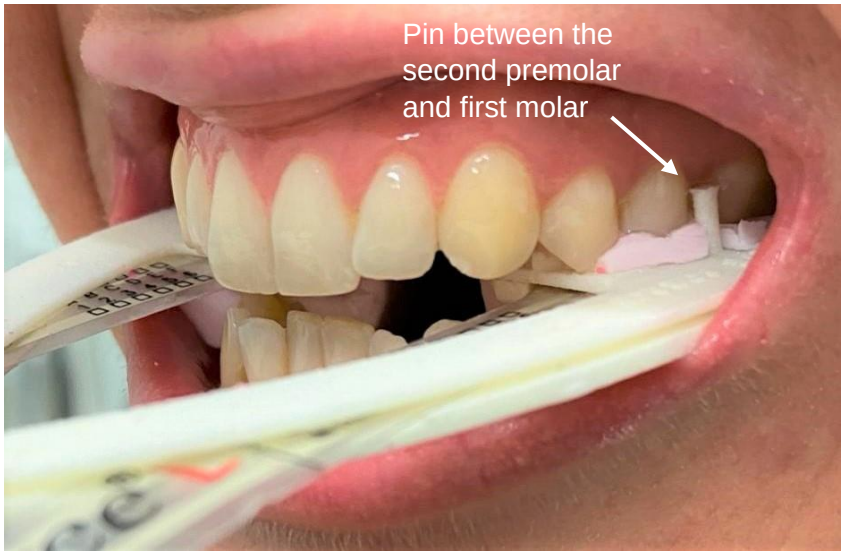


Figure 21: Intraoral placement of the BiteFork®, the pin is located between the first molar and second premolar.

These silicone bite blocks were stored, along with the sensor holders, in small, numbered boxes in the laboratory of the University, where the measurements took place (Weisskircher, 2013). The two functionally separated sensors ensure an independent but simultaneous recording of bite force for each side of the jaw (Giannakopoulos et al., 2018; Rauer et al., 2019).

Before starting each measurement, calibrations must be performed to ensure the repeatability and reliability of the acquired data. The calibration installation is visualized in *Figure 22*. For this purpose, the sensors are placed in the calibration box and a force between 200-250 Newton (N) is adjusted. In a fast, constantly increasing motion the calibration box is closed on the sensors until the force reaches 200-250 N.

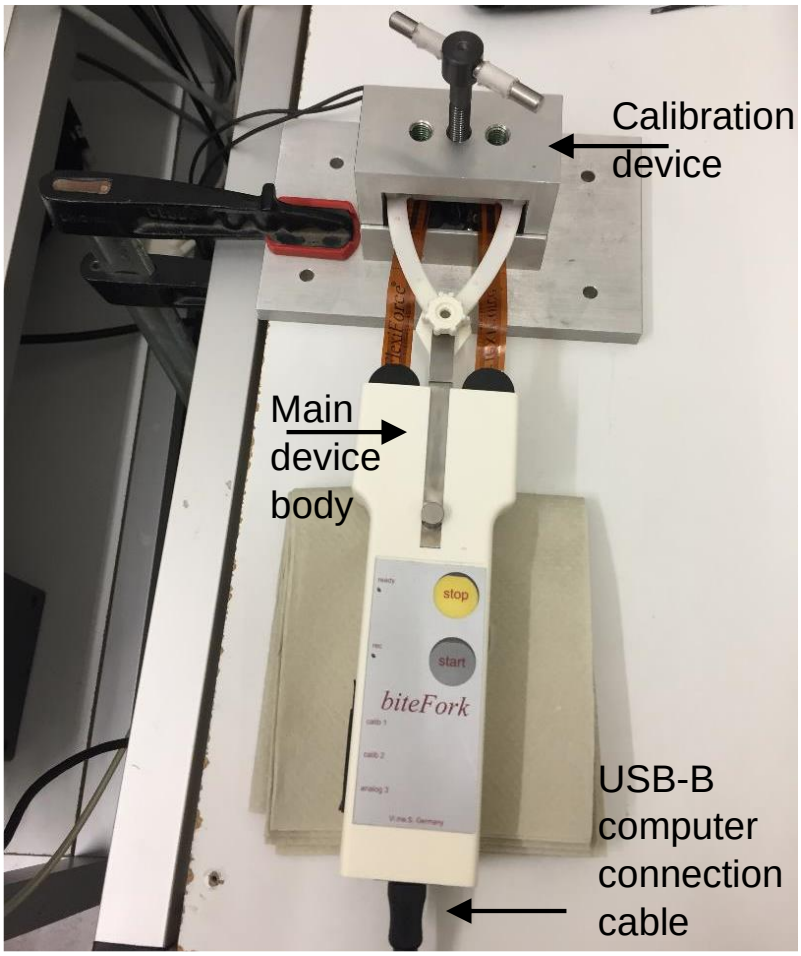


Figure 22: Installation for calibrating the BiteFork®, the main device body, calibration device and USB-B cable.

The transfer process from force to a numeric value is described by Weisskircher et al. as follows: "*When the resistance at the sensor head decreases, the base potential increases. The device digitizes the measured voltage (sampling rate 1000 samples/s, 10 bit), converts it into Newton and displays them as a graph on a PC display or as numeric maximum force values on the display of its' hand piece.*" (Weisskircher, 2013).
Subsequently, a dynamic graph on the computer shows the transfer of force from the sensor for each of the two functionally separated sensors. Ideally, they should not severely divert from each other. A maximum angle of 45 degrees is considered acceptable, according to the manufacturer's recommendation (personal communication with Dr. Weisskircher). If this range is exceeded, a repetition of the calibration is needed. *Figure 23* shows a successful calibration example.

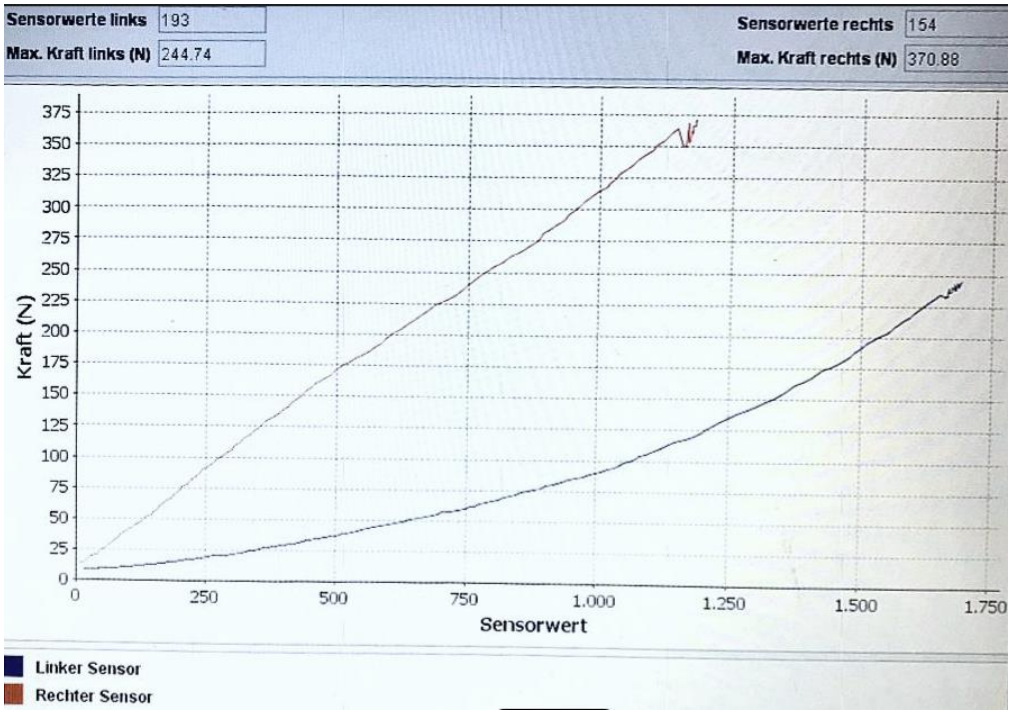


Figure 23: Calibration of BiteFork® in the calibration box. The right sensor (red) reached a calibration value of 371 N and left sensor (blue) reached a calibration value of 245 N, with an angle < 45 degrees.

2.6. Study protocol

The active study part included four appointments for each participant. All four appointments took place in the oral physiology laboratory of the Department of Prosthodontics of the University Clinic in Würzburg. Initially, volunteering subjects were selected according to the preset eligibility criteria after a preliminary examination (T0). If the inclusion criteria were met, participants were informed about the study protocol and duration of five weeks. Furthermore, they received an information sheet about the home use of GrindCare® and after clarifying all questions regarding the study protocol, their participation or other safety issues, they signed a written consent form for their voluntary participation and photo-documentation during the experiment. All participants were in contact with the study examiners and the principal investigator via WhatsApp or E-mail, for occurring questions or premature termination. An important requirement from the participants was to maintain their usual sleeping habits, including the use of occlusal splints, during their participation in the study. At the end of the initial investigation, subjects were handed out the GrindCare® device and were instructed to wear it in its diagnostic mode (i.e., with stimulation level set at zero) for one week.

For accomplishing the purpose of finding electromyographic variables which may differ depending on the diagnosis or degree of bruxism, surface EMG was recorded bilaterally on the masseter and anterior temporalis muscles. The first (T1), second (T2), and third (T3) sessions encompassed the laboratory EMG measurements controlled by simultaneous force feedback. One week after T0 the first laboratory EMG session took place in T1. After that the control group was instructed to continue wearing the inactive GC device while asleep for further two weeks. The CES option of the GrindCare® device was activated for the intervention group according to our randomized list to set back all additional masticatory muscle activity. An EMG session for monitoring the EMG variables followed in both groups two weeks after intervention in T2. During the last two weeks of the study, the whole study sample returns to wearing the inactive GC, followed by the last EMG session in T3. The last part of the study aims at investigating whether stopping the CES intervention would result in the same muscle activity as before the intervention. The study protocol is visualized in *Figure 24*.

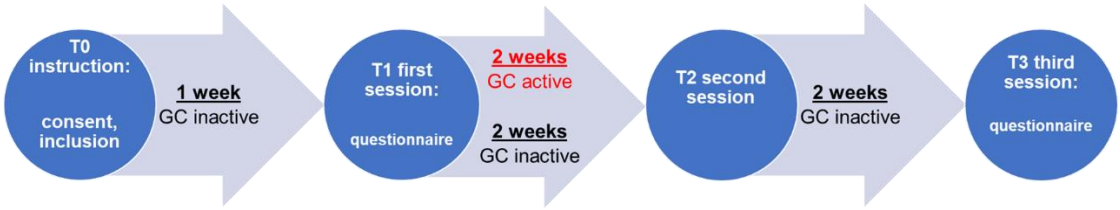


Figure 24: Study protocol.

Throughout the study, neither the investigator nor the participants had access to the GrindCare® data or to the subject's group allocation. Therefore, an external examiner, not participating in this EMG study was present at the T0 appointment and was responsible for all GrindCare® related tasks in T1, T2, and T3. This method ensured that both parties, investigator and subject, were blinded and not influenced by the GrindCare® results during all EMG measurements. One issue that occurred towards the end of the study, was the participation of fewer male than female subjects which made the matching a demanding task. In order to solve this issue, the external examiner selected the last subjects additionally according to gender and age. This method enabled the maintenance of the blind experiment for the examiners throughout the whole study.

2.6.1. T0: subject orientation session

The external examiner who was not participating in the laboratory EMG recordings fulfilled the following tasks:

1. Filter eligible participants (according to inclusion/exclusion criteria and by assessing the TMD pain screener).
2. Information about the study protocol (study period of five consecutive weeks, EMG measurements in T1, T2 and T3, GrindCare® use during the first week).
3. Instruction about the GrindCare® device:

   All subjects, regardless of the group, wore the inactive GrindCare® device, on the side of the temporalis determined by a second randomization list, for the first seven consecutive nights. The external examiner further instructed that the temple must be cleansed using 70% alcohol pads and be allowed to dry before applying the GC pad. While the skin surface is drying, the disposable gel pads shall be adhered to the GC according to the user's manual. With the aid of the template, the subject is advised to adhere the stimulator to the skin always at the same position with the help of the individual template. If the placement was successful, a short beep becomes audible. Only then is data recorded from GrindCare®. In case the silicone protection cover falls off, it is advised to push the minus button until a short beep resonates to make sure the device is not active in terms of stimulation during the first week. After each use (when waking up), the stimulator shall be placed in the Grinddock to ensure the data is transferred and the stimulator is charged and ready for use next night. Subjects were instructed to leave the Grinddock plugged in throughout the entire study time.
4. Clarifying all occurring questions (also during the other appointments).
5. Collect the signed informed consent for voluntary participation and photo-documentation.
6. Sort the subjects into different groups (by randomization list and match according to gender and age).
7. Handing out the diary, GrindCare®, and questionnaires (OBC, general questionnaire):

The GrindCare® diary enabled the recording of occurring problems (e.g., while adhering, or in case the stimulator fell off), and is attached in the Appendix *(Table 30*). Participants were asked to fill out the questionnaires at home and were advised to return them after seven days, along with the GrindCare® device to the first laboratory EMG session (T1).

8. Coverage of the GC Stimulator:

   The two buttons which regulate the intensity of the biofeedback were covered by an elastomer A-silicone FutarD (Kettenbach GmbH & Co. KG), in order to prevent its activation.

9. Preparation of the individual template as shown in *Figure 15*, to facilitate reproducible positioning of the GC on the skin overlying the temporalis muscle.

2.6.2. T1: First session

Preparations before the electrodes' placement

Before the participant came to the first session, all nine electrodes were trimmed at the lateral edge to make them thinner and two reference points were punched out, the central and upper/lower point. A new folder was created for each participant on the personal computer for saving all EMG and BiteFork® data. Furthermore, all relevant information and deviations from the protocol were noted in the dedicated laboratory book. A numbered box was prepared for every subject with their number from the randomization list to store the silicone bites and the sensor holders for the BiteFork® device. The last step in preparation for measurement was the assembly of the BiteFork® device, placing both sensors in between the sensor holders, adhering both parts with denture fixative cream, and starting the calibration of the BiteFork®, as explained in chapter 2.4.2. When the subject came to the first session, the external examiner received the questionnaires and the GrindCare® device in a separate room, extracted and saved the data to a paired mobile phone and afterwards copied those into a special folder before deleting them from the GrindCare® device. This step made ensures that there was always enough storage space in the Grindare® device for the upcoming fourteen nights of recording. Fourteen new alcohol and gel pads were placed in the GrindCare® box, for the following two weeks.

Meanwhile, the subject was seated in the oral Physiology Laboratory room in front of a laptop connected with the BiteFork® device to visually control the feedback screen bar. Once more, the temple and masseter were cleaned using alcohol pads. After disinfecting the examiner's hands and by using gloves, the temporalis and masseter were palpated and marked for electrode placement.

Electrode placement

The masseter contour was identified manually and marked with a pen while letting the subjects contract the muscle by clenching their teeth. *Figure 25* shows the contracted masseter of a proband, in the process of identifying the masseter.

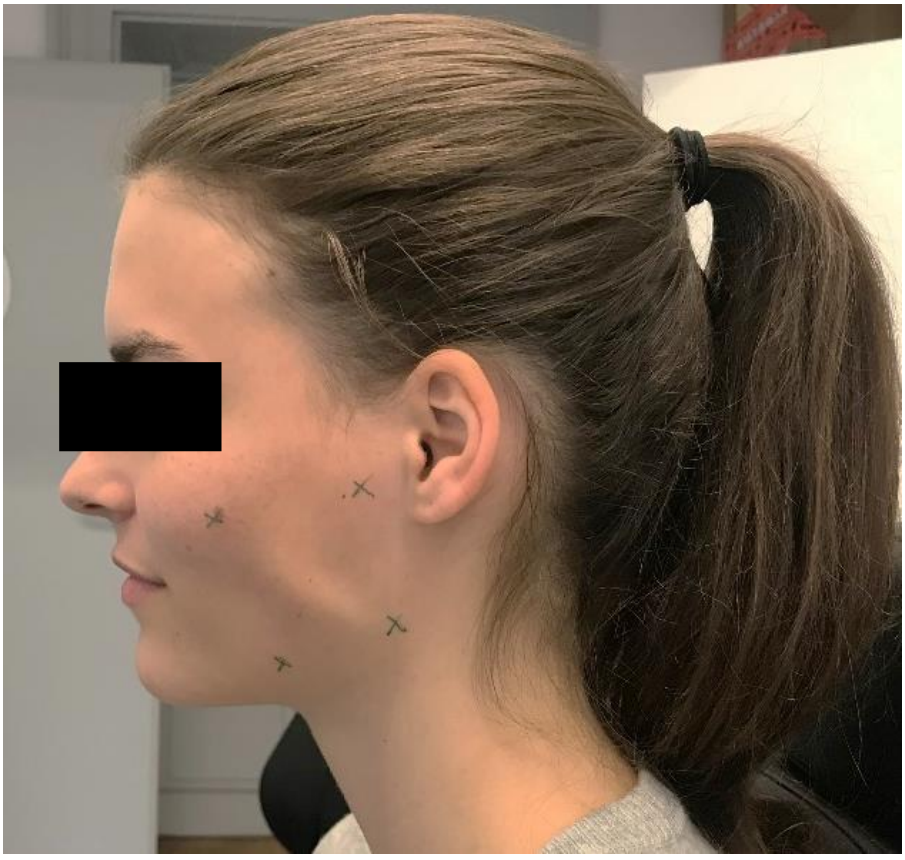


Figure 25: T1; Identifying the masseter muscle.

After connecting the four outermost border points of the muscle (1&3, 2&4), two diagonals result which intersect in the center of the muscle, the “punctum maximum”, as shown in *Figure 26*.

Figure 26: T1; The “punctum maximum”. 1 and 2: boundaries of the masseter origin, 3 and 4 boundaries of the masseter insertion.

This point determines the center of the middle (2nd and 5th -) electrode, which was applied first and lies over the belly of the muscle. The outer two electrodes were applied parallel to the direction of the muscle fibers, right next to the middle one. Therefore, all six electrodes were placed within the boundaries of these lines since these lines represent the outer edge of the masseter muscle. Two further electrodes were applied to the most prominent part of the anterior temporalis muscle, one on each side. The lateral view of the electrode placement is shown in *Figure 27* and the frontal view in *Figure 28*. The reference electrode was positioned on the neck over the seventh cervical vertebra.

Figure 27: T1; Electrode placement on the masseter and temporalis, lateral view.

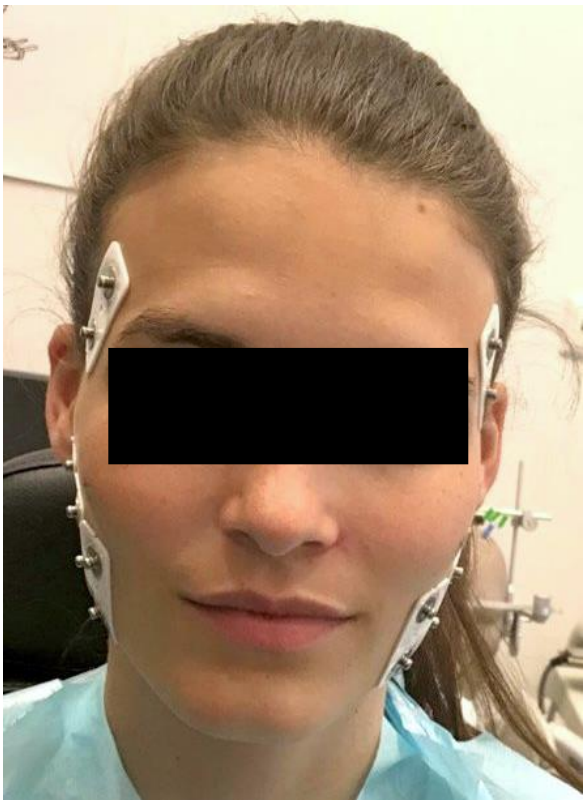


Figure 28: T1; Electrode placement on the masseter and temporalis, frontal view.

To enable a reproducible measurement, a coordinate system was drawn. The x-axis was represented by a line between the “tragus” and the “angulus oculi lateralis”. The y-axis was a line that runs vertically through zero (“tragus”). This coordinate system is displayed in *Figure 29*.

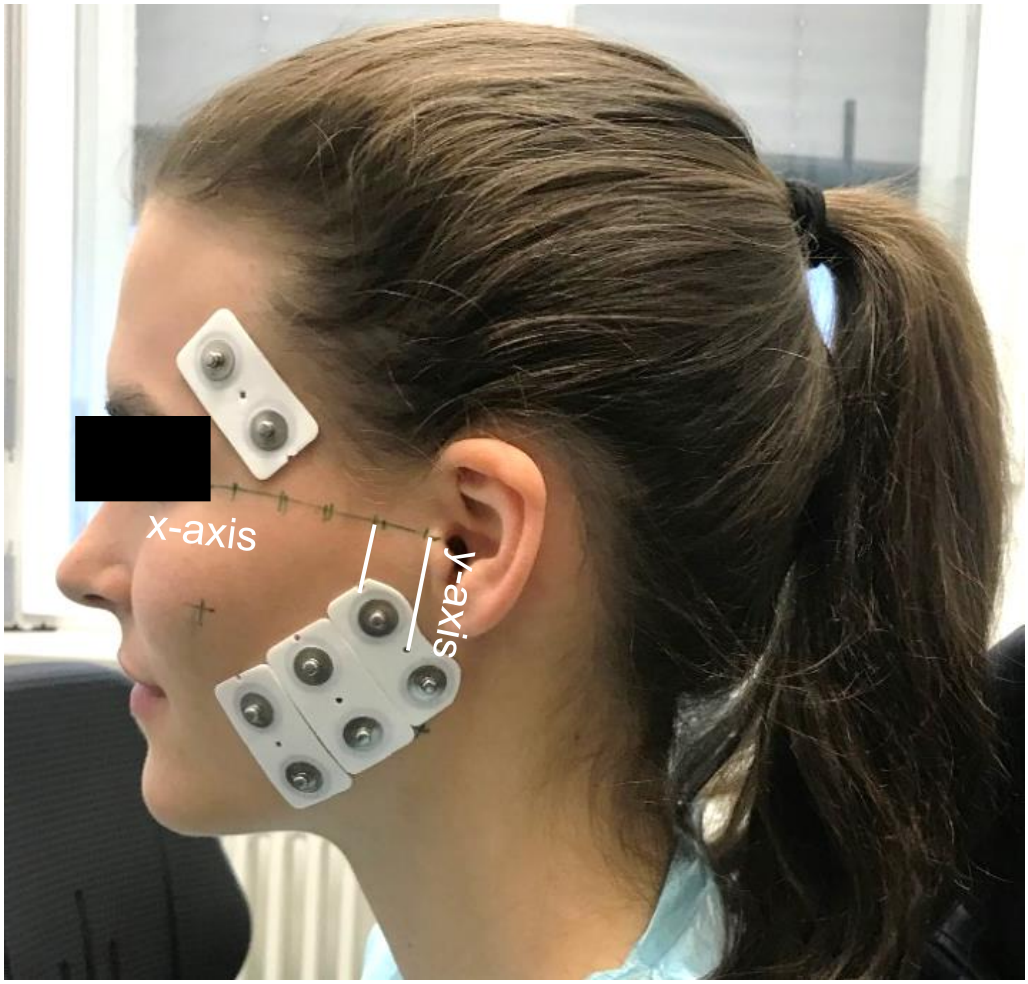
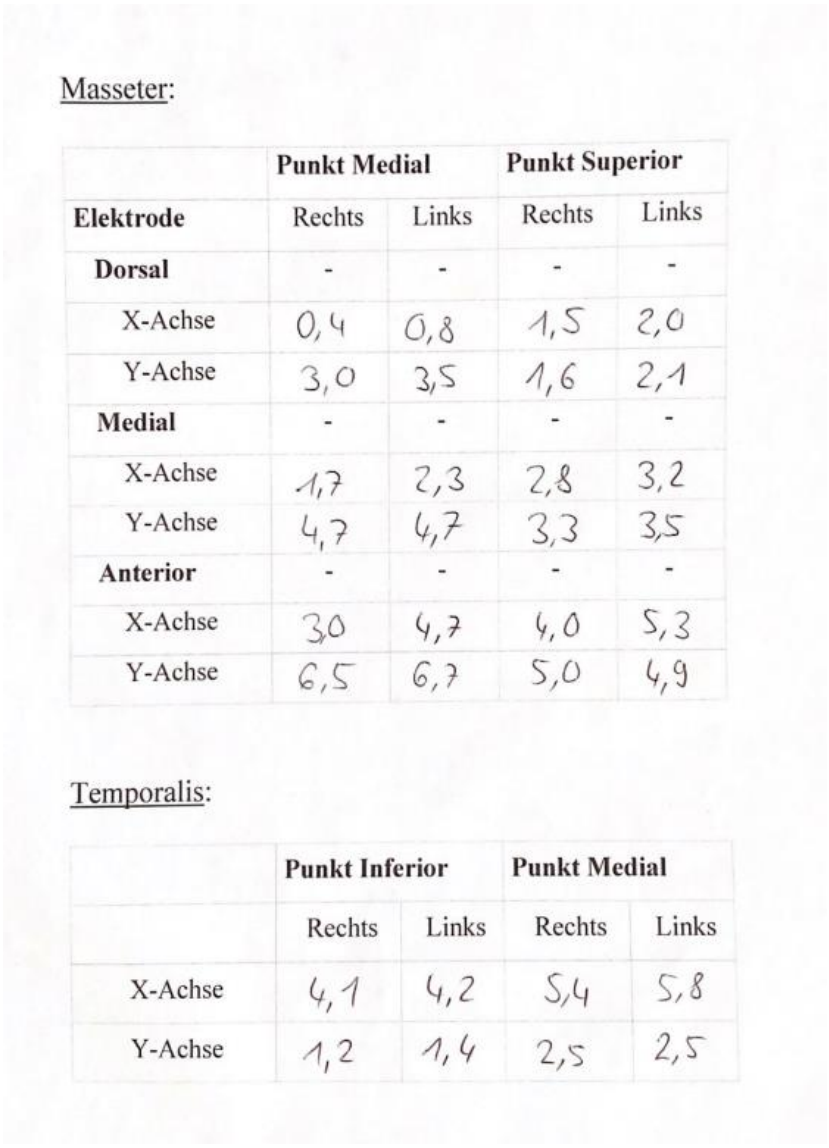


Figure 29: T1; Electrode location in the coordinate system; lateral view.

For unequivocal placement of the electrodes, two reference points are necessary to pinpoint their location in the coordinate system. The first point was the electrode center (10mm to the upper electrode edge/ 7 mm to the lateral electrode edge) and the second was the upper electrode point (1 mm/ 7 mm), as shown in *Figure 30*. A slight variation was needed in terms of the temporalis electrode as the upper central point was not located on the defined x-axis (no extension possible due to anatomic reasons). Therefore, the lower central point was used according to the above-explained scheme (1mm to the lower electrode edge/ 7mm).

