NielAsher.

Advanced Trigger Point Techniques

Trigger Point Therapy Course

Spondylolisthesis

WELCOME

Spondylolisthesis can cause significant amounts of pain and discomfort. Even though 80% of cases can be effectively managed[1] with conservative measures, patients are often left confused, in pain, and compelled to seek help. This course is designed to help you understand what spondylolisthesis is, and to offer you a clear hands-on pathway for its treatment and management. We passionately believe that an understanding of trigger points and how to use them will give you valuable extra tools for treatment. Weaving trigger points into your massage or soft tissue routines can have truly profound effects. Combine this with self-help, stretching, and advice on lifestyle modification, and you should be able to help the majority of those in pain.

At the end of this course, you will find a reflective learning exam. This is not a 'pass or fail' test, but a mechanism to see whether you have understood the information and can apply it for the good of your patients.

We are excited to share this information with you, and don't forget that if you have any questions, we are here to support you.

CONTENTS

WHAT IS SPONDYLOLISTHESIS?

Types of spondylolisthesis[2]:

☐ Type I: Congenital spondylolisthesis

☐ Type II: Isthmic spondylolisthesis

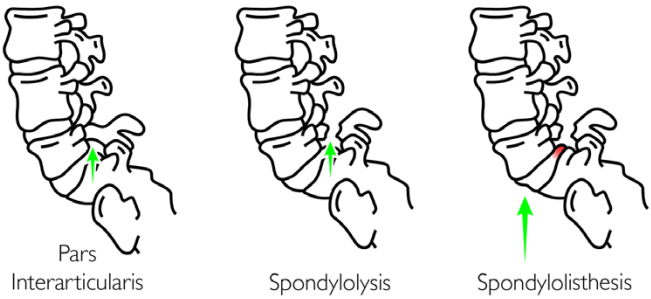
☐ Type III: Degenerative spondylolisthesis

☐ Type IV: Traumatic spondylolisthesis

☐ Type V: Pathologic spondylolisthesis

☐ Type VI: Postsurgical

Spondylolisthesis is a fracture and slippage of a spinal vertebra (usually lower lumbar). It can develop for a range of reasons and can vary in severity; the majority fall into one of two categories: traumatic or degenerative. Whatever the underlying reason, the result is that the vertebrae literally slip either forward on the one below (anterolisthesis) or backwards (retrolisthesis). This can sometimes be felt as a palpable 'step' as you run your fingers down the patient's spine.



Congenital

Congenital spondylolisthesis is due to dysplastic sacral or lower lumbar segments. Dysplastic facets or abnormal orientation of the facet joints are the cause for spondylolisthesis.

Traumatic/Isthmic

Isthmus spondylolisthesis is rare – it is often due to displacing a small fracture in a piece of bone called the pars interarticularis (spondylolysis). Pars interarticularis defect often occurs because the bone didn't fully form from its infantile cartilage (this can be due to a lack of folic acid during pregnancy, amongst other things). Traumatic spondylolisthesis usually affects the lower part of the spine, most commonly the 5th lumbar vertebra (82%), but sometimes the 4th lumbar vertebra[3].

The usual mechanism is that a child or youth falls heavily onto the coccyx region and the cartilaginous pars splits. This type of fracture usually occurs around 5-7 years of age; however, the symptoms may not be felt until adulthood. Usually a traumatic slippage is a grade I or II (see later) and is well adapted and compensated for by the body over time. However, it is not uncommon to see young boys (mainly) of 14/15 years old coming to you with acute spondylolisthesis-induced low back pain. Younger patients are at higher risk than older patients for **developing progressive spondylolisthesis**. The risk for progression in **adults** is **rare** when the main problem is at L5.

Degenerative

Usually due to aging, a degenerative spondylolisthesis happens where the spine's support system (bones, joints, and ligaments) weaken and are no longer able to maintain alignment. Degenerative spondylolisthesis is most common in the 5th decade onwards, and usually occurs at the L4/5

zone because that's mechanically weaker due to the maximal angle of the lumbar lordosis.

This is sometimes also called a 'stress fracture'. It occurs more commonly in **females**, with a **5:1 female to male ratio**. The incidence increases after age 40 years.

