Thermographic Assessment of Reversible Inferior Alveolar Nerve Deficit

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The purpose of this study was to investigate thermography's potential as a diagnostic alternative for evaluating neurosensory deficits of the inferior alveolar nerve. Electronic thermography was used to evaluate the alterations in facial thermal patterns attendant to a conduction defect of the inferior alveolar nerve induced in 12 subjects using 2% lidocaine. The rates of onset and duration of sensory block, as visualized by thermography, were related to the results of conventional neurosensory testing. Comparison of the rate of response change within each measurement system revealed that changes in facial skin temperature manifest the induced deficit earlier than discriminative tests. Also, the prolonged elevation of thermal asymmetry suggested that electronic thermography has the ability to detect subtle changes in nerve function that are not discernible by physical neurosensory tests relying on patient response. Although cutaneous temperature increases were highest in the field of observation near the sensory distribution of the mental nerve, an inexplicable warming of the contralateral side of the face and neck was also observed. These attendant findings emphasize the need for further studies on the pathophysiologic mechanisms of facial thermal changes to better understand thermography's diagnostic accuracy and clinical utility for monitoring inferior alveolar nerve dysfunction.
J OROFACIAL PAIN 1994;8:375–383.

Neurosensory deficit of the inferior alveolar nerve (IAN) is a common and distressing consequence of traumatic or iatrogenic injury. Reports on the incidence of IAN dysfunction encountered in patients undergoing oral and maxillofacial surgery range from 3% following surgical removal of impacted third molars up to 70% following ramal sagittal osteotomy.[1-4] Conventional neurosensory testing used to detect and monitor sensory impairments of the IAN include the two-point discrimination test, the static light touch test, thermal discrimination test, and the pin-prick test.[5] However, these tests have low interrater reliability and are qualitative at best because they are based solely on the patient's subjective assessment of symptoms.[6,7] The determination of the need, nature, and timing of neurosurgical therapy is hampered by this lack of diagnostic objectivity. Subjective neurosensory testing criteria render it difficult to predict which nerves will recover spontaneously and which will need surgical intervention. Additionally, the poor predictability of conventional testing complicates determination of surgical intervention efficacy. The lack of standardized neurological assessment methods also has medicolegal repercussions. Basing the sensory examination solely on the patient's subjective assessment of symptoms compounds the difficulty in determining whether an individual's expressed complaint

of paresthesia or anesthesia nerve deficit is a result of organic nerve damage, psychogenic factors, or even malingering. Consequently, there is a need for more reliable, sensitive, and objective testing measures to document and monitor IAN function or dysfunction.

Electronic thermography, which can record small cutaneous temperature alterations, has been proposed as a safe, noninvasive diagnostic test for evaluating peripheral nerve dysfunction.[8] As an index of nerve dysfunction, thermography relies on detecting thermal variations in cutaneous sensory segments, alterations that manifest the changes in sympathetic activity resulting from nerve impairment. Modern computerized thermography units provide quantitative temperature measurements, eliminating the subjectivity previously associated with the visual interpretation of thermographic images. The purpose of this study was to investigate thermography's potential as a diagnostic alternative to conventional neurosensory tests. A temporary pharmacologic block of the IAN was used to ascertain whether conduction deficits manifest as detectable and quantifiable changes in facial thermal patterns. Changes in thermal patterns were related to conventional neurosensory testing to determine the comparative sensitivity of thermography in identifying and monitoring IAN dysfunction.

Materials and Methods

Twelve healthy subjects aged 21 to 30 years were chosen from a pool of volunteers. Informed consent was obtained from each subject according to the protocol approved by the Institutional Review Board. Prestudy screening consisted of a detailed medical history and head and neck examination. Exclusionary criteria included any evidence of acute or chronic orofacial lesions, a history of orofacial trauma or temporomandibular disorders, or a subjective neurosensory deficit following oral or facial surgical procedures.

