



by James Woessner, MD, PhD

**Understanding the organic and physiological patterns of referred pain helps to identify the true origin of pathology and inform proper treatment.**

# **REFERRED PAIN**

## **VS.**

# **ORIGIN OF PAIN**

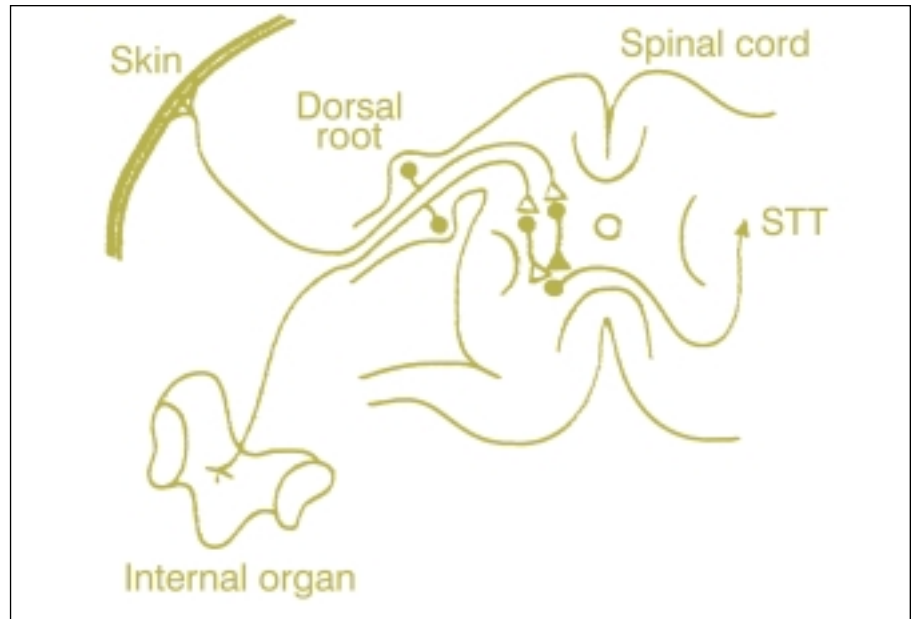
# **PATHOLOGY**

**R**eferred pain, as defined by Anderson, is “pain felt at a site different from the injured or diseased organ or body part.”<sup>1</sup> Radiating pain, however, is not defined by Anderson; radiating pain is more commonly used in connection with pain perceived in somatic nerve and spinal nerve root distributions (i.e. the dermatomes that all physicians learn early in their training). Merskey and Bogduk specify that “referred pain is pain perceived in a region that has a nerve supply different from that of the source of pain,”<sup>2</sup> which indicates that radiating pain is completely different (the author does not find that excluding radiating pain from referred pain useful; radiating pain is just a subcategory of referred pain).

Bellenir adds “Antidromic” into the definition, noting that visceral and somatic nerve cells may synapse on the same neuron at the spinal cord.<sup>3</sup> With chronic stimulation, “the impulse will spill over. . . into the somatic nerve.” Warfield and Fausett also calls it “heterotopic” pain and state that “referred pain is a phenomenon that is frequently encountered and is most baffling.”<sup>4</sup> Added meaning is conveyed by Khalsa, who defines referred pain as “pain that exists in a location other than the immediate area of the spasm”<sup>5</sup> without defining limits, or specific distributions. However, according to Khalsa, the range of the main pain should not be larger than the receptive field, which varies in size depending on the area of the body.

It has been said by the IASP Subcommittee on Classification that “Pain is always subjective. . . .”<sup>2</sup> Yet if the clinician does not understand a presenting pain pattern, where the pain is already considered “subjective,” the chances of justly handling and treating the patient are limited. Indeed, if psychogenic (eg. “subjective”) pain and referred pain become synonymous, then the physician may stop looking for the originating pathology and not provide proper treatment or any treatment at all. The patient is likely to slip into a downward spiral of “doctor shopping.”

However, it must be said that all pain “is always real.” Thus, diagnosing pain pathology — in the face of referred pain that may be perceived as worse than the origin of the pain — becomes a daunting challenge. An understanding of the pain pathophysiology with familiarity of referred pain possibilities, coupled with a



**FIGURE 1.** Note that the pain pathways from the skin surface and from an internal organ pass very close to each other at the dorsal horn. Via ephatic transmission (analogous to an electrical short) causes the brain to mistake pain from the internal organ for pain from the skin (Smith, 2000).<sup>13</sup> Reprinted with permission.

thorough history and physical examination, is essential in making an appropriate and potentially correct diagnosis.

### Referred Pain Characteristics

The best known referred pain patterns originate from viscera and myofascial trigger points. Each type is presented in more detail below.

Ombregt has provided more precise principles limiting and defining referred pain.<sup>6</sup> These principles are paraphrased as follows:

1. radiation is related to spinal segmental,
2. perceived pain site and pathology are on the same side of midline,
3. usually felt deeply,
4. referred distally within a dermatome, but not necessarily throughout the whole dermatome (the author has agreed with this interpretation above),
5. may be contiguous with or may be separated from pain origin.

The author proposes a sixth principle: namely that the site of perceived pain is not tender, whereas the site of pathology is tender. Central pain phenomena do not necessarily fit completely within these criteria, but it is still useful to understand the similarities.

