The Neurology of Posture

The Peripheral and Cranial Nerve Work of Jean-Pierre Barral and it's Relation to Pain, Neurogenic Inflammation and Structure

This paper began with a workshop led by Jean-Pierre Barral, D.O. on treating cranial and peripheral nerves. As I took his techniques into my practice, and my palpation skills improved, new questions arose daily. I would often observe nerve fibers, which initially had the consistency of hard plastic or copper wire and were often very painful, that would soften in a matter of seconds to minutes to the feel of jell-o, along with a diminution or disappearance of the sensitivity.

Nerves may have a mechanical (as well as an electrochemical) influence on skeletal relationships and hence on the body's ability to relate effectively to the gravity field.

Dr. Barral talked very little about the neurophysiology behind his work. He had some cryptic remarks about "current of injury" and a description of the anatomy of a nerve bundle, which I will discuss later on. But what makes nerve fibers hard and taut he didn't say. Nor did he offer to explain how the manipulation he taught caused them to soften and lengthen. Barral has a book out in French which presumably answers some of these questions. (Unfortunately, French is not a talent of mine.)

Academic papers don't generally start with a personal narrative, but academic papers don't often set out to correlate some subjective palpatory experience with the experimental results involving neurotransmitter assays or rats who give their sciatic nerve to the advancement of science. We owe much to the rats. They've given a lot.

The Search for Truth

I spent my summer vacation catching up on pain research. I began with some online articles on neurogenic inflammation. Please understand that "neurogenic inflammation" was hardly a concept 10 years ago. So this was new. I got a couple collections of articles on fibromyalgia and one on chronic pain — many, many pages on neurotransmitters. Several concepts popped up that I was unfamiliar with. I realized a lot had happened in the last 10 years. It didn't have much to do with my palpatory questions, but the concept of "central sensitization" kept intriguing me. It was tempting to see central sensitization as a bigger version of "facilitated pathway" which was a formulation in the '70's by Irwin Korr that neurons that fired together like to do it more often — that there was an ease in neural familiarity.

Central sensitization, briefly, is the concept that pain pathways which send frequent barrages into the area of synapse in the spinal cord cause a change in the milieu of the cord. This change in the neurotransmitter soup makes it easier for nociceptive (for now,

read “pain”) input into the spinal cord to fire the neurons that travel up the cord to the brain (2nd order neurons). And if the neurons to the brain can fire more easily, it’s possible to experience more pain for less input.

So in an extreme case, like fibromyalgia, you can see that the entire cord might be lit up with central sensitization. If you step back a bit you may also see that what we have is a hotbed of activity being stirred up by nociceptive input, and that nociceptive input is itself made easier by the hotbed of activity. All of this had seemingly nothing to do with the path I had set for myself, which was to show a nice, neat connection between neurogenic inflammation and the behavior of neurons under my fingers. But I was hooked.

Then I ran across the term “dorsal root reflexes”, which led to another whole series of Google searches. The “dorsal root” itself (not the reflex) is simply the collection of sensory nerves that enter the spinal cord at a single vertebral level. There is a dorsal root ganglion, where the cell bodies live, just outside the spine. We’ll go over this in more detail with pictures and everything. But the dorsal root reflex was something new for me.

This reflex happens when our hotbed of activity causes nerves to fire “backwards” - so to speak. The nociceptive fibers which bring information in from the periphery are now being fired at the proximal end, i.e. at the spinal cord, to take information back out to the tissue where they originated. But in some cases when the hotbed of activity gets stirred up enough, the mechanism is not so discriminating. It can trigger neurons at other spinal levels or different neurons at the same level. It can even be on the other side of the body.

Researchers create an arthritis in the left knee of our courageous rat and, behold, soon after an arthritic condition emerges on the right knee... unless the nerve to the right leg is severed.

What these nociceptive fibers bring back down to the peripheral tissue is the stuff that causes inflammation — neurogenic inflammation. You start to get the picture? Nerve cells that transmit nociception in one direction are creating inflammation in the other. In the laboratory researchers create an arthritis in the *left* knee of our courageous rat and, behold, soon after an arthritic condition emerges on the *right* knee... unless the nerve to the right leg is severed. Then nothing happens on the right knee. The rat doesn’t walk so well, but hey.

If you’re where I was at this stage of the discovery, there is probably, like the sun beyond the horizon, an early sense of the enormity of the potential of these connections. There is this sensitization process and a reflex that sends antidromic impulses to the periphery, which means inflammation can be created, well, anywhere.

About Barral’s Technique

The technique that Dr. Barral teaches is, on the surface, disarmingly simple. Treat the nerve about the way you would treat a liver with visceral manipulation. In fact, Barral

began the class having us treat a liver as a way to free up the right shoulder - because both are innervated by the 5th cervical nerve. He had us do a compress-release technique on the liver and then transfer it to the nerves. That's when it starts to get complicated. The hardest parts for me have been learning to palpate all the ways that nerves can feel and then learning how to engage them in all their various presentations.

