Dermoneuromodulation treatment manual
Self-published June 2007 by Diane Jacobs, PT.

IMAGES
All nerve images that appear in this book were made by me adapted from images from anatomy sources (Gray's, Netter, and Clemente) and all treatment diagrams were drawn from photos taken by Eric Matheson PT at a previous DNM workshop. The cadaver photo was taken by me. Please feel free to reproduce any image in this manual with a mention of its source.
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Overview of Dermoneuromodulation

This treatment system addresses soft tissue dysfunction (i.e., tension patterns, palpable tightness in tissue) and tenderness in tissue as felt by the patient. The two often overlap, although they may not. I chose the term “dermoneuromodulation” as a way to avoid falling into conceptual traps and pitfalls re: other soft tissue methods. I think a good case could be made that all forms of manual therapy are neuromodulatory in their effects, and since no one takes the skin off a patient prior to treatment, all manual therapies are derno as well.

It has been noted clinically that successfully reducing peripheral pain where it can be found and verified by both practitioner and by patient, will improve clinical outcomes such as range of motion. Anecdotally, patients report reduced pain, greater ease of movement, better strength, and improved perception of themselves within their own physicality. A study is underway to test these outcomes and hopefully verify them objectively.

The DNM system takes into account cutaneous nerves. A cadaver study has demonstrated the directional orientation of subcutaneous skin ligaments that convey neural structures to the most superficial outer layer of the arm, as shown below. Note a slight resemblance to rigging on a sailing ship.

Figure 1 Figure: Dissected nerves, colored, with labels placed near to them

The treatment rationale includes providing the nervous system with novel stimuli to assist it to function more easily and economically. Ordinary mechanical pain (from movement deficiency) becomes decreased, usually markedly. Follow up homework includes movement suggestions, but
not usually any “exercise” as such. This approach is consistent with neurodynamic theory and pain theory.

Some techniques are borrowed from the plethora that exists, but nothing has been retained in any sort of “pure form”. Many techniques are completely original. All techniques are suggestions only. Once you learn how to engage the nervous system and “feel” it self-correct, you will undoubtedly learn your own easiest ways to go about treating your patients with simple hands-on methods. So you may regard this manual like a set of training wheels.

Some interesting facts about skin and its innervation

- Skin weighs as much as the skeleton (BodyWorlds exhibit)
- Skin contains 10 times the amount of blood flow necessary for its own maintenance
- The cutis/subcutis is up to a half inch thick in the upper arm
- The cutis/subcutis layer has six definitive layers of circulation
- Cutaneous nerves are mixed sensory and motor, but their motor fibers are all autonomic

Seriously, try avoiding these nerves that embed into the underside of skin. Do you think it is even possible? There are estimated to be 45 miles (72km.) of nerves in the human body (BodyWorlds exhibit).

Some thoughts on Neurodynamics

How is dermoneuromodulation consistent with concepts in neurodynamics? Let’s look at David Butler’s book, The Sensitive Nervous System, for some neurodynamic concepts:

1. The nervous system is a continuum:
   - all functions of the nervous system are interdependent
   - its electrical, mechanical and chemical connectedness is unique – alter one of these and all can be altered
   - any change in one part or function of the system can have far reaching effects in remote parts
2. The nervous system is built to move.
3. Neurons (comprising 2% of the whole body but requiring 20% of available oxygen) require ample blood flow:
   - for nutrition (high oxygen demand)
• for clearing away of metabolic by-products
4. The vasculature to the neural structures itself benefits from movement. It will be slack and tortuous in some positions, in some zones, and on tension other places, depending on position.
5. The nervous system includes the brain, and the brain likes novel stimuli.

All these concepts apply to the nervous system that is directly below the cutis/subcutis as much as they apply to the large nerves and spinal cord.
About tunnel syndromes

In the book *Tunnel Syndromes: Peripheral Nerve Compression Syndromes* (3rd Ed., 2001), the authors (Marko M. Pećina, Jelena Krmpotić-Nemainić, Andrew D. Markiewitz) state that:

1. Tunnel syndromes can occur with no apparent “cause” (e.g., a tumor pressing into a nerve, etc.) – many are “idiopathic”
2. Function of a nerve can be dramatically altered without needing to be compromised in terms of space: diverse factors can adversely affect nerves function, such as
   - Inflammatory changes that thicken a neural wall and reduce blood supply,
   - Edema secondary to hormonal changes from pregnancy, menopause, birth control pills, hypothyroidism (how about the damaging effects of diabetes on autonomic neurons?)
   - Anatomical variations coupled with ordinary movement or repetitive movement (how about insufficient movement?)
3. Ischemic changes first affect sensory fibers; if they continue motor fibers will be affected
4. Pain is the most common symptom (there is still time to turn things around at this stage)
5. Anatomic variations create restricted mobility for a nerve between its origin and its course through its tunnel
6. X-rays don’t show soft tissue variations which are the compressive factors in many cases
7. A nerve that abruptly enters a new tissue produces a fulcrum on which external forces can act.

For more on peripheral nerve response to entrapment or other mechanical loading and deformation see the paper, *BIOLOGICAL RESPONSE OF PERIPHERAL NERVES TO LOADING: PATHOPHYSIOLOGY OF NERVE COMPRESSION SYNDROMES AND VIBRATION INDUCED NEUROPATHY*, included in this manual.

The take-home point I want to stress about dermoneuromodulation, and about treating this multitude of neural tunnels that exist right under the skin layer, is that when we take care of any part of the nervous system, even a superficial part, we will most times be taking care of its depth as well - with less pain people will move better. When they are encouraged to move, and discover they can, they will. When they learn that movement will help them keep pain under control long term (will “feed” their nerves), they learn to plug movement into their lives more easily.

The movement I teach patients is based on Barrett Dorko’s corrective movement, i.e., movement that elicits characteristics of correction; warmth, softening, spontaneity and effortlessness. This form of movement is also consistent with the principles of neurodynamics we looked at above, especially in terms of providing the CNS with novel stimuli.

REFERENCES:

1. Butler D; Sensitive Nervous System, Noigroup Publications, 2000
3. Standring S; Gray’s Anatomy 39th Ed.

ONLINE:

1. Dorko, B; Characteristics of Correction; [http://www.barrettdorko.com/articles/characterc.htm](http://www.barrettdorko.com/articles/characterc.htm)
2. Melzack R; Pain and the neuromatrix in the brain; J Dent Educ. 65(12): 1378-1382 2001 [http://www.jdental.org/cgi/content/abstract/65/12/1378](http://www.jdental.org/cgi/content/abstract/65/12/1378)

Overview of Cutaneous Innervation

In this section I want to present a simple enough model of the body that you will get how it innervates itself, so that it can “feel” the outside world and the inside world, and adjust appropriately, take action.

Our trunk body plan is segmented. We have this in common with worms and fish. It is reasonable to suspect that our body plan came from some ancestor we all three have in common. All vertebrates are our cousins, therefore, and fish were the first on the planet. The human segmented part develops and is innervated exactly the same way as all vertebrate bodies do and are, from back to front, from rostral to caudal.

Arms, legs and even heads are later add-ons. (We will confine ourselves to the limbs for now). Limb buds grow out from the body wall. Hands form first, and contain the median, ulnar and radial nerves. Likewise, the feet form first, containing the tibial nerve. These appendages lengthen away from the body, and as they do, the nervous system adds more innervation to deal with both motor and sensory supply, i.e., more motor supply added for muscle control, and more sensory supply for the skin area over them. The splits come off the plexuses more and more proximally until all the skin on the outside of the arms and legs has innervation. In this way we end up with a lot of nerves from above the plexuses proper that innervate only skin, especially around the outside of the pelvis and over the shoulder girdle. I.e., skin over the shoulders is innervated by long cutaneous nerves from the superficial cervical plexus, an “outer” version of the brachial plexus. The skin over the pelvis and outer thigh is innervated (“outer”-vated?) by long single nerves from T12 and the upper lumbers.

Specifics of skin treatment

Let’s be clear that we are not treating “skin” as such - we are treating working relationships within and between and among the following structures:

1. The nervous system
2. The relationship between the cutis/subcutis layer and its neural tunnels (skin ligaments) and the neural structures that are conveyed through them
3. The relationship between the muscle layer beneath the neural structures and cutis/subcutis.
4. Cutaneous nerves. These are highly accessible, sensitive, and immediately responsive to handling. Input through them goes immediately via fast myelinated fibres to the S1 representational area of the cortex, so the patient can help guide you as to what they are feeling. The information also reaches non-conscious parts of the CNS where non-conscious responses begin to occur immediately.

The relationships we are treating are physical, functional and physiological, simultaneously.

Skin is right out on the surface of the body, and the cutaneous nervous system is anchored right into its underside, so why not use it as a handle? It is a stretchy, elastic covering with a thick slippery underside. It slides around quite readily. As we slide it, the angle and tension on the
cutaneous twigs that convey through it are changed, which stimulates mechanoreceptors. This is proposed as a neurodynamic explanation for the effectiveness of the application.

One can use positioning of various parts of the body to add another dimension of mechanoreceptive stimulation to the system being treated. One can combine skin stretch with positioning.

A Closer Look at Skin Dynamics

Skin covers everything and is slippery:

![Skin Dynamics Diagram](image)

Figure 3 This is a schematic of a cross section of a conceptual “normal” skin neurodynamic. One cutaneous twig is shown conveyed to skin via a skin ligament

Figure 4: This is a schematic of differential layers that may occur when an underlying layer is moved due to muscular contraction. Note mechanical distortion of the neural sleeve.

If there is muscle tension pulling on the suspension system from beneath, the nerve and the neural tunnel are pulled in the same direction under the skin, creating the usual hypoxic threat to the nociceptors. Could this situation be misconstrued as a “trigger point”? What good would direct pressure on something like this do? Why not relieve the hypoxia first, by pulling the skin into some direction of comfort?

As soon as the skin is comfortable, the tenderness and palpable “tightness” vanishes as if it were never there. The “tenderness” can be right over an underlying nerve, large enough to palpate. Such a structure might feel like a cord or string under the skin. Such “cords” vanish easily when skin is stretched in a direction that pleases the brain. The real trick is to stay there, hold everything in the shut off position, until the whole system, peripheral and central, “resets”. This takes 2 to 5 minutes, usually.
Why skin stretch?

One can stretch skin in an infinite number of ways, but I prefer a low angle of entry, i.e., lateral skin stretch. This choice is the one most likely to engage and stimulate Ruffini endings in skin. These are slow adapting mechanoreceptors that fire continuously to lateral stretch. Theoretically this puts a constant non-nociceptive input into the nervous system to help it downregulate nociception from mechanical deformation.

1. Lateral skin stretch can be applied simply by placing both hands on the skin, engaging with it, then applying a small distracting force through it.
2. Lateral stretch can be applied with one hand, by using a “balloon” technique.