Pathologic spondylolisthesis

Can occur as a result of any bone lesion that might weaken the posterior bony structures. Generalized skeletal diseases including osteomalacia, syphilitic disease, and Von Recklinghausen disease are some reported causes. Bony destructive lesions including tumor or infection are other potential causes.

Spondylolisthesis facts (Nabil Al Ali et al 2005)[4]:

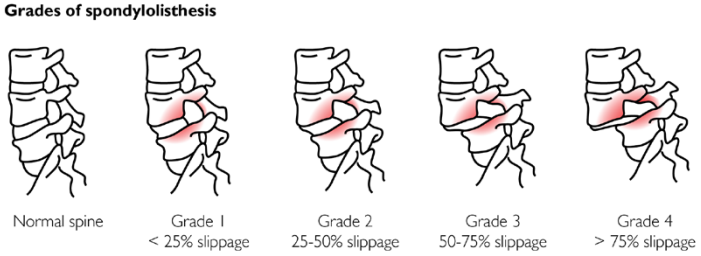
- **The L4-L5 interspace is affected 6-10 more times than any other level**
- **Approximately 82% of cases of isthmic spondylolisthesis occur at L5-S1**
- **Another 11.3% occur at L4-L5**
- **Degenerative spondylolisthesis occurs more frequently with increasing age, especially after 40**
- **Congenital/dysplastic spondylolisthesis has been documented in children as young as 3.5 months**
- **Heavy athletic activities may predispose some athletes to developing pars defects[5]**
- **Isthmic spondylolytic defects affect roughly 1.1% of black females**
- **The most commonly affected group is the white male, with an incidence of 6.4%**

- **Arkara Plains Indians and Aleut people groups have a very high incidence of spondylolytic defects, due to a combination of genetic and environmental factors[6]**
- **Degenerative spondylolisthesis affects black females more commonly than white females (females more affected than males)**

Symptoms

Patients typically have complaints of pain in the back with intermittent pain to the legs. Spondylolisthesis can often cause muscle spasms, or tightness in the hamstrings.

Symptoms will depend on a number of factors, age, chronicity, and the degree/grade of slippage. We grade slippage from I-IV according to the percentage of slippage, and as the bone slips it can also pull on the intervertebral disc, which can be a source of pain. Remember that most spondolistheses occur at the L5/S1 level. Anatomically, most of the spinal nerves have exited to the lower extremities by the L3/4 zone (the cauda equina starts at L2/3), so any nerve damage or symptoms will tend to be less serious than if the slippage was higher up. It can affect one side (unilateral) or both sides (bilateral), and often presents as sciatica or pseudosciatica. If severe, it can also cause **cauda equine syndrome** (CES). CES is potentially very serious, and may present as a loss of bladder or bowel control/regulation, in which case you must urge the patient to seek **immediate medical/surgical intervention**.



Symptoms of spondylolisthesis may include:

- Difficulty standing straight
- Back or buttock pain
- Pain that runs from the lower back down one or both legs (sciatica)
- Numbness or weakness in one or both legs
- Difficulty walking
- Leg, back, or buttock pain that gets worse on bending over or twisting
- Loss of bladder or bowel control, in rare cases
- Pain getting up from a chair
- Morning pain and stiffness
- Back pain getting worse as the day goes on
- Pain usually is provoked by activity, particularly back extension activities
- Poor tolerance of activities requiring excessive spine loading, including running and jumping
- Sitting usually is better tolerated
- A large percentage of patients with spondylolysis are asymptomatic. Progression of a spondylolisthesis also may occur without symptoms

What are the symptoms telling us?

Pain is a signal that something is wrong – it's part of our 'protect and defend mechanism'. In spondylolisthesis, back pain is the most common symptom; this is mainly due to overloaded lumbar erector spinae and or multifidus muscles.

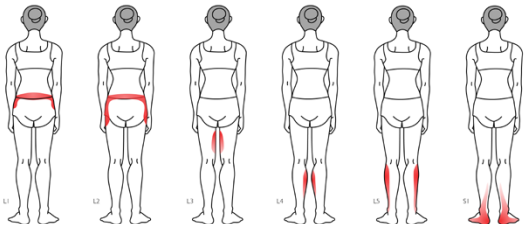
The muscles are being asked to do two jobs at the same time – the normal job of maintaining posture (multifidus) and extending the flexed spine (erector spinae) **PLUS** the job of stabilizing around the fracture site.