Structural Relations

For the better part of a year I've been using Barral's techniques and discovering their strengths and limitations. I've had many surprises. The first surprise, as I mentioned, was how hard and rigid even tiny nerves can become. In that state they are practically impervious to stretch.

Another — more profound — surprise was to discover that joints are often restricted, not from *myo*fascial tension, but from *neuro*fascial restriction. — from the fact that nerves, when they become swollen and hard, have the consistency of tendons. And many of the places where clients asked, "Is that a muscle knot", and I said "yes", are not muscle after all, but swollen nerves. I've reached the point in my work that I check for neural tension *before* I look at myofascial or skeletal relations.

What's even more interesting, when I use Barral's techniques, these "tendons" lose their tendon-like quality and go back to being more like neural tissue. When they do, the joints they cross behave normally. (OK, Now do I have your attention?) We have a lot of discussion to demonstrate this, but it looks like inflammation is created within the restricted space of the nerve bundle, and what was the longitudinal elasticity of the nerve became taken up by the transverse increase in diameter. This means that nerves may have a mechanical (as well as an electrochemical) influence on skeletal relationships and hence on the body's ability to relate effectively to the gravity field. While a significant part of the big picture, it is only a part.

Schleip's Contributions

Robert Schleip has been at the forefront of our community for years in searching for the neurological role in the changes that take place during manipulation of fascia. He is also, no doubt, a Google Master. In his paper "Fascial plasticity – a new neurobiological explanation: Part 1"[22] he puts forth an argument which examines the possible role of Group IV mechanoreceptors in changes which occur during manipulation.

Group IV fibers play a large role in my discussion: however, I will focus on their nociceptive/inflammatory properties. Inflammation within the nerve trunk may cause changes in the body habitus, as I have noted, by creating tensile structural elements from nerve trunks, and, I will argue, by exciting motor neurons carried in those nerve trunks, causing contraction in the muscles they innervate.

The Theatre

The drama of inflammation/nociception/pain takes place in the larger theatre of immune and stress response, which include reactions from and to central centers, such as immune cytokines (messenger molecules) traveling to the brain during infection and making you feel sick. Responses to stressful events can trigger neurogenic inflammation by way of either 1) the sympathetic nervous system or 2) the Hypothalmic-Pituitary-Adrenal (HPA) axis. The HPA axis is an endocrine corridor that plays an essential activation role throughout life and is particularly important during the bonding experience of infancy.[3]

It is clear to me, in my limited experience, that the work with peripheral and cranial nerves is an avenue into this theatre. How much of an effect it can ultimately have, I have no answer. I am surprised daily by the global responses to very local interventions.

The goal of this paper to introduce the concepts of neurogenic inflammation and central sensitization in as non-technical way as possible and to show the relevance to working with peripheral nerves and, finally to suggest that these phenmena may be central to the practice of somatic manipulation. As this research began, I was encountering new concepts daily. To organize these in a coherent way requires some logistical planning. I'll present the concepts as follows:

- Inflammation in general and neurogenic inflammation in particular
- The neural framework of nociception - how the various cell types are related
- Primary afferents - from the periphery to the spinal cord
- The dorsal horn of the cord and secondary afferents
- What is central sensitization?
- Dorsal root reflexes - the key to neurogenic inflammation
- Ectopic stimulation - excitation of a nerve cell in mid-fiber
- The anatomy of a nerve trunk
- How immune cells interact with neural tissue
- CRPS - Complex Regional Pain Syndrome
- The relevance of all this to somatic practice

Embryology

It always helps to start at the beginning. At the beginning of the third week of life, the embryo is a tiny disc of about a quarter of a millimeter across and shaped rather like a pita bread suspended between two fluid-filled balloons - the amniotic sac at its back and the yolk sac in front.

A midline is established (cause unknown) and a groove develops in the ectodermal layer (the future dorsal side) of the pita bread, that will be the basis of the future nervous system. The sides of the groove arch up and join, forming a tube. That tube is the pre-

cursor to the spinal canal - the opening at the center of the spinal cord. I don't know how many cells there are in .25 mm but it's not a lot — compared with the trillions of cells that will develop from them.

Cells begin to divide and climb over one another, phalanx upon phalanx. They march forward with brothers-in-arms forming the columns that make up the spinal cord and the neural axis. The embryo grows longer and the neural columns stay connected. Cells in the same column came from the same progenitor cell, so these cells are brothers and sisters, and cousins and uncles. They're family, they talk together, they have lunch. So if you have a chronically inflamed nerve in your calf, maybe you can feel sensation in your arm when you press on the calf. Cell columns talk. When they get excited, they talk more. Maybe they're Italian.

Inflammation

Inflammation is essential to the existence of higher life forms. It is a complex process and a very central process in the body's interaction with the environment. As you consider our discussion of the inflammatory process and the ways it apparently goes wrong, it may help also to think of the response time that an organism, such as a human being, for example, has to recognize an invader and find a way to destroy it. We will see that the system has a certain amount of "overkill" built into it, which is designed precisely to get the whole organism responding in emergency. We'll also see where that very responsiveness is *the problem* when things don't resolve in a timely manner.

