REVIEW

Masticatory muscle pain and disordered jaw motor behaviour: Literature review over the past decade

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Summary The clinically important relationship between masticatory muscle pain (MMP) and disordered jaw motor behaviour is subject of this concise, evidence-based review of the literature that was published during the past 10 years. Mainly based on studies that used some sort of experimental MMP (e.g., the intramuscular injection of noxious substances like hypertonic saline), it was concluded that MMP has pronounced effects on jaw motor functions like maximal clenching and mastication. The pain-related modulation of oral reflexes further illustrated the effects of MMP on masticatory motor control. Protecting the painful muscle tissues against further damage and allowing for time to heal the damaged tissues by immobilization of the masticatory system seem to be the key outcomes of these effects. Further, MMP was shown to influence the cervical motor system, which may partly explain the mechanism behind the frequently observed co-occurrence of pain in the neck and the jaw. Finally, it was concluded that, even though the evidence is not yet conclusive, also remote pain (non-MMP) can modulate jaw motor behaviour, which indicates the involvement of central mechanisms in this modulation.

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Introduction

Many patients with musculoskeletal pain in the orofacial region present themselves to their dentist not only with a pain complaint but also with complaints of disordered jaw function, like a limitation in their mandibular movements. For the dentist, it is important to understand the nature of the relationship between these two types of clinical problems. Among others, a proper understanding will yield a more accurate clinical diagnosis for both types of problems and will improve the dentist’s insight into the aetiology. In turn, this will enable better treatment choices.

Most pain complaints in the orofacial region are due to dental causes (e.g., pulpitis, periodontal pain) and trauma. These pains usually have an acute nature at the time when patients present themselves to their dentist, and are relatively easy to diagnose and to treat. Of the chronic pain conditions in the orofacial region, temporomandibular pains constitute the most common diagnostic group. Among this group of musculoskeletal pain conditions, the most frequently occurring diagnosis is masticatory muscle pain (MMP).

During the past decades, the relationship between MMP and disordered jaw motor behaviour has been studied and reviewed extensively; see, for example, the reviews by Lund et al., Lund, Stohler, and Svensson and Graven-Nielsen. Unfortunately, previous reviews only seldom give the literature search strategy that was used. In a time where evidence-based dentistry is the norm, the selection of eligible papers should be controllable for the reader. The purpose of the present paper therefore was to provide a concise, evidence-based review of studies to the functional consequences of MMP that were published during the past 10 years, i.e., in the decade after the appearance of the landmark publications by Lund et al. and Lund on the motor function of patients suffering from temporomandibular pain.

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Clinical and experimental masticatory muscle pain

Classically, clinical MMP (i.e., myogenous temporomandibular pain) is divided into two subgroups
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based on the duration of the painful condition: (sub)acute MMP (<6 months) and chronic (persisting) MMP (>6 months). Like other musculoskeletal pain problems, chronic MMP is clinically characterized by its usually mild intensity, its provocation or exacerbation by voluntary muscle contraction, and its fluctuating nature. This fluctuating nature of chronic MMP, however, renders several studies to the relationship between MMP and disordered jaw motor behaviour difficult to interpret: the pain may sometimes have been absent during the assessment of the motor behaviour of interest. To ascertain that sensations of MMP are actually present at the time of the assessment of its functional consequences, many researchers use techniques to experimentally elicit MMP in healthy subjects.

MMP can be elicited experimentally by excessive jaw functions like intense and prolonged clenching and chewing. This will lead to haemodynamic changes, viz., a reduced blood flow in the masticatory muscles. In many instances, the resulting localized hypoxia causes immediate, ischaemic muscle pain. Accumulation of metabolites is, at least partly, responsible for this muscle pain. As an example, gum chewing was shown to induce ischaemic muscle pain in male and female MMP patients as well as in healthy women, the thus provoked MMP persisting longer in females than in males. In another study, prolonged gum chewing was shown to aggravate subsequent ischaemic MMP elicited through maximal voluntary clenching (MVC). Unfortunately, ischaemic MMP generally has a fast-transient character, lasting only for a few minutes. This makes it difficult to apply this provocation technique in studies to the functional consequences of MMP.

