The Influence of Deep (Odontogenic) Pain Intensity, Quality, and Duration on the Incidence and Characteristics of Referred Orofacial Pain

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This study examined the effects of the intensity, quality, and duration of odotogenic pain on the incidence, pattern, and clinical characteristics of pain referral in the orofacial region. Four hundred consecutive patients reporting with posterior toothache to the dental emergency clinic were included. Patients completed a standardized clinical questionnaire consisting of a numerical rating scale for pain intensity and chose verbal descriptors from a list of adjectives describing the quality of their pain. In addition, patients indicated sites to which pain referred by drawing on a mannequin of the head and neck. Pain intensity was found to significantly affect the presence of referred pain ($P < .005$). However, neither duration nor quality of pain influenced the incidence of referred pain. Finally, pain referral occurred in vertical laminations as indicated on mannequin drawings, but these were not found to be diagnostic because of extensive horizontal overlap. The association of intensity and referral is attributed to central nervous system hyperexitability causing expansion of receptive fields and spread and referral of pain.
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key words: toothache, odontogenic pain, referred pain, deep pain

Referred pain is felt in an area distant from the actual source of the pain and is commonly observed in orofacial pain conditions including toothache.[1–3] Toothache is a highly prevalent orofacial pain condition and is manifest in a wide array of clinical pain patterns.[4] Referred pain from toothache may obscure accurate identification of the offending tooth. For example, in the presence of pulpal pathology, patients may complain not only of pain in the offending tooth, but they may also report widely dispersed pain in other orofacial structures.[5–8]

The precise mechanisms of referred pain are not definitively known, but it is thought that convergence of afferent neurons is an important factor.[1,9] According to this theory, afferent neurons from different anatomic regions converge and synapse on a common pool of secondary neurons in the medullary dorsal horn.[10] Nociceptive impulses are transmitted from the periphery, converging on fewer and fewer common neurons, ultimately stimulating an area of the medullary dorsal horn and/or higher central nervous system structures. Thus, nociceptive input may be perceived by the cortex as arising from any one of several of the neighboring peripheral regions innervated by the primary afferent neurons. Thus, convergence of afferent nociceptive neurons in the trigemi-

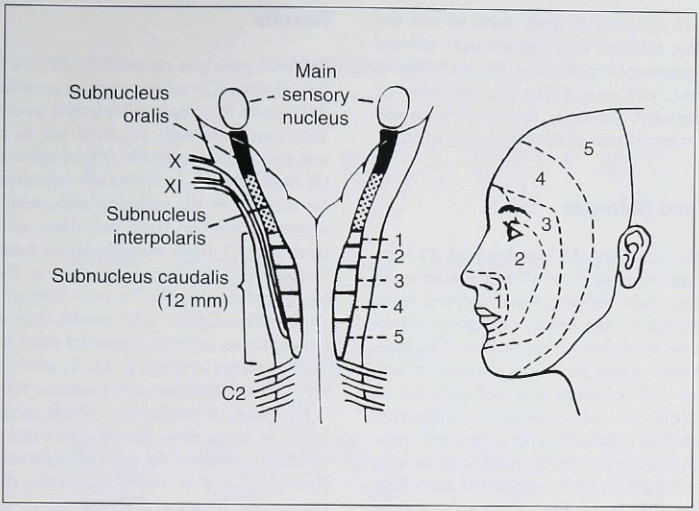

Fig 1 Rostrocaudal somatotopic organization of the subnucleus caudalis *(left)*. Corresponding relationship of the subnucleus caudalis to cutaneous input from primary sensory afferents on the face *(right)*. (Reprinted with permission from Bonica JJ. The Management of Pain, vol 1, ed 2. Philadelphia: Lea & Febiger, 1990.)

nal sensory complex has been implicated as a reason for poor localization of deep orofacial pain.