This causes muscle fatigue and low grade constant aching, which is also one of the reasons why the lower back pain is often worse during the day.

As the muscles fatigue, the pressure on the spinal cord and/or its neurovascular structures may increase and cause more leg pain and/or hamstring tension.

Patients often present with slight forward (flexion) bending (the Phalen-Dickson sign). This posture leads to buttock pain as the gluteus maximus and medius and hamstring muscles become similarly engaged in protecting and stabilizing the lower back and hips through their myofascial attachments. Over time, this leads to chronic tight clenched buttocks, often with spasms and pain; this buttock clenching tension can be seen clearly during examination. Gluteal muscle spasm and tension can in turn lead to tension in the piriformis muscles, which engorge and then press upon the sciatic nerve (causing sciatica) or its blood supply (pseudosciatica).

Numbness: May be one sided (unilateral) or bilateral. It is usually felt down the back of the legs, but this depends on where the spondylolisthesis is located. Each nerve that exits the spine has a specific radiating pattern or dermatome. See chart below. Often the leg pain is not directly related to pressure on the nerves, but may be coming from muscular trigger points and associated tight muscles. Tight muscles can press on the delicate blood supply to the nerves and mimic nerve pain (myogenic neuropathy). In these cases, trigger point (NAT) therapy can be very, very effective.



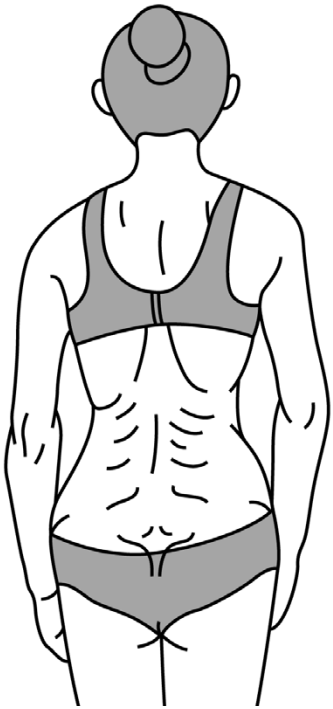
EXAMINATION AND TESTING

In most cases, it is not possible to see visible signs of spondylolisthesis by examining the patient; however, there are a few tests you can do. The patient may present with a waddling gait. This may be noted secondary to hamstring tightness producing a shortened stride length. The patient also often presents with tight clenched buttocks. A step or bump in the spinous processes can sometimes be felt as you stroke down the spine.

Typical Spondylolisthesis postures

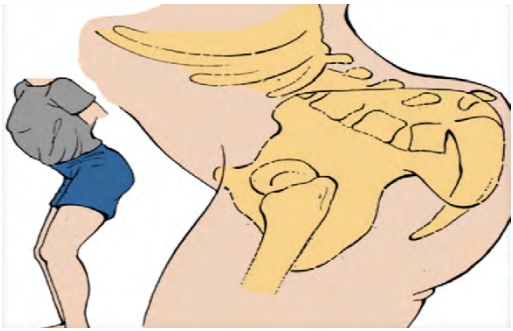
12 year old girl
Isthmic spondylolisthesis

63 year old woman degenerative spondylolisthesis



The Phalen-Dickson sign

With increasing slippage, the sacrum becomes relatively more vertical, impairing hip extension and compelling the patient to walk with a knee-flexed, hip-flexed gait.



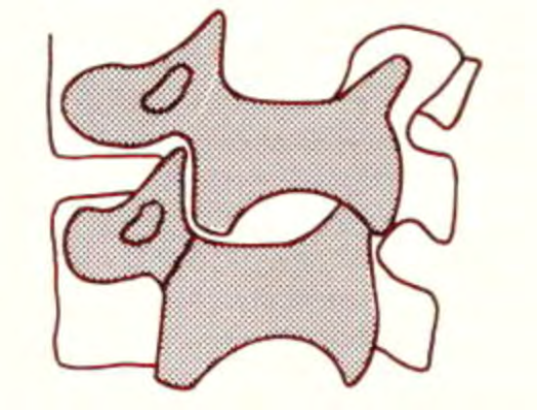
One-legged hyperextension test (stork test):

Positive one-legged hyperextension test, while standing on one leg and bending backward; pain is experienced in the same side (ipsilateral) of the low back.