Vasodilation

Inflammation is initiated either by cell injury, by an immune response to apparent non-self, or by neuropeptides secreted by nerve cells - thus the term "neurogenic". In all three scenarios, the first step involves an alteration of the blood supply to the area. When there is cell damage, vasoactive chemicals flow from the rupture in the cell membrane into the intercellular space and cause dilation in the arterioles and constriction in the venuoles.[16] In an immune response to a bacterial or viral intruder, the immune cell releases cytokines (a category of messenger molecule) to do the same job. In neurogenic inflammation the job is done by a neuropeptide called Substance P (as well as others). ("Substance P" is easier to remember than "calcitonin gene-related peptide".) In each case the vessels dilate, blood pools in the area and the flow of the blood is slowed.

The pooling blood creates the heat and redness associated with, for example, a sprain. The same vasoactive molecules cause the cells in the capillary walls to loosen their grip on each other which allows immune cells and larger molecules to "leak" into the intercellular space. This creates osmotic pressure for plasma to follow. The fluid pooling causes edema - the swelling that accompanies injury.

In the drama of inflammation there are a couple of subplots. One is chemotaxis. Messenger chemicals get released from various cells in this process. Some have jobs to notify neutrophils (a particular kind of lymphocyte) and fibroblasts (the carpenter's union) that there's a party going on.

Other messenger molecules interact with the free nerve endings of nociceptors to continue to remind you that inflammation is happening. This sometimes-poignant message you experience as pain. In fact when the inflammation first started, when cell products leaked out of the cell, several of them were quite skilled at notifying you also that damage was occurring.

Breakdown and Repair

There is one sub-plot that is particularly important — especially when the inflammation becomes chronic. Part of what goes on while your body-part is swelling and throbbing is the sub-drama of breakdown and repair. Cells, such as fibroblasts, and molecules, such as fibrinogen (which is a precursor to fibrin (which makes collagen)) are released into the mix. And a category of enzymes called "matrix metallo-proteinases" (MMP) are particlularly important when the inflammation becomes chronic.

MMP's have the job of breaking down tissue, collagen and elastin, that has been damaged, so the repair crews can build new stuff. But when the inflammation does not resolve — periodontitis is a good example — MMP's start tearing down collagen that your body would have preferred remained in place - like the ligaments that hold your teeth in your jaw. You can name any of the chronic "itis's". These guys are unpleasant fellows to have around in chronic inflammation. To the degree that neural stimulation contributes to chronicity, it is important in this discussion.

Neurogenic inflammation

Neurogenic inflammation is a special breed of inflammation. It is initiated in tissue by substance P and other neuropeptides which are secreted by particular nerve fibers. While it plays a role in all inflammatory processes, neurogenic inflammation can occur where no tissue injury has taken place and no immune response to any invader has occurred. Which means it can start its own party, then invite the other players. How it does this we'll get to shortly, but it apparently plays a leading role in several of major chronic inflammatory conditions, from arthritis to migraine to fibromyalgia. It also plays a role in chronic nerve pain or "neuropathic pain."

It is tempting to think of the phenomena I discuss as "medical" cases, as events that happen to the relatively few. However, I find hot, swollen nerves in almost everyone I work on. I suspect that neurogenic inflammation and inflammation in nerve trunks are the rule rather than the exception.

A Primer on Nomenclature and Depolarization

Whoever devised the names for nerve fibers was not a marketing guy. Fibers are categorized by diameter and transmission speed, in a slightly confusing combination of Roman numerals, Greek letters and Arabic letters and numerals. I'll use the Roman I-IV to differentiate the sensory fibers. I and II are the DSL lines of nerve transmission, large diameter, heavily myelinated fibers that carry proprioceptive information, position sense, and light touch. Group III is thinly myelinated, slower than DSL and carries nociceptive and other sensory information. Group IV has no myelin, dial-up speed and is dedicated to nociception. Hey, someone has to do it. Group IV's are also known as C fibers. Just remember "C fibers make Substance P", and you'll have the essence of neuroscience.

When neurons fire, it's an electrical event. The presence of large protein molecules inside the cell causes a strong negative charge. The firing of neurons which synapse on the cell in question changes the strength of the charge. When that negative charge approaches neutral an electrical spike called an action potential (AP) is generated and travels the length of the fiber. Excitatory synapses (like the C fiber synapses) add positive charge and bring the cell closer to firing. Inhibitory synapses, such as descending neurons from the brain which modulate the effects of C fibers, increase the negative charge and move the secondary cell farther from firing. It is the balance between excitation and inhibition which determines when and if the cell fires.

Something else you should know: After a primary cell fires, the changes it induces in the secondary cell degrade over time. If primary cells fire rapidly enough there is a summation of the effect called, no surprise, "temporal summation". Or if another chemical were to change (prolong) the degradation in the secondary cell, it would have the same effect.

Depolarization

Nerve tissue is excitable by any of a number of factors occurring in the inflammatory conditions described, Many of the chemicals floating about can cause depolarization (nerve firing), which is experienced as pain if C fibers are excited or as spasm if motor nerves are involved. In addition mechanical forces like over-stretching and compression cause nerve cells to fire.

