Infrared Thermal Imaging As A Tool In Pain Management - An 11 Year study, Part I of II

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Summary

The anatomical tests such as magnetic resonance imaging (MRI), computed tomography (CT), and physiological tests such as electromyography (EMG) and nerve conduction velocity (NCV) tests have been the main diagnostic tools applied in the management of somesthetic (somatic) pain. The above tests usually are not informative in the diagnosis of neuropathic pain. The neurovascular involvement in neuropathic pain requires tests such as Infrared thermal imaging (ITI) and Quantitative sudomotor axon reflex test (QSART) that address autonomic (e.g., thermal) changes for a more accurate diagnosis and treatment. This is a study of the role of ITI in the diagnosis and management of pain.

Bales Scientific Thermal Processor and Agema Cameras were used for this study of 3,265 successive patients. A review of our experience with Infrared Thermal Imaging (ITI) and its role in pain management was conducted, and compared with the recent medical literature. The study was limited to the role of ITI in the management of complex chronic pain.

Sloppy technique, and poor background in basic neurophysiologic training, have contributed to poor utilization and interpretation of ITI. For the ITI to be accurate and clinically useful, proper technique, standardization, and proper clinical correlation are the minimal requirements. The basic physiology of autonomic thermoregulation is outlined in detail to help the clinician to properly understand and interpret the test. The dysfunction of thermal sensory nerves cannot be detected by EMG or NCV and excluding the ITI test may mislead the clinician to diagnose the condition as "psychogenic" or "functional.

ITI provides useful clinical information when applied with proper technique. It provides diagnostic and therapeutic information limited to diseases involving autonomic, neurovascular, and neuroinflammatory changes. Conversely, it cannot be expected to help diagnose nerve injuries with nomicrovascular involvement such as somesthetic nerve injuries. Proper teaching and understanding ofthermoregulation helps the clinician to obtain indispensable information from this test

Key Words - CRPS, Headache, Sympathectomy ,Thermography

Infrarotthermographie als Hilfsmittel im Schmerzmanagement - eine 11 Jahres-Studie. 1.Teil

Anatomie orientierte Untersuchungen wie die Magnetresonanzdarstellung (MRI) oder die Computertomographie (CT) und physiologische Tests wie die Elektromyographie (EMG) und die Bestimmung der Nervenleitgeschwindigkeit (NLG) gelten als die wichtigsten diagnostischen Methoden, um den somatischen Schmerz zu beurteilen. Diese Untersuchungen versagen jedoch bei der Beurteilung des neuropathischen Schmerzes. Bedingt durch die neurovaskulären Veränderungen des neuropathischen Schmerzes bedarf der Methoden desr Infrarothermographie und der quantitativen Beurteilung des sudomotorischen Axonreflexes, um die Veränderungen des autonomen Nervensystems zu Zweck einer korrekten Diagnose und Behandlung zu beurteilen. Die vorliegende Untersuchung beschreibt die Rolle der Infrarotthermographie in der Diagnose und dem Management von Schmerzsyndromen.

Infrarotkameras von Bales (Scientific Thermal Processor) und Agema wurden in dieser Studie an 3265 Patienten eingesetzt. Ein Überblick unserer Erfahrungen mit der Infrarrotthermographie und ihrer Rolle im Management von Schmerzsyndromen wird gegeben und mit der aktuellen medizinischen Literatur in Beziehung gesetzt. Dabei war der Einsatz der Infrarotthermographie auf das Management komplexer chronischer Schmerzsyndrome beschränkt.

Mangelnde Technik und unzureichende Kenntnisse der elementaren Grundlagen der Neurophysiologie habe n

zu einer insuffizienten Anwendung und Auswertung von Infrarotthermogrammen beigetragen. Korrekte und standardisierte Durchführung und Korrelation mit den klinischen Befunden sind die minimalen Voraussetzungen, um die Infrarotthermographie verlässlich und mit klinischem Nutzen einzusetzen.Die grundlegende Physiologie der autonom-nervösen Thermoregulation wird im Detail beschrieben, um zu gewährleisten, dass der Kliniker die Thermographie richtig anzuwenden und zu interpretieren weiß. Die Fehlfunktion der thermosensiblen Nerven kann durch EMG und NLG nicht erfasst werden. Wenn die Thermographie in diesen Fällen nicht eingesetzt wird, kann der Kliniker fälschlicherweise zur Diagnose eines "psychogenen" oder "funktionellen" Syndroms gelangen.

Die korrekt durchgeführte Infrarot-Thermographie liefert nützliche klinische Informationen. Allerdings sind die diagnostischen und therapeutischen Aussagen auf Krankenheitsbilder mit autonomen, neuro-vaskulären oder entzündlichen Veränderungen beschränkt. Im umgekehrten Fall, kann nicht erwartet werden, dass die Thermographie bei Nervenverletzungen ohne begleitende Gefäßreaktion zur Diagnose beträgt. Kenntnisse der Thermoregulation erlaubt es dem Kliniker, die unverzichtbare Information aus dieser Untersuchung zu gewinnen.

