Intraoperative Thermographic Monitoring During Neurogenic Thoracic Outlet Decompressive Surgery

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This article reports the use of thermography to monitor 123 plexus decompressions for neurogenic thoracic outlet syndrome. The diagnosis and management of this disease continues to be controversial. Questions about pathologic mechanisms, the extent and frequency of muscular entrapment, scar, and interdigitations, as well as their relative contributions, remain. Thermographic visualization of the operated extremity allowed us to map and correlate thermal changes with specific surgical manipulations, as well as to analyze the tissues resected to better answer these questions. Initial thermal abnormalities indicating, usually, ulnar entrapments or irritation, normalized sequentially as discrete entrapments were resected. Thermographic monitoring continues to provide surgically useful information in one third of operations.

Introduction

Thoracic-outlet operations used to treat neurogenic pain derived from the brachial plexus have been controversial for decades.[1,2] One of the central difficulties involved in the surgery is identifying the underlying pathophysiology of this entrapment neuropathy. We have found that infrared imaging provides information, as well as answers, to this difficulty.[3,4]

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Skin temperature in a constant environment is exquisitely sensitive to perturbations of the nerve roots, which provide sensory innervation to the imaged skin surface.[5] Irritation or entrapment of a specific nerve root is reflected on the dermatomal skin surface within seconds.

This can be repeatedly and dramatically evidenced intraoperatively by any irritation of a specific root or exposed peripheral nerve secondary to localized traction or pressure with a resultant cooling of the subtended dermatome within seconds. Recovery may occur within seconds, but, more often, requires several minutes, corresponding to the extent of the irritation. Neurolysis and/or decompression of cervical and lumbosacral nerve roots in spinal disease results in comparable increases in skin temperature using thermography.[6]

We report the significance and extent of the multiple entrapments occurring in thoracic outlet syndrome.

Materials and Methods

A total of 123 patients were studied during decompressive thoracic-outlet operations from May 1990 through January 1994. All had primary positional pain in the affected upper one fourth of their body for 2 to 3 years at the time of surgery. The age range was 25 to 45 years (avg, 32.8 years) with an 8:1 female preponderance.

Following initial trials in decompressive neurosurgical procedures, we began monitoring surgical decompression of the brachial plexus in the thoracic outlet because of the inherent difficulties mentioned previously.

A draped visual tunnel was prepared at the same time as the operative site and was continuously monitored during the surgical procedure. An Inframetrics Model no. 520 infrared camera was positioned approximately 1 m away from the dorsum of the affected hand while the supraclavicular ipsilateral decompression took place.

The hand was continuously monitored on a small visual display with a digitized color scale, making temperature changes easily discernible. Compressive neuropathy of the various roots and trunks of the brachial plexus was clearly reflected in temperature asymmetries or decreases, as imaged in the dorsum of the affected hand, most often involving the ulnar distribution. These changes were correlated with the specific surgical intervention taking place. Temperature changes, both in degree and location, were reported to the surgeon. Temperature changes, as displayed on the monitor, were reported both in degree, as well as to location, to the surgeon. Deviations from symmetry indicated which nerve roots continued to be compressed and/or irritated.

Proper positioning and immobilization of the affected arm was important during the surgical procedure because any traction during the procedure (i.e., the shoulder dropping on release of the superficial musculature) was reflected in a global cooling and degradation of the infrared image. Proper positioning and visualization should be possible during alternate approaches, such as the transaxillary one.

Alterations were recorded on color film, which we have found to be more convenient for retrospective analysis than video tape. Controls consisted of 15 surgical decompressions that were monitored bilaterally, using two infrared cameras, visualizing the dorsa of both hands during the course of the procedure.

Results

Preoperative

Preoperative infrared imaging (thermography) revealed varied thermal asymmetries in the affected upper extremity. Characteristically, focal deficits and symmetries worsened with repetitive activity of the symptomatic extremity. Normal images had smoothly varying thermal contour without focal decreases and were symmetrical with the contralateral extremity.

Controls

In the 15 controlled cases, all with unilateral symptoms, where the dorsa of both hands were

Table I. Incidence & extent of dermatomal temperature increases following resection.