Another way to elicit experimental MMP may be the provocation of delayed onset muscle soreness (DOMS). It is known from limb muscles that repeated episodes of heavy skeletal muscle contractions may elicit DOMS, especially when eccentric contractions (i.e., stretching of contracting muscles) are part of the exercise. DOMS is characterized by pain that appears approximately 24 h after the exercise and lasts for several days. The words that are commonly used to qualify DOMS (e.g., 'tense', 'taut') resemble those, used to qualify chronic MMP. These characteristics make DOMS very suitable for studies to the functional consequences of MMP. However, there is a lack of studies in which DOMS was successfully elicited in human masticatory muscles. This may be due to difficulties in performing eccentric contractions of the jaw-closing muscles. Likewise, repeated submaximal clenching at 25% MVC failed to induce a progressive increase in MMP during a 5-day study period. Apparently, other pain provocation techniques are needed to further explore this subject.

Hence, to be certain of pain being present in the masticatory muscles when assessing its functional consequences, many researchers nowadays use chemical provocation techniques, like the intramuscular injection (bolus or continuous infusion) of hypertonic saline, glutamate, or capsaicin. The resulting pain is present for a sufficiently long duration to perform the experiments that are needed to test its functional consequences. However, one should keep in mind that chemically induced, acute MMP cannot readily be compared with clinical, usually chronic MMP, even though the subjective descriptions of chemically induced MMP resemble those of clinical MMP.

Masticatory muscle pain and disordered jaw motor behaviour

Below, the following jaw motor functions will be dealt with in relation to MMP: bruxism, postural EMG activity, maximal clenching, and mastication. In addition, the effects of MMP on masticatory motor control will be assessed by reviewing relevant oral reflex studies. Both subjects will be preceded by a brief description of some MMP-related changes in vegetative functions like respiration and blood circulation.

Vegetative functions

There is a scarcity of studies to the vegetative consequences of MMP. Experimental MMP (viz., a bolus infusion of hypertonic saline) caused elevated values of many standard respiration variables (e.g., respiration rate, minute ventilation volume, and inspiratory and expiratory flow rates) during the first 5 min following the infusion, while sustained MMP, assessed between 5 and 10 min after the infusion, still yielded an elevated respiratory rate. In addition, the autonomic regulation of intramuscular blood flow yielded a lower re-perfusion during the recovery phase after submaximal clenching in chronic MMP patients than in healthy control subjects.

Bruxism

The possibility of a causal relationship between MMP and bruxing habits during sleep has been the subject of a previous review. It was concluded that there is insufficient evidence to confirm or refute the existence of such relationship. Recent studies now suggest that bruxing habits during sleep do not cause MMP. Rather, the presence of clinical or experimen-
tal MMP seems to be associated with less masticatory muscle activity during sleep than the absence of MMP. However, these findings are not conclusive: the relationship between MMP and sleep bruxism needs further attention. In addition, more research is needed to clarify the purported causal relationship between MMP and daytime clenching.

Postural activity

Classically, the onset and maintenance of MMP was understood in terms of the so-called vicious cycle pain model: increased postural activity would cause MMP, which in turn yielded a further increase in postural activity, etcetera, thus setting up a vicious cycle. Experimental pain, induced by hypertonic saline or mustard oil injections in rat masseter muscles as well as by hypertonic saline injections in human jaw-closing muscles, indeed caused an increase in postural activity. However, the induced hyperactivity did not last longer than the pain response of the infusion itself, viz., maximally 10 min. Likewise, a study in MMP patients and healthy control subjects provided only little support for the use of postural EMG data to accurately distinguish MMP patients from controls, even though for some muscle sites higher postural activities were found in the MMP patient group. Chandu et al. and Bodéré et al. confirmed this latter finding. In short, the above-cited studies only partly support the classical point of view that the onset and maintenance of clinical MMP may result from a vicious cycle mechanism.

Maximal clenching

Experimental MMP, induced by hypertonic saline, has been shown to cause a reduction in maximal voluntary clenching (MVC) capacity, as measured both in the EMG output and in the force output. This finding is in line with the so-called pain-adaptation model, as formulated by Lund et al. and Lund. In short, this model states that in the presence of pain (e.g., MMP), agonist muscle activity is decreased (e.g., jaw-closing muscle activity during clenching) while antagonist muscle activity is increased (e.g., jaw-closing muscle activity during mouth opening). These immobilizing effects may have the consequences of preventing further damage of the painful muscle tissues and of promoting healing and restitution. This model can explain not only the pain-related behaviour of entire muscles or muscle groups but also that of single motor units: with a constant force output of the masseter muscle with experimentally induced pain (capsaicin injection), a decreased firing rate of single motor units was found. The constant force was probably maintained by a significant increase in the twitch force amplitude that was observed during capsaicin-induced pain; the twitch force amplitude being a measure of the mechanical properties of the single motor units.

