Thermographic Assessment of Neuropathic Facial Pain

Steven B. Graff-Radford, DDS
Assistant Research Dentist
Section of Orofacial Pain and Occlusion
University of California, Los Angeles
School of Dentistry
Los Angeles, California
and Director of Head and Neck Section
The Pain Center
Cedars-Sinai Medical Center
Los Angeles, California

Marie-Christine Ketelaer, LSD
Postdoctoral Resident
Section of Orofacial Pain and Occlusion

Barton M. Gratt, DDS
Professor
Section of Oral Radiology

William K. Solberg, DDS, MS
Professor
Section of Orofacial Pain and Occlusion

University of California, Los Angeles
School of Dentistry
Los Angeles, California

Correspondence to:
Dr Steven B. Graff-Radford
Section of Orofacial Pain and Occlusion
43-009 University of California, Los Angeles
School of Dentistry
10833 Le Conte Avenue
Los Angeles, California 90024–1668

Ongoing pain, intermittent sharp pain, or intermittent dull aching pain around the teeth can evoke the suspicion of tooth pathology. However, when no dental cause can be found clinically or radiographically, the differential diagnosis involving neuropathic pain and pulpal pathology is still a challenge. Neuropathic facial pains are still too often misdiagnosed as tooth pain of dental origin, resulting in unnecessary dental extraction or endodontic therapy. The purpose of this study was to determine if electronic thermography was able to differentiate neuropathic facial pains presenting as toothache from pulpal pathology. Electronic thermography was used to compare asymptomatic subjects and subjects with neuropathic facial pains. Asymptomatic subjects and subjects with trigeminal neuralgia, pre–trigeminal neuralgia, and pulpal pain without periapical pathology showed no thermographic difference in the territory of the pain complaint when compared to the opposite nonpainful side. Patients with sympathetically maintained traumatic trigeminal neuralgia (atypical odontalgia) and half of the group with sympathetically independent traumatic trigeminal neuralgia presented with "hot" thermograms. The other half of the patients with sympathetically independent traumatic trigeminal neuralgia displayed "cold" thermograms in the area of their pain complaints. Electronic thermography was the least selective test for the group showing "cold" thermogram patterns (80% agreement with the thermographic characterization criteria). These data suggest that electronic thermography may be helpful in differentiating neuropathic pains from pulpal pathology.

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Neuropathic facial pains are often misdiagnosed as tooth pain of dental origin. Despite the lack of physical findings, dental treatments of such "toothaches" sometimes consist of tooth extraction or root canal treatment. Other patients with vague toothaches receive the stigma of "psychogenic pain." The number of persons having possible neuropathic pains has been underscored by the results of a study by Campbell et al,[1] who described approximately 5% of patients undergoing root canal therapy in combination with apical surgery as having persistent pain.

The major differentiation of neuropathic orodental pain from otherwise diagnosable toothache (TA) comes from the clinical history. Indeed, it is not unusual that pulpal testing and other commonly used dental examination techniques fail to clearly establish the diagnosis. Considering this diagnostic dilemma, it would be helpful to have additional testing that shows adequate sensitivity and specificity in differentiating TA from neuropathic pain. Electronic thermography (ET) may be of help in the validation of a physiologic process.[2] Essentially, ET is sensitive to slight changes in

cutaneous temperature and has been proposed as a safe, noninvasive diagnostic test for evaluating damage to the nervous system.[3–8] Thermography has also been used previously as a method for assessing tooth vitality on the basis of the blood supply rather than the nerve supply.[9] Through software applications, variations in levels of heat emission are conveniently documented by ET for temperature patterns of chosen regions of body surface.

Patients with painful neuropathies may have abnormal or asymmetrical skin temperatures in the region of pain or nerve injury. Recent advances in the understanding of neurogenic inflammation, sympathetically maintained pain, and antidromic vasodilatation secondary to nerve trauma suggest that quantitating ET may lead to a better understanding of the mechanism producing the painful symptoms.[7,10–14] The present study used ET to investigate the presence of skin temperature deviations in patients presenting with various neuropathic facial pains. Patients diagnosed with sympathetically maintained traumatic trigeminal neuralgia (SMTN) (judged by the authors to be atypical odontalgia), sympathetically independent traumatic trigeminal neuralgia (SITN), trigeminal neuralgia (TN), pre–trigeminal neuralgia (PTN), and pulpal pain in the absence of periapical pathology (TA) were compared with asymptomatic subjects using ET.

