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Thermography and the sensory dermatome

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Abstract. The basic neuroanatomic and physiologic aspects of the sympathetic outflow to the limbs are reviewed and correlated with the somatic sensory dermatomes. The recent literature is considered. A thermocouple thermometer was used to test 30 normal patients and 87 patients with clinically proven nerve root lesions. Thermographic imaging of the sensory dermatome is not plausible, and thermography is not recommended for clinical documentation of painful conditions of the neck, back, or limbs.

Key words: Thermogram – Sensory dermatome – Sympathetic dermatome – Intervertebral disc – Spine abnormalities

Recent articles and presentations on thermography and the musculoskeletal system have claimed strong correlation of thermograms with myelograms, computed tomography (CT) scans, and electromyograms (EMG), in documented herniated discs and surgically-proven cases of nerve root irritation. In 1968 Edeiken described infra-red thermographic patterns in the lumbar spine [3], with asymmetrical temperature changes in the lower lumbar region that correlated clinically with myelograms in a high percentage of cases. These changes were verified in 1976 by Raskin [15], although with less enthusiasm. Both authors speculated that increased temperature changes associated with herniated discs might result from muscular spasms producing surface changes by vascular convection.

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In 1978 Ching and Wexler believed that thermal changes in the limbs associated with nerve root lesions should correspond to Keegan's sensory dermatomes [1] and assumed that sensory fibers to the skin could somehow convey sympathetic impulses to the corresponding blood vessels. Using AGA Thermovision, with 1° C isotherms in the limbs, in addition to lumbar thermography, he claimed a 93% clinical correlation with lumbar disc herniations.

In 1982 Pochaczevsky and coworkers described the use of liquid crystal thermography [10–13] apparently believing in the possibility of thermal imaging of the sensory dermatome. He concluded that " the thermographic examination of extremities of paraspinal dermatomes can accurately determine the anatomical site of involvement in spinal root compression syndromes," and that "it also serves to document objectively the nature of the subjective complaint of pain."

In 1983, a small but well-controlled double-blind study was performed by Mahoney et al. [8] using AGA Thermovision. It showed no significant correlation of thermographic features in 25 patients with proven herniated discs undergoing chemonucleolysis; it also demonstrated significant asymmetry in the limbs in 25 normal subjects.

In 1983 and 1984 numerous papers were presented claiming significant correlation between thermography and clinical findings in patients with nerve root lesions. An interesting presentation by Uematsu, "Imaging the Sensory Dermatome" [20], presented significant temperature differences in peripheral nerve lesions, and from this information concluded that "thermographic imaging of the sensory dermatome is practical for use in clinical application."

Some theories on the mechanism of these changes include:

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1. "As a nerve root is irritated, it sends mixed spurious messages along the course of a nerve, causing capillary blood vessels to spasm" [21].
2. "The longitudinal extension along the sensory dermatome is an expression of the degree of pressure on the sinovertebral nerve (recurrent meningeal nerve) vis-a-vis its autonomic component, and does not require nerve root compression" [22].
3. Sometimes complete block of the sympathetic fibers of the sinovertebral nerve can allow unopposed parasympathetic fibers to produce vasodilitation in the extremity dermatomes [Course material: Thermography, 1984).

The claims for thermography include:
1. "For the sake of comparing it to something else such tests as a myelogram or EMG are used, but they are at best partial standards. Because of the unique information it provides, the thermogram is its own standard" [23].
2. "Thermography can detect nerve root irritation that may be clinically missed by another more prominent symptomatic level, or may even be clinically subliminal" [21].
3. A patient with a positive thermogram at the L4-5 level subsequently demonstrated a thoracic neurilemmoma from T-7 to T-11. This was "obviously pressing on the L-5 nerve *tract*" (of the cord) [22].
4. Changes in the hand in cases of carpal tunnel syndrome are best seen on the dorsum of the hand [Course material: Thermography, 1984].