Schlüsselwörter: CRPS, Kopfschmerz, Sympathektomie ,Thermographie

Introduction

This is a review of our 11-year experience with the application of Infrared thermal imaging (ITI) in 3,265 patients suffering from chronic pain. This study focuses on the application of ITI as a diagnostic and therapeutic guide.

Terminology

The nociceptive pain sensation is divided into two distinct categories: Neuropathic (Table 1) and somesthetic (somatic) pain. The neuropathic pain is associated with thermal (vasomotor) changes. These changes are in response to the afferent noxious impulses of unmyelinated sensory nerves (1). This is in contrast to the common somatic (somesthetic) pain which is usually not accompanied by circulatory dysfunction.

The somesthetic pain is characterized by involvement of afferent somatic (spinothalamic) nerves usually with no circulatory disturbance. The somatic pain has a dermatomal pattern (Fig. 1) in the distribution of nerve roots and nerve trunks. In contrast, the thermatomal distribution (Fig. 1) of neuropathic pain (2,3) follows an arterial distribution such as femoral, carotid or brachial arteries. In pathologic states, hypo- and hyperthermic changes are recorded by ITI which can be quite helpful in the selection of a proper treatment protocol.

The neuropathic pain, by virtue of involving the neurovascular structure, is accompanied by circulatory (Thermal) changes leading to a different type of pain such as causalgia (4), deafferentation and sensitization (5), as well as abnormally evoked pain: e.g., hyperpathic (protopathic) regional pain (6),and allodynic pain evoked by even minimal tactile stimulation (7). These are characteristic pains accompanied by

Table 1,
Diseases; in which neuropathic pain may occur.

Mononeuropathy	Amputation stump pain Causalgia Diabetes mellitus Neuroma Plexus avulsion Postherpetic neuralgia Traumatic Vasculitis
Mononeuropathy multiplex	Diabetes
Polyneuropathy	Alcohol, Nutritional neuropathy Chemotherapy Diabetes Ehrler Danlos Syndrome Fabré disease HIV Hypothyroidism Vitamin deficiencies
Cancer	
Neurosyphilis (Tabes)	
Trigeminal neuralgia	

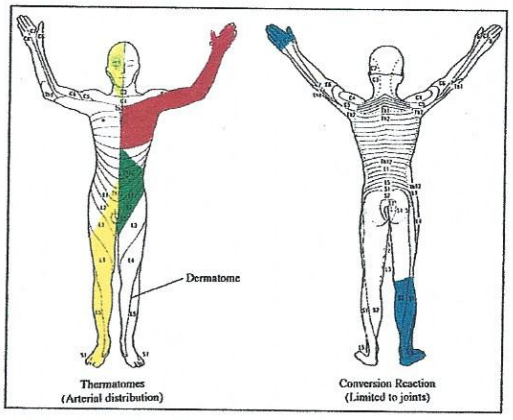

Figure 1
In neuropathic pain the sensory loss shows thermatomal (vascular) distribution in contrast to the dermatomal (radicular) distribution of the somatic pain. Conversely, the malingering sensory loss is limited to the joints.
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neurovascular dysfunction (8) of any etiology (e.g., post herpetic neuralgia, diabetic mononeuropathy, etc.

The ITI exclusively measures temperature changes of the body. Hence, its diagnostic value is limited to the study of nerve dysfunction with microvascular involvement (neuropathic pain). The thermal regulation is achieved by coordination of multiple anatomic areas of central and peripheral nervous system (PNS). The PNS contribution is modulated by afferent impulses from microscopic small c-fiber thermoreceptors) (9,10,11). Such minute unmyelinated nerves cannot be tested with anatomical tests such as CT and MRI, nor with somatic type of physiological tests such as electromyography (EMG), and nerve conduction velocity (NCV). The NCV cannot study the microcirculatory neuropathic function. It studies the function of the large trunk myelinated nerve fibers which are part of the somatic (e.g., spinothalamic) nervous system (12). In contrast, ITI evaluates sympathetic thermoregulatory function more comprehensively than sweat test. The sweat test e.g., QSART measures the function of a minority (less than 10%) of cholinergic nerves in the sympathetic system.

History

In ancient medicine, physicians were taught to measure temperature by hands. This insensitive and inaccurate method is still applied by physicians with poor knowledge of physiology. Approximately Four decades B.C., wet mud salves were used to detect surface body temperature. Hippocrates advocated the method. By the end of the 16th century, Galileo devised a "thermoscope" as a tool in patient care. John Herschel was the first to perform Thermography by using a prism and a piece of paper soaked with alcohol and impregnated with lamp-black. By the early 1950's, thermal recoding was applied by US forces in the Korean war. Dr. Ray Lawson, and later Professor E. F.J. Ring, and others (13-16) reported clinical application of thermography. By 1982, thermal imaging was accepted as a new laboratory test in Japan. At least in 2 states (California and Florida) the worker's compensation courts have accepted the utilization of ITI for the diagnosis of CRPS.