Figure 30: Electrode center and upper electrode point.

These coordinates were noted on a sheet of paper for each participant in the first session, thus ensuring reproducibility in electrode position for all examination sessions. The coordinate system sheet is shown in *Figure 31* and the original document is attached in the appendix (*Figure 48*).



Masseter:

	Punkt Medial		Punkt Superior	
Elektrode	Rechts	Links	Rechts	Links
Dorsal	-	-	-	-
X-Achse	0,4	0,8	1,5	2,0
Y-Achse	3,0	3,5	1,6	2,1
Medial	-	-	-	-
X-Achse	1,7	2,3	2,8	3,2
Y-Achse	4,7	4,7	3,3	3,5
Anterior	-	-	-	-
X-Achse	3,0	4,7	4,0	5,3
Y-Achse	6,5	6,7	5,0	4,9

Temporalis:

	Punkt Inferior		Punkt Medial	
	Rechts	Links	Rechts	Links
X-Achse	4,1	4,2	5,4	5,8
Y-Achse	1,2	1,4	2,5	2,5

Figure 31: Coordinate system sheet of the presented proband in Figure 32.

When subjects came in for the second and third EMG session, only the coordinate system sheet was necessary to find the exact position of the electrodes according to their initial placement. *Figure 32* shows the drawing of the coordinate system on the face of the proband and *Figure 33* the electrode re-placement.

Figure 32: T2; Coordinate system for electrode re-placement; lateral view.

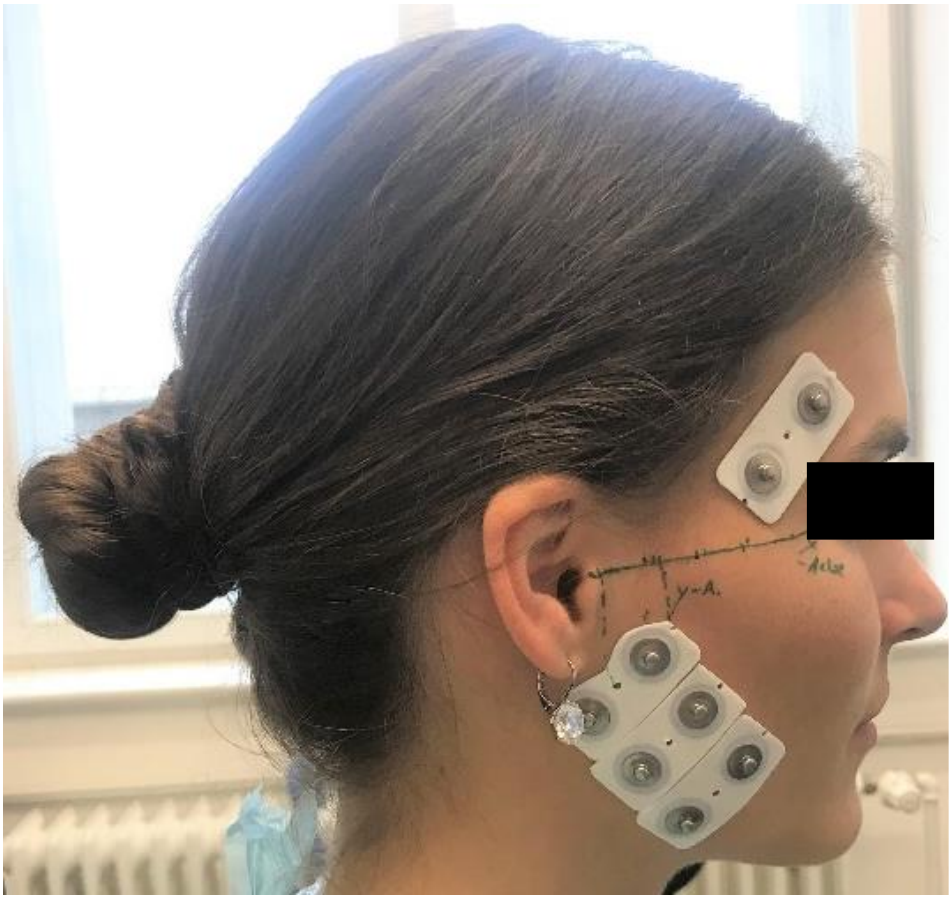


Figure 33: T2; Electrode re-placement with the coordinate system.

Measurement

The second examiner recorded the subject's relevant information and transferred the coordinates on the prepared coordinate system sheet. Lastly, the two silicone impressions with 2- 3 mm thickness were fabricated for the BiteFork®, placing each on the right and left sensor holder at the mandibular side between the first molar and the second premolar and at the maxillary side between the second premolar and the first molar (*Figure 21*). After use, they were disinfected with 70% alcohol and stored for the following session. Participants were instructed to hold the BiteFork® device parallel to the ground with their hands while performing the tasks, under visual control of a bar on the feedback screen. This bar corresponds with the vertical force (bite force) exerted on the sensors located between the participants back teeth.

The BiteFork® software enables the definition of a target force window, hence the color of the displayed bar changes from red to green as soon as the bite force lies within the predefined level (*Figure 34*).

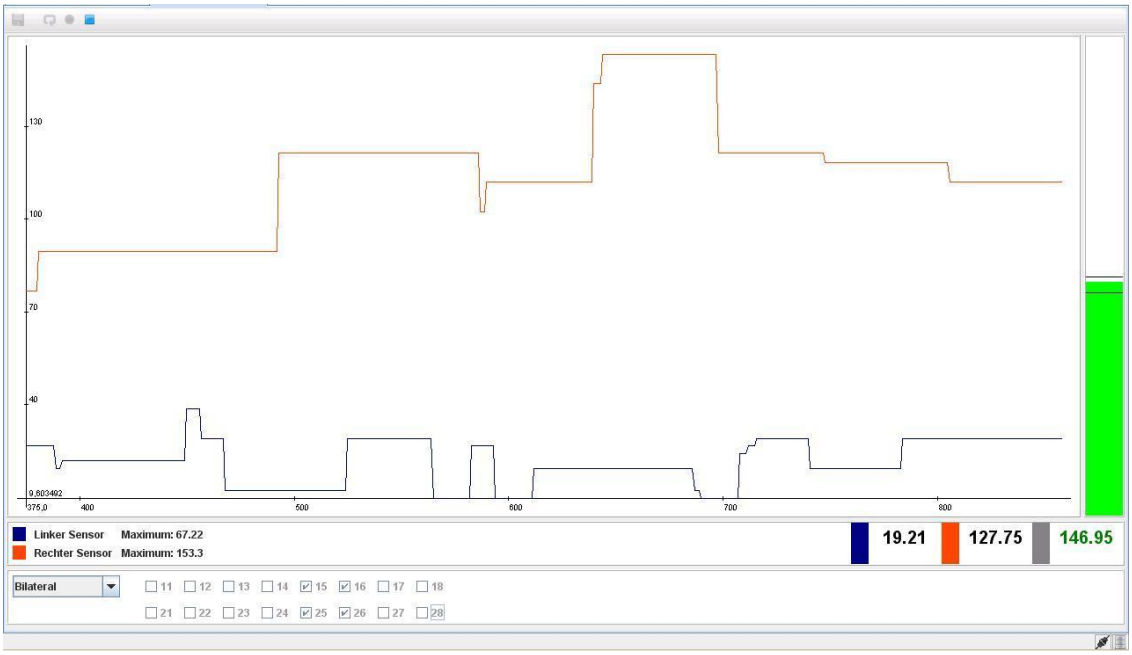


Figure 34: BiteFork® screen with target force window (140-150 N). The green bar in the target force window, achieved through biting on the sensor holders.

The EMG measurement was performed in triplicate for each of the three different bite force windows: 45-50N, 95-100N, 140-150N. Submaximal bite forces (SBF) were chosen as they represent the masticatory system's regular physiological activity (Schindler et al., 1998). Prior to each bite force level, subjects performed a test round to ensure their capability to complete the task and train the balancing of the bite force bar using the BiteFork® device and software. Afterwards, the EMG electrodes were connected to the amplifier/EMG device (*Figure 35*).

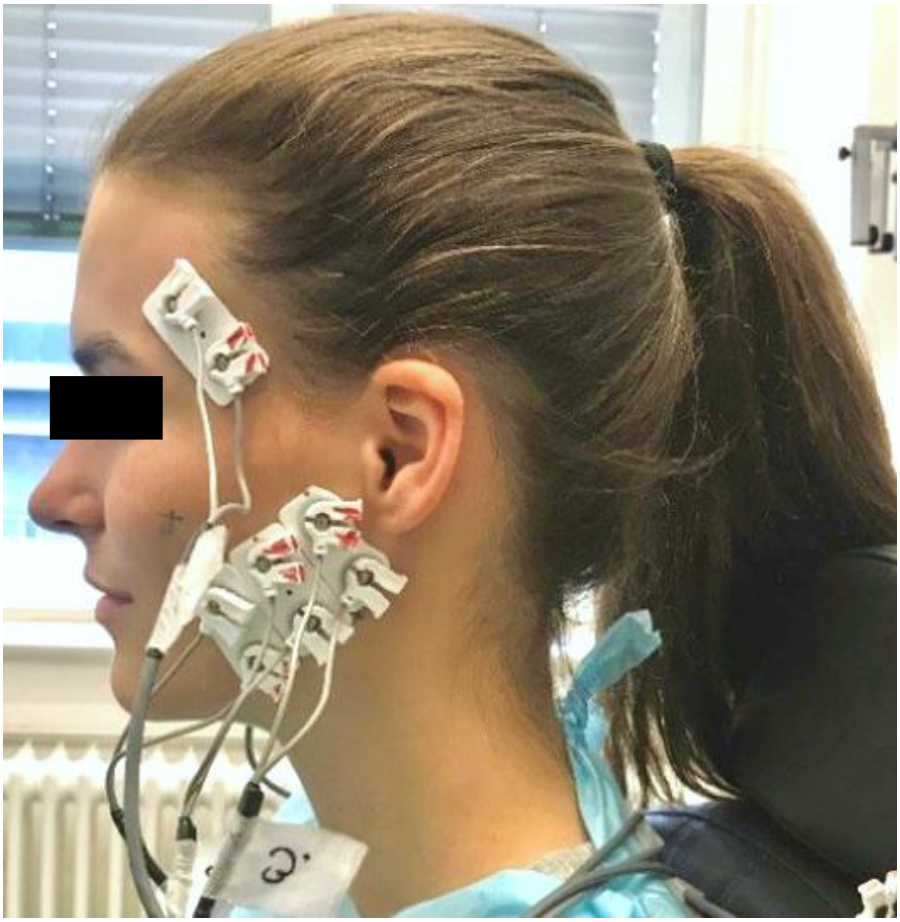


Figure 35: Wired electrodes on the temporalis and masseter during the EMG session.

The EMG recording was consequently activated and the baseline EMG activity of all eight muscles was controlled for artifacts or interferences of the signal. The examiner gave the following instructions per repitition: “Start” for the participant to begin teeth clenching, “Hold” as soon as the participant has reached the desired bite force level, and “Stop” after succesfully completing 5 seconds of recording. *Figure 36* shows the proband during measurement, holding the Bitefork® for force feedback with simultaneous EMG.

Figure 36: BiteFork® placement for force feedback with simultaneous EMG.

After three succesful repetitions at the same bite force level, the BiteFork® was taken out of the mouth for a short break of 90 seconds and the participant was advised to rest, in order to avoid muscle fatigue. Meanwhile, the three repetitions were saved on the PC and the BiteFork® was dried from saliva to avoid artefacts. All EMG recordings at the three bite force levels were performed according to this scheme and by using the following numbering of the eight electrode channels: 1st: right masseter anterior, 2nd: right masseter medial, 3rd: right masseter posterior, 4th: left masseter anterior, 5th: left masseter medial, 6th: left masseter posterior, 7th: temporalis right, 8th: temporalis left, resulting in three repetitions for each of the three levels (*Figure 37).*

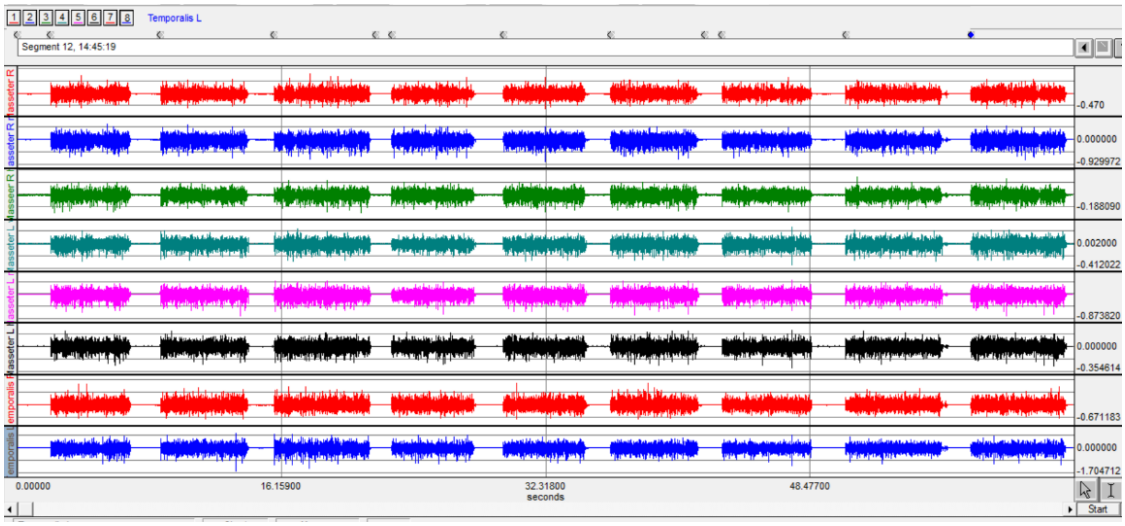


Figure 37: Illustration of a complete EMG recording, triplicate measurement at three different force levels during submaximal static contraction.

After completing all nine recordings, a second task was performed after a further break of 90 seconds. This test was to measure the maximum voluntary contraction using wet dental cotton rolls instead of the BiteFork®. A test round was not performed, to prevent muscles from fatiguing. The examiner placed the cotton rolls between the second premolars and first molars on each side of the jaw. During this test, also performed in triplicate with a 10 second break in-between, the examiner instructed the subjects to bite with all their force and encouraged them vocally to continue biting throughout the 5 seconds of recording (*Figure 38*).

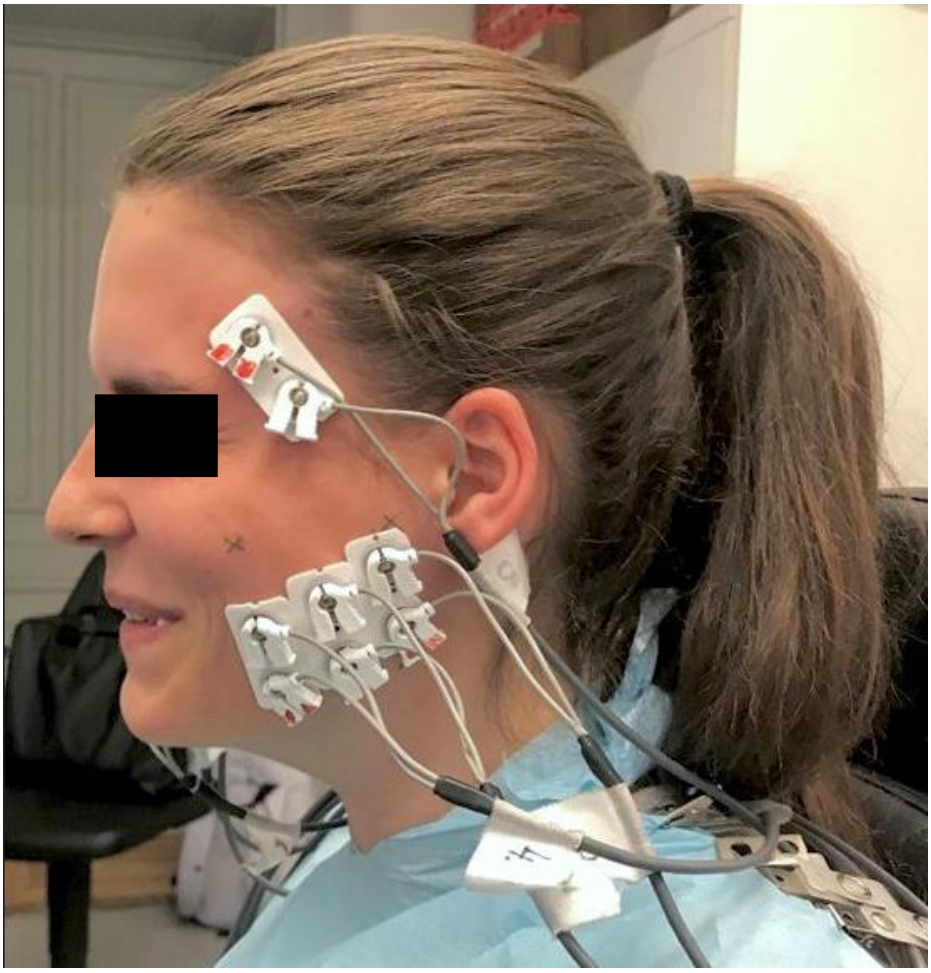


Figure 38: Maximum voluntary contraction/ clenching on dental cotton rolls.

After a short break of 90 seconds, followed the third task, which was fatigue testing. Subjects were instructed to exert their utmost bite force while clenching on the dental cotton rolls and were vocally encouraged to sustain the bite force for 30 seconds. In order to not exceed the scope of this dissertation, the fatigue test is not further evaluated within this thesis, as it shall be analyzed in detail in a separate publication. At the end of the session and after ensuring that all data were securely stored, the EMG wires and the electrodes were removed, and the face of the participant was cleaned from all lines and points by using alcohol. In a separate room, the external examiner informed the participants about the procedures in the following two weeks and whether they were in the inactive or intervention group, in order to ensure the blinding of the main investigator. For the intervention group, the silicone coverage of the buttons of the GrindCare® devices was removed and the level of stimulation adjusted to a noticeable but not painful or potentially sleep-disrupting grade.

In case the stimulation disrupted sleep or was too low, subjects were advised to increase or decrease the level of stimulation accordingly and were instructed to note it in their GrindCare® diary.

2.6.3. T2: Second session and T3: Third session

T2 Second session:

The procedure of session two was identical to session one. The electrodes were placed according to the coordinate sheet as described in T0.

At the end of the measurement, the buttons of the GrindCare® devices were covered with silicone once more (for the subjects of the intervention group) at stimulation level zero. Furthermore, subjects were instructed to inactivate their GrindCare® device every night by pushing the minus button until the level zero sound sounded, in case the silicone cover fell off. The last questionnaires were handed out and subjects were instructed to fill them out after the last time they applied the GrindCare® device.

T3 Third Session:

Measurements were identical to T1 and T2. At the end of the five trial weeks, participants returned the questionnaires, the GrindCare® device, and the GrindCare® diary. In appreciation of the invested time, every participant received small expense allowances, i.e., dental products like dental floss, toothbrushes, and toothpastes, kindly provided by Oral B (Procter & Gamble GmbH, Schwalbach am Taunus, Germany) and GUM® (Sunstar Suisse S.A., Etoy, Switzerland).

2.7. Data processing

2.7.1. Signal cleansing

All 27 generated EMG datasets, resulting from the EMG recordings at the three bite force levels that were performed in triplicate in each of the three sessions, had to be processed, i.e., remove of artifacts, isolate the steady signal needed for the comparisons, and cut to equal length. Moreover, the bite force datasets recorded with the BiteFork® software had to be time-matched with the EMG datasets, to enable the identification of the EMG signal that corresponds to the desired bite force. These files were referred to as the RMS % MVC at submaximal bite force 1-3 (SBF 1-3).

For that purpose, the EMG and BiteFork® data were imported into a custom-made software program based on Julia Programming Language (Julia v1.7) (Bezanson et al., 2012), implemented with the support of a computer scientist (Dr. Nikolaos Bakas, Associate Research Scientist, The Cyprus Institute). This software automatically inverted the bite force and EMG datasets to ensure that the end of these datasets, where the absence of bite force and the return of EMG signal to baseline could be identified, were well matched. Afterwards they were compared and cut concurrently so that they reached the same length. The software cut 500 ms of each inversed end (= start of the recording) to remove the initially unstable EMG signal parts and keep the more stable signal obtained during isometric contraction at the desired force level. The quality of the resulting recording was visually controlled by the main investigator and assessed whether 500 ms were sufficient to remove the uncontrolled dynamic contraction part. If insufficient, these datasets were processed again to cut bigger part of the data on both sides respectively. Furthermore, extreme outliers of the force values characterized by discordant values or signal peaks with a threefold difference to the standard deviation were removed, as these values would strongly influence further analysis. After time-matching, cutting the initial recordings, and removing the outliers the software identified the parts of the force signal that were the most stable. The EMG signal corresponding to the stable force signal was kept and all other data were discarded. At the end, the EMG data were put together and inverted, again. Lastly, all maximum voluntary contraction (MVC) data files were controlled for artifacts, characterized by abnormal signal peaks, and cleaned using the Acquire software which also stored the raw data.

2.7.2. Calculation of the root mean square (RMS)

After receiving the final SBF and MVC EMG datasets from the software, all 27 repetitions from each of the eight channels and all MVC data files were converted using the root mean square (RMS) algorithm, defined as the square root of the mean of the squares of a set of values. This full-wave rectification enables further data analysis and comparison since the raw EMG has the same amount of positive and negative values, resulting in a mean value of zero. This transformation was accomplished by the Acquire software. The RMS of the EMG signal was computed using the “Integrate” transformation in a Root Mean Square Average over three samples configuration. It consequently automatically integrated the selected channel after adjusting the settings in “integrate setup”.

2.7.3. Normalizing EMG

As already mentioned in chapter 2.4, the RMS EMG must be normalized to a reference value (MVC) to obtain the percent of maximum contraction capacity of the time of measure and allow quantitative comparison in between subjects and sessions (Konrad, 2005). Afterwards, the same custom-made software was implemented to conduct the following tasks.

2.7.3.1. Percent of MVC

The rectified (with the RMS algorithm) MVC files contain three repetitions for each of the eight channels, with an average duration of 3 s. For each repetition, the software determines the maximum (peak) of the MVC and isolates a 400 ms interval, 200 ms prior, and 200 ms post signal peak. From these intervals mean values (MV) are calculated, one for each of the three repetitions per channel. In turn, the program computes the average of the three mean values, resulting in eight MVC mean values for each session. The normalization is consequently computed with the respective mean values for each session. Each value of the SBF data is divided by the corresponding mean value, resulting in "percent of MVC". Lastly, a mean value is again calculated from the three repetitions of one level. The final results are three mean values per session for each channel, as presented in the overview of *Figure 39*.

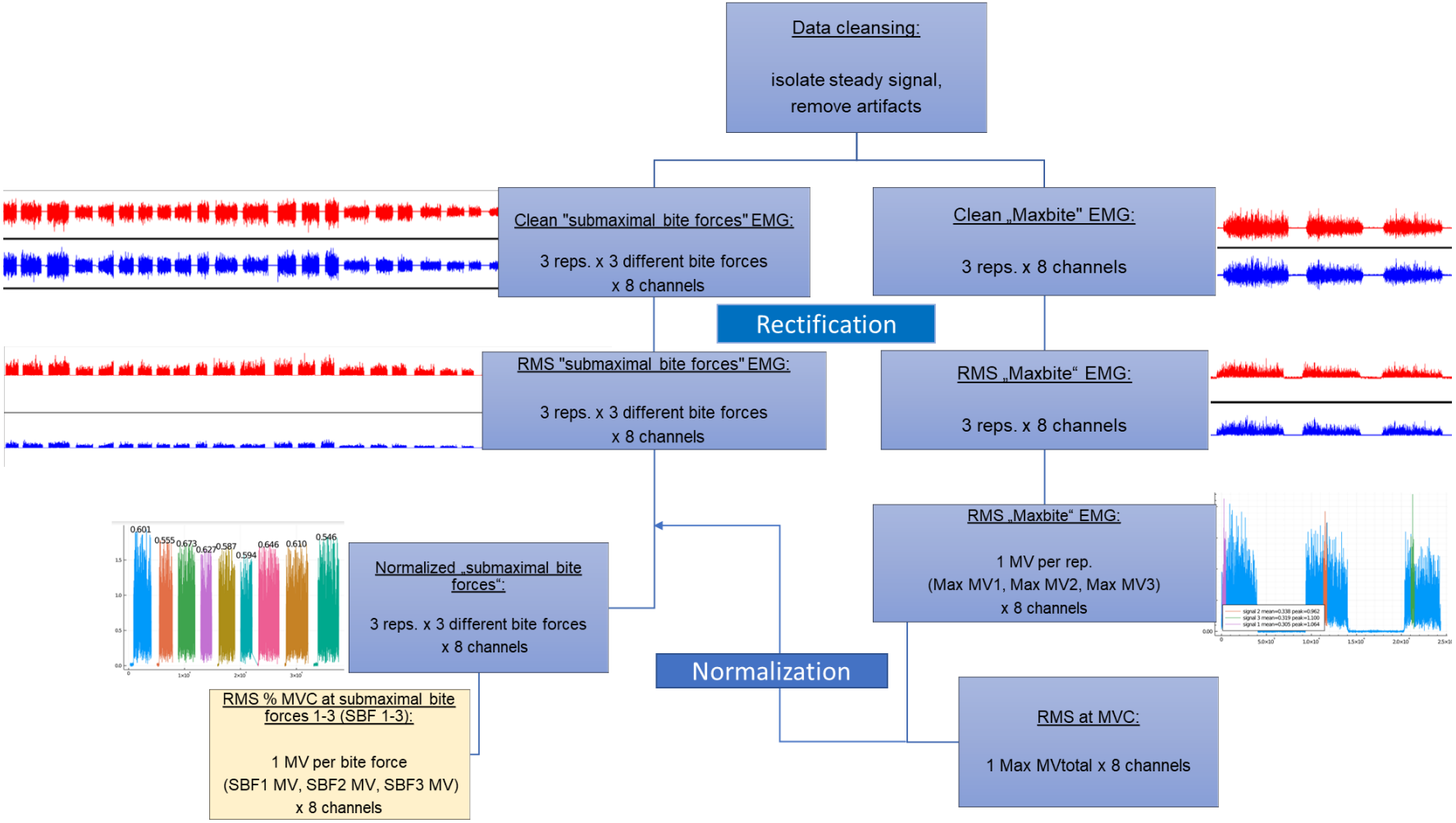


Figure 39: Scheme of data processing followed for each of the three recording sessions.