Thermography and neurosensory testing were carried out in a cool, draft-free environment maintained at 21°C ± 1°C to minimize skin-temperature changes resulting from environmental fluctuations. In each subject, a testing area was outlined on the skin (5 cm in diameter, situated 6 mm inferior to vermilion border of lower lip) to approximate the area of sensory loss following IAN injury. The subjects were seated in a positioning chair, and a baseline facial thermogram was taken at an imaging sensitivity of 0.1°C using frontal projections of the chin and lip (Fig 1). A tongue blade held between the occlusal plane of the teeth helped to clearly delineate the area being investigated and to ensure replicability of patient position in the field of view of the thermographic scanner.

Because each point on the skin has multiple innervations and shows receptor specificity, a combination of physical neurosensory tests was used to monitor nerve function.[9] Prior to each testing session, the individual neurosensory tests were demonstrated on a distant site, such as the subject's forearm, to evaluate the subject's comprehension of the test and appropriate response. Subjects were asked to close their eyes during testing to eliminate any visual clues. To minimize interrater variation, all neurosensory testing and thermographic imaging were performed by the same set of investigators.

A reversible pharmacologic block of the IAN served as experimental model to evaluate the temporal alterations in nerve function and concomitant facial temperature changes. After establishing baseline thermographic and neurosensory data for the testing site, each volunteer received a unilateral mandibular block with 2 mL of plain 2% lidocaine hydrochloride. Following injection, sequential thermographic images and sensory evaluations were made at 5-minute intervals until sensory levels returned to baseline or near baseline values (approximately 2 hours after injection).

Electronic thermography was conducted using an Agema 870 thermovision unit (Agema, Secaus, NY) incorporating an infrared scanner, a control unit, a thermal image computer TIC-8000, and image processing software (Meds 1.0, Agema). The thermograms were stored on a computer disc and subsequently digitized to obtain quantitative thermal data for analysis. After demarcating the testing site on the individual thermograms, the degree of thermal asymmetry (ΔT) between each postinjection thermogram and the baseline thermogram was recorded and compared (Figs 1 and 2).

The light touch discrimination (LT) test was carried out with a series of 20 nylon monofilaments marked from 1.65 to 6.65 (Pressure Anesthesiometer, Research Designs, Houston, TX). Each number represents the logarithm of 10 times the force in milligrams required to bend the monofilament. The two finest monofilaments (1.65 and 2.36) were applied via two "quick bounces" to the stimulus area, whereas the heavier monofilaments were applied by a slower, one-touch motion. Filament size was determined as the variable that was perceived by the patient at least two times out

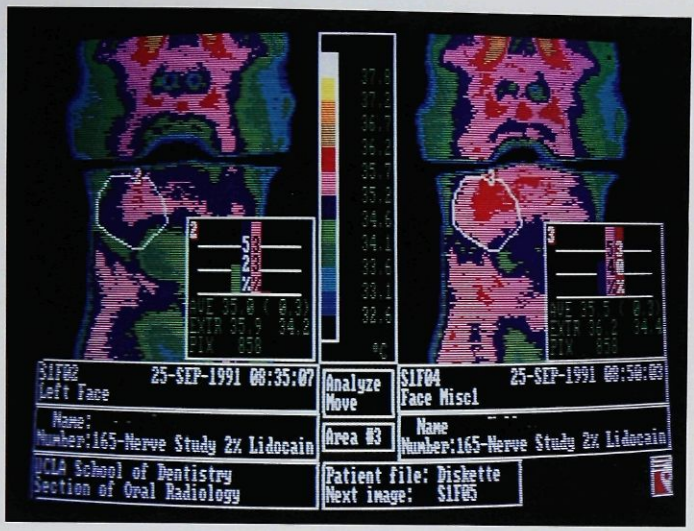

Fig 1 Preinjection (t = 0 min) and postinjection (t = 5 min) thermograms obtained at an imaging sensitivity of 0.1°C. The thermal patterns of the testing site have been demarcated and digitized to permit comparative analysis.