Temperature Increase	NL	AS	MS	SM	SU	C7	IN
Incidence, %	84	78	29	11	53	18	35
Extent of mean temperature increase, °C	1.1	0.9	0.7	0.8	0.5	0.7	0.8
SD	0.7	0.4	0.3	0.3	0.3	0.3	0.3

NL = neurolysis; AS = anterior scalene; MS = middle scalene; SM = scalenus minimus; SU = superficial muscles and fat pad; C7 = elongated C7 transverse process; IN = interdigitations; SD = standard deviation.

monitored continuously, the temperature changes during the operation in the contralateral hand (i.e., the non-symptomatic hand) were consistently less than 0.25°C. This was less than or equal to the changes in the body core temperature, as monitored intra-esophageally by the anesthesiologist. Because of the relatively constant temperature in the nonsymptomatic extremity, additional controls were thought to be unnecessary.

Operative (Pathological)

The affected (experimental) upper extremity showed at least a 2.5°C increase in vascular heat during the course of the decompressive surgery with the normalization of the initial, predominantly ulnar, asymmetries.

Following anesthetic induction for surgery, these asymmetries became more pronounced (following a biphasic thermal response during the initial 20 minutes of induction, then remaining stable), affecting most consistently the ulnar dermatomes.

During the surgical procedure, thermographic normalization (increased temperature and symmetry) occurred following specific surgical events. These interventions, with the corresponding temperature increases, are shown in the attached table (Table I).

For example, not all patients experienced a change in temperature with each operative step, indicating that further intervention was necessary. Thus, if scalenectomy showed little or no change, resection of the middle scalene, exploration for a minimus, proximal interdigitations, or as yet nonevident perineural fibrosis was indicated. Overall, this kind of information was thought to be surgically significant in one third (more so initially) of the decompressions.

Each individual surgery differed to some degree reflecting the multiple pathologies involved. One patient might have a dramatic temperature increase with the resection of an obvious interdigitation while another might not. Similarly, a large, loosely adherent perineural fibrotic strand could show no change when removed, but a small (millimeters) adherent fibrosis, when removed, could show a 2°C warming. However, there were significant trends and commonalities as indicated below.

We have not observed any relationship between the first rib and irritation of the lower roots of the plexus. There was no increased heat emission following rib resection. However, an elongated C7 transverse process, with associated myofascial anomalies, was shown to significantly irritate the mid-plexus (C6–7) in 18% of cases. Removal of these anomalies resulted in significant warming, suggesting the importance of elongated C7 transverse processes as a site of initiation and/or attachment of compressive tissues affecting the plexus. We have also found that first rib remnants in thoracic-outlet operations have consistently provided foci of attachment to the elongated C7 transverse process. The resection of these elements resulted in significant thermographic warming.

We have found that the most consistent increase in temperature occurs following neurolysis, which occurred in 89% of cases. Perineural fibrosis/scarring is the most frequently encountered pathology in these patients and is probable cause for a significant, if not major, component of the consequent symptoms.

The next most frequent source of entrapment, based on the temperature increase following resection, was the anterior scalene in 78% of cases, which has long been considered a primary motor in driving this pathosis. Recent evidence from a number of groups has documented the muscle fiber transformation in the anterior scalene in this disorder, resulting in muscles that are more easily tonically contracted, exerting a compressive force on the underlying plexus.[7-9] A similar mechanism has been described in an animal model with current research indicating that pathology is initiated on a microscopic level involving the perineural tissues in which histologically documented changes occur with similar levels of compressive force.[10]

That this compression need be quite minimal in nature is substantiated by the changes that we have noted in a significant number of affected individuals with simple release of the tissues overlying the scalene fat pad, including the omohyoid, accomplished in the preliminary stages of the operative intervention supraclavicularly (see Table I).

Additionally, thermographic observations corroborate the important contribution of aberrant muscular interdigitations, occurring in 35% of cases, arising from the anterior, middle scalene, or scalenus minimus with varied courses, often attaching to Sibson's fascia or the first rib and, as previously mentioned, frequently arising from either an enlarged C7 transverse process or a first rib remnant.

These myofascial bands usually have a very significant fibrous component, which can, over time, contract due to evolving collagenous cross-

linking, thus slowly exerting a more constrictive entrapment. We observed that the perineural fibrosis often extends into the periforaminal space, necessitating a maximal nerve root dissection. This neurolysis can often be monitored by thermographic imaging. Periforaminal fibrosis may account for the large number of individuals with thoracic-outlet symptoms, who are believed to exhibit primarily a cervical spine disorder. Resection of visible post-ganglionic afferent sympathetic fibers has been done routinely. It has resulted in a temperature increase in only 2 cases, underscoring the importance of compression-incited pathosis as compared to aberrant extra-nerve root sympathetic activity.