2.8. Statistics

The generated RMS % MVC of the matched subjects are metric values and descriptively represented by mean values and standard deviations in bar graphs for the graphical depiction of the numeric data, by using the software IBM® SPSS® Statistics 22.0 (Machines, 2013). For the inferential statistics, parametric and non-parametric tests were used. Parametric tests underlie specific assumptions about the parameters of the subject population, for instance, the assumption of a normal distribution. Non-parametric tests, however, are “distribution-free tests” as they do not assume that the data is normally distributed or follow a specific distribution. The only non-parametric test used in this study was the Friedman’s ANOVA, for the RMS at MVC, which examines whether more than two related groups differ and corresponds to the non-parametric version of a one-way repeated-measures ANOVA. The assumption which the non-parametric tests must meet is that the dependent variable is ordinally scaled. For the parametric tests, the independent samples t-test between groups was used to compare and objectify the data of both groups, intervention and control.

Furthermore, the triplicate repetitions of the same experiment during different periods allow a single factor repeated measures analysis of variance (rmANOVA) which is the most suitable variance analysis for matched (“balanced”) study designs with repeated measures. This statistical test analyzes if the mean values of each level differ with time, in T0, T1, T2, with and without intervention. The t-test and ANOVA premise a normal distribution of values and belong to the parametric tests. Normality was verified by using the Q–Q (quantile-quantile) plots. Lastly, the statistical significance level (*p*-value) was set on $p \leq 0.05$.

3. Results

The study started with one GrindCare® device and gradually the number of devices increased to ten in order to accelerate the data acquisition. Three to four study participants were measured each week and the measurement period was completed after approximately 18 months. 53 participants fulfilling the eligibility criteria were included in the study. 49 participants could complete the full study protocol. No adverse effects were reported by any of the study participants regarding any of the employed measurement methods. Since our subjects had to be matched, only 40 of these 49 probands were further evaluated in statistical tests. The selection was made by the external examiner based on matching pairs in the control- and intervention group. From the initial study sample, there were two dropouts before they began their first GrindCare® week, due to refusal of shaving off the beard for the EMG measurement. Further two probands reported pain in the TMD pain screener at the end of the examination period and one proband could not adhere the GC correctly, which resulted in excluding them from the final study sample.

3.1. Descriptive statistics

3.1.1. Study sample

Table 11 summarizes the demographic characteristics of the study participants who successfully completed the study protocol.

Table 11: Number of participants, sorted according to gender and age. Age min.= minimum, max.= maximum, mean, and SD= standard deviation.

Gender	*N*	*Age (min.)*	*Age (max.)*	*Age (mean)*	*SD*
Female	24	21.44	32.94	24.86	2.89
Male	16	21.25	39.56	27.81	5.40

20 participants were recruited for each group (*N*=40), with more female (*N*=24) than male (*N*=16) participants. The average age was equal for women and men.

The proportions of bruxers and non-bruxers within the study sample according to the different diagnoses for sleep bruxism, i.e., definite diagnosis (GC) and possible diagnosis (general questionnaire) is shown schematically in the following graph, *Figure 40*. The GC diagnosis is based on the 19 EMG/h for five nights as cutoffs (Stuginski-Barbosa et al., 2016).

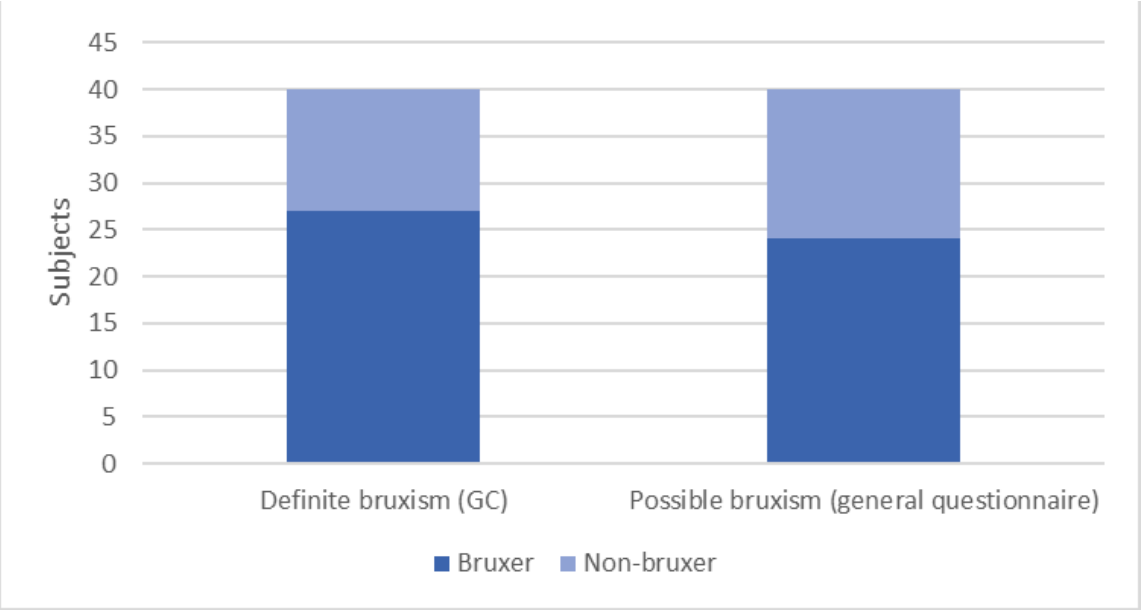


Figure 40: Proportions of bruxers and non-bruxers among the study sample, according to the definite diagnosis (GC measurement) and possible diagnosis (general questionnaire) in T1.

Among the included participants (*N*=40), there was a sleep bruxism proportion of 67.5 % (definite bruxers to non-bruxers 27:13), as per GrindCare® measurement in T1. According to the general questionnaire, the amount of possible bruxers were 60% (possible bruxers to non-bruxers 24:16).

3.1.2. EMG Data: Mean values at submaximal bite force

Figure 41, Figure 42, and *Figure 43* illustrate the mean values and standard deviations of the EMG activity (RMS % MVC at the first- third level of submaximal bite force) for the intervention and control group, in all three measurement sessions (T1-T3). The x-axis presents the eight muscle parts of the active (intervention) and inactive (control) group, while the y-axis shows the mean values and standard derivations of RMS as percent of MVC. The numeric data that belong to the graphs below are attached in the Appendix (*Table 31, Table 32, Table 33).*

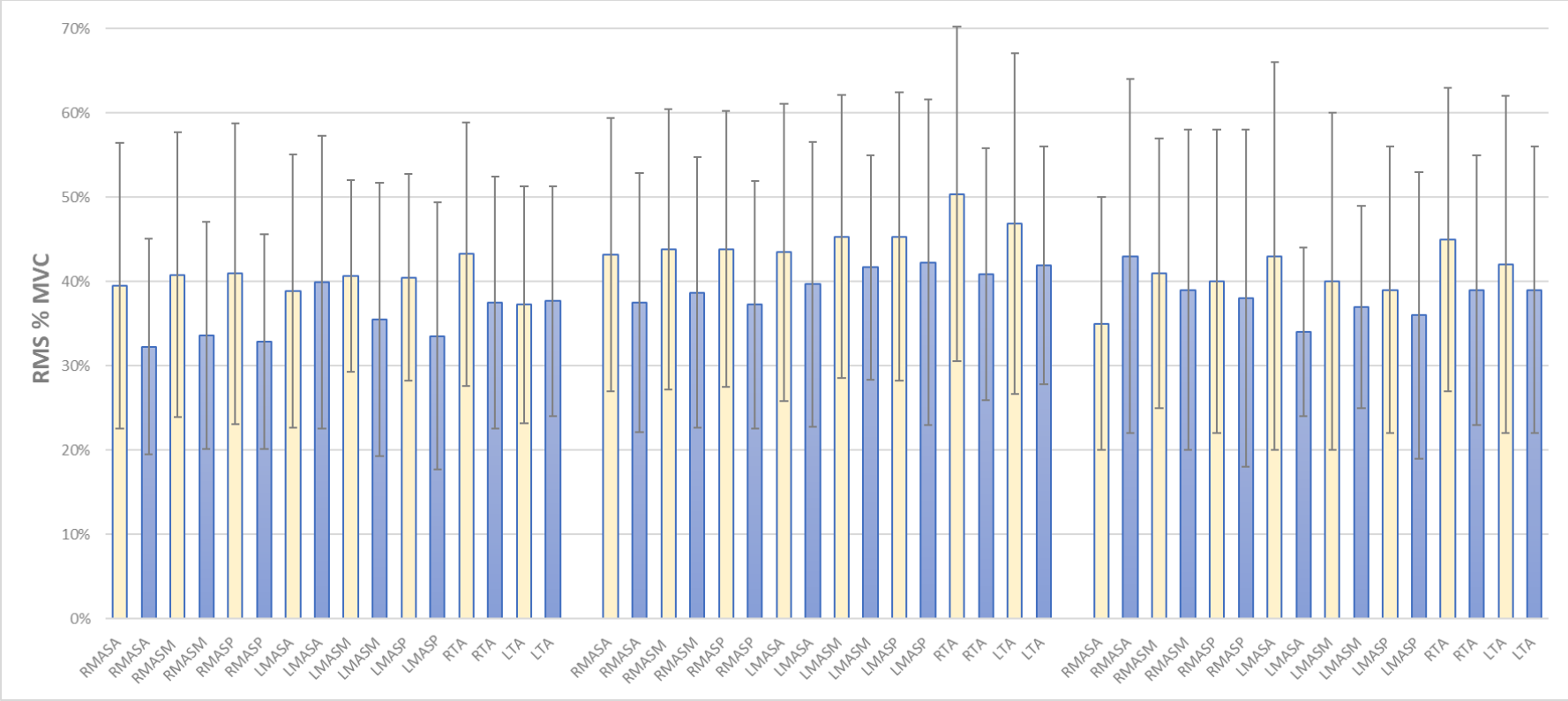


Figure 41: Mean values and standard derivations of the RMS % MVC at the first submaximal bite force in T1, T2, and T3 for the eight studied muscle regions and both study groups; yellow: intervention group, blue: control group.
RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

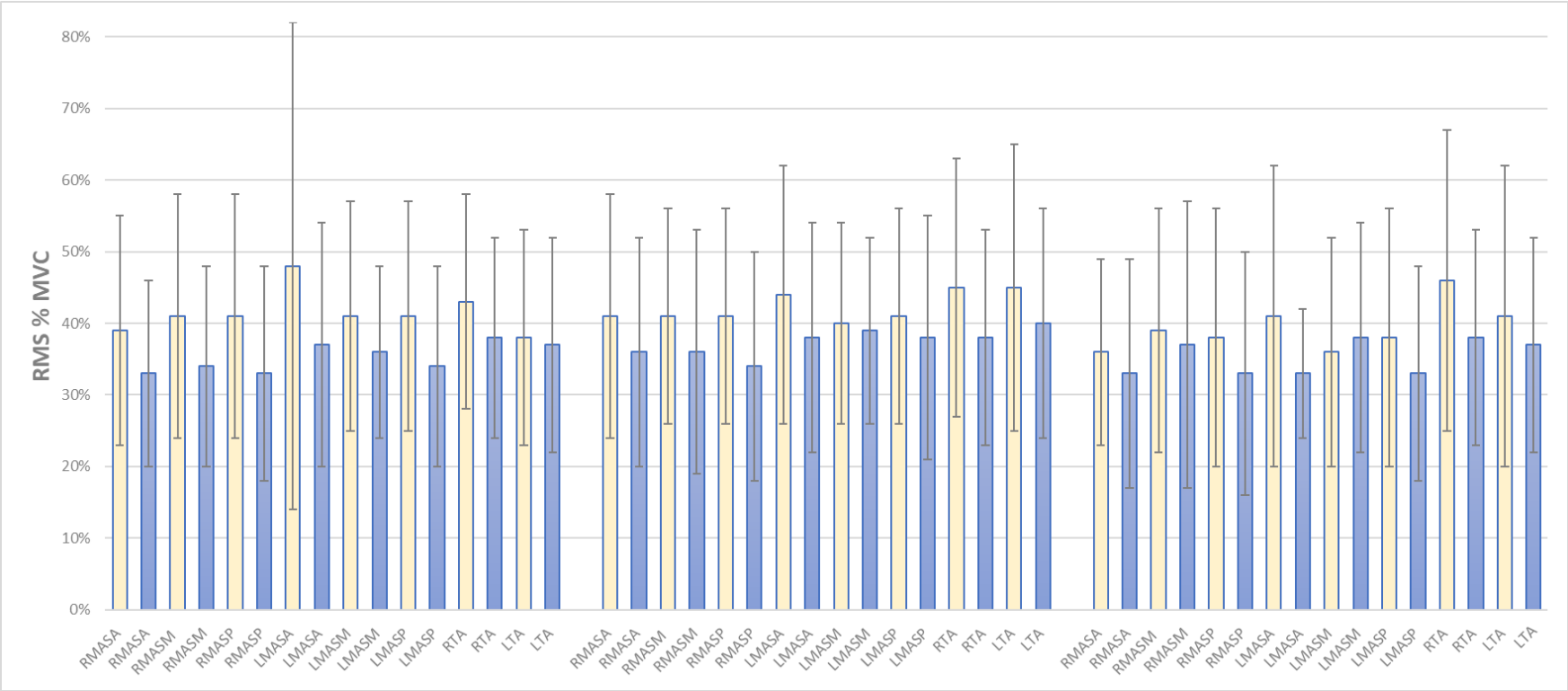


Figure 42: Mean values and standard derivations of the RMS % MVC at the second submaximal bite force in T1, T2, and T3 for the eight studied muscle regions and both study groups; yellow: intervention group, blue: control group.
RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

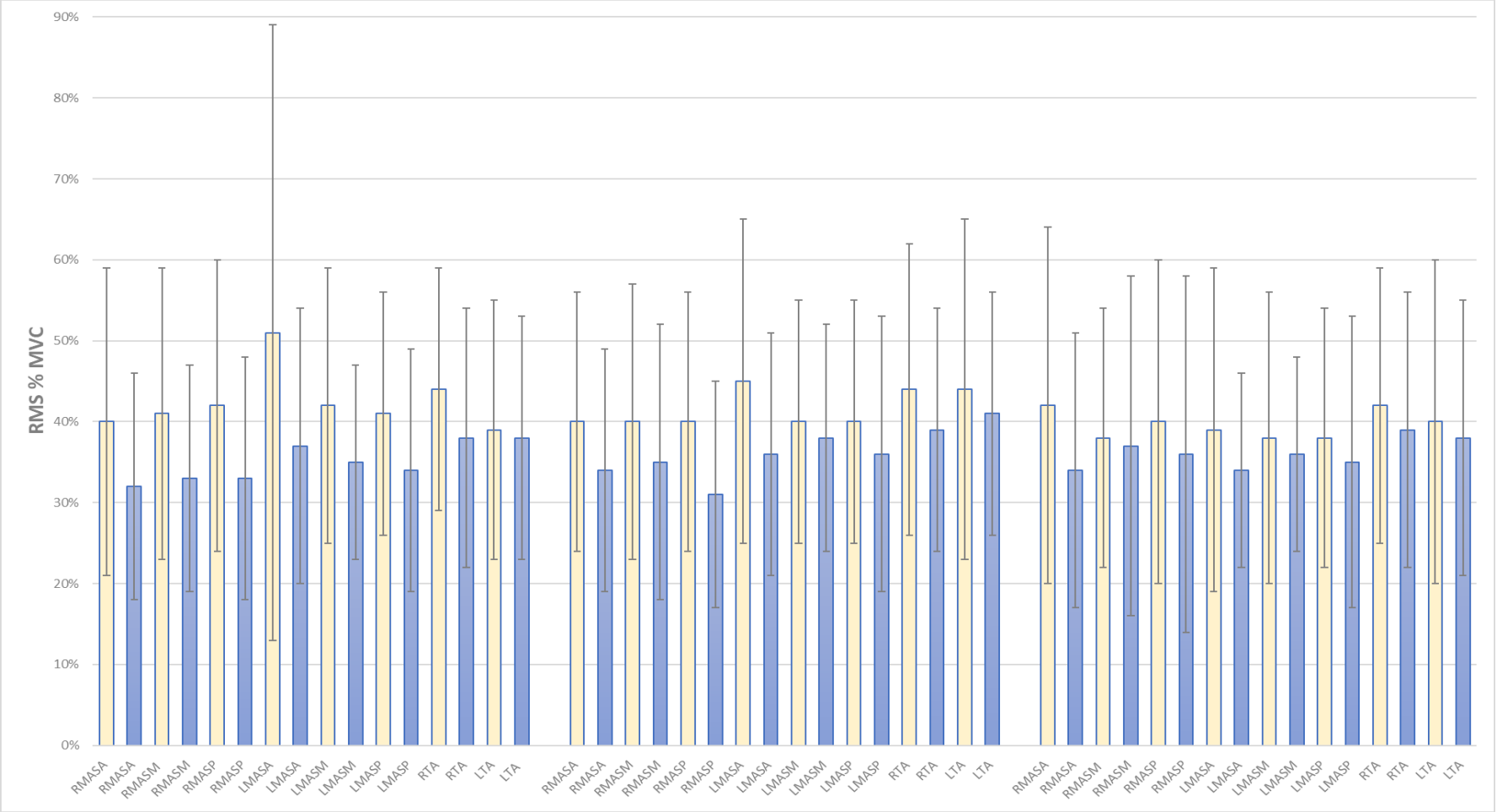


Figure 43: Mean values and standard derivations of the RMS % MVC at the third submaximal bite force in T1, T2, and T3 for the eight studied muscle regions and both study groups; yellow: intervention group, blue: control group.
RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

It is apparent that in all three sessions, the mean values of the active group are similar to the mean values of the inactive group. For the active group, the mean values of the RMS % MVC at first submaximal bite force resolve around a value of 40% MVC in T1 and increase slightly in T2, variating between 45-50% MVC. The decrease in T3 ranges between 35-43 % MVC. The inactive group seems to have a broader range between 32-42%.

The mean values of RMS % MVC at second submaximal bite force of both groups (active and inactive) do not differ in T1, T2, and T3, ranging between 37-47 % MVC and 32-40% MVC respectively. The mean values of the RMS % MVC at the second and third submaximal bite force of the active group are close to those of the inactive group. The active group starts high, decreasing along with each following measurement session (up to 50% MVC in T1, 45% MVC in T2, and 40% MVC in T3). The inactive group, however, remains more consistent (31- 40% MVC).

3.1.3. Mean values of the BiteFork® data

The bite force in Newton (N) measured by the BiteFork® between the back teeth of the participants is summarized for all three force levels. *Table 12* displays the sum of the mean values of the two BiteFork® sensors (right and left sensor) along with their respective standard deviations of all participants for the first, second and third force level and their triplicate repetition according to the measurement session (T1, T2, and T3).

Table 12: Sum of the mean values and standard deviations of the right and left BiteFork® sensors in Newton (N), N=40 (all subjects). Displayed according to the measurement session (T1, T2, and T3), the bite force level (level 1, 2 and 3) and the triplicate measurement.

Measurement session	**Force level 1**			**Force level 2**			**Force level 3**		
	1st rep.	*2nd rep.*	*3rd rep.*	*1st rep.*	*2nd rep.*	*3rd rep.*	*1st rep.*	*2nd rep.*	*3rd rep.*
T1	141.53	141.60	140.87	141.66	141.84	141.87	141.37	141.49	140.82
T2	141.74	141.64	141.56	142.34	141.85	142.00	142.17	142.66	142.22
T3	140.52	141.52	141.72	141.28	141.65	141.92	142.27	141.78	141.52
Total MV + StDev.	141.41 +/- 4.83			141.82 +/- 4.43			141.81 +/- 5.85		

The table above shows a total mean value of 141-142 N for all levels, contrary to the initial study plan which intended a clear distinction between three bite forces, ranging between 45-50N, 95-100N, 140-150N. The calculation for the actual exerted bite force (141-142N) is explained below.

3.1.3.1. Analysis of the BiteFork® data

The comparison of the BiteFork® data and the visual comparison of the EMG data (RMS % MVC at submaximal and maximal bite force) revealed that mean values of all three submaximal bite forces show no difference, despite the different bite force levels set. Based on that finding, a validation of the BiteFork® system was necessary. A Universal Testing Machine (ProLine, ZwickRoell GmbH & Co. KG, Ulm, Germany) was used to verify the BiteFork® device.

Unfortunately, the testing results did not match with the settings of the device. The two sensors measured different values which were significantly lower than the initially selected 50N, 100N, and 150N (real values: 25N - 40N - 65N). Although the manufacturer had confirmed the ability of the device to measure bite force in the selected range (50-150N) reliably and the calibration of the sensors followed the instructions of the manufacturer, the BiteFork® display/software falsely indicated different bite forces than measured in reality. The calibration of the BiteFork® data with the Universal Testing Machine allowed the conversion of the measured BiteFork® data to Newton to enable insight into the amount of the real exerted force, which amounted to 50N on average (25N per side, since the measurement was bilateral).

Assuming that bite force and EMG correlate linearly and using the Universal Testing Machine data, a calculation of the amount of the maximal bite force was possible. Hence, the maximal bite force amounted to 450N on average. One probable explanation of this phenomenon is that the actual bite forces of 25N, 40N, and 65N are below the sensors' capacity and the device's low resolution was unable to distinguish between the three different submaximal bite forces. The assumption is that the low resolution of the device did not allow for the grading set and therefore measured most likely only the lowest force possible to measure.

Consequently, the bite force could not be adequately presented. For the data analysis, the original study protocol was adapted, i.e., regarding the submaximal bite force levels as three distinct forces, but not the values 50/100/150N, and expanded by the new knowledge regarding the submaximal bite force levels as having practically the same bite force (25N per side) throughout the whole experiment. This was done as the subjects were informed that with each task the bite forces increased and truly reported differences in the effort required.

3.1.4. EMG data: Mean values of RMS at maximal bite force

The EMG activity during MVC serves as the reference value for calculating the submaximal RMS % MVC that enables the comparison between probands. *Table 13* illustrates the mean values of the rectified maximal bite forces in Volt (RMS) exerted by each subject in each measurement session (T1- T3).

Table 13: EMG data (V), mean RMS values of the maximal bite force; eight muscle parts of each subject in T1, T2 and T3. RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Probands	Muscle areas for each measurement session (T1-3)																							
	RMASA			RMASM			RMASP			LMASA			LMASM			LMASP			RTA			LTA		
	T1	*T2*	*T3*	*T1*	*T2*	*T3*	*T1*	*T2*	*T3*	*T1*	*T2*	*T3*	*T1*	*T2*	*T3*	*T1*	*T2*	*T3*	*T1*	*T2*	*T3*	*T1*	*T2*	*T3*
Prob. 1	0.17	0.22	0.28	0.22	0.30	0.29	0.14	0.20	0.30	0.15	0.19	0.25	0.21	0.29	0.30	0.20	0.23	0.27	0.28	0.34	0.54	0.34	0.45	0.35
Prob. 2	0.18	0.16	0.16	0.32	0.22	0.29	0.21	0.18	0.27	0.16	0.04	0.19	0.19	0.17	0.26	0.19	0.19	0.20	0.22	0.22	0.22	0.23	0.19	0.18
Prob. 3	0.31	0.26	0.33	0.30	0.28	0.43	0.14	0.11	0.18	0.40	0.10	0.18	0.28	0.24	0.33	0.14	0.09	0.12	0.30	0.34	0.33	0.43	0.44	0.40
Prob. 6	0.57	0.50	0.40	0.57	0.42	0.47	0.30	0.20	0.22	0.58	0.46	0.45	0.53	0.38	0.51	0.27	0.20	0.22	0.56	0.48	0.40	0.73	0.43	0.47
Prob. 7	0.29	0.22	0.21	0.34	0.22	0.28	0.17	0.13	0.11	0.32	0.34	0.33	0.35	0.39	0.46	0.22	0.15	0.24	0.23	0.24	0.25	0.30	0.27	0.29
Prob. 8	0.71	0.55	0.63	0.53	0.46	0.53	0.23	0.19	0.16	0.46	0.30	0.29	0.53	0.38	0.34	0.23	0.16	0.17	0.54	0.47	0.51	0.37	0.32	0.31
Prob. 10	0.25	0.44	0.25	0.30	0.43	0.27	0.20	0.42	0.13	0.07	0.4	0.10	0.37	0.44	0.42	0.18	0.44	0.17	0.15	0.3	0.22	0.16	0.44	0.19
Prob. 11	0.31	0.46	0.19	0.50	0.51	0.31	0.21	0.24	0.24	0.13	0.34	0.08	0.43	0.53	0.54	0.27	0.26	0.26	0.26	0.39	0.38	0.30	0.42	0.26
Prob. 12	0.20	0.20	0.24	0.22	0.29	0.23	0.15	0.16	0.10	0.11	0.10	0.16	0.33	0.33	0.32	0.19	0.14	0.19	0.24	0.26	0.30	0.26	0.27	0.24
Prob. 13	0.15	0.13	0.20	0.22	0.25	0.23	0.12	0.10	0.12	0.22	0.06	0.24	0.29	0.30	0.33	0.11	0.11	0.13	0.19	0.35	0.27	0.26	0.29	0.33
Prob. 14	0.45	0.30	0.36	0.32	0.27	0.28	0.21	0.17	0.13	0.08	0.22	0.17	0.31	0.22	0.18	0.21	0.14	0.10	0.27	0.29	0.32	0.31	0.28	0.28
Prob. 15	0.16	0.17	0.16	0.30	0.35	0.30	0.27	0.23	0.31	0.13	0.11	0.11	0.36	0.54	0.49	0.30	0.30	0.34	0.41	0.33	0.39	0.39	0.26	0.27
Prob. 16	0.45	0.74	0.49	0.52	0.74	0.59	0.21	0.24	0.20	0.12	0.26	0.17	0.41	0.71	0.52	0.17	0.35	0.21	0.30	0.33	0.26	0.27	0.37	0.31
Prob. 17	0.35	0.32	0.20	0.26	0.34	0.23	0.10	0.11	0.09	0.09	0.10	0.04	0.46	0.41	0.22	0.16	0.13	0.12	0.43	0.39	0.36	0.37	0.41	0.31
Prob. 18	0.53	0.42	0.56	0.63	0.39	0.55	0.22	0.17	0.26	0.17	0.27	0.30	0.81	0.64	0.86	0.60	0.32	0.39	0.33	0.25	0.29	0.56	0.32	0.41
Prob. 19	0.22	0.27	0.18	0.29	0.33	0.37	0.16	0.10	0.15	0.24	0.09	0.30	0.36	0.30	0.36	0.20	0.12	0.19	0.31	0.25	0.28	0.36	0.31	0.36
Prob. 20	0.31	0.30	0.28	0.41	0.27	0.30	0.13	0.16	0.13	0.10	0.17	0.13	0.35	0.25	0.23	0.15	0.11	0.16	0.40	0.45	0.48	0.37	0.36	0.36
Prob. 21	0.30	0.29	0.29	0.36	0.47	0.41	0.10	0.19	0.12	0.16	0.30	0.33	0.45	0.43	0.38	0.15	0.16	0.18	0.23	0.28	0.26	0.20	0.21	0.20
Prob. 22	0.22	0.27	0.25	0.27	0.30	0.35	0.13	0.16	0.17	0.07	0.05	0.21	0.20	0.31	0.27	0.10	0.15	0.13	0.19	0.21	0.23	0.11	0.15	0.17
Prob. 23	0.27	0.29	0.35	0.27	0.40	0.31	0.13	0.20	0.16	0.13	0.04	0.04	0.15	0.20	0.22	0.12	0.13	0.15	0.26	0.30	0.32	0.21	0.24	0.26
Prob. 24	0.77	0.60	0.54	0.65	0.52	0.53	0.21	0.23	0.30	0.70	0.45	0.13	0.68	0.38	0.41	0.28	0.19	0.24	0.40	0.37	0.26	0.33	0.40	0.26
Prob. 25	0.25	0.26	0.18	0.28	0.29	0.26	0.14	0.14	0.16	0.31	0.26	0.31	0.25	0.23	0.23	0.17	0.15	0.13	0.46	0.38	0.34	0.48	0.48	0.49
Prob. 26	0.34	0.4	0.31	0.41	0.44	0.35	0.26	0.4	0.24	0.41	0.42	0.37	0.32	0.4	0.32	0.22	0.4	0.18	0.42	0.45	0.42	0.48	0.44	0.48
Prob. 27	0.46	0.61	0.56	0.51	0.62	0.55	0.18	0.20	0.24	0.61	0.79	1.03	0.49	0.45	0.70	0.18	0.15	0.23	0.35	0.34	0.42	0.25	0.44	0.41
Prob. 28	0.23	0.20	0.16	0.27	0.30	0.16	0.12	0.12	0.08	0.07	0.08	0.04	0.37	0.31	0.23	0.17	0.12	0.17	0.23	0.23	0.18	0.25	0.15	0.18
Prob. 29	0.19	0.16	0.22	0.22	0.24	0.26	0.10	0.20	0.14	0.06	0.06	0.07	0.24	0.33	0.38	0.14	0.18	0.25	0.27	0.22	0.38	0.32	0.28	0.46
Prob. 30	0.61	0.68	0.58	0.54	0.89	0.77	0.21	0.30	0.34	0.20	0.25	0.25	0.69	0.86	0.81	0.26	0.30	0.23	0.22	0.34	0.27	0.42	0.56	0.70
Prob. 31	0.34	0.39	0.28	0.35	0.37	0.23	0.21	0.30	0.20	0.11	0.24	0.16	0.37	0.37	0.49	0.18	0.19	0.27	0.20	0.28	0.25	0.25	0.26	0.23