Ectopic depolarization means the action potential begins as a point along the fiber instead of at the periphery for sensory nerves or in the cord for motor nerves. The action potential spreads in both directions. In the C fiber this means ectopic stimulations does double duty causing pain at one end and inflammation at the other. When the AP travels in the usual direction, it is called a prodromic impulse. The AP traveling in the other direction is antidromic.

Since we're interested in structural effects, I want to point to the effects of ectopic firing on the motor system. We haven't talked much about motor nerves. There are two types of motor nerves in the nerve trucks that we're discussing: 1) large diameter "alpha"

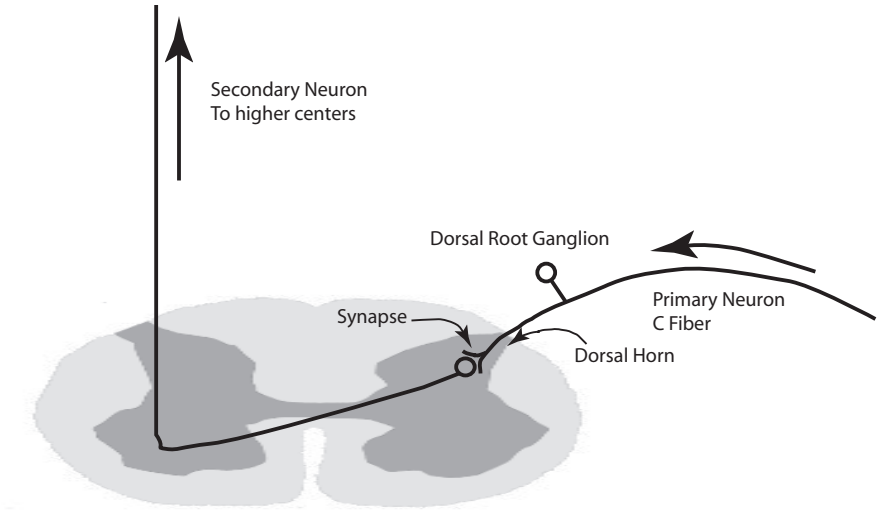
(back to the Greek) motor neurons which innervate muscle to create contraction directly, and 2) small diameter “gamma” motor neurons which innervate spindle cells which set the tone of muscles. Excitation of either one of these motor neurons changes muscle excitability and thus skeletal relations.

There are several mechanisms that help perpetuate the experience of pain at the spinal level which have to do with CNS “plasticity”. Plasticity refers to the ease with which a particular pathway is fired. We’ll mention the phenomena of “windup” and “central sensitization” and a mechanism by which larger non-nociceptive fibers come to trigger second order spinal fibers.

Nociception

We’ve been using “nociception” almost as a synonym for pain. It’s related to pain, but it’s not synonymous. Pain is a perception. Nociception is the neural response to a noxious stimulus - which could be excess heat, excess cold, pressure or response to molecules of inflammation. As was mentioned before, nociception is carried over smaller diameter neurons groups III and IV (C fibers).

Our discussion begins with primary afferent (which means sensory) cells - the ones that convey information from the periphery to the spine. An individual C-fiber is a microscopic filament. It has free nerve endings which are stimulated by a variant of chemicals - especially the products of cell injury — unlike most other afferent fibers. Group I’s and II’s are connected to receptor cells which transduces pressure, tension, chemicals or heat. C fibers also repond to mechanical and heat - sometimes nociceptively, sometimes not.



C fibers get most of the attention in discussions of neurogenic inflammation. What makes them so interesting? It turns out C fibers go both ways. Like all sensory fibers, C fibers send an action potential (AP) from the periphery to the central nervous system. The cell bodies of the C fibers lie just lateral to the spinal cord in structures called dorsal

root ganglia (DRG). They then project a short axon into the spinal cord to the “dorsal horn” of the spinal cord, where they synapse with ascending fibers that carry APs to the brain stem and the brain.

But C fibers also transmit information back down the fiber to the tissue they innervate [11]. The information is not in the usual form of neurotransmitters which stimulate postsynaptic cells. In the peripheral tissue via antidromic impulse C fibers release neuropeptide molecules — Substance P (SP), Calcitonin Gene Related Peptide (CGRP) and various generic neuropeptide names.

Substance P and CGRP are both potent vasodilators, which causes immediate redness and swelling, Substance P, in addition, activates other inflammatory cells - in particular mast cells. If you’ve been blessed with allergies, you probably know mast cells. Think of mast cells as little spiny drugstores. When stimulated by SP or other molecules, they empty their shelves into the interstitial fluid, a dollop of histamine, a little serotonin, some prostaglandin, and a bolus of nerve growth factor.

Nerve growth factor is interesting stuff. In the embryo, its job is to enable the budding axons to find the right muscles to attach to. In the adult among other things, it stimulates C fibers to fire and may cause nerves to arborize and lengthen.[13] On a palpatory level, it’s very clear that inflamed nerves are sometimes double the length as they are in people without inflammation. So notice, C fibers secrete SP into peripheral tissue which degranulates masts cells, which turn around and cause C fibers to fire as well as increase their reach, thereby increasing the possibility of being stimulated.