The aim of the present study was to determine if ET is able to differentiate between groups of patients with suspected nonodontogenous toothache from those having overt pulpal pathology, and specifically, to determine if thermography can differentiate pulpitis from SMTN, TN, PTN, and SITN.

Materials and Methods

Fifty-eight consecutive patients presenting with neuropathic pain at the Pain Center, Cedars-Sinai Medical Center, Los Angeles, were studied. They were compared with a control group including 22 adults (greater than 20 years of age) who were selected according to the following criteria: they had a negative health history, defined as good health without history of pain, facial skin problems, and blemishes, and with a negative clinical examination. The toothache group with overt pulpal pathology was selected partly from the Oral Diagnosis Clinic at University of California, Los Angeles, School of Dentistry. The patients in the pain subgroups were diagnosed by the same clinician using the inclusion criteria described previously by Graff-Radford[15] and Gratt et al[8] (Table 1).

Thermography

Electronic thermography was conducted using an Agema 870 thermovision unit (Agema, Secaus, NY) that included an infrared scanner, a control unit, MEDS 1.0 software, and a thermal image computer TIC-8000 linked to a color monitor and coupled to a 35-mm camera with color print film. Room conditions for ET examinations included a draft-free environment (no windows or open doors), with temperature control (ranging from 20°C to 22°C), variable lighting, a patient-positioning chair, a head-positioning device, and a small hand-held electric fan.

Facial Imaging

All subjects were given prethermographic examination instructions according to the recommendations of the Academy of Neuro-Muscular Thermology.[16] Facial thermograms were taken of the 80 subjects using frontal projections, at 0.5°C imaging sensitivity (0.1°C accuracy). Before the examination, each patient's face was cleared of hair (tied back). The face was wiped with a damp cloth and then air dried using a small electric fan. A 15-minute period was allowed for facial temperature equilibration, and a series of facial thermograms was made and stored on computer disk for analysis.

Computer Analysis

Mathematical analysis was made from electronically generated images using an Agema TIC-8000 computer, software, and a color monitor. Built-in computer applications facilitated individual mapping of any zone of the face and allowed comparison with the contralateral side. Absolute temperature measurements of individually selected anatomic zones or regions, mirrored comparisons, and differences in mean temperatures (ΔT values) were facilitated by the advanced anatomically oriented computer software (Agema).

Data Analysis

Analysis determinations of mean values and ΔT values were calculated for each subject. Thermographic imaging was analyzed by one author (BMG), and subjects were assigned to the following categories based on their thermographic patterns:

1. Normal ($0.0°C \leq \Delta T \leq 0.25°C$)
2. Equivocal ($0.26°C \leq \Delta T \leq 0.35°C$)
3. Abnormal ($0.36°C \leq \Delta T$)

Table 1 Inclusion Criteria for SMTN, SITN, TN, PTN, and TA*

SMTN	No obvious local cause
	No abnormality shown on radiograph
	Continuous or almost continuous pain in the tooth or surrounding alveolar structure
	Pain present longer than 4 months
	Associated hyperesthesia
	Somatic block equivocal†
	Sympathetic block decreases pain > 60%
SITN	No obvious local cause
	No abnormality shown on radiograph
	Continuous or almost continuous pain in a tooth or surrounding alveolar structure
	Pain present longer than 4 months
	Associated hyperesthesia
	Somatic block positive†
TN	No obvious local cause
	No abnormality shown on radiograph
	Intermittent sharp or electriclike pain localized at one or more divisions of the trigeminal nerve†
	Pain triggerable by trivial stimuli
	Somatic block positive
PTN	No obvious local cause
	No abnormality shown on radiograph
	Intermittent dull toothache†
	Somatic block positive
	Pain triggerable by trivial stimuli
TA	Obvious local cause†
	No abnormality shown on radiograph
	Continuous or almost continuous pain in the tooth or surrounding alveolar bone
	Somatic block positive

*Graff-Radford[15] and Gratt et al.[8]
†Criteria unique to that subclassification.
SMTN = sympathetically maintained traumatic trigeminal neuralgia; SITN = sympathetically independent traumatic trigeminal neuralgia; TN = trigeminal neuralgia; PTN = pre–trigeminal neuralgia; TA = pulpitis.