Material and methods

Since thermal imaging of the sensory dermatome appeared to be anatomically impossible, a review of the literature on the sympathetic and sensory innervation of the limbs was undertaken. In addition, 31 normal subjects and 87 patients with definite neurologic deficits were independently examined. Using a Diateck 500, YSI Telethermometer Series 43A and a Series 396 Atkins 39658-J thermocouple digital thermometer (Figs. 1–3) temperatures of the back and limbs were recorded. Examinations were performed primarily in the office, with women in examination gowns and men in shorts. Room temperature ranged from 70–73° F, and patients were allowed to stabilize for at least 10 to 15 min. Temperatures were recorded in the back to the nearest 0.1° F. In the limbs, temperatures were tested in any areas of existing sensory impairment. Records of previous surgery and documentation of neurologic deficits were precise. Patients with occlusive or spastic vascular disease were excluded.

Results

Normal temperatures in the back ranged from 90.2–94.5° F. No lumbar stripe (increased temperature midline) was noted except with artificial cooling. The gluteal cleft was consistently hotter, 93–95° F, but no consistent increased temperature was found over the sacroiliac joints. Normal temperatures of the thighs ranged from 88–92° F, calves 81–88° F, feet 75–87° F, forearms 82–87° F, and fingers 71–82° F.

At room temperature average normal temperatures range from 92° F in the back to 78° F in the fingers and feet, consistent with the rule that the temperature of the farthest extremities will approximate a midpoint between body temperature and external temperature. Temperatures are frequently quite symmetrical in the back and limbs. In the back, only slight asymmetry is noted (0.2–0.3° F). Progressing distally, the asymmetry increases to 1.0–2.0° F in the feet and fingers.