Prob. 32	0.31	0.20	0.22	0.51	0.42	0.47	0.25	0.21	0.17	0.39	0.12	0.09	0.68	0.45	0.39	0.36	0.17	0.24	0.49	0.25	0.27	0.45	0.22	0.32
Prob. 33	0.20	0.17	0.23	0.17	0.24	0.23	0.10	0.14	0.10	0.05	0.03	0.05	0.18	0.17	0.14	0.09	0.11	0.08	0.09	0.17	0.18	0.14	0.12	0.15
Prob. 34	0.26	0.23	0.22	0.36	0.34	0.27	0.24	0.21	0.20	0.11	0.06	0.06	0.31	0.35	0.23	0.19	0.15	0.12	0.28	0.29	0.22	0.37	0.40	0.30
Prob. 35	0.06	0.08	0.09	0.10	0.07	0.09	0.05	0.04	0.05	0.03	0.02	0.05	0.12	0.08	0.11	0.07	0.06	0.09	0.16	0.14	0.20	0.14	0.15	0.17
Prob. 36	0.18	0.19	0.16	0.35	0.35	0.34	0.24	0.28	0.12	0.05	0.08	0.06	0.28	0.20	0.27	0.21	0.15	0.21	0.37	0.26	0.27	0.23	0.19	0.25
Prob. 37	0.29	0.29	0.37	0.36	0.23	0.31	0.20	0.13	0.13	0.28	0.24	0.07	0.30	0.27	0.29	0.11	0.10	0.10	0.19	0.21	0.22	0.18	0.23	0.18
Prob. 38	0.30	0.31	0.44	0.26	0.26	0.26	0.12	0.12	0.17	0.18	0.18	0.08	0.24	0.21	0.31	0.12	0.11	0.12	0.12	0.18	0.18	0.09	0.10	0.12
Prob. 43	0.58	0.51	0.82	0.51	0.45	0.73	0.42	0.35	0.37	0.64	0.66	0.19	0.65	0.53	1.01	0.55	0.43	0.56	0.45	0.37	0.28	0.36	0.28	0.30
Prob. 46	0.37	0.65	0.53	0.31	0.37	0.30	0.18	0.18	0.15	0.16	0.21	0.27	0.32	0.53	0.45	0.17	0.27	0.19	0.19	0.26	0.21	0.25	0.25	0.18
Prob. 47	0.47	0.20	0.47	0.38	0.25	0.34	0.21	0.13	0.13	0.22	0.08	0.13	0.46	0.31	0.31	0.22	0.17	0.17	0.30	0.28	0.22	0.22	0.21	0.18
Prob. 48	0.34	0.32	0.41	0.44	0.48	0.56	0.23	0.19	0.21	0.10	0.12	0.45	0.39	0.34	0.37	0.22	0.11	0.16	0.25	0.22	0.18	0.25	0.23	0.18
Prob. 49	0.51	0.48	0.39	0.36	0.32	0.33	0.20	0.20	0.17	0.17	0.12	0.36	0.33	0.32	0.34	0.20	0.20	0.22	0.40	0.27	0.30	0.23	0.22	0.24
	T1								T2									T3						
Total MV +/- Stdev.	0.29 +/- 0.15								0.28 +/- 0.15									0.28 +/- 0.15						

The total mean value of the maximal bite force resolves around 0.28-0.29 V which corresponds to 450N according to the BiteFork® device, as explained above in chapter 3.1.3.1.

3.2. Inferential statistics

For statistical analysis various tests and comparisons were implemented. *Figure 44* presents a general overview of the subsequent analysis.

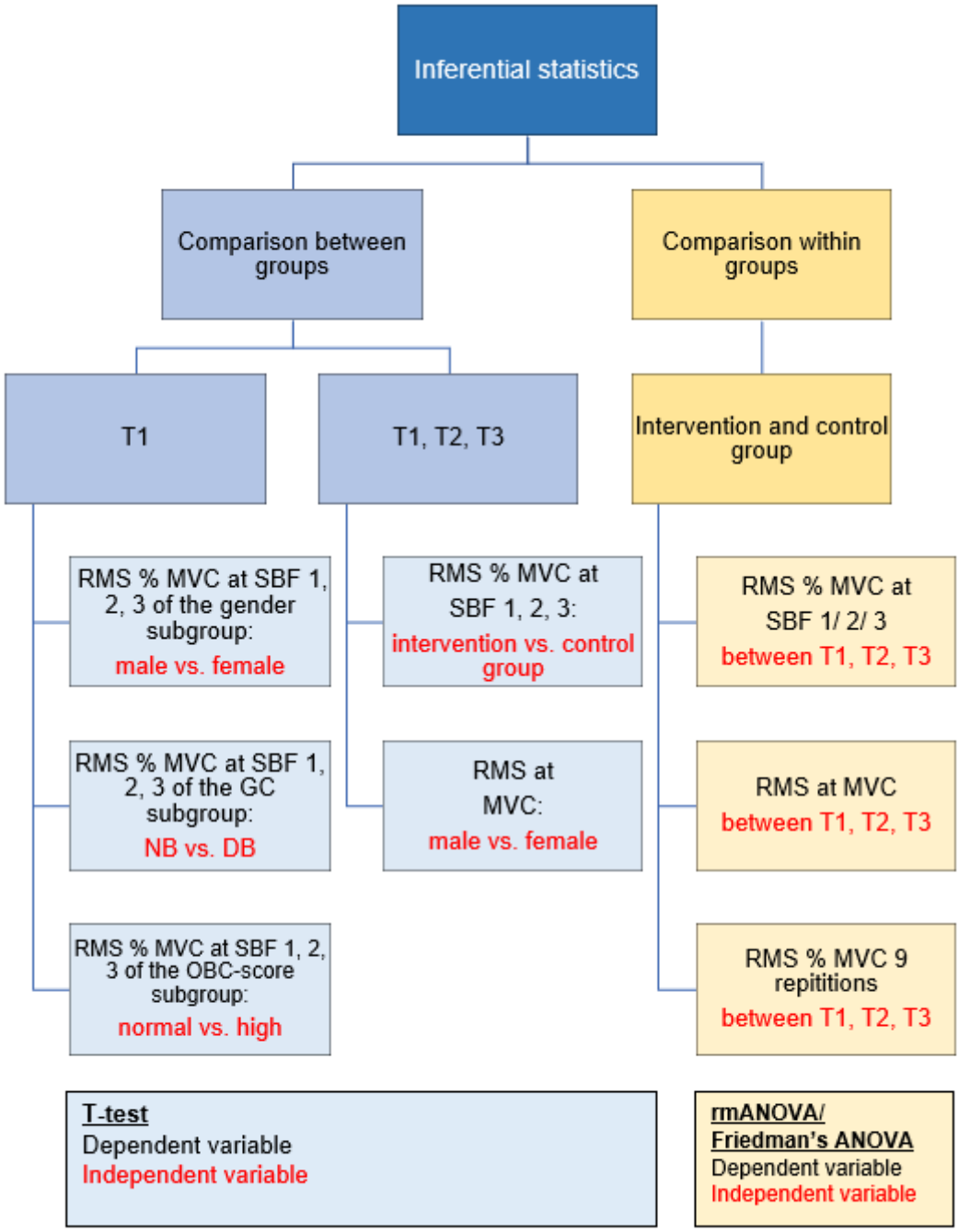


Figure 44: Inferential statistics, variables compared, and tests applied.

3.3. Normal distribution of the metric study variables

The t-test and ANOVA premise a normal distribution of values which was verified using Q–Q (quantile-quantile) plots, as most variables are close to the diagonal. *Figure 45* shows one example of a Q-Q plot for the right masseter anterior (inactive group).

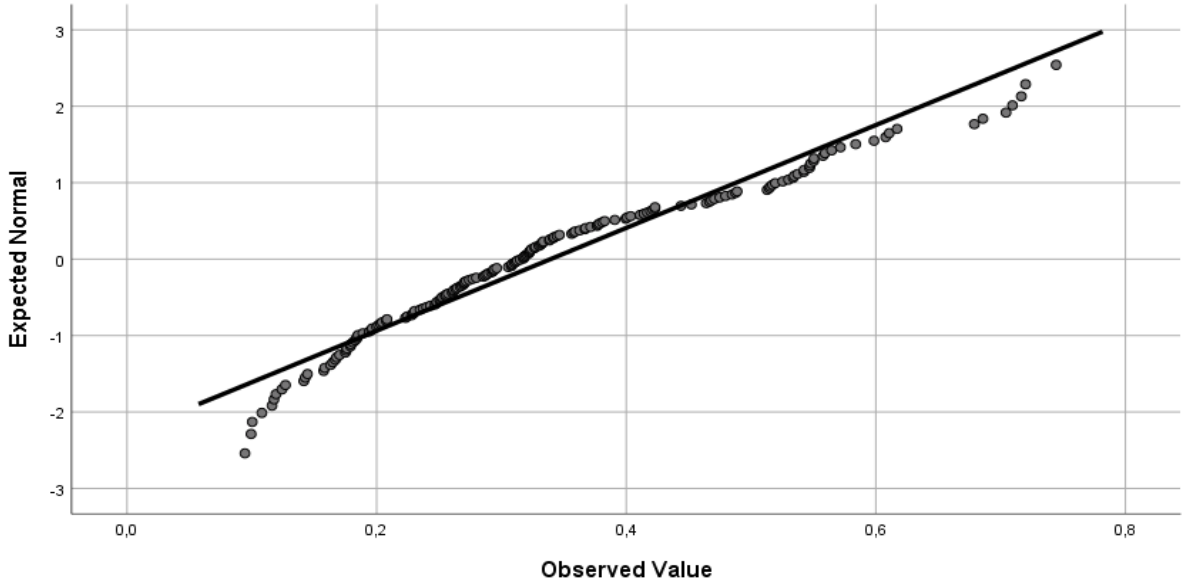


Figure 45: Normality confirmation with a Q-Q plot for RMASA (right masseter anterior); inactive group.

3.4. Tests between-groups

3.4.1. Comparison of RMS % MVC at submaximal bite force tasks

As mentioned above, the study sample was classified into the intervention - and control group. However, several other sample characteristics could be factors of heterogeneity within the study sample, such as gender (female and male) and bruxism behavior, verified by GrindCare® and OBC-score. The characteristics gender, definite bruxism, and possible bruxism of the study sample function as independent variables, influencing the comparison of the resulting RMS % MVC of each muscle area (dependent variable). Differences between these categories (male vs. female, definite bruxer vs. non-bruxer, and low vs. high OBC score) were tested with t-tests in T1, to explore whether there are significant differences regarding the aforementioned sample characteristics at the beginning of the study. *Table 14* displays the performed tests and results.

Table 14: T-test; Comparison of the RMS % MVC of the eight studied muscle regions, for various study sample subgroups, at all three submaximal bite force levels (SBF 1-3) in T1.
p= Statistical significance (p-value), t= t-variable, df= degrees of freedom. Statistical significance was set at p≤0.05. The study sample is grouped according to gender: male vs. female; GrindCare® (GC) diagnosis: definite bruxer vs. non-bruxer; and OBC score: low vs. high.
RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Sample subgroups	RMS % MVC	Muscle areas																							
		RMASA			RMASM			RMASP			LMASA			LMASM			LMASP			RTA			LTA		
		t	*df*	*P*	*t*	*df*	*P*	*t*	*df*	*P*	*t*	*df*	*P*	*t*	*df*	*P*	*t*	*df*	*P*	*t*	*df*	*P*	*t*	*df*	*p*
Gender (male vs. female)	*SBF1*	-0.75	38	0.46	-0.70	38	0.49	-0.92	38	0.36	-1.32	38	0.20	-0.96	38	0.34	-0.84	38	0.40	-1.45	38	0.16	-1.35	38	0.18
	SBF2	-1.12	38	0.27	-1.06	38	0.30	-1.27	38	0.21	-0.87	38	0.39	-1.07	38	0.29	-0.90	38	0.37	-1.80	38	0.08	-1.79	38	0.08
	SBF3	-0.99	38	0.33	-1.03	38	0.31	-1.39	38	0.17	-0.81	38	0.42	-0.97	38	0.34	-1.27	38	0.21	-1.90	38	0.07	-1.84	38	0.07
GC diagnosis (definite bruxer vs. non-bruxer)	*SBF1*	1.01	38	0.32	1.26	38	0.22	0.67	38	0.51	1.75	38	0.09	1.45	38	0.15	0.72	38	0.48	0.90	38	0.37	1.52	38	0.14
	SBF2	1.20	38	0.24	1.45	38	0.15	0.80	38	0.43	1.01	38	0.32	1.52	38	0.14	0.99	38	0.33	1.02	38	0.32	1.68	38	0.10
	SBF3	0.62	38	0.54	0.98	38	0.33	0.40	38	0.69	0.71	38	0.48	1.02	38	0.31	0.68	38	0.50	0.56	38	0.58	1.07	38	0.29
OBC score (low vs. high score)	*SBF1*	0.36	38	0.72	0.17	38	0.87	0.05	38	0.96	-0.02	38	0.99	0.78	38	0.44	0.36	38	0.72	1.07	38	0.29	0.81	38	0.42
	SBF2	0.25	38	0.81	0.07	38	0.95	-0.08	38	0.93	0.04	38	0.96	0.78	38	0.44	0.38	38	0.71	0.97	38	0.34	0.77	38	0.45
	SBF3	0.10	38	0.92	0.07	38	0.95	0.01	38	1.00	-0.11	38	0.91	0.58	38	0.56	0.55	38	0.58	0.74	38	0.46	0.65	38	0.52

The first analysis studied the difference between the variable gender entailing the groups male and female, regarding their RMS % MVC. The second independent variable was “bruxism diagnosis”, measured by the portable GrindCare® device. This GC diagnosis is based on the mentioned 19 EMG/h for five nights as cutoffs (Stuginski-Barbosa et al., 2016). Furthermore, the study participants were grouped according to the subjectively perceived bruxism behavior as expressed with the OBC score, which could be high (score > 24) or low (score ≤ 24). The performed t-tests examined whether the RMS % MVC (dependent variable) depends on the gender of the subject, the bruxism activity, or the OBC-score (independent variables) in T1. Since neither subgroup (male vs. female, non-bruxers vs. bruxers, high vs. low OBC-score) showed any statistically significant differences ($p > 0.05$), the data could be merged into one pool for the intervention group and one for the control group. Hence, both study groups were considered relatively homogeneous regarding gender and bruxism activity at baseline. Due to the matching of both groups, the data profiles have been listed together in the tables below.

3.4.2. Comparison of RMS % MVC (intervention vs. control group)

The between-groups analysis (intervention vs. control group) compared the respective RMS % MVC at submaximal bite force tasks of each muscle area by using a t-test. The statistical variables (*p*-value, t-value, and df) are listed according to the measurement session (T1-3) in the tables below (*Table 15* presents the RMS % MVC at SBF1, *Table 16* at SBF2, and *Table 17* at SBF 3).

Table 15: T-test; Comparison of the RMS % MVC of the eight studied muscle regions at submaximal bite force level 1 for the intervention vs. control group. p= statistical significance (p-value), t= t-variable, df= degrees of freedom. T1-3 indicate the timepoints. Statistical significance was set at p≤0.05 and is denoted with asterisk (*). RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	***p* (T1)**	***p* (T2)**	***p* (T3)**	***t* (T1)**	***t* (T2)**	***t* (T3)**	***df* (T1-3)**
RMASA	0.085	0.236	0.197	-1.817	-1.223	-1.338	19
RMASM	0.121	0.272	0.759	-1,623	-1.132	-0.311	19
RMASP	0.109	0.128	0.742	-1.683	-1.593	-0.334	19
LMASA	0.807	0.440	0.165	0.248	-0.789	-1.443	19
LMASM	0.199	0.308	0.522	-1.331	-1.047	-0.652	19
LMASP	0.077	0.433	0.583	-1.871	-0.801	-0.558	19
RTA	0.156	**0.033***	0.245	-1.479	-2.302	-1.20	19
LTA	0.918	0.313	0.632	0.104	-1.036	-0.487	19

Table 16: T-test; Comparison of RMS % MVC of the eight studied muscle regions at submaximal bite force level 2 for the intervention vs. control group. p= statistical significance (p-value), t= t-variable, df= degrees of freedom. T1-3 indicate the timepoints. Statistical significance was set at p≤0.05. RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	***p* (T1)**	***p* (T2)**	***p* (T3)**	***t* (T1)**	***t* (T2)**	***t* (T3)**	***df* (T1-3)**
RMASA	0.144	0.242	0.538	-1.522	-1.207	-0.627	19
RMASM	0.132	0.290	0.771	-1.574	-1.088	-0.296	19
RMASP	0.111	0.143	0.450	-1.670	-1.530	-0.772	19
LMASA	0.196	0.263	0.149	-1.341	-1.154	-1.505	19
LMASM	0.248	0.816	0.778	-1.193	-0.236	0.285	19
LMASP	0.110	0.579	0.349	-1.677	-0.564	-0.959	19
RTA	0.175	0.115	0.123	-1.408	-1.652	-1.615	19
LTA	0.848	0.288	0.548	0.194	-1.093	-0.612	19

Table 17: T-test; Comparison of the RMS % MVC of the eight studied muscle regions at sub-maximal bite force level 3 for the intervention vs. control group. p= statistical significance (p-value), t= t-variable, df= degrees of freedom. T1-3 indicate the timepoints. Statistical significance was set at p≤0.05. RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	***p* (T1)**	***p* (T2)**	***p* (T3)**	***t* (T1)**	***t* (T2)**	***t* (T3)**	***df* (T1-3)**
RMASA	0.122	0.243	0.189	-1.617	-1.206	-1.362	19
RMASM	0.118	0.307	0.896	-1.638	-1.050	-0.133	19
RMASP	0.123	0.091	0.602	-1.615	-1.783	-0.531	19
LMASA	0.142	0.155	0.310	-1.532	-1.482	-1.043	19
LMASM	0.124	0.676	0.463	-1.608	-0.424	-0.749	19
LMASP	0.104	0.477	0.592	-1.707	-0.726	-0.545	19
RTA	0.141	0.258	0.490	-1.535	-1.166	-0.705	19
LTA	0.915	0.579	0.711	-0.108	-0.565	-0.376	19

The negative t-values outline that the control group appears to have constantly lower mean RMS % MVC at SBF 1 compared with the intervention group for the first sub-maximal bite force task. The differences, however, were only statistically significant for the right temporalis anterior in T2 (*p*-value= 0.033), without adjustment for multiple testing.

The statistical analysis of the RMS % MVC EMG values at SBF2 and SBF3 has led to similar findings, indicating no significant differences ($p > 0.05$) between the intervention and control group but with constantly negative t-values.

3.4.3. Comparison of RMS at MVC (male vs. female group)

An independent samples t-test compared the RMS at maximal bite force (MVC) between male and female participants. The results are demonstrated in *Table 18.*

Table 18: T-test; Comparison of the RMS at MVC of the eight studied muscle regions for the two gender subgroups (male vs. female). p= statistical significance (p-value), t= t-variable, df= degrees of freedom. T1-3 indicate the timepoints. Statistical significance was set at p≤0.05 and is denoted with asterisk (*). RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	***p* (T1)**	***p* (T2)**	***p* (T3)**	***t* (T1)**	***t* (T2)**	***t* (T3)**	***df* (T1)**	***df* (T2)**	***df* (T3)**
RMASA	**0.050***	0.057	**0.009***	-2.028	-1.965	-2.747	38	36	38
RMASM	0.992	0.255	0.496	-0.010	-1.158	-0.688	38	36	38
RMASP	0.640	0.186	0.859	-0.471	-1.349	-0.179	38	36	38
LMASA	0.923	0.222	0.395	-0.098	-1.243	-0.860	38	36	38
LMASM	0.518	0.523	0.291	-0.653	-0.645	-1.070	38	36	38
LMASP	0.995	0.397	0.694	-0.006	-0.857	-0.397	38	36	38
RTA	0.567	0.696	0.719	0.578	-0.394	0.362	38	36	38
LTA	0.071	0.625	0.329	1.855	0.493	0.988	38	36	38

Overall, male participants exert significantly higher RMS values at MVC than female participants for the right masseter anterior at T1 (*p*-value: 0.050) and T3 (*p*-value: 0.009). The right masseter anterior at T2 shows the same tendency but was not statistically significant. The other muscle areas were not statistically significant either.

3.5. Tests of within-subject effects

3.5.1. Comparison of RMS % MVC between T1, T2 and T3

The following rmANOVA compares the RMS % MVC of each muscle area at SBF1-3 (dependent variable) with time (T1-3) (independent variable) within intervention and control group. The results indicate whether the active GrindCare® has influenced the RMS % MVC of the intervention group and whether the effects are reversed after this intervention stopped. Since there was no statistically significant difference in the rmANOVA for the intervention or control group, the results are joined together in the tables below. Prior to using rmANOVA for statistical analysis, following assumptions have been tested and verified to allow the application. The first requirement is the normal distribution of the data which was already evaluated above. The second assumption is sphericity, to which a repeated measures ANOVA (within-subject factors) is susceptible most when violated. This requirement can be tested by the Mauchly test, using the software SPSS. The results of the Mauchly test are presented in *Table 19*.

Table 19: rmANOVA; Mauchly's test of sphericity; Analyzing the sphericity of RMS % MVC of the eight studied muscle regions at submaximal bite force level 1-3 (SBF1-3). p= statistical significance (p-value). Statistical significance was set at p≤0.05 and is denoted with asterisk (*). RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle area	**RMS % MVC at SBF1:** ***p-values***	**RMS % MVC at SBF2:** ***p-values***	**RMS % MVC at SBF3:** ***p-values***
RMASA	0.275	0.826	0.122
RMASM	0.876	0.805	0.783
RMASP	0.770	0.799	**0.022***
LMASA	0.566	**0.007***	**0.002***
LMASM	0.431	0.592	0.209
LMASP	0.865	0.172	0.134
RTA	0.695	0.687	0.201
LTA	0.546	0.642	**0.036***

In our case, the Mauchly's test indicated that the assumption of sphericity has not been violated for the big majority of the data, as there was no statistical significance ($p > 0.05$). Therefore, a change of the degrees of freedom was not required. Three datasets of the muscle parts RMASP and LMASA, however, have violated the sphericity ($p \leq 0.05$). They were addressed using the Greenhouse-Geisser test. However, the results did not change, which is why the correction is not further elaborated. The following tables *(Table 20, Table 21, Table 22)* present the key findings of the repeated measures ANOVA, displaying the results of the RMS % MVC at SBF 1, 2, and 3 with time (T1-T3).