The percentage of agreement with thermographic characterization for normal and abnormal thermograms was calculated for each group.

Results

The control subjects and subjects with TA, PTN, and TN showed no significant thermographic difference in the field of the pain complaint when the painful area of the skin was compared with the opposite control area (Tables 2 to 4). Patients with SMTN (Table 5) and half of the SITN group (Tables 6 and 7) presented with abnormal "hot" thermograms with a 100% agreement with our thermographic criteria. Electronic thermography was the least selective test (80%) to detect cold thermograms in the group of patients with SITN. Three of 15 subjects in the cold SITN subgroup presented with an equivocal thermogram (Table 7).

Discussion

Our findings suggest that ET is helpful in the differential diagnosis of neuropathic facial pains from toothache of pulpal origin where no periapical lesion is found. Our results show that abnormal thermographic image helps to differentiate SMTN and SITN from TA. However, ET did not aid in the differentiation of PTN and TN from TA. Further, the SITN group is unique among the groups investigated because subjects with SITN displayed better hot or cold ET patterns. It is noteworthy that all patients in the SMTN and SITN categories had abnormal thermograms, and the difference from thermal normalcy appears to be almost 1°C. The relevance of this finding must be explored in further studies (Table 8).

We assume that the mechanism for hyperalgesia and focal pain are different for those patients displaying hot versus cold thermal emission. One

Table 2 Thermography Results for Subjects With TN

Sex	Age (years)	Pain duration (months)	Location	Affected (°C)	Unaff (°C)	ΔT (°C)	ET pattern
M	58	16	R cheek	34.4	34.6	–0.2	normal
M	65	64	Unilat face	33.8	33.9	–0.1	normal
F	53	36	R V3	33.9	34	–0.1	normal
F	49	24	L V2	31.8	31.8	0.0	normal
M	76	36	R V3	34.1	34.2	–0.1	normal
M	52	75	R V3	33.8	34.0	–0.2	normal
Mean	59	41.8		33.6	33.8	–0.1	normal
SD	10	23		0.9	1.0	0.1	

R = right; L = left; V2 = dermatomal zone of the second division of the trigeminal nerve; V3 = dermatomal zone of the third division of the trigeminal nerve.

Table 3 Thermography Results for Subjects With TA

Sex	Age (years)	Pain duration (months)	Location	Affected (°C)	Unaff (°C)	ΔT (°C)	ET pattern
F	56	6	R V2	32.5	2.3	+0.2	normal
F	30	1	Tooth 17	33.9	34.0	–0.1	normal
F	60	3	Tooth 18	33.5	33.5	0.0	normal
F	22	3	Tooth 19	33.8	33.6	+0.2	normal
M	33	5	Tooth 12	33.0	33.0	0.0	normal
M	53	2	Tooth 14	34.5	34.5	0.0	normal
M	40	1	Tooth 14	35.0	35.3	–0.3	normal
F	47	30	R jaw (not chin)	33.3	33.5	–0.2	normal
F	45	19	Tooth 5	36.5	36.5	0.0	normal
Mean	42.9	7.7		34.0	34.0	0.0	normal
SD	12.73	10.0		1.2	1.3	0.2	

R = right; V2 = dermatomal zone of the second division of the trigeminal nerve.

Table 4 Thermography Results for Subjects With PTN

Sex	Age (years)	Pain duration (months)	Location	Affected (°C)	Unaff (°C)	ΔT (°C)	ET pattern
F	45	27	L V2	32.1	32.0	+0.1	normal
M	66	11	L V2	32.8	32.9	–0.1	normal
F	43	31	R V2	33.0	33.0	0.0	normal
F	48	20	R V3	32.1	32.1	0.0	normal
Mean	22.3	51		32.5	32.5	0.0	normal
SD	11	8.8		0.50	0.52	0.08	

L = left; R = right; V2 = dermatomal zone of the second division of the trigeminal nerve; V3 = dermatomal zone of the third division of the trigeminal nerve.

