Are Jaw and Facial Reflexes Modulated During Clinical or Experimental Orofacial Pain?

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A variety of jaw and facial reflexes can be evoked by orofacial mechanical or electrical stimuli. Because of its possible diagnostic utility in the management of pain and dysfunction of the masticatory system, the exteroceptive suppression that can be evoked in the masseter and temporalis muscles has been particularly investigated. A review of the different studies emphasizes the crucial importance of the area stimulated and the type of stimulation used to evoke the reflex. More recent studies have applied the necessary standardization of stimulus intensity, clenching levels, recording procedures, and unbiased interpretation of the reflex components in muscle electromyographic (EMG) activity. Controversial results have been reported regarding the differences in these inhibitory (and excitatory) reflex responses between temporomandibular disorder or headache patients and controls. Even if the absence of a second inhibitory phase in the masseteric EMG activity of the patients is a frequent finding, its sensitivity and specificity as a diagnostic tool for myogenous pain or bruxism remain to be tested. Controlled studies on the duration of the second exteroceptive suppression period in tension-type headache patients could not confirm the initially reported difference between patients and asymptomatic subjects. Studies that involve experimentally induced muscle pain could provide better insight into the characteristics of the afferent fibers and synaptic circuitry that are involved in the jaw and facial reflexes.
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The exteroceptive suppression of electromyographic (EMG) activity in the jaw-closing and facial muscles, as well as the monosynaptic jaw-jerk reflex, have been extensively studied as a possible quantitative or qualitative tool for the diagnosis of pain syndromes of the masticatory system. The first part of this review will cover the problems that occur with standardization of stimulus parameters and with recording and interpreting the results obtained from patients with muscle or joint pain or temporomandibular dysfunction. It will also illustrate the pitfalls that occur in this kind of neurophysiologic examination with regard to clinical diagnosis, inclusion/exclusion criteria for the group under study, and standardization of the experimental procedure.

As a consequence of these drawbacks, several groups decided to study the trigeminal and facial reflexes in a laboratory environment first, under strictly controlled conditions, on the presumption that experimental pain might give a better insight as to how to interpret observations in clinical pain patients. The progress that they have made in the study of jaw and facial reflexes in relation to experimental pain will be discussed in the second part of this review. Finally, the most advanced information on the possibilities and limitations of reflex studies in clinical practice will be discussed, and some questions and suggestions for further research will be formulated.

Clinical Diagnosis of TMDs and the Use of Trigeminal Reflexes

Temporomandibular disorders (TMD) is a collective term that embraces a number of clinical conditions with similar symptoms of pain, limitation, and/or interference during jaw movement. Part of the conflicting results reported here might be related to the long-standing confusion with regard to clear definitions and classification. More recently, different subgroups have been defined according to validated criteria (Research Diagnostic Criteria[1]). A classification has been proposed for subdividing TMDs and related conditions into three groups: *(1)* myofascial pain with or without limitation of movement; *(2)* anterior disc displacement with or without reduction during mandibular movement; and *(3)* arthralgia, arthritis, and arthrosis. In future studies, the application of these criteria to patient groups under study will certainly enhance the clear interpretation of the results obtained in reflex studies.