In addition to convergence, spatial and temporal summation are important in producing referred pain, which suggests that the intensity of the stimulus influences the scope of referred pain.[11] Overall, a strong nociceptive barrage is thought to contribute to central nervous system hyperexcitability, which may be responsible for spread and referral of pain outside of normal dermatomal distributions.[1,12]

Another mechanism reported to result in the referral of pain has recently been described.[13,14] After acute noxious stimulation of deep tissues, dorsal horn nociceptive neurons may actually develop expanded receptive fields. Moreover, these neurons may also change in regard to the character of the stimulation necessary to affect their discharge. These alterations may reflect rapid changes occurring in the central nervous system in response to noxious stimulation and may influence the extent and character of referred pain states.[15]

Orofacial afferent neurons synapse in the trigeminal sensory nucleus, which is the medullary analog of the spinal dorsal horn with which it is contiguous.[9] The trigeminal nucleus is subdivided rostralcaudally into subnucleus oralis, interpolaris, and caudalis, with the majority of afferent nociceptive neurons synapsing on cell bodies located in the nucleus caudalis.[9] The presence of a somatotopic or layered vertical arrangement of cell bodies in nucleus caudalis has been reported, whereby cutaneous stimulation of specific orofacial areas results in neuronal activity in corresponding areas of the nucleus caudalis.[16] For example, sensory innervation near the midline around the mouth and nose is represented by cells in the most rostral part of the nucleus, whereas the innervation of more posterior regions of the face and jaws ends progressively in more caudal parts of the nucleus. This results in a so-called *onion peel* distribution of sensory afferents (Fig 1).

Although the somatotopic organization of the nucleus caudalis is based on studies of cutaneous orofacial stimuli, extensive neuronal convergence from deep and superficial orofacial tissues on medullary dorsal horn neurons has been demonstrated.[14] Therefore, it was hypothesized that patterns of pain referral from teeth would correspond to the vertical segments described above and noted in Fig 1.

The aims of the present study were to test the hypothesis that referred odontogenic pain follows a vertical segmentation pattern in the teeth, jaws, head, and face, and to examine the influence of toothache intensity, duration, and quality on the incidence of referred pain in the subjects studied.

Materials and Methods

Four hundred patients (183 males and 217 females) with ages ranging from 17 to 73 years were included in the study. Subjects were recruited from patients reporting to the dental emergency clinic with complaint of posterior toothache. The incidence of anterior toothache in this population was relatively low and therefore was not included in this study. Inclusion criteria were *(1)* subjective report of posterior toothache and *(2)* clinical verification of pulpal or periapical pathology in one tooth to which pain could be attributed according to criteria described by Cailleteau.[17] Exclusion criteria were pain attributed to *(1)* anterior teeth, *(2)* involvement of multiple posterior teeth, *(3)* gingival tissues, and *(4)* nonodontogenic sources such as sinusitis, temporomandibular disorders, or salivary gland pathology. All examinations were conducted by the principal author.

Prior to examination, a clinic questionnaire regarding demographics, health history, and duration and timing of toothache were completed. Patients were asked to describe the nature and quality of their pain by selecting from a list of pain descriptors adapted from the McGill Pain Questionnaire. Pain intensity was measured by having patients circle the appropriate number on a numerical rating scale ranging from 0 to 10 in whole integer units, anchored on each end by the words "no pain" and "extreme pain," respectively. In addition, subjects circled painful intraoral areas on an illustration of the mouth and teeth and outlined any areas of pain felt in extraoral facial, neck, or head areas by drawing on a mannequin.

The chi square goodness-of-fit test was applied to test the degree of association between the presence of referred pain and each of the variables of gender, duration of pain, and specific verbal descriptors of pain. The mean score on the numerical rating scale for pain intensity was compared between those patients with referred pain and those without, using a two-sample t test. Finally, composite diagrams were constructed from patient drawings depicting the pain referral pattern to the face, head, and neck for each posterior tooth.

Results

Referred pain was reported by 89.8% (n = 359) of the subjects. Chi square analysis revealed no gender differences in report of referred pain (P = .283). The most commonly reported site of pain referral was to adjacent teeth (80.0% of subjects, n = 287). Of those, 24.0% (n = 69) also reported pain referral to teeth in the opposite arch, with 42 subjects reporting referral from maxillary to mandibular teeth and 27 from mandibular to maxillary teeth. Extraoral pain referral occurred in the jaw, face, head, and neck in 74.7% (n = 268) of the subjects with referred pain. The results demonstrate that pain tends to refer from painful teeth in broad vertical segments as noted in Fig 2, where referral patterns are outlined for each posterior tooth studied.