NOTES:
HEAD AND NECK

This is a relatively easy region to treat. A typical patient will usually look stressed through the neck. Their neck rotation may be restricted. They may have no awareness of it, or they may be all too aware of it and have tried everything. They often have headache. They will complain they can’t find a good pillow. Often their shoulders will be raised, and traps will feel hard. This is usually defense, not defect. The nervous system is using the muscular system to try to unload neural tissue.

OCCIPITAL NERVES:

On assessment, the patient will usually have a lot of tenderness at points on the occipital ridge. They may or may not also have a headache. They may or may not have restricted chin tuck.

1. Patient is comfortably supine with neck in neutral (Figure 6.).
2. Slide palpating fingers gently in behind head/occipital ridge, find the nastiest sore spot, to be treated first. Keep a monitoring finger over the sore spot. It will feel tight and hard and usually tender to the patient. Do not press it, just touch it.
3. With your other hand, gather up skin on the opposite occipital protuberance, as if you were going to make a bit of a ponytail with it. Slowly. The skin will stretch in an infinite number of vectors all round the whole head. If it makes it easier, you can visualize the head as a balloon.
4. Go slowly. Let the patient’s brain register what you are doing. Remember their scalp skin doesn’t have the same density of receptors your fingertips do, so give their nervous system time to process your intervention.
5. At some point, your monitoring finger on the sore spot will register either a sudden or gradual softening. Use that information to stop moving, and just hold statically whatever you’ve gathered to that point. There you are, with a bunch of skin gathered up in one hand and a soft, less tender spot under the other finger.
6. Wait. After about 20 seconds, check with the patient to see if the spot feels more comfortable to them, by poking it a bit and asking them to give you sensory feedback. "How is the tenderness? Still as tender or any better?" Usually it's better, but wait in that position for another minute or two. Remember this pain, like all pain and brain function, especially hindbrain functional change, is a process through time, like all of nature, not a machine with an on-off switch. It takes time for a new pattern to establish itself. Time is what your patient
needs from you. It's humbling, but the truth is that's ALL they really need from you, once
you've got their layers lined back up with themselves comfortably.

7. After about two minutes, slowly let go. Press the spot again, carefully. It will most times feel
"normal", i.e., perfectly homogenous with the rest of the tissue (to you) and not tender (to the
patient). You will have successfully persuaded the patient's own brain to dismantle a nociceptive driver.
Congratulations. One down and probably several more to go.

8. Move on to the next one. By now the patient is usually relaxed and their nervous system is eager to
track more input from you. Clear the occipital ridge (see picture at right); usually about 4 spots will do it.
The more lateral ones generally require being held in a (tiny) bit of extension and rotation. By tiny I mean one
or two degrees off neutral.

Movement will often be fuller after, particularly head-nod. If not, some activation of deep neck
flexors will likely help; e.g., contract-relax.

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**DORSAL RAMI OF NECK**

While you're back there, you might treat these cutaneous nerves along the vertebrae of the neck.
Simply superficially skin stretch longitudinally on each side, for a few minutes. One set of fingers holds the skin caudal at the CT junction, while the other set of fingers pulls the skin upward along the spinous processes.

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NOTES:
**ANTERO-LATERAL SUPERFICIAL CERVICAL PLEXUS:**

This is a very extensive cutaneous plexus. While the patient is still supine, check/clear the whole antero-lateral cervical plexus. This is easy, because it's superficial and attached to the underside of the platysma. On assessment, the skin at the sides and front of the neck will feel "tight". SCM will feel "hard" to the touch.

2. Roll the patient's head gently to one side or the other, just a few degrees. Position the lateral borders of your hands on the skin of the neck, one hand along the lateral border of SCM, the other along the medial border of SCM. Let your hands sink gently into the skin, just far enough to get a grip on the top layer of the skin.
3. When you feel sufficiently "glued" to the patient's skin, drag your hands in different directions.
   Specifically, pull the skin under the lateral border hand down toward the sternoclavicular notch. Pull the skin under the medial border hand upward toward the angle of the jaw. At the first tiny tug of resistance, stop moving and hold your (tiny amount of) skin tension.
4. There should be no indentation of the tissue going on. Think of gently pulling the skim off heated milk by pulling it sideways.
5. Wait.
   In about 30 seconds, you might feel a relaxation in the skin.
6. The patient's brain is responding to your input.
   Now you can take up a bit more slack, just a bit. Hang in there for a couple minutes, then let go slowly. Now the side you treated should feel a lot "looser."
7. Repeat on the other side.

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**SUPRACLAVICULAR NERVES:**

These drape down from the cervical plexus over the clavicles over the upper chest wall. On assessment, the skin just inferior to the clavicles will feel “tight”.

1. Sit at the side of the patient. Lay the outside edge of one hand along the bottom edge of the

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clavicle, and lay the outside edge of the other hand along the top edge of the clavicle. Let your hands sink into, and glue onto the skin a bit.

2. Pull the skin below the clavicle toward you. Push the skin above the clavicle toward the sternoclavicular notch.

3. Hold for a few minutes. Let go slowly and retest; the skin above, below and over the clavicle should feel more yielding. Treat the other side too.

### SPINAL NERVE ROOTS

You can test these easily in a comfortable manner as a group. The “protectors” in here will be the scalenes --- if these are contracted the neck will feel “stiff”, won’t have any “translation” or lateral glide available. You will not need to do mobilization or anything joint-based here to get lateral movement, you can do balloon technique instead.

**Testing:**

1. Carefully palpate for the transverse processes. Let your fingers stick to the skin.
2. Slide the skin up onto the anterior surface of the transverse processes, into the soft tissue, to gauge how “slidey” or tight the tissues are /skin is.

**Treating:**

1. Target the tighter of the two sides first, and put your monitoring fingers onto the most turgid bit you can feel.
2. With the opposite hand on the opposite side, gather skin layer and grasp it, gently and slowly pulling it into a bunch.
3. Soon you will feel the tissue under your monitoring hand yield and soften, as you displace the mesodermal part of the neck over toward it.
4. Check with your patient to ensure they are comfortable.
5. Hold for a few minutes. Take up any slack that presents itself.
6. Treat both sides.
7. Retest for improved amplitude of side glide.
8. Definitely teach your patient how to explore their new neck movement. Teaching them to find and practice ideomotor movement is the most beneficial approach in my opinion.
NOTES:

We are still focused on neck, but also, we want to minimize the amount of time spent changing position. So I recommend checking and treating something else while you still have your patient in supine, namely the axillary nerve. This properly belongs in the shoulder section, so you’ll see it there as well.

**AXILLARY NERVES**

These are directly from the brachial plexus, posterior cord. The axillary nerve swoops down behind the shoulder, has to get through a narrow quadrangular space through teres and triceps, then enters the arm from the posterior axilla to supply deltoid. It has a cutaneous branch called Upper Lateral Cutaneous Nerve of the Arm. Bend your patient’s arm up, humerus at 90°, hand resting on the forehead. Palpate the posterior border of the axilla. On assessment, there will be a hard and/or tender point or band or cord in the vicinity.

1. Monitor tender spot with one hand. With the other hand grasping the forearm, roll the skin over the rest, in an outward direction (toward supination).
2. You might also find it useful to lift the whole arm directly up at the same time, toward the ceiling slightly.
3. As soon as you feel the spot soften, that will be the right place to hold the arm for a few minutes.
4. Release s-l-o-w-l-y.

Now, you can have your patient sit up for a moment. Have another look at their range. It should be easier, especially head tilt, and rotation, but the shoulders may still look raised and have “knots”. Time to treat prone.
NECK AND SHOULDER

Now we will deal with the classic “knots”. Sometimes the knots are tender, sometimes not. But take raised shoulders as a sign that the nervous system is trying to defend the neural array/brachial plexus within, keep oxygen going into it, not as a postural defect that requires fixing, or the knots as something that have to be massaged out.

Some nerves (deep, buried motor nerves) need to be treated indirectly, so you will add in some positional techniques. While you're at it you can be treating the cutaneous nerves of the arm. But for now, stay focused on the neck. The goal is to have the shoulders be relaxed. They won’t relax if there is too much tension on the neural structures in the neck, but you’ve taken care of those. Likewise the neck can’t relax if there is too much tension in the shoulder zones, so now we’ll learn to take care of these.

In the course of treating accessory and dorsal scapular nerves you will also be helping to oxygenate intercostobrachial nerve, from T2, and cutaneous to the axilla/medial side of arm; and upper and lower lateral cutaneous nerves of the arm. Furthermore, in the treatment position (modified quadruped) you’ll be able to treat the elbow and forearm cutaneous nerves.

Have your patient lay prone, with face in a face hole. It should stick out from the end of the bed so there's room for the arm to hang freely in front of the bed. Be sure the angle of the faceplate is comfortable so they can focus their attention on what you're doing instead of wasting time being/feeling uncomfortable. (The suprascapular nerve will require a bit different set-up).

Prone with a freely hanging arm gives you a lot of options for dermoneuromodulation combined with arm positioning.

ACCESSORY NERVE (relaxing traps)

1. Arm hangs forward off the edge of the bed.
2. Sit down close to the floor on a low stool, so you can comfortably handle the dangling arm.
3. With one hand, palpate the cranky spot, up in the top of the trap. With the other, gently grasp/let your hand glue onto the skin of the upper arm, postero-medial aspect, just about the level where the deltoids insert. Drag the skin layer, s-l-o-w-l-y, around the mesoderm of the arm, into internal rotation. (The whole point of doing all this slowly is to give her brain a sensory feast of being able to differentiate itself and its nerve and ectodermal skin from the mesodermal entrapping tissue.)
4. Pretty soon you’ll be able to feel the cranky place "relax". If it doesn’t, something else is going on, maybe the patient is experiencing discomfort somewhere and is unconsciously struggling. If so, stop, let go, help her find
a more comfortable way to be, maybe a pillow under the bosom. whatever then try again. Don't give up. Once the cranky spot doesn't feel cranky anymore, to either you or her, hold her arm there and wait, about two minutes.

**DORSAL SCAPULAR**

1. Same position and handling, but this time, take the arm further into relative elevation and outwardly AND/OR inwardly rotate it. Both good.
2. You can monitor as shown in the picture, or along the side of the neck.
3. Feel free to improvise a bit, ask for feedback, let the patient tell you how they would like you to move the arm, how much, how long to hold it there. You might need to go into different angles, or up past neutral into the extension zone. Let it be as interactive as it needs to be. Once patients get a taste of what this is about they can guide you better.

**NOTES:**
SHOULDER AND ARM

Congratulations. We’ve helped the nerves all through the tissues of the neck to be able to breathe better, and we will now move to the shoulder region. Sidelying is a good position to treat shoulder problems, providing lots of opportunity to test without moving the patient. Saves time.