Jaw-Jerk Reflex

Downward tapping on the chin while subjects maintain voluntary activity of the jaw-closing muscles results in a myotatic reflex (the jaw jerk), with reflex contraction of the jaw-closers; no response is seen in the jaw-opening muscles. This monosynaptic reflex was first described by Hoffman,[2] and it has been extensively reviewed.[3–6] The latency of this excitatory response has been found to be between 6.1 and 11 ms (for review see De Laat[7]). This range may be a result of interindividual variability, but can probably also be explained in part by the evaluation technique used to determine the onset latency.[8] The intraindividual difference in latency between left and right sides, by contrast, is assumed to be extremely small (0 to 1 ms), and the unilateral absence of the jaw-jerk reflex or a side difference in latency larger than 1 ms generally implies a neurologic disease (for review see Kimura et al[9]). The amplitude of the reflex is highly variable, not significantly different in patients with TMDs, and depends on the force and direction of the chin tap, the age of the subject,[10] and the level of EMG background activity.[11] In 14 patients with temporomandibular dysfunction, the overall amplitude of the jaw-jerk reflex after chin tap was comparable to the values obtained in normal subjects; the latency was significantly longer and the amplitude was significantly smaller when the affected side was compared with the asymptomatic side in the same subject.[12] It was remarkable that in six of the patients, the latency asymmetry exceeded 1 ms. The authors concluded that if such a side difference is found in TMD patients, a neurologic examination should be performed; a similar finding in a neurologic clinic would not automatically imply neurologic disease and would warrant stomatognathic examination. A more recent study[13] confirmed the finding that shorter latencies and smaller amplitudes were present on the painful side.

Using a bite fork that pulled the mandible downward, Büchner et al[14] studied the jaw-jerk reflex in symptom-free subjects and in patients with myogenous temporomandibular dysfunction before and after treatment. Significant asymmetry was found in many of the patients when the amplitude of the reflex was corrected for the actual background EMG activity. This difference decreased after successful treatment. Comparison between groups, however, could not show significant differences; this was confirmed in a subsequent study of the same group.[15] When patients who suffered from unilateral jaw muscle pain were compared with a group that had bilateral pain, Cruccu et al[16] reported a smaller jaw-jerk reflex on the painful side, and even the absence of the reflex in 6 out of 25 patients. The apparent variability, however, increases the risk for false-positive diagnoses,[17] and consequently the authors concluded that the finding had an uncertain clinical value.

Silent Period After Chin Tap

The jaw-jerk reflex in the masseter and temporalis muscles is followed by a period of decreased activity (the so-called silent period [SP][2,4,6,18]). Similar to the jaw-jerk reflex itself, the large interindividual and intraindividual variability of the latency and duration of the response has been attributed to various factors: *(1)* the operational definition of the SP, which varied between authors; *(2)* intraindividual variability[19,20]; *(3)* standardization of the methods of stimulation[21,22]; and *(4)* the interpretation of the reflex parameters, including experimenter bias.[23] Higher voluntary clenching levels resulted in shorter SPs,[22,24] and an increase in the vertical dimension and variation of the location of the EMG electrode appeared to change the reflex.[21]

The interpretation of the neurophysiologic background of the SP is complicated and has been extensively reviewed.[4,6,25] It was originally suggested to result from a crossed autogenic inhibition that arises in the Golgi tendon organs[26]; it was later explained as a pause in jaw-closing muscle spindle activity.[27] The stimulus, however, may activate a diverse range of receptors, including cutaneous, bone, inner ear, and apparently also periodontal receptors in the periodontal ligament and bone since the SP after chin tap shortens after quadrant-local anesthesia.[28] Intraoral stimuli also result in similar reflexes, which further complicates interpretation.

Several studies have evaluated the SP after the jaw-jerk reflex in patients who suffered from pain and dysfunction of the masticatory system, and a prolonged SP[29–32] and shortening to "normal ranges" after successful (occlusal) therapy[29,33–35] have been claimed. However, more recent reports did not find any difference between patients and controls.[36,37] Furthermore, a 3-year longitudinal study[38] found no significant difference in SP duration among patients who had achieved complete, partial, or no relief from symptoms. These controversial results might be attributed at least in part to the methodologic difficulties mentioned earlier.[19,23] In particular, the differences with regard to SP duration between patients and controls could be linked to the clenching levels used; higher clenching levels result in a decreased duration of the SP, and since TMD patients cannot produce the same amount of bite force/clenching, the duration of their SP is prolonged.[37,39] The latency of the SP that occurs after chin tap is increased when older subjects are compared with younger subjects,[40] and most patient studies have not controlled for age. Variations in the critical interpretation of the latency of the silent period might also affect its suggested use in the study of peripheral neuropathy.[41,42]