Duration of toothache, which ranged from 24 hours to more than 30 days, was not significantly related to whether the patient experienced referred pain or not (P = .069). Similarly, there was no association between the presence of referred pain and the use of particular verbal descriptors of the quality of pain (P = .233). The two-sample t test indicated a significantly higher mean pain intensity score among those subjects with referred pain, compared with those without referred pain (P < .005) (Fig 3). The mean pain intensity for the referred pain group was 7.2, compared with a mean of 6.1 for those subjects without referred pain.

Discussion

The results indicate that pain severity is the most reliable predictor of referred pain, and they support earlier findings suggesting that stimulus intensity influences pain referral.[11] Additionally, our observations confirm those of a recent study[18] of the effects of injection of an algogenic substance in the human temporalis muscles. This recent study demonstrated that with increasing intensity of nociceptive barrage, pain referral was more likely to occur. Our findings are also consistent with those of Stohler and Lund,[19] who demonstrated that with increasing intensity of acute and tonic experimental noxious stimuli, pain radiated from the site of stimulation to distant ipsilateral areas of the face. Finally, our observations confirm those of Wolff,[3] who reported that experimental tooth pain in humans results in widely dispersed referral in the head and neck.

The relationship between intensity of pain and the tendency for referral is at least in part attri-

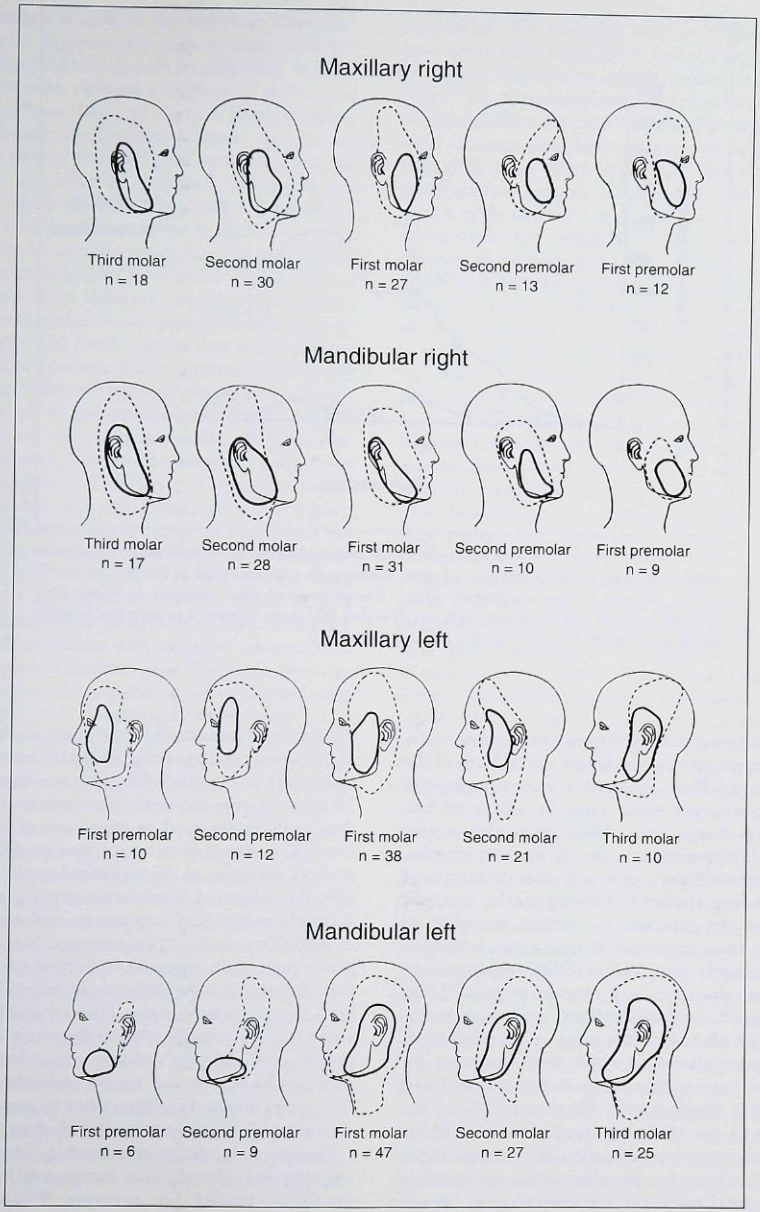

Fig 2 Extraoral referral patterns from maxillary and mandibular teeth on the right side and left side. The solid lines outline the most commonly reported pattern of pain referral, and the dotted lines outline the less commonly reported reference areas.