Kosek and Hansson have specifically found that, “referred pain is most likely a consequence of misinterpretation [by the

perceptor] of the origin of input from the stimulated focal pain area, due to excitation of neurons somewhere along the neuroaxis with projected fields in the referred pain area. . . . [and] suggests that the divergence of the input is not reciprocally arranged.”<sup>7</sup>

Before enumerating and describing the various known referred pain patterns, the complexity of pain generation and propagation needs to be reviewed.

### Pain Generators

The author, in a prior article,<sup>8</sup> gives a detailed description of nociceptive, neuropathic and central pain and the neural pathways involved. For nociceptive pain, stimulation must occur at the free nerve endings with various types of signals being transmitted along several basic nerve fiber types. Neuropathic pain, on the other hand, is generated by the dysfunctioning pain nerves themselves. Central (“perceptor”) pain describes dysfunctional perception of pain by neurons in the spinal cord and/or brain. One superficially easy way to distinguish nociceptive and local neuropathic pain from psychosomatic, central, and referred pain is local tenderness, hyperalgesia and/or allodynia.

Above and beyond their identity, there are some basic principles of nerve distribution and anatomy that must be under-

stood to follow the concepts presented here. To understand the generation of nociceptive pain, one must first identify the location of free nerve endings of the sympathetic C-fibers and the A-delta fibers, since the associated free nerve endings are really the only places where nociceptive pain is generated. Radiculopathy is a special case where sensory and motor nerve dysfunction may occur, but we are only concerned with the sensory portion of the radicular dysfunction that presents pain.

Butler presented photographic proof of myriad distribution of fine nerves on the spinal dura.<sup>9</sup> Such evidence strongly implies similar patterns of different densities of these nerves occurring on many tissue planes or interfaces throughout the body. Distribution of nerve fibers on the spinal dura does not specifically assure us that free nerve endings also occur there. On the other hand, it is highly likely that such free nerve endings do, in fact, occur in this potential space, or more generically this tissue plane, as they do in tissue planes throughout the body.

Therefore, it is reasonable to expect that insults (mechanical, chemical or thermal) to the free nerve endings in tissue planes throughout the body may result in pain patterns that are completely consistent with the specific location of the impact on those nerve endings and, therefore, considered to be anatomically and physiologically valid.

There must be an origin of pain pathology before referred pain can be perceived. Some of these patterns of referred pain are well recognized, while others seem rather esoteric.

### Factors Favoring Referred Pain

Ombregt, in describing “factors favouring reference of pain,” concluded that, from pooled experience, stronger central and/or proximal deep (vs. superficial) stimuli more likely cause the perception of pain beyond the pathology.<sup>6</sup> Sclerotomal referred pain is more likely to occur than myotomal referred pain, and much more likely than bone pain to occur. This order of occurrence may be generally inversely related to intensity and pain-related dysfunction.

Marcus adds that “tenacious” pain stimulation is more likely to be referred, superficial pain is more likely to be localizable (less likely referred), deep (excluding bone) is more likely referred, soft tissue

referred pain is less localizable (i.e. more likely referred), and distal pathology is more localizable than proximal.<sup>10</sup>

Although the author, in the course of his practice, has encountered patients with specifically localized central pain, the general rule is that as the pathology is more proximal — progressing from peripheral nerve to nerve trunk; to nerve root; to spinal cord; to brain — the pain is perceived as more generalized, especially as duration increases (i.e. becomes more chronic).

*“(for) patients with specifically localized central pain, the general rule is that as the pathology is more proximal — progressing from peripheral nerve to nerve trunk; to nerve root; to spinal cord; to brain — the pain is perceived as more generalized...”*

### Referred Pain Mechanisms

Various authors (Ombregt, Marcus, Rachlin, etc.) discuss the embryologic basis for referred pain.<sup>6,10,11</sup> Certainly, the referred pain mechanisms must have a relationship to nerve pathways and networks. These pathways and network are geometrically and positionally related to where the precursor structures occurred in the embryo and how these structures migrated during growth, development and maturation. Thus, referred pain patterns have an evolutionarily ancient and developmentally individual relationship to dermatomes, myotomes, sclerotomes and viscerotomes (the “-tomes” are discussed in more detail in subsequent sections below). Percepton pathway and network pathology can also be better under-

stood in the same way.

Rachlin<sup>11</sup> refers to Selzer and Spencer, who suggested five mechanisms for referred pain:<sup>12</sup>

1. “Convergence-Projection” describes one neuron receiving impulses from two sources, i.e. peripheral neurons, resulting in the central pathways not being able to distinguish between the sources.

2. “Peripheral Branching of Primary Afferent Nociceptors” points out that single neurons are very long narrow tubes that may have various branches coming from different peripheral sources, again making it impossible for central pain pathways to distinguish the source.

3. “Convergence-Facilitation” is best illustrated by Figure 1, where ephatic transmission (analogous to electrical “shorting” between two proximate wires) occurs when nerves from two different body areas are in close proximity, resulting in signals from the viscera being transmitted along an associated spinothalamic tract to be perceived in the brain as coming from the skin.