Mastication

In line with the pain-adaptation model, experimental MMP, caused by hypertonic saline injections in both masseter muscles, yielded an increased EMG activity of the jaw-closing muscles in the antagonist phases of mastication, and a reduced EMG activity of these muscles during the agonist phases. Interestingly, using fine-wire electrodes, the reduction in jaw-closing muscle activity during the agonist phase was less pronounced when measurements were taken at a larger distance from the location of the hypertonic saline infusion. The functional consequence of these findings may be a reduction in the mobility of the jaw during mastication. Surprisingly, no significant changes in jaw movement characteristics (i.e., the kinematic parameters displacement, velocity, and duration) due to hypertonic saline-induced experimental MMP could be observed, although it was shown that breaking of hard food with painful muscles of MMP patients took longer than with pain-free muscles of healthy controls.

Reflexes

Oral reflexes play an important role in masticatory motor control. Since oral reflexes are well-suited for controlled experimental studies, the modulation of these reflexes by MMP has been studied extensively (for reviews, see Göbel and Dworschak and Svensson). Most reflex studies dealt with the early and late exteroceptive suppression periods (‘ES1’ and ‘ES2’) in isometrically and submaximally contracting masseter and temporalis muscles. The exteroceptive suppression periods can be evoked by external stimuli, like the electrical or mechanical stimulation of peri-oral and intra-oral tissues. Also, the proprioceptive jaw-stretch (‘jaw-jerk’) reflex was studied frequently. Jaw-stretch reflexes can be evoked from the muscle spindles within the jaw closers by mechanical displacement of the mandible.

Experimental MMP, caused by injections of hypertonic saline into the jaw-closing muscles, reduced the size of the second (late) inhibitory period of the exteroceptive inhibitory reflex in these muscles that was peri-orally evoked by non-painful electrical stimulation of the skin over the mental nerve. The amplitude of the short-latency excitatory part
of the jaw-stretch reflex did not differ significantly between MMP patients and healthy controls.\textsuperscript{44} On the other hand, continuous hypertonic saline infusion caused a significant increase in both the early (onset after 9—10 ms post-stimulus) and the late (duration from 25 to 40 ms post-stimulus) excitatory jaw-stretch reflex responses.\textsuperscript{45,46} Taken this evidence together, it can be concluded that during isometric, submaximal contractions of the jaw-closing muscles, experimental MMP may cause a reduced inhibition of the jaw-closing muscles’ inhibitory reflex and an increased excitation of the jaw-stretch reflex in these muscles. Both effects contribute to an increased stiffness of the masticatory muscles while in pain, which results in a reduced mobility of the jaw. This fits, at least in part, the pain-adaptation model,\textsuperscript{9,10} because jaw opening is thus being hampered in the presence of jaw-closing MMP.

**Remote pain and disordered jaw motor behaviour**

From the above paragraph, it can be gathered that MMP influences jaw motor behaviour. The question that remains is whether or not it is a prerequisite that the pain, that modulates this behaviour, originates from the jaw muscles themselves, or whether it is merely the presence of any pain (including remote pain; non-MMP) that may cause this modulation.

Many studies, but not all, show modulatory effects of remote pain on masticatory motor control. Topically applied capsaicin to the skin over the masseter muscle did not influence the masseter inhibitory reflex evoked by non-painful electrical tooth pulp stimulation.\textsuperscript{47} Further, the size of the second inhibitory period, as evoked by peri-oral electrical stimulation, was found to be normal in patients with chronic tension-type headache.\textsuperscript{48,49} Also Wang et al.\textsuperscript{43} did not observe any significant changes in the characteristics of the exteroceptive inhibitory reflex in the jaw-closing muscles during painful infusion of hypertonic saline into the anterior tibialis muscle. On the other hand, however, carefully controlled studies showed that remote experimental pain in the arm or hand, as caused by an inflated pneumatic cuff or an immersion in cold water, respectively, does cause a significant reduction of the size of the second inhibitory period of the exteroceptive inhibitory reflex in the jaw-closing muscles, as evoked by non-painful electrical stimulation of the upper lip,\textsuperscript{50} by non-painful mechanical taps on a central incisor,\textsuperscript{51,52} or by non-painful ‘ramp and hold’ (push) stimuli against a central incisor.\textsuperscript{53} These latter studies reinforce the possibility of modulatory effects of remote pain on masticatory motor control.

