Pain Patterns and Mandibular Dysfunction Following Experimental Trapezius Muscle Pain

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Aims: To clarify the effects of experimental trapezius muscle pain on the spread of pain and on jaw motor function. **Methods:** *In 12 male subjects aged 25 to 35 years, experimental pain was induced in the superior border of the trapezius muscle by injecting 0.5 mL of hypertonic (6%) saline. The control infusion consisted of a 0.5-mL isotonic (0.9%) saline solution. Pain intensity was evaluated on a visual analog scale (VAS). An experimental (EX) and a control (CT) injection were administered to the subjects in a randomized sequence.* **Results:** *Pain intensity as scored on the VAS increased immediately after the EX injection and decreased gradually after reaching a peak of 68.0 ± 16.1 mm at 60 seconds after injection. The VAS scores in the EX condition were significantly higher than after the CT condition from 30 to 330 seconds after injection (P < .05, analysis of variance [ANOVA]). Mean (± SD) maximal unassisted mouth opening before injection in the EX condition was 54 ± 5.7 mm and decreased immediately after the injection, reaching a low of 47.8 ± 5.1 mm. A gradual recovery to normal was then observed. This reduction of mouth opening in the EX condition was significant compared with the CT condition from immediately after the injection to 60 seconds after the injection (P < .05, ANOVA). According to the subjects, pain spread most often to the infra-auricular zone (n = 6), and the posterolateral part of the neck (n = 10).* **Conclusion:** *The present results suggest that experimental trapezius muscle pain can spread over a wide area and is also accompanied by a temporary reduction of mouth opening.* J OROFAC PAIN 2005;19:119–126

Key words: experimental pain, jaw motor function, limited mouth
        opening, pain spreading, trapezius muscle

Patients with temporomandibular disorders (TMD) experience pain around the temporomandibular joint (TMJ) and in the jaw muscles, but they also report associated pain in the shoulder or neck area.[1] Myofascial trigger points in the shoulder and neck muscles have been discussed as possible sources of referred facial pain.[2] Convergence of trigeminal and cervical input on second-order neurons, in addition to central excitatory effects resulting from nociceptive input, have been suggested as possible explanations for this referred pain.[3] Afferent nociceptive input was found to induce co-contraction of masticatory muscles in TMD patients[4] and in studies using experimental jaw muscle pain.[5,6] In animals, application of inflammatory irritants to cervical paraspinal muscles results in excitatory effects in both jaw and neck muscles.[7–9] Consequently, one could hypothesize that

myofascial pain in the shoulder and neck muscles could have an influence on jaw motor function.

Experimentally induced muscle pain can be used to gain a better understanding of the mechanisms involved in acute and chronic muscle pain, which may subsequently lead to a better characterization, prevention, and management of pain. Intramuscular infusion of hypertonic saline has proven to be an efficient method for the study of deep pain, because the quality of the induced pain mimics clinical muscle pain.[10–13] Continuous infusion of hypertonic saline into muscles[14–17] and electrical stimulation of muscles[18–22] are widely used to induce muscle pain. Injection of isotonic saline[13] offers an acceptable control situation for the saline-induced muscle pain.

In the orofacial region, a large number of studies using experimental pain have been reported. In several studies, hypertonic saline was injected into the masticatory muscle, and motor responses in the orofacial region were observed.[23–26] In addition, the influence of experimentally induced pain in the orofacial region on various muscle reflexes was studied.[26–32] There is, however, little information on the influence of experimental pain in the neck and shoulder muscle on motor activity in the orofacial region. Ashton-Miller et al[33] performed a study using a hypertonic saline injection in the sternocleidomastoid muscle and measured electromyographic (EMG) activity of the cervical muscles. Madeleine et al[34] performed a similar experiment in trapezius muscle and measured pain intensity, but they did not investigate the effect on jaw motor function.

The present experiments were conducted to clarify the effects of experimental trapezius muscle pain both on pain spread and on jaw motor function. Part of this study was presented previously in Japanese.[35]

Materials and Methods

Subjects

The subjects were 12 males (25 to 35 years old, mean age ± SD: 27.9 ± 2.7 years) who had a complete dentition and no symptoms of pain in the neck, shoulder, or stomatognathic system. The exclusion criteria are listed in Table 1. The Human Subjects Ethics Committee of the Nihon University School of Dentistry at Matsudo approved the experimental protocol, and written informed consent was obtained from each participating subject.