Table 20: rmANOVA; Comparison of RMS % MVC of the eight studied muscle regions at submaximal bite force 1 between T1, T2, and T3. Type III Sum of Squares, df= degrees of freedom, Mean Square, F-value, p-value= statistical significance, observed power. Statistical significance was set at p≤0.05 and is denoted with asterisk (*). RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle region	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*p*	*Observed Power*
RMASA	0.041	2	0.021	1.955	0.149	0.393
RMASM	0.033	2	0.016	1.716	0.187	0.350
RMASP	0.028	2	0.014	1.363	0.262	0.285
LMASA	0.021	2	0.010	0.673	0.513	0.159
LMASM	0.072	2	0.036	4.598	**0.013***	0.763
LMASP	0.116	2	0.058	5.681	**0.005***	0.850
RTA	0.057	2	0.028	4.661	**0.012***	0.769
LTA	0.097	2	0.049	5.633	**0.005***	0.847

Table 21: rmANOVA; Comparison of RMS % MVC of the eight studied muscle regions at sub-maximal bite force 2 between T1, T2, and T3. Type III Sum of Squares, df= degrees of freedom, Mean Square, F-value, p-value= statistical significance, observed power. Statistical significance was set at p≤0.05 and is denoted with asterisk (*). RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle region	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*p*	*Observed Power*
RMASA	0.031	2	0.015	1.957	0.148	0.394
RMASM	0.005	2	0.002	0.258	0.773	0.089
RMASP	0.008	2	0.004	0.412	0.664	0.114
LMASA	0.075	2	0.038	1.484	0.233	0.308
LMASM	0.017	2	0.008	0.939	0.395	0.207
LMASP	0.024	2	0.012	1.365	0.261	0.286
RTA	0.003	2	0.002	0.229	0.796	0.085
LTA	0.060	2	0.030	3.520	**0.034***	0.640

Table 22: rmANOVA; Comparison of RMS % MVC of the eight studied muscle regions at sub-maximal bite force 3 between T1, T2 and T3. Type III Sum of Squares, df= degrees of freedom, Mean Square, F-value, p-value= statistical significance, observed power. Statistical significance was set at p≤0.05. RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle region	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*p*	*Observed Power*
RMASA	0.004	2	0.002	0.196	0.822	0.079
RMASM	0.000	2	0.000	0.014	0.986	0.052
RMASP	0.012	2	0.006	0.518	0.598	0.132
LMASA	0.104	2	0.052	1.703	0.189	0.348
LMASM	0.010	2	0.005	0.504	0.606	0.130
LMASP	0.004	2	0.002	0.228	0.797	0.084
RTA	0.002	2	0.001	0.126	0.882	0.069
LTA	0.032	2	0.016	1.673	0.194	0.342

The one-way rmANOVA mostly found no statistically significant differences between the three measurement sessions (*p*-value>0.05). However, the RMS % MVC at first SBF of the left masseter posterior, the right temporalis anterior and the left temporalis, and the RMS % MVC at second SBF of the left temporalis anterior were found statistically significant (LMASM11<LMASM31<LMASM21; LMASP11<LMASP31< LMASP21; RTA11<RTA31<RTA21; LTA11<LTA31<LTA21; LTA12<LTA32<LTA22). The observed power lies below the desired power of 0.800 for statistical tests. A lower desired power indicates that it is less likely to detect a difference when one veritably exists.

3.5.2. Comparison of the RMS at MVC between T1, T2 and T3

The RMS at MVC were very constant between the three measurements (T1, T2 and T3) for the whole study sample. Therefore, a statistical test was performed comparing the RMS at MVC in T1, with T2, and T3 for all probands. Normality was verified with the Shapiro-Wilk's test and Q-Q plots. For the muscle areas RMASA, LMASA, LMASM, LMASP, RTA, and LTA normality could not be confirmed due to a lower *p*-value than the predefined cutoff value of 0.05. Therefore, the nonparametric equivalent to the rmANOVA (Friedman test) was used for these muscle parts, as outlined in *Table 23*.

Table 23: Friedman's rmANOVA; Comparison of RMS at MVC for six of the studied muscle regions in T1, T2 and T3. df= degrees of freedom, p= statistical significance (p-value). Statistical significance was set at p≤0.05 and is denoted with asterisk (*). RMASA=right masseter anterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	**RMS at MVC**	**Percentile**			***chi-square***	***df***	***p***
		25%	***50% (median)***	***75%***			
RMASA	T1	0.220	0.305	0.450	0.658	2	0.720
	T2	0.200	0.290	0.465			
	T3	0.203	0.280	0.433			
LMASA	T1	0.1000	0.160	0.302	0.667	2	0.717
	T2	0.0800	0.145	0.262			
	T3	0.0800	0.170	0.297			
LMASM	T1	0.280	0.350	0.458	3.361	2	0.186
	T2	0.248	0.330	0.435			
	T3	0.263	0.335	0.458			
LMASP	T1	0.150	0.190	0.220	10.255	2	**0.006***
	T2	0.120	0.150	0.200			
	T3	0.130	0.185	0.237			
RTA	T1	0.220	0.275	0.400	0.843	2	0.342
	T2	0.238	0.280	0.342			
	T3	0.220	0.270	0.355			
LTA	T1	0.230	0.285	0.370	0.764	2	0.538
	T2	0.218	0.275	0.400			
	T3	0.183	0.275	0.358			

The muscle parts RMASA, LMASA, LMASM, RTA, and LTA did not show a statistically significant difference ($p > 0.05$) for the measurement interval T1-T3. However, for the left posterior masseter the median values in T1, T2 and T3 were significantly ($p=0.006$) lower than the value 10.255 in chi-square statistics. In order to identify which measurement time point differs, a post-hoc test was performed, i.e., a multiple comparison procedure versus the control group T1 via the Dunnett's method. The results are summarized in *Table 24*.

Table 24: Dunnett's method; Comparison of RMS at MVC for LMASP (left masseter posterior) between T1, T2 and T3. Diff of Ranks, q' ratio, p= statistical significance (p-value). Statistical significance was set at p≤0.05.

Comparison LMASP	*Diff of Ranks*	*q' ratio*	*p*
T2 vs T1	22.500	3.650	Yes
T3 vs T1	1.500	0.243	No

The post-hoc test shows that the significant difference appears between T1 and T2, i.e., week 1 and 3, after the GrindCare® intervention (LMASP2 > LMASP1). Between the intervals T2 and T3, the difference is no more significant, signifying that the RMS at MVC returned to the starting condition after the effect was withdrawn.

The two muscle parts RMASM, RMASP met the assumptions of a normal distribution and equal variances (Shapiro-Wilks, $p > 0.05$). Therefore, a parametric one-way rmANOVA was performed for the right middle masseter and right posterior masseter, presented in *Table 25*, *Table 26*.

Table 25: rmANOVA; Comparison of RMS at MVC for RMASM (right masseter medial), RMASP (right masseter posterior) between T1, T2 and T3.
Interval, mean, standard deviation (SD), standard error of mean (SEM).

Muscle areas	*Interval*	*Mean*	*SD*	*SEM*
RMASM	T1	0.362	0.128	0.0203
	T2	0.364	0.151	0.0245
	T3	0.359	0.147	0.0233
RMASP	T1	0.185	0.0679	0.0107
	T2	0.182	0.0628	0.0102
	T3	0.180	0.0747	0.0118

Table 26: rmANOVA; Comparison of RMS at MVC for RMASM (right masseter medial), RMASP (right masseter posterior) between T1, T2 and T3.
Type III Sum of Squares, df= degrees of freedom, Mean Square, F-value, p= statistical significance (p-value), observed power. Statistical significance was set at p≤0.05.

Muscle areas	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*p*	*Observed Power*
RMASM	0.000274	2	0.000137	0.0290	0.971	0.050
RMASP	0.000409	2	0.000204	0.132	0.876	0.050

The mean values do not show a significant change over the measurement interval T1, T2, and T3 among the treatment groups, for both muscle parts. Furthermore, the observed power was 0.05 and therefore far below the desired value of 0.80. Hence, the performed experiment with the existing study sample size is not likely to detect a difference.

3.5.3. Comparison of the RMS % MVC between T1, T2 and T3

Since the study protocol was not performed at the planned submaximal force levels and the real submaximal bite force proved to be around 50N, measured in triplicate, another within-group test was performed. A parametric and nonparametric rmANOVA was conducted to compare the RMS % MVC during T1, with T2, and T3 with one another, to test for potential training effects for each muscle region. The Friedman test was chosen when the normality test failed (Shapiro-Wilk; p≤0.05) and the parametric ANOVA when the normality test passed (p > 0.05). Normality was verified with the Shapiro-Wilk's test and Q-Q plots. For the muscle areas RMASA, LMASA, LMASM, LMASP, RTA, and LTA normality could not be confirmed due to a lower *p*-value than the predefined cutoff value of 0.05. The following *Table 27* shows the results of the performed Friedman test for these muscle regions.

Table 27: Friedman's rmANOVA; Comparison of RMS % MVC for seven of the studied muscle regions between T1, T2, and T3. df= degrees of freedom, p= statistical significance (p-value). Statistical significance was set at p≤0.05 and is denoted with asterisk (*).
RMASA=right masseter anterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

		Percentile					
Muscle areas	***Interval***	***25%***	***50% (median)***	***75%***	***chi-square***	***df***	***p***
RMASA	T1.1	0.0743	0.106	0.140	9.874	8	0.274
	T1.2	0.0760	0.103	0.131			
	T1.3	0.0700	0.101	0.145			
	T2.1	0.0762	0.124	0.165			
	T2.2	0.0698	0.115	0.152			
	T2.3	0.0655	0.111	0.152			
	T3.1	0.0720	0.0943	0.151			
	T3.2	0.0685	0.0884	0.160			
	T3.3	0.0658	0.0950	0.161			
RMASM	T1.1	0.0813	0.118	0.164	16.982	8	**0.030***
	T1.2	0.0783	0.122	0.158			
	T1.3	0.0831	0.112	0.180			
	T2.1	0.0913	0.131	0.200			
	T2.2	0.0801	0.113	0.180			
	T2.3	0.0756	0.117	0.187			

	T3.1	0.0880	0.112	0.183			
	T3.2	0.0786	0.103	0.168			
	T3.3	0.0761	0.107	0.177			
LMASA	T1.1	0.0299	0.0632	0.104	5.747	8	0.676
	T1.2	0.0333	0.0643	0.119			
	T1.3	0.0360	0.0646	0.114			
	T2.1	0.0270	0.0563	0.104			
	T2.2	0.0284	0.0620	0.0922			
	T2.3	0.0277	0.0570	0.104			
	T3.1	0.0328	0.0576	0.0870			
	T3.2	0.0323	0.0573	0.0842			
	T3.3	0.0330	0.0579	0.0826			
LMASM	T1.1	0.0903	0.125	0.163	12.919	8	0.115
	T1.2	0.0836	0.128	0.174			
	T1.3	0.0791	0.136	0.166			
	T2.1	0.0898	0.137	0.193			
	T2.2	0.0830	0.124	0.179			
	T2.3	0.0858	0.118	0.175			
	T3.1	0.0865	0.126	0.185			
	T3.2	0.0806	0.122	0.185			
	T3.3	0.0820	0.113	0.184			
LMASP	T1.1	0.0481	0.0615	0.0877	13.656	8	0.091
	T1.2	0.0447	0.0601	0.0920			
	T1.3	0.0428	0.0627	0.0922			
	T2.1	0.0477	0.0697	0.0904			
	T2.2	0.0432	0.0597	0.0804			
	T2.3	0.0450	0.0553	0.0785			
	T3.1	0.0465	0.0669	0.0854			
	T3.2	0.0397	0.0652	0.0828			
	T3.3	0.0674	0.0674	0.0855			
LTA	T1.1	0.0596	0.0983	0.155	9.544	8	0.299
	T1.2	0.0749	0.0989	0.160			
	T1.3	0.0683	0.0929	0.165			
	T2.1	0.0749	0.116	0.173			
	T2.2	0.0674	0.114	0.168			
	T2.3	0.0682	0.117	0.152			
	T3.1	0.0631	0.104	0.153			

	T3.2	0.0667	0.0920	0.148			
	T3.3	0.0697	0.0919	0.150			
RTA	T1.1	0.0754	0.108	0.157	18.147	8	**0.020***
	T1.2	0.0720	0.115	0.158			
	T1.3	0.0700	0.110	0.152			
	T2.1	0.0915	0.126	0.159			
	T2.2	0.0765	0.113	0.151			
	T2.3	0.0788	0.110	0.153			
	T3.1	0.0851	0.111	0.144			
	T3.2	0.0745	0.109	0.147			
	T3.3	0.0765	0.101	0.147			

RMASA, LMASA, LMASM, LMASP, and LTA were analyzed by the Friedman's test and showed no significant difference between all RMS % MVC values in all measurement sessions. The right middle masseter and right anterior temporalis, however, showed significant differences ($p = 0.03$ and 0.02 respectively) between the median values and the chi-square (chi square (8) RMASM: 16.982, chi square (8) RTA: 18.147). Pairwise tests were performed with a Tukey test, which is a post-hoc test used to compare all possible group combinations. For the right middle masseter, three pairwise comparisons showed statistically significant differences: T2.1 > T3.3; T2.1 > T1.1; T2.1 > T2.3, while for the right temporalis significant differences were found for the following combinations: T2.1 > T3.3, T2.1 > T1.3; T2.1 > T3.2, and T2.1 > T1.2.

Since the RMASP confirmed a normal distribution, a parametric ANOVA was conducted and the results are outlined in the following tables (*Table 28, Table 29).*

Table 28: rmANOVA; Comparison of RMS % MVC for RMASP (right masseter posterior) between T1, T2 and T3.
Interval, mean, SD= standard derivation, SEM= standard error mean.

Muscle areas	*Interval*	*Mean*	*SD*	*SEM*
RMASP	T1.1	0.0647	0,0314	0,00497
	T1.2	0.0644	0.0306	0.00484
	T1.3	0.0653	0.0336	0.00532
	T2.1	0.0705	0.0325	0.00528
	T2.2	0.0645	0.0295	0.00479
	T2.3	0.0615	0.0294	0.00477
	T3.1	0.0656	0.0287	0.00454
	T3.2	0.0598	0.0275	0.00435
	T3.3	0.0622	0.0291	0.00460

Table 29. rmANOVA; Comparison of RMS % MVC for RMASP (right masseter posterior) between T1, T2 and T3.
Type III Sum of Squares, df= degrees of freedom, Mean Square, F- value, p= statistical significance (p-value), observed power. Statistical significance was set at p≤0.05.

Muscle	*Type III Sum of Squares*	*df*	*Mean Square*	*F*	*p*	*Observed Power*
RMASP	0.00329	8	0.000411	1.754	0.086	0.345

The ANOVA showed no significant differences (p = 0.086) and the observed power (0.345) was below the desired 0.80.

4. Discussion

4.1. Summary of the main results

Summing up the main results it can be stated that there were no substantiating statistically relevant differences in the analyzed data. Examining the subgroups in T1 has confirmed that there was no heterogeneity in the two main groups, as the subgroups gender and bruxism activity (OBC score and GC activity) did not render statistically significant results in terms of RMS % MVC. The RMS % MVC at submaximal bite forces were mostly not significant between the groups, except for the right anterior temporalis in T2. The comparison of RMS at maximum voluntary contraction between men and women showed higher values for the right masseter anterior in the male subgroup. The analysis within the groups primarily revealed no differences for the intervention and control group and were therefore analyzed together. Within the whole study population, the RMS % MVC at SBF1 of the left masseter medial, left masseter posterior, right temporalis anterior, left temporalis anterior, and the RMS % MVC at SBF2 of the left temporalis anterior significantly increased between T1 and T2 and decreased in T3. Furthermore, the RMS at MVC within the whole study population was statistically significant for the left posterior masseter. Lastly, the comparison of RMS % MVC at 50 N between T1, T2 and T3 revealed significant differences for the right middle masseter and right anterior temporalis. Most importantly, the overall observed power was consistently below the desired value of 0.80, which makes our results prone to Type II error.

This study was designed for basic research on bruxism behavior, aiming to advance our knowledge of the EMG patterns of bruxers and thus enable facilitation of the diagnosis. Our hypothesis that the RMS % MVC of subjects with high and low bruxism activity differ in baseline and after use of CES could not be verified. Furthermore, CES intervention did not affect these EMG parameters.

As this is yet a novel field of dental medicine, there is only limited scientific data available. Therefore, it is important to evaluate the used materials, methods, and results in order to guide future research.

4.2. Discussion of materials and methods

4.2.1. Study sample

Our study sample was recruited randomly via social media platforms, friends, and recommendations of people who participated. Participants without painful symptoms or in need of dental care were selected for this study. Among the included participants (*N*=40), there was a sleep bruxism proportion of 67.5 % (GrindCare® measurement), while the amount of possible bruxers were 60% (general questionnaire). As already mentioned above, the GC data are more reliable as they do not underly the influenceability and subjectivity of the probands. However, both measurement techniques rendered a high amount of bruxers compared to epidemiological data. Current literature states that the prevalence of possible sleep bruxism is around 12.8 ± 3.1 % (Lavigne et al., 2008; Lobbezoo et al., 2012). By using social media for the recruitment of subjects, primarily young people (mean age 25 for women and 28 for men) volunteered for the study, which may be the reason for the high percentage of bruxers, as studies have shown that the prevalence peak for adult bruxism lies between the second until the third decade of life (Peroz et al., 2019). However, this aspect is negligible since this study was planned as a cohort study. Furthermore, the experimental trial entailed more female than male participants since there were various drop-outs due to the requirement of shaving off the beard for application of the EMG-electrodes. However, this unequal distribution is insignificant as each participant was matched with the same-gender and -age counterpart.

4.2.2. Subjective bruxism assessment – questionnaires

As already introduced above, the OBC was developed as a tool for assessing bruxism behavior as a risk factor for TMD by the DC/TMD, therefore there is only limited availability on studies including pain-free subjects. It was proven that the OBC is high in its reliability (Donnarumma et al., 2018), well understood by subjects, and in concordance with EMG patterns (Ohrbach et al., 2008). Donnarumma et al. have shown that in the OBC functional activities are higher for non-TMD patients than for TMD patients (Donnarumma et al., 2021). This finding generally confirms the good properties of the OBC for healthy individuals.

However, a possible limitation of the OBC questionnaire is the retrograde report of oral behaviors over a prior month (Markiewicz et al., 2006). Many studies have already

applied the OBC translated into different languages to their study protocol before. Besides the various studies by Ohrbach et al., who have compared the validity of the different behavioral patterns to the EMG (Markiewicz et al., 2006; Ohrbach et al., 2008), there is the study by Bucci. et al. who investigated the correlation of AB and occlusal sensitivity (Bucci et al., 2019). The relatively recent study by Yurttutan et al. has analyzed the efficacy of occlusal splints and Botulinum Toxin for the treatment of bruxism also by applying the OBC (Yurttutan et al., 2019). Furthermore, Donnarumma, Ohrbach and Lobbezoo et al. have performed a study about TMD patients using the combination of the DC/TMD Axis I and II questionnaires for TMD diagnostics and the OBC (Donnarumma et al., 2021). However, not all studies could confirm the good reliability as the examination between self-report and a portable EMG device showed a lack of association between these two parameters (Prasad et al., 2021).

Summarizing the subjective assessment method via questionnaires it is important to outline that different questionnaires come to different results regarding bruxism, due to subjectively perceived factors. A major factor which influences the results and scores is the fluctuation of bruxism by nature (Giannakopoulos et al., 2013). Another aspect is that the information obtained is retrospective at a single observation point, making it difficult to remember the exact occurrence of the oral behavior (Manfredini et al., 2013). Further influences are the current psychological condition (Pintado et al., 1997), the dentist calling the attention of the patient on attrition facets, and the lack of sounds during clenching, which can falsify the results of the questionnaire (Giannakopoulos et al., 2013; Koyano et al., 2008).

4.2.3. Intraoral force measurement

The BiteFork® device used in our study is a digital assessment tool for the bilateral bite force exerted by each patient. The force measurement is necessary to warranty the same test conditions for all subjects. Despite the calibration of the BiteFork® according to the instructions of the manufacturer, it somehow did not correctly measure the desired bite forces. The device probably recorded the lowest detectable force within the recording range which had a detrimental impact on the outcome.

The EMG data analysis showed that the EMG signal did not follow the intended steps corresponding to physiological chewing forces of 50N, 100N, and 150N. After validating the BiteFork® device independently, it was apparent that the actual bite force amounted

around 50N (25 per side). In 1999 Eckhard Stengel evaluated the maximal vertical chewing force as a function of the food and the number of chewing strokes of five healthy male and five healthy female subjects. He found that the greatest exerted force in the course of mastication was measured during the initial chewing stroke, regardless of whether these forces occurred vertically or horizontally. His findings were consistent with the current literature, reporting that chewing forces are food-specific and reflect different textural properties. According to his findings, the force of 50 Newton (25 N on each side) represents the chewing of a three-minute cooked carrot and kohlrabi (Stengel, 1999). This puts our not intended BiteFork® measurement in the context of mastication of very soft food. Since the manufacturer had already completed the validation process and confirmed the reliability of the device, a revalidation beforehand seemed unnecessary at the time.

Furthermore, the general design of the BiteFork® is based on Force Sensing Resistors (FSR) which have proven their reliability in past studies. Fernandes et al. has compared the strain-gauge bite fork with a conductive polymer pressure-sensing resistor, similar to the one applied in this study. The values obtained have shown no statistically significant differences in bite force levels between 50 to 300 N and the reproducibility was around 93%. Both devices, the strain-gauge bite fork and the conductive polymer pressure-sensing resistor, have proven to assess the bite force sufficiently in terms of clinical accuracy and precision. Although it has been reported that potential disadvantages of the FSR are the nonlinear and load-rate dependent properties of the sensor, caused by the nonlinearity of the force-sensing resistor and damage of the surface material of the sensor (Fernandes et al., 2003).

4.2.4. Electromyographic measurement

4.2.4.1. Sleep bruxism EMG assessment

The acquisition of sleep bruxism events was ensued by the GrindCare® device. However, the gold standard for monitoring sleep bruxism behavior remains the PSG recording combined with the EMG of masticatory muscles, EEG, ECG, EOG, audio and video recordings to differentiate between bruxism and other oral behaviors during sleep.

The implementation of our study protocol in a sleep laboratory was not possible due to the large amount of participants in addition to the observation period of five weeks, apart from the high cost and the burden for the patients.

Furthermore, it might have influenced the sleep habits as a consequence of the foreign sleep environment. Based on these aspects, it was more convenient for subjects to use home devices due to their proven ability to overcome these obstacles while remaining reliable. So far, only BiteStrip®, BruxOff®, and GrindCare® affirm high validity compared to PSG recordings (Manfredini et al., 2014). BiteStrip® can only monitor the masseteric activity for 5 hours in a single measurement, which makes it rather less suitable (Giannakopoulos et al., 2013). GrindCare® is superior in that respect as it can measure several consecutive nights while also offering a stimulation mode. Studies have shown that the use of GrindCare® has resulted in a 90% specificity in comparison to PSG for a reliable definitive sleep bruxism diagnosis (Stuginski-Barbosa et al., 2016). The 90% specificity is reached when the cutoffs for sleep bruxism are set for 18/19EMG/h for three/five nights of GrindCare® measurement (Stuginski-Barbosa et al., 2016). Nevertheless, a recent overview by Thymi et al. has shown the large variety of thresholds and grading criteria for defining sleep bruxers (Thymi et al., 2021), which highlights the difficulty in finding universal guidelines to measure RMMA and to ensure the comparability of studies.

Current question of research is the clinical relevance of RMMA in regard to bruxism (Manfredini et al., 2019; Manfredini et al., 2020), as negative clinical health outcomes can be associated with RMMA but are not limited to them (Thymi et al., 2021). Background EMG activity, intensity and timing, amplitude of activity, and variability of activity over time are also thought to be relevant parameters, although the extent is unknown at present (Baad-Hansen et al., 2019). Instead of diagnosing RMMA solely, the objective of future bruxism research should be more focused on correlating muscle activity in EMG to specific bruxism-associated symptoms and to define corresponding thresholds (Manfredini et al., 2019; Thymi et al., 2021). The EMG devices selected should be classified according to the consistent pattern, i.e., what type of MMA outcomes they are able to assess (Thymi et al., 2021).

Regarding the intervention (CES), other techniques have proven to be effective in reducing sleep bruxism events. For sleep bruxism, auditory, electrical, vibratory, and even taste stimuli can be used for biofeedback (Lobbezoo et al., 2008; Murali et al., 2015; Shetty et al., 2010). However, all effects are transient and some result in frequent arousals which may lead to severe side effects (i.e., excessive daytime sleepiness) (Lobbezoo et al., 2008; Shetty et al., 2010).

In contrast to that intervention technique, GrindCare® intervenes whenever a bruxism pattern is measured, reducing RMMAs and allowing symptomatic improvement. It does not aim for a conscious activity change but reduces RMMA through reflex activation (Desmedt et al., 1976; Godaux et al., 1975; Jadidi et al., 2008; Peroz et al., 2019). GrindCare® is therefore superior to the other stimuli as it has proven to be more reliable than the biofeedback approach (Manfredini et al., 2015b) while least affecting the sleeping habits and allowing an observation of the change of behavior close in time to the actual experience. Although other studies have indeed found a reduction of SB-related motor activities (Jadidi et al., 2008; Jokubauskas et al., 2018; Manfredini et al., 2015b; Needham et al., 2013; Sato et al., 2015; Shimada et al., 2019) our study could not verify these findings. A possible reason is our larger sample size. While Needham's sample size was *N*=19, Sato's was *N*=13, and Jadidi's was *N*=14, our study entailed *N*=40 participants. Besides, a further impact on the effectiveness of reducing SB episodes is the intervention interval.

Our study protocol has scheduled an active GrindCare® period of two weeks. After starting our trial there has been a recommendation as part of a consensus paper by Lobbezzo recommending four weeks of active stimulation (Lobbezoo et al., 2019). A dissertation by Niklas Becker has found no significant reduction of EMG episodes/hour after two weeks of intervention. In consultation with the manufacturer, they consequently changed their study protocol from two weeks of intervention to four which significantly reduced the EMG episodes/hour after the fourth week (Becker, 2021). A possible learning effect remains unclear, as decreased EMG parameters were found to be transient after the intervention had finished (Jokubauskas et al., 2018; Raphael et al., 2013), while others do claim that the EMG scores remain reduced (Jadidi et al., 2013).

4.2.4.2. EMG recordings in the laboratory setting

An EMG recording can be performed in different ways (intramuscular/surface electrodes; monopolar/bipolar electrodes) depending on the variables under investigation, as each method gives distinct information. Regarding the bipolar measurement of the surface EMG, the electrodes were placed in fiber direction and were favored over the monopolar measurement since it has a higher interference immunity (Kluth et al., 2013). In contrast to portable EMG devices, EMG recording in laboratory settings cannot detect bruxism behavior close in time to the experience. It is therefore not a real-time measurement which might influence the interpretation of the results. In order to obtain information about single muscle fibers with a high spatial resolution (e.g., investigating the origins of the action potential and recruitment patterns of motor units) it is recommended to use intramuscular needles and wires and a higher sampling rate. The conduction of intramuscular needles requires precise information about the location of the muscles, for example, by using a magnetic resonance imaging (MRI) and an expert to insert the needle. While surface EMG is more manageable and does not require much preparation, it instead delivers information about the superficially located muscles with a low spatial resolution. It is a result of the superimposed MUAP of many motor units.

Since the aim was to investigate the overall differences of EMG (RMS % MVC) of subjects with different degrees of bruxism, a surface EMG was used. According to the Henneman principle and confirmed by Farella et al., the deep masseter (pars profunda) is preferably recruited for low bite forces, e.g., 25N. The higher the exerted force, the more the recruitment pattern shifts towards activation of the superficial masseter (pars superficialis) (Farella et al., 2002). This plays a major role in the decision of whether to apply a surface or an intramuscular EMG. It seems apparent that higher forces activate the superficial masseter and require a surface EMG, as the active region is closer to the electrode and therefore provides a stronger signal (Henneman et al., 1965). Since the SBF was unknowingly low (25N per side) an intramuscular EMG could have provided more information about the probably predominantly activated deep masseter.