The American Academy of Neurology (AAN) (17) in 1990 reviewed the utilization of Thermal imaging in neurologic practice. It emphasized the importance of proper technique. It found the test not useful for the diagnosis of radiculopathies, entrapment neuropathies, headaches, stroke, and transient cerebral ischemia (17). As discussed in the present paper, the ITI is not useful for diagnosis of transient ischemic attacks, entrapment neuropathy (18), and disc herniation (19), but can contribute information to diagnosis and treatment of neuropathic pain due to neurovascular dysfunction.

The ITI Puzzle

The ITI has been abused, and over -and underused in the past three decades. Improper technology on one hand, and poor understanding of the basic anatomy and physiology of the autonomic nervous system (ANS) has contributed to exclusion of this test, depriving the patient of proper diagnosis and treatment.

The erroneous expectation of a single test to identify the cause of a complex clinical syndrome can lead the physician to deem the test useless. Such a complex syndrome is properly diagnosed by careful history taking and clinical correlation rather than by applying a single test. As an example, MRI of the spine may show an innocuous disc bulging or protrusion unrelated to the patient's pain. Whereas only 28-30% of the patients suffering from chronic back pain are due to disc pathology, over 80% of such patients are diagnosed as disc disease, leading to unnecessary surgery (20,21).

Technical Aspects

The dynamic state of flux of the sympathetic system plays an important role in balancing and compensating the external versus internal fluctuations of body temperature. Accurate recording of this sophisticated temperature regulation requires impeccable technique, reproducible, consistent recording, and a controlled laboratory environment. Unfortunately, the physician rarely enters the laboratory to obtain further, more sensitive, and accurate pictures than the standard technique done by the technician (Fig.2). ITI can provide further information leading to correct diagnosis.

The infrared camera records the infrared electromagnetic spectrum (Table 2). At the short wave boundary (Table 2) the infrared spectrum starts at the visual perception limit of deep red. At the opposite extreme of long wavelength frequency, it borders, and blends with, microwave-radio wavelength. The infrared band is

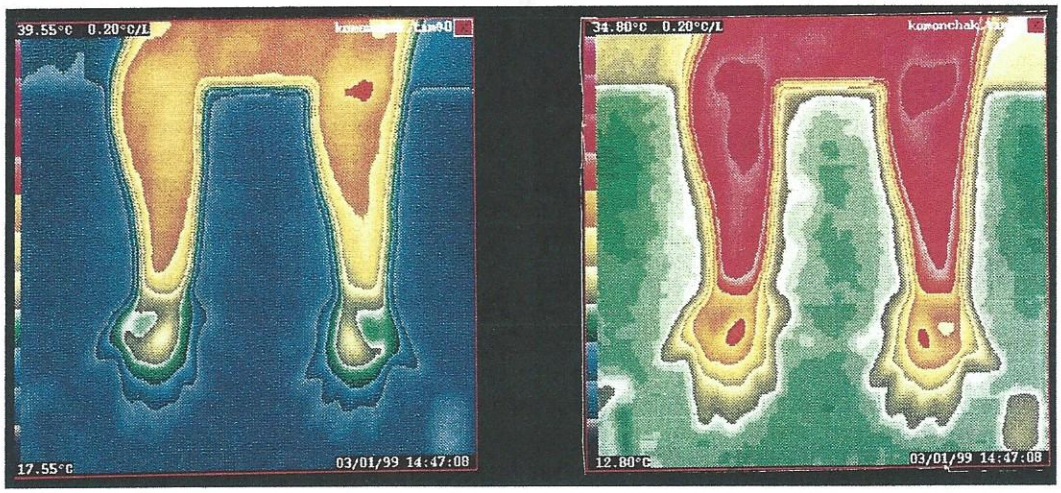


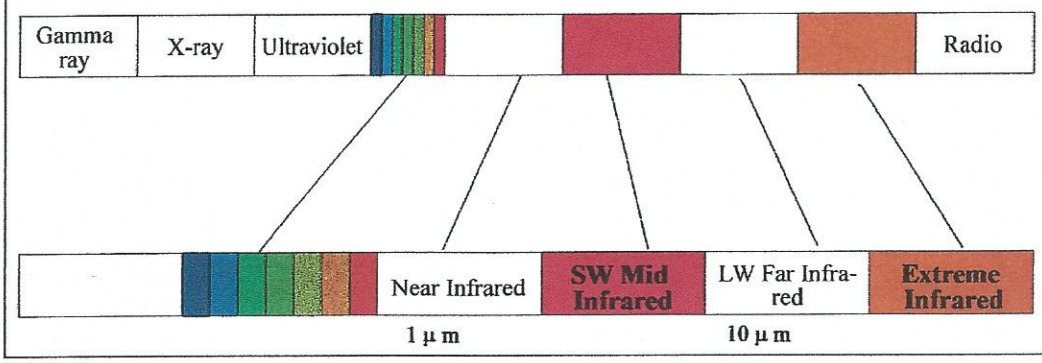
Figure 2

Central hyperthermic areas of entrance and exit in electrical injury. The permanent hyperthermic damage is surrounded by vasoconstrictive hypothermia. Only after increasing the thermal sensitivity (right) the lesions were identified. This "button hole" sign is exclusively seen in electrical injury.

