Masticatory muscle pain: Causes, consequences, and diagnosis

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Chapter 7

General discussion

Causes of masticatory muscle pain

Masticatory muscle pain is one of the subclassifications of temporomandibular disorders (TMD). The causes of temporomandibular disorder (TMD) pain have been a matter of debate for decades. In the middle 30s of the past century, pain in the pre-auricular area was described as a syndrome consisting of various symptoms like pain within and around the ears, tinnitus, impaired hearing, and a burning sensation in the throat and tongue and at the side of the nose (Costen, 1934). Back then, these symptoms were associated with the loss of occlusal support, thereby putting the etiological focus on the patient's dental occlusion and articulation (Costen, 1934). This etiological concept resulted in the management of TMD patients by means of extensive and irreversible rehabilitation of the patient's dental occlusion. Later on, the role of dental occlusion in the pathogenesis of TMD pain was more and more critically evaluated, while at the same time oral parafunctions were introduced as possible causes for the initiation and perpetuation of TMD pain (Schwartz, 1955). Moreover, Schwartz was the first to mention that pain in the orofacial region could also originate from the masticatory muscles. In the following years, the psychophysiological model was proposed for the etiology of TMD pain, mentioning oral parafunctions as the most important factor initiating TMD pain (Laskin, 1969). In this model, psychological factors were proposed to initiate parafunctions, thereby causing pain.

For disease in general, the importance of psychosocial factors was later on recognized in the so-called biopsychosocial model of disease (Engel, 1977), which is still considered to explain the etiology of TMD pain best (Suvinen et al., 2005). In the biological part of the biopsychosocial model, the role of oral parafunctions is considered pivotal in the initiation and perpetuation of muscle pain in the masticatory system (Greene, 2001). Epidemiologic studies have indicated an association between parafunctions (like bruxism) and TMD signs and symptoms (Johansson et al., 2004; Magnusson et al., 2000). In order to understand the underlying mechanisms, several experimental studies have been performed; for a review see (Svensson et al., 2008). In these studies, the protocols used included series of isometric and concentric contractions of the masticatory muscles in an attempt to mimic the muscles' contractions as they occur during parafunctional activities in humans. The results indicated that although it is possible to induce muscle fatigue and pain in the masticatory muscles, these symptoms disappear shortly after the cessation of the exercises. In the clinic, however, patients report longer lasting symptoms that are present for weeks or even months. In limb muscles, unaccustomed exercises can provoke long lasting pain, known as delayed onset muscle soreness (DOMS) (Cheung et al., 2003). Several attempts have failed to provoke similar symptoms in the masticatory muscles (Svensson and Arendt-Nielsen, 1995). A possible explanation for the fast resolution of the provoked symptoms in masticatory muscles is these muscles' unique composition compared to limb muscles: the highly compartmentalized organization of the muscle fibres

makes them indeed fatigue-resistant (van Eijden and Turkawski, 2001). Therefore, the role of oral parafunctions in the initiation and perpetuation of TMD pain is not yet clear.

In **Chapters 2, 4,** and **5** of this thesis, the role of oral parafunctions in the etiology of persistent TMD pain was investigated. Persistent pain is pain that lasts longer than the expected recovery period of the muscle tissue damage. Two different experimental protocols were used in order to mimic human parafunctional activities, both aiming to investigate the possible provocation of signs and symptoms similar to those reported by TMD-pain patients. In **Chapter 2**, participants were asked to chew at high speed on a big bolus of hard chewing gum for 100 minutes in total. This exercise resembled the habit of gum chewing, albeit with larger bolus size and higher velocity than usual. The results indicated that immediately after the provocation part, most patients, but not all, developed signs and symptoms that led to the diagnosis of myofascial pain according to the Research Diagnostic Criteria for TMD (RDC/TMD) (**Chapter 2**). Even though most of the participants had recovered 24 hours after the provocation part, two of them still received the diagnosis of myofascial pain 1 day after the provocation. Clearly, there was considerable variability in the response of the participants to the experimental protocol. Unfortunately, no further data regarding the psychosocial status of the participants in this study were collected that may have given an explanation for this variability.

In **Chapters 4** and **5**, a newly developed exercise protocol was applied to load the masticatory muscles in a way that better resembles the bruxism activities that are frequently reported by TMD-pain patients. This protocol included an eccentric component in the muscle contractions and was successful in provoking a longer lasting TMD diagnosis of myofascial pain according to the RDC/TMD in 70% of the participants. This diagnosis was present for a longer period of time compared to the protocol described in **Chapter 2**, and resembled more the clinical condition of patients with TMD complaints. Since clinically, eccentric contractions can occur during bruxism activities of the masticatory muscles, it supports the suggestion that such contractions play a role in the initiation of TMD pain.