Discussion

Thermography, when coupled with clinical findings, is useful in the diagnosis of thoracic outlet syndrome. Objective thermal asymmetries can be documented consistently in the absence of other objective indicators, such as electrodiagnostics or imaging.

Twenty-one of the surgical patients (those living locally) underwent preoperative thermography. The same abnormalities were present intraoperatively and the improvements seen during decompression were present 2 months postoperatively, barring significant reinjury. The 3 reinjured patients showed an increased worsening compared to the final intraoperative levels.

The exquisite sensitivity of dermal blood flow to perturbations in the spinal nerve supplying the relevant sensory dermatome makes infrared monitoring (thermography) useful in neurovascular entrapment decompressions. Response to further insult or relief from compressive irritation occurs within seconds. As a result, the rapid warming following the removal of perineural scarring prompted histopathologic studies, which revealed the rich peptidergic (calcitonin gene–related peptide, SubP) innervation of the scar and a possible peptidergic mechanism for perpetuated inflammation that could explain the chronic pain.[11]

Decreases in vascular heat emission in the dorsum of the hand, presumably indicating increased irritation, have been traced to plexus traction occurring during the procedure. These are effectively prevented by improved immobilization and support for the affected extremity during the surgery. Thermographically prompted intraoperative suggestions for further surgical intervention followed a learning curve for the first 20 procedures. Subsequently, suggestions have been found useful in 1 of 3 operations.

These results confirm previous observations. There are multiple mechanisms for this entrapment neuropathy. This becomes apparent during surgical decompression when peripheral heat emission, as visualized in the dorsum of the affected hand, progressively increases in a step-like fashion during the procedure as different structures are resected or removal. Thus, in most cases, the 2.5°C increase at the end of the surgery has occurred in roughly 0.5°C steps involving resection of the anterior and middle scalenes, muscular interdigitations, fibrous strands, adherent fibrous tissue, and perineural scarring. In a subset of cases, 1.5 to 2.0°C increases were obtained from resection of the anterior scalene, thick abnormal fibrous interdigitations, or extensive periforaminal fibrosis (see Table I).

It appears that the initial insult occurs in the vascular endothelium (probably vasa nervorum or vasa vasorum) with platelet-derived tachykinins followed by monocyte/macrophage re-

Table II. Number of cases by category.

Left vs Right	Bilateral Symptoms	Repetitive Work Stress Injury	Redo	Total
58:65	36	51	23	123
	(29%)	(41%)	(19%)	

cruitment, aberrant collagen deposition, and aberrant growth factor expression. The central factors in this hypothesized positive feedback loop are changes in the vascular endothelium and the establishment of aberrant peptidergic nerve fibers. Pain production is probably very similar to the National Institute of Health animal hyperesthesia/dystrophy model involving very light multiple ligatures around large nerve trunks with alternations in the perineural space and the adjoining extracellular matrix.[12,13]

Depending on which components of this model are overexpressed, the various observed "motors" for brachial plexus irritation could be explained. Thus, hypertrophy and muscle fiber transformation of any of the scalenes, the constrictive effect of anomalous muscular interdigitations, variously situated fibrous bands, and perineural fibrosis can all occur to various degrees, explaining the high individual variability of actual pathologic conditions seen. The possible proliferation of cytokine-secreting neurons (see above) as a common factor bears further investigation. This takes into account the mechanism of physical injury, anatomic predisposition, osseous and myofascial anomalies, and a possible neuronal predisposition to repetitive work stress injury.

It is of interest that 41% of the individuals who came to surgery for this disorder have a clear-cut history of repetitive work injury (Table II). Their symptoms were initiated and progressively worsened in work environments requiring the relatively fixed repetitive use of the adversely positioned upper extremity.

Summary

Intraoperative infrared monitoring for neurogenic thoracic outlet syndrome appears to be a sensitive means of determining the site of the brachial plexus entrapment. It is noninvasive, easily used, and provides reassurance that the intervention has relieved existing entrapments.

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