The shoulder girdle could be looked at as a large soft tissue funnel applied to the trunk, with the arm bones protruding through the neck of the funnel. The wide part of the funnel is attached posteriorly all the way down to the pelvis, the last 4 ribs, and every vertebra up to T6 in the back (lat dorsi), and anteriorly to midline and around ribcage in the front (pec). We have trapezius covering up lat from T6 level down to T12. Suspended within this funnel is a mobile scapula. This arrangement provides a lot of stable and large range, but can entrap any of the nerves that have to navigate through it.

We will start with the axilla. If an arm cannot lift up easily when passively lifted, it is often because it is being held down from underneath. Latissimus (and its nearby companion serratus anterior) have so many nerves associated with them, mostly the lateral cutaneous nerves of the trunk, that for workbook purposes I will refer to the muscles rather than the nerves. But bear in mind the point is to unload the lateral cutaneous nerves of the trunk. This movement/excursion issue must be addressed before the shoulder nerves can become mobile and oxygenated. Feel free to start with this area first when faced with someone with neck pain. I do quite often.

These techniques work well for all those “rotator cuff” pains that keep people from recovering all their rotation.

LATERAL CUTANEOUS NERVES OF THE BODY WALL

First, have a look at shoulder range, and test the ability of lat to lengthen. See the picture to the right.

There should be full elevation such that the arm is easily into 180° of flexion-abduction. Chances are pretty good that if someone has had neck/shoulder pain for awhile, this will be restricted. It may even be a contributing factor.

If there is not full excursion, if the movement feels sticky, or hurts, or feels like the arm wants to pull forward, or if the patient rolls backwards to accommodate, treat the sidewall.

Treatment:
1. Ask the patient to leave their elbow pointing up toward the ceiling, and to rest their hand on their head somewhere, and to abdominally breathe. Explain to them what you plan to do.
2. Place your hands softly onto the ribcage top and bottom. Give your hands a moment to stick to their skin. Waiting just a few seconds means you need to exert less force less deeply (more finesse).

3. Slowly stretch your hands apart, just enough to take up skin slack. Wait. Usually the patient’s system will provide you with more slack to take up after a little while. Take it up as it presents itself. Do not overstretch.

4. After a minute to two minutes, slowly let go, lift your hands away, and fold their arm back down straight along their body. Let the brachial plexus refresh itself. Then retest. It’s not uncommon to gain 30 or 40 degrees of arm elevation, just with this simple intervention.

If you do not get the expected huge increase in range, then look a bit closer at scapular movement and/or pec excursion.

Serratus anterior may be entrapping the lateral cutaneous nerves up at the top, especially on gym attenders, or maybe the subscapular nerves could use some movement. Set up treatment as shown at right.

**SUBSCAPULAR NERVES**

1. Tell your patient what you want to do. Ask them to abdominally breathe. Place the heel of your hand comfortably along the lateral border of the scapula.

2. Slowly press it medially toward the other blade, taking up slack as it presents itself. Do NOT be in any hurry. If this takes 5 minutes, then spend 5 minutes.

3. It helps to use the supporting hand, up on the elbow, to pull some skin up the back of the upper arm toward the elbow. Always be thinking up new ways to move-slide cutaneous nerves, in this case, the posterior cutaneous nerve of the arm.

4. Sometimes you can feel the scapula start to slide easily all of a sudden. The patient will usually say something like, "Ooh, that feels so good." Other times, this won’t happen. It probably has less to do with your treatment and more to do with how well they can perceive their own body. Whatever. Do your best and move on.

**NOTES:**
LATERAL PECTORAL NERVES

Lots of people whether they go to the gym or not, will have a lack of elevation because of tension in the front of the shoulder.

Treatment:

1. Explain what you want to do. Let the patient’s elbow lean against you. Take the full weight of the arm; ask them to give it to you. You can tuck their elbow under your own arm somewhere.
2. Ask them to abdominally breathe and relax as much as possible.
3. Grasp the whole anterior compartment of the axilla in two hands, and let your hands and their brain have a moment to “say hello” to each other. Then gently squeeze into it. Take your time. Take up slack as it presents itself. You are not just on a great big pec tendon, you are affecting some very sensitive and responsive neural tissue, including the intercostobrachial nerve cutaneous to the axilla.
4. Give this lots of time, and when it feels like it can’t lengthen anymore, let go slowly. Now on retest the arm should be able to go further.

MUSCULOCUTANEOUS NERVE

This nerve comes off the brachial plexus, pierces through coracobrachialis, supplies it, biceps and brachioradialis, then changes its name to lateral cutaneous nerve of the forearm. Treat the front of the shoulder if there still seems to be some tightness present.

Treatment:

Apply a light skin stretch to the front of the upper arm as shown at right.

NOTES:
SUPERFICIAL CERVICAL PLEXUS

What? Again? Didn’t we move off the neck a long time ago? Yes, but this plexus is extensive, and some of those supraclavicular nerves drape down over the shoulder like a little shawl.

Treatment:

You won’t really know if you are treating these or as above, musculocutaneous. The one (superficial) overlaps the other (deeper) so it hardly matters. Just get on the skin and stretch it gently.

SUPRASCAPULAR NERVE

On assessment, there will be a persistent tender place above the spine of the scapula, and pain on movement. This is the only nerve that requires its own unique treatment position, prone at the edge of the bed. Ask your patient to move over to the edge of the bed so that the arm can hand over the side instead of off the front end. This will mean the neck will need to be turned - whichever direction is most comfortable is fine. Maybe you can place a towel roll to protect the neck from turning too far, and allow your patient more support for relaxation. You could also place a wedge under their hip to keep them from feeling they might roll off the bed, especially an obese patient.

Treatment:

1. The arm hangs off the side of the bed.
2. With one hand palpate the cranky spot under or in the skin over supraspinatus. With the other, slowly lift the arm a bit out, and back, into abduction extension. About 45 degrees is good. Take the arm gently into external rotation (or internal, whichever works better).
3. The palpated spot should be starting to let go. Hold the arm in that position.
4. To help it along, stretch the patient’s skin gently down the radial side of the forearm, down by the wrist somewhere. The spot up top will let go completely. Hold that pose for a couple minutes.

---

**SUPRASCAPULAR NERVE (cont.)**

You can let your patient use the face hole again. Suprascapular nerve supplies supraspinatus (you’ve already treated that branch), and infraspinatus, to which we will now turn our attention. It also sends a slip to the AC joint, which we will get to.

On assessment, you will feel hard tense bands under your fingers that feel like they are part of infraspinatus. Maybe these are just patches of muscle defending the inferior portion of the suprascapular nerve. In any case they let go quite readily the following way:

*Treatment:*

Setup is the same as it was for the dorsal scapular nerve (see picture left), only the arm can be lower, and at a 45º angle forward/outward, and resting on your knee.

1. With one hand, palpate the infraspinatus zone, and select the area of worst tightness to monitor.
2. Place the other over the radial side of the forearm; roll the forearm into outward rotation until you feel things soften under the monitoring hand. It helps to pull the skin down over the forearm, which affects the lateral cutaneous nerve of the forearm. See if inward rotation is helpful. If so, use it too.
3. Feel free to make slight adjustments in your position, pressure, or angle, to take advantage of slack that will open up.

A variation as shown at right, will hopefully help take care of a pesky sore spot that is often found right at the medial superior tip of the scapula. This could be a dorsal cutaneous nerve of the T spine.

Setup is the same as above, only this time the elbow is bent and the forearm is resting on your lap.
**Treatment:**

1. Bend the elbow to 90 degrees and rest it on your knee. With one hand, use a light finger to palpate a very sharply tender sore spot right on the superior/medial corner of the scapula. Rest your finger there - do not aggravate the soreness by pressing it.
2. Grasp the arm just above the elbow, skin layer only, and slide it gently around the rest of the arm into internal rotation direction.
3. The rest of the arm will eventually follow but it seems important to let the patient's brain experience the lag time. Meanwhile, you can feel through your palpating finger some motion in the tissue layers. You'll know when to stop moving the arm - the sore spot will suddenly soften, and not hurt anymore.

---

**SUPRASCAPULAR NERVE (cont.) “Pencil technique”**

There is one more application I want to include when considering the SS nerve; any new unsharpened and erasered pencil will do just fine.

On assessment, the patient’s elevation range will still look restricted, and/or they may still feel discomfort on horizontal adduction, the classic sign for AC problems. Maybe they don’t have an AC problem, maybe they have an hypoxic suprascapular nerve.

**Treatment:**

1. Set up as at left, patient prone, arm by side.
2. Carefully press the eraser end of a new pencil into the space between the acromion process and lateral end of the clavicle.
3. Go as far as burying the whole eraser into the tissue but go slowly and wait for the patient to feel comfortable with each increment before adding another.
4. Sometimes it is helpful to pull the middle finger caudally at the same time. I don’t know much about myo-osseus kinetic chains, but this might be one. For whatever reason, it helps the process along. If this was indeed your patient’s main restriction, when you recheck, they will have much improved range and decreased discomfort.
**CUTANEOUS NERVES OF THE ARM**

When you have the patient prone with the arm hanging, you can get in around it on all sides and treat any of the cutaneous nerves in it, from any direction, using balloon technique.

The picture to the right shows cutaneous branches of the radial nerve being treated. The left hand is monitoring for softening, and the right hand is doing the grasping.

Feel free to explore the arm and treat what you might find. The elbow in particular is easy to treat in this position.

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**AXILLARY NERVES**

These are directly from the brachial plexus, posterior cord. The axillary nerve swoops down behind the shoulder, has to get through a narrow quadrangular space through teres and triceps, then enters the arm from the posterior axilla to supply deltoid. It has a cutaneous branch called Upper Lateral Cutaneous Nerve of the Arm. Bend your patient’s arm up, humerus at 90°, hand resting on the forehead. Palpate the posterior border of the axilla. On assessment, there will be a hard and/or tender point or band or cord in the vicinity.

1. Monitor tender spot with one hand. With the other hand grasping the forearm, roll the skin over the rest, in an outward direction (toward supination).

2. You might also find it useful to lift the whole arm directly up at the same time, toward the ceiling slightly.

3. As soon as you feel the spot soften, that will be the right place to hold the arm for a few minutes.

4. Release s-l-o-w-l-y.

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NOTES:
TREATING THE TRUNK

This is surprisingly easy with DNM. You could visualize the trunk more less as a slightly flattened cylinder, with vertical rows of cutaneous nerve exit points. The rows of exit points are often where the tender points will be located.

One is treating the cutaneous nerves with DNM, and they occupy only the outer layer or two. It is worth discussing back pain for a few minutes as it is such a common problem.

Take a look at the way the cutaneous innervation is set up in the body wall. The dorsal cutaneous rami are the first branches to split off from nerve root (which then becomes, by definition, ventral). The dorsal cutaneous roots:

1. branch off main root at a sharp angle (more vulnerable)
2. must negotiate several barriers, including vertebral transverse processes and sheets of dense fascia.
3. supply skin that spans the distance between the tip of the spinous process over to where the back becomes side, at which point the lateral cutaneous nerve, posterior branch (from the ventral root) takes over the skin.