4. “Sympathetic Nervous System Activity” suggests that either restricted blood flow to an area, due to increase efferent C-fiber transmission, causes pain in that area or causes the release of substances that sensitize nerve endings in an area of perceived pain so that hyperesthesia or allodynia occur (this is repeated here for completeness, but the author does not find that this possibility makes much sense; if it did, then tenderness should occur in the area of referred pain without other cause).

5. “Convergence or Image Projection at the Supraspinal Level” describes proximity of neurons in central locations (rather than at the dorsal root) via ephatic transmission or some similar mechanism so that pain is perceived in one area while the stimulation comes from another.

The following are additional possibilities of pain-referral mechanisms:

1. phantom pain; this phenomenon is discussed in the labeled section below.

2. embryologic relationship of the internal organs to spinal levels, which is then directly related to sympathetic chain levels. The importance of the embryologic levels may reflect organization in the central nervous system. In addition, the main nerve fiber type of the sympathetic nerve system is the C-fiber (i.e. a primitive, unmyelinated pain fiber).

Referral Pattern	Underlying Organic/ Physiologic Distribution	Suggested sources for pain referral mappings
<b>Dermatomes</b>	pain nerves at spinal nerve roots	<ol style="list-style-type: none"> <li>1. Moore, 1999 — dermatomes depicted next to peripheral nerve distributions.<sup>14</sup></li> <li>2. Kopf-Maier, 2001 — depictions of dermatomes.<sup>15</sup></li> <li>3. Bonic &amp; Loeser, 2001 — specific mapping of the sensory distributions of nerves from spinal segments and anatomic locations of the innervating nerves.<sup>16</sup></li> <li>4. Brass &amp; Dingle, 1983 — when compared to some of the others, the dermatome distribution can appear to be whole nerve root level off.<sup>17</sup></li> </ol>
<b>Myotomes</b>	pain nerves myofascial tissue planes	<ol style="list-style-type: none"> <li>1. Coda &amp; Bonica — mappings of referred pain from muscle intentionally injected with an experimental substance known to cause pain.<sup>18</sup></li> </ol>
<b>Sclerotomes</b>	pain nerve at the attachment points of tendons, ligaments, cartilage on bone, to some only at the spinal facet joints	<ol style="list-style-type: none"> <li>1. Hackett, 1958 — mapped pain referred from ligamentous and tendon attachments.<sup>19</sup></li> <li>2. Fischer, 2002 — ligamentous trigger point referral patterns.<sup>20</sup></li> </ol>
<b>Viscerotomes</b>	pain nerves lining internal organs refer to other structures possibly by “shorting” of nerves via ephatic transmission as they pass in close proximity at the dorsal horn	<ol style="list-style-type: none"> <li>1. Coda &amp; Bonica, 2002 — a complete depiction of the referral patters of internal organs.<sup>18</sup></li> <li>2. Hardy &amp; Naftel, 1997 — each is, to some degree different.<sup>21</sup></li> <li>3. Andersen, 2002 — each is, to some degree different.</li> </ol>
<b>Thermotomes</b>	referred pain patterns related to the circulatory distribution of sympathetic nerves, which transmit pain signals afferently and autoregulate circulation efferently	<ol style="list-style-type: none"> <li>1. Hooshmand, 2000 — unique, but shows generalized patterns that have been previously difficult to interpret.<sup>22</sup></li> </ol>

**TABLE 1.** Pain referral patterns and identification of underlying organic/physiologic distributions.

3. along these pathways, neuropathic pain can also be referred and, in some cases, may indicate that the nerve is trying to normalize, to heal. Certainly, dead neurons do not transmit pain or any other impulse.

4. central pain syndromes could very well fit into the same category as phantom pain. Both central hypersensitization syndrome and deafferent pain syndrome are consistent with total amputation, and represent pain syndromes with and without, respectively, nerve impulses of any sort coming from the periphery. In other words, the pathology or dysfunction may be in the neurons of the central nervous system, not necessarily just in the brain; collectively, the author calls this system the perceptron.

5. wide dynamic range neurons and interneurons of the spinal cord represent neuropathic dysfunction that could, by specific complex mechanisms, end with the perception of pain where there is no pathology; the pathology, in this case, would be in the spinal cord.

6. sympathetic chain pathology is the same as the spinal cord pathology. We may eventually identify Wide Dynamic Range (WDR) neurons of the sympathetic

chains; we will probably come up with a different name.

7. patchy brain modulation of pain, i.e. antinociception, could well leave the brain appreciating pain, where there is no pain with or without a reason, i.e. nerve impulses of any kind coming from elsewhere.

Healing nerves and tissue may also cause pain through the following mechanisms:

1. inflammation is part of the healing process and the natural chemicals involved are caustic to pain nerve endings. The dilemma here is if you stop the pain, specifically with anti-inflammatory medications, do you stop the healing?

2. muscle spasms or cramping muscles may decrease circulation; ischemia causes pain by promoting a caustic microenvironment around nerve endings. In addition, the spasming/cramping muscles may create pressure on the A-delta and C-fibers nerve endings that exist in the myofascial tissue planes.