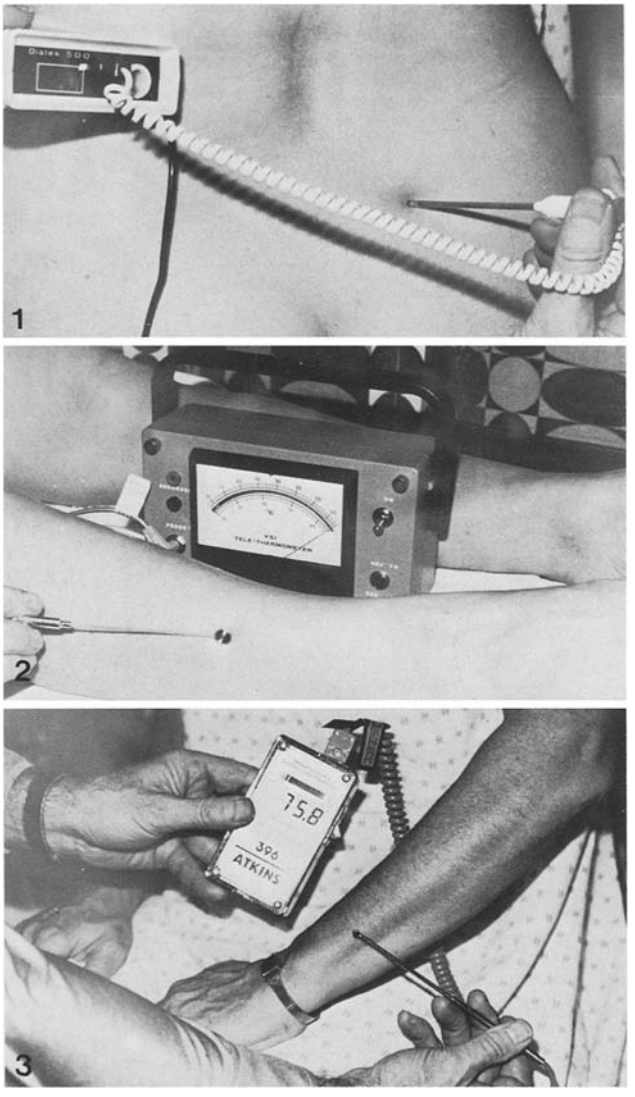

Fig. 1. Diatech # 500

Fig. 2. Yellow scientific instrument # 43TA

Fig. 3. Atkins scientific instrument # 39658

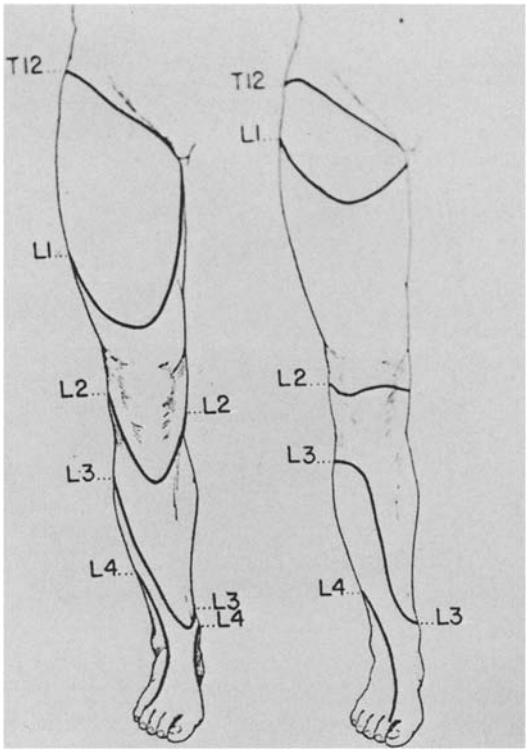

Fig. 4. Richter's sympathetic dermatomes and his representation of Foerster's sensory dermatomes. Reprinted from Ref. 17

In no case could correlation be made between temperature change and a proven neurologic deficit. Some general asymmetry of the limbs was noted on occasions, but it was similar to variations in the normal controls. Statistical analysis was not performed, since no patients showed temperature changes in dermatome distribution, and there was nothing to analyze.

Discussion

The distribution of the sympathetic nervous system to the trunk and limbs has interested many investigators [5].

It is established that impulses from the temperature control centers in the hypothalamus and the vasomotor centers in the medulla oblongata influence sympathetic preganglionic neurons of the intermediolateral cell column and other regions in spinal segments T 1 through L 2. Preganglionic myelinated fibers pass through the ventral roots to reach the sympathetic trunk via the white rami communicantes, either synapsing at the same level or traversing the trunk to more cranial or caudal ganglia before synapsing with postganglionic neurons. The sympathetic chain ganglia above T 1 and below L 2 (occasionally C 8 and L 3) receive preganglionic fibers from the central thoracolumbar outflow only by way of the sympathetic trunk. Each preganglionic fiber innervates up to 100 postganglionic neurons [18]. Postganglionic fibers are distributed to the limbs primarily via gray rami to the roots of the brachial, lumbar, and lumbosacral plexuses. These postganglionic fibers then provide innervation to all the vasculature of the limbs through the peripheral nerves, with the exception of some vasomotor fibers that travel in perivascular plexuses to the very large vessels (subclavian, branchial, iliac, femoral, obturator, etc.). Separate postganglionic fibers innervate the sweat glands