In the image below and above, note the 180° branch angle that not only the dorsal cutaneous but all the cutaneous nerves of the trunk must cope with as they surface, split, and bend backwards and forwards to run within the cutis/subcutis layer of the trunk.

The cutaneous branches surface through latissimus dorsi or through trapezius, or through their respective fascial attachments, neither of which are innervated from the back they cover, i.e., both of them completely bury the actual spinal muscles as they attach to the spinous processes.

They then supply the skin layer. What do we suppose might happen if the lats are pulling one way, the traps another, and some other deeper layer of the back still another? Perhaps those dorsal roots become tugged from below, and end up tractioned from within, with mechanical deformation/hypoxia. I’m not suggesting that all back pain is due to peripheral entrapment of the dorsal rami, but I am saying that a lot of back pain can be downregulated by a patient’s nervous system if these branches are attended to.
Treatment of dorsal rami:

1. Patient is prone. Investigate hardness and/or tenderness of tissues just off midline. There will almost always be some sort of tight patch, somewhere, in either the upper or lower back.

2. Select your spot to target. Monitor it with one set of fingertips. With the other set, settle onto skin exactly across the spine from where the first fingers are.

3. Once your fingerprints have become attached to the skin, pull the skin on the opposite side of the spine from where you are monitoring, away from the spine at a 90º angle.

4. After a few minutes let go slowly. Move to the next spot.

5. Repeat steps 1-4 as necessary, over the thoracic, lumbar and sacral spine. It is useful for the superior cluneal nerves as well. The anterior cutaneous nerves from ventral roots can be treated this way as well.

I’ve found the exercises created by Tomas Hanna (founder of Somatics) useful for helping people gain increased awareness of how to help their trunks elongate, move, expand, and breathe.
**Treatment with side bending:**

The patient is still prone. Just distal to the TL junction, one side will feel a lot harder/tighter than the other.

1. Place a wedge under the non-tight side, below the hip, effectively “banking” your patient up on the non-tight side.

2. Sit down on the wedged side. With one hand, palpate the “tight” area. With the other, take hold of the opposite side foot, by its lateral border.

3. Slowly load into the foot, pulling it toward you. It may not have to move much at all before you can feel an unloading happen under your palpating fingers. Once the back feels softer, wait.

4. If your patient’s foot gets uncomfortable, hook a finger over the outside of the end of the fibula, and pull down a bit of skin, keeping tension all the way up to the back while you unload the foot a little. The whole letting go process may take as long as 5 minutes.

5. When things feel done, slowly let go. Check the two sides. They will usually feel more even.

**Treatment with arm raising:**

Patient is still prone. The zone between T6 and T12 has an extra layer, as trap overlaps lat (as if those dorsal cutaneous roots didn’t have enough to contend with already).

1. Palpate and find out which side feels “tighter” and/or more tender. Stand on that side.

2. Monitor the tightness with one hand, lightly. With the other, slowly lift the patient’s arm up, elbow in extension, slowly. You may only have to lift a little, but with some people it will be a lot. Usually it will be somewhere between 45 and 60 degrees. When you feel something move under the palpating hand, that’s your signal to stop lifting. Just hold the arm up at that level.

3. If you internally rotate the lifted arm, you will be tensioning trap. If you externally rotate it, you will be tensioning lat. Figure out which way works better in each case. Sometimes one way will, other times, for other patients, the other will.

4. Choose whichever way provides the patient with the most softening. Hold the arm in that position for about 2 minutes.
**Treating the ribcage:**

This is the only time, in my experience, that adding a bit of pain is necessary for down regulating pain, necessary only because there is no kinder way to do it. Fortunately, we have something called DNIC (diffuse noxious inhibitory control) to explain the results. It is useful for people who look like they cannot get a good breath or expand easily.

1. Patient is supine. Place one hand behind the back, just to one side of the spinous process. Find a tender point there. This will be the most medial tip of a medial dorsal cutaneous nerve root.

2. With the other set of fingers, find a tender point along the edge of the breastbone, within the same segment of the body. This will be the medial potion of the anterior cutaneous branch. It will be tender.

3. Carefully press the two spots simultaneously, and hold. This will feel like “pain” to the patient, but they will downregulate rapidly. Only stay there for 15-30 seconds before moving on to the next segmental level. Their breathing does the mobilizing of the neural tunnel.

4. Patients usually feel that breathing is much easier after, that their ribcages feel more expanded, lighter.

**Treating the DCN with skin stretch of the arm**

The patient is supine.

1. Slide one hand behind their back, and feel the tissue beside the spine. Find what you want to treat.

2. With the other hand pick up the patient’s arm and fold it across them. Place your free hand on the back of their arm, and stretch the skin of its posterior aspect toward their elbow, until you feel the area you are palpating soften. Stay there, hold for at least two minutes.

3. The arm can be placed at literally any angle of a semicircle, from down along their side (for places in the upper back) to up over their ear (their head turned away) for lower back, whichever position feels the best. I find this particularly effective for upper back treatment.

These are just a few ways of treating the skin layer with and without added positioning. Discover your own ways. Adding wedges in and underneath the ribcage and or pelvis, sometimes both, will often facilitate your work.
It never hurts to remind ourselves to explain to patients what we plan to do, and why, especially in this area of the body. Be sure they feel comfortable with being treated in this manner. Ask them to tell you immediately if they feel discomfort of any sort at any time, so that you can change your grip slightly, or modify the approach somehow. I think it’s best to treat from lateral to medial.

We could look at the pelvis as a pair of large curved scapulas joined at the middle, but much less mobile, and as fused to the sacrum as it is possible to be without actually being fused. Cutaneous nerves have to get around this big bony fused part, coursing along the inside of it and around the outside of it, to get to the legs. Several long cutaneous nerves emerge from the low back, descend obliquely downward within the body wall to emerge at the pelvis on the sides or in front of the hips through the inguinal ligaments. There are numerous opportunities for entrapments, especially in a sedentary job, appy scars, leg crossing, etc.

We’ll start in supine, and with the nerve that comes out T12 and travels cutaneously around the top of the iliac crests. It’s called subcostal nerve (see p. 31).
SUBCOSTAL NERVES

Usually one side is more affected than the other. The patient in standing will look like their pelvis tips forward on one side or backward on the other. This is defense, not defect.

On supine assessment, there will be a real tight/hard spot at the top of one of the crests compared to the other. It may feel quite tender or just tight to the patient.

Treatment:

1. Stand on side opposite to target ilium. Put the bed to the correct height, put one foot up on the bed, place your patient’s legs up over your knee. (I often use a piece of yoga mat or carpet underlay to provide friction, promote more relaxation.)

2. Get comfortable. Reach over the patient and find, then monitor, the tender point.

3. Place the other hand across the bottom of the feet; slowly pull the feet away from the target side. The thighs will naturally roll slightly toward the target side. Turn both legs several degrees away from the target side, as if they were a large crank.

4. When the tender point stops being tender, and softens, wait in whatever position you are in for as long as it takes, for a change to occur.

5. You can finesse the process by pulling the skin over the lateral malleolus distally, and holding it there.

LATERAL CUTANEOUS NERVE OF THE THIGH

The next nerve in is the lateral cutaneous nerve of the thigh, a branch of the iliohypogastric in many anatomy books. Entrapment of this nerve is common. It leads to pain and/or numbness of the lateral anterior thigh.

For this, and other inguinal cutaneous nerves, a bolster is very useful. It permits comfort for the patient, and a modified quadruped position for the patient’s nervous system. Learn to palpate these through clothing. They are sensitive enough that you’ll know when you’re on one, just by patient response.
Treatment:

1. As shown at the right, locate and monitor the lateral cutaneous nerve. If it’s in trouble, it will feel hard and usually, but not always, tender.

2. Monitor it with one set of fingertips, and place the other hand on the skin at the front of the thigh about 2 inches distal. Let your fingerprints stick to the patient’s skin.

3. Once the contact is sufficient, traction the skin layer only, distal toward the knee, in line with the angle of the thigh.

4. The tender zone will soften and any tenderness will suddenly disappear. Wait.

5. Give it a few minutes, then slowly release.

INGUINAL LIGAMENT AREA

Next we move to the area of the inguinal ligament. The inguinal ligament is created when embryologically the abdominal wall folds over on itself and a lower edge is created. Several cutaneous nerves surface through it, and innervate the skin over it. As you palpate along the inguinal ligament, you will find the next tender spot which will likely be the femoral nerve, as this the next one medial.

FEMORAL

Treatment:

Treatment is exactly the same as for the lateral cutaneous nerve, just a few centimeters more medial.
ILIOPINGUINAL NERVE

Treatment

1. The tender spot will be high up in the groin, near or on the adductor attachment to the superior ramus. If you have a skeleton in the room, show them on the skeleton first where your hands are going to be placed, and why. Have a picture handy of where the nerve emerges. Get their consent once again.

2. Treatment is exactly the same as for the other inguinal cutaneous nerves, although for this one have the patient’s leg flexed-abducted, and support the knee with your body so your patient can relax. The other leg is still over the bolster, and the foot of the leg being treated can rest its lateral border against the bolster.

OUTSIDE OF THE HIP

Now we’ll have our patient turn on their side, target hip uppermost.

There are a number of things you can do to treat in this position that will contribute to a good outcome. One of the simplest is balloon technique over the trochanter. There is a bursa under the skin in that area, and although I haven’t seen a picture of the neural plexus that innervates it, I have seen its vascular plexus at BodyWorlds. I expect there’s a neural one too. In any case there are twigs from the lateral cutaneous nerve to deal with, and often the lateral most superior cluneal nerve will reach this far.

Treatment:

1. Palpate the trochanter and the layers stretched over it. There will usually be a few sore spots either behind it or in front or both
2. Select a sore spot to treat, monitor it with one hand.
3. With the other, use the web of your hand to gather the skin in a horizontal version of the balloon technique, basically a large but non-nociceptive pinch. Gather the skin slowly. Wait
for it to let you go further. Hold for a few minutes. Let go slowly. Quite large increases in range in the hip can be obtained in this simple way, sometimes, on some people that look like they might be ready for THR.

**OUTSIDE OF THIGH**

See the section on KNEE (p. 37)

**SUPERIOR CLUNEAL NERVES**

These are long dorsal rami from the lumbar spine that drape obliquely down over the back of the iliac crests, through narrow fibrous tunnels. They are cutaneous to the skin over the buttocks, and can reach down as far as the trochanter on some people.

*Treatment in side lying:*

1. Palpate outside of pelvis superior to trochanter to locate any tender spots.

2. If you find one, monitor it, and lay the other hand flat along the outside of thigh. Wait for the skin on your hand to stick to the skin of the patient’s thigh.