3. improper healing of any tissue can reasonably contort it and cause nerve dysfunction. For example, nociceptive pain could come from pressure on the nerve

endings by various configurations of scar tissue, while neuropathic pain could come from the changed anatomy/physiology that result in changes in the chemical micro-environment, or by changes in the anatomy of the long, skinny tube that is the peripheral neuron.

### Referred Pain Patchiness

In addition to the complex referral patterns implied in the above sections, if the nociceptive pathology is patchy or complex, we can expect that the pain referral patterns would be made further complex by the complexity of the mother pain.

The various plexuses of the body, e.g. brachial plexus, may be the best to illustrate the patchiness of tissue plane adhesions that can complicate the anatomic and/or physiologic mechanisms causing the focal pain patterns and the consequent referred pain patterns. If we visualize spreading white glue over the weave of the brachial plexus and then tug and push the surrounding tissue, we can imagine the free pain nerve endings being stimulated at least mechanically and making complex patterns of adhesions that result in more complex pain



patterns and ultimately even more complex referred pain.

Because the small pain nerves (i.e. A-delta and C-fibers that coat the nerve roots, plexuses, nerve trunks, cords) divisions and axon accumulations of peripheral nerves are in close proximity to the fibers more distal down that distribution, the brain, by mechanisms mentioned elsewhere in this article, can be fooled into thinking that the origin of the pain is indeed more distal.

**Other Diagnostic Considerations**

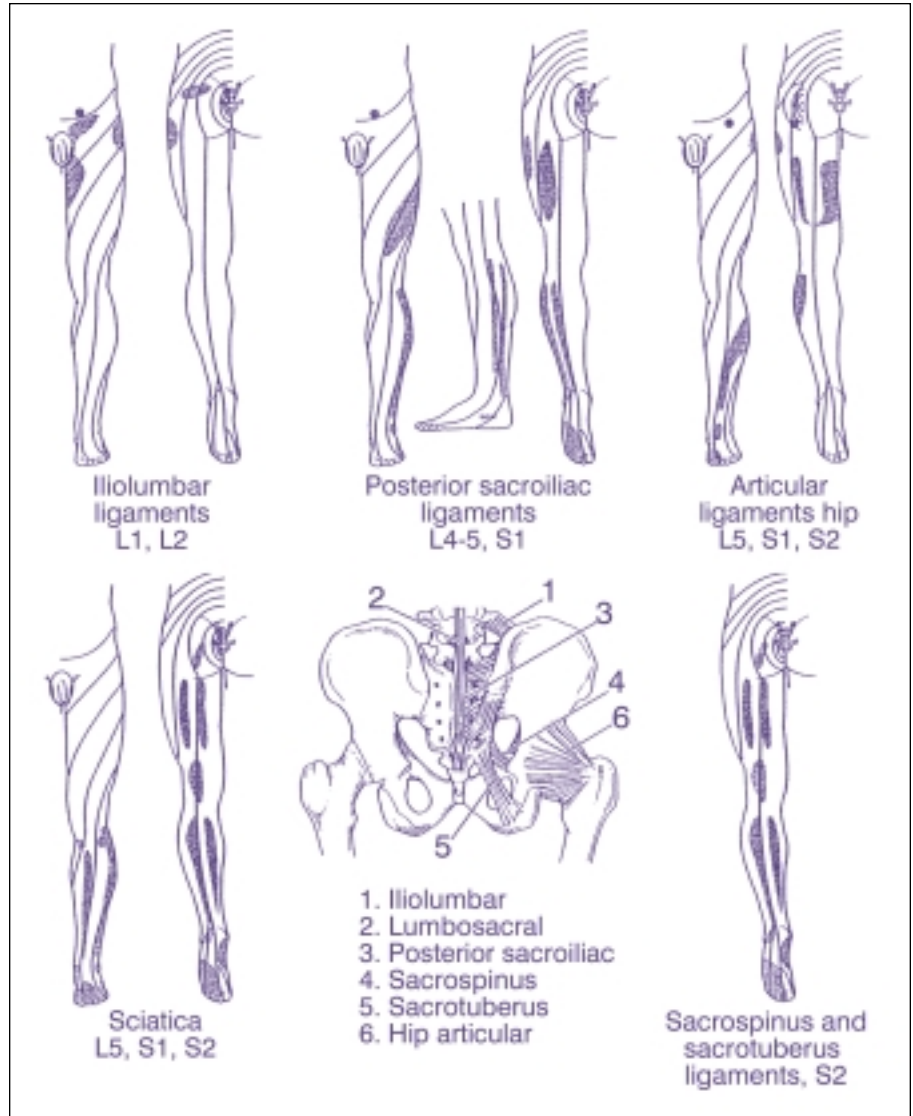
Mappings of referred pain are, by necessity, averages of numerous individual variations in the way small nerve branches grow and develop. This can result in the general boundary between distributions being millimeters or even centimeters different between individuals. Not only do the borders become more erratic, but these overlapping distributions also makes the borders fuzzy. Further, with tissue damage and adaptations, it is easy to imagine that these borders would change over an individual's life time.

It is easy to see with interdigitating peripheral nerve distributions that slight differences in position and/or function could easily result in great specific differences between individuals. Just as the referred visceral pain patterns vary among different individuals, referred pain patterns logically vary between individuals depending on the exact anatomy of the nerve pathways. Because there are also several little-known patterns of referred pain, it is not surprising that most practitioners do not know or are completely unaware of the number of patterns in which referred pain can manifest itself.