Mechanical Tooth Stimulation

If a standardized tap is delivered to a maxillary incisor while subjects maintain a constant clenching level, a sequence of inhibitory[4,43,44] and excitatory reflexes[45] occurs in the masseteric EMG. These reflex changes have been analyzed in a series of averaged and rectified sweeps by means of a computer program[46] that automatically calculates the latencies and durations of the different components of the poststimulus EMG complex (PSEC). The PSEC appears to be mediated through periodontal[47] and inner ear[48] receptors. Shorter latencies and durations, and decreased amplitudes result from increasing the background activity.[49] Reproducible early and late inhibitions (with latencies of around 14 and 45 ms, respectively) that alternate and are sometimes followed by periods of increased EMG activity could be evoked at low clenching levels and by light, nonpainful taps. The excitatory components have been shown to be of a reflex nature and not to reflect rebound effects.[48]

Over time, the morphology of the PSEC appears to be very reproducible in normal subjects. In a study that compared a group of symptom-free subjects to a group of bruxers and patients with temporomandibular dysfunction, the second inhibition always appeared to be absent in bruxers and was significantly smaller or absent in patients with myogenous pain.[50] In contrast to the SP after chin tap, this effect cannot be attributed to lower background activity in the patients because the smaller and shorter reflex changes were associated with higher clenching levels (see above). There has been some indication that after successful treatment the amplitude of the second inhibition tends to normalize again.[51] In addition, the second inhibition may be absent during a fatigue-inducing clench, but present both before and immediately after the clench.[52] The sensitivity and specificity of the presence/absence of the second inhibition still needs to be assessed in a controlled study; in view of the difficulties mentioned earlier,[17] the PSEC has not been advocated as a diagnostic tool in clinical situations.

Air-Jet Stimulation of Perioral and Intraoral Sites

Only one study has used a puff of air as a stimulus, and the experiments were performed in symptom-free subjects.[53] After stimulation of perioral sites, two distinct periods of inhibition (with latencies of 16 and 60 ms, respectively) were observed in the masseter and temporalis muscles. Intraoral application of the air jet (hard palate or tongue) evoked a single period of suppression with a latency of 50 ms. The threshold for suppression was higher for cutaneous stimuli than for mucosal stimuli.

Intraoral and Extraoral Electrical Stimulation

In many studies of exteroceptive suppression (ES), intraoral or extraoral electrical stimulation has been applied. Conflicting results were initially reported regarding the presence of an early inhibition, an early and late inhibition, or only a late inhibition (for review see De Laat[7]). Cadden and Newton[54] showed that intraoral stimuli in symptom-free subjects most often results in two inhibitory periods (with a latency of around 14 and 47 ms, respectively), which are sometimes fused at higher stimulation intensities. For extraoral stimulation, the exact location of the stimulus appears to be crucial; electrical pulses to the vermilion border of the lip produced a single inhibition with a latency of 25 ms, whereas stimulation of the hairy skin of the lip produced only the late inhibition (with a latency of around 48 ms). In the majority of subjects tested, electrical stimuli applied across the commissura of the lip (implicating stimulation of both the second and third branches of the trigeminal nerve) or to the mental nerve area[55] consistently resulted in a double ES.[56]

The intensity of stimulation appears to be important when weak stimuli are used,[57] but not at higher intensities.[56] A constant clenching level should be maintained while the stimuli are delivered, and most studies advocate 50% or 100% of the maximal voluntary contraction (MVC). Türker and Miles[58] suggested the control of muscle excitation through the firing rate of a single motor unit. To decrease the intraindividual variability and the experimenter bias,[56] a series of sweeps should be analyzed after averaging and rectification.[59–61] To facilitate better comparison among future studies, specific parameters for stimulation, recording, and analysis have been recently proposed.[62]