Still, the transition from pain initiation to pain perpetuation is not yet fully understood. In people practising sports, an imbalance between training and recovery (i.e., overtraining) has been shown to result in long lasting muscle pain (Lehmann et al., 1993). Especially repetitive eccentric muscle contractions are considered responsible for micro-trauma to the muscles (Friden and Lieber, 1998), and consequently for the development of DOMS. When eccentric contractions are repeated daily and with incomplete periods of recovery, this could, together with the influence of other factors included in the biopsychosocial model, contribute to the chronification of pain. In the masticatory system, oral parafunctions that occur on a daily basis may thus yield the typical clinical spectrum of signs and symptoms as reported by chronic TMD patients.

Consequences for motor function

Jaw-muscle pain is known to influence muscle function, causing a disordered jaw motor behaviour (Lobbezoo et al., 2006). The exact interaction between pain and muscle function is, however, not yet fully understood. In the past, several theories have been proposed for the interaction between pain and motor function. The first one is known as the "vicious-cycle" or "pain-spasm-pain cycle" theory (Travell et al., 1942). According to this theory, pain is provoked by muscle spasm and afterwards, pain reflexly produces more muscle spasm which leads to a self-perpetuating cycle. Apart from explaining the consequences of pain on muscle function, this theory also tries to explain the etiology of pain. However, experimental studies could not confirm several aspects of this theory, for the example the presence of spasm in chronic pain patients. Therefore, it is questioned whether the "pain-spasm-pain cycle" theory really represents the mechanism present in patients suffering from pain (Roland, 1986). According to the "vicious cycle" theory, muscle spasm is considered the result of muscle overload. Afterwards, in a cascade of spasm-pain-spasm events, pain may perpetuate and eventually become chronic (Travell and Simons, 1983).

In the early 90's, the "pain adaptation" model, was proposed (Lund et al., 1991). In this model, pain causes an increased activity of the antagonistic muscles and simultaneously a decreased activity of the agonistic muscles. The "pain-adaptation" model predicts that in the presence of pain, the mobility of the painful body part is decreased in order to avoid further damage and to allow time for recovery. This can be considered the immediate benefit for the pain patient. However, the fact that the non-affected muscles adjust their activity in order to compensate for the disturbed activity of the affected muscles, can have negative long-term consequences for the health of the individual. This can create secondary pain in the muscles that are not affected at the first instance (Hodges and Tucker, 2011). The "pain-adaptation" model seemed more plausible than the "vicious-cycle" theory, because it only deals with the effects of pain on the motor function of the musculoskeletal system. However, subsequent experimental studies could not confirm the pain adaptation model in full (Hodges and Tucker, 2011) . Therefore, new theories appeared (Hodges and Tucker, 2011; Murray and Peck, 2007), which appreciate the fact that changes in muscle function due to pain are individualized. Depending on the different components of pain (i.e., sensory-discriminative, motivational-affective, and cognitive-evaluative) that are expressed in certain conditions, the modulation of the motor response can be different. These changes aim to minimize pain and maintain homeostasis at the same time (Murray and Peck, 2007)..

Regarding the influence of pain on motor function, the results of this thesis contributed to increasing our insight regarding this topic. The decreased maximum voluntary bite force (MVBF) in the presence of jaw-muscle pain (**Chapter 4**) corroborates the results of previous experimental studies which showed that when pain is present, the MVBF decreases (Wang et al., 2000a). This finding is in line with the "pain-adaptation"

model, whereby the decreased activity of the agonistic muscles is thought to prevent further damage to the masticatory system (Lobbezoo et al., 2006). However, in contrast to other experimental provocation protocols, no changes in the jaw-stretch reflex amplitude were found (**Chapter 2**). The only study corroborating these findings is an experimental protocol that was also based on a brief chewing exercise (van Selms et al., 2005). This contradicts, though, the results of several experimental studies that provoked jaw-muscle pain with injections of painful substances in the masticatory muscles and found a facilitation of the stretch reflex amplitude when pain was present (Cairns et al., 2003; Svensson et al., 2003; Wang et al., 2000b; Wang et al., 2002; Wang et al., 2004). This reflex facilitation was thought to protect the painful muscles according to the "pain adaptation" model. One reason explaining these contradictory results could be the lower intensity of pain provoked with the chewing exercises (**Chapter 2**) as compared with that provoked with injected substance-induced jaw-muscle pain. A more likely explanation, however, could be the fact that the evoked fatigue and pain after chewing exercises occurs more naturally, allowing the masticatory system to better adapt to the gradually increasing pain. Therefore, the stretch-reflex amplitude will probably not change under clinical pain conditions.