subdivided to "near infrared" (0.75 to 3μm), "middle infrared "(3-6μm), "far infrared" (6-15μm), and "extreme infrared" (15-100μm). The standard ITI units measure the far infrared (FIR) wave length. To accurately record and measure subtle temperature changes requires a " blackbody" box. The "blackbody" is an object capable of absorbing all radiation and equally emitting any wave length (Kirchhoff Law).

The thermal imaging laboratory requires minimum space of 3.5x4.5 meters; Room temperature of 70-72°F; No shiny (e.g., Linoleum) or smooth plastic floor to avoid heat and light mirror effect; The interior of the laboratory should be infrared absorptive. Walls should be insulated at minium rate of R=19Hr-Ft 2 - °F/BTU; Room humidity of 50-70 %. The air conditioner should provide laminar down flow ranging no more than 1°F (1.8 °C) temperature. The patients are cooled down in a 20-21°C steady state room for 30 minutes of equilibration without clothing. No prior smoking for at least 90 minutes, no sun bathing lotion, no application of ice packs or heat, no acupuncture, laser therapy, EMG needle test, or transcutaneous electric nerve stimulation (TENS) for at least 48 hours prior to the thermographic testing. The patient should not wear any type of jewelry during the test. A standard sensitivity of 24-34°C is the usual staring point. Standardization of technique, and consistent reproduction of the results are essential for accuracy of the test. A standardized ambient temperature of

Table 2.
The relationship of electromagnetic spectrum to the infrared band.



21-22° C (70-72°F), a minimum of 2-3 sets of reproducible measurements in a cool, controlled temperature, no smoking, and no perfumes for at least 1 hour before the test, are the minimal technical requirements (22) to achieve a reliable comparative side to side (delta T) of ± 0.2 - 0.3° C (23). The review of ITI done prior to patients being referred to our clinic have revealed the tendency for inflexible routine baseline temperature measurements, rather than adjusting the sensitivity gauge to achieve a more accurate test (1).

Physiology

The sympathetic nervous system is an integral part of the comprehensive central and peripheral nervous system playing a role in

1. TEMPERATURE REGULATION of the body (skin and viscera) (24-27),

2. CONTROL OF VITAL SIGNS (blood pressure, pulse, and respiration), and

3. MODULATION OF IMMUNE SYSTEM.

The temperature regulation is achieved by modulating heat loss and heat preservation through dermal and sub-dermal circulatory (sympathetic) and sweating (cholinergic) changes. In contrast to the fish, the "warm blooded" animals can only survive in the stable, narrow - range band of the internal environment temperature - milieu interne (28). The dermal and subdermal structures provide a grid style of vertical and horizontal vascular shunting system which protects the body against excess ambient heat by wasting the body heat through vasodilation and sweating. The same system does the opposite in excess ambient cold environment by conserving heat, and by cooling the skin surface. The deep tissue circulation plays a major role in the protection of the internal environment (homeostasis). In cold temperature, skin vasoconstriction increases the deep tissue heat and circulation to prevent core hypothermia. Chronic pathologic sympathetic up- regulation causes the vicious circle of persistent dermal vasoconstriction, and increased deep circulation in bone and muscles, leading to osteopenia and muscle weakness (29).

The sympathetic system modulates the cellular immune function (30), leading to modulation of cellular neurogenic inflammation in pathologic conditions (30-45). In severe and chronic stages of sympathetic dysfunction, neuro-inflammation results in bulbous lesions (33), pelvic inflammatory disease (PID), interstitial cystitis (46), and sterile abscess (47). The regional neuroinflammatory edema leads to impingement of the peripheral nerves mislead the clinician to mistake the disease for conditions such as carpal tunnel (38,48,49), Dupuytren's contracture (50) (Table 3), thoracic outlet (TOS), tarsal tunnel, and myofascial syndrome (39). The surgical trauma or repetitive trauma due to sympathetic ganglion blocks (Table 3), in turn aggravates the inflammation (12, 40, 51-54), becoming a new source of neuropathic pain and leading to spread of the disease (42,55). ITI can spare the patient from unnecessary surgery (12,36,37). The primary afferent sensory neuron plays a major role in modulation of excitatory, and pro-inflammatory neuropeptides such as substance P (SP) (,43,44,56-66), nitric oxide (NO) (67-87), and calcitonin gene-related peptide (CGRP) (88), as well as inhibitory hormones such as corticotropin-releasing hormone, opioid peptides, such as dynorphin

Table 3
The role of ITI in selection of treatment modalities.