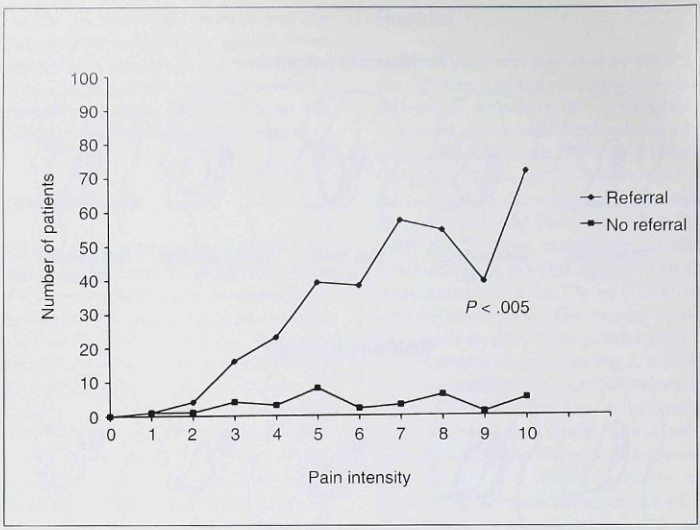

Fig 3 Number of patients who either did or did not report referral of pain plotted against a numerical rating scale for intensity of pain. Rating scale was anchored at 0 with "no pain" and at 10 with "worst imaginable pain." Comparison of pain intensity in those who reported referral and those who did not revealed that pain referral was reported significantly more often ($P > .005$) as pain intensity increased.

butable to extensive convergence of primary orofacial afferents into the medullary dorsal horn of the spinal cord.[9] Thus, nociceptive input from painful teeth converges onto specific areas of the medullary dorsal horn. These areas also receive sensory information from the masticatory muscles, temporomandibular joints, and other orofacial and surrounding tissues.[10] Consequently, noxious input may be perceived by cortical structures to originate from any or all of those areas, leading to the commonly observed inability of patients to accurately localize deep sources of pain.[20] The convergence of primary afferent neurons onto second-order neurons is the basis of the projection-convergence theory,[21] which would account for referred pain within normal dermatomal and myotomal distributions. However, 75% of the subjects in our study reported pain in extradermatomal and extramyotomal distributions. Possible explanations for this observation are found in experimental evidence indicating that an intense nociceptive barrage results in central nervous system hyperexcitability, which in turn may be responsible for extradermatomal spread and referral of pain in humans.[12,22] In a study of the effects of pulpotomy on cat teeth, Hu et al[23] reported that alterations in nucleus oralis neurons lasted up to 15 days. During the initial postoperative period, these authors observed an expansion of the receptive field in dorsal horn nociceptors to include two or three divisions of the trigeminal nerve. In addition, they observed spontaneous activity as well as a rapidly habituating response to orofacial stimuli in medullary dorsal horn neurons. Similar findings[22] have been reported in human studies and may account for the widespread referral of pain seen in our study sample. These reports demonstrate why the identification of the actual source of pain in orofacial pain conditions may be clinically difficult. Moreover, our results highlight the need for careful diagnostic efforts prior to institution of treatment for painful orofacial pathology.

Despite a significant relationship between pain intensity and referral, other factors may have influenced our results. For example, Wilson et al[24] examined patients with another orofacial pain condition, temporomandibular disorders, and reported that high levels of somatization, as measured by

the somatization scale of the Symptom Checklist-90-R,[25] plus high-intensity pain, strongly predicted widely dispersed muscle pain on palpation. In addition, those with elevated somatization scales were three times more likely to report a painful placebo site. Although the findings of these authors are based on *chronic* orofacial pain conditions, it is not known if somatization may have influenced our findings, which are based on reports of tooth pain ranging from hours to months. In addition, we did not control for anxiety, which has also been shown to influence reactions to painful stimuli.[26] In a comparison of the influence of acute versus chronic noxious stimulation of the masseter muscle on anxiety, Stohler and Lund[19] found that acute stimulation produced no significant elevations, while tonic noxious stimulation produced elevations in anxiety in two thirds of the studied sample. The authors attributed the difference in anxiety scores of subjects with tonic pain to the subjects' inability to predict the level and duration of pain they would experience once the tonic stimulation had begun. This is in contrast to the rapid escalation and decrease in intensity experienced with an acute stimulus. Thus, it is reasonable to predict that the tonic stimulation in our study cohort, which ranged from 1 to more than 30 days, may have caused elevated levels of anxiety with unknown consequences on the report of referred pain. Therefore, the relationship of anxiety and the report of referred pain from painful teeth remains to be investigated.