Table 1 Exclusion Criteria

Exclusion Criteria
Previous treatment for orofacial pain
Currently receiving medication
History of orthodontic treatment
Metabolic disease (eg, diabetes, hyperthyroidism)
Neurological disorders (eg, dyskinesia, trigeminal neuralgia)
Vascular disease (eg, migraine, hypertension)
Neoplasia
History of drug abuse
Recent facial or cervical trauma (eg, whiplash)
Psychiatric disease

Experimental Procedure

A single-blind, randomized, repeated-measures study design was selected to examine the effect of experimental trapezius muscle pain. Pain was induced by infusing a 0.5-mL hypertonic (6%) saline solution. The condition induced was considered the experimental (EX) condition. The control infusion consisted of a 0.5-mL isotonic (0.9%) saline solution. The condition induced was known as the control (CT) condition. Each subject received 1 EX injection and 1 CT injection. The sequence of the EX and CT conditions was randomized, and the subjects did not know which substance was injected. The superior border of the upper trapezius muscle was chosen as the site of infusion. A 25-gauge needle was inserted into the muscle (1.5 to 3.5 cm deep) of the pars descendens of the left trapezius muscle (2 cm laterally from the midpoint between vertebra C7 and the acromion). The injection was performed over a 15-second period. Prior to hypertonic and isotonic saline injections, the skin was infiltrated with 0.1 mL of carbocaine (10 mg/mL, Astra). The second injection was administered 4 weeks after the first one to minimize memory of the first injection as much as possible.

Measurement

The evolution of the pain intensity caused by the injection was evaluated every 30 seconds from immediately after injection to 600 seconds postinjection with a 100-mm visual analog scale (VAS) that ranged from 0 ("no pain at all") to 100 ("the most intense pain you can imagine"). The distance from the zero point was measured with sliding calipers to within 1 decimal point. The maximum unassisted mouth opening as defined by the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD)[36] was also measured with sliding calipers to within 1 decimal
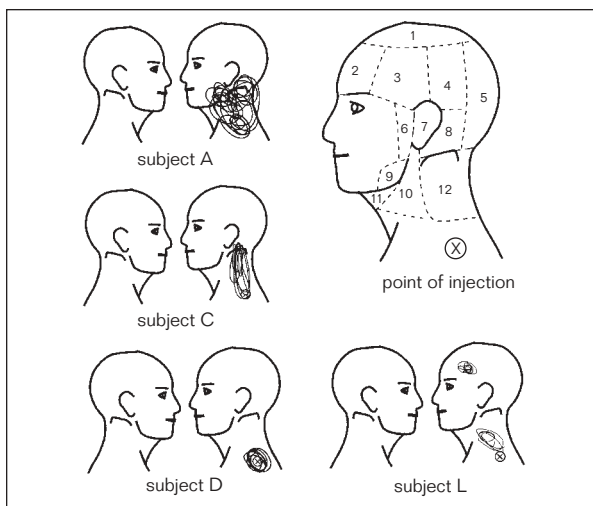
Fig 1 Evaluation of the distribution of pain spread. The drawings shown, made by subjects A, C, D, and L, are typical of the drawings made. The areas of pain spread were evaluated using a plastic template on which the face was divided into anatomical zones *(upper right)*. This template was superimposed on the subject's drawing. For subject A, pain spread into zones 5, 6, and 8 to 12. For subject C, pain spread into zones 8 and 12, for subject D, there was no spread of pain, and for subject L, pain spread into zones 3, 10, and 12.
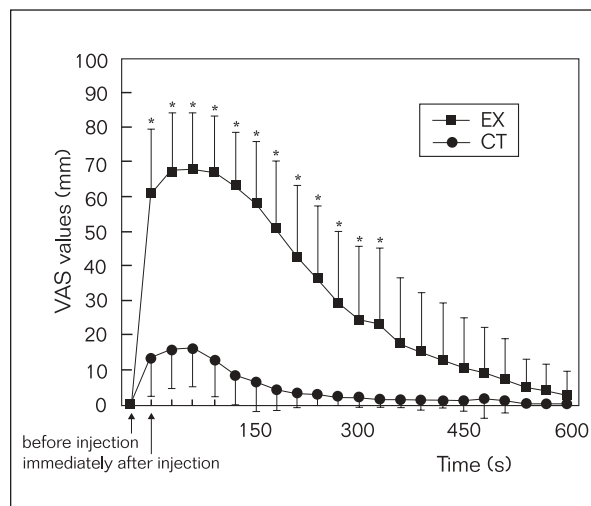