1. Identification of "Virtual Sympathectomy". (Permanent hyperthermia due to damage from repetitive ganglion block needle insertion). ITI spares the patient from further blocks.
2. Alpha-receptor supersensitivity to circulating Nor-ep shows diffuse hypothermia indicating the futility of any other chemical, radiofrequency, or surgical procedures
3. ITI identifies the permanent hyperthermia in the injured extremity. The apex, central part of permanent sympathetic nerve damage is surrounded by hypothermia. Any form of needle insertion, nerve block, or topical Clonidine skin patch application to the damaged nerve area aggravates and enlarges the lesion. The treatments should be applied proximally at epidural and paravertebral levels of spine corresponding to the area of nerve damage.
4. Thermal evidence of neurovascular instability on ITI proves advanced stage of sympathetic dysfunction non-responsive to sympathetic ganglion block or sympathectomy.
5. ITI identifies referred-pain focal hypothermic area. Massage or nerve block in this focus relieves the pain.

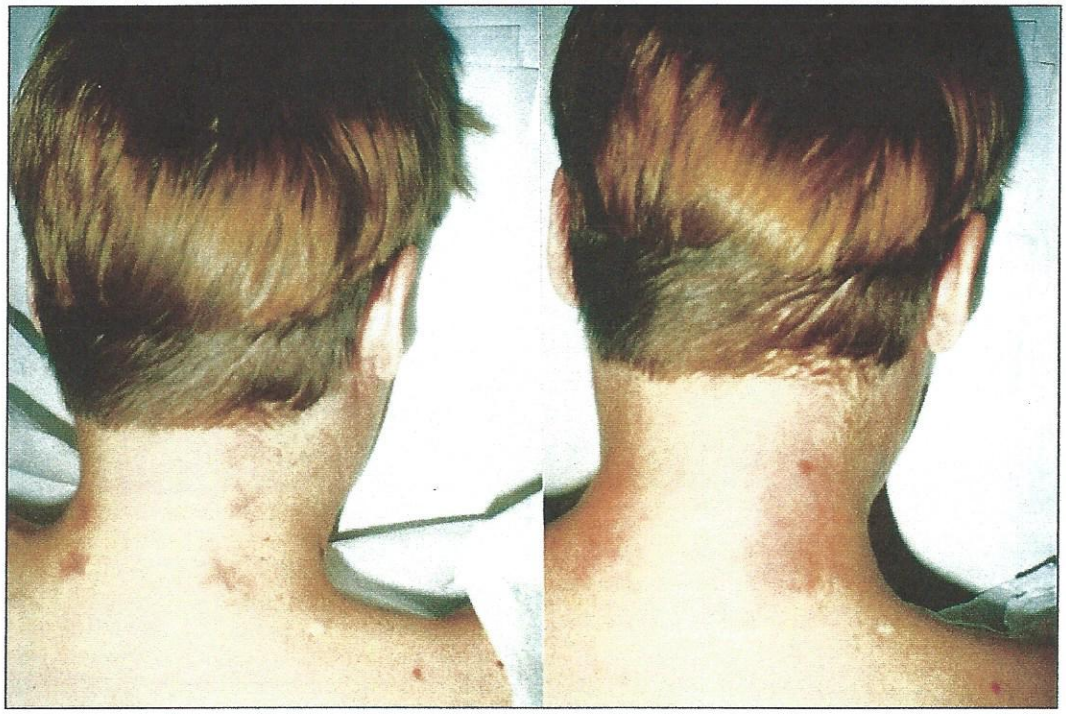


Figure 3
Cervical neuropathic pain represented with hypothermia on ITI in the paravertebral area. Gentle pressure exerted over the cervical spine (Left) revealed reactive release of inflammatory chemicals and blushing of the skin in the hypothermic area. Treatment with paravertebral nerve blocks (Right) provides pain relief, and dissemination of irritative substances (SP, NO, etc). Massage therapy after block enhances the recovery.

(59). Usually, in chronic stage, the referred pain such as seen in shoulder-hand syndrome, results in antidromic spread (89) of the inflammatory substances (59) causing secondary involvement of the paravertebral sensory nerves. The sympathetic dysfunction leads to inflammatory response in the extremity, as well as in the epidural and paravertebral regions of spine. ITI helps identify these inflammatory changes. Epidural (90) and paravertebral (91) nerve blocks in these regions help relieve the inflammation (Fig. 3). Such blocks achieve pain relief, as well as anti-inflammatory effect through injection of minute (2.5 to 10 mg) doses of depomedrol® (methylprednisolone) (12,92).