3. Slowly drag the skin layer distally toward the knee, *without* pressing in. The tenderness should diminish easily in the tender spot. Hold a few minutes.

*Treatment through levering:*

1. With one hand, monitor the tender spot. With the other, grasp the patient’s foot.

2. Carefully lift it, and ask your patient to stay relaxed, to not try to help. As you lift the foot, the knees will stay together and the leg will go into internal rotation.

3. Block the patient’s heel with your body and take the forefoot into adduction/inversion. When you feel a softening occur, hold in
position for a few minutes.

4. Do exactly the same thing again, but this time, take the foot into the opposite direction, abduction/inversion.

Figure 50

Treatment prone:

1. Find and monitor the tender spot at or near the iliac crest to one side of the sacrum.

2. Place the other hand on the trochanter on the same side as the tender spot. Lean straight down into the back of the hip, slowly. Take pauses. Wait for the patient’s body to let you proceed.

3. Slowly release after a few minutes.

Figure 51

There are lots of other ways to get at these, so don’t limit yourself to these few suggestions that there happen to be pictures for. Try holding the leg at different angles. Try skin stretch across the sacrum. Try whatever makes sense.

OBTURATOR NERVE

This nerve is deep and medial, descends through the pelvis and can become entrapped high up inside the pelvic floor. Lucky for us, it has a cutaneous patch on the medial thigh, just above the knee, where we can most easily get our hands on something it embeds into.

On assessment in standing, when viewed from behind, the patient’s knee will look like it doesn’t fully extend or the thigh internally rotate fully, compared to the other.

Treatment:

1. Patient is prone. Palpate the inner side of the thigh a few inches above the knee. It will feel as though a tight thin cable is suspended within the tissues. Often it will feel tender.

2. Sit down, get comfortable, lay your arms out along the length of the leg.

Figure 52

Figure 53 BALLOON TECHNIQUE VARIATION
(see left) and with both hands grasp the skin over the inner thigh.
3. Slowly pull the skin up and around the thigh into internal rotation.
4. Stretch the skin you have in contact with your forearms, distally and proximally, simultaneously.
5. After a few minutes it will feel like the whole leg lengthens and softens. Let go slowly, then retest. It will feel like the tight thin cable is gone, and tenderness will be gone as well. Reassess in standing. Usually the knees will rotate more evenly.

Other parts of the obturator nerve can be accessed through pelvic floor work.

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**PUENDAL NERVE**

Treating the pelvic floor and the nerves that can become entrapped within it may seem a bit daunting, but don’t let any part of the body daunt you. Simply understand what you are about to do and why, provide clear explanation to the patient, and wait for permission.

First, let’s look at the neural component: the pudendal nerve supplies sensation to much of the pelvic floor. It is motor to the sphincters and to the autonomic aspects of the genitalia. People who have sensory disturbances, i.e., pain, burning, or paresthesias, that are worse with sitting, may well benefit from treatment.

The internal rotators of the hip are buried in the pelvic floor as well, with their neural components.

Right where the pudendal nerve comes out through Alcock’s canal, just medial to the ischial tuberosity, is a good place to treat. Treatment can be successfully accomplished through a light layer of clothing.

**Treatment:**

1. This is for both male and female pelvic floors. You have already explained to the patient what you want to do and how, and have obtained their permission. Chart it. Make sure you have lots of time. You must not rush this.

2. Patient is supine, their knees bent up and supporting each other. Feet are flat on the table. You may want to place some pieces of yoga mat under the feet to keep them from sliding, and help your patient be able to relax, and not have to use any leg muscles to keep their knees up. Get gravity to do all the work whenever possible.

3. Take your time. Sit by the side of the patient, on the side you’ll be treating. Place one set of fingers on the skin of their ischial tuberosity, the other set on their iliac crest.

4. Let your fingers sink in for a little while. See if you can feel the patient’s breathing through your hands.
5. When you are ready, straighten out your fingers and wrist. Slide the skin over the IT medially, without losing contact. Sink your fingers into the tissue just medial to the IT. Take pauses to let the tissue soften. Then proceed with a directly rostral pressure.

6. This is a sensitive area, and will take awhile to treat. Go slow and be sure you have your elbow lined up behind your forearm so you don’t strain your wrist. The more you go slowly, the less work it will be. Get feedback from your patient. They will tend to unconsciously tighten in spite of themselves, so remind/ask them to relax again from time to time.

7. This does not have to be uncomfortable at all, if you and the patient are working as a team. It’s also ok to take a rest, letting go completely. You can rest your and the patient’s body can reoxygenate. When you add pressure a second time, the nervous system will have likely “learned” to let your fingers in easier, and your patient will have “learned” to relax better.

8. This is clearly an indirect method rather than directly cutaneous, but it will accomplish the goal, i.e., will help slide the pudendal nerve further out of its tunnel entrapment. If more sophisticated pelvic floor work is required, refer out to someone with more advanced training and the scope to do internal work.

NOTES:
KNEE

This large and often problematic area can be treated simply and effectively for pain and motor problems that may mimic orthopaedic problems, simply by removing possible neural confounding factors. We’ve already looked at treating the outer thigh, and inner thigh. The knee itself is innervated from behind, by a branch of the obturator and genicular branches from the tibial.

OBTURATOR NERVE

This nerve is deep and medial, descends through the pelvis and can become entrapped high up inside the pelvic floor. Lucky for us, it has a cutaneous patch on the medial thigh, just above the knee, where we can most easily get our hands on something it embeds into.

On assessment in standing, when viewed from behind, the patient’s knee will look like it doesn’t fully extend or the thigh internally rotate fully, compared to the other.

Treatment:

1. Patient is prone. Palpate the inner side of the thigh a few inches above the knee. It will feel as though a tight thin cable is suspended within the tissues. Often it will feel tender.

2. Sit down, get comfortable, lay your arms out along the length of the leg, (see left) and with both hands grasp the skin over the inner thigh.

3. Slowly pull the skin up and around the thigh into internal rotation.

4. Stretch the skin you have in contact with your forearms, distally and proximally, simultaneously.

5. After a few minutes it will feel like the whole leg lengthens and softens. Let go slowly, then retest. It will feel like the tight thin cable is gone, and tenderness will be gone as well. Reassess in standing. Usually the knees will rotate more evenly.
**SAPHENOUS NERVE**

This long and extensively branched nerve that stems from the femoral, supplies the skin over the medial side of the knee picking up where obturator leaves off. It extends into the lower leg, giving off an infrapatellar branch that swings laterally. It reaches all the way to the medial foot, supplying skin (see below).

The main nerve can be treated much the same way as the obturator, in prone, with hands placed lower. The tender spot is usually over the medial knee joint. It can be treated in supine too if you prefer.

The actual patellar plexus and infrapatellar branch can be treated together (see further on).
PATELLAR PLEXUS

The pictures to the right show the saphenous nerve, cutaneous to the medial knee and lower leg.

The picture below shows more of the contributors to the patellar plexus, namely the intermediate cutaneous nerve of the thigh, also from femoral nerve.

Treatment:

1. Palpate carefully around the patella. Note any spots that feel tight or tender.

2. Choose one of them to treat. Monitor with one finger, and with the other hand use the patella itself as a small lever or crow bar, pressing down on its opposite side, slowly. When you feel softening under the monitoring finger, hold for a few minutes.

3. Slowly release. You can do this all round the patella.

NOTES:
TIBIAL NERVE

Treatment:

1. Patient is lying supine. Palpate back of the knee.

2. There will usually be a tender and or tight spot just below the crease, medial side. Target that.

3. Keep a finger on that spot, and carefully bring the leg down off the edge of the bed (be sure to put some padding there), and let it rest on your knee. The patient’s knee will be in slight flexion.

4. Place your working hand on the front of the lower leg, near the ankle. Carefully press it medially and into a small bit of medial rotation at the same time, as if to “gap” the knee joint laterally, but you aren’t going to press hard enough; instead, soft tissue will be tensioned.

5. The medial side of the lower leg will be pressed into the bed. Tender/tight spot will relax. Stay with it. After a few minutes, slowly let go, and put the patient’s leg back up on the bed. Upon checking, there should feel like the tissues are all more mobile.

COMMON FIBULAR (PERONEAL) NERVE

The sural nerve is cutaneous to CFN and is being treated at the same time.

Treatment:

1. Palpate tender and or tight spot over the top of the fibula.

2. Monitor it while you perform a balloon technique with the other hand.

See Manual pages for Lower Leg, Ankle and Heel for the rest of the fibular nerve considerations.
**POSTERIOR CUTANEOUS NERVE OF THIGH**

This nerve is an extensive branch of the sciatic.

*Treatment:*

1. In prone, longitudinal skin stretch.
2. In supine, balloon technique.

**OUTSIDE OF THIGH**

**LATERAL CUTANEOUS NERVE BY THE KNEE**

Many of us have gotten confused trying to treat something out here called the iliotibial band. ITBs are blamed for everything and are mostly innocent of all charges. The ITB can’t do anything by itself as it is a passive structure. Instead, think, how can I relieve neural structures that pass through lateral quads and lateral hamstrings that attach to it from underneath, and which innervate the skin over it? I have a few suggestions.

Apart from the usual ortho tests, you can spot tension in the outside part of the leg in standing, simply by looking. The patient will prefer a wide base. If you ask them to stand with their feet together, they’ll often say they feel pain in the hips or pelvis somewhere. Maybe their pelvis will torque one way or another. On a prone patient, on palpation of the outer side of the thigh, one side will feel more “tight” than the other, or maybe both sides will feel “tight”. They might not even like to lay prone, because it hurts their “back”. If this is the case, place some blocks under their hips to lift them up a little. Help them relax so they can focus on their *good* sensations.
Treatment:

1. With one hand, palpate the outside off the leg. With the other, grasp the foot and bend the knee to about 90°, or maybe a little more. Rotate the hip slightly, into inward rotation, by bringing the foot out of the sagittal plane.

2. Ask your patient repeatedly to tell you if they experience any knee discomfort throughout. I’ve treated using this technique for years and have never had any problem with knees, because I check/treat the knees prior and am sure beforehand they can handle the forces I’m about to apply. **TIP: DO THE SAME.**

   Even so, it’s good practice to ask your patient to let you know if they are experiencing any discomfort at any time, during ANY technique, especially this one, where you’ll be adding considerable load.

3. Next, torque the foot, s-l-o-w-l-y, into either internal or external rotation, whichever feels like the best choice for that patient. Go slow so you can choose properly. Or else do both.

4. After you’ve decided how to hold the foot, load some body weight down through the heel, straight down into the knee. I know it seems like sacrilege, but you will find that the overwhelming majority of people will find this pressure quite comfortable. Load in carefully, by stages, always checking with your patient for their comfort level, until – and only until - you feel their leg relax and the ITB area soften. Then hold them there for a few minutes.

5. When you are done, decompress, lift yourself off their foot, carefully bring their lower leg back up into sagittal plane, and lower the foot to the bed, straightening the leg slowly.