Some interesting studies illustrate the functional connections between the muscles of the jaw and those of the neck/shoulder region. For example, the second inhibitory period was reduced in the electrically evoked exteroceptive inhibitory reflex in both the temporalis muscle and the trapezius muscle by hypertonic saline-induced experimental pain in one of these muscles, reflecting a pain-mediated functional connection between the trigeminal and upper cervical neural systems.\textsuperscript{54} Likewise, the amplitude of the short-latency excitatory response of the proprioceptive jaw-stretch reflex increased not only after painful glutamate injections in the masseter muscle, but also after such injections in the splenius muscle.\textsuperscript{55} Functionally, neck injury patients (e.g., whiplash-associated disorder) showed derangements in the control of mandibular opening-closing movements.\textsuperscript{56}

Conversely, MMP can also influence the motor behaviour of the cervical system. Painful bradykinin injections in the masseter muscle of the cat caused an increased static fusimotor drive to the muscle spindle system in the dorsal neck muscles.\textsuperscript{57} Besides demonstrating the existence of a reflex connection between the trigeminal and cervical systems, this observation may, at least in part, explain the mechanism behind the frequently observed co-occurrence of pain in the neck and the jaw. Finally, in MMP patients, increased postural activities were observed in the sternocleidomastoid and trapezius muscles,\textsuperscript{58} which also suggests that pain in the jaw muscles may have functional consequences for the muscles in the neck/shoulder region.

Taken all evidence together, it can be hypothesized that, even though the evidence is not conclusive, also non-MMP (remote pain) can modulate jaw motor behaviour. The fact that remote pain can influence jaw motor behaviour suggests that central mechanisms may be involved in this modulation.

**Discussion**

Both MMP and non-MMP have effects on jaw motor behaviour as well as on vegetative functions like respiration and blood circulation. As far as the effects of pain on jaw motor behaviour are concerned, protecting the painful muscle tissues against further damage and allowing for time to heal the damaged tissues by immobilization of the masticatory system seem to be the key outcomes of these effects. Most findings thereby corroborate, at least in part, the pain-adaptation model, as formulated by Lund et al.\textsuperscript{9}
and Lund. The clinical consequence of these pain-dysfunction relationships is that when treating patients with both MMP and a disordered jaw function, like a muscle-determined limited maximum mouth opening, the treatment of MMP may also yield an improved jaw function. In everyday clinical practice, this is a frequent observation indeed.

It should be noted that in MMP patients, the above reasoning is especially feasible in the presence of acute muscle pain conditions, where injury-inflicted tissue damage constitutes the actual disease state. However, for chronic muscle pain conditions, where the pain itself rather than the tissue damage constitutes the disease, this explanation is less applicable. Indeed, many of the studies on chronic MMP patients that were included in the present review yielded different findings than the studies to the same jaw motor function that used experimental MMP, like the absence of a change in the amplitude of the short-latency excitatory part of the jaw-stretch reflex in the study by Kitagawa et al. More research is needed to fully explain the observed motor behaviour in chronically painful muscles.

Another issue that should be pointed out is recent evidence suggesting that the classical method of quantifying reflexes (i.e., counting the number of neuron discharges following stimulation) may generate significant errors. Instead, Türker and Powers recommend that the rate of discharge of a neuron be used to accurately display the neuron’s excitability at the time of discharge. Future studies to the effects of MMP on masticatory motor control should consider this recommendation as to avoid erroneous outcomes that cannot be interpreted.

Most evidence for the relationship between MMP and disordered jaw motor behaviour comes from studies in which the pain was elicited chemically in otherwise healthy individuals. The main advantage of this approach is the certainty that MMP is actually present when the physiopathological measurements are being taken. Further, using initially pain-free subjects yields study samples in which psychological disorders are less likely to be present than in chronic pain patients. The earlier-mentioned main disadvantage (viz., that chemically induced, acute MMP cannot readily be compared with clinical, usually chronic myogenous temporomandibular pain), however, should always be kept in mind when interpreting such experimental studies.

References

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