Peripheral and Central regulation of Deep and Surface Temperature

The sympathetic system participates in regulating the core body temperature within a narrow band. The normal blood flow to the skin is 200 ml/mm which is 4% of the cardiac output (93). This output far exceeds the baseline oxygen and nutrient requirements of skin. The rich arteriovenous anastomoses in acral areas of the palm of hands, feet, and axilla, allow a large volume of blood to flow through the skin. This leads to hyperthermia and heat emission. The parallel anatomical structure of large arteries and veins in the extremities allows countercurrent exchange of heat leading to superficial vasoconstriction, and simultaneous shunting of blood from the superficial to the deep venous systems, leading to surface heat preservation (93).

Central Nervous System Thermoregulation

The thermal changes in blood circulation are detected by neurons in the preoptic nuclei of the hypothalamus (94). The thermoreceptors for cold - and warmth sensations in the skin and perivascular areas play an important role in this temperature detection (94). The posterior hypothalamus modulates proper heat-generation or dissipation mechanisms. The inhibition of sympathetic output results in cutaneous vasodilation and heat loss (94). Moreover, the same inhibition results in more cholinergic sweating (by 1/10 of nerve fibres in the efferent

sympathetic nerves). On the opposite extreme, cold exposure stimulates increased sympathetic output from posterior hypothalamus leading to vasoconstriction, and heat conservation (94). The shunting of blood into the deep venous system acts as an insulator in the subcutaneous fat layers between the blood and the cold ambient temperature. The protective effect of subcutaneous fat is important. The obese individuals can maintain a higher internal temperature on cold exposure. They also have chronically cooler skin temperature (95,96). The above multiple factors contribute to maintenance of a constant core temperature of approximately 37° C against a range of external temperature fluctuations - usually between 15° C and 54° C (60° F- 128° F) (97).

ITI Recording of Deep Temperature Changes

Thermal imaging in medicine addresses the thermal variations in superficial and deep structures of the body . Even though the old literature has claimed that ITI studies the surface skin temperature , as claimed by the U.S.Federal Register (98), to a depth of 6 mm. The research conducted by Elam, et al (99) has shown the test to be informative in evaluating deep temperature changes as well. The skin is an almost perfect radiator of the deep heat. This radiator helps prevent hyperthermia and damage to internal organs (specifically the brain). This radiator, with 98% emissive efficiency, allows the deep heat, that is conducted through the tissue and convectet by blood vessels, to radiate and dissipate in the ambient environment (99). This heat radiation is recorded by ITI (Fig. 2). Different methods have been applied to study superficial and deep circulation (e.g., scintigraphic bone scanning, and ITI) (100). The first clinical application of ITI was to record the thermal changes of deep structures such as breast cancer (101-103), and arthritis (104,105). ITI in Paget's disease has shown pathological hyperthermia in deep structure of the sacral-sacroiliac region. The ITI in Paget's disease showed direct correlation with improvement and reversal of bone changes and pain after proper treatment (106). This is another example of the sensitivity of ITI in identifying the deep tissue pathology (99).

Limitations of ITI

The ITI provides accurately measurable information regarding subtle thermal changes (100). The ITI shows any old or new sympathetic nerve damage or dysfunction, thus confusing the examiner and demanding careful and proper clinical correlation. The examiner interprets the old lesion as the main pathology. The confusing results due to multiple old and new life time injuries may mislead the physician to end up losing faith in ITI. Only proper history taking and interpretation can solve such a problem. However, in a double blind study the above example of ITI will be tagged as invalid.

Another source of confusion is spread of the thermal changes to the contralateral extremity - making it difficult to compare the delta T between the two extremities. As the disease becomes chronic and the sympathetic thermal dysfunction becomes bilateral (1,61,107), the ITI shows identical bilateral temperature changes causing difficulty in diagnosis. This is true both in standard ITI, and in cold stress tests (108,109). The bilateral representation of central sympathetic temperature regulation is modulated at the following centers: The first center is at the chain of sympathetic ganglia on each side of spine. These ganglia relay the transmission of pain or thermal impulses horizontally and vertically(21,110) (Fig. 3). This bilateral integration of the impulses at the sympathetic chain level serves the purpose of transmitting the stressful impulses to the rest of the body, and coordinating the sympathetic stress regulation (21). The second center is at the spinal cord level (61) where the temperature modulation is exerted symmetrically and bilaterally with side to side temperature variation (delta-T) of ± 0.2 - 0.3° C (23). This explains the spread of thermal changes to the contra- lateral side (1). The next relay center for neuropathic afferent nerves and temperature regulation is the hypothalamic modulation centers (107). Finally, at the cerebral hemispheric level, the Central autonomic network (CAN) exerts its influence on vasomotor, visceromotor, neuroendocrine, cardiovascular, and pain modulation. The CAN includes the limbic system-specifically the mesial frontal and insular cortex, amygdala, stria terminalis hypothalamus, and nucleus solitarius (111,112).