When you re-palpate the outside of the thigh, it will feel much softer, as though you have wrung out a lot of water out of the tissues. Most likely the softness is due to a lot of reflexive softening of motor structures. In any case, now they might be able to keep those outsides softer with some attentive exercise.
LOWER LEG, ANKLE, HEEL

SURAL NERVE

Much of the cutaneous lateral lower leg, and lateral heel, are supplied by the sural nerve.

Treatment:

This is quite straightforward. With patient in either prone or supine position, you can balloon the leg anywhere along the length of it.

Treatment using heel:

Using the heel as a lever is simple to do and mechanically affects any nerve in any container passing the ankle, including sural

1. Palpate the back of the leg. Find a tight and or tenderspot to monitor with one hand.

2. With the other, grasp the heel slowly, squeeze it, and turn the soft tissue around the calcaneus, slowly.

3. Patients usually really like this. Hold the tissue turned for as long as you can feel reactions going on.

4. Repeat in the opposite direction.
FIBULAR NERVES

The outer aspect of the anterior leg and top of the foot are cutaneously innervated by these branches (see right). The deeper branch passes under the retinaculum overlying the anterior ankle.

Treatment:

1. Balloon technique around the ankle and hindfoot works quite well for the more superficial branch.

Treatment for the deeper branch

1. Position your patient in sitting, on the edge of the bed. You will sit on a low stool facing them. The foot NOT being treated can rest on a chair.
2. Place the foot you are treating on your knee.
3. Locate and monitor a tender spot in the anterior bend of the ankle a bit lateral to midline.
4. Place the other hand firmly around the calcaneus. With your knee operating as a “third hand”, take the whole foot into some passive dorsiflexion. Externally or internally rotate the skin and outer tissues over the heel, whichever way works better to turn off the tenderness you are monitoring.
5. Hold for a few minutes. Release your grip slowly.

This technique is recommended for tarsal tunnel syndrome as well.

PLANTAR NERVES

Treatment:

Plantar neural tunnels do well with heel twists and shearing types of maneuvers applied to the sides of the feet, or applied to plantar and dorsal surfaces in a circular manner.
Treatment Tips:

In general;
1. Skin on the front of the tibia seems to prefer to be stretched distally.
2. Skin on the front of the fibula likes to be stretched proximally.
3. On the back of the leg, the opposite applies: The skin down along the fibula prefers to be stretched caudally, and along the tibia, proximally.
4. The skin over the heel seems to prefer external rotation forces. But in also likes quite heavy inversion.
5. The skin on the front of the knee seems to prefer to be stretched down medially and up laterally.

I expect these directional preferences likely have to do with the direction of skin ligaments/cutaneous neural “twigs” entering cutis/subcutis from below.

It’s useful to use stretchy tape in these same directions to continue DNM over the next few days following a treatment.
SOME IDEAS ON TAPING FOR DNM

Figure 77

Figure 76
APPENDIX


BIOLOGICAL RESPONSE OF PERIPHERAL NERVES TO LOADING: PATHOPHYSIOLOGY OF NERVE COMPRESSION SYNDROMES AND VIBRATION INDUCED NEUROPATHY

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Introduction
Nerve compression syndromes involve peripheral nerve dysfunction due to localized microvascular interference and structural changes in the nerve or adjacent tissues. Although a well known example is compression of the median nerve at the wrist (e.g., carpal tunnel syndrome) other nerves are vulnerable (e.g., ulnar nerve at the wrist or elbow, spinal nerve roots at the vertebral foramen, etc.).

When tissues are subjected to pressure, they deform and create pressure gradients, redistributing compressed tissue toward areas of lower pressure. Nerve compression syndromes usually occur at sites where the nerve passes through a tight tunnel formed by stiff tissue boundaries. The resultant ‘confined space’ limits tissue movement and can lead to sustained pressure gradients. Based on case reports, space occupying lesions (e.g., lumbroical muscles, tumors, cysts, etc.) can cause nerve compression injury, as can conditions associated with the accumulation of fluid (edema) or extracellular matrix in soft tissues (e.g., pregnancy, congestive heart failure, acromegaly, myxedema hypothyroidism, muscle compartment syndromes etc.). Although nerve injuries related to vibration occur near the region of vibration exposure, they may be manifested at constriction sites. Other conditions, such as diabetes mellitus may increase the susceptibility of the nerve to compression. In addition, an inflammatory reaction may occur which may impair the normal gliding of the nerve. Basic knowledge of the microanatomy of the peripheral nerve and the neuron and their complex reactions to compression are essential to understanding, preventing and treating nerve compression injuries.

Structure and Function of Peripheral Nerves

Microanatomy
The neuron consists of the nerve cell body which is located in the anterior horn of the spinal cord (motor neuron) or in the dorsal root ganglia (sensory neuron), and of a process extending into the periphery – the axon – which is surrounded by Schwann cells arranged in a longitudinal continuous chain forming myelinated nerve fibers (Figure 1 [from Lundborg G. and Dahlin L 1996]). Between the Schwann cells, non-myelinated nerve fibers are located in a large number. Myelinated and non-myelinated nerve fibers are organized in bundles, called fascicles, and surrounded by a mechanical strong membrane consisting of laminas of flattened cells, the perineurial membrane. The bundles are usually organized in groups, held together by a loose connective tissue called the epineurium. In between the nerve fibers and their basal membrane is located an intrafascicular connective tissue – the endoneurium. The amount of the connective tissue components may vary between various nerves and also between various levels along the same nerve. For example, nerves located superficially in the limb or parts of the peripheral nerve that cross a joint contain an increased quantity of connective tissue, possibly as a response to repeated loading (Sunderland 1978).

The normal propagation of impulses in the nerve fibers as well as the communication and nutritional transport system in the neuron – axonal transport – require a sufficient energy supply. The peripheral nerve therefore contains a well developed microvascular system in all connective tissue layers of the nerve (Lundborg 1970, 1975). The vessels approach the nerve trunk segmentally and these vessels have a coiled appearance so that the vascular supply is not impaired during the normal gliding or excursion of the nerve trunk. When the vessels reach the nerve trunk they divide into branches running longitudinally in various layers of the epineurium and also form numerous collaterals to vessels in the perineurial sheath. When the vessels pass through the perineurium into the endoneurium, which primarily contains capillaries, they often go through the perineurium obliquely thereby constituting a possible “valve mechanism” (Lundborg 1970, 1975).

The perineurial layer and the endoneurial vessels play an important role in protecting the nerve fibers in the fascicles. The endoneurial milieu is preserved by a blood-nerve barrier, and the tissue pressure in the fascicle – endoneurial fluid pressure – is slightly positive (Myers et al. 1978). This is obvious when there is injury to the perineurium; following a
transsection a “mushrooming” effect is observed. There are no lymphatic vessels in the epineurial space, therefore problems occur when an edema is formed in the endoneurial space. Following such an edema the pressure in the fascicle may increase and rapidly interfere with the endoneurial microcirculation (Lundborg and Dahlin 1986). The epineurial vessels are more vulnerable than the endoneurial vessels to trauma and even to surgical handling of the nerve.

The neuron itself is, as mentioned above, a unique cell with the cell body and the extending process (axon). The length of the axon may be 10 to 15,000 times the diameter of cell body. Therefore, there is a need for an intraneuronal transport system – axonal transport – where essential products are produced and constantly transported from the nerve cell body down the axon (anterograde transport), and disposal materials and trophic factors are also transported in the opposite direction (retrograde transport) (Grafstein and Forman 1980). The axonal transport consists of various components where fast axonal transport (up to around 410 mm per day) involves various enzymes, transmitter substance vesicles and glycoproteins and the various slow components (up to 30 mm per day) involve mainly cytoskeletal elements such as subunits of microtubules and neurofilaments. It should be noted that axonal transport is energy dependent and disturbances in axonal transport may be involved not only in the development of diabetic neuropathy but also in nerve compression injuries (Dahlin et al. 1986).

Normal Gliding of Nerve Trunks

Outside the peripheral nerve trunk there is a conjunctive like “adventitia” that permits an excursion of the nerve trunk that is a feature of normal nerve functioning during, for example, joint movements. Such an extraneural gliding surface together with the normally occurring sliding of fascicles against each other in deeper layers – intraneural gliding surfaces – make the normal gliding of the nerve possible. The median and ulnar nerve may glide 7.3 and 9.8 mm respectively during full elbow flexion and extension, and the extent of excursion of the nerve just proximal to the wrist is even more pronounced (14.5 and 13.8 mm respectively) (Wilgis and Murphy 1986). In relation to the flexor retinaculum the median nerve may move up to 9.6 mm during wrist flexion and to a slight degree in wrist extension but the nerve also moves during finger movements (Millesi et al. 1990).


REFERRED PAIN OF PERIPHERAL NERVE ORIGIN: AN ALTERNATIVE TO THE "MYOFASCIAL PAIN" CONSTRUCT

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Although painful conditions of varying degrees of severity involving the soft tissues (i.e., muscles, tendons, ligaments, and peripheral nerves) occur frequently, their underlying pathogenesis is poorly understood. During the 19th century, these conditions were called muscular rheumatism or fibrositis to distinguish them from conditions such as articular rheumatism, which primarily involve joints (1). Chronic forms of muscular rheumatism were attributed to inflammation of a “peculiar” kind affecting the fibrous tissues around joints; this inflammation was found in tendons, bursae, ligaments, fascia, nerve sheaths, muscles, and peroneum (2). Others only used the term rheumatism when they wished to denote the presence of non-specific inflammation involving voluntary muscle fibres (1,3).

In the early part of this century, the English neurologist Gowers (4) championed the concept of “fibrositis” as a painful inflammatory disorder of the fibrous structure of muscle spindles (at that time the only known sensory structures in muscle). He taught that “fibrositic” inflammation could spread by direct fascial extension to involve nearby tendons, joints, and nerve sheaths (interstitial neuritis), thus unifying the two conceptual models of the 19th century. By the 1930s his views had gained general acceptance. The committee on “arthritis and allied conditions” appointed by the British Medical Association in 1933 recognized the following subgroups of fibrositis: intramuscular and facial; periarticular; bursal and tenosynovial; subcutaneous (panniculitis); and perineuritic (5). The clinical sequela attributed to “perineuritis” included radiating pain, paresthesiae, cutaneous hyperesthesia, tenderness in muscles and joints in the sensory area served by the nerves involved, and tenderness over the site of the nerve itself, which was attributed to the involvement of the nervi nervorum. Motor and sensory impairments were uncommon (5).

Over the next 50 years, clinicians attempting to unravel the complex nature of muscular rheumatism focused their attention on palpatory findings in and around voluntary muscles, almost to the exclusion of a possible contribution to pain from peripheral nerves or other tissues. The concept of the “fibrositic nodule,” described as “an area in the substance of a muscle or its tendinous sheath which gives rise to pain either in the same locality or referred to a distance when stimulated” (6), was discarded when it became clear that it lacked pathological support (7). In addition, as knowledge of spinal disc pathology increased, it was argued that so-called fibrositic lesions in muscles could be explained as secondary or referred phenomena (8).