What makes Chronic Pain Chronic?

What Goes on in the Dorsal Horn?

The dorsal horn refers to the section of the spinal cord where sensory nerves synapse with nerves that travel up the cord. For the most part, sensory cells and pathways in the CNS occupy in the dorsal part of the nervous system, motor cells use the ventral.

The dorsal horn is arranged in layers from outer to inner where different types of afferent fibers terminate. The large diameter neurons terminate deeper at lamina 4-5, while nociceptors do their thing at 1 and 2. Sometimes neurons from deeper levels send sprouts into Laminae 1 and 2, which means that innocuous touch can synapse with and set off — or help to set off — second order nociceptive fibers.

To get a picture of what happens in the spine, think of the thousands of nerve endings that terminate in the dorsal horn at each vertebral level. Many of them are large-diameter, high-speed myelinated fibers that carry proprioceptive information and deliver it to deeper levels of the grey matter of the cord. In the more superficial layer the C fibers and Group III (slightly larger thinly myelinated) fibers terminate.

C fibers express the neuro-transmitter glutamate and our friend Substance P into the synapse (Recall SP goes both ways.) It's role in the dorsal horn is different than at the periphery. SP is secreted only with persistent nociceptive bombardment, which is the topic of the next section.

Central Sensitization

Take a Bad Song and Make it Badder (with apologies to the Beatles)

Let me explain central sensitization. The kid across the street has a new Fender Stratocaster (guitar) and a big amp that he plays in his garage. When he plays Chet Atkins, it's mildly annoying but tolerable. But when he gets into Jimi Hendricks, something else takes over. For one thing, when he plays particularly hard and fast, the reverb unit on his amplifier, instead of holding the note for a second or two, now holds each note for minutes, sometimes hours. And the harder he plays, the more the sensors on his guitar pick up the noise room and amplifies it. This is called feedback.

Usually when he gets too loud his father yells out the back door and that inhibits his excitement. But when he really gets rocking, he puts on earphones and that inhibits his father's inhibition. That's how neural circuits work. A pathway gets excited, an interneuron creates some inhibition, then a higher center comes along and inhibits the inhibition. So pretty soon there's one long feedback screech coming out of his garage.

There are some thirty houses on your block and each of them has a kid with a guitar. And there are wires that go from each garage to the two on either side. Now when the first kid gets into Jimi, now and then it starts to fire off the amplifier in one or another of the garages next to him. And sometimes the kid in *that* garage gets excited and he starts playing Jimi. Pretty soon anytime anyone plays a note it launches a blast of feedback — sometimes from every garage on the block. This is called Strato-myalgia, the acoustic version of fibromyalgia.

When a nociceptor barrage of the CNS reaches a certain frequency a phenomenon called "windup" occurs as SP is released along with glutamate into the dorsal horn causing slow [20] synaptic potentials which allow temporal summation of second order neurons. To explain, glutamate is released and fires neurons ascending the cord whenever the firing is rapid enough to trigger an action potential. SP provides a longer lasting charge enabling the glutamate receptors more time before the signal decays.

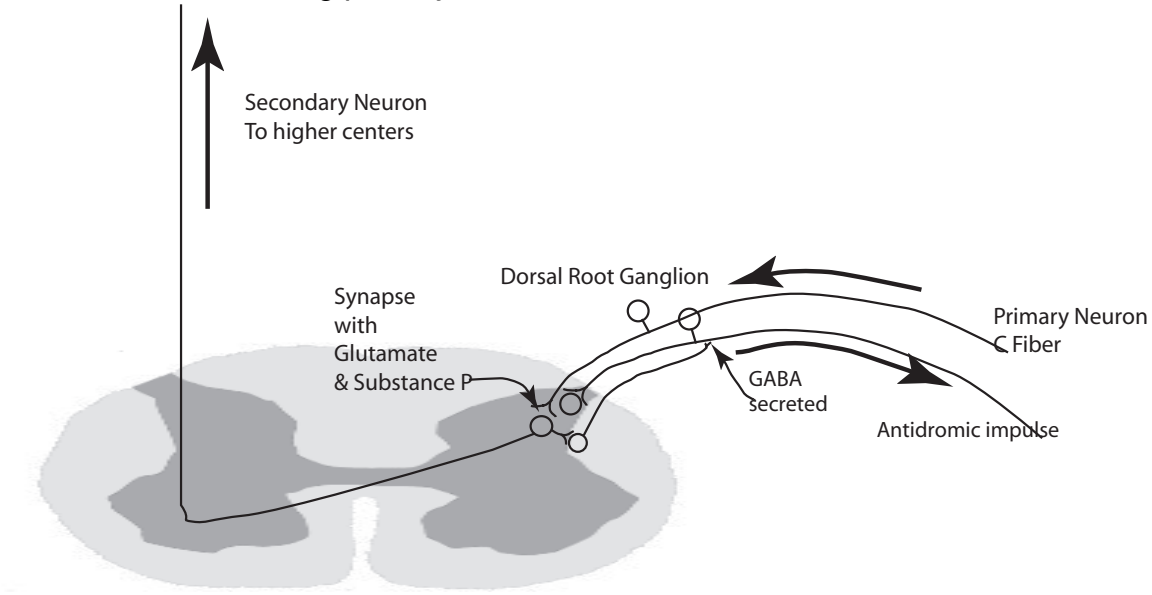
Moreover, central sensitization initiates signal cascades, which are the neural equivalent of the reverb. This means the signal is preserved by interneurons, enabling the initial signal to be amplified and prolonged for hours. This is hyperalgesia - where a painful stimulus initiates a reaction out of proportion to the stimulus. Additionally, receptors other than nociceptors may trigger responses, so that innocuous stimuli such as a light touch may cause painful experience - a process called allodynia. Following peripheral nerve injury, larger afferents which typically are found at deeper layers, lamina II and IV,