Significance of Hyperthermic Regions

In early stage of nerve dysfunction, the involved area is hyperthermic due to release of destructive cytokines (21,47). After a few weeks, the hyperthermic area shrinks. In some cases

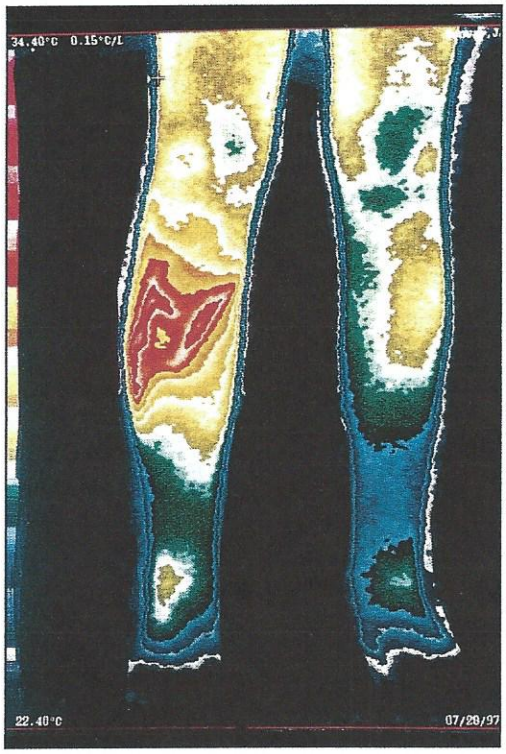


Figure 4
A previously undiagnosed right leg arteriovenous malformation (AVM)over 27mm deep, complicated by CRPS (RSD). ITI identified the deep lesion and spared the patient from the scheduled sympathectomy. Vascular surgery corrected the condition.

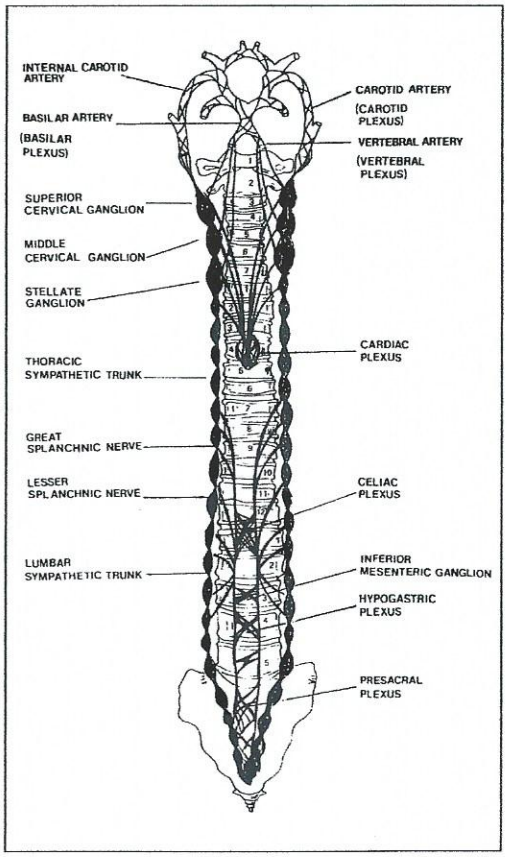


Figure 5
The paravertebral chain of ganglia transmit the neuropathic pain and abnormal sympathetic dysfunction vertically (e.g., from foot to hand and vice versa), and horizontally (from side to side). This explains the remote symptoms and thermal manifestations of CRPS patients.

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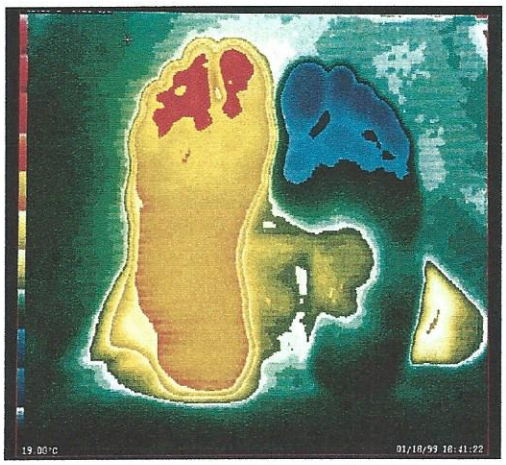

Figure. 6

CRPS nerve damages to right toes after "neuroma exploration". The sympathectomy did nothing for the pain. ITI spared the patient from the scheduled chemical sympathectomy. The left foot showed compensatory hypothermia after sympathectomy.

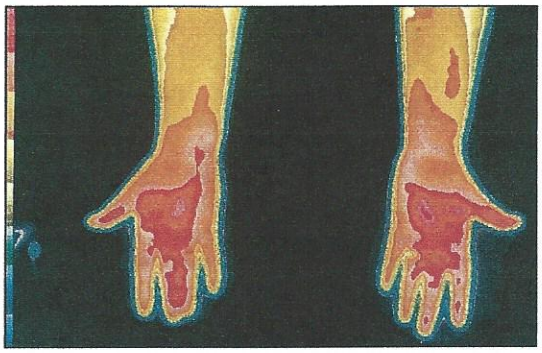

Figure 7
"Virtual sympathectomy" secondary to repeated stellate ganglion nerve blocks leading to permanent sympathetic nerve damage and hyperthermia (heat leakage) in upper extremities. The ITI spared the patient from further sympathetic nerve blocks.