However, those who still adhered to the belief that many localized or regional chronic pain syndromes are attributable to more subtle pathological changes occurring solely within voluntary muscle put forward the construct of the myofascial pain syndrome (9). More recently, another, competing, construct has been redefined to account for chronic and widespread musculoskeletal pain, namely fibromyalgia syndrome (10). The main diagnostic criterion for this syndrome is the presence of a defined number of “tender points” at predetermined anatomical sites. We have argued elsewhere that this construct conveys no pathophysiological insights, having been derived by a process of circular argument (11). Moreover, some
authors have recognized that there may be considerable clinical overlap between the two syndromes (12,13). In view of the controversial and complex nature of these pain syndromes, a critical analysis of the prevailing hypotheses is justified to clarify the situation.

In this article, the hypothesis that pain arising from trigger areas within muscles is of primary myofascial origin is critically examined. It will be shown on epistemological, clinical, and pathophysiological grounds that the myofascial pain syndrome (MPS) construct is invalid and that the phenomena it purports to explain are better understood as secondary hyperalgesia of peripheral neural origin.

THE "MYOFASCIAL PAIN" CONSTRUCT

The major sources for the synthesis provided herein are the principal writings of the proponents of MPS (9,14-19). Definition and basic phenomenology

Myofascial pain has been described as "the most common cause of chronic pain" (17). Introduced in 1952 after a decade of research, and developed since by Travell and her co-workers (9,15), MPS has been defined as a regional pain syndrome with two major components: (a) the trigger point, a localized area of deep muscle tenderness or hyperirritability; and (b) a predictable, discrete reference zone of deep aching pain, which may be located in the immediate region of or remote from the trigger point (TrP), may be quite extensive, and is worsened by palpation of the TrP.

Triggers points have been described in skin, joint capsules, ligaments, periosteum as well as in muscles and their fasciae. Myofascial TrPs are said to be located within palpable taut bands, purported to represent shortened muscle fibres. On "snapping" palpation or needleling of a myofascial TrP, a local twitch response can be elicited. This clinical sign is accompanied by an irritable electromyographic response. A muscle containing a TrP exhibits antalgic inhibition when tested for strength and is also intolerant to stretch. In a seeming contradiction, muscle stretching is recommended as being efficacious treatment for myofascial pain. Relief of pain requires "inactivation" of the relevant trigger area, by physical (needleling or stretch) or chemical (local anaesthetic) means.

Travell and Simons (15) insist that the "specific muscle or muscle group that causes the symptoms should be identified." More recently, Simons (19) has defined MPS as "primarily a dysfunction of one or more specific muscles" (emphasis added). The constancy of distribution of pain referred from individual muscles is said to enable the clinician to "work backward" and thus locate the TrP(s) responsible for particular pain patterns.

One of our main criticisms of the construct of myofascial pain is that its major proponents have incorporated their preferred hypothesis of causation within the definition. As will be shown elsewhere in this article, this error in reasoning has limited the discussion of other explanations for the various clinical phenomena observed in these syndromes. Metaphysics of trigger points

The TrP is said to "cause" (16) or have "the propensity to cause" (18) or "the responsibility for causing" (17) local and referred pain. It has even been suggested that TrPs may at times "refer" hypoesthesia or anaesthesia instead of pain (19). Trigger points may be "active," "latent," "satellite," or "secondary." Active TrPs are more likely to be found in musculature of the neck, shoulder, and pelvic girdles and in the muscles of mastication. They can occur in multiple locations in any one muscle; their site(s) can vary from person to person and their irritability is said to vary from hour to hour and from day to day.

A TrP is considered latent or dormant if it is not causing referred pain. Latent TrPs can be found in asymptomatic subjects, in whom the TrPs are nonetheless said to restrict movement and cause weakness in the affected muscle (20). Latent TrPs are said to accumulate with advancing age (14).

Satellite TrPs are those that can be found in muscles within the pain reference zone of another TrP. Secondary TrPs develop in muscles that are either synergists or antagonists of the muscle that contains the primary TrP. Synergists are said to be overloaded when they substitute for the affected muscle and antagonists are said to be overloaded when they counter its tautness.

Initiation

It was originally proposed that myofascial TrPs may be initiated by "direct trauma to muscle or joint, chronic muscular strain, chilling of fatigued muscle, acute myositis, arthritis, nerve root injury, visceral ischemia or dykinesia, and hysteria" (9). These same factors, plus resumption of normal activity after periods of immobility, are also said to be capable of activating latent TrPs. A latent TrP may even be activated during therapy: as one set of muscles is being stretched, their antagonists, which presumably contain the latent TrP, are shortening.

Myofascial pain is now mainly ascribed to an initial insult to muscle fibers, either from macrotrauma or repetitive microtrauma (16). The consequences of such an insult may include release of such substances as histamine, serotonin, kinins, and prostaglandins which may then activate nociceptors and cause reflex muscle contraction.

However, this proposition of muscle injury lacks empirical support. Muscle pain and damage following eccentric contractions have been extensively studied (21). In normal subjects, complete recovery is the rule and no long lasting
Dermoneuromodulation

effects have been noted. Unless muscle strains are severe (e.g., complete tears) or associated with deep haematoma formation, recovery is complete. Sever distraction or contusion injuries are common in sport but no evidence has been presented that such well-defined acute injuries are antecedents of MPS. Furthermore, electromyography of painful muscles (22) and thermographic studies of the tissues overlying them (23) have not demonstrated abnormalities in TrPs. Muscle biopsy studies of TrPs have also been largely unrewarding in terms of muscle inflammation or damage.

Perpetuation

The chronicity of pain that follows the activation of a myofascial TrP has been explained by a feedback cycle maintained by bombardment of the central nervous system (CNS) by impulses from TrPs themselves: that is, they become self-perpetuating. However, remote lesions in joints or chronic visceral disease and dysfunction may also provide noxious input into this cycle, as may emotional stimuli, chronic infection, various metabolic disturbances, and even dietary deficiencies (14).

As the painful muscles in MPS are electrically silent, the presence of muscle spasm that may reflect ectopic impulse formation seems most unlikely (22). Furthermore, the efferent arm of the proposed vicious cycle has been tested. Mense (24) found that gamma-motoneuron activity was diminished rather than increased in muscles with carrageenan-induced injury and concluded that the proposed vicious-circle models "have to be considered as working hypotheses rather than explanations of known mechanisms.

Spread

Spread of pain is attributed to latent TrPs being activated or to active myofascial TrPs "metastasizing" to sites within or outside of the pain-reference zone of the original TrP(s) (18). Travell (14) postulated a chain reaction whereby an ever-increasing number of satellite TrPs come into being, causing complex overlapping patterns of pain.

Reliability of TrP phenomena

When blinded as to diagnosis, those expert in the field of MPS were able to detect active TrPs in only 18% of examinations of subjects with a MPS diagnosis (25). In the same study, expert assessments for taut bands and muscle twitch responses were also found to be unreliable. These findings call into question the internal validity of the construct.

Treatment

Inactivation of the TrP by physical and chemical means would be predicted if the TrP is indeed a site of primary hyperalgesia. However, reports of the efficacy of this approach are only anecdotal; inactivation has not been subjected to formal trial. Furthermore, the persistence of using the recommended approach in the face of clinical inefficacy, along with the continuing failure over time to reveal a reasonable anatomical or pathophysiological basis for so doing, is not only irrational but also fails to acknowledge powerful placebo effects (26) and the wider psychosocial context of chronic pain (27). Objections to MPS construct

The definition of MPS incorporates a preferred hypothesis of causation. This logical error has resulted in a system of diagnosis and treatment that has become popular but remains entirely anecdotal. Moreover, the proposition that myofascial pain and TrPs are intimately related constitutes circular reasoning: that is, by virtue of its form this proposition must always be true (Table 1).

In their efforts to preserve the centrality of the myofascial TrP, myofascial pain theorists have lowered the number and nature of predisposing, precipitating, and perpetuating factors to be open-ended and to encompass the full spectrum of aetiology, including the untestable psychogenic level. (16,17). This serves only to perpetuate the circularity of the reasoning.

Perhaps in an attempt to provide external validity, researchers have said that TrPs arise from muscle damage, despite electrical silence and the lack of histological or biochemical evidence. Furthermore, there is neither support from an animal experimental model (24) nor from studies of human muscle injury (21). Trigger points are nonetheless said to be maintained via the CNS, not only by their own activity but also by a legion of processes associated with afferent neural input. Spread of pain is attributed to the activation of latent TrPs or to the metastasis of TrPs. This teleological argument is physiologically unsound.

Taken together, the tenets of the MPS construct arise out of circular reasoning, which should condemn MPS as epistemologically unacceptable.*MYOFASCIAL PAIN* VERSUS PERIPHERAL NEURAL PAIN

The argument that follows explores a putative relationship between "myofascial pain" and pain of peripheral neural origin. We show that the explanation for peripheral neural involvement in MPS, which depends on nerve compression by "taut bands," is speculative and unconvincing. Application of current concepts of the physiology of nociception can lead to an alternate construct. Differential diagnosis of MPS

The differential diagnosis of myofascial pain, as proposed (14,16), includes a variety of painful and somewhat loosely defined neurological conditions such as thoracic outlet syndrome (28), radiculopathies, and polyneuropathies. Their differentiation from myofascial pain is said to be facilitated by the presence of accompanying neurological deficits (particularly those matching a peripheral nerve or root distribution) and electrodiagnostic abnormalities (15). Although a
Dermoneuromodulation

fundamental distinction has been made between TrP pain (deep and aching) and pain of peripheral neural origin (prickling, tingling, and numbing). Dalton and Jull (29) were not able to distinguish between somatogenic and neurogenic cervicobrachial pain when they relied solely on the characteristics of pain. Moreover, peripheral neural pain can occur without neurological deficit (30) and without conventional electrodiagnostic abnormality (31).

By contrast, when neurological deficit (often accompanied by electrodiagnostic abnormality) accompanies MPS, it has been ascribed to peripheral nerve entrapment by the taut band containing the TrP (16,19). The taut band is said to cause an overall shortening of the involved muscle, which then, in turn, can lead to a “secondary” nerve entrapment syndrome (32). The dual propositions that neurogenic mechanisms can activate myofascial TrPs and that myofascial TrPs can cause neurogenic pain add up to a circular argument. Furthermore, the neurological literature does not include the TrP taut band as a recognized anatomical cause of entrapment neuropathy (33,34).