Fig 2 VAS values of pain intensity following injection of hypertonic saline (6%, EX condition) and isotonic saline (0.9%, CT condition). Values are means (± SD) for 12 subjects. The mean VAS for the EX condition was significantly higher than that of the CT condition from immediately after injection to 330 seconds afterward (*$P < .05$, ANOVA), and the highest score (68.0 ± 16.1 mm) was recorded at 60 seconds after injection.

point. The subjects were asked to bring the mandible into a comfortable position and to open their mouth as wide as possible, even if pain would occur. The edge of the slide calipers was placed at the incisal edge of the right maxillary central incisor, and mouth opening was measured vertically to the labioincisal edge of the opposing mandibular incisor. Measurements were performed every 30 seconds from immediately before injection to 600 seconds postinjection. After completion of all measurements, the subjects were asked to make drawings of the distribution of their pain on a picture of a face[37] (Fig 1). Afterward, a plastic template divided into zones (Fig 1) based upon "referred pain areas" described for the trapezius muscle,[2] the superficial sensory distribution of the trigeminal and upper cervical nerves,[38] and the usually reported distribution of TMD complaints,[39,40] was superimposed on the same picture. The total number of zones covered by the subject's pain drawings was counted to quantify the distribution.

Statistical Analysis

Descriptive statistics were used to summarize the VAS values and the range of mouth opening. A 2-way repeated-measured analysis of variance (ANOVA) (Tukey method) was applied to test the difference of the means ± SD between CT and EX conditions over time. Pearson's correlation test was used to analyze the relation between the maximum pain intensity and the amount of pain spread (ie, total number of zones on the facial map). The level of statistical significance was set at $P < .05$. All analysis was conducted using SPSS.

Results

Time-courses of Changes of VAS for Pain Intensity

The time-courses of changes of pain intensity in CT and EX conditions after the injection of saline are shown in Fig 2. In EX, the pain intensity

Table 2 Spread of Pain After Injection of Hypertonic Saline in the Left Trapezius Muscle

Zone	Subject												Total (per zone)
	A	B	C	D	E	F	G	H	I	J	K	L	
1. Parietal region													0
2. Frontal region						X							1
3. Anterior temple						X					X	X	3
4. Posterior temple						X					X		2
5. Occipital zone	X	X							X				3
6. Preauricular zone	X												1
7. Auricular zone													0
8. Retro-auricular zone	X	X	X		X			X					5
9. Mandibular angle	X								X				2
10. Infra-auricular zone	X	X						X	X		X	X	6
11. Submandibular zone	X	X						X	X				4
12. Posterolateral part of neck	X	X	X		X	X		X	X	X	X	X	10
Total mean (per person)	7	5	2	0	2	4	0	4	5	1	4	3	3.1 ± 2.2

X = the zones covered by the subject's pain drawings.
See Fig 1 for the locations of the zones.

increased immediately after the injection and gradually decreased after reaching a peak of 68.0 ± 16.1 mm at 60 seconds after injection. In CT, the intensity also reached its peak (16.8 ± 12.0 mm) at 60 seconds after injection and slowly decreased afterward. The VAS scores in the EX condition were significantly higher than those in the CT condition in the time interval between 30 to 330 seconds after injection ($P < .05$, ANOVA). The perception of pain intensity varied greatly among the subjects, as reported pain intensity peaks ranged from 42.9 mm to 89.8 mm.

Pain Distribution Zone

The distribution and the total of pain zones noted on the anatomical maps after hypertonic saline injection are shown in Table 2. The zones of pain most often mentioned were the posterolateral part of the neck (n = 10), infra-auricular zone (n = 6), retro-auricular zone (n = 5), submandibular zone (n = 4), occipital zone (n = 3), and anterior temple (n = 3). Three of the subjects felt pain in 5 or more zones, and 2 felt no pain at all in the orofacial region. Consequently, pain appeared to spread frequently into the region of the temporomandibular joint.

Correlation Analyses

The correlation between the maximum pain intensity following injection of hypertonic saline and the total number of zones into which the pain spread in each subject is shown in Fig 3. A significant positive correlation was found between the maximum pain intensity and the total number of pain zones (Pearson's correlation test, r = 0.669, $P = .0174$). There was no significant correlation between maximum pain intensity and maximum mouth opening before injection, the maximum mouth opening after injection, or the reduction of mouth opening (from before injection to afterward).