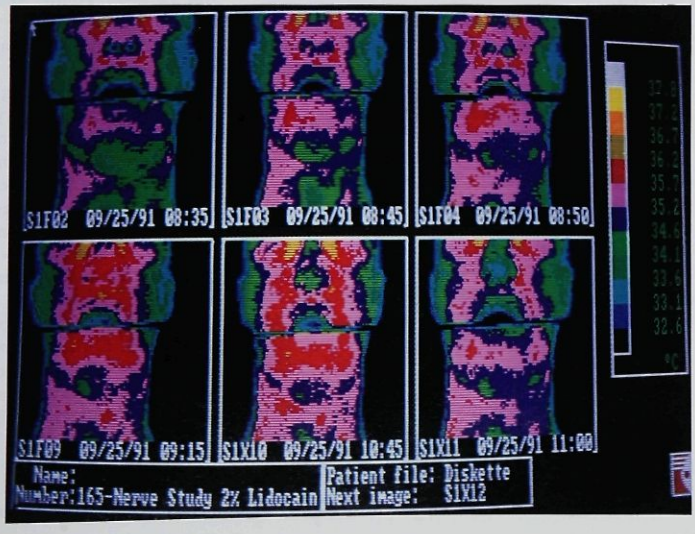

Fig 2 Temporal changes in facial thermal patterns subsequent to unilateral IAN block (imaging sensitivity = 0.1°C).

of three.[9] Patient responses to this test were scored on a scale (1 to 20) to correspond to filament number.

The two-point discrimination (2PD) test utilized the 2-Point Pressure Anesthesiometer (Research Designs).[9] This anesthesiometer consists of a set of seven devices with separation of the parallel plastic filaments ranging from 2 to 14 mm; each delivers a uniform force of 3.6 g. Starting with the largest separation, the devices were applied sequentially to the test site until the subject could no longer discriminate two separate points. Patients' responses ranged from 2 to 14 for this test based on filament separation.

The Minnesota Thermal Disk (MTD) tested for thermal discrimination by using two discs (copper MTD and polyvinyl chloride MTD) (Rochester Electro Medical, Tampa, FL) of different relative perceived coolness ranges. Each disc was applied to the testing site for 2 seconds, and a yes/no testing method was used to record the subject's ability to discriminate between these discs.[10]

The pin-prick (PP) test evaluated the subject's ability to detect sharpness.[5] Testing was accomplished by a sterile hypodermic needle applied to the test site. Subjects' responses were determined using a yes/no testing system.

Data Analysis

To analyze the relative onset of nerve block, the rate of response change for each subject and measurement method was compared over three time intervals: from *1)* time of injection (t = 0) to 5 minutes postinjection; *2)* 5 to 10 minutes postinjection; and *3)* 10 to 15 minutes postinjection. Paired *t* tests were used to compare whether the rates of change during a time period were statistically different from the rate (of the same measurement method) in the time period that followed. Two-tailed significance tests were evaluated at the 5% level.

Additionally, patterns of neurosensory deficit duration were compared within each of three measurement methods (thermography, light touch, and two-point distance). A subject's rate of response change during an interval representative of established IAN deficit (from t = 15 to t = 30 minutes postinjection) was paired with the subject's rate of change in the later portion of the measurement period (from t = 30 until the end of individual data collection).

Results

Figures 3a to 3e illustrate the mean profile of the temporal changes in measurement scores for the individual neurosensory tests. Despite different measurement scales and varying rates of change, the patterns of response change for each test approximated the nerve conduction changes expected after an IAN block. In general, the tests employing a continuous measurement scale (thermography, LT, 2PD) appeared to be more sensitive indicators of conduction deficit onset and duration. Neurosensory tests (MTD, PP) that utilized a discreet measurement scale (yes/no response) lacked this sensitivity. In most subjects, the baseline scores for the MTD and PP tests did not change until 20 minutes postinjection. At least two patients did not vary in their response to the MTD and PP tests. Hence, these tests were excluded from further analysis.

