The Temporomandibular Joint Pain Dysfunction Syndrome and The Orofacial Pain

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INTRODUCTION

Headache and orofacial pain due to the temporomandibular joint (TMJ) pain dysfunction syndrome is a cause of human suffering often resulting from major diagnostic and therapeutic difficulties. These difficulties arise from the fact that the syndrome is rather little known among dentists as well as other medical specialists. The main symptom of the dysfunction syndrome is pain, on account of which the patient seeks medical advice. Depending upon the speciality of the therapist, the leading features of treatment will derive from disciplines such as gnathology, prosthetics, oromaxillar surgery, algesiology, neurology, psychiatry and psychology.

The pain of TMJ dysfunction syndrome is thought to be of myofascial origin. However, the etiology of myofascial pain is still a matter of controversy. Although increase of muscle tone is emphasized as a major contributing factor, there are three established theories, each of which attempts to explain the mechanism of TMJ pain dysfunction syndrome from a different perspective:

1. The theory of occlusal disharmony attributes pain to increased muscle tone resulting from faulty dental contact or occlusal interference, e.g. due to dislocation of one or both condyles from their hanging position in the mandibular joint. Such dislocation is responsible for ventral displacement of the mandible and simultaneously, permanent overstrain of masticatory muscles — particularly the Mm. pterygoidei laterales. In the course of chewing and speaking these muscles become painful and spastic; which further enhances discoordination and limitation of mandibular motion.

2. The psychological theory proposed by Laskin holds that

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psychosomatic stress alone can cause increased muscle tone and eventually lead to muscle spasm in the absence of occlusal disharmony.

3. The trigger point theory assumes that the pain may result from stimulation of trigger points which develop in muscles for unclear reasons.26,35,36

Our investigations showed, however, that pain in this syndrome occurs due to stimulation of trigger points during long speech, chewing of solid food, clenching of teeth during sleep and passive pressure on masticatory muscles during sleep in the lateral or prone position. A common characteristic of these patients is increased psychological and somatic tension.

The diagnostic criteria of the TMJ pain dysfunction syndrome have been defined previously by Laskin17. Several factors indicate that TMJ dysfunction pain is of muscular origin. The pain is difficult to describe and localize; it is perceived as deep pain such as in muscular spasm. The pain can be elicited by palpation of trigger points. Stimulation of masticatory and other muscles with hypertonic saline causes the same type of pain.13,18,29,32 Pain originating in trigger points is perceived in the muscle itself or in distant areas, referred to as reference zones. Pressure exerted on an active trigger point causes the pain to radiate to the respective reference zone which can be described by the patient and recorded. It should be noted that there is a high correlation between trigger points and acupuncture points.19,28,33 The reference zones of the various masticatory muscles are distributed throughout the head and face. In the present study, reference zones of masticatory muscles were examined to investigate the nature of the TMJ pain dysfunction syndrome. Since the influence of psychological stress on muscle tone of masticatory muscles has been well documented17, special consideration will not be given to this factor as a cause of headache and facial pain in the present paper.

METHODS

Twelve patients, and seven volunteers including the authors participated in the study after providing informed consent. The present methods have been described in detail elsewhere.28,29 Briefly: reference zones were established using 10% saline. Pain elicited by hypertonic saline was abolished by lidocaine (1 ml 1% Xylocain, Astra Chemicals) 15 seconds later. One injection of hypertonic saline per muscle in 2–4 muscles of the masticatory apparatus was performed in each subject — long time intervals between injections being meticulously preserved. The observed reference zones were not uniform — a fact attributed to differences among subjects with respect to muscular innervation,
Fig. 1. Reference zones of referred myofascial pain elicited by stimulation of trigger points (TP Nos. 1 – 4) in the temporal muscle. A cross marks each trigger point and the stippled area indicates recorded reference zones. Heaviness of the stippled area corresponds to the intensity of referred pain.

Fig. 2. Reference zones of referred myofascial pain elicited by stimulation of trigger points (Nos. 1 – 4) in the masseter muscle.
Fig. 3. Reference zones of referred myofascial pain elicited by stimulation of trigger points in the lateral pterygoid (upper and lower portion) and medial pterygoid muscles.

Fig. 4. Reference zones of referred myofascial pain elicited by stimulation of trigger points in the anterior and posterior heads of digastric muscle.
Fig. 5. Reference zones of referred myofascial pain elicited by stimulation of trigger points in the clavicular and sternal portions of the sternocleidomastoid muscle. Further trigger points have been found in the upper and middle part of the muscle.
amount of injected saline and pain threshold. Areas were therefore only included in reference zone, subject to agreement in several subjects.

RESULTS

The reference zones of masticatory muscles are shown in Fig. 1–6. The reference zones of muscles and given portions of a muscle extended into different dermatomes and innervation areas. The reference zones of the first three trigger points of the temporal muscle were projected to areals within the field of innervation of the trigeminal nerve; that of the fourth trigger point within the C2–C3 dermatome as well as the ear lobe.