send sprouts upward into the synaptic areas of nociceptive neurons, thereby enabling innocuous signals to trigger nociceptive responses.[18]

I think it's difficult to grasp the effects of central sensitization even with my enactment. Those effects can be profound. They can shape the way a life is lived, a body is built. They can shape the way you take in the morning sun and how you treat your children. Central sensitization can cause a screaming feedback loop of pain, the formation of neurogenic inflammation, which might show up as severe muscle pain or perhaps your migraine.

Dorsal Root Reflexes - Sensory nerves act like motor nerves.

The nervous system is kept in balance by by a continuous tug-of-war between excitatory and inhibitory cells and transmitters substances. One of the unusual chemicals in the dorsal horn soup is GABA, which acts as an inhibitor of second order neuron firing [6] while at the same time exciting primary neurons in an antidromic fashion.[11]



This antidromic impulse is the dorsal root reflex (DRR), sometimes called axo-axonal reflexes. Normally the action potentials (AP) in sensory nerves travels from peripheral to central. However, as has long been known, nerve impulses can be triggered anywhere along the course of the nerve by chemical or mechanical insult creating ectopic stimuli. The DRR is a specialized form of ectopic stimulus and has profound effects. Among the interneurons within the dorsal horn of the cord, are GABA secreting nerves which loop back and excite the sensory nerves where they enter the dorsal root ganglion.[11] The AP travels back down the fiber causing the release of SP at the initial site of C fiber. SP, as you may recall, sets off more inflammation.

The interneurons that cause DRR's aren't very precise in which axons they stimulate. They sometimes depolarize neurons at higher or lower vertebral levels or cross the cord to the contralateral side of the body. Arthritis, especially rheumatoid arthritis, typically

attacks the same joint on both sides of the body, whether it's a wrist, a knee, whatever. But if there is nerve damage to the other limb, the arthritis never takes hold there. In laboratory animals, if the nerve is severed to *either* the inflamed limb or to the other side, the process is aborted.

Neuropathic pain

There is a swirl of terminology surrounding these processes, and precise definitions are at best malleable. Nociceptive pain is, as we've described, generated by activation of Group III and IV (C) fibers and is typically initiated by injury to tissue or other inflammatory origin. Neuropathic pain is produced by damage to or pathological changes in either the peripheral or central nervous system.[18] Neuropathic pain can be caused by peripheral nerves being stretched, crushed or cut.

Other researchers [8] have differentiated between pain that is sore locally because of activation of nociceptors in the nervi nervorum and distant pain which is produced by the local inflammation acting on the longer fibers, which they call neuropathic as being "pain due to dysfunction of the nervous system".

The extreme cases are easy to comprehend. A nerve that is crushed or stretched evokes constant "burning" or "electric" pain from fibers that never stop firing. Several clients have come to my office with severe back pain clearly related to cutaneous nerves being chronically over-stretched. The pain is unremitting and months to years in duration. Probably we would call it "neuropathic". If you stretched your wrist and tweaked the ulnar nerve, and it was sore for a couple days, it's clear that the nervi nervorum is doing its job. However, things get a little grey sometimes when you try to determine if nerves are doing their jobs or just being dysfunctional.

Anatomy of a Nerve Bundle

So far we have answered a lot of questions, but still have not addressed the one we began the paper with, namely, how is it that nerves get hard, taut and painful in the first place, and why do they soften with Barral's technique. Part of the answer to the first part hinges on the unique construction of nerve tissue, and part may involve unusual properties of nerve fibers and their relations with immune cells. The majority of this discussion comes from a thorough review of immune and glial behavior in the nerve bundle by Watkins and Maier.[27]

Like the rest of the tissues of the body, nerve fibers are wrapped in fascia, first the individual fibers (the endoneurium) then bundles of fibers (the perineurium), and the bundles of bundles (the epinurium). Within each layer of the fascial sheaths is a vascular (vasa nervorum) and nerve (nervi nervorum) supply . It is these structures that participate in the phenomenon of intra-neural inflammation and edema.

The nervi nervorum (NN) has been shown to carry nociceptive fibers.[21] There seems to be some debate about their role in more serious pain patterns.[8] Nevertheless, it has been demonstrated that they depolarize with chemical or mechanical stimulation and that they secrete neuropeptides - principally CRGP. CRGP, we noted earlier, is a potent vasodilator and inflammatory precursor.