Table 1 Comparison of Average Rates of Change for Thermography, Light Touch, (LT) and Two-Point Discrimination (2PD) Tests*

	Thermography (SD)	LT (SD)	2PD (SD)
Rate of change (min)			
0–5	0.053 (0.032)	0.267 (0.375)	0.150 (0.284)
5–10	0.017 (0.025)	0.900 (0.851)	0.300 (0.302)
10–15	–0.004 (0.025)	0.283 (0.346)	0.125 (0.186)
Difference in rates between intervals (min)			
0–5 and 5–10	0.037 (0.042)	–0.633 (0.790)	–0.150 (0.444)
5–10 and 10–15	0.021 (0.035)	0.617 (0.863)	0.175 (0.391)

* Rates of change are not comparable between methods because of differences in measurement scales.

Table 1 shows the relative rate of onset of the neurosensory deficit for thermography, LT, and the 2PD tests. Thermography revealed a rapid increase in thermal asymmetry in the initial postinjection period (0 to 5 minutes) that slowed down over the next 5 minutes and appeared to stabilize 10 to 15 minutes postinjection. The difference in the mean rates of change observed in the first and second 5-minute intervals was statistically significant ($P = .011$), whereas the difference in the rates of change between the second and third intervals was marginal ($P = .062$). In contrast, the rate of response change for LT test in the first 5-minute interval was much slower than the rate in the next 5 minutes ($P = .018$), and slowed down again in the third 5-minute interval postinjection ($P = .031$). The 2PD test appeared to lag behind the thermography and LT tests in determining the onset of the sensory block. The average rate of change in the first and second 5-minute intervals was the same ($P = .267$)

Figs 3a to 3e Average of responses for thermography and the individual neurosensory tests across time. Direct comparisons are not possible due to different measurement scales.

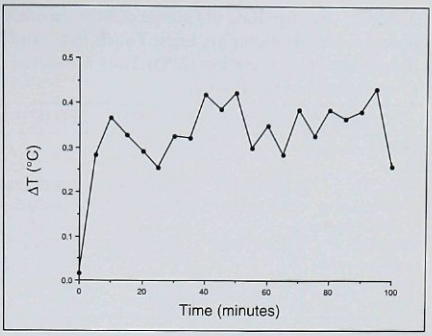

Fig 3a Thermography.

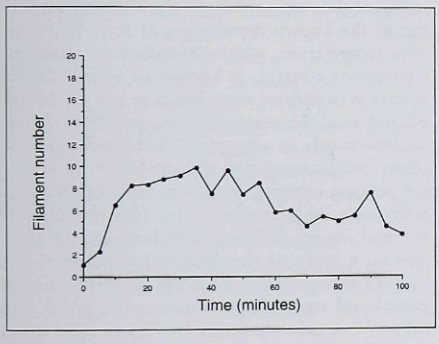

Fig 3b Light touch test.

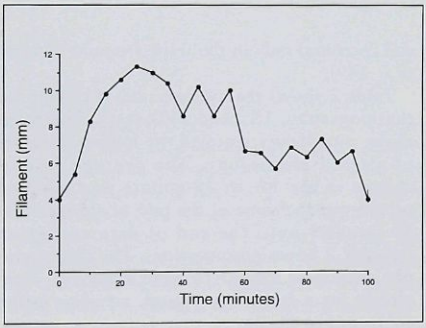

Fig 3c Two-point discrimination test.

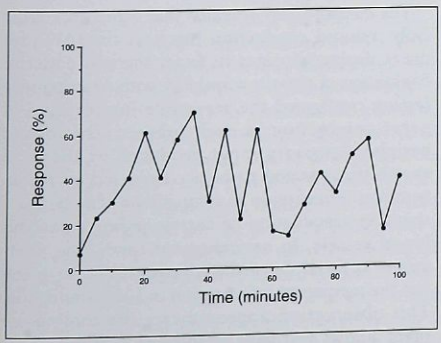

Fig 3d Minnesota Thermal Disk test.