Table 5 Thermography Results for Subjects With SMTN

Sex	Age (years)	Pain duration (months)	Location	Affected (°C)	Unaff (°C)	ΔT (°C)	ET pattern
M	61	12	R chin	35.3	34.9	+0.4	hot
F	60	6	L chin	34.9	34.5	+0.4	hot
F	72	18	L V2, V3,	36.3	34.2	+2.1	hot
			L neck	35.5	34.7	+0.8	hot
F	35	60	R V3	35.3	34.7	+0.6	hot
F	37	60	R ant V2	35.3	34.7	+0.6	hot
F	81	360	R ant V2,	35.5	33.3	+2.2	hot
			R V3	35.1	34.2	+0.9	hot
F	54	12	L chin	34.0	33.3	+0.7	hot
F	66	5	L V2,	36.1	33.0	+3.1	hot
			L neck	35.9	33.9	+2.0	hot
F	74	12	R ant V2	32.9	31.5	+1.4	hot
			R chin	33.3	32.6	+0.7	hot
F	44	180	Mixed	33.9	33.4	+0.5	hot
			Bilateral	33.7	33.2	+0.5	hot
Mean	58	72.5		34.9	33.7	1.1	hot
SD	16	114.2		1.0	1.0	0.8	

R = right; L = left; V2 = dermatomal zone of the second division of the trigeminal nerve; V3 = dermatomal zone of the third division of the trigeminal nerve.

Table 6 Thermography Results for Subjects With SITN

Sex	Age (years)	Pain duration (months)	Location	Affected (°C)	Unaff (°C)	ΔT (°C)	ET pattern
F	66	66	Dental implant	34.9	34.1	+0.8	hot
F	67	24	R L mouth P	35.0	34.5	+0.5	hot
F	31	7	R V3	34.2	33.4	+0.8	hot
M	51	9	R ant cheek	34.7	34.2	+0.5	hot
M	41	missing	L nose	35.5	34.3	+1.2	hot
M	67	missing	Upper lip	35.7	35.3	+0.4	hot
F	38	60	R upper later				
			incisor	34.4	33.8	+0.6	hot
F	69	60	L V3	34.6	33.7	+0.9	hot
F	30	48	L mouth	34.5	33.6	+0.9	hot
F	74	111	R V3	34.7	33.4	+1.3	hot
F	62	62	L lip	34.4	33.8	+0.6	hot
F	44	44	L cheek	35.9	34.1	+1.8	hot
F	66	24	L chin	34.7	34.3	+0.4	hot
F	62	40	L V2	34.7	32.9	+1.8	hot
Mean	55	41.5		34.9	34.0	0.9	hot
SD	15	30		0.5	0.6	0.5	

R = right; L = left; V2 = dermatomal zone of the second division of the trigeminal nerve; V3 = dermatomal zone of the third division of the trigeminal nerve.

might hypothesize that hot thermograms due to injury or trauma would either indicate neurogenic inflammation or tissue inflammation produced by release of substance P and calcitonin gene–related peptide (CGRP).[17] Cold thermal emissions suggest a decrease in blood flow due to peripheral vasoconstriction. This is perhaps secondary to catecholamines binding new adrenergic receptors (α_1) that have sprouted on vessels around the site of the trauma.[17-20] These new receptors are bound by cir-

Table 7 Thermography Results for Subjects With SITN

Sex	Age (years)	Pain duration (months)	Location	Affected (°C)	Unaff (°C)	ΔT (°C)	ET pattern
F	41	60	L cheek	33.8	34.7	–0.9	cold
F	56	60	L angle mand	34.1	34.7	–0.6	cold
F	56	60	L V2	33.7	34.0	–0.3	equivocal
M	33	24	R V3	34.4	35.1	–0.7	cold
M	41	12	L cheek	34.5	35.2	–0.7	cold
F	26	24	L cheek	33.9	34.7	–0.8	cold
F	77	77	L lower lip	34.7	35.2	–0.5	cold
F	31	31	L ant V2	32.7	33.8	–1.1	cold
F	69	69	R post neck	32.8	34.1	–1.3	cold
F	48	48	R post neck	31.1	33.5	–2.4	cold
F	64	64	L V2	31.4	31.8	–0.4	cold
M	79	60	R V3	33.0	33.5	–0.5	cold
F	38	41	R face	33.6	34.0	–0.4	cold
F	70	123	Bilat ant V2	33.4	33.7	–0.3	equivocal
M	45	22	L V2	34.4	34.7	–0.3	equivocal
Mean	52	51.1		33.4	34.2	–0.7	cold
SD	17	29.0		1.1	0.9	0.5	