The maximum clenching bite force is an individual and major variable needed to normalize the acquired RMS data. It is an indicator of the harmonic synergy of the masticatory system and can be altered due to internal influences, e.g., pain, temporomandibular disorders, gender, age, craniofacial morphology, occlusal factors, and external influences, such as recording devices and techniques (Koç et al., 2010).

The criteria pain, TMD, dental treatment need, and incomplete dental arch were set as exclusion criteria to eliminate these influences. Furthermore, the subjects were matched according to gender and age in order to be able to compare each proband with his/her respective match, while ensuring the same initial conditions for both groups (intervention and control group). However, it is inevitable that factors such as craniofacial morphology, time of the experiments, and previous chewing activity may have affected the study's outcome.

Studies state many different values when it comes to the maximal clenching force. There are various techniques to obtain the maximal bite force, either by biting on dental cotton rolls or in intercuspation. In contrast to biting on dental cotton rolls, the maximal bite force in intercuspation has shown significant reductions in EMG activity, possibly due to avoidance patterns (activation of periodontal nociceptors). Considering this phenomenon, obtaining maximum voluntary contraction with cotton rolls is recommended as it enables a balanced, consistent, and repeatable contraction. At the same time, the MVC on occlusal surfaces might be displaced onwards (controlled predominantly by the temporalis muscle) or backwards (masseter controlled) (Ferrario et al., 2006; Hellmann et al., 2011). Current literature have equivocal results but more studies tend to find a higher exertion of force for males than females (Helkimo et al., 1977; Koç et al., 2010), which generally ranges between 300-600N (Bakke, 2006). One example is Helkimo et al.'s study, who found that the maximal bite force measured in the molar region for men was 382 N and the corresponding value for women was 216 N (Helkimo et al., 1977). Comparable to our study design is the one by Jirakittayakorn et al.'s who also included male and female participants to investigate MVC to consequently design a portable EMG device. The results confirm that men can provide higher MVC values than women (Jirakittayakorn et al., 2014). Nevertheless, many studies are reporting higher values, such as 496N (Stengel, 1999) and 550N (Gibbs et al., 1981).

The mean value of the maximal bite force of this study was 450N (the calculation is explained above in 3.1.3.1) which is within the range of values reported in the literature. In terms of the gender-related maximal bite force, there was mostly no statistically relevant difference between men and women which is in conformity with a study performed by Hellmann et al. (Hellmann et al., 2011). However, men exerted higher forces in the right masseter anterior. The research in available data did not render a possible explanation for this finding.

4.2.5. Study protocol

Our study protocol included male and female participants, all matched according to gender and age. This “balanced” study design allows the assumption that each participant has an equal counterpart in the opposing group, limiting the heterogeneity of participants and therefore results, plus ensuring adequate comparisons. Furthermore, it improves efficiency when controlling for the matching factors (e.g., gender) (Pearce, 2016). The improved precision enables a reduction of the source population which consequently facilitates the subject recruitment. One major aspect that affected the recruitment of subjects was the relatively long observation period. The five weeks interval influenced the willingness to participate most notably among the male participants. Although an equal distribution of male to female participants was aimed, most men were unwilling to shave their beards for five weeks, resulting in dropouts.

4.2.6. Methodological limitations

One of the limiting factors of this study was that by using social media to recruit subjects, primarily young people volunteered which might be the reason for the low age mean, the unwillingness to shave the beard, and the higher rate of women participating. However, the main limitation seems to be the use of the BiteFork® device. The display of erroneous bite forces led to a change in the interpretation of the study results. The ultimately measured bite force of 50 N (25N per side) lies below usual physiological chewing forces and poses possibly the main influencing factor for the outcomes presented in this study. Studies have shown that low bite forces rather activate the deep masseter (pars profunda) instead of the superficial masseter (pars superficialis) (Farella et al., 2002). Therefore, the validity of the surface EMG is limited, as it is less suitable than an intramuscular EMG in measuring the electromyographic activity of muscle parts further away from the superficial electrode.

Moreover, the use of GrindCare® intervention could not provide a considerable change in the MMA of the subjects which is inconsistent with current literature (Jadidi et al., 2008; Needham et al., 2013). Lastly, a further limitation for the outcomes is the GrindCare® intervention interval of two weeks instead of the later recommended four weeks. An extension to a 4-week intervention may have enabled a cessation of the bruxism behavior.

4.3. Discussion of outcomes

4.3.1. Outcomes

- The comparison between the subgroups gender (male and female), bruxism activity expressed as OBC score (high and low scores) and GrindCare® activity (non-bruxers and definite bruxers) did not render statistically significant results in terms of RMS % MVC.
- The between-groups (control vs. intervention group) comparison regarding the RMS % MVC at submaximal bite forces was mostly not statistically significant, except for the right anterior temporalis in T2 which was significantly (*p*-value: 0.033) higher in the intervention group.
- The comparison of the RMS at MVC between men and women mostly showed no statistically relevant differences. Nevertheless, for the right masseter anterior men had significantly higher RMS values at T1 (*p*-value: 0.050) and T3 (*p*-value: 0.009).
- The within-groups comparison regarding the RMS % MVC at the submaximal bite forces between T1, T2, and T3 mostly showed no significant changes. Only the RMS % MVC at SBF1 of the left masseter medial (*p*-value= 0.013), left masseter posterior (*p*-value= 0.005), right temporalis anterior (*p*-value= 0.012), and left temporalis anterior (*p*-value= 0.005) significantly increased between T1 and T2 and decreased in T3 (LMASM11 < LMASM31 < LMASM21; LMASP11 < LMASP31 < LMASP21; RTA11 < RTA31 < RTA21; LTA11 < LTA31 < LTA21). The RMS % MVC at SBF2 of the left temporalis anterior (*p*-value= 0.034) additionally increased between T1 and T2 and decreased in T3 (LTA12 < LTA32 < LTA22).
- The within-groups comparison regarding the RMS at MVC between T1, T2, and T3 of the whole study population overall showed no significant changes. The RMS at MVC of the left posterior masseter, however, increased between T1 and T2 (*p*-value= 0.006), i.e., week 1 and 3 post-baseline, after the GrindCare® intervention. During interval T2 and T3, the difference was no more significant.

- In the analysis of the triplicate repetitions of RMS % MVC at 50 N between T1, T2, and T3 the right middle masseter and right anterior temporalis showed statistically significant differences (*p*-value = 0.03 and 0.02 respectively), increasing between T1 and T2 and decreasing between T2 and T3. RMASM T2.1 > T3.3, T2.1 > T1.1, T2.1 > T2.3; RTA T2.1 > T3.3, T2.1 > T1.3, T2.1 > T3.2; T2.1 > T1.2.
- The overall observed power was consistently below the desired value of 0.80, which makes our results prone to Type II error.

4.3.2. Outcome statement

The abovementioned results suggest that participants with increased bruxism activity according to the GC measurement render the same RMS % MVC as those with no-/low bruxism activity and that no considerable change can be achieved by intervening with a CES device for the intervention time of two weeks. Our hypothesis that patients with high definite bruxism differ from those with low definite bruxism regarding EMG parameters at baseline and after using CES could not be verified. Consequently, no return to baseline was observed. Furthermore, it is apparent that in this study, men and women mainly generate similar maximal clenching forces (similar EMG activity during MVC). Isolating the entire statistically significant findings, they do not seem to offer clinically relevant conclusions. In summary, the results of this experiment showed no differences in EMG parameters relevant for a valid bruxism diagnosis which is inconsistent with current literature (Hellmann et al., 2011; Palinkas et al., 2016). This finding is possibly due to the low exerted bite force, as further discussed below.

In order to interpret the study findings, it is inevitable to analyze the observed power which represents the probability of finding a difference based on the obtained sample size, if there is indeed any actual difference to be found. For our study, the observed power was lower than the desired cutoff value of 0.8. A larger sample size would therefore provide more certainty about the statistical significance of differences between the RMS % MVC values of participants with high and low bruxism activity. However, the evaluation of the number of participants needed to reach a valid conclusion requires a power analysis which was not part of this pilot study. Possible limitations and underlying reasons for these observations are discussed below.

4.3.3. Motor adaptation of the masticatory muscles after training

Training effects or motor adaptation are the change of motor actions due to regular exercise (Shemmell et al., 2005), by modification of the muscle activation pattern due to cortical reorganization (Lotze et al., 2003; Muellbacher et al., 2001). These training-induced adaptation processes do not change the motor action per se but modify variables such as the recruitment pattern and optimize the performance of a muscle. Once the motor action has been adapted to the new neuromuscular performance, it cannot retrieve the initial state unless the initial behavior is readapted with skill acquisition (Shemmell et al., 2005). The main objectives of this study were the evaluation and comparison of motor tasks on masseter and temporal muscles to find EMG parameters which can be related to bruxism and its' probable training effect. However, no effects of training were observed. Although there are available studies which investigate the motor adaptation of masticatory muscles in contrast to other skeletal muscles after simulated training, none has measured the changes of masticatory muscle activity regarding bruxism per se as a training. The aforementioned studies are presented below, assuming that there might be similarities between bruxism and training for masticatory muscles.

4.3.3.1. Plasticity of the masticatory system

One of the most established studies in the field of motor adaptation of the masticatory system is the investigation of the plasticity of the tongue after tongue-task training and an investigation of the masticatory muscles after isometric contraction. They found that with increased training, there have indeed been changes regarding the plasticity of the tongue and alterations in EMG activity (Peck et al., 2010; Svensson et al., 2006). They also concluded that exercise modifies motor activation strategies to optimize the performance of the same motor task. Peck et al., however, found that after four weeks of training there were no further changes in the EMG activity compared to the inactive control group and concluded that a therapeutic effect, which changed the motor control pattern, had occurred (Peck et al., 2010).

4.3.3.2. Motor adaptation of masticatory muscles as a function of time

In 2015, Kumar et al. have investigated the effects of short-term training by splitting a chocolate intraorally into two equal halves and recording the accuracy of the split and vertical jaw movements during the task. The results indicate that training a motor action induces skill acquisition and improvement of jaw movements (Kumar et al., 2015) even after short-term training. Furthermore, Hellmann et al. have shown that masticatory muscles adapt fast when coordination tasks are performed and therefore concluded that force-controlled balancing tasks at 100N, 200N, and 300 N can train masticatory muscles thoroughly, even after short-term intervals. Interestingly, the lower bite force of 50 N showed no statistically significant effects. In that study, training effects were visualized by a reduction of EMG activity while the reduction amounted to 29 % after merely two weeks and 40% ten weeks post-baseline (Hellmann et al., 2011).

Using our current study protocol, subjects with high and low bruxism activity were not significantly different in RMS % MVC, although a connection between a training effect due to repeated motor actions (e.g., bruxism) seems apparent. As might be reasonably expected, the constant exertion of motor actions leads to a change of recruitment patterns of the masticatory muscles of bruxers. Therefore, an optimization of performing tasks and hence a lower EMG activity should be anticipated (Palinkas et al., 2016) which is not in concordance with our study findings. This circumstance could be due to two aspects according to available literature, either the short intervention of 2 weeks instead of the later published recommendation of 4 weeks (Becker, 2021), or the rather low bite force of 50 N (25 N on each side). The latter aspect probably affected the study outcome more than the selected interval of 2 weeks, as the decisive factor in regard to the short interval is the excellent plasticity of the masticatory muscles even after a short-term intervention at 100, 200, and 300 N, as shown by Hellmann et al. (Hellmann et al., 2011). However, the low bite force of 50 N (25N on each side) represents the chewing of a three-minute cooked carrot and kohlrabi (Stengel, 1999) which is hardly representative for physiological bite force.

Furthermore, Hellman et al. have found significant changes for maximal bite force in intercuspation but not on dental cotton (Hellmann et al., 2011). One possible explanation for this phenomenon is that initial redundant motor units gradually become inactive and allow other motor units, which are biomechanically better suitable for performing the task, to take over, i.e., resulting in a change of the recruitment pattern. The plastic properties of the masticatory muscles enable this long-lasting neuromuscular adaptation.

The maximal bite force in intercuspation, in contrast to biting on dental cotton rolls, has shown significant reductions in EMG activity possibly due to avoidance patterns (activating periodontal nociceptors) (Ferrario et al., 2006; Hellmann et al., 2011). In our study, dental cotton rolls were chosen as they provide a more consistent variable than intercuspal maximal bite force (Hellmann et al., 2011; Hugger et al., 2008). Even though the selected technique might be the “state of the art” for measuring the maximal bite force, this study could not find any statistically significant differences regarding the RMS at MVC between bruxer and non-bruxer which is consistent with some studies (Hellmann et al., 2011; Palinkas et al., 2016), but inconsistent with other literature (Koç et al., 2010). Furthermore, this study could not verify the finding of Koç et al. that men exert higher maximal bite forces than women (Koç et al., 2010).

4.3.3.3. Effect of pain on motor adaptation

A ubiquitous and persistently studied aspect of oral health is the occurrence of pain and its influence on masticatory muscle performance. In 2010 Boudreau et al. have investigated the influence of pain on human motor learning and have found that pain does have a negative impact on motor learning but cannot prevent it per se (Boudreau et al., 2010). Based on this knowledge it becomes more reasonable why subjects with painful symptoms were excluded from this bruxism study. The aim was to not negatively influence the skill acquisition by use of the GrindCare® intervention due to pain (Boudreau et al., 2010). During our EMG measurement session, it was of great importance to allow the subject to rest in between tasks, to prevent pain and fatigue and, thus, negatively influence the outcomes.

4.3.4. Heterogeneity of the masticatory muscle physiology

4.3.4.1. Heterogeneity of masticatory muscle fiber types

One of the studies most relevant in comparison to ours is the study by Farella et al., who have investigated the functional diversity of the different fiber types of masticatory muscles by using electromyographic power spectra in relation to the bite force (for 25 N, 50N, 100 N, and 200 N). At a bite force level of 25N, the mean power frequency (MPF) values of the posterior temporalis were significantly lower than those of the masseter and anterior temporalis.

With increased bite force, the MPF values of the masseter decreased, while those of the temporalis did not change. They explain their findings by recruitment of different fiber types, leading to the assumption that the deep masseter (pars profunda) and anterior temporalis muscles have relatively higher proportions of type I fibers as their MPF was higher for low forces. In contrast, the superficial masseter (pars superficialis) and posterior temporalis contain more type II fibers, as the MPF was low (Farella et al., 2002).

According to the Henneman principle, the deep masseter, which is further away from the electrode and therefore contributes with a weaker signal, and anterior temporalis are preferably activated at lower bite forces. In comparison, the superficial part of the masseter, which is respectively closer to the electrode and therefore contributes with a stronger signal, is activated at higher forces (Henneman et al., 1965). Further studies confirm the distinct functional properties of different fiber types (Koolstra et al., 1988; Windhorst et al., 1989). Farella et al. explain that this heterogeneous recruitment phenomenon could be the underlying reason for the significant decrease in MPF (Farella et al., 2002). This study mirrors the already investigated heterogeneous activation capability of the masticatory muscles which has often been reported (Blanksma et al., 1997; Farella et al., 2002; Terebesi et al., 2016; Türp et al., 2002; Van Eijden, 1990). The studies outlined above illustrate that the variable “EMG activity” is significantly reduced after exercise. This finding is not in conformity with our study findings, as the RMS % MVC of bruxers and non-bruxers did not differ significantly and neither did the intervention with the active GrindCare® three weeks post-baseline. The study by Farella et al. is highly relevant as a comparison, as it has shown that the deep masseter generates higher EMG activity for low bite forces (25N) than the superficial masseter (Farella et al., 2002).

Furthermore, Van Eijden and Blanksma have pointed out that the masseter appears to differ not only on intramuscular but also on intraregional level showing different EMG activities in the anteroposterior direction of the deep masseter (Blanksma et al., 1997). Although the study by Van Eijden demonstrates that static vertical bite force activates the right and left sides equally and shows no intraregional differences, he did not correlate the existing findings to concrete bite forces. In addition, the BiteFork® system interferes slightly with the vertical dimension of occlusion which nevertheless changes neuromuscular mechanisms (Terebesi et al., 2016) and influences the habitual cooperation of muscles and intramuscular regions. The results of the abovementioned studies might suggest that intramuscular fine-wire electrodes might have been preferable to detect differences in EMG activity for the low exerted bite force of 25N per side.

4.3.4.2. Heterogeneity of the MM in relation to bite force

In 1990 Van Eijden investigated the correlation between the direction of bite force and the activity of the right and left masticatory muscles. On average, a constant vertical bite force activates the right and left muscles equally, while the masseter and posterior temporal muscle show the most variety in activity during variously directed bite forces (Van Eijden, 1990). Blanksma et al. have studied the partitioning of the masticatory muscles by acquiring data from six regions of the temporal and three regions of the masseter muscle during laterodeviations, protrusion/retrusion, and opening/closing movements. The results confirm a functional partition of the masseter muscle into an anterior deep, posterior deep, and superficial part (Blanksma et al., 1992). In 1993 Van Eijden already analyzed the EMG activity during selected motor tasks, for example static bites, and found muscle region-specific differences for the temporalis and masseter muscle. For instance, the anteroposterior differences in activation of the deep masseter have not been found for the superficial part. Furthermore, the study has shown that the EMG peak is often passed on from the deep to the superficial masseter and vice versa, at various tasks (Van Eijden et al., 1993). Interestingly, Schindler et al. have also investigated the correlation between the muscle region preferably active during various motor tasks by simulating clenching and grinding forces and found a heterogeneous activation of the masseter muscle (Schindler et al., 2005).

These outcomes point out the highly complex neuromuscular mechanisms of particularly the masseter. They also confirm that an intramuscular EMG should have been preferably used at these low bite forces and if so, a measurement of the complete anteroposterior masseter might have been necessary to record the desired signal. Besides, a comparison of symmetry between the right and left muscles including intraregional comparisons might have revealed differences that could not be found within the current scheme of analysis. For instance, the fatigue testing and symmetry index might have given further insight into the activation patterns of these muscles but were not evaluated in this dissertation. A further examination of the data will nonetheless be conducted.

4.3.4.3. Impact of SB on the EMG of the masseter and temporalis muscle

One of the few studies which compare the EMG of patients with SB and the control group without painful symptoms is from Ruhland, in 1988.

They performed an EMG of the masseter muscle on 19 volunteers (7 possible bruxers and 12 control group, verified via questionnaire) and found that bruxers' power spectra of clenching and chewing activities were significantly different from the control group (Ruhland et al., 1988). The questionnaire used in our study was the OBC and therefore one of our outcomes evaluated differences in RMS % MVC between subjects with high and low oral behaviors. However, no statistically relevant differences regarding RMS % MVC were found in this study, although the spectral density was not evaluated. A more relevant study is the recent one by Palinkas et al. from 2016. Their complex study protocol entails different tools for bruxism research, such as BiteStrip®, polysomnography, EMG in laboratory setting, ultrasound, gnathodynamometry, and entails a sample of 45 SB subjects and 45 subjects in the control group. They investigated the impact of SB on the EMG of the masseter, temporalis, and maximal bite force. The EMG findings of this study are highly relevant and summarized in the following abstract (Palinkas et al., 2016):

- During rest, SB subjects show a decreased EMG activity for both masseter and temporalis muscle.
- With time and compared to the control group, bruxers tend to have a higher number of active motor units, a decrease of the firing frequency of motor neurons, and a decrease in myoelectric activity, as a consequence of extended neuromuscular activity. Accumulation of lactate and pH decrease are underlying causes which can change the physiology of muscular structure (Cecílio et al., 2010).
- The temporalis muscles react the most to psychological stress with increased myoelectric activity and are more active than the masseter at rest, for both bruxers and non-bruxers.
- Lower EMG activities were recorded for SB compared with the control group during dynamic and static contraction (such as rest, protrusion, right and left laterality, dental clenching with Parafilm M, habitual chewing with peanuts, and raisins). This may be explained by a “deactivation and de-recruitment” (Palinkas et al., 2016) of motor units to prevent muscle injury in the process of muscle fatigue.

Our outcomes could not confirm the lower EMG activities for bruxer, or any differences as such. This circumstance is likely to be associated with the bite force as well, since the exerted bite force was adjusted to 50 N, while the motor tasks performed by the subjects in the study by Palinkas et al. are all physiological motor tasks at physiological force.

4.3.5. Limitation of results

One of the main limitations of the outcomes also refers to the subject sample. The observed power for the selected study sample (groups: intervention *N*=20, control *N*=20) of all performed tests was lower than the desired value of 0.80. Consequently, the findings are inconclusive since it cannot be excluded that while maintaining the current study protocol, a more extensive study sample would have revealed a statistically significant difference.

The low bite force of 50N probably activated the deep masseter which would have been better detected by using an intramuscular EMG. Furthermore, the bite force of 50 N is insufficient for changing the EMG activity, as proven by (Hellmann et al., 2011). The heterogeneity of the masseteric recruitment patterns requires a large variety of tests to detect possible differences. Another scheme of computing, for instance, a symmetry index or fatigue testing, might have revealed these differences. These tests should be further elaborated but will not be part of this dissertation.

4.4. Conclusion

One main finding of this study was that no differences in the RMS % MVC were observed between the subgroups gender and bruxism activity (GC and OBC-score) at baseline. Hence, the two study groups (intervention and control group) were considered roughly homogeneous. The analysis of the RMS at MVC basically showed no differences apart from RMASA which was more active in the male group at T1 and T3, and LMASP in both groups, which differed between T1 and T2. The comparison of the RMS % MVC values between the three timepoints (T1, T2, and T3) within both groups and the comparison at each timepoint between groups (intervention vs. control) was not significant, with few exceptions (within groups: LMASM, LMASP, RTA, and LTA for SBF1; LTA for SBF 2; between groups: RTA in T2).

The comparison of RMS % MVC at 50N between T1, T2, and T3 resulted in a significant difference for RMASM and RTA. However, these findings do not seem to have major clinical relevance.

It can be stated that this study has not observed any substantiating differences between the EMG values of participants with high - and low bruxism activity. Our hypothesis which supposes that subjects with high and low bruxism activity differ in RMS % MVC could not be verified.

Furthermore, CES does not seem to influence the studied EMG parameters in the two weeks intervention time while the EMG activity neither differed between the control and intervention group, nor between the probands with high and low bruxism activity. The heterogeneity of the masseteric recruitment patterns requires a large variety of tests, such as a symmetry index or fatigue testing, to possibly reveal existing differences. The results of this study may provide data for a sample size calculation for future studies in bruxism research.

4.5. Future directions

For future research on the differences between subjects with high and low bruxism activity, it is proposed to extend the GrindCare® intervention to 4 weeks according to current recommendations, to increase the possibility of successful cessation of the bruxism behavior. Potential differences shall still be detected by a surface EMG while simultaneously performing the tasks with a larger submaximal bite force, such as 150N - 300N. The forces shall differ distinctively so that the resolution of the device allows the measurement of clearly different forces, within the measurement range of the sensors. As for the study sample, a larger number of matched subjects is required to improve the observed power.

5. Abstract

The main objective of this study was to test whether subjects with different degrees of bruxism differ regarding EMG parameters and whether CES intervention affects those parameters. The hypothesis was that CES influences EMG parameters and after its' cessation, all EMG parameters return to baseline (exposure–response relationship).

For this purpose, forty subjects were examined, 16 men and 24 women, matched for age and gender and assigned randomly in the intervention (*N*=20) and control group (*N*=20). The procedure was as follows: 1-week inactive GC (*N*=40), 2 weeks inactive/active GC (*N*=20/*N*=20), 2 weeks inactive GC (*N*=40). Each interval was followed by a surface EMG recording from eight muscle parts (right and left anterior -, medial -, and posterior masseter and right and left anterior temporalis) under force-controlled feedback (BiteFork®) with three submaximal bite forces. The resulting EMG activity is expressed as RMS % MVC and RMS at MVC. The statistics is performed with t-test, one-way rmANOVA, and Friedman rmANOVA on ranks, according to the distribution of the data. The significance level was set at $p \leq 0.05$.

The results generated from the within-groups and between-groups comparison were mostly not statistically significant and could therefore not offer clinically relevant conclusions.

However, it cannot be excluded that a higher submaximal bite force and an extended intervention interval would have rendered different outcomes. The insufficient study sample resulted in a low observed power which makes the findings prone to Type II error. It can be concluded that this study did not find any substantiating differences between the EMG values of participants with various bruxism activity and that CES could not influence the studied EMG parameters in the two weeks intervention time.

Our hypothesis which supposes that subjects with high and low bruxism activity differ in RMS % MVC could not be verified. However, with the gained knowledge, it is recommended to further elaborate a definite bruxism diagnosis by using portable EMG devices.

5.1. Zusammenfassung

Das Hauptziel dieser Studie bestand darin zu prüfen, ob sich Probanden mit unterschiedlichem Bruxismusgrad hinsichtlich der EMG-Parameter unterscheiden und darüber hinaus, ob bedingte elektrische Stimulation diese beeinflusst. Die Hypothese lautete, dass die bedingte elektrische Stimulation die EMG-Parameter beeinflusst und nach Einstellung alle EMG-Parameter zum Ausgangswert zurückkehren.

Zu diesem Zweck wurden vierzig Probanden untersucht (16 Männer und 24 Frauen), die nach Alter und Geschlecht gematcht und zufällig in die Interventions- (*N*=20) und die Kontrollgruppe (*N*= 20) eingeteilt wurden. Das Studienprotokoll begann mit einer Woche inaktivem GC (*N*=40), gefolgt von zwei Wochen inaktivem/aktivem GC (*N*=20/*N*=20) und endete mit zwei Wochen inaktivem GC (*N*=40). Nach jedem Intervall erfolgte eine Oberflächen-EMG-Aufzeichnung der acht Muskelpartien (rechter und linker anteriorer -, medialer - und posteriorer Masseter, sowie rechter und linker anteriorer Temporalis) unter Kraft-kontrolliertem Feedback (BiteFork®). Die statistische Auswertung erfolgte mittels t-Test, einseitiger rmANOVA und Friedman rmANOVA, je nach Verteilung der Daten. Das Signifikanzniveau wurde auf $p \leq 0,05$ festgelegt.

Die Ergebnisse aus den Vergleichen waren statistisch nicht signifikant und konnten daher keine klinisch relevanten Schlussfolgerungen liefern.

Dennoch kann nicht ausgeschlossen werden, dass eine höhere Beißkraft und ein längeres Interventionsintervall zu anderen Ergebnissen führen könnten. Die kleine Stichprobe führte zu einer geringen Teststärke, wodurch ein Risiko für einen Fehler 2. Art besteht. Es lässt sich schlussfolgern, dass diese Studie keine begründeten Unterschiede zwischen den EMG-Werten von Teilnehmern unterschiedlicher Bruxismusaktivität gefunden hat, und außerdem, dass die bedingte elektrische Stimulation in dem zweiwöchigen Intervall zu keiner Veränderung der EMG-Parameter geführt hat.