Time-courses of Changes of Maximum Mouth Opening

The time-courses of maximal mouth-opening changes in the CT and EX conditions are shown in Fig 4. Maximum mouth opening at baseline was approximately 54 mm in both conditions. In the CT condition, the mouth opening remained stable. In the EX condition, by contrast, it decreased immediately after injection and reached the lowest value of 47.8 ± 5.1 mm. A gradual recovery to normal occurred by approximately 450 seconds.
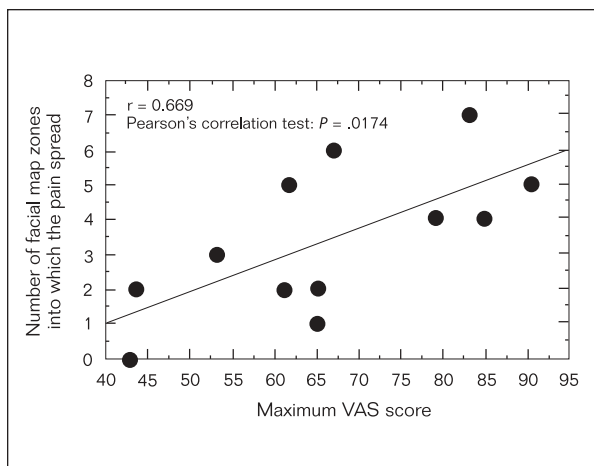
Fig 3 Correlation between the maximum pain intensity (measured on a VAS) following injection of hypertonic saline and the total number of facial map zones to which the pain spread in each subject.

Fig 4 Maximum mouth opening following injection of hypertonic saline (6%, EX condition) and isotonic saline (0.9%, CT condition). Mean values (± SD) are shown for the 12 subjects. The maximum mouth opening at baseline was approximately 54 mm, and was significantly reduced in EX compared with CT from immediately after injection to 60 seconds after (*$P < .05$, ANOVA). The lowest value in EX was 47.8 ± 5.1 mm (immediately after injection).

The reduction of mouth opening in the EX condition was significant compared with the CT condition ($P < .05$, ANOVA) from immediately after the injection until 60 seconds after injection.

Discussion

Distribution of Reported Pain

In the present study, the injection of hypertonic saline into the upper trapezius muscle in 12 normal male subjects induced pain at the base of the neck in 10, in accordance with the findings of Steinbrocker et al.[41] Arendt-Nielsen and Svensson[42] have used the definition "pain felt at a site remote from the site of origin/stimulation" for referred pain. According to their definition, pain referred to the orofacial region (anterior temple) was induced in only 3 of 12 subjects in the present study. The pain distribution presented in the anatomical map should then be considered widely spread local pain. Madeleine et al[34] induced experimental pain by injection of hypertonic saline into the trapezius muscle, and the reported pain intensity was comparable to the present results. However, spreading of pain around the temporo-
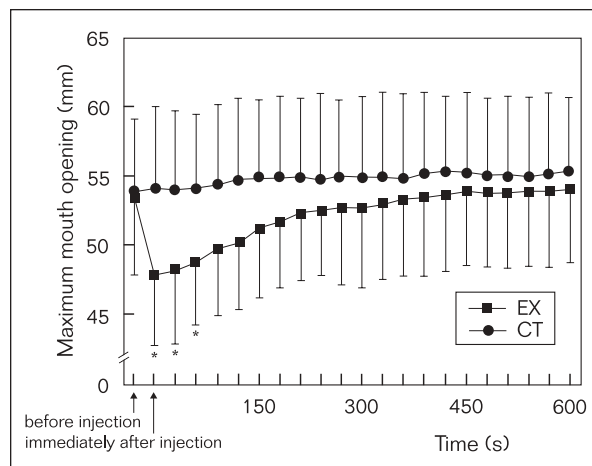
mandibular region was reported more frequently in the present study. One reason for this may be differences in the study designs. Madeleine et al[34] questioned subjects about the pain distribution 5 minutes after injection, at a moment where still a quarter or more of pain intensity remained. Residual pain may have influenced the ability of the subjects to calmly judge the location of pain. In the present study, the subjects were questioned 10 minutes after injection, when pain had almost completely disappeared, and possibly, the subjects could more calmly consider the pain distribution. Another explanation may be that pain spread further in the time interval between from 5 and 10 minutes after injection.