However, on clinical grounds alone, there appears to be an intimate relationship between MPS and defined neuropathology. This relationship is worth exploring in terms of current understanding of nociceptive mechanisms. Characteristics of myofascial pain

The pain attributed to myofascial TrPs is described as deep, dull, and aching, varying in intensity from mild to severe and occurring either at rest or only on motion (Table 2). These are the characteristics of deep somatic pain. By the 1930s, it had been long known that pain arising in deeply situated joints was often referred to anatomically distant structures. The seminal clinical experiments carried out by Lewis (35) and Keligren (36) convincingly demonstrated the same phenomenon for pain arising in other deep musculoskeletal tissues, such as muscles, ligaments, and periosteum.

According to Keligren (36), "The diffuse pain from a given muscle is always distributed within certain regions, though the distribution within these limits varies from individual to individual, and according to the part of the muscle stimulated" and “pain from muscle may be confused with pain arising from other deep structures such as joints and tests:"

Some caution is therefore necessary before a mechanically provoked pain response is attributed to a particular structure or structures. Afferents from muscles that are the sites of referred pain and tenderness are the very ones that converge centrally onto spinal neurones that could be involved in processing information from a region of deep damage, thus leading to central summation effects (37).

Vasconstriction, hypoesthesia, dermographia, and hyperhidrosis have been observed in the skin overlying a region of deep pain. These phenomena appear to be reflexly induced concomitants of somatic referred pain (38). Peripheral neural pain

The connective tissues of human peripheral nerves are well-innervated. They derive their nerve supply from axons within the nerve and from fibres accompanying the extrinsic vessels that provide its nutrition (39). As well as regulating intraneural microcirculation, this intrinsic nerve system, the nervi nervorum, is thought to have a nociceptive function (40).

Two types of pain, present singly or in combination, have been described in patients with peripheral neuropathy: "nerve trunk pain" and "dysesthetic pain" (41). The former pain has been described as aching, knife-like, or tender, whereas the latter has been described as burning, tingling, searing, crawling, drawing, or electric. Nerve trunk pain is therefore indistinguishable from pain described as myofascial (see Table 2). Nerve trunk pain has been attributed to increased activity in mechanically or chemically sensitized nociceptors within the nerve sheath, while dysesthetic pain has been attributed to damaged nociceptive afferent axons themselves.

Nerve trunk pain characteristically follows the course of the involved nerve, which is found to be tender, whereas dysesthetic pain is felt in its peripheral sensory distribution (41). However, when pain of nerve origin is severe, it can be felt in regions outside the sensory distribution of the particular nerve (33,34).

Peripheral neural pain may be associated with neurological deficit, but it can be accompanied by a hyperaesthetic syndrome, which includes both allodynia (pain due to a normally non-painful stimulus) and hyperalgesia (an increased response to a normally painful stimulus) (42-44). The term peripheral neuropathic pain has recently been suggested to embrace the combination of positive and negative symptoms in patients in whom pain is due to pathological changes or dysfunction in peripheral nerves or nerve roots (45).

Pain with the characteristics of "nerve trunk pain" has been described by patients with irritative cervical (46) and lumbar (47) radicular lesions, with brachial neuropathy (40), and following peripheral nerve injury (48).

Most nerve pain syndromes commence with symptoms more in keeping with an irritative than a destructive process (49,50). Local tenderness is commonly found over nerve trunks at sites of entrapment or metabolic insult; this tenderness has been attributed to sensitization of free nerve endings within neural connective tissue (nervi nervorum) (40). Such specific tender points over peripheral nerves, palpation of which could cause distant pain, was reported over a century ago (51). It has recently been suggested that radiating pain and other sensory phenomena could originate from ectopic neural pacemaker nodules formed at a site of entrapment (52). Tenderness has also been noted over motor bands (zone of innervation) and muscles in association with cervical and lumbar radicular pain without gross physical signs of denervation (53). Neuropathic pain states are frequently accompanied by abnormalities in functioning of the sympathetic nervous system (54). Referred neural pain
Intraneural stimulation of muscle fascicles within the median and ulnar nerve trunks of normal volunteers has been shown to refer pain both distally to muscles within the innervation territories of each nerve, and proximally to deep structures (muscle and bone) in segmentally related regions outside the innervation territory of each nerve (53,56).

Recounting his personal and clinical experience, Ochoa (57) described both local elbow pain and referral of pain into the ipsilateral scapular region following mechanical stimulation of an entrapped ulnar nerve at the elbow. In his own and the other cases, none of the distal symptomatology typical of ulnar neuropathy was present.

Thus, peripheral neural tissue is a rich source of local and potential referred pain. Anatomical concordance of myofascial TrPs and peripheral nerves

Some TrPs said to be myofascial could be situated in an adjacent hyperalgesic nerve trunk. For example, the discrete upper-limb pain syndromes attributed to TrPs in the middle finger extensor, the extensor carpi radialis, and the supinator muscles can equally be attributed to TrPs in the radial or posterior interosseous nerve trunks. The TrP said to be situated in the pronator teres muscle coincides with the median nerve, and the pain projected there from into the thenar muscles follows the course of the median nerve in the forearm. TrPs in the flexores digitorum referring pain into the hand may represent a tender compressed median nerve in the proximal forearm. MPSs in the shoulder girdle region may represent entrapment of the suprascapular nerve, the long thoracic nerve, the axillary nerve, and the dorsal scapular nerve, as the pain-reference zone of the TrPs follow the course of these nerves. In the lower limb, MPSs have been attributed to TrPs close to the sciatic, tibial, and superficial and deep peroneal nerves.

ALTERNATIVE EXPLANATIONS FOR MPS PHENOMENA

Alternative explanations for MPS phenomena are summarized in Table 3.

TrPs as sites of secondary hyperalgesia:
The weight of evidence does not support myofascial TrPs as the anatomical sites of pain origin. By contrast, the presence of hyperalgesia in muscles that are structurally and electrically normal suggests that it must be secondary (referred) hyperalgesia (58). This hyperalgesia could be due to peripheral mechanisms such as antidromic activation or sensitization of nociceptive afferents (59) or, more likely, to a state of central sensitization, including spontaneous firing and expansion of the receptive fields of nociceptive dorsal horn neurones (60).

Spread of pain:
Latent, metastasising, and secondary TrPs lack supporting evidence, as does nerve entrapment by taut bands. The spread of pain is more likely to be the consequence of altered central nociceptive processes and enlarged receptive fields in response to ongoing nociception or ectopic impulse generation (60).

Intolerance of muscle to stretch:
The taut bands described in muscles containing TrPs may represent reflex spasm secondary to nociception in structures innervated by the same spinal segment (8). The intolerance to stretch could also be explained as a reflex response to the stretching of adjacent hyperalgesic neural tissue.

Chronicity of pain:
MPS theorists attribute chronicity of pain to the self-perpetuating propensity of TrPs, usually in the presence of an assortment of other factors such as a short leg, poor posture, somatoform pain disorder, chronic infection, and secondary gain-all of which are teleological arguments. Alternatively, it has been shown that the altered central processing held responsible for secondary hyperalgesia may be maintained by nociception elsewhere possibly including, of course, peripheral neural structures (61).

Hypoesthesia:
There are two explanations for hypoesthesia in MPS theory: compressive neuropathy by a taut band or a referred phenomenon reflecting the downward modulation of receptive fields in the pain reference zone of the TrP (19). Irrespective of the particular entrapping mechanism, it is accepted that hypoesthesia results from the loss of sensory afferents due to nerve compression at the site of an entrapment (33). However, hypoesthesia has also been attributed to a functional block occurring at spinal or higher levels associated with a peripheral neural pain state (43).

Vasomotor and sudomotor disturbances:
Disturbances of sympathetic efferent function that have been described in association with MPS have also been recognised as reflexly induced accompaniments of neuropathic pain states.

CONCLUSION

The construct of MPS, as proposed to explain chronic, deep, aching, poorly localized pain, not only lacks internal and external validity but also is epistemologically unsound. The emphasis on the primacy of the TrP phenomenon has directed attention away from other possible explanations. By contrast, there are anatomical and physiological grounds to suggest that the phenomenon of the TrP, on which depends the theory of MPS, is better understood as a region of secondary hyperalgesia of peripheral nerve origin. This proposition is testable to achieve external validity for the described clinical phenomena.
TABLE 1. Problems with the MPS hypothesis

| Definition of syndrome incorporates hypothesis of causation. |
| TrPs lack clinical reliability and validity. |
| Predisposing, precipitating, and perpetuating factors are legion. |
| Histological, biochemical, and electrical evidence of primary muscle pathology is lacking. |
| There is no support for the MPS hypothesis from animal experimental models or human muscle injuries. |
| Trigger points are an operational concept elevated to the status of theory by circular reasoning. |

TABLE 2. Comparison of peripheral neural pain with myofascial pain

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Myofascial Pain Syndrome</th>
<th>Peripheral neural pain nerve trunk variety</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain Descriptors</td>
<td>Deep, dull, aching</td>
<td>Deep aching</td>
</tr>
<tr>
<td>Tenderness</td>
<td>TrP in muscle (active or latent)</td>
<td>Nerve trunk (local)</td>
</tr>
<tr>
<td></td>
<td>TrPs satellite/secondary</td>
<td>Somatic referred</td>
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<tr>
<td>Associated phenomena</td>
<td>Sympathetic dysfunction</td>
<td>Sympathetic dysfunction Neuropathic phenomena</td>
</tr>
<tr>
<td>Electrodiagnostic abnormality</td>
<td>Usually absent</td>
<td>Usually absent</td>
</tr>
<tr>
<td>Therapeutic implications</td>
<td>Desensitisation (inactivation of TRPs)</td>
<td>Nerve decompression</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Treat neuropathic pain</td>
</tr>
</tbody>
</table>

TABLE 3. Pathophysiological explanation for the phenomena of myofascial pain syndrome

<table>
<thead>
<tr>
<th>Phenomena</th>
<th>MPS theory explanation</th>
<th>Referred explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperalgesia</td>
<td>TrPs (primary hyperalgesia)</td>
<td>Secondary (referred)</td>
</tr>
<tr>
<td>Spread of pain</td>
<td>Activation of TrPs nerve entrapment by taut band</td>
<td>Sensitisation of nervi nervorum central sensitisation</td>
</tr>
<tr>
<td>Intolerance of muscle to stretch</td>
<td>Contracture of taut band</td>
<td>Reflex spasm, secondary to nociceptors elsewhere</td>
</tr>
<tr>
<td>Chronicity</td>
<td>Self perpetuation, many other factors</td>
<td>Maintenance by nociception elsewhere. Central sensitisation</td>
</tr>
<tr>
<td>Cutaneous correlates</td>
<td>Nerve entrapment</td>
<td>Nerve compression itself</td>
</tr>
<tr>
<td>Hypoesthesia</td>
<td>Reflex efferent phenomena</td>
<td>Reflex referred phenomena</td>
</tr>
<tr>
<td>Vasomotor/Sudomotor</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
REFERENCES