L = left; R = right; V2 = dermatomal zone of the second division of the trigeminal nerve; V3 = dermatomal zone of the third division of the trigeminal nerve.

culating catecholamines or by norepinephrine secreted by sympathetic nervous system activity (SMTN group) (Fig 1). Other patients without obvious sympathetic overactivity (local somatic nerve block stops the pain) may have a sprouting of neuropeptide Y receptors on their vessels[17] that will bind neuropeptide Y and circulating catecholamines, and will then show also a cold thermogram pattern (SITN group) (Fig 2). Myers et al[20] reported that circulating neuropeptide Y is derived from both sympathetic system and platelet activation, and they proposed the role of neuropeptide Y in the platelet-vascular interaction. This interaction will contribute to the vasoconstriction of the blood vessels.

Hot thermal emissions were seen uniformly in the SMTN group. This may be due to the chronicity of the symptoms (the SMTN group included patients having their symptoms for a longer period of time than the SITN group when tested), which may result in impairment of the sympathetic fibers and lead to a peripheral vasodilatation. Janig and McLachlan[17] also stated that in some cases of skin reinnervation, neurogenic vasodilatation can override the vasoconstriction induced by simultaneous stimulation of postganglionic vasoconstrictor axons, and thus result in blood vessel vasodilatation (Figs 3 and 4).

Analysis of the thermographic changes may be of diagnostic importance with regard to future treatment of neuropathic pain. Should there be different mechanisms causing diverse temperature changes, one could hypothesize that different treatments could be developed to affect these mechanisms. In this study, the presentation of normal thermograms was seen in patients with TN. Could this be secondary to the intermittent nature of the pain and the lack of pain at the time of imaging? Most of the TN patients were pain free at the time of imaging. Or could this be due to a mechanism that does not evoke peripheral blood flow changes? Rappoport and Devor[21] reported that the primary

Table 8 Thermography Results for the Different Diagnostic Groups

Diagnosis	n	Mean age	ET pattern	Thermal accuracy (%)
SMTN	10	58	Hot	10/10 (100%)
SITN	14	55	Hot	14/14 (100%)
SITN	15	51	Cold	13/15 (80%)
TN	6	42	Normal	6/6 (100%)
PTN	4	51	Normal	4/4 (100%)
TA	9	40	Normal	8/9 (89%)
Asymptomatic	22	47	Normal	22/22 (100%)

Normal ET pattern: $0.0°C \leq \Delta T \leq 0.25°C$
Equivocal ET pattern: $0.26°C \leq \Delta T \leq 0.35°C$
Abnormal ET pattern: $.36°C \leq \Delta T$

Figs 1 to 4 Central nervous system = CNS; norepinephrine = NE; calcitonin gene–related peptide = CGRP; substance P = SP; neuropeptide Y = NPY.

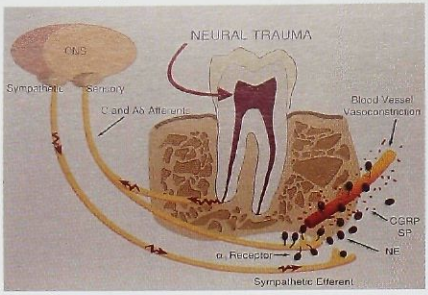

Fig 1 (SMTN with cold thermogram) Following neural trauma, sensory input is transmitted to the CNS via Aδ and C fibers. This input produces sensitization of the CNS. The α_1 receptors sprout on peripheral nociceptors following trauma. When these α_1 receptors are activated by the release of NE from the sympathetic terminals, nociceptors are activated and pain develops.[13] Adrenergic receptors on peripheral blood vessels will also bind NE, which causes vasoconstriction and skin cooling, resulting in a "cold" thermogram. The CGRP and SP are present in the nerve trauma area and stimulate the release of NE from sympathetic efferent fibers.