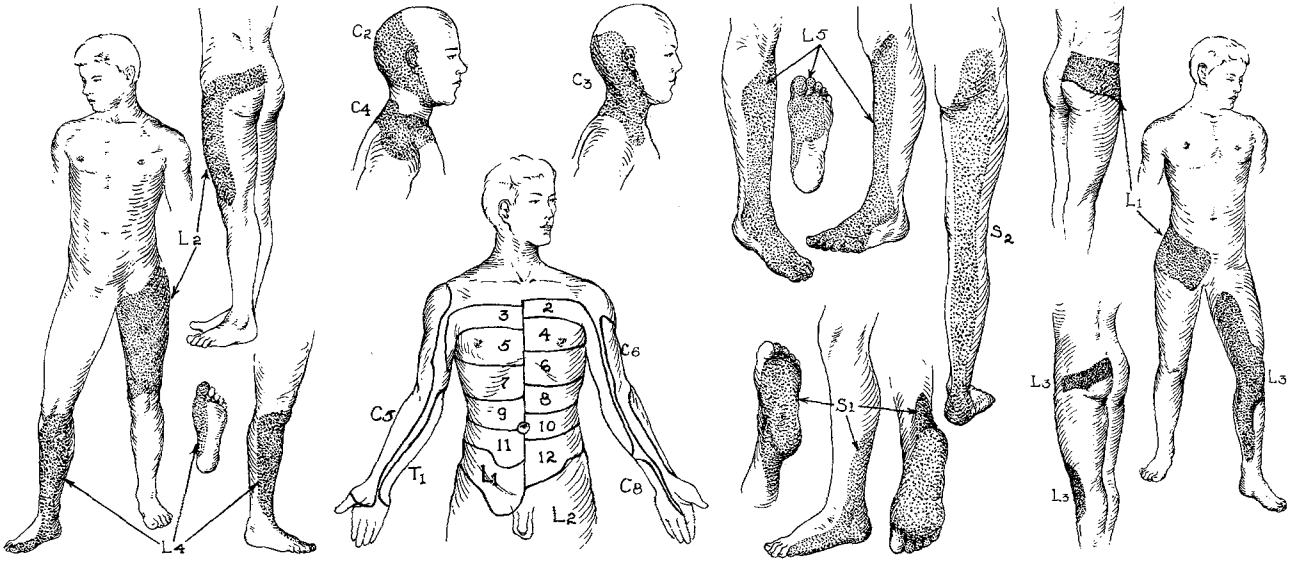

Fig. 5. Foerster's sensory dermatomes as translated by Fender. Fender FA: Foerster's "Scheme of the dermatomes" (1939) Arch Neurol Psychiatr 41:688

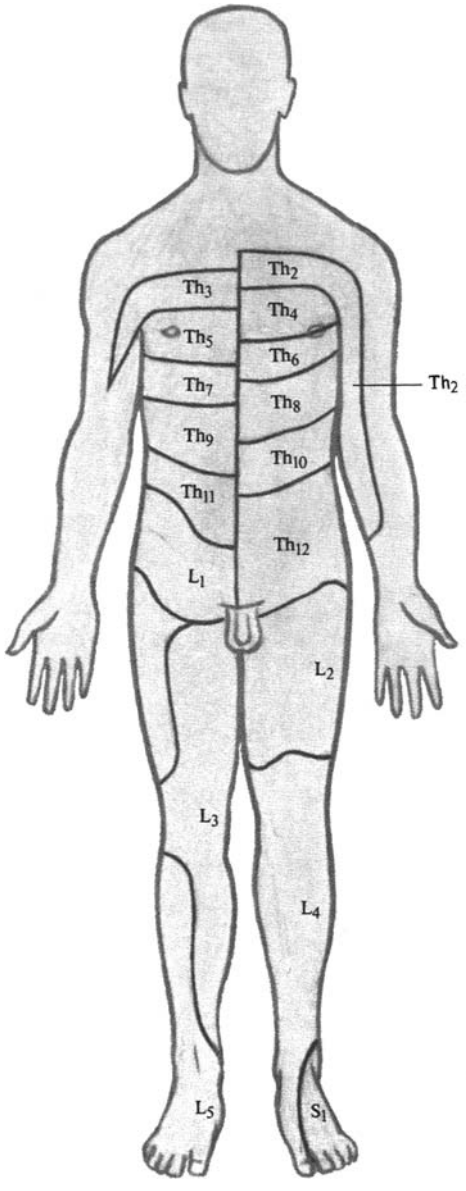

Fig. 6. Foerster's sensory dermatomes as represented by Keegan. Reprinted from Ref. 6

and arrector pili muscles by the same pathways. Thus, blocking the ulnar or median nerve at the elbow will produce a sympathetic blockade, including sudomotor, pilomotor, and vasomotor function, corresponding roughly to the nerve distribution.

Despite reports to the contrary, efforts to map specific somatic sympathetic dermatomes have been unsuccessful. The pioneering works of Folkow and Randall et al. [5, 14] have remained basically unchallenged. In 1945 Richter and Woodruff attempted to map the lumbar sympathetic dermatomes, using electric skin-resistance measurements, in patients who had undergone sympathectomy at various lumbar levels [17]. He described dermatomes which corresponded roughly to Foerster's sensory dermatomes, with the same indirect method of measurement, using the upper level of impairment only. His work, however, is flawed by the small number of cases of the most critical levels (L 4, four cases; L 5, four cases; S 1, two cases). This is crucial, considering the extreme variability of the lumbar chain. His findings are not reproduced by other authors. He erred in referring to Foerster's dermatomes as "sensory and vasomotor." His diagram does not show Foerster's overlapping dermatomes appropriately (Figs. 4–6), and his method of measuring sudomotor function has not produced reliable results for other investigators.