To our surprise, the duration of toothache did not influence the presence of referred pain in this study, which is in disagreement with the prevailing theory that central nervous system hyperexcitability and spread and referral of orofacial pain is influenced by the duration of nociceptive input.[27] Hu et al[23] examined brainstem neurons subsequent to pulpotomy in cat teeth and observed a rapidly habituating response to orofacial stimuli. These changes in neuronal properties were highly significant, but only in the immediate postoperative period. Thus, prolonged nociceptive discharge from painful teeth at relatively low or variable intensities may result in a similar habituation of dorsal horn neurons. Alternatively, other intrinsic modulating influences such as central opioid depression[28,29] may vary among individuals such that central nervous system hyperexcitability may be of short duration in some but prolonged in others, leading to widespread variation in clinical reports of pain referral.

Acute versus chronic orofacial pain conditions may be distinguished by asking patients to identify the quality of their pain, using standardized verbal descriptors. For example, acute and chronic pain associated with masticatory musculature is most frequently described as aching, tender, and throbbing.[19] The most commonly used descriptors in the current study were also "throbbing" and "aching," although these were not found to be used significantly more often than other descriptors in those with or without referred pain. Our subjects used a wide array of verbal descriptors to characterize their pain, regardless of the presence or absence of referred pain. The tendency to select multiple verbal descriptors is consistent with other reports in acute and chronic orofacial pain in both the experimental and the clinical settings.[19] Thus, it appears that deep nociceptive input in the orofacial region is often described as aching or throbbing, regardless of the offending tissues, the duration of the pain, or the presence or absence of pain referral. Future studies should employ the use of standardized measurements of the sensory and affective dimensions of pain such as the McGill Pain Questionnaire[30] to examine possible relationships among those dimensions of pain and report of referred pain.

Figure 2 illustrates a distribution of vertical referral of pain with considerable horizontal overlap, which is consistent with observations in other orofacial pain conditions.[19,31] Despite similarities in referral patterns between the present study and those in experimental studies,[19] the referral of pain reported in the present study was more inclusive, involving not only the ipsilateral dental arches, jaws, and face, but large areas of the ipsilateral head and neck as well. Thus, the present study supports the hypothesis that referred odontogenic pain (deep pain) generally follows a vertical distribution consistent with an onion skin or lamination pattern reported in cutaneous orofacial tissues,[32] but with significant overlap in horizontal and vertical directions. This expansive overlap illustrates the improbability of accurately identifying the actual source of pain based on referral patterns. In conclusion, it appears that a focus of orofacial nociceptive input (eg, toothache) can produce widespread referral of pain to a variety of ipsilateral sites in the head and neck region consistent with those found in experimental studies of tooth pulp stimulation.

Our conclusions should be interpreted in light of the fact that we did not include a control or comparison group with which to compare our findings. For example, it would be instructive to compare the referral patterns of patients with acute and chronically painful sinusitis to determine the extent to which pain referral varies with other orofacial diseases. However, comparison of the results

of other studies involving experimental and clinical cohorts reveals similar patterns of pain referral, suggesting that our data is representative of referral patterns seen in other masticatory pain conditions.[3,19,31] In addition, the influence of medications taken by our study cohort may have altered our findings such that intensity and perhaps duration of pain were diminished, with possible effects on sensory-discriminative as well as cognitive and affective reports of pain. Finally, future studies should employ standardized and validated measures of pain assessment to avoid incorporating investigator bias. In the present study, patients were asked to characterize their pain using a clinically useful but limited list of verbal descriptors chosen by the investigator, restricting patient choices, perhaps resulting in response bias.