It may be noted that individual reference zones can overlap (e.g. trigger points No. 2 and 4 of M. masseter). It is striking that stimulation of the upper and lower dorsal trigger points of the masseter muscle (No. 1 and 3) caused pain in the temporal and the periocular area — which also corresponds to the reference zone revealed by stimulation of the temporal muscle. Furthermore, pain was also perceived in the upper and lower teeth. Pain referred to the upper and lower teeth may also occur after dental extraction and is, in our opinion, responsible for dental phantom pain. Noteworthy is the pain from the posterior head of the digastric muscle, which was referred to the occipitocervical crossing immediately behind the ear. The present results show that referred pain from this muscle can occur in the face and head.
DISCUSSION

The distribution of reference zones found in the present study largely agrees with observations made by Laskin.\textsuperscript{17} However, there were some differences with respect to reference zones of the clavicular and sternal portions of the sternocleidomastoid muscle as reported by Travell\textsuperscript{34}: in the present study, pain from the clavicular portion of the muscle radiated from the neck down to the elbow.

The overlapping of the observed pain patterns elicited by trigger point stimulation may reflect involvement of afferent fibers of muscles and probably also afferent fibers of specific and nonspecific articular nerves\textsuperscript{40} passing through these muscles. It should be kept in mind, however, that the nociceptive coding in the central nervous system may be rather poorly differentiated. In any case, the nociceptive input from various muscles seems to converge at the same neurons, producing a particular pain pattern. It is therefore mandatory to remember that pain involving broad parts of the head and face can originate in the masticatory muscles. Similarly, dental pain may actually represent referred pain, which has nothing to do with dental disease. In such a case, it is a grave mistake to extract healthy teeth. Referred pain related to TMJ dysfunction syndrome should particularly be considered in patients with good dental hygiene. Pain of this syndrome can involve broad areas, such as headache or even hemilateral facial pain. In contrast to migraine, mild forms of TMJ dysfunction syndrome affect the temporal, periocular or periauricular region; in special cases, pain can occur bilaterally. It seems unlikely that TMJ pain patients would profit from betablocker medication.

Therapy-resistant TMJ pain dysfunction syndrome is often accompanied by extensive pathology of the cervical spine. For this reason, the pathological input from both temporomandibular and intervertebral joints must be considered. Such input from cervical intervertebral joints is mostly caused by vertebrogenic blockage, which has been defined as a "reversible state of anomalous function of joint mobility without organic lesion".\textsuperscript{39} "Joint play"\textsuperscript{20} is abolished during such functional anomaly of joints.

Rissanen has observed distinct degenerative changes of ligamentous apparatus in patients over thirty years of age.\textsuperscript{23} However, most of the present patients with facial pain due to TMJ pain dysfunction syndrome were over forty years of age. This may reflect the fact that the incidence of degenerative processes of the cervical spine increases with advanced age.

In our opinion, facial pain due to TMJ pain dysfunction syndrome originates not exclusively from impaired coordination of the masticatory apparatus, but also from lesions of cervical spine structures and statodynamic disturbances of the entire spine. Schimek reported previously\textsuperscript{27} that 39 out of 43 patients with
therapy-resistant facial pain due to TMJ pain dysfunction syndrome had cervical lesions in the form of cervical instability or discopathy and the remaining patients had deceleration trauma of the cervical spine. Cervical instability is characterized not only by disc lesions, but also by damage to the intervertebral joints and ligaments\textsuperscript{12,38}, and was therefore termed the “syndrome of three joints”\textsuperscript{41}. We feel that with the exception of trauma such damage is a consequence of muscular discoordination and statodynamic disturbance of the entire spine. Such pathological conditions develop through functional impairment involving the pelvic ring and the sacral bone which together may be regarded as the basis of the spine. During blockage of sacroiliac joints, which is accompanied by a relative difference of leg length, the sacral bone is mispositioned in the pelvic ring. In the erect position, the cranial surface of the sacral bone consequently assumes an oblique position\textsuperscript{27,31} towards the prominently blocked side and the spine develops compensatory scoliosis. The scoliosis usually involves rotation of the vertebrae. This process progresses cranially and ends at the C\textsubscript{1} vertebra, were the skull prevents further dynamic adaptation of the spine. This evidence was forthcoming from meticulous examination of the dynamics of the spine in our patients with therapy-resistant TMJ pain dysfunction syndrome. The vertebrogenic blockage involved several vertebrae, occurred most often at the C\textsubscript{0}–C\textsubscript{1} segment, and extended down to C\textsubscript{4}–C\textsubscript{6}.\textsuperscript{27,28,31} The present clinical observations confirm the previous reports: the removal of vertebrogenic blockage at C\textsubscript{0}–C\textsubscript{2} abolished blockage of the sacroiliac joint. On the other hand, it was possible to remove vertebrogenic blockage in the upper cervical spine of some patients by relieving blockage of sacroiliac joints.

Furthermore, the trigeminal nuclei reveal close relationship between afferent impulses from masticatory muscles, neck muscles, facet joints of the cervical spine and temporomandibular joints. For this reason, lesions of the cervical spine increase not only the muscle tone in the cervical area, but also influence the tone of masticatory muscles. Involvement of the cervical spine should therefore always be considered when encountering incoordination of masticatory muscles and discoordination of mandibular movement.