In 1955, Randall et al. [14] surgically exposed the entire sympathetic chain from L-1 through L-5. Silver clips were placed to aid radiologic location of the rami, roots, and ganglia, which were electrically stimulated. Spinal anesthesia prevented spontaneous sympathetic activity, and measurements of sweating, using iodine and starch bands, were made at the thighs, calves, ankles, and feet. After dissection of the chains, a detailed microscopic study was correlated with drawings made during surgery. Sweat patterns varied significantly among individuals, with no evidence of a dermatome distribution pattern. Stimulation of upper lumbar ganglia frequently produced significant sweating of the soles and ankles, with minimal sweating proximally. In other patients, generalized sweating patterns developed with stimulation of lower level ganglia. They conclude that sudomotor, pilomotor, and vasomotor activity are disassociated, since these sympathetic functions are carried by separate fibers. Randall et al. [14] admitted the lack of a practical method of measuring vasomotor function, and specifically showed that in both dogs and humans, no evidence of crossover was found during stimulation of the sympathetic trunks.

In 1974 Normell [9] studied 57 paraplegics, relating sensory, vasomotor, and sudomotor levels (Fig. 7). Using AGA 680 Thermovision, thermocouples to measure room temperature, core temperature, and rectal temperature, with clear plastic covering sheets to measure sweating, he immersed the arms in hot and cold baths, stimulating and comparing sudomotor and vasomotor levels with known sensory levels. He confirmed Randall et al. [14] findings, and found significant disassociation between vasomotor and sudomotor levels. He confirmed the expectation of more proximal sympathetic levels in the paraplegics above T 4, and more

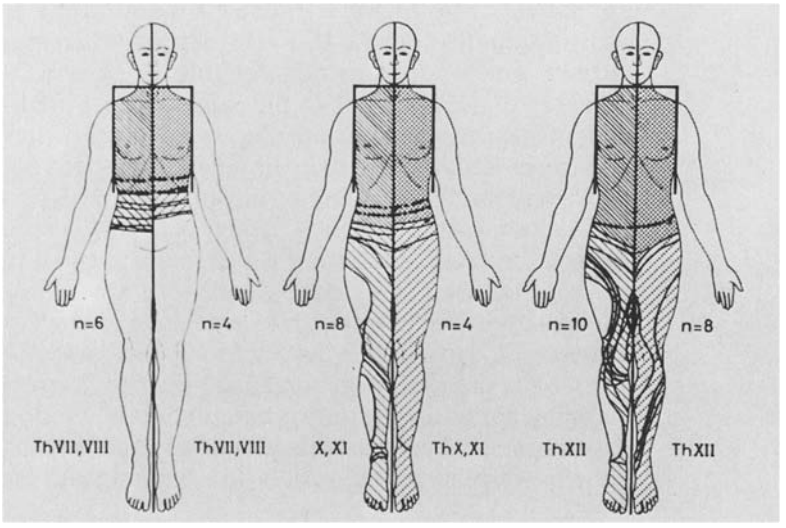

Fig. 7. Sensory, vasomotor, and sudomotor levels in paraplegics. n = number of cases. ThVII, "etc." = sensory level. Reprinted from Ref. 9

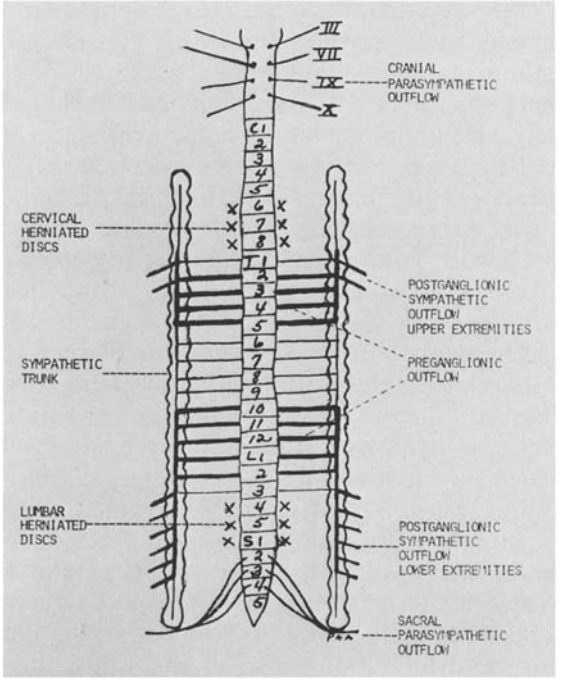

Fig. 8. Diagram: Preganglionic sympathetic and parasympathetic outflow

distal sympathetic levels below T 10, with marked individual variation. His findings are consistent with preganglionic innervation of the lower limbs from spinal segments T 10 to L 2 (Fig. 8). Randall et al. [14], Normell [9], Folkow et al. [5], and Kuntz [7] have all mentioned rare cases which suggest sympathetic outflow distal to L 2.