The NN is stimulated by stretch or compression. It occupies primarily the outer sheaths, the peri- and epineurium, where these forces are more likely to occur. So the NN is a credible candidate for creating the conditions under investigation – swelling and pain. It is notable that the majority of places where nerves get into trouble, except in more serious neurological problems, is where the nerves cross joints — in other words, where the NN is likely to be stretched.

Unlike other tissues of the body, the vascular supply to the deep layers of the nerve trunk is protected by the blood-nerve barrier, similar to the blood-brain barrier.[27] So while the epineurium is well supplied with fenestrated capillaries that allow immune elements to freely circulate, the same is not true of the endoneurium, which is generally impenetrable to immune cells and other agents of inflammation. An exception is that T-cells have access to all levels of the nerve trunk.

Thus immune access generally is limited by the integrity of the blood-nerve barrier which is, however vulnerable to damage by stretching, crushing or cutting. Damage to the nerve fiber itself releases proteins which are recognized by the immune system as non-self, so the fiber itself is often a victim of immune attack on the proteins.

Fibroblasts and glial cells are also part of the defensive array and both are present at all levels. Fibroblasts engage in cleanup and repair of damaged myelin and other cellular debris, and glial cells are known primarily in their support role. Schwann cells are glial cells that produce the myelin sheath. But glial cells also have phagocytic capabilities - which means basically they kill by eating. Schwann cells also secrete messenger molecules for other immune cells. It's a busy life down there by the neuron.

The very structure that protects nerves becomes a problem once inflammation gets started. The fascial coverings limit expansion and increase the internal pressure which further compromises the neural tissue. As the inflammatory process progresses, myelin is de-laminated and cell necrosis can occur.

Immune processes in nerve bundles do not necessarily damage nerve fibers; however they can alter fiber excitability, causing ectopic foci. The increase of pressure can also compress the vasa nervorum which can lead to ischemia (lack of oxygen).

The Quieting

The first experience of palpating a hard, swollen nerve is surprising. There are times when nerves are virtually indistinguishable from a boney ridge. The first time you will undoubtedly have the feeling that you are touching muscle or tendon. We've not been

trained to palpate nerves, so it is a surprise to discover how prevalent they are and how wrong you've been in your assessment of what's under your fingers.

What occurs during treatment is probably like what occurs to a hardened kidney or liver during visceral manipulation. As you engage the tissue, whether or not you precisely follow the motility, a softening begins, sometimes slowly, sometimes quite precipitously.

The longer the chronicity, the longer it takes to get relief. That seems to be the case, though I've not tabulated data. What exactly happens as the tissue softens is difficult to guess. I can say it is not merely a local phenomenon. Typically if the segment of nerve I'm working on doesn't respond, I will working way downstream - e.g. on a cutaneous branch - or way upstream, which may cause a release of the whole nerve. I was once able to get a jaw — which is innervated by the motor division of the Trigeminal n. — to let go, by gently working the sensory branches of the Trigeminal that innervate the face.

One hypothesis I have considered is that the gentle pressure somehow alters the fluid dynamics of inflammation, possibly causing edema to be taken up by the vasculature. This mechanical model, however, doesn't readily explain the changes that take place several feet away.

Nerves and Structure

Neurogenic inflammation has undoubted effects on structural relations, though not desirable effects. It has been shown in several studies that neurogenic inflammation is capable of creating arthritis in laboratory animals. [3, 11, 27] It has also been shown that knee joint manipulation has an analgesic effect on rats with induced arthritis in the ankle.[11] Rats get manual manipulation, humans get drugs.

I mentioned earlier that nerve trunks probably had a mechanical effect on joints and thus on skeletal relationships. I have not compiled case studies and goniometric measurements, as you might expect if I were publishing this in a peer-reviewed journal. So I report my observations in anecdotal fashion to illustrate my claims.

My first realization of the mechanical effect of nerves on joints involved a case diagnosed as plantar fascitis. My client's foot was quite painful just anterior to the calcaneous and resistant to dorsiflexion. At that time I would sit with my anatomy book next to my table to acquaint myself with nerve pathways. (Actually, I still do that.) The plantar nerves travel right through the painful area and on compression seemed to be the source of the pain. The plantar nerves derive from two branches of the tibial nerve which descend the medial calcaneous and then duck under the heel.

I began gently working the tibial branches, which were hard as copper wires, until the elasticity returned. Then I worked the plantar nerves, especially the medial. Soon my client was able to stand on it without pain. As she walked the flexion came back into her ankle as if I'd done the two knuckles down the achilles. [As a warning to anyone who wants to try what I've described, I don't recommend Rolfing nerves. It's very easy to

flare up an inflamed nerve and clients get very unhappy when that happens. I had a few accidents when I started out.]

I've worked, I think, with six cases of plantar fascitis. Based on my results, I would say "plantar fascitis" may sometimes be a misdiagnosis. It is possibly a neurogenic inflammation that has been instigated by the stretching of the nerves. I've had excellent results in half of the cases and moderate results in the rest. In two of the cases heel pain is part of a larger pain and inflammation pattern.