As neuropathic processes illustrated above, referred pain can result from neuropathology anywhere along the neural pathway, in the peripheral nerves proximal to the pathology, at the nerve roots, along spinal tracts, and also probably in the sympathetic chains and in the brain.

**Referred Pain Patterns**

Pain referral patterns have been mapped by various authors and identified as "dermatomes," "myotomes," "sclerotomes," "viscerotomes," and "thermotomes," depending on the underlying organic/physiologic origin of pain (see Table 1). The following sections describe



**FIGURE 2.** Fischer (2002) shows ligamentous trigger point referral patterns overlain on lower extremity dermatomes.<sup>20</sup> The similarities are interesting, but probably not directly related in the sense that dermatomes are somatic sensory nerve distributions and trigger point pain referral patterns are more related to sympathetic C-fiber distributions. Reprinted with permission.

these patterns in greater detail.

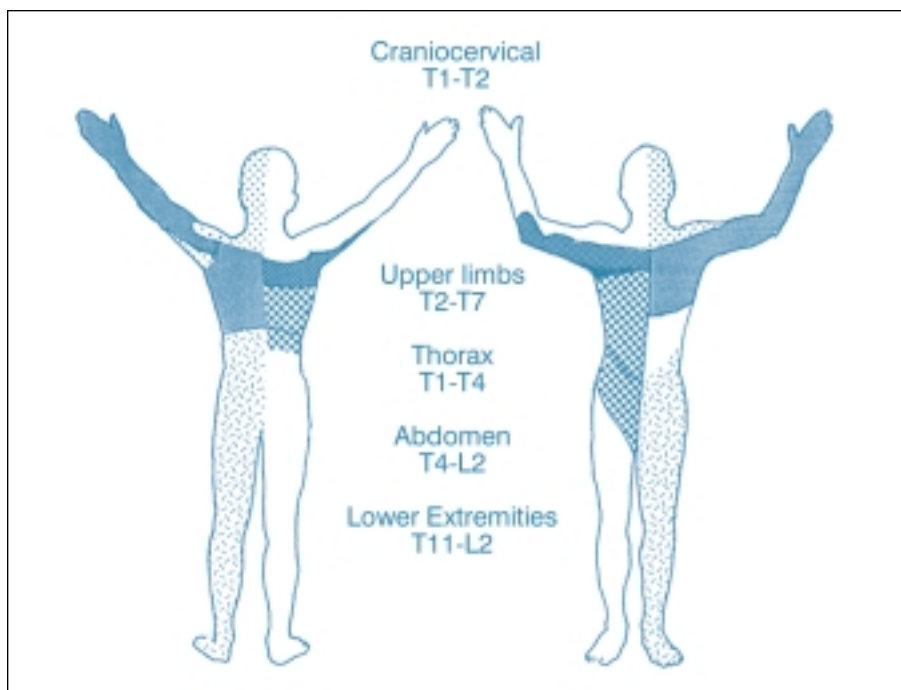
**Dermatomes**

Trained and licensed healthcare professionals are aware of the meaning of dermatomes as distributions of the somatosensory fibers that come from specific nerve roots. Most are also cognizant that every individual may have different specific distributions. It is observed that the peripheral somatosensory innervated areas do not exactly overlap with the dermatomes, suggesting that axons of a distal peripheral nerve probably come from more than one nerve root.

Recognizing that published dermatome maps are representations of average or

common distributions, one example is the C8 nerve root innervation of the lateral aspect of the fourth digit (the ring finger) is in the median nerve distribution, which is mostly made up of axons from the C6 nerve root.

Most physicians can usually determine the presence of a pure acute radiculopathy. A plexopathy or peripheral mononeuropathy distribution of altered nerve function may occur, and the majority of physicians suspect common plexus and peripheral nerve injuries. On the other hand, plexopathy and peripheral nerve injury distributions of symptoms can show up in complex patterns and have complex patterns of referred pain, which few physi-



**FIGURE 3.** Mappings referred to as “thermatomes” (Hooshmand, 2000).<sup>22</sup> Reprinted with permission.

cians can readily understand.

The radiating component of radicular pain is technically “referred pain.” This type of “referred pain” is not a nociceptive process, it is neuropathic, even if momentary. Pain with such a specific distribution seems unlikely to even be central. Radicular pain also typically radiates along a dermatome, and therefore, could also be called dermatomal pain. Dermatomal pain suggests nerve root involvement from a herniated disc or other physical or chemical pathology at the nerve root exit from the spinal canal.

Sometimes myofascial pain referral patterns may follow dermatomes to some degree as shown in Figure 2. Fischer has nicely diagrammed the overlap of myofascial trigger point pain referral patterns with typical dermatomal patterns<sup>20</sup> (see Figure 2). Variations from these typical patterns can be expected due to patchy pathology and specific anatomic differences between unique individuals. There is much to be investigated and considered before an integrated theory really useful to Pain Management can be advanced.