Few experiments have been made with the arms. However, findings in high paraplegics and results from thoracic sympathectomies for upper extremity vascular disease, confirm that sympathetic fibers arise from T 2 to T 6 spinal segments, and reach the arms mainly by way of the cervicothoracic (stellate) ganglion. Other investigators [16] have produced a sympathetic response in the hands to stimulation as distal as T 9 and T 10.

Additional postganglionic fibers reach the roots of the brachial plexus via the vertebral and middle cervical ganglia. The second and sometimes the third thoracic ganglia supply postganglionic fibers to the first thoracic nerve by way of Kuntz's nerve. No sympathetic outflow originates at any cervical spinal segment.

Sensory dermatome mapping has been more successful. Head's sensory dermatomes of visceral referred pain are used by all clinicians. Foerster's dermatomes (1939) are widely used in evaluation of chronic neurologic lesions. Keegan, in 1948, developed a somatic chart, made possible by Mixter and Barr's discovery of the herniated disc in 1934 [6] (Fig. 9). Keegan evaluated 1,556 patients with single nerve root lesions; 826 verified by surgery or X-ray. He disproved Foerster's statement that single nerve root lesions produce no sensory change, but he did admit some overlap of dermatomal distribution. He used normal volunteers to map the dermatomes of the arms, using local novacaine blocks of the individual cervical roots.

Folkow showed that there are no vasodilator fibers in the dorsal roots [4], and no clear evidence of vasodilator sympathetic fibers to the skeletal muscles in man has been found. The vasodilatation produced experimentally by stimulation of the dorsal roots is best explained by antidromal response, and the possible release of a peptide substance, producing a reaction similar to the axon reflex flare

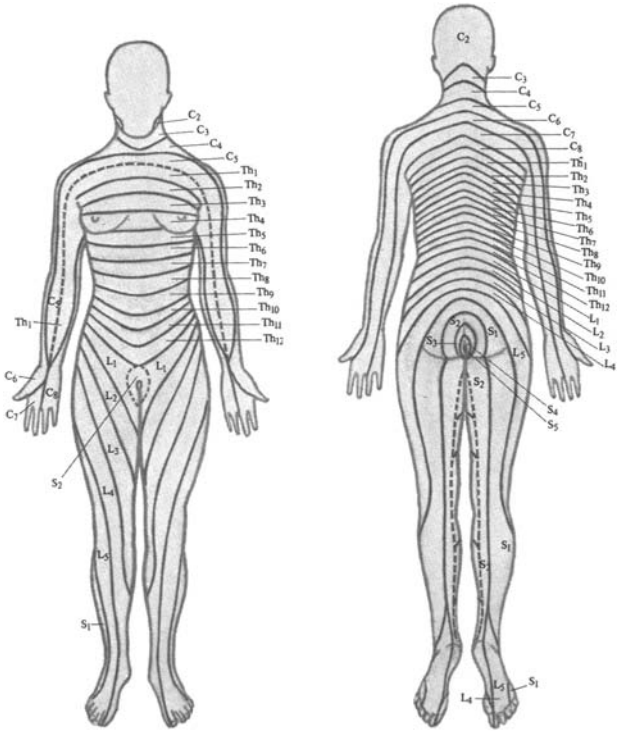

Fig. 9. Keegan's original sensory dermatomes. Reprinted from Ref. 6

of the triple response. Vasodilatation in skeletal muscles, at least in man, is probably the result of local metabolic factors rather than direct innervation.

Pathways involved in reflex dystrophy, causalgia, and peripheral nerve injuries are not within the scope of this article, but sensory and sympathetic reflex spinal arches may be relevant. High circulating levels of norepinephrine are described. Temperature changes are not dermatomal. In biofeedback training, temperature of the fingers has been found to range from 72–93° F, and varies widely with tension states [19].