McGrath et al[63] concluded that the reflexes that occur after electrical stimulation of the tooth pulp cannot be considered a real correlate of pain sensation. Similar to the findings of De Laat et al,[50] these authors found that there were more single inhibitory periods (ES1) in patients with myofascial pain, while double or fused inhibitions (ES2) were present in a symptom-free group.[64] In a study that compared TMD patients with controls, stimulation of the intraoral mucosa of the lower lip at 6 times the perception threshold (T) resulted in a recognizable two-phase inhibition, which sometimes merged.[65,66] No difference was observed regarding the sensory and reflex thresholds (approximately $4 \times T$), but different reflex responses were observed in both the surface EMG and in single motor unit recordings; in TMD patients, ES1 appeared more often and was more resistant to changes in excitability level, while ES2 was only present in a minority of the sweeps. In symptom-free subjects, by contrast, ES1 was small or absent, and ES2 was mostly present and remained at higher excitation levels. These observations were attributed to changes in the input to one or more interneurons that link the afferent limb of the reflex to the masseteric motoneurons. Unfortunately, no distinction was made with regard to the type of TMD in the few patients tested. The lack of strict inclusion and exclusion criteria might also explain the controversial results of Raudino[67]; no clear trend for the presence/absence of ES2 was observed in a mixed group of patients who experienced temporomandibular dysfunction and/or pain.

Both ES1 and ES2 can be observed when the region of the mental nerve is stimulated extraorally. Hussein and McCall[68] reported longer SPs in TMD patients compared to controls, but the results were not corrected for clenching level. They also illustrated that SPs after electrical stimulation appeared to be shorter at 100% of MVC than at 50%, which might explain the differences observed among patients. Using the same technique, Cruccu et al[69] reported normal reflexes in patients with Huntington's chorea, dystonia, and parkinsonism. In patients with unilateral masticatory spasm, SPs were absent. Using double shocks, the recovery of the first and second SPs was studied in TMD patients and in controls,[17] and no differences were observed with regard to the presence/absence of ES2 or the recovery cycle.

The technique of stimulation across the commissura has been frequently performed, and it has focused particularly on the ES2 in the temporalis muscle. Originally, the absence of or decrease in temporalis ES2 was promoted as a diagnostic tool for chronic tension-type headache.[70–73] Unfortunately, and similar to the findings about the masseteric SP in TMD patients, the initial enthusiasm about ES2 duration as a diagnostic tool decreased, probably because the analysis of single sweeps can lead to very high intraindividual variability in the same recording session[68,74] and over days.[73,74] Using a very strict methodology, Bendtsen et al[56] were able to limit the intraobserver and interobserver variation through the analysis of averaged and rectified sweeps, but they confirmed the intraindividual variability to be around 20% (ES2 duration). In this study, the latencies and durations of ES2 were similar when the temporalis and masseter muscles were compared within the same subject, but the interindividual variability was high (36.7% for temporalis muscles and 43.7% for masseter muscles). Repeating the experiment after 1 week resulted in a longer ES2 in the same subjects, perhaps because the arousal of the subjects' sense of the electrical stimulation decreased during the second session. Consequently, it was suggested that a practice session be performed before actually starting a longitudinal study. Most importantly, it was concluded that the ES2 duration cannot be used as a diagnostic tool because of the high interindividual variability; this was later confirmed in a controlled study on tension-type headache patients,[75] where no differences between the groups were observed. Because the treatment of tension-type headache patients with amitriptyline resulted in a shorter ES2, modulation both by serotoninergic and noradrenergic pathways has been suggested.[76] However, in another study neither aspirin nor placebo changed the latency of ES2 in migraine or tension-type headache patients, but aspirin did result in a significant increase in ES2 duration.[77]