Radicular pain is, by definition, pain that originates at the cervical, thoracic, lumbar or sacral nerve roots. Theoretically, pain down the extremity would not be necessary in order for low back to be radicular; on the other hand, tradition

dictates that there be a radiating component associated with a diagnosis of radiculopathy. As shown by Fischer (see Figure 2), myofascial trigger point referral pain patterns may be the remarkably similar.<sup>20</sup>

Distinguishing these possibilities requires a physical examination by a knowledgeable practitioner to get the correct diagnoses and include all origins of pain pathology. In the author’s opinion, pressure on the free pain nerve endings around the nerve root should be enough for discogenic pain to be radicular. Myofascial trigger point pain can be detected by looking for classic myofascial trigger points, as per Travell and Simons.<sup>23</sup>

### Myotomes

Along with viscerotomes, myofascial trigger point referral patterns are very common and have been mapped by Drs. Travell and Simons.<sup>23,24</sup> As stated above, Fischer has tried to fit these patterns into traditional dermatomes.<sup>20</sup>

The author’s personal experience with referred myofascial trigger point pain occurred about seven years ago. He eventually discovered that his left middle scalene muscle trigger point was referring pain to the extensor muscles in his left forearm. The perceived pain in his forearm did not change to the better or worse with rubbing and massaging those extensor muscles; in other words, these muscle

seemed to hurt, but were not tender. Within seconds of doing ischemic compression to that scalene trigger point with his left thumb, the pain went completely away. Frequent retreatment was necessary over the first few months; now once a month or so is sufficient.

Trigger points may develop from direct impact on the tissue itself or may develop as a secondary response to referred pain.<sup>25</sup> Considering the embryologic relationships of myotomes and neural pathway compensation can help one understand why myofascial trigger points occur at sites of soft tissue pathology.

Note that chronic myofascial pain and fibromyalgia may occur simultaneously, or one may grade into the other. These diseases are completely different at the gestalt level (focal vs. systemic), as well as microscopically. It also been established that referred pain does not occur in classic fibromyalgia,<sup>11</sup> where tender points do not refer or radiate.

### Sclerotomes

According to Rachlin, sclerotomes are pain referral patterns from sites of enthesopathy, i.e. pathology of the collagenous attachments (tendons, ligaments, cartilage, etc.) to bones generated by neurogenic inflammation.<sup>11</sup> Neurogenic inflammation occurs locally, when antidromic nerve signals cause the release of inflammatory chemicals.

Referral of pain from pathology at facet joints, where collagenous tissue is attached to the bones of the facet joints, is a specific subtype of sclerotomal referred pain. Cox<sup>26</sup> indicates that Lora and Loy notes this specific referred pain pattern by artificially stimulating facet joints.<sup>27</sup>

While Rachlin<sup>11</sup> emphasizes spinal segment sensitization, this phenomenon can be better understood by remembering that the sympathetic C-fiber networks are involved and result in a more widespread and fuzzy picture, much like the thermatomes<sup>22</sup> or Butler’s<sup>9</sup> representation of dura-generated pain patterns.

### Viscerotomes

Visceral referred pain is probably the most widely recognized, while still being little understood of all of the referred pain patterns. Lingappa & Farey, in fact, describe “referred pain” as “the phenomenon in which injury to internal organs causes pain that localizes, in part, to surface structures or other organs clearly dis-

tinct from the site of primary injury.” “Typically, the pain is referred to other structures that have the same embryonic origin.”<sup>28</sup> While traditional meanings of “referred pain” are restricted to visceral pain, technically the definitions above fit several other pain conditions, as indicated elsewhere throughout this article.

Cousins refers to these patterns as “viscerotomes.”<sup>29</sup> Visceral pain is difficult for the human brain to locate, because the pain is “referred” to the skin via ephatic transmission (analogous to an electrical short) and/or that “many different afferent sensory nociceptive neurons synapse with the same ascending fibers in the spinal cord” causes the brain to mistake pain from the internal organ for pain from the skin and/or nearby subcutaneous tissues and possibly deeper structures.<sup>28</sup> Lingappa & Farey also suggest that the brain generally will have more recent memory of surface/subcutaneous pain and will “ignore” deep pain until an inciting event occurs.<sup>28</sup> Angina with pain referred to the left arm is a classic, well-known example.

While “activation of visceral pain receptors does not always give rise to a sensation of pain,”<sup>30</sup> the norm, in this context, is to at least expect pain, and sometimes expect pain referral patterns, that can be misinterpreted if not recognized. Pain that becomes rapidly generalized implies perforation and leakage of fluid into the peritoneal cavity. Biliary pain can radiate from the right inferior scapula. Pancreatic and abdominal aneurismal pain may radiate to the back. Ureteral colic classically is referred to the groin and thigh.

Following is a more complete list of some referred visceral pain patterns with a brief description of the respective pain referral patterns. It is assumed there is no dextroposition of the internal organs. Note that one must expect that each patient will display variations on these generalizations.

- Lungs — pain is referred in a collar-like band completely around the neck from about the C6 to T3 levels.
- Diaphragm — pain is referred in a pattern similar to the lungs.
- Heart — pain can be referred to the area around the mouth, but is more commonly referred over the left chest and contiguously down the anterior left arm and directly to the mid-back between the scapula from T4 to T7.

- Gallbladder — pain is referred to superior and lateral right shoulder, offset superior similar in size and circular shape to the superficial distribution of the axillary nerve.