Sessle et al[3] have suggested that referred pain results from the convergence of input from a site subject to irritation, with input from another site without tissue irritation. When information transmitted to the second-order neurons is relayed to the higher brain centers, additional nociception might be signaled by adjacent converging neurons. The higher centers are unable to distinguish the exact site where the noxious stimuli came from and may identify the wrong source. As a result, the somatosensory cortex perceives 2 pain locations or a larger distribution of perceived pain. In the pres-

ent experimental setup, 2 regions of pain were reported: the trapezius region, which represents the true source of nociception, and the orofacial region, which was not an actual source of pain. In some of the subjects of the present study, the irradiation or spread of the pain sensation as indicated on the anatomical map overlapped the region where TMD symptoms usually are reported, which underscores the necessity of differential diagnosis between TMD and neck or shoulder problems.

Pain Intensity

As shown in Fig 2, pain intensity reached its peak within 1 to 2 minutes after injection; the peak mean of 68.0 ± 16.1 mm on the 100-mm VAS is comparable to a previous report.[34] The reported peak pain intensity in the present subjects ranged from 42.9 mm to 89.8 mm. Aspects associated with such a range of pain intensity could be genetic sensitivity to pain and/or psychological factors.[43] In addition, the sites and extent of pain differed greatly among individuals, which might suggest important interindividual variation in convergence at the level of the trigeminal spinal tract nucleus. There was no significant correlation between maximum pain intensity and maximum mouth opening before injection, the maximum mouth opening after injection, or the reduction of mouth opening (from before injection to afterward) (data not shown). The lack of correlation may be the result of the great interindividual variation in mouth opening in symptom-free subjects, and the variation in the subjects' reactions to experimental pain. However, a significant positive correlation was found between the maximum pain intensity and the total number of pain zones (Fig 3). This finding could mean that subjects with high pain sensitivity experience spread of pain to a broader area, illustrating differences regarding convergence or projection systems of pain. Consequently, this might have implications for a different treatment approach of patients with more widespread pain.

Maximum Mouth Opening

The mean maximum mouth opening reported for young males is 55 mm,[44] which is comparable to the present findings. Experimental pain induced a reduction of mean maximum mouth opening from 54 to 47.8 mm, although the injected trapezius muscle is seemingly irrelevant in the control of mouth opening. The amount of mouth opening reduction also has clinical significance in view of

the relevance of Smallest Detectable Difference.[45] The underlying mechanism for this reduction is not clear. Previously, it was suggested that head posture, which is controlled by the trapezius muscle, correlates with mouth opening.[2] Different head postures can lead to EMG changes in the digastric muscle during mouth opening,[46,47] and it is widely accepted that mouth opening reflects the coordination between the suprahyoid and lateral pterygoid muscles.[48,49] Furthermore, Eriksson et al[50] have suggested that functional jaw movements are the result of the activation of jaw as well as neck muscles, leading to simultaneous movements in the temporomandibular, atlanto-occipital, and cervical spine joints. In the subjects of the present study, it is possible that the left trapezius muscle became strained as pain was induced by the injection. This could hypothetically result in sagittal or horizontal imbalance between the muscles that maintain head posture[51] and decrease the possibility of moving the head freely during mouth opening. As a consequence, coordinated activity of the affected muscles with the surrounding muscles might be disturbed, resulting in reduced mouth opening.

However, in considering the relation between pain and mouth opening, it should be mentioned that pain itself may affect the masticatory muscle activity by central effects. Afferent nociceptive input from the head or neck muscles may excite efferent (motor) neurons, resulting in co-contraction of masticatory muscles,[4–9] which also may explain the observed reduction of mouth opening. The influence of pain as such on mouth opening limitation, regardless of the source of pain, could not be evaluated in the present study, since experimental pain at a remote site was not induced.

Limitation of the Study

The major limitation of the present study is the selective use of male subjects. Since the authors were not fully aware of the effect of injecting hypertonic saline in the trapezius muscle, and, in a pilot experiment, saw short-term but serious pain reactions in a female volunteer, it was decided to limit the study to male subjects. This also eliminated possible female hormone–related influences or fluctuations in pain sensitivity and/or pain report.[52,53] However, chronic myofascial pain is much more common in females compared to males. Consequently, future studies should also include female subjects, and the gender-related confounding factors mentioned previously will need to be taken into account.

Conclusions

The present results suggest that experimental trapezius muscle pain spreads to the posterior neck and posterior temporal zone in most subjects and produces referred pain in the temporomandibular region of some subjects. In addition, trapezius experimental pain was accompanied by a reduction of mouth opening. Future studies need to focus on gender differences, interindividual differences, psychological factors involved in the perception of referred pain, the effect of remote pain on mouth opening, and the interaction of neck and shoulder muscles with the stomatognathic system.

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