Blink Reflex

Stimulation of the supraorbital nerve[9,78] or the mental nerve[79,80] results in a reflex contraction of the orbicularis oculi muscle, which normally consists of an ipsilateral response (R1) and a bilateral response component (R2) that are mediated via tactile A-beta fibers.[81] The latencies of these blink reflex components are considered to be pathologically delayed when they exceed 13 and 41 ms, respectively. The blink reflex provides information on the trigeminofacial reflex arc, and it has been studied in patients with brain stem lesions,[82] dementia,[83,84] and lesions of the inferior alveolar nerve.[85] A late response (R3) component has also been described[69,86,87] after noxious stimuli in symptom-free subjects, and it is probably mediated via thin unmyelinated A-delta fibers.[88] Recently, this R3 component has been studied in patients with burning mouth syndrome (BMS).[89] In 50% of the patients, even non-noxious stimulation resulted in an R3 component; this was only rarely the case in the control group. In addition, the threshold for the tactile R1 component appeared to be significantly higher in the patients, which indicates neurophysiologic changes in the somatosensory/motor function of BMS patients.

Jaw and Facial Reflexes During Experimental Pain

Figure 1 summarizes the variations and confounding factors discussed for patient studies. These methodologic difficulties could be reduced in experiments that are performed in a controlled laboratory environment. In this way, the methodology could be optimized and standardized. Moreover, working with symptom-free volunteers and the use of a stimulus whose nature and mediating afferents are better understood has provided better insight into the observed reflex changes.

1. Stimulus parameters
- Modality
- Intensity
- Application site
- Repetition rate (interstimulus interval, number of stimuli)
- Control of background contraction
- Automatic triggering

2. Signal recording and processing
- Electrode placement
- Filtering
- Averaging of multiple sweeps
- Rectification
- Statistical determination of cutoffs (confidence interval)
- Automatic versus experimenter detection of reflexes
- Latency and duration determination, experimenter bias

3. Patient population
- Homogeneous groups, clear inclusion/exclusion criteria
- Adequate control groups
- Clinical pain intensity, duration, location
- Psychologic state
- Sample size

Fig 1 Summary of variations or confounding factors in studies that involve reflexes in TMD or orofacial pain patients.

H Reflex in Masseter and Temporalis Muscles

The H reflex has been used especially in limb muscles to study motoneuron excitability, presynaptic, recurrent, and reciprocal inhibition, and the effect of various conditioning stimuli (for review see Schieppati[90]). In contrast to limb muscles, reports on the H reflex in the trigeminal system were scarce[91–93] until a monopolar stimulation technique was developed[94]; this technique enabled the production of a clear M response and H reflex in the masseter muscle,[95,96] and a heteronymous H reflex in the ipsilateral temporalis muscle.[95,97] As in most homonymous muscles, masseteric H reflex decreases parallel the increase of the M response at higher stimulation intensities. The heteronymous temporalis H reflex, however, persists and reaches a plateau. In this way, the heteronymous H reflex serves as an excellent tool for the study of motoneuron excitability in the trigeminal system. With increasing background activity, the heteronymous H reflex increases,[98] and it is also modulated by changes in the head position and the asymmetric tonic neck reflex.[99] Godaux and Desmedt[93] observed a decrease in the masseteric H reflex while vibration was applied to the chin of their subjects. During jaw movement in cats, the H reflex is modulated according to the phase of jaw movement (facilitation during jaw closing and inhibition during jaw opening).[100]

In limb muscles, the H reflex has also been studied with different kinds of experimental pain.[101–103] No changes with regard to reflex latency or amplitude were observed during the experimental pain state when compared to the nonpain situation. Similarly, the relief of knee pain did not change the H reflex in the quadriceps femoris.[104]

In the trigeminal system, a recent study[105] applied electrical stimuli to the masseteric nerve at 150% of the intensity needed to produce a maximal M response in the masseter muscle. The symptom-free subjects maintained 50% of their MVC, and a series of 20 stimuli were delivered before, during, and after periods of experimental muscle pain that was induced by a standardized infusion of hypertonic saline (5%) in the contralateral masseter muscle.[106] At this clenching level and high level of stimulation, the acute jaw muscle pain could not modulate motoneuron excitability, as measured by the heteronymous temporal H reflex.