- Liver — pain is referred in a similar pattern to the heart, but only on the right hemi-body.

- Stomach — pain is referred just to the right of midline in the epigastric area and to the mid-back, just below the referred angina from T7 to T9.

- Ovaries — pain is referred to the skin area immediately over the ovaries anteriorly and directly posteriorly, but more lateral.

- Appendix — pain is referred to McBurney’s point in the right hypogastric area.

- Kidneys — pain is referred to the skin area somewhat below the kidneys, posteriorly only, and medial to the posterior referred ovarian pain; there is also an area half way down the right lateral thigh, the right chest just to the right of the lower sternum.

- Ureters — pain is referred to an anterior band across the pelvis, including the groin and the genitals, but not extending around to the back.

- Bladder — pain is referred to a continuous area encompassing the sacrum from S2 down to the upper medial thighs.

Drewes has described some of the dynamics of viscerotomes (visceral referred pain patterns). His observations can be summarized as follows:<sup>31</sup>

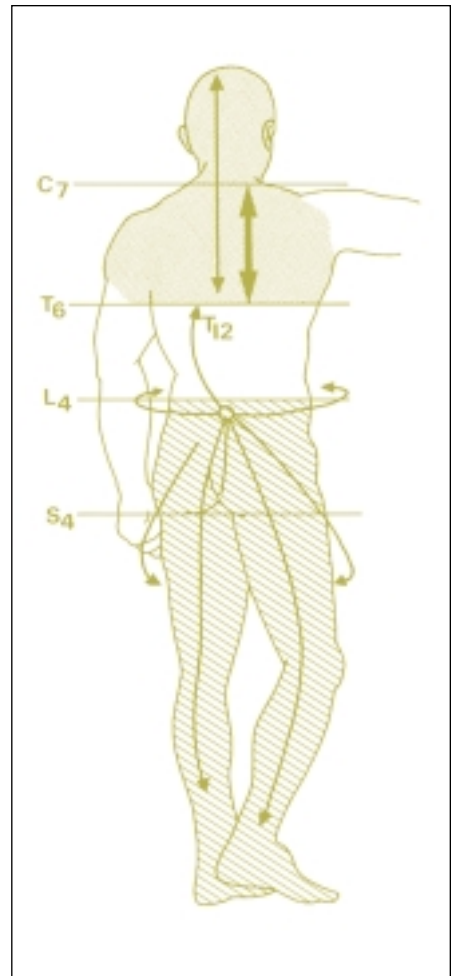
- 1) referred pain does not have to be separated from the area of pathology,
- 2) referred pain spreads over time to a maximum, and
- 3) there is a great deal of variation between individuals.

Drewes also indicates that the site of pain stimulation, as well as the referred pain, are perceived to rise and fall almost coincidentally in intensity over a few minutes. During the first half of this time course, the referred pain is actually perceived as worse than the primary pain.<sup>31</sup>

The origin of pain in classic visceral referred pain patterns can be explained by the distribution of small pain nerves over the viscera of the body.

### Thermotomes

Hooshmand coined the term “thermatomes” to describe pain patterns related to the circulatory distribution of sympathetic nerves. These relatively



**FIGURE 4.** Pain referred from the spinal dura is reminiscent of the thermatomes in being diffuse, but these referral patterns are unique (from Butler, 1991).<sup>9</sup> Reprinted with permission.

amorphous distributions (see Figure 3) are consistent with the observation that these C-fiber nerve pathways end up seeing pain “through fogged glass.”<sup>22</sup>

### Referred muscular pain

Rachlin discusses “referred pain zones.”<sup>11</sup> He states that “referred pain is a manifestation of spinal segmental sensitization.” This spinal segmental sensitization observation does make mechanistic sense and fits into the broad category of being a neuropathic phenomenon. The sensitized neurons at the nerve roots are dysfunctional pain neurons. While the author knows of no direct evidence, deductive reasoning suggests that impulses via ephatic or similar means are transmitted from the site of nociception to the neurons innervating the area of referred pain.

“Myotomal” pain involves the myofascial tissue planes in and around muscles

groups. While “myotomal” may not be the correct description, when muscles were injected with hypertonic saline, which is an experimental substance known to produce pain, the above-mapped patterns of referred pain emerged.<sup>18</sup> We would expect that these would be the same referred pain patterns as myofascial trigger points. Strangely, gross inspections reveals no clear congruence or overlap, possibly indicating that myofascial trigger point referral patterns operate by some different mechanism.

### Dura-Generated Pain

Butler<sup>9</sup> has used Cyriax’ map of referred pain from the spinal dura,<sup>32</sup> which is also probably related to stimulation/irritation of the sympathetic C-fibers on the dura (illustrated in Figure 4), and is reminiscent of the therratones, but these are mutually distinct and unique patterns (compare Figures 3 & 4). These patterns are far removed from the spinal segmented patterns of the other -tomes related patterns. This figure certainly illustrates the concept that the C-fiber pain is seen by the brain through “fogged glass.”

Not only are these pain referral patterns poorly accepted, but the origin of pain pathology as being the spinal dura is even less recognized. A physician could reasonably consider this referred pain pattern as “non-physiologic” without knowledge of this possibility. Certainly, with this pain origin and referral pattern as a possibility, the physician must not take such a presentation lightly, nor write the patient off as having a “psychogenic” pain problem.