I've used this technique on many different joints with good results. Restrictions in the popliteal space, hallux limitus (big toe), shoulder and neck resolve nicely. I've worked on the tiny nerves that innervate the small intervetebral muscle of the neck in a way that I like better than unwinding. On scoliotic spines, the twigs of dorsal rami are typically inflamed on the convex side of the curve. Release causes noticeable improvement of the curve. The last two instances, releasing vertebrae, may be less representative of mechanical restriction by nerve bundles, than of reducing the amount of innervation to the muscle.

I have several pages of clients' descriptions of their experience of this work on my website at: dhazen.com/neuropages/Client.html

I have a strong hunch that many of the effects we see when doing Rolfing strokes are the result of the direct action on cutaneous or major nerve bundles. I get better results sometimes working the femoral nerve than I have doing psoas work. Since the femoral nerve lies directly over the psoas, maybe we have all been doing neural work without being aware of it.

CRPS - Complex Regional Pain Syndrome

CRPS has more-or less-replaced the name RSD - reflex sympathetic dystrophy - since it was discovered that RSD was not caused by a sympathetic excess. In fact most articles point to a lack of sympathetic activity to the injured area rather than an increase. The condition has a group of symptoms, in addition to hyperalgesia and allodynia, which seem to be related to sympathetic excess - redness, sweating, vasoconstriction, and vasodilation.

There are some novel attempts to account for these phenomena. One is an argument that lack of sympathetic input (which has been demonstrated)[7] causes a supersensitivity to adrenergic (sympathetic neurotransmitters) agents and to a formation of adrenergic receptors on C fibers. This means that receptor sites are more sensitive to sympathetic neurotransmitters in the bloodstream and create bizarre effects without direct stimulation of nervous stimulation. It also means that C fibers become sensitive to these circulating neurotransmitters and when fired create nociceptive firing to the cord and vasoactive substances at the periphery - though instead of our friend Substance P, a different neuropeptide, CGRP, is found.

The mystery deepens and there is still much to find out about CRPS. I have included it because the one case I've worked with responded much better than I had hoped using the technique I've been discussing. While I can't rule out an accident or an act of God, fairly well-established symptoms resolved in 3-4 sessions. The client went from barely able to touch her foot to the ground to walking without support. The cute little patch of hair that had developed on the dorsum of her foot disappeared.

The question is how. The mechanism is more speculative that the explanations for the condition. Several writers consider the sympathetic lack to be centrally driven, yet the sympathetic-like symptoms resolved quickly by addressing peripheral nerves. Obviously I won't know if my success was a fluke without more cases.

Immune and Stress Connections to Neurogenic Inflammation

While not exactly relevant to the central discussion of this paper, immune connections between inflammation and the brain and the way that physiologic and psychological stress affect neurogenic inflammation are part of a larger picture which also impacts structure. Stress has been clearly shown to cause inflammatory processes via neuropeptides.[3] And cytokines, the messenger molecules of the immune system are involved in inflammation and in two-way communication with the brain.[14] These relations will undoubtedly play a role when we look, at some future time, at more global effects of this work.

Discussion

This has been a wide-ranging exploration. We began simply enough — with an attempt to account for the palpatory experience of nerve fibers, which I noted varied widely - from dense to boggy and swollen to feeling like plastic or metal wire. That experience is closely connected with the client's experience of pain — ranging from achy discomfort to intolerable blinding pain, though the pain reported correlates only loosely with the felt quality.

We have looked at a range of neural phenomena which are connected in one way or another with the palpatory quality of nerve bundles and the physiological behavior of nerves. What has occurred in my own experience of the peripheral nerve work has led me far from where I began.

To review, I described the inflammatory process and the special instance of neurogenic inflammation. The concept of nociception was presented and the role of C fibers functioning as primary afferents and the neuropeptides they secrete - in particular, substance P. Dorsal root reflex and ecptopic stimuli cause the release of substance P at the distal end of the C fiber. This led to vasodilation and mast cell degranulation which caused neurogenic inflammation.

The excitation in the dorsal horn and the spinal cord generally lead to another level of complexity as the nociceptive signal is passed on to second order neurons traveling to the brainstem and the cortex. Central sensitization leads to a globalization of effects. However, it has been less the spread of *neurogenic inflammation*, than the spread of *inflammation of nerves* which has concerned us here.

The immune response adds yet another layer of complexity as immune cytokines trigger inflammatory effects and changes in brain chemistry. The discussion of the HPA axis and sympathetic input completes a cursory overview of a vast interlocking network of messenger molecules and excitatory cells that enable our bodies to function in an environment that is not always friendly.

I have suggested that the swollen, rigid quality of some nerve fibers - especially those that are painful to the client - can possibly be explained by endoneural inflammation. If that is the case, then the use of Barral's technique has a quieting effect on the inflammatory process, for symptoms subside and the fibers soften from its use. This technique offers a potential for widespread effects in the area of pain relief and structural change.

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