Correlating the sensory and sympathetic innervation of the limbs, the sympathetic outflow is clearly not represented in the roots of the spinal nerves that supply the branchial, lumbar, and lumbosacral plexuses (Figs. 10 and 11). Therefore, irritation of spinal nerve roots C5, C6, C7, C8, and L4, L5, S1, by herniated discs, spinal stenosis, arachnoiditis, etc., cannot produce temperature changes in the limb dermatomes since these roots contain no sympathetic fibers. Single nerve root lesions at these levels produce no measureable sympathetic change at any level. There is no mechanism by which sensory nerve stimulation can produce a sympathetic response in dermatome distribution. Sinovertebral (recurrent meningeal) stimulation cannot produce vasoconstriction of the corresponding sensory dermatome via antidromal stimulation of sensory fibers [22]. The increased temperature changes in the lumbar region described by Edeiken et al. [3] in patients with herniated discs remain unexplained, but the author himself does not feel they are sufficiently specific to be clinically useful. Muscle spasm with vascular convection is not considered a likely cause [2]. Peptide substance released through sensory root stimulation would raise rather than lower temperatures, and there is no experimental basis for this speculation.

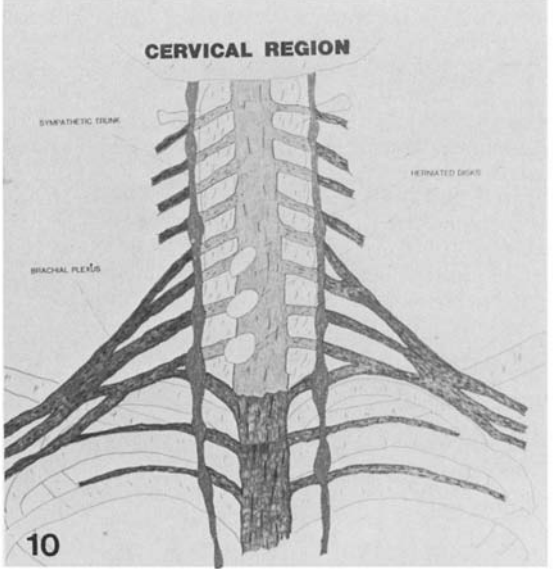

Fig. 10. Cervical region. Relationship of sympathetic outflow to herniated disks

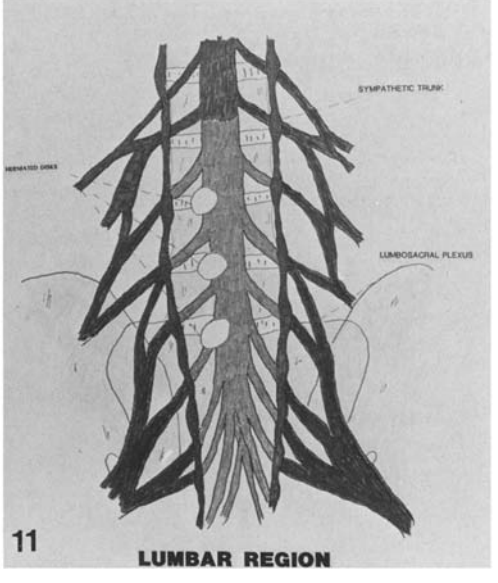

Fig. 11. Lumbar region. Relationship of sympathetic outflow to herniated disks

Explanation of thermography changes

Errors in measurement of both liquid crystal and infra-red thermography are difficult to prevent. Liquid crystal thermography color changes are related to pressure; the plate must be applied equally in all areas, a delicate undertaking. To avoid glare during photography the plate must be removed prior to exposure. Color change begins immediately, and if the plate is not lifted evenly, colors will be asymmetric.

Using color-coded isotherms with AGA Thermovision, differences of as little as 0.1° to 0.2° C will produce asymmetrical patterns. Since each color is ordinarily a one-degree range, and since temperature variation in the extremities is frequently 0.5° C or more, asymmetry of 1° C isotherms should be expected more often than not.

In the lumbar region, thermal focusing techniques such as alcohol baths produce hairline changes on black and white infra-red monitors [22]; these are otherwise undetectable and quite different from the changes described by Edeiken et al. [3] and Raskin [15]. They resemble small blood vessels in the dermis. Any correlation of these changes with underlying disc lesions, presumably as the result of muscle spasm, are highly suspect.

Conclusions

1. Thermographic imaging of the sensory dermatome is not plausible.
2. A research thermocouple thermometer provides an excellent means to verify thermographic features.
3. Further study is required before thermography is used clinically to document painful conditions of the neck, back, and limbs.
4. There are no predictable sympathetic dermatomes.

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