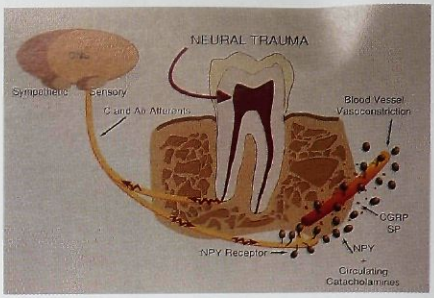

Fig 2 (SITN with cold thermogram) Following neural trauma, sensory input is transmitted to the CNS via Aδ and C fibers. This input produces sensitization of the CNS. It was postulated that this CNS sensitization causes NPY receptors to sprout on the peripheral nociceptors and blood vessels around the nerve trauma. The NPY and circulating catecholamines then bind these NPY receptors and produce vasoconstriction of the blood vessels, resulting in a "cold" thermogram.

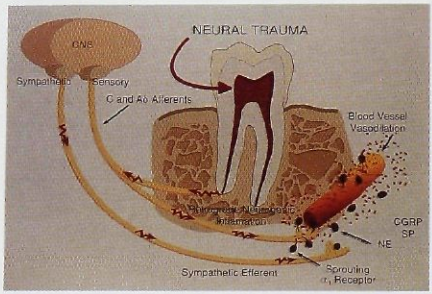

Fig 3 (SMTN with hot thermogram) Following neural trauma, sensory input is transmitted to the CNS via Aδ and C fibers. This input produces sensitization of the CNS. Sympathetic efferent activity causes the release of NE, which binds to α_1 receptors that have upregulated on the peripheral nociceptors and blood vessels following the trauma. This activity would cause vasoconstriction and a cooling of the skin. However, the CNS sensitization may also produce neurogenic inflammation, a retrograde release of SP and CGRP in the area of the nerve trauma. These neuropeptides have a vasodilating effect on the peripheral blood vessels surrounding the nerve trauma. In this case, the neurogenic inflammation effect is postulated to be greater than the vasoconstrictive effect of NE; therefore, vasodilatation and a "hot" thermogram result.

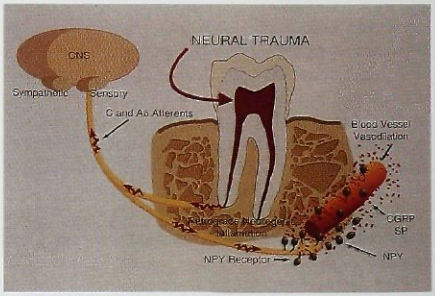

Fig 4 (SITN with hot thermogram) Following neural trauma, sensory input is transmitted to the CNS via Aδ and C fibers. This input produces sensitization of the CNS. It was postulated that the retrograde neurogenic inflammation mediated by SP and CGRP is greater than the vasoconstrictive effect of NPY and circulating catecholamines. The resultant vasodilatation causes a "hot" thermogram.

abnormality in trigeminal neuralgia resides in the trigeminal root ganglion (TRG) and in the trigeminal root, rather than in the skin or the central nervous system. They reported that the findings on examination are normal, except for the usual hyperesthesia in the trigger zone of the attack. They concluded that if antidromic firing in the C fibers of the TRG occurs, neurogenic vasodilatation should be triggered. Paroxysms are too brief to evoke a visible flare reaction in the periphery. The neural inflammation producing toothache (pulpitis) may also result in a normal thermogram because it is taking place within the pulpal canal, and therefore not producing skin tissue vasodilatation. This may not be the case in pulpitis with periapical pathology, where spreading inflammation in the tissues should result in a hot peripheral skin temperature.

The SMTN group showed 100% asymmetrical heat emission patterns. These subjects are very carefully selected as being unresponsive to somatosensory nerve block, which poses a problem in the differentiation of SMTN and the subgroup of "hot" SITN subjects.