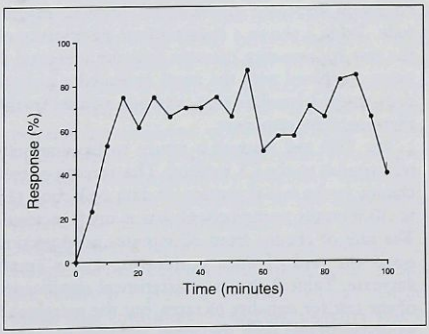

Fig 3e Pin-prick test.

Table 2 Average Rate of Change of Measurement Scores for Thermography, Light Touch (LT), and Two-Point Discrimination (2PD) Tests Measured Over Period of Observation

	Thermography (SD)	LT (SD)	2PD (SD)
Rate of change (min)			
15–30	−0.002 (0.008)	0.058 (0.195)	0.028 (0.078)
30 to end of data collection	−0.001 (0.003)	−0.060 (0.093)	−0.048 (0.058)
Difference in rates between intervals (min)	0.001 (0.008)	0.118 (0.220)	0.076 (0.121)
P value	0.732	0.089	0.06

* Rates of change are not comparable between methods because of differences in measurement scales.

and increased only in the third 5-minute interval ($P = .149$).

Table 2 shows the average rate of change for thermography, LT, and 2PD tests. Thermography consistently revealed the longest duration of altered physiology. The average rate of change in the 15- to 30-minute postinjection period was the same as the rate of change from 30 minutes until the end of data collection (roughly 2 hours postinjection). The thermography responses appear to have stabilized completely for a long time period, an observation confirmed by Fig 3a.

In comparison, the LT test showed a quicker return to baseline than thermography. The average rate of change in the 15- to 30-minute postinjection period increased gradually. The rate of change from 30 minutes postinjection until the end of data collection decreased gradually by the same magnitude. Table 2 shows a nonstatistical significance of the test for equality of rates, but the marginal *P* value combined with the trend indicated a general drift toward baseline that was not evident in the thermography responses.

The 2PD test showed a return to baseline pattern similar to the LT method. The average rate of change in the initial portion of data collection (15 to 30 minutes postinjection) was a small increase. The rate of change from 30 minutes postinjection until the end of data collection was a small decrease. Table 2 shows nonstatistical significance of the test for equality of rates, but the marginal P value combined with the trend indicates a general drift toward baseline.

Discussion

The subjectivity and variability associated with conventional neurosensory tests for IAN function derive largely from their reliance on patient perception and response. Differences in the examiner's experience and skill with a particular technique further compounds the veracity of test results. This lack of diagnostic objectivity makes it difficult to predict the ability of an injured IAN to recover spontaneously or to find out the need, timing, or efficacy of surgical intervention. Consequently, it is necessary to explore other measurement methods that could potentially facilitate reliable and objective monitoring of IAN function and dysfunction.

The conceptual appeal of thermography as an alternative or adjunct to conventional neurosensory tests lies in its ability to provide direct measurements of skin temperature at segments corresponding to the known distribution of sensory nerves. Skin temperature, which depends on sympathetic vasomotor control, is known to reflect disturbances in peripheral nerve function and can be displayed as a thermographic image.[11–14] Technical improvements in electronic thermography now allow visualization and quantification of surface temperature changes, decreasing the subjective bias previously associated with the interpretation of thermal images. With such technologic advancements, a body of thermographic patterns has evolved that corresponds to the clinical finding of peripheral nerve impairment in the trunk and limbs.[11,14] A previous study by Gratt et al[15] found that normal subjects demonstrated a high level of thermal symmetry. By extrapolation, altered facial thermal patterns in related cutaneous sensory segments may have diagnostic utility in monitoring trigeminal nerve dysfunction.