Another contribution of the present thesis to the body of knowledge involves the methodology used to analyse the amplitude of the jaw-stretch reflex. In **Chapter 3**, it was proven that the method of normalizing the reflex amplitude with the averaged rectified EMG activity of the 100-ms pre-stimulus period makes the reflex amplitude independent of the location of the electrodes, provided that they are placed along the muscle fibers. This finding is important, because it indicates that if the jaw-stretch reflex amplitude is acquired and analysed according to the technique described in **Chapter 3**, it is a reliable measure, because it is not influenced by the position of the electrodes over the muscle belly. In the studies that dealt with the effects of substance-induced pain on the jaw-stretch reflex, the reflex amplitude was also determined as described in this chapter. (Cairns et al., 2003; Svensson et al., 2003; Wang et al., 2000b; Wang et al., 2002; Wang et al., 2004). Therefore, the reasons for the contradictory results regarding the modulation of the stretch-reflex amplitude after pain provocation should be searched in physiological mechanisms, and not in the methodology of the reflex recording itself.

Consequences for clinical practise

An important part of a TMD patient's evaluation is establishing the correct diagnosis. Currently, several classification systems for the diagnosis of TMD pain exist (Dworkin and LeResche, 1992; Merksey and Bogduk, 1994; Spitzer et al., 1987; Spitzer and Skovron, 1995). The most widely used one is the Research Diagnostic Criteria for TMD (RDC/TMD) (Dworkin and LeResche, 1992). In the RDC/TMD, the diagnosis mostly depends on the reaction to palpation of the masticatory muscles and temporomandibular joints. The role of palpation in the diagnosis of other musculoskeletal disorders and

conditions like fibromyalgia, though, has been criticised (Wolfe, 2003). Two recent, large-scale multicenter studies (Truelove et al., 2010; Visscher et al., 2009) concluded that the validity of the RDC/TMD is not acceptable. Indeed, the results reported in **Chapter 6** indicate that palpation tests are more related to comorbid factors like regional pain and somatization than to the local TMD-pain complaint. As an alternative to palpation, the use of the dynamic/static tests has been proposed (Naeije and Hansson, 1986). Moreover, it was recently proposed that the use of the "familiar pain" concept could further improve the validity of diagnostic tests (i.e., a diagnostic approach that takes into account the fact whether or not patients recognize the provoked pain as their pain complaint). In **Chapter 6**, a study to the diagnostic ability of palpation and of dynamic/static tests is described, incorporating the influence of comorbid factors, like depression and other pain complaints, on the outcome of the clinical tests. The study demonstrated that only the dynamic/static tests, with "familiar pain" as outcome measure, were uniquely related to the presence of local TMD-pain complaints and not influenced by comorbid factors. The outcome of palpation tests was mostly related to comorbid factors; not to the local TMD complaints of the patients. Hence, in patients with a chronic orofacial pain complaint, in whom comorbid factors like widespread pain or depression are regularly present, a positive reaction to palpation does not necessarily indicate the actual presence of a TMD pain. Since the physical examination of the RDC/TMD mainly relies on palpation tests of the masticatory system, this finding has important implications for the interpretation of current studies in the field of TMD. For example, in studies to patients with chronic orofacial pain complaints, the presence of TMD-pain is likely to be overestimated (false positive RDC/TMD diagnoses). When such patients are included in studies to treatment outcome, treatment success may be underestimated. Therefore, future diagnostic systems for musculoskeletal disorders, among which TMD, should incorporate dynamic/static tests, with "familiar pain" as outcome measure.

Concluding remarks

The studies included in this thesis showed that masticatory muscles' overloading can initiate signs and symptoms of TMD pain in healthy individuals (**Chapters 2, 4, and 5**). The provoked pain influenced both sensory and motor functions of the masticatory system, causing a decrease in pain pressure thresholds (**Chapter 2, 5, and 6**) and variable effects on motor control: while the MVBF decreased when pain was present (**Chapter 5**), the reflex amplitude was not affected (**Chapter 2**).

For the recording of the jaw-stretch reflex, a technique based on the normalization of the raw data was used. This technique was proven to provide an objective measure of the reflex amplitude that was not dependent on the location of the electrodes for the reflex recording (**Chapter 3**).

Regarding pain, it is known that it is a multidimensional experience, affecting both the somatic and psychosocial aspects of an individual's life. In order to diagnose TMD pain, assessment of the somatic and psychosocial condition of the patient (Axis I and Axis II, respectively) is therefore mandatory. The findings from the present thesis (**Chapter 6**) indicate that diagnostic tests for TMD are negatively influenced by a patient's comorbidity. This influence decreases when the presence of familiar pain is used as outcome measure of the clinical tests. The only tests that were not influenced by the presence of comorbidity were the dynamic/static tests with the use of familiar pain as their outcome.

The results of the present thesis have contributed to further unravel the factors influencing the diagnosis of musculoskeletal pain. Moreover, it increased the understanding of the causes of musculoskeletal pain and of its consequences on jaw-motor function. It remains for future research to further elucidate the factors influencing the progression of pain from acute to chronic. If these mechanisms will be defined, this could lead to the development of an etiological management of TMD pain.

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