Poststimulus EMG Complex After Mechanical Tooth Stimulation

When ice water was used as a remote noxious stimulation to the hand of subjects in which the PSEC was evoked by mechanical taps on a central incisor, decreased latencies and amplitudes of both the early and late inhibitory phases were observed.[107] The effects were more clear after harder taps than after soft taps, which led the authors to suggest that PSEC components that were mediated through high-threshold mechanoreceptor afferents were especially involved. Because the taps were never reported to be painful, the authors concluded that the observed changes were not a result of pain or autonomic reactions, but were mediated at the level of the brain stem. Interestingly, when subjects increased their level of attention by performing mental exercises during the stimulus, a similar attenuation of both inhibitory components of the PSEC was observed.[108]

Intraoral and Extraoral Electrical Stimulation

As mentioned earlier, the masseteric inhibitory reflex can be evoked at non-noxious levels of tooth pulp stimulation.[109–111] This contradicts an earlier report that states that noxious stimuli were necessary to evoke the reflex response.[112] Increasing the stimulus intensity from non-noxious to noxious levels resulted in longer inhibitions with shorter latencies.[111] This indicates that the reflex certainly has a nociceptive component.

Capsaicin (the active substance in red peppers) has also been used as a stimulant. It selectively affects C fibers and depletes these fibers of substance P in the dorsal horn, and also induces an expansion in the sensory receptive field of the afferent neurons; these effects are similar to those induced by sectioning a nerve.[113] Application of capsaicin to the ipsilateral cheek of healthy volunteers resulted in a clear dissociation between the sensory and reflex effects; the ratings of the tooth pulp–evoked sensation were significantly reduced after capsaicin application, while the masseteric reflexes did not differ from the control situation. This points to a lack of convergence between cutaneous C fibers and tooth pulp–elicited reflex circuits.[111] This finding is in line with the observed dissociation between orofacial sensory estimates and inhibitory reflexes after the administration of narcotics[63] or under hypnotic anesthesia.[114]

The longer latency inhibitory component that occurs after upper lip stimulation is thought to be modulated through a premotoneuronal reflex arc; the inhibition is forcefully reduced by the application of painful cold or hot stimuli to the hand,[115] by the anticipation of painful electrical stimuli to the ankle,[116] and even by increased attention.[117] If subjects perform painful arm exercises under ischemia, the inhibition is also reduced by more than 40%; it returns to control values after 5 minutes.[118] In contrast to the effects after the combination of ischemia and exercise, neither ischemia alone nor exercise alone influenced the reflex. This led the authors to conclude that remote nociceptive—not non-nociceptive—deep somatic afferents could mediate this influence.

The relationship between the stimulus intensity and the exteroceptive suppressions ES1 and ES2 that occur after electrical stimulation of the mental nerve or the corner of the mouth is far from clear. Desmedt and Godaux[119] reported an equal threshold for ES1 and ES2, and longer and deeper suppressions when the stimulus intensity was increased. By contrast, Yu et al[44] stated that ES1 has a lower threshold, while more recent studies have found a lower threshold for ES2.[120,121] Apparently the reflex can be observed at non-noxious levels of stimulation,[55] but ES2 in particular seems to be related to nociception.[99,112] The duration of ES2 is reduced by tonic experimental pain (cold pressor test),[56] conditioning stimuli (radiant heat applied to the ipsilateral cheek),[122] and experimental muscle pain.[123]

Blink Reflex and Experimental Pain

Both the R2 and R3 components of the blink reflex have been studied under experimental pain conditions. The R3 component appears to always be linked with pain sensation, but it is easily habituated, and it is also modulated by attention and distraction.[86] Whereas the threshold to evoke R2 is approximately the tactile threshold, the R3 threshold is always higher and is associated with reported pain.[87] In addition, this threshold is increased in patients with hypertension compared to controls.[124] Recently, R2 and R3 components have been investigated after both electrical and laser radiant heat stimuli.[125] The R2 and R3 responses were comparable after the two kinds of stimuli when the time needed for the laser stimulus to become painful was taken into account. This allowed the authors to conclude that the same nociceptive fibers are stimulated or that the electrically evoked and laser-evoked responses result from the same multireceptive neurons in the dorsal horn and midbrain. Homotopically applied radiant laser heat resulted in the inhibition of R2 and the facilitation of the R1 component.[122] A similar modulation of R2 has been described after the induction of ischemic muscle pain.[126]