Unsere Hypothese, dass sich Probanden mit hoher und niedriger Bruxismusaktivität im EMG (RMS % MVC) voneinander unterscheiden, konnte nicht verifiziert werden.

6. References

Abbink, J., van der Bilt, A., Bosman, F., & van der Glas, H. (1999). Speed-dependent control of cyclic open-close movements of the human jaw with an external force counteracting closing. *J Dent Res, 78*(4), 878-886.

Abe, S., Yamaguchi, T., Rompre, P., De Grandmont, P., Chen, Y., & Lavigne, G. (2009). Tooth wear in young subjects: a discriminator between sleep bruxers and controls? *Int J Prosthodont, 22*(4), 342-350.

Abraham, J., Pierce, C., Rinchuse, D., & Zullo, T. (1992). Assessment of buccal separators in the relief of bruxist activity associated with myofascial pain-dysfunction. *Angle Orthod, 62*(3), 177-184.

Ahlberg, J., Savolainen, A., Rantala, M., Lindholm, H., & Könönen, M. (2004). Reported bruxism and biopsychosocial symptoms: a longitudinal study. *Community Dent Oral Epidemiol, 32*(4), 307-311.

Al-Wayli, H. (2017). Treatment of chronic pain associated with nocturnal bruxism with botulinum toxin. A prospective and randomized clinical study. *J Clin Exp Dent, 9*(1), 112-117.

Amorim, C., Paes, F., santos de Faria Junior, N., de Oliveira, L., & Politti, F. (2012). Electromyographic analysis of masseter and anterior temporalis muscle in sleep bruxers after occlusal splint wearing. *J Bodyw Mov Ther, 16*(2), 199-203.

Baad-Hansen, L., Thymi, M., Lobbezoo, F., & Svensson, P. (2019). To what extent is bruxism associated with musculoskeletal signs and symptoms? A systematic review. *J Oral Rehabil, 46*(9), 845-861.

Bakke, M. (2006). Bite Force and Occlusion. *Semin Orthod, 12*(2), 120-126.

Becker, N. (2021). *Untersuchung zum Effekt eines elektrischen Stimulus des Musculus temporalis auf orofaziale Schmerzen bei Patienten mit Bruxismussymptomen.* Universität Greifswald

Bezanson, J., Karpinski, S., Shah, V., & Edelman, A. (2012). Julia: A fast dynamic language for technical computing. *arXiv preprint arXiv:12095145*.

Binboğa, E., Tok, S., Catikkas, F., Guven, S., & Dane, S. (2013). The effects of verbal encouragement and conscientiousness on maximal voluntary contraction of the triceps surae muscle in elite athletes. *J Sports Sci, 31*(9), 982-988.

Blanksma, N., Van Eijden, T., & Weijs, W. (1992). Electromyographic Heterogeneity in the Human Masseter Muscle. *J Dent Res, 71*(1), 47-52.

Blanksma, N., Van Eijden, T., Van Ruijven, L., & Weijs, W. (1997). Electromyographic heterogeneity in the human temporalis and masseter muscles during dynamic tasks guided by visual feedback. *J Dent Res, 76*(1), 542-551.

Bloor, D., Donnelly, K., Hands, P., Laughlin, P., & Lussey, D. (2005). A metal–polymer composite with unusual properties. *J Phys D Appl Phys 38*(16), 2851.

Boudreau, S., Hennings, K., Svensson, P., Sessle, B., & Arendt-Nielsen, L. (2010). The effects of training time, sensory loss and pain on human motor learning. *J Oral Rehabil, 37*(9), 704-718.

Bracci, A., Djukic, G., Favero, L., Salmaso, L., Guarda-Nardini, L., & Manfredini, D. (2018). Frequency of awake bruxism behaviours in the natural environment. A 7-day, multiple-point observation of real-time report in healthy young adults. *J Oral Rehabil, 45*(6), 423-429.

Bucci, R., Koutris, M., Lobbezoo, F., & Michelotti, A. (2019). Occlusal sensitivity in individuals with different frequencies of oral parafunction. *J Prosthet Dent, 122*(2), 119-122.

Burdette, B., & Gale, E. (1990). Reliability of surface electromyography of the masseteric and anterior temporal areas. *Arch Oral Biol, 35*(9), 747-751.

Carlsson, G., Egermark, I., & Magnusson, T. (2003). Predictors of bruxism, other oral parafunctions, and tooth wear over a 20-year follow-up period. *J Orofac Pain, 17*(1), 50-57.

Carra, M., Bruni, O., & Huynh, N. (2012). Topical review: sleep bruxism, headaches, and sleep-disordered breathing in children and adolescents. *J Orofac Pain, 26*(4), 267-276.

Castroflorio, T., Farina, D., Bottin, A., Piancino, M., Bracco, P., & Merletti, R. (2005). Surface EMG of jaw elevator muscles: effect of electrode location and inter-electrode distance. *J Oral Rehabil, 32*(6), 411-417.

Castroflorio, T., Bargellini, A., Rossini, G., Cugliari, G., Deregibus, A., & Manfredini, D. (2015). Agreement between clinical and portable EMG/ECG diagnosis of sleep bruxism. *J Oral Rehabil, 42*(10), 759-764.

Cecílio, F., Regalo, S., Palinkas, M., Issa, J., Siéssere, S., Hallak, J., et al. (2010). Ageing and surface EMG activity patterns of masticatory muscles. *J Oral Rehabil, 37*(4), 248-255.

Conti, P., Stuginski-Barbosa, J., Bonjardim, L., Soares, S., & Svensson, P. (2014). Contingent electrical stimulation inhibits jaw muscle activity during sleep but not pain intensity or masticatory muscle pressure pain threshold in self-reported bruxers: a pilot study. *Oral Surg Oral Med Oral Pathol Oral Radiol, 117*(1), 45-52.

De la Hoz-Aizpurua, J., Diaz-Alonso, E., LaTouche-Arbizu, R., & Mesa-Jimenez, J. (2011). Sleep bruxism. Conceptual review and update. *Med Oral Patol Oral Cir Bucal, 16*(2), 231-238.

Desmedt, J., & Godaux, E. (1976). Habituation of exteroceptive suppression and of exteroceptive reflexes in man as influenced by voluntary contraction. *Brain Res, 106*(1), 21-29.

Dessem, D., Iyadurai, O., & Taylor, A. (1988). The role of periodontal receptors in the jaw-opening reflex in the cat. *J Physiol, 406*(1), 315-330.

Donnarumma, V., Cioffi, I., Michelotti, A., Cimino, R., Vollaro, S., & Amato, M. (2018). Analysis of the reliability of the Italian version of the Oral Behaviours Checklist and the relationship between oral behaviours and trait anxiety in healthy individuals. *J Oral Rehabil, 45*(4), 317-322.

Donnarumma, V., Ohrbach, R., Simeon, V., Lobbezoo, F., Piscicelli, N., & Michelotti, A. (2021). Association between waking-state oral behaviors, according to the Oral Behaviors Checklist, and TMD subgroups. *J Oral Rehabil, 48*(9), 996-1003.

Dreyer, P., Yachida, W., Huynh, N., Lavigne, G., Haugland, M., Svensson, P., et al. (2015). How close can single-channel EMG data come to PSG scoring of rhythmic masticatory muscle activity. *J Dent Sleep Med, 2*(4), 147-156.

Dube, C., Rompre, P., Manzini, C., Guitard, F., De Grandmont, P., & Lavigne, G. (2004). Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. *J Dent Res, 83*(5), 398-403.

Eberhard, L., Braun, S., Wirth, A., Schindler, H. J., Hellmann, D., & Giannakopoulos, N. (2014). The effect of experimental balancing interferences on masticatory performance. *J Oral Rehabil, 41*(5), 346-352.

Egermark, I., Carlsson, G., & Magnusson, T. (2001). A 20-year longitudinal study of subjective symptoms of temporomandibular disorders from childhood to adulthood. *Acta Odontol Scand, 59*(1), 40-48.

Ella, B., Ghorayeb, I., Burbaud, P., & Guehl, D. (2017). Bruxism in Movement Disorders: A Comprehensive Review. *J Prosthodont, 26*(7), 599-605.

Farella, M., Van Eijden, T., Baccini, M., & Michelotti, A. (2002). Task-related electromyographic spectral changes in the human masseter and temporalis muscles. *Eur J Oral Sci, 110*(1), 8-12.

Fernandes, C., Glantz, P., Svensson, S., & Bergmark, A. (2003). A novel sensor for bite force determinations. *Dent Mater, 19*(2), 118-126.

Fernandes, G., Franco, A., Siqueira, J., Goncalves, D., & Camparis, C. (2012). Sleep bruxism increases the risk for painful temporomandibular disorder, depression and non-specific physical symptoms. *J Oral Rehabil, 39*(7), 538-544.

Ferrario, V., Tartaglia, G., Galletta, A., Grassi, G., & Sforza, C. (2006). The influence of occlusion on jaw and neck muscle activity: a surface EMG study in healthy young adults. *J Oral Rehabil, 33*(5), 341-348.

Frame, J., Rothwell, P., & Duxbury, A. (1973). The standardization of electromyography of the masseter muscle in man. *Arch Oral Biol, 18*(11), 1419-1423.

Gallo, L., Lavigne, G., Rompré, P., & Palla, S. (1997). Reliability of scoring EMG orofacial events: polysomnography compared with ambulatory recordings. *J Sleep Res, 6*(4), 259-263.

Garnick, J. (1975). Reproducibility of the Electromyogram. *J Dent Res, 54*(4), 867-871.

Gesch, D., Bernhardt, O., Mack, F., John, U., Kocher, T., & Alte, D. (2005). Association of Malocclusion and Functional Occlusion with Subjective Symptoms of TMD in Adults: Results of the Study of Health in Pomerania (SHIP). *Angle Orthod, 75*(2), 183-190.

Giannakopoulos, N., & Schmitter, M. (2013). Diagnostik von Bruxismus. *Zahnmedizin up2date, 7*(02), 155-167.

Giannakopoulos, N., Rauer, A., Hellmann, D., Hugger, S., Schmitter, M., & Hugger, A. (2018). Comparison of device-supported sensorimotor training and splint intervention for myofascial temporomandibular disorder pain patients. *J Oral Rehabil, 45*(9), 669-676.

Gibbs, C., Mahan, P., Lundeen, H., Brehnan, K., Walsh, E., Sinkewiz, S., et al. (1981). Occlusal forces during chewing--influences of biting strength and food consistency. *J Prosthet Dent, 46*(5), 561-567.

Godaux, E., & Desmedt, J. (1975). Exteroceptive suppression and motor control of the masseter and temporalis muscles in normal man. *Brain Res, 85*(3), 447-458.

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 sleep bruxism: a randomized clinical trial. *Chiropr Man Therap, 22*(1), 43.

Gonzalez, Y., Schiffman, E., Gordon, S., Seago, B., Truelove, E., Slade, G., et al. (2011). Development of a brief and effective temporomandibular disorder pain screening questionnaire: reliability and validity. *J Am Dent Assoc, 142*(10), 1183-1191.

Greven, M., Onodera, K., & Sato, S. (2015). The use of the BruxChecker in the evaluation and treatment of bruxism *J CranioMandib Func, 7*(3), 1-11.

Guaita, M., & Högl, B. (2016). Current Treatments of Bruxism. *Curr Treat Options Neurol, 18*(2), 10-10.

Gupta, R., Luthra, R., & Mehra, A. (2017). Nocturnal bruxism: A review *Int J Appl Dent Sci, 3(4): 225-229*.

Helkimo, E., Carlsson, G., & Helkimo, M. (1977). Bite force and state of dentition. *Acta Odontol Scand, 35*(6), 297-303.

Hellmann, D., Giannakopoulos, N., Blaser, R., Eberhard, L., Rues, S., & Schindler, H. (2011). Long-term training effects on masticatory muscles. *J Oral Rehabil, 38*(12), 912-920.

Henneman, E., Somjen, G., & Carpenter, D. (1965). Functional significance of cell size in spinal motoneurons. *J Neurophysiol, 28*, 560-580.

Hollowell, D., & Suratt, P. (1989). Activation of masseter muscles with inspiratory resistance loading. *J Appl Physiol (1985), 67*(1), 270-275.

Holmgren, K., Sheikholeslam, A., & Riise, C. (1993). Effect of a full-arch maxillary occlusal splint on parafunctional activity during sleep in patients with nocturnal bruxism and signs and symptoms of craniomandibular disorders. *J Prosthet Dent, 69*(3), 293-297.

Hosman, H., & Naeije, M. (1979). Reproducibility of the normalized electromyographic recordings of the masseter muscle by using the EMG recording during maximal clenching as a standard. *J Oral Rehabil, 6*(1), 49-54.

Hugger, A., Hugger, S., & Schindler, H. (2008). Surface electromyography of the masticatory muscles for application in dental practice. Current evidence and future developments. *Int J Comput Dent, 11*(2), 81-106.

Huynh, N., Manzini, C., Rompre, P., & Lavigne, G. (2007). Weighing the potential effectiveness of various treatments for sleep bruxism. *J Can Dent Assoc, 73*(8), 727-730.

Im, Y., Han, S., Park, J., Lim, H., Kim, B., & Kim, J. (2017). Repeatability of measurements of surface electromyographic variables during maximum voluntary contraction of temporalis and masseter muscles in normal adults. *J Oral Sci, 59*(2), 233-245.

Jadidi, F., Castrillon, E., & Svensson, P. (2008). Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. *J Oral Rehabil, 35*(3), 171-183.

Jadidi, F., Nørregaard, O., Baad-Hansen, L., Arendt-Nielsen, L., & Svensson, P. (2011). Assessment of sleep parameters during contingent electrical stimulation in subjects with jaw muscle activity during sleep: a polysomnographic study. *Eur J Oral Sci, 119*(3), 211-218.

Jadidi, F., Castrillon, E., Nielsen, P., Baad-Hansen, L., & Svensson, P. (2013). Effect of contingent electrical stimulation on jaw muscle activity during sleep: a pilot study with a randomized controlled trial design. *Acta Odontol Scand, 71*(5), 1050-1062.

Jirakittayakorn, N., & Wongsawat, Y. (2014). *An EMG instrument designed for bruxism detection on masseter muscle.* Paper presented at the The 7th 2014 Biomedical Engineering International Conference.

Johansson, A., Omar, R., & Carlsson, G. (2011). Bruxism and prosthetic treatment: a critical review. *J Prosthodont Res, 55*(3), 127-136.

Jokubauskas, L., & Baltrusaityte, A. (2018). Efficacy of biofeedback therapy on sleep bruxism: A systematic review and meta-analysis. *J Oral Rehabil, 45*(6), 485-495.

Kato, T., Thie, N., Huynh, N., Miyawaki, S., & Lavigne, G. (2003). Topical review: sleep bruxism and the role of peripheral sensory influences. *J Orofac Pain, 17*(3).

Kluth, K., Steinhilber, B., & Nesseler, T. (2013). Oberflächen-Elektromyographie in der Arbeitsmedizin, Arbeitsphysiologie und Arbeitswissenschaft. *Zeitschrift für Arbeitswissenschaft, 67*(2), 113-128.

Koç, D., Doğan, A., & Bek, B. (2010). Bite force and influential factors on bite force measurements: a literature review. *Eur J Dent 4*(2), 223-232.

Konrad, P. (2005). The abc of emg. *A practical introduction to kinesiological electromyography, 1*(2005), 30-35.

Koolstra, J., Van Eijden, T., Weijs, W., & Naeije, M. (1988). A three-dimensional mathematical model of the human masticatory system predicting maximum possible bite forces. *J Biomech, 21*(7), 563-576.

Koyano, K., Tsukiyama, Y., Ichiki, R., & Kuwata, T. (2008). Assessment of bruxism in the clinic. *J Oral Rehabil, 35*(7), 495-508.

Kuhn, M., & Türp, J. (2018). Risk factors for bruxism. *Swiss Dent J, 128*, 118-124.

Kumar, A., Grigoriadis, J., Trulsson, M., Svensson, P., & Svensson, K. (2015). Effects of short-term training on behavioral learning and skill acquisition during intraoral fine motor task. *Neuroscience, 306*, 10-17.

Lavigne, G., Kim, J., Valiquette, C., & Lund, J. (1987). Evidence that periodontal pressoreceptors provide positive feedback to jaw closing muscles during mastication. *J Neurophysiol, 58*(2), 342-358.

Lavigne, G., & Montplaisir, J. (1994). Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. *Sleep, 17*(8), 739-743.

Lavigne, G., Rompre, P., & Montplaisir, J. (1996). Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res, 75*(1), 546-552.

Lavigne, G., Rompre, P., Poirier, G., Huard, H., Kato, T., & Montplaisir, J. (2001). Rhythmic masticatory muscle activity during sleep in humans. *J Dent Res, 80*(2), 443-448.

Lavigne, G., Kato, T., Kolta, A., & Sessle, B. (2003). Neurobiological mechanisms involved in sleep bruxism. *Crit Rev Oral Biol Med, 14*(1), 30-46.

Lavigne, G., Khoury, S., Abe, S., Yamaguchi, T., & Raphael, K. (2008). Bruxism physiology and pathology: an overview for clinicians. *J Oral Rehabil, 35*(7), 476-494.

Lee, S., McCall, W. J., Kim, Y., Chung, S., & Chung, J. (2010). Effect of botulinum toxin injection on nocturnal bruxism: a randomized controlled trial. *Am J Phys Med Rehabil, 89*(1), 16-23.

Lobbezoo, F., Lavigne, G., Tanguay, R., & Montplaisir, J. (1997). The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. *Mov Disord, 12*(1), 73-78.

Lobbezoo, F., & Naeije, M. (2001a). A reliability study of clinical tooth wear measurements. *J Prosthet Dent, 86*(6), 597-602.

Lobbezoo, F., & Naeije, M. (2001b). Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil, 28*(12), 1085-1091.

Lobbezoo, F., Van Der Zaag, J., Van Selms, M., Hamburger, H., & Naeije, M. (2008). Principles for the management of bruxism. *J Oral Rehabil, 35*(7), 509-523.

Lobbezoo, F., Ahlberg, J., Manfredini, D., & Winocur, E. (2012). Are bruxism and the bite causally related? *J Oral Rehabil, 39*(7), 489-501.

Lobbezoo, F., Ahlberg, J., Glaros, A., Kato, T., Koyano, K., Lavigne, G., et al. (2013). Bruxism defined and graded: an international consensus. *J Oral Rehabil, 40*(1), 2-4.

Lobbezoo, F., Ahlberg, J., Raphael, K., Wetselaar, P., Glaros, A., Kato, T., et al. (2018). International consensus on the assessment of bruxism: Report of a work in progress. *J Oral Rehabil, 45*(11), 837-844.

Lobbezoo, F., Aarab, G., Ahlers, M., Baad-Hansen, L., Bernhardt, O., Castrillon, E., et al. (2019). Consensus-based clinical guidelines for ambulatory electromyography and contingent electrical stimulation in sleep bruxism. *J Oral Rehabil, 47*(2), 164-169.

Loomans, B., & Opdam, N. (2018). A guide to managing tooth wear: the Radboud philosophy. *Br Dent J, 224*(5), 348-356.

Lotze, M., Braun, C., Birbaumer, N., Anders, S., & Cohen, L. (2003). Motor learning elicited by voluntary drive. *Brain, 126*(4), 866-872.

Lund, J., & Olsson, K. (1983). The importance of reflexes and their control during jaw movement. *Trends Neurosci, 6*, 458-463.

Macedo, C., Silva, A., Machado, M., Saconato, H., & Prado, G. (2007). Occlusal splints for treating sleep bruxism (tooth grinding). *Cochrane Database Syst Rev*(4), Cd005514.

Machines, I. (2013). IBM SPSS statistics for windows, version 22.0. *IBM Corp Armonk, NY*.

Madani, A., Abdollahian, E., Azangoo Khiavi, H., Radvar, M., Foroughipour, M., Asadpour, H., et al. (2013). The efficacy of gabapentin versus stabilization splint in management of sleep bruxism. *J Prosthodont, 22*(2), 126-131.

Manfredini, D., Landi, N., Tognini, F., Montagnani, G., & Bosco, M. (2004a). Occlusal features are not a reliable predictor of bruxism. *Minerva Stomatol, 53*(5), 231-239.

Manfredini, D., Landi, N., Romagnoli, M., & Bosco, M. (2004b). Psychic and occlusal factors in bruxers. *Aust Dent J, 49*(2), 84-89.

Manfredini, D., & Lobbezoo, F. (2009). Role of psychosocial factors in the etiology of bruxism. *J Orofac Pain, 23*(2), 153-166.

Manfredini, D., & Lobbezoo, F. (2010). Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod, 109*(6), 26-50.

Manfredini, D., Winocur, E., Guarda-Nardini, L., & Lobbezoo, F. (2012). Self-reported bruxism and temporomandibular disorders: findings from two specialised centres. *J Oral Rehabil, 39*(5), 319-325.

Manfredini, D., Winocur, E., Guarda-Nardini, L., Paesani, D., & Lobbezoo, F. (2013). Epidemiology of bruxism in adults: a systematic review of the literature. *J Orofac Pain, 27*(2), 99-110.

Manfredini, D., Ahlberg, J., Castroflorio, T., Poggio, C., Guarda-Nardini, L., & Lobbezoo, F. (2014). Diagnostic accuracy of portable instrumental devices to measure sleep bruxism: a systematic literature review of polysomnographic studies. *J Oral Rehabil, 41*(11), 836-842.

Manfredini, D., Guarda-Nardini, L., Marchese-Ragona, R., & Lobbezoo, F. (2015a). Theories on possible temporal relationships between sleep bruxism and obstructive sleep apnea events. An expert opinion. *Sleep Breath, 19*(4), 1459-1465.

Manfredini, D., Ahlberg, J., Winocur, E., & Lobbezoo, F. (2015b). Management of sleep bruxism in adults: a qualitative systematic literature review. *J Oral Rehabil, 42*(11), 862-874.

Manfredini, D., Bracci, A., & Djukic, G. (2016a). BruxApp: the ecological momentary assessment of awake bruxism. *Minerva stomatol, 65*(4), 252-255.

Manfredini, D., Arreghini, A., Lombardo, L., Visentin, A., Cerea, S., Castroflorio, T., et al. (2016b). Assessment of anxiety and coping features in bruxers: a portable electromyographic and electrocardiographic study. *J Oral Facial Pain Headache, 30*(3), 249-254.

Manfredini, D., Ahlberg, J., Wetselaar, P., Svensson, P., & Lobbezoo, F. (2019). The bruxism construct: From cut-off points to a continuum spectrum. *J Oral Rehabil, 46*(11), 991-997.

Manfredini, D., Ahlberg, J., Aarab, G., Bracci, A., Durham, J., Ettlin, D., et al. (2020). Towards a Standardized Tool for the Assessment of Bruxism (STAB)-Overview and general remarks of a multidimensional bruxism evaluation system. *J Oral Rehabil, 47*(5), 549-556.

Markiewicz, M., Ohrbach, R., & McCall, W. J. (2006). Oral behaviors checklist: reliability of performance in targeted waking-state behaviors. *J Orofac Pain, 20*(4), 306-316.

Matsumoto, H., Tsukiyama, Y., Kuwatsuru, R., & Koyano, K. (2015). The effect of intermittent use of occlusal splint devices on sleep bruxism: a 4-week observation with a portable electromyographic recording device. *J Oral Rehabil, 42*(4), 251-258.

McNair, P., Depledge, J., Brettkelly, M., & Stanley, S. (1996). Verbal encouragement: effects on maximum effort voluntary muscle: action. *Br J Sports Med 30*(3), 243-245.

Merletti, R., & Parker, P. (2004). *Electromyography: physiology, engineering, and non-invasive applications* (Vol. 11): John Wiley & Sons.

Milosevic, A., Agrawal, N., Redfearn, P., & Mair, L. (1999). The occurrence of toothwear in users of Ecstasy (3, 4 MethyleneDioxyMethAmphetamine). *Community Dent Oral Epidemiol, 27*(4), 283-287.

Muellbacher, W., Ziemann, U., Boroojerdi, B., Cohen, L., & Hallett, M. (2001). Role of the human motor cortex in rapid motor learning. *Exp Brain Res, 136*(4), 431-438.

Murali, R., Rangarajan, P., & Mounissamy, A. (2015). Bruxism: Conceptual discussion and review. *J Pharm Bioallied Sci, 7*(1), 265-270.

Needham, R., & Davies, S. (2013). Use of the Grindcare® device in the management of nocturnal bruxism: a pilot study. *Br Dent J, 215*(1), E1-E1.

Ohmure, H., Oikawa, K., Kanematsu, K., Saito, Y., Yamamoto, T., Nagahama, H., et al. (2011). Influence of experimental esophageal acidification on sleep bruxism: a randomized trial. *J Dent Res, 90*(5), 665-671.

Ohrbach, R., Markiewicz, M., & McCall, W. J. (2008). Waking-state oral parafunctional behaviors: specificity and validity as assessed by electromyography. *Eur J Oral Sci, 116*(5), 438-444.

Ommerborn, M., Giraki, M., Schneider, C., Schäfer, R., Gotter, A., Franz, M., et al. (2015). Diagnosis and quantification of sleep bruxism: New analysis method for the Bruxcore Bruxism Monitoring Device is suitable for clinical use. *J CranioMandib Func, 7*, 135-150.

Ottenhoff, F., van der Bilt, A., van der Glas, H., & Bosman, F. (1992). Control of elevator muscle activity during simulated chewing with varying food resistance in humans. *J Neurophysiol, 68*(3), 933-944.

Paesani, D., Andersen, M., & Arima, T. (2010). Bruxism: Theory and Practice. *Quintessence Int, 1/2011*, 560

Palinkas, M., Bataglion, C., De Luca Canto, G., Machado Camolezi, N., Theodoro, G., Siéssere, S., et al. (2016). Impact of sleep bruxism on masseter and temporalis muscles and bite force. *Cranio - J Craniomandib Pract, 34*(5), 309-315.

Paredes-Madrid, L., Matute, A., Bareño, J., Parra Vargas, C., & Gutierrez Velásquez, E. (2017). Underlying Physics of Conductive Polymer Composites and Force Sensing Resistors (FSRs). A Study on Creep Response and Dynamic Loading. *Materials (Basel), 10*(11), 1334.

Park, M., Ahn, K., & Jung, D. (2003). Botulinum toxin type A treatment for contouring of the lower face. *Dermatol Surg, 29*(5), 477-483.

Pearce, N. (2016). Analysis of matched case-control studies. *BMJ, 352*, i969.

Peck, C., Wirianski, A., & Murray, G. (2010). Jaw motor plasticity in health and disease. *Comput Methods Biomech Biomed Engin, 13*(4), 455-458.

Peroz, I., Bernhardt, O., Kares, H., Korn, H., Kropp, Lange, M., et al. (2019). Diagnostik und Behandlung von Bruxismus. *083-027*.