At this time, it is believed that ET may be useful in differentiating neuropathic orofacial pain from pulpitis. Particularly in the clinical context of toothache without obvious local cause, the positive finding of asymmetrical thermographic heat emission might cause the clinician to ponder whether to go forward with irreversible dental procedures. Despite the interesting results of this investigation, further clinical investigation is needed before ET can be routinely recommended as a diagnostic tool for toothache.

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Resumen

Evaluación Tormográfica del Dolor Facial Neuropático

El dolor en curso, agudo e intermitente, o el dolor sordo intermitente alrededor de los dientes puede evocar la sospecha de la existencia de una patología dental. Sin embargo, cuando no se puede determinar clínica o radiográficamente la etiología dental, el diagnóstico diferencial relacionado al dolor neuropático y a la patología pulpar es todavía un desafío. Los dolores faciales neuropático muy a menudo son todavía mal diagnosticados como odontalgia de origen dental, lo cual trae como resultado extracciones dentales o endodoncias innecesarias. El propósito de este estudio fue el de determinar si la termografía electrónica era capaz de diferenciar los dolores faciales neuropáticos que se presentaban como odontalgias a causa de patología pulpar. La termografía electrónica fue utilizada para comparar los sujetos asintomáticos y los que tenían dolores faciales neuropáticos. Las personas asintomáticas y las personas con neuralgia del trigémino, neuralgia pretrigeminal y dolor pulpar sin patología periapical no mostraron diferencias termográficas en la zona donde estaba el dolor al compararse con el lado opuesto sin dolor. Los pacientes con neuralgia del trigémino (odontalgia atípica) y la mitad del grupo que presentaba neuralgia trigeminal traumática independiente simpática presentaron termogramas "calientes." La otra mitad de los pacientes con neuralgia del trigémino traumática independiente simpática revelaron termogramas "frios" en el área donde se presentaba el dolor. La termografía electrónica fue el examen menos selectivo para el grupo que mostraba un patrón de termograma "frío" (un acuerdo del 80% con las normas de caracterización termográfica). Esta información indica que la termografía electrónica puede ser útil para diferenciar los dolores neuropáticos de la patología pulpar.

Zusammenfassung

Thermografische Beurteilung von neuropathischen Gesichtsschmerzen

Anhaltender Schmerz, intermittierender schneidender Schmerz oder intermittierender stumpfer Schmerz kann den Verdacht auf eine Zahnpathologie erwecken. Wenn weder radiologisch noch klinisch eine dentale Pathologie gefunden werden kann, so bleibt die Herausforderung der Differentialdiagnose zwischen neuropathischem Schmerz und einer Pulpapathologie. Neuropathische Gesichtsschmerzen werden noch immer häufig als Zahnschmerzen dentalen Ursprungs fehldiagnostiziert. Das Ziel dieser Studie war es, zu bestimmen, ob elektronische Thermographie fähig sei, zwischen neuropathischen Gesichtsschmerzen, die als Zahnschmerzen imponieren und Zahnschmerzen mit pulpaler Pathologie zu differenzieren. Die elektronische Thermographie wurde angewandt, um asymptomatische Probanden und Subjekte mit verschiedenen Arten von neuropathischem Gesichtsschmerz zu vergleichen. Bei asymptomatischen, sowie bei Subjekten mit trigeminaler Neuralgie, prätrigeminaler Neuralgie und pulpalen Schmerzen ohne periapikale Läsionen konnte thermographisch kein Unterschied zwischen der schmerzhaften und der nichtschmerzhaften gegenüberliegenden Seite gefunden werden. Patienten mit atypischer Odontalgie und die Hälfte der Gruppe mit sympathischer unabhängiger traumatischer Neuralgie zeigten "heisse" Thermogramme. Die andere Hälfte der Patienten mit sympathischer unabhängiger traumatischer Neuralgie zeigten "kalte" Thermogramme in der Zone ihrer Beschwerden. Die elektronische Thermographie war der am wenigsten selektive Test für die Gruppe mit "kalten" Thermogrammen (80% Übereinstimmung mit den Charakterisierungskriterien der Thermographie). Diese Daten lassen die elektronische Thermographie hilfreich erscheinen für die Differenzierung zwischen neuropathischen Schmerzen und pulpaler Pathologie.