Conclusions

A diverse range of elicitable reflexes in the jaw muscles is apparent. The importance of the locus of stimulation as well as its nature (mechanical, electrical, chin tap, etc) calls for studies to specify a clear methodologic outline since different reflex patterns can emerge. It is intriguing that comparable sequences of inhibitory waves, which alternate and are sometimes followed by periods of increased EMG activity, can be observed after mechanical stimulation of single teeth, after electrical stimulation of the corner of the mouth and the mental nerve area, and even after light tactile stimulation of the perioral skin with a puff of air. The first inhibition of this reflex sequence involves postsynaptic inhibitory mechanisms, and it is probably served by a disynaptic reflex arc, through supratrigeminal interneurons[4,127]; it appears to be stable under the different conditions tested. However, the background EMG activity is always an important factor because an increase in the voluntary drive will result in smaller reflexes. In TMD patients, the difference in background activity has been especially noted as the main explanation for the longer SPs observed after chin tap, and it may also play a crucial role in the different amplitude or latency of the jaw-jerk reflex in patients with unilateral TMD-related pain.

The inhibition that occurs at a longer latency (described as ES2 after electrical stimulation of the mental nerve, the commissura, or tooth pulp; the reflex after electrical stimulation of the upper lip; the second inhibition of the PSEC, etc) is influenced by a variety of conditioning factors: level of attention, level of arousal, remote experimental pain, remote non-noxious stimulation, etc. All of these factors may explain why in some patient groups a decreased inhibition was reported in comparison to controls. Similar pitfalls, however, as those encountered in the early studies that used chin tap may arise. In this respect some suggestions regarding future research should be considered:

- A clear description of the stimulus is necessary and it would be advantageous to specify the locus of stimulation in a detailed way. Most studies to date appear to have used the mental nerve area or the perioral region (commissura, upper lip). It is also important to record the sensory effects of the stimulus (using visual analogue scales and descriptive questionnaires) in addition to its intensity. Detailed study of the "curves" of the different reflexes using increasing intensities (expressed in physical as well as psychophysical units) and different levels of constant background activity (expressed as percentages of MVC or as a constant clenching force) are needed in both symptom-free subjects and in patients to provide a framework and normative data. Even if an electrical stimulus has significant characteristic advantages, the fact that apparently similar responses can be evoked by more "natural" mechanical or tactile stimulus should not be forgotten.
- The use of experimental setups that automatically trigger the stimulus when a constant clenching level is present is advocated. This would minimize any experimenter or subject-related bias. With regard to the level of background activity, it could be speculated that the modulation of a reflex response will be more difficult if the tonic drive is very high or when high intensity stimuli are used. Studies that use lower clenching levels with the whole range of stimulus intensities would address this problem.

- Examining the overall "shape" or configuration of the reflexes (single, double, merged, etc), or studying the frequency of occurrence of the reflex parts, or the variability of occurrence is suggested instead of trying to identify differences with respect to latency, duration, degree of suppression, etc, of the reflexes.
- If patients are to be studied longitudinally, it would be better to familiarize them with the experimental procedure before beginning the study. This would decrease the side effects of arousal, attention, etc.
- Clear inclusion and exclusion criteria based on recognized diagnostic descriptions should be used to form homogeneous patient groups. Adequate control groups, controlling for age and gender, should always be included.

In addition to the important clarification that clinical neurophysiology can offer in brain stem lesions or neurologic disease, studies that particularly involve experimental pain have helped in the understanding of the characteristics of the afferent fibers and synaptic circuitry that are involved in the jaw and facial reflexes. The data from studies on patients with TMDs or tension-type headache, on the other hand, remain controversial and appear not to support the use of these reflexes as a diagnostic tool in daily practice at this point in time.