The current study revealed that a pharmacologically induced conduction block of the IAN produces distinct changes in facial thermal patterns. Evaluation of test-site sensibility with neurosensory testing confirmed the reversible interruption of nerve conduction in each subject. The loss of peripheral sensory input produced by the local anesthetic appeared to be accompanied by a concomitant interruption in sympathetic activity and a resulting vasodilation of corresponding cutaneous blood vessels. In an indirect manner, the alterations in facial thermal patterns seemed to manifest the temporary interruption in IAN conduction. This observation approximates the findings of other studies that have established a close correlation of temperature patterns changes with altered

function of known cutaneous sensory nerves.[14,16] Furthermore, the premise that mechanical compression by the injection volume could be partly responsible for the thermal changes has been repudiated in experiments by Brelsford and Uematsu.[14] Saline injection in a nerve's vicinity did not produce any detectable effect on the vasomotor activity as evidenced by thermography.

The ability to measure subtle differences in nerve function is an expression of the sensitivity of a particular neurosensory test. To establish the relative sensitivity of thermography, the rates of response change and the duration of altered response were related to the individual neurosensory tests. Because each test had a different measurement scale, direct cross comparisons were not possible. However, comparisons of the rates of change within each measurement scale allowed characterization of each method's onset patterns and duration. Individual analysis of the speed of deficit onset in the initial 15 minutes postinjection showed that the rate of measurement change from baseline was fastest for thermography. An elevation in facial temperatures appeared to be the earliest indicator of the induced IAN deficit. The LT and 2PD tests were relatively slower in revealing the onset of the nerve deficit. In contrast, the baseline scores for the MTD and PP tests did not change until 20 minutes postinjection, and in two subjects, these scores did not change at all. This lack of response sensitivity suggests that the MTD and PP testing measures may be unreliable monitors of IAN function, an assumption corroborated by other studies.[17,18]

The individual tests also varied in terms of the duration of their response change measured over the period of observation. Thermography scores rapidly increased and remained high for the duration of the neurosensory testing. The LT, 2PD, MTD, and PP tests revealed a gradual increase in scores that returned to near baseline levels within 2 hours. One explanation for this disparity in manifesting the duration of nerve dysfunction might be thermography's ability to visualize the cutaneous temperature alterations resulting from extremely small physiologic changes. Due to its high sensitivity, thermography can plausibly detect residual defects in nerve conduction that are not discernible by tests predicated on the patient's perception. In this regard, the PP and MTD tests are particularly disadvantaged because they record subject response as either normal or abnormal. This restrictive data collection format does not permit these tests to report subtle or borderline changes in nerve function. Alternatively, the prolonged increase in thermal asymmetry may be due to a preferential and prolonged decrease of the sympathetic component over the sensory component.[19]

The extent of altered thermal patterns was greater than anticipated and could not be explained by the current understanding of the sympathetic nerve supply of the face. The sympathetic nerve supply to facial skin is believed to come from the superior cervical ganglion via the arterial blood supply in the branches of the internal and external carotid branches. Therefore, the thermal asymmetry was expected to reflect the unilateral nerve block. Although temperature increases were highest in the field of observation near the sensory distribution of the mental nerve, the thermal asymmetry also involved the contralateral side of the face and extended to the neck. Through some mechanism yet unclear, the nerve deficit appeared to cause a bilateral interruption of sympathetic tone in all subjects. Because the thermal changes were observed at levels above, below, and adjoining the actual site of cutaneous sensory loss, matching a thermal abnormality specifically to a trigeminal nerve dermatome would appear to be difficult.

Thermography's ability to indicate nerve function in a functional sphere has clinical promise and needs to be explored further. Albeit indirectly, changes in facial skin temperature appear to be more sensitive indicators of subclinical or clinical abnormalities in IAN function than the discriminative tests used currently. The initial results in this study show that neurosensory examination, based on the analysis of thermal patterns, is likely to disclose that neurologic dysfunction is more frequent than currently reported. However, determination of thermography's diagnostic utility and accuracy is complicated by the lack of an ideal reference standard, the complexity of facial skin temperature patterns, and the limited understanding about the sympathetic dermatomes of the face. As with other physiologically based tests, alterations in thermal patterns do not always follow a clear-cut pattern. The warming of the contralateral side of the face was an inexplicable but consistent observation in all subjects. Furthermore, an inadequate understanding of biologic thermoregulatory processes influencing skin temperature contributes to the large variation in the diagnostic-accuracy data (sensitivity and specificity) reported by various researchers.[20] Hence, further experimental and clinical studies are needed to clarify the pathophysiologic mechanisms that contribute to thermal changes and to establish thermography's diagnostic utility for monitoring IAN function.