Piccoli, L., Besharat, L., Cassetta, M., Migliau, G., Di Carlo, S., & Pompa, G. (2014). Tooth wear among patients suffering from mental disorders. *Ann Stomatol (Roma), 5*(2), 52.

Pintado, M., Anderson, G., DeLong, R., & Douglas, W. (1997). Variation in tooth wear in young adults over a two-year period. *J Prosthet Dent 77*(3), 313-320.

Prasad, S., Paulin, M., Cannon, R., Palla, S., & Farella, M. (2019). Smartphone-assisted monitoring of masticatory muscle activity in freely moving individuals. *Clin Oral Investig, 23*(9), 3601-3611.

Prasad, S., Ramanan, D., Bennani, H., Paulin, M., Cannon, R., Palla, S., et al. (2021). Associations among masticatory muscle activity, physical activity and self-reported oral behaviours in adult women. *Clin Oral Investig*, 1-11.

Preston, J. (1987). Glossary of prosthodontic terms. *J Prosthet Dent, 58*(6), 713-762.

Rantanen, V., Ilves, M., Vehkaoja, A., Kontunen, A., Lylykangas, J., Mäkelä, E., et al. (2016). *A survey on the feasibility of surface EMG in facial pacing.* Paper presented at the 2016 38th Annual International Conference of the IEEE Engineering in Medicine and Biology Society (EMBC).

Raphael, K., Sirois, D., Janal, M., Wigren, P., Dubrovsky, B., Nemelivsky, L., et al. (2012). Sleep bruxism and myofascial temporomandibular disorders: a laboratory-based polysomnographic investigation. *J Am Dent Assoc, 143*(11), 1223-1231.

Raphael, K., Janal, M., Sirois, D., & Svensson, P. (2013). Effect of contingent electrical stimulation on masticatory muscle activity and pain in patients with a myofascial temporomandibular disorder and sleep bruxism. *J Orofac Pain, 27*(1), 21-31.

Raphael, K., Janal, M., Sirois, D., Dubrovsky, B., Klausner, J., Krieger, A., et al. (2015). Validity of self-reported sleep bruxism among myofascial temporomandibular disorder patients and controls. *J Oral Rehabil, 42*(10), 751-758.

Raphael, K., Santiago, V., & Lobbezoo, F. (2016). Is bruxism a disorder or a behaviour? Rethinking the international consensus on defining and grading of bruxism. *J Oral Rehabil 43*(10), 791-798.

Rauer, A., Giannakopoulos, N., Hellmann, D., Hugger, S., Schmitter, M., Schindler, H., et al. (2019). Physiotherapy home exercises and occlusal splint therapy for myofascial TMD pain. *Cranio - J Craniomandib Pract*, 13.

Rintakoski, K., Ahlberg, J., Hublin, C., Broms, U., Madden, P., Könönen, M., et al. (2010). Bruxism Is Associated With Nicotine Dependence: A Nationwide Finnish Twin Cohort Study. *Nicotine Tob Res, 12*, 1254-1260.

Rompre, P., Daigle-Landry, D., Guitard, F., Montplaisir, J., & Lavigne, G. (2007). Identification of a sleep bruxism subgroup with a higher risk of pain. *J Dent Res, 86*(9), 837-842.

Ruhland, J., Jeutter, D., Ackmann, J., Hoge, H., & Jodat, R. (1988). *Acquisition and analysis of electromyograms of the human masseter muscle (bruxism detection).* Paper presented at the Proceedings of the Annual International Conference of the IEEE Engineering in Medicine and Biology Society.

Saletu, A., Parapatics, S., Anderer, P., Matejka, M., & Saletu, B. (2010). Controlled clinical, polysomnographic and psychometric studies on differences between sleep bruxers and controls and acute effects of clonazepam as compared with placebo. *Eur Arch Psychiatry Clin Neurosci, 260*(2), 163-174.

Sato, M., Iizuka, T., Watanabe, A., Iwase, N., Otsuka, H., Terada, N., et al. (2015). Electromyogram biofeedback training for daytime clenching and its effect on sleep bruxism. *J Oral Rehabil, 42*(2), 83-89.

Schindler, H., Stengel, E., & Spiess, W. (1998). Feedback control during mastication of solid food textures--a clinical-experimental study. *J Prosthet Dent, 80*(3), 330-336.

Schindler, H., Rues, S., Türp, J., Schweizerhof, K., & Lenz, J. (2005). Activity patterns of the masticatory muscles during feedback-controlled simulated clenching activities. *Eur J Oral Sci, 113*(6), 469-478.

Shemmell, J., Forner, M., Tresilian, J., Riek, S., Barry, B., & Carson, R. (2005). Neuromuscular adaptation during skill acquisition on a two degree-of-freedom target-acquisition task: isometric torque production. *J Neurophysiol, 94*(5), 3046-3057.

Shetty, S., Pitti, V., Satish Babu, C., Surendra Kumar, G., & Deepthi, B. (2010). Bruxism: a literature review. *J Indian Prosthodont Soc 10*(3), 141-148.

Shiffman, S., Stone, A., & Hufford, M. (2008). Ecological momentary assessment. *Annu Rev Clin Psychol, 4*, 1-32.

Shim, Y., Lee, M., Kato, T., Park, H., Heo, K., & Kim, S. (2014). Effects of botulinum toxin on jaw motor events during sleep in sleep bruxism patients: a polysomnographic evaluation. *J Clin Sleep Med, 10*(3), 291-298.

Shimada, A., Castrillon, E., & Svensson, P. (2019). Revisited relationships between probable sleep bruxism and clinical muscle symptoms. *J Dent, 82*, 85-90.

Stapelmann, H., & Türp, J. (2008). The NTI-tss device for the therapy of bruxism, temporomandibular disorders, and headache - where do we stand? A qualitative systematic review of the literature. *BMC Oral Health, 8*, 22-22.

Stassi, S., Cauda, V., Canavese, G., & Pirri, C. F. (2014). Flexible tactile sensing based on piezoresistive composites: A review. *Sensors, 14*(3), 5296-5332.

Stengel, E. (1999). *Physikalische Beschreibung des Kauvorganges zur Bewertung der Textur fester Lebensmittel*: na.

Stone, A., & Shiffman, S. (1994). Ecological momentary assessment (EMA) in behavorial medicine. *Ann Behav Med, 16*(3), 199–202.

Stuginski-Barbosa, J., Porporatti, A., Costa, Y., Svensson, P., & Conti, P. (2016). Diagnostic validity of the use of a portable single-channel electromyography device for sleep bruxism. *Sleep Breath, 20*(2), 695-702.

Svensson, P., Romaniello, A., Wang, K., Arendt-Nielsen, L., & Sessle, B. (2006). One hour of tongue-task training is associated with plasticity in corticomotor control of the human tongue musculature. *Exp Brain Res, 173*(1), 165-173.

Tartaglia, G., Moreira Rodrigues da Silva, M., Bottini, S., Sforza, C., & Ferrario, V. (2008). Masticatory muscle activity during maximum voluntary clench in different research diagnostic criteria for temporomandibular disorders (RDC/TMD) groups. *Man Ther, 13*(5), 434-440.

Terebesi, S., Giannakopoulos, N., Brustle, F., Hellmann, D., Turp, J., & Schindler, H. (2016). Small vertical changes in jaw relation affect motor unit recruitment in the masseter. *J Oral Rehabil, 43*(4), 259-268.

Thie, N., & Lavigne, G. (2001). Bruxism and orofacial movements during sleep. *Dent Clin North Am, 45*(4), 657.

Thymi, M., Lobbezoo, F., Aarab, G., Ahlberg, J., Baba, K., Carra, M., et al. (2021). Signal acquisition and analysis of ambulatory electromyographic recordings for the assessment of sleep bruxism: A scoping review. *J Oral Rehabil, 48*(7), 846-871.

Toussaint, M., Luthringer, R., Schaltenbrand, N., Carelli, G., Lainey, E., Jacqmin, A., et al. (1995). First-night effect in normal subjects and psychiatric inpatients. *Sleep, 18*(6), 463-469.

Treacy, K. (1999). Awareness/relaxation training and transcutaneous electrical neural stimulation in the treatment of bruxism. *J Oral Rehabil, 26*(4), 280-287.

Türp, J., Schindler, H., Pritsch, M., & Rong, Q. (2002). Antero-posterior activity changes in the superficial masseter muscle after exposure to experimental pain. *Eur J Oral Sci, 110*(2), 83-91.

Valiente Lopez, M., Van Selms, M., Van der Zaag, J., Hamburger, H., & Lobbezoo, F. (2015). Do sleep hygiene measures and progressive muscle relaxation influence sleep bruxism? Report of a randomised controlled trial. *J Oral Rehabil, 42*(4), 259-265.

Van der Meulen, M., Lobbezoo, F., Aartman, I., & Naeije, M. (2006). Self-reported oral parafunctions and pain intensity in temporomandibular disorder patients. *J Orofac Pain, 20*(1), 31-35.

van der Zaag, J., Lobbezoo, F., Wicks, D. J., Visscher, C. M., Hamburger, H. L., & Naeije, M. (2005). Controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. *J Orofac Pain, 19*(2), 151-158.

Van Eijden, T., Koolstra, J., Brugman, P., & Weijs, W. (1988). A feedback method to determine the three-dimensional bite-force capabilities of the human masticatory system. *J Dent Res, 67*(2), 450-454.

Van Eijden, T. (1990). Jaw Muscle Activity in Relation to the Direction and Point of Application of Bite Force. *J Dent Res, 69*(3), 901-905.

Van Eijden, T., Blanksma, N., & Brugman, P. (1993). Amplitude and Timing of EMG Activity in the Human Masseter Muscle during Selected Motor Tasks. *J Dent Res, 72*(3), 599-606.

Victor, L., Valeriu, F., Dumitru, R., Gheorghe, B., & Paula, F. (2017). Coffee consumption influence upon the clinico-neurophysiological manifestations of primary sleep bruxism. *Mold Med J, 60*(3), 17-22.

Wang, L., & Han, Y. (2013). Application of carbon nanotube filled silicone rubber composite in stress measurement during ramped loading with low compression speed. *Sens Actuators A Phys, 201*, 214-221.

Wang, L., & Li, J. (2014). A piezoresistive flounder element based on conductive polymer composite. *Sens Actuators A Phys, 216*, 214-222.

Weisskircher, H. (2013). Maximum bilateral masticatory forces in patients with and without pain Maximale bilaterale Beißkräfte bei Schmerz-und Nichtschmerzpatienten. *Cranio - J Craniomandib Pract, 5*(1), 19-39.

Wetselaar, P., Lobbezoo, F., Koutris, M., Visscher, C., & Naeije, M. (2009). Reliability of an occlusal and nonocclusal tooth wear grading system: clinical use versus dental cast assessment. *Int J Prosthodont, 22*(4), 388-390.

Windhorst, U., Hamm, T., & Stuart, D. (1989). On the function of muscle and reflex partitioning. *Behav Brain Sci, 12*(4), 629-645.

Winocur, E., Gavish, A., Voikovitch, M., Emodi-Perlman, A., & Eli, I. (2003). Drugs and bruxism: a critical review. *J Orofac Pain, 17*(2), 99-111.

Winocur, E., Uziel, N., Lisha, T., Goldsmith, C., & Eli, I. (2011). Self-reported bruxism-associations with perceived stress, motivation for control, dental anxiety and gagging. *J Oral Rehabil, 38*(1), 3-11.

Woda, A., Pionchon, P., & Palla, S. (2001). Regulation of mandibular postures: mechanisms and clinical implications. *Crit rev oral biol med, 12*(2), 166-178.

World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. (2013). *Jama, 310*(20), 2191-2194.

Yap, A., & Chua, A. (2016). Sleep bruxism: Current knowledge and contemporary management. *J Conserv Dent 19*(5), 383.

Yoshida, K. (1998). A polysomnographic study on masticatory and tongue muscle activity during obstructive and central sleep apnea. *J Oral Rehabil, 25*(8), 603-609.

Yurttutan, M., Sancak, K., & Tüzüner, A. (2019). Which treatment is effective for bruxism: Occlusal splints or botulinum toxin? *J Oral Maxillofac Surg, 77*(12), 2431-2438.

Zani, A., Lobbezoo, F., Bracci, A., Ahlberg, J., & Manfredini, D. (2019). Ecological momentary assessment and intervention principles for the study of awake bruxism behaviors, Part 1: general principles and preliminary data on healthy young italian adults. *Front Neurol 10*, 169.

Zucconi, M., & Ferri, R. (2014). Assessment of sleep disorders and diagnostic procedures. *Eur Sleep Res Soc, 23*, 95-110.

Appendices

Table 30: GrindCare® diary (German).

	Gab es <u>Probleme</u> bei der Anwendung von GrindCare®?			**Gab es irgendwelche besonders <u>stressigen</u> Ereignisse (Klausur, Termine, etc.)?**
	Nein	Ja	Welche?	Bitte betreffende Tage ankreuzen
1. Nacht				
2. Nacht				
3. Nacht				
4. Nacht				
5. Nacht				
6. Nacht				
7. Nacht				
Messung				
1. Nacht				
2. Nacht				
3. Nacht				
4. Nacht				
5. Nacht				
6. Nacht				
7. Nacht				
8. Nacht				
9. Nacht				
10. Nacht				
11. Nacht				
12. Nacht				
13. Nacht				
14. Nacht				
Messung				
1. Nacht				
2. Nacht				
3. Nacht				
4. Nacht				
5. Nacht				
6. Nacht				
7. Nacht				
8. Nacht				
9. Nacht				
10. Nacht				
11. Nacht				
12. Nacht				
13. Nacht				
14. Nacht				
Messung				
Geschafft! :)				

Diagnostic Criteria for Temporomandibular Disorders
TMD-Schmerz-Screener

1. Wie lange hielt innerhalb der letzten 30 Tage der Schmerz in Ihrem Kiefer oder Schläfenbereich auf beiden Seiten an?

    a. Kein Schmerz
    b. Schmerz kommt und geht
    c. Schmerz ist immer da

2. Haben Sie innerhalb der letzten 30 Tage Schmerzen oder Steifigkeit im Kiefer beim Aufwachen gespürt?

    a. Nein
    b. Ja

3. Haben innerhalb der letzten 30 Tage folgende Aktivitäten einen Schmerz in ihrem Kiefer oder Schläfenbereich auf beiden Seiten beeinflusst (d.h. gelindert oder verschlimmert)?

    A. Harte oder zähe Nahrung kauen
        a. Nein
        b. Ja

    B. Den Kiefer öffnen oder vorwärts oder seitwärts bewegen.
        a. Nein
        b. Ja

    C. Angewohnheiten des Kiefers wie die Zähne aufeinander halten, pressen, knirschen, oder Kaugummi kauen
        a. Nein
        b. Ja

    D. Andere Aktivitäten des Kiefers wie Sprechen, Küssen oder Gähnen
        a. Nein
        b. Ja

ZAHNKLINIK
PROTHETIK

Figure 46: TMD-Pain Screener (German).

Fragebogen

*Bitte füllen Sie diesen Fragebogen sorgfältig aus. Machen Sie bitte immer nur **ein** Kreuz in eine Zeile und beantworten Sie **alle Punkte**, lassen Sie keinen aus!*

I. Allgemeine Fragen

1	Bitte geben Sie Ihr Geburtsdatum an: (TT/MM/JJJJ)			_____
2	Bitte geben Sie Ihr Geschlecht an:	w		m
3	Sind Sie Links – oder Rechtshänder/in?	rechts		links
		ja	**nein**	Wenn ja: Gesagt hat es mir:
4	Wurde Ihnen gesagt oder haben Sie selbst bemerkt, dass Sie **im Schlaf** mit den Zähnen pressen oder knirschen?			_____ _____
5	Schlafen Sie in einer Position, wo der Unterkiefer durch Druck belastet wird? (z.B. auf dem Bauch oder der Seite schlafen)			
6	Haben Sie gemerkt oder wurde es Ihnen gesagt, dass Sie während stressiger Situationen, Situationen, in denen Sie sich konzentrieren müssen, oder wenn Sie etwas Schweres tragen wollen, die Kiefer aufeinanderpressen oder mit den Zähnen knirschen?			_____ _____
7	Haben Sie schon eine abgeschlossene kieferorthopädische Behandlung hinter sich?			Ich bin zurzeit in kieferorthopädischer Behandlung
8	Haben Sie Ohrgeräusche oder Ohrklingen?			
9	Haben Sie schon einmal gemerkt, dass Sie bevorzugt auf einer Seite kauen?		So etwas ist mir noch nicht aufgefallen	Wenn ja: rechts links beide gleich
10	Nehmen Sie regelmäßig Medikamente ein?			Welche? _____ _____ _____
11	Rauchen Sie?			Wie viele Päckchen pro Tag? _____
12	Trinken Sie viel Kaffee?			Ungefähr wie viele Tassen? _____

ZAHNKLINIK PROTHETIK

Figure 47: General questionnaire (German).

Elektroden-Koordinaten

Nr./Proband(-in)/ Geb.datum:

Masseter:

	Punkt Medial		**Punkt Superior**	
Elektrode	Rechts	Links	Rechts	Links
Dorsal	-	-	-	-
X-Achse				
Y-Achse				
Medial	-	-	-	-
X-Achse				
Y-Achse				
Anterior	-	-	-	-
X-Achse				
Y-Achse				

Temporalis:

	Punkt Inferior		**Punkt Medial**	
	Rechts	Links	Rechts	Links
X-Achse				
Y-Achse				

Figure 48: The coordinate system sheet.

Table 31: Mean values (MV) and standard derivations (SD.) of the RMS % MVC at the first sub-maximal bite force in T1, T2, and T3 for the eight studied muscle regions of the intervention and control group. RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA= left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	**Intervention group: MV (SD)**			**Control group: MV (SD)**		
	T1	**T2**	**T3**	**T1**	**T2**	**T3**
RMASA	0.40 (0.17)	0.43 (0.16)	0.35 (0.15)	0.32 (0.13)	0.38 (0.15)	0.43 (0.21)
RMASM	0.41 (0.17)	0.44 (0.17)	0.41 (0.16)	0.34 (0.13)	0.39 (0.16)	0.39 (0.19)
RMASP	0.41 (0.18)	0.44 (0.16)	0.4 (0.18)	0.33 (0.13)	0.37 (0.15)	0.38 (0.2)
LMASA	0.39 (0.16)	0.43 (0.18)	0.43 (0.23)	0.4 (0.17)	0.4 (0.17)	0.34 (0.10)
LMASM	0.41 (0.16)	0.42 (0.13)	0.40 (0.20)	0.36 (0.11)	0.45 (0.17)	0.37 (0.12)
LMASP	0.4 (0.15)	0.45 (0.17)	0.39 (0.17)	0.34 (0.12)	0.42 (0.19)	0.36 (0.17)
RTA	0.43 (0.16)	0.50 (0.20)	0.45 (0.18)	0.37 (0.14)	0.41 (0.15)	0.39 (0.16)
LTA	0.37 (0.14)	0.47 (0.20)	0.42 (0.20)	0.38 (0.14)	0.42 (0.14)	0.39 (0.17)

Table 32: Mean values (MV) and standard derivations (SD.) of the RMS % MVC at the second submaximal bite force in T1, T2, and T3 for the eight studied muscle regions of the intervention and control group.
RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA=left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	**Intervention group: MV (SD)**			**Control group: MV (SD)**		
	T1	**T2**	**T3**	**T1**	**T2**	**T3**
RMASA	0.39 (0.16)	0.41 (0.17)	0.36 (0.13)	0.33 (0.13)	0.36 (0.16)	0.33 (0.16)
RMASM	0.41 (0.17)	0.41 (0.17)	0.39 (0.17)	0.34 (0.14)	0.36 (0.17)	0.37 (0.20)
RMASP	0.41 (0.17)	0.41 (0.15)	0.38 (0.18)	0.33 (0.15)	0.34 (0.16)	0.33 (0.17)
LMASA	0.48 (0.34)	0.44 (0.18)	0.41 (0.21)	0.37 (0.17)	0.38 (0.16)	0.33 (0.09)
LMASM	0.41 (0.16)	0.40 (0.14)	0.36 (0.16)	0.36 (0.12)	0.39 (0.13)	0.38 (0.16)
LMASP	0.41 (0.16)	0.41 (0.15)	0.38 (0.18)	0.34 (0.14)	0.38 (0.17)	0.33 (0.15)
RTA	0.43 (0.15)	0.45 (0.18)	0.46 (0.21)	0.38 (0.14)	0.38 (0.15)	0.38 (0.15)
LTA	0.38 (0.15)	0.45 (0.20)	0.41 (0.21)	0.37 (0.15)	0.40 (0.16)	0.37 (0.15)

Table 33: Mean values (MV) and standard derivations (SD) of the RMS % MVC at the third submaximal bite force in T1, T2, and T3 for the eight studied muscle regions of the intervention and control group.
RMASA=right masseter anterior, RMASM=right masseter medial, RMASP=right masseter posterior, LMASA= left masseter anterior, LMASM=left masseter medial, LMASP=left masseter posterior, RTA=right temporalis anterior, LTA=left temporalis anterior.

Muscle areas	**Intervention group: MV (SD)**			**Control group: MV (SD)**		
	T1	**T2**	**T3**	**T1**	**T2**	**T3**
RMASA	0.40 (0.19)	0.40 (0.16)	0.42 (0.22)	0.32 (0.14)	0.34 (0.15)	0.34 (0.17)
RMASM	0.41 (0.18)	0.40 (0.17)	0.38 (0.16)	0.33 (0.14)	0.35 (0.17)	0.37 (0.21)
RMASP	0.42 (0.18)	0.40 (0.16)	0.40 (0.20)	0.33 (0.15)	0.31 (0.14)	0.36 (0.22)
LMASA	0.51 (0.38)	0.45 (0.20)	0.39 (0.20)	0.37 (0.17)	0.36 (0.15)	0.34 (0.12)
LMASM	0.42 (0.17)	0.40 (0.15)	0.38 (0.18)	0.35 (0.12)	0.38 (0.14)	0.36 (0.12)
LMASP	0.41 (0.15)	0.40 (0.15)	0.38 (0.16)	0.34 (0.15)	0.36 (0.17)	0.35 (0.18)
RTA	0.44 (0.15)	0.44 (0.18)	0.42 (0.17)	0.38 (0.16)	0.39 (0.15)	0.39 (0.17)
LTA	0.39 (0.16)	0.44 (0.21)	0.40 (0.20)	0.38 (0.15)	0.41 (0.15)	0.38 (0.17)

License:

Table 34: "Cut-off values and grading criteria for defining sleep bruxers" (Thymi et al., 2021).

License number	**5370101489071**
License date	Aug 15, 2022
Licensed content publisher	John Wiley and Sons
Licensed content publication	Journal of Oral Rehabilitation
Licensed content title	Signal acquisition and analysis of ambulatory electromyographic recordings for the assessment of sleep bruxism: A scoping review
Licensed content author	Peter Svensson, Gilles Lavigne, Daniele Manfredini, et al
Licensed content date	May 2, 2021
Licensed content volume	48

I List of abbreviations

AB	Awake bruxism
ANOVA	Analysis of variance
CES	Contingent electrical stimulation
CI LL	Lower limit of the 95% confidence interval
DC/TMD	Diagnostic criteria for temporomandibular disorders
ECG	Electrocardiography
EEG	Electroencephalography
EMA	Ecological momentary assessment
EMG	Electromyography
EOG	Electrooculography
FSR	Force Sensing Resistor
GC	Grindcare®
MM	Masticatory muscle
MMA	Masticatory muscle activity
MPF	Mean power frequency
MUAP	Motor unit action potentials
MVC	Maximum voluntary contraction
OA	Oral appliances
OBC	Oral Behavior Checklist
OR	Odds ratio
PSG	Polysomnography
RDC	Research Diagnostic Criteria
RMMA	Rhythmic masticatory muscle activity
RMS	Root mean square
SB	Sleep bruxism
SBF	Submaximal bite force
STAB	Standardized Tool for the Assessment of Bruxism
TMD	Temporomandibular disorder

II List of Figures

III List of Tables

IV Acknowledgment

First of all, I would like to express my deepest appreciation to my supervisor, PD. Dr. med. dent. N. N. Giannakopoulos for providing guidance, feedback, and motivation throughout this project. Your constant interest in research, knowledge in the field of bruxism, and the resulting infectious enthusiasm in wiring these cables made accomplishing this thesis a great pleasure. Thank you!

Many thanks to Prof. Dr. med. dent. Marc Schmitter, who enabled this clinical study in his department, and provided for the materials and laboratory.

I also wish to thank Sunstar, for providing the GrindCare® devices and the support staff from Sunstar Germany, Marianne Seger, for her fast assistance with all occurring queries.

To Dr. Patrick Trautner who has helped practice the study protocol; Christoph Huber and Vivien Frommer, for the best research colleagues; and Nura, Robin, and Lina, for their enormous support: major thanks!

My special gratitude goes to Prof. Dr. Kathleen Wermke, who arranges the seminar for doctoral students and makes every effort to impart knowledge to the students.

Furthermore, I would like to thank my partner for the inexhaustible assurance and encouragement in this thesis, and life. The best. Thank you for your constant support.

Lastly, this endeavor would not have been possible without my parents, who supported me throughout every stage of my studies, and even more in my thesis. Although I did not have my thesis written with a typewriter like my father did, it was probably as much trial and error. Now as I am filling the last page, I would like to thank you most genuinely for proofreading, your motivation, and suggestions along the way.

V Publications and congress participation

Publications:

Obid N, Frommer V, Huber C, Schindler HJ, Schmitter M, Giannakopoulos NN. The effect of contingent electrical stimulation (CES) on the self-report of bruxism. (Accepted on the 04/07/23)

Frommer V, Obid N, Huber C, Schmitter M, Schindler HJ, Giannakopoulos NN. Correlation of different instruments for bruxism diagnosis. J CranioMandib Func. (2/2023) 101-117

Congress Participations:

Obid N, Frommer V, Huber C, Schmitter M, Giannakopoulos N. (12/02/22). Verändert bedingte elektrische Stimulation die Bruxismus Selbstangabe? 55. Jahrestagung der Deutschen Gesellschaft für Funktionsdiagnostik und -therapie, Bad Homburg; (Poster)

Frommer V, Obid N, Huber C, Schmitter M, Schindler HJ, Giannakopoulos NN. (12/02/22). Wie korrelieren verschiedene Methoden der Bruxismus Diagnostik miteinander? 55. Jahrestagung der Deutschen Gesellschaft für Funktionsdiagnostik und -therapie, Bad Homburg. (Presentation)

Obid N, Frommer V, Huber C, Schindler HJ, Schmitter M, Giannakopoulos NN. (06/24/22). Does contingent electrical stimulation affect the self-report of Bruxism? (presentation). International Association for Dental Research General session, Virtual experience. (Presentation)