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Resumen

¿Son los Reflejos Faciales y Mandibulares Modulados Durante el Dolor Orofacial Clínico o Experimental?

Se pueden evocar una variedad de reflejos faciales y mandibulares por medio de estímulos eléctricos o mecánicos. Debido a la posible utilidad diagnóstica en el manejo del dolor y la disfunción del sistema masticatorio, se ha investigado particularmente la supresión exteroceptiva que puede ser evocada en los músculos temporales y maseteros. La revisión de los diferentes estudios enfatiza la importancia crucial del área estimulada y el tipo de estimulación usada pare evocar el reflejo. Los estudios más recientes hen aplicado la estandarización necesaria de la intensidad del estímulo, los niveles de apretadura con los dientes, los procedimientos registrados y la interpretación imparcial de los componentes reflejos en la actividad electromiográfica muscular. Se han reportado resultados controversiales en relación a las diferencias en las respuestas de estos reflejos inhibitorios (y excitatorios) entre pacientes que sufren de desordenes temporomandibulares o de cefaleas, y los controles. Aún si la ausencia de una segunda fase inhibitoria en la actividad electromiográfica masetérica de los pacientes es un hallazgo frecuente su sensitividad y especificidad como instrumentos de diagnóstico para el dolor miógeno o bruxismo, todavía tienen que ser probadas. Los estudios controlados sobre la duración del segundo período de supresión exteroceptivo en la cefalea debida a la tensión, no pudieron confirmar la diferencia reportada inicialmente entre pacientes y personas asintomáticas. Los estudios que involucran el dolor muscular inducido experimentalmente podrían proveer un discernimiento mejor en cuanto a las características de las fibras aferentes y el sistema de circuitos sinápticos que están envueltos en los reflejos mandibulares y faciales.

Zusammenfassung

Werden Kiefer- und Gesichtsreflexe während Klinischen oder Experimentellen Orofazialen Schmerzen Moduliert

Eine Vielzahl von Kiefer- und Gesichtsreflexen kann durch mechanische oder elektrische Stimuli hervorgerufen werden. Wegen des möglichen diagnostischen Nutzens in der Behandlung von Schmerzen und Dysfunktionen des Kausystems wurde die exterozeptive Suppression, die in den Masseter- und Temporalismuskeln hervorgerufen werden kann, besonders untersucht. Ein Rüchblick von verschiedenen Studien hebt die entscheidende Wichtigkeit des stimulierten Areals und des Stimulationstyps, der angewendet wurde, um den Reflex auszulösen, hervor. Neuere Studien haben die notwendige Standardisierung der Stimulusintensität, der Stufe des Pressens, des Aufzeichnungsablaufes und der unbeeinflussten Interpretation der Reflexkomponenten in der elektromyographischen Muskeaktivität (EMG) verwendet. Unterschiedliche Ergebnisse wurden hinsichtlich der Differenzen in diesen inhibitorischen (und exzitatorischen) Reflexantworten zwischen den Patienten mit temporomandibulären Erkrankungen oder Kopfschmerzen und den Kontrollpersonen berichtet. Auch wenn die Abwesenheit einer zweiten inhibitorischen Phase in der EMG-Aktivität der Masseteren bei den Patienten ein häufiger Befund darstellt, bleibt seine Sensitivität und Spezifität als ein diagnostisches Werkzeug für myogene Schmerzen oder Bruxismus zu testen. Kontrollierte Studien über die Dauer der zweiten exterozeptiven Suppressionsperiode beim Spannungstyp-Kopfschmerz konnten die ursprünglich berichtete Differenz zwischen Patienten und asymptomatischen Personen nicht bestätigen. Studien, welche experimentell induzierte Muskelschmerzen enthalten, könnten bessere Einsicht in die Charakteristika der afferenten Fasern und synaptischen Kreisläufe, welche an den Kiefer- und Gesichtsreflexen beteiligt sind, liefern.