Acknowledgment

This project was supported by BRS Grant RR05304 from the Biomedical Research Support Grant Program, DRR, National Institutes of Health.

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Resumen

Evaluación termográfica de la deficiencia reversible del nervio alveolar inferior

El propósito de este estudio fue el de investigar el potencial de la termografía como una alternativa diagnóstica en la evaluación de las deficiencias neurosensoriales del nervio alveolar inferior. Se utilzó la termografía electrónica para evaluar las alteraciones en los patrones térmicos faciales concomitantes a un defecto en la conducción del nervio alveolar inferior inducido en 12 personas por medio del uso de lidocaína al 2%. Los valores del principio y a la duración del bloqueo sensorial representados en la termografía, fueron relacionados a los resultados obtenidos de exámenes neurosensoriales convencionales. Las comparaciones de los valores de cambio en la respuesta dentro de cada sistema de medida revelaron que los cambios en la temperatura dérmica facial demostraban la deficiencia inducida mas temprano que los exámenes discriminativos. También, la elevación prolongada de la asimetría térmica indicó que la termografía electónica tiene la habilidad de detectar cambios tenues en la función nerviosa que no son perceptibles por medio de exámenes neurosensoriales físicos que dependen de la respuesta del paciente. Aunque los aumentos de la temperatura cutánea fueron mayores en el campo de observación cerca de la distribución sensorial del nervio mentoniano, también se observó un calentamiento inexplicable del lado contralateral de la cara y el cuello. Estos hallazgos concomitantes enfatizan la necesidad de realizar mas estudios sobre los mecanismos patofisiológicos de los cambios térmicos faciales para entender mejor la exactitud diagnóstica de la termografia y su uso clínico para el monitoreo de la disfunción del nervio alveolar inferior.

Zusammenfassung

Thermographische Beurteilung von reversiblem Gefühlsausfall des N. alveolaris inferior

Das Ziel dieser Studie war, das Potential der Thermographie als diagnostische Alternative in der Evaluation von neurosensorischen Ausfällen des N. alveolaris inferior zu untersuchen. Elektronische Thermographie wurde benutzt, um die Veränderungen der Wärmeverteilung im Gesicht auszuwerten, welche bei einer Blockade des N. alveolaris inferior durch Lidocain (2%) bei 12 Personen entstanden ist. Beginn und Dauer des sensorischen Ausfalls, welche durch Thermographie sichtbar gemacht worden sind, wurden mit den Resultaten von konventionellen neurosensorischen Tests in Beziehung gebracht. Der Vergleich der Zeitmuster der durch die Anästhesie hervorgerufenen Reaktionen zeigte, dass Änderungen der Hauttemperatur im Gesicht den induzierten Ausfall früher erkennen liessen als diskriminative Tests. Ausserdem legte das verlängerte Auftreten einer thermischen Asymmetrie nahe, dass elektronische Thermographie die Fähigkeit hat, kleinste Veränderungen der Nervenfunktion wahrzunehmen, die bei neurosensorischen Tests, welche sich auf Patientenantworten verlassen, nicht festzustellen sind. Obwohl die Hauttemperaturanstiege im untersuchten Feld am grössten im Versorgungsbereich des N. mentalis waren, wurde auch eine unerklärbare Erwärmung der kontralateralen Seite von Gesicht und Nacken beobachtet. Diese zusätzlichen Beobachtungen betonen die Notwendigkeit von weiteren Studien über die pathophysiologischen Mechanismen bei Änderungen der Gesichtstemperatur, um die diagnostische Genauigkeit der Thermographie und ihre klinische Anwendung zur Prüfung von Dysfunktionen des N. alveolaris inferior besser zu verstehen.