The upper cervical spine is not just a static organ but also a receptor, which influences the balance and coordination of movement of the whole body.\textsuperscript{7,11} The input from the C\textsubscript{0}–C\textsubscript{3} segments affects almost all sensory and motor innervation of the head.\textsuperscript{10,11} In addition, the input and output from neurons of both the upper cervical spinal cord and medula oblongata coordinate movements of the body, head, and temporomandibular joint; such as chewing and speaking. Disturbances in the upper cervical region can therefore influence the entire spine, pelvis, extremities and also abdominal and thoracic organs; as well as vice versa.\textsuperscript{1}
Coordinative impulses to the masticatory apparatus are propagated mainly through trigeminal and partly facial nerves. Neurophysiological studies showed that cytoarchitectonic and functional structure of the pars caudalis of the trigeminal nerves remarkably resembles the structure of dorsal horns; some authors therefore speak of "medular dorsal horn" as a continuation of the "spinal dorsal horn".\textsuperscript{7,22} The caudal nucleus of the trigeminal nuclei extends down to the upper third of the C\textsubscript{2} segment and receives afferent fibers directly from the cervical root of segments C\textsubscript{1}—C\textsubscript{4}, and indirectly from C\textsubscript{4}—C\textsubscript{7}.\textsuperscript{5,14} For this reason, there are synaptic connections between cervical roots and the trigeminal nuclei; The pathological input from afferent fibers of cervical roots resulting, for example, from functional disturbances or lesions in the craniocervical region, is transmitted to the trigeminal nuclei; facial pain may therefore be related to pathological influences from cervical segments. Subsequently, muscular tone of masticatory muscles increases, which causes myofascial pain in the head and face.

We propose that the development of trigger points is a reflectory process due to pathological input from mechanoreceptors and nociceptors of zygoapophyseal joints of the spine, temporomandibular joints, and capsules of these joints.\textsuperscript{27,30,32} Type I and II mechanoreceptors and nociceptors are not only distributed over intervertebral joints, but also over temporomandibular and other joints.\textsuperscript{8,15,16,21,30,37,40} The abnormal input from these receptors increases the tone of the whole muscle, or some portions of it.\textsuperscript{32} We feel that afferent stimuli from any intervertebral joint can raise the tone of muscles innervated from a particular segment and cause the development of trigger points.\textsuperscript{27,32} For example, we consistently detected trigger points in muscles innervated by the C\textsubscript{5} segment (Mm. trapezius, deltoideus, supraspinatus, sternocleidomastoideus, etc.) in patients with acute vertebrogenic blockage at this segment. These trigger points disappeared immediately or within a short period of time after successful manual therapy of the acute blockage. Such a dramatic effect was lacking in patients with chronic vertebrogenic blockage, since abnormal chronic input from joint receptors caused alteration of the respective muscles. In this context, we are introducing the term "arthromyofascial pain syndrome" for conditions involving trigger points and referred pain which can be attributed to increased muscle tone as a result of chronic pathological input from the mechanoreceptors and nociceptors of joints of the cervical spine and other joints.

For the development of trigger points in masticatory muscles it is necessary that the pathological inputs from receptors of TMJ and one or several zygoapophyseal joint capsules reach the dorsal horn of the spinal cord. The cervical intervertebral joints have the highest polysegmental sympathetic innervation.\textsuperscript{3,4} For this
reason, symptoms of sympathetic overactivity occur concomitantly in the reference zones of masticatory muscles, e.g. redness of skin or eyes, lacrimation, nasal congestion and visual disturbances. These symptoms manifest homolaterally, however.

We conclude that in addition to the occlusal disharmony, psychosomatic stress and trigger point stimulation, the most important factor in the development of TMJ pain dysfunction syndrome is the disturbance of statics and dynamics of the cervical spine. In contrast to the previous theories mentioned above, one single factor is rarely solely responsible for the pain in this syndrome. Thus therapy of TMJ pain dysfunction syndrome should not be restricted to gnathological management, but should also include relaxation procedures (EMG-biofeedback, autogenic training), physiotherapy and chiropraxis. In complicated cases neurosurgery and orthopedic operations are necessary in order to remove the pathological input from cervical vertebral joints and corresponding muscles.

**SUMMARY**

The reference zones of myofascial pain originating in masticatory muscles were investigated in twelve chronic pain patients and seven healthy volunteers. The results of the study indicate that headache associated with temporomandibular joint pain dysfunction syndrome not only results from malfunctioning of the masticatory apparatus, but may also result from malfunctioning of the cervical spine. Facial pain may be a sequela of organic lesions of the cervical spine and is invariably accompanied by headache. Furthermore, in patients with headache and facial pain, functional pathology (abnormal statics and dynamics) of other parts of the spine was observed. For these reasons it seems mandatory to search for derangements of the entire spine in patients suffering from the temporomandibular pain dysfunction syndrome, particularly in cases of failed dental/functional therapy.

**REFERENCES**

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