References

1. Fields HL. Pain. New York: McGraw-Hill, 1987:82–94.
2. Fricton JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: A review of clinical characteristics of 164 patients. Oral Surg Oral Med Oral Pathol 1985;60:615–623.
3. Wolff HG. Headache and Other Head Pain. New York: Oxford Press, 1948:29–30.
4. Lipton JA, Ship J, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. J Am Dent Assoc 1993;124:115–121.
5. Robertson S, Goodell H, Wolff HG. The teeth as a source of headache and other pain. Arch Neurol Psychiatry 1947; 57:277–283.
6. Howell FV. The teeth and jaws as sources of headache and facial pain. In: Dalessio DJ (ed). Wolff's Headache and Other Head Pain, ed 5. New York: Oxford Press, 1987:255–65.
7. Hutchins HC, Reynolds OE. Experimental investigation of the referred pain of aerodontalgia. J Dent Res 1947;26:3–8.
8. Glick DH. Locating referred pulpal pains. Oral Surg 1962; 15:613–626.
9. Sessle BJ. The neurobiology of facial and dental pain. J Dent Res 1987;66:962–981.
10. Sessle BJ, Hu JW, Amano N, Zhong G. Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and non-nociceptive neurones in trigeminal subnucleus caudalis (medullary dorsal horn) and its implications for referred pain. Pain 1986;27:219–235.
11. Torebjork HE, Ochoa JL, Schady W. Referred pain from intraneural stimulation of muscle fascicles in the median nerve. Pain 1984;18:145–156.
12. Dubner R. Neuronal plasticity and pain following peripheral tissue inflammation or nerve injury. In: Bond MR, Charlton JE, Woolf CJ (eds). Pain Research and Clinical Management, vol 4 [Proceedings of the VIth World Congress on Pain, 1990, Amsterdam, The Netherlands]. Amsterdam, The Netherlands: Elsevier, 1991:263–276.
13. Hoheisel U, Mense S. Long-term changes in discharge behavior of cat dorsal horn neurones following noxious stimulation of deep tissues. Pain 1989;36:239–247.
14. Hu JW, Sessle BJ, Raboisson P, Dallel R, Woda A. Stimulation of craniofacial muscle afferents induces prolonged facilitatory effects in trigeminal nociceptive brainstem neurones. Pain 1992;48:53–60.
15. Mense S. Nociception from skeletal muscle in relation to clinical muscle pain. Pain 1993;54:241–289.
16. Bonica JJ. General considerations of acute pain. In: Bonica JJ (ed). The Management of Pain, vol 1, ed 2. Philadelphia: Lea and Febiger, 1990:159–179.
17. Cailleteau JG. Diagnosis and management of toothaches of dental origin. In: Falace D (ed). Emergency Dental Care. Baltimore, MD: Williams and Wilkins, 1995:25–66.
18. Jensen K, Norup M. Experimental pain in human temporalis muscle induced by hypertonic saline, potassium, and acidity. Cephalalgia 1992;12:101–106.
19. Stohler CS, Lund JP. Effects of noxious stimulation of the jaw muscles on the sensory experience of volunteer human subjects. In: Stohler CS, Carlson DS (eds). Biological and Psychological Aspects of Orofacial Pain, vol 29. Craniofacial Growth Series. Ann Arbor, MI: Univ of MI, 1994:55–74.
20. Dubner R. Introductory remarks: Basic mechanisms of pain associated with deep tissues. Can J Physiol Pharmacol 1991;69:607–609.
21. Ruch TC. Visceral sensation and referred pain. In: Fulton JF (ed). Textbook of Physiology. Philadelphia: Saunders, 1946:385–401.
22. Coderre TJ, Katz J, Vaccarino AL, Melzack R. Contribution of central neuroplasticity to pathological pain: Review of clinical and experimental evidence. Pain 1993; 52:259–285.
23. Hu JW, Dostrovsky J, Lenz Y, Ball G, Sessle BJ. Tooth pulp deafferentation is associated with functional alterations in the properties of neurons in the trigeminal spinal tract nucleus. J Neurophysiol 1986;16:50–68.
24. Wilson L, Dworkin SF, Whitney C, LeResche L. Somatization and pain dispersion in chronic temporomandibular disorder pain. Pain 1994;57:55–61.
25. Derogatis LR. SCL-90-R: Administration, Scoring and Test Procedures Manual-II for the Revised Version. Towson, MD: Clinical Psychometric Research, 1983.
26. Peck K. Psychological factors in acute pain management. In: Cousins MJ, Phillips BD (eds). Acute Pain Management. Edinburgh: Churchill Livingstone, 1986.
27. Okeson JP. The central processing of pain. In: Okeson JP (ed). Bell's Orofacial Pains, ed 5. Chicago: Quintessence, 1995:65.
28. Sessle BJ, Hu JW, Yu X-M. Brainstem mechanisms of referred pain and hyperalgesia in the orofacial and temporomandibular region. In: Vecchiet D, Albe-Fessard, Lindblom U (eds). New Trends in Referred Pain and Hyperalgsia, Pain Research and Clinical Management, vol 7. Amsterdam, The Netherlands, 1993:59–71.
29. Chiang CY, Hu JW, Sessle BJ. Parabrachial area and nucleus raphe magnus induced modulation of nociceptive and non-nocicepetive trigeminal subnucleus caudalis neurons activated by cutaneous or deep inputs. J Neurophysiol 1994;71:2430–2445.
30. Melzack R. The McGill Pain Questionnaire: Major properties and scoring methods. Pain 1975;1:277–299.
31. Travell JG, Simons DG. Myofascial Pain and Dysfunction. The Trigger Point Manual, vol 1. Baltimore: Williams and Wilkins, 1983.
32. Kunc Z. Significance of fresh anantiomeric data on spinal trigeminal tract for possibility of selective tractotomies. In: Knighton RS, Dumke PR (eds). Pain. Boston: Little, Brown, 1966:351–366.