If we think of the possible evolutionary origin of the sympathetic chains, which in lower animals transmit all efferent and afferent nerve impulses, those pathways (i.e. the sympathetic chains) may very well be able to reestablish transmission pathways in compensation when normal pathways are lost, much like the development of collateral circulation in strokes.

### Head and Facial Pain

Pains around the head and neck are commonly referred, however, these are seldom appreciated as such, probably because of the short distances involved. Particularly in migraine headaches, phenomena similar to referred pain occurs in addition to the referred pain, i.e. visual and other sensations that are perceived without a distal initiating stimulus.<sup>28</sup>

Guyton & Hall observed that:

- nasal sinus and eye headaches radiate to a wide area around the eyes from below the nose and up to mid-forehead.
- cerebral vault headaches occur frontally to parietally at the ear.
- brainstem and cerebellar vault headaches occur from the ear through the entire occiput.<sup>33</sup>

As for the referred pain component, the origin is probably around vessels that are vasodilating and vasoconstricting on the meninges, and subsequently the pain radiates to behind the eyes, usually unilaterally. Throbbing is, by definition, relat-

sected tailbone.

Note that, as with all pains with central components, non-existence of the perceived origin of pain pathology makes no difference in the perception of pain, which is certainly true of all referred pains. However, it does not necessarily follow that all referred pain has a central dysfunctional component. Further, stump and neuroma pains post-amputation are not referred pains, and therefore, should not be mistaken for phantom pain. There seems to be surprising confusion about these pains versus phantom pain.

Repeating the above meaning, it may be possible to have phantom pain of a body part that is not missing as evidenced by abdominal pain in spinal cord injured patients. However, there is also the possibility that this pain may be “real” and actual pain from the perceived site of pain, where pain nerve impulses pass through some other continuous pathways to the central nervous system, such as through the sympathetic chains.

### Central Hypersensitization vs. Deafferent Pain

Central hypersensitization syndrome merely describes the situation in which central neurons are sensitized such that normally sub-threshold pain impulses are perceived as pain in widespread regions of the body. From the author’s own experience with small fiber,<sup>34</sup> it is clear that these perceptions can be patchy. Pain need not be perceived as coming from everywhere. Central hypersensitization can also be described as widespread hyperalgesia or allodynia, i.e. the patient is very tender, more in some places than others. Specific mechanisms for this behavior are reviewed by Rachlin.<sup>11</sup> Rachlin also presents evidence that fibromyalgia fits into this rather wide category, based on current knowledge.

Likewise, deafferent pain syndrome need not be manifest as “whole body” pain; it can be patchy. The contrast here is, like phantom pain, no peripheral input is theoretically necessary in a pure deafferent pain situation. These patients are non-tender, or not remarkably tender. They seem to be detached from the world, but preoccupied by their pain. This type of pain, on the other hand, can be very specific and focal. This syndrome, in the author’s opinion, describes precisely “phantom pain” of a body part that is still physically present, but not sending pe-

*In a sense, phantom pain is the ultimate “referred pain.” Perception of the pain is obviously not where the pain is originating, since there cannot be peripheral pain nerve stimulation.*

ed to the heart beat, thus, stimulating and radiating in synchrony with the beating of the heart.

### Phantom Pain

Phantom sensations and pain merely mean that the brain perceives the existence of a body part from which no nerve impulses could possibly be emanating, such as from an amputated limb, and is a well-described phenomena. In a sense, phantom pain is the ultimate “referred pain.” Perception of the pain is obviously not where the pain is originating, since there cannot be peripheral pain nerve stimulation. The author has even had a patient with phantom coccygis of a re-



ripheral pain signals to the central nervous system.

## Conclusion

Understanding referred pain is important in determining the true origin of pain pathology so that proper diagnosis and treatment occurs. The complexities of different referred and radiating pain patterns presented in this article demonstrate that understanding pain requires specialized and diverse knowledge and wide experience. Suggesting that complaints are “non-anatomic” or “non-physiologic” may very well be a clear indication of the diagnostician’s limitations rather than a true reflection of a patient’s pathology or psychological state.

In the defense of these well-meaning and intelligent health care providers, it really does require a specially trained physician to artfully and effectively decide the primary cause of a patient’s pain problem and to pick the best and most effective treatment early in their care. This diagnostic exercise is the essential first step in deciding on a theoretically-based and pragmatically-possible and effective treatment plan.

Interestingly enough, these different pain etiologies and patterns are most directly helpful in dealing with nociceptive pain. In other words, these pain sources and referral patterns basically represent normal neurophysiologic functioning and, by and large, provide the patient and the physician with wonderful information for determining a good working diagnosis and treatment modalities for nociceptive pain. However, reality is much more complex.

Based on available data and experience, the author concludes that referred pain is neuropathologic and not nociceptive. The referred pain phenomenon itself is apparently a “dyna-”neuropathic process — that is, pain nerve dysfunction. This conclusion, then, categorizes referred pain as a problem with the wires/wiring or it can also be central, i.e. the “perceptron” as described in the author’s prior article.<sup>8</sup> ■

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