(47) the hyperthermia persists due to permanent damage to the thermosensory nerve fibers (1) (Fig. 4). This bodes a poor prognosis.

Traditionally, hyperthermic areas recorded on ITI have been ignored due to the universal old and partially true dictum emphasizing the importance of hypothermic foci as the main sign of sympathetic dysfunction. This has resulted in the examiner usually not paying attention to hyperthermic foci which are equally significant. Usually, the hyperthermic areas point to either irreversible damage to the sympathetic system in the traumatized focus, or a referred pain area undergoing a backlog of neural transmission of the antidromic afferent sensory nerve fibers (88) to the spinal cord in the form of algogenic chemicals such as nitric oxide (NO) (67-87,113,114), SP (43,44,56-58,60), CGRP (44,115), and oxidative stress agents (116) (Fig. 5). These algogenic pro-inflammatory chemicals play a major role in the function of immune regulation, and when accumulated in large doses cause movement disorders such as tremor, and pro-inflammatory referred pains such as headaches, neck pain, back pain (12,113). Traumatic procedures such as surgical ex ploration(12), needle insertion in hands or feet for nerve blocks, or EMG needle insertion should not be applied to the damaged hyperthermic area in the extremity which may lead to further deterioration and aggravation of the condition (22,117,118). On the other hand, the above treatment should be applied to the referred pain areas in cervical, thoracic and lumbar regions which have undergone no focal nerve damage but are reflecting the backlog and accumulation of cytokines in the path from the distal extremity nerve damage to the dorsal horn of the spinal cord (88) (Fig. 3). This identification of hyperthermic nerve injury is a major therapeutic contribution of ITI.

Physiologic hyper - and hypothermia are the reflection of the dynamic function of sympathetic system (22) to achieve homeostasis. These changes are the end-results of multiple factors such as hyperthermia due to damage to thermoregulatory sympathetic nervous system (117,118) (Fig. 2). The up-regulation of the sympathetic system leads to vasoconstriction and hypothermia. The down-regulation or damage of this system leads to a dermal hyperthermic focus surrounded by a compensatory hypothermia (1) (Fig. 6).

The referred pain phenomenon may be accompanied by hypo - or hyperthermia in paravertebral regions (Fig.3). This form of hyperthermia is due to accumulation of pro-inflammatory cytokines outlined above. These cytokines play a major role in development of inflammation (42,55), movement disorder (119-122), and immune regulation (44, 59,113). In contrast, in chronic late stages, the hyperthermic area becomes more focal and quite small in size surrounded by compensatory hypothermia of the rest of the region (12). The contralateral normal side also undergoes compensatory hypothermia. The end result is two cold extremities with no statistically significant thermal difference (delta T). This problem can only be solved by proper clinical correlation.

Significance of Hypothermic Regions

Sympathetic dysfunction causes sympathetic up-regulation and regional hypothermia. This phenomenon has been blamed as Alpha-receptor supersensitivity to circulatory norepinephrine (NE) after prolonged denervation(24,25). This phenomenon is usually seen in the sympathectomized limb. The limb, instead of being warm, becomes colder after surgery due to end - organ supersensitivity to alpha receptors (24, 25,123) (Fig. 6). This is a major cause of sympathectomy failure (Fig. 6). ITI identifies this form of hypothermia, sparing the patient from further harmful surgical treatment(12) (Fig. 6). In this regard, ITI points to the futility of sympathectomy. Commonly, as the sympathetic system becomes chronically dysfunctional, the prolonged pathologic vasoconstriction yields to inconsistent tonus of the vasomotor nerves. This leads to the development of neurovascular dysfunction, mottling, and instability (124). This refers to blotching, and fluctuation of skin temperature. ITI identifies this condition more accurately, and spares the patient from sympathectomy and ganglion nerve blocks (Fig. 6) which cannot be expected to help an unstable and failed stage of sympathetic dysfunction (1,12).

The Role of ITI in Pain Management

The proper identification of hyper - and hypothermic areas guides the clinician in management of pain, more accurate diagnosis, and avoidance of further traumatizing the already damaged nerves by avoiding unnecessary surgical procedures (51-53),(Fig. 7), or improper nerve blocks. ITI can identify these areas of nerve dysfunction in paravertebral regions of

the spine (91) in form of hypothermic foci. Epidural nerve blocks (with bupivacaine and depo-medrol) in these regions provide both somatic and sympathetic pain relief (12,21,91). According to Stein (88) the cytokines and inflammatory chemicals are transmitted via spinal nerves to the spinal cord and vice versa, modulating the spinal cord function of nociception. The therapeutic effect of these blocks lasts 8-12 weeks (1,12,21) versus ganglion blocks which last a few hours or days.

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