Resumen

La Influencia de la Intensidad, Calidad y Duración del Dolor Profundo (Odontogénico) en la Incidencia y Características del Dolor Orofacial Referido

Este estudio examina los efectos de la intensidad, calidad y duración del dolor odontogénico en la incidencia, patrón y características clínicas del dolor referido en la región orofacial. Se incluyeron 400 pacientes consecutivos de una clínica odontológica de emergencia que presentaban odontalgias en las partes posteriores. Los pacientes completaron un cuestionario clínico estandarizado que consistía de escalas de registro numérico para la intensidad del dolor, a la vez que permitía seleccionar palabras de una lista de adjetivos que describían la calidad del dolor. Además, los pacientes indicaban los sitios a donde el dolor se refería, dibujando sobre un maniquí que correspondía a la cabeza y cuello. Se encontró que la intensidad afectaba significativamente la presencia del dolor referido ($P < 0,005$). Sin embargo, ni la duración ni la calidad del dolor influenciaron la incidencia del dolor referido. Finalmente, el dolor referido ocurría en forma de laminados verticales como se indicó sobre los dibujos de los maniquíes, pero no tenían un valor diagnóstico debido a superposiciones horizontales extensas. La asociación entre la intensidad y la referencia del dolor es atribuida a la hiperexcitabilidad del sistema nervioso central lo que causa una expansión de los campos receptivos lo mismo que la diseminación del dolor.

Zusammenfassung

Der Einfluss von tiefer (odontogener) Schmerzintensität, -qualität und -dauer auf die Inzidenz und Eigenschaften von übertragenem orofazialem Schmerz

Diese Studie untersuchte die Effekte der Intensität, Qualität und Dauer von odontogenem Schmerz auf die Inzidenz, das Muster und die klinischen Eigenschaften von Schmerzübertragung in der orofazialen Region. Es wurden 400 aufeinanderfolgende Patienten einer zahnärztlichen Notfallklinik mit Zahnschmerzen im Seitenzahngebiet untersucht. Die Patienten füllten einen standardisierten Fragebogen aus, welcher eine VAS (Visual analog scale) für die Schmerzintensität enthielt, sowie eine verbale Beschreibung des Schmerzes (Auswahl aus einer Liste von Adjektiven) für die Schmerzqualität. Zusätzlich mussten die Patienten Regionen von Schmerzausstrahlung (übertragener Schmerz) auf einer Abbildung des Kopf/Hals-Bereichs einzeichnen. Es zeigte sich, dass die Schmerzintensität eine signifikante Auswirkung auf das Auftreten von übertragenem Schmerz hatte ($P < 0.005$). Schmerzdauer und -qualität beeinflussten aber das Auftreten von übertragenem Schmerz nicht. Die Schmerzübertragung geschah in vertikalen Schichten wie auf den Zeichnungen angegeben, hatten aber keinen diagnostischen Wert wegen grosser horizontaler Überlappung. Die Beziehung zwischen Intensität und Schmerzübertragung wird einer zentralnervösen Hypererregbarkeit zugeschrieben, welche eine Expansion rezeptiver Felder und die Ausbreitung und Übertragung von Schmerzen verursacht.