Diagnostic tests[7]

1. Radiography:

Spondylolisthesis is easily identified using plain radiographs A/P, lateral, and oblique. Lateral (side) view of lumbar spine is especially useful in the detection of spondylolisthesis. Oblique x-ray also clearly shows the appearance of bilateral pars defects – this resembles a Scottie dog with a collar (the collar is the pars defect).



2. Computed Tomography:

CT SCANNING axial or sagittal image of the lumbar spine can be performed with or without contrast enhancement.

3. Magnetic Resonance Imaging (MRI):

Has the distinct advantage of imaging the spine in any plane. Typically, the axial (up/down) and sagittal (side view) planes are used.

DIFFERENTIAL DIAGNOSIS - What Else Could It Be?

Differential diagnosis is an essential tool to assess the risk of any problems when patients come to the clinic for treatment. 90% of musculoskeletal pain is benign in origin, but it's important to know more about that 10%. Early detection and treatment can facilitate effective recovery.

We highly recommend using the pathological sieve **CDFIMNRT** to sift through the potential alternatives to spondylolisthesis; of course, it is possible to have two or more conditions occurring at the same time. This sieve is a quick and easy format that you can apply to all complaints.

PATHOLOGICAL SIEVE	
SPONDYLOLISTHESIS	
CONGENITAL	Spondylolysis, anomalies of the lumbar or sacral vertebrae such as sacralization or lumbarization
DEGENERATIVE	Spinal bulging or prolapsed disk, spinal stenosis (narrowing of the vertebral nerve exits), arthritis of the spine pressing (bars and/or osteophytes) on the nerves or ligaments, spinal fracture (or crush fracture)
FUNCTIONAL	Sacroiliac joint injury/dysfunction/derangement, Overuse/sitting/cross-legged/high heeled shoes, piriformis syndrome, ischial tunnel syndrome
INFECTIVE	Tuberculosis, osteomalacia, syphilitic disease, and Von Recklinghausen disease
METABOLIC	Osteoporosis, hyperparathyroidism
NEOPLASTIC	Primary neoplastic, secondary neoplastic – spinal fracture (or crush fracture)

RETICULO-ENDOTHELIAL	Neuropathy: e.g., Mononeuropathy monoplex, Systemic Lupus (SLE) Erythematosis, multiple myeloma
TRAUMATIC	Fracture, accident

Red flags

- Pain that develops gradually, and slowly gets worse and worse over days or weeks
- Constant back pain that is not eased by lying down or resting
- Pain that travels to the chest, or is higher in the back behind the chest
- Weakness of any muscles in a leg or foot
- Lack of feeling (numbness) in any part of your bottom or leg
- If you have taken steroid tablets for more than 3 months
- Numbness around the back passage (anus) and the saddle area
- Bladder symptoms such as loss of bladder sensation: loss of bladder control, incontinence, loss of sensation when passing urine
- Incontinence (faeces, stools, or emotions)

Symptoms that may indicate a non-spondylolisthesis fracture in the spine:

- Back pain following major trauma such as a road accident or fall from a height
- Back pain following minor trauma in people with osteoporosis

WHICH MUSCLES ARE AFFECTED BY SPONDYLOLISTHESIS?

- Piriformis
- Lumbar erector spinae
- Gluteus medius
- Gluteus maximus
- Multifidus
- Hamstrings

Muscles overview

As we have discussed, the brain/body is forced to adjust to the new situation of a permanently 'slipped vertebrae'. Spondylolisthesis will affect the whole body – note the flexed knees in the pictures above. There will often be changes through the hip flexors and 'core abdominal muscles causing weakness. However, it is mainly the local muscles that bear the brunt of the work, such as the multifidus and lumbar erector spinae.

Erector spinae

Interestingly, and contrary to what some of us have been taught, the erector spinae don't hold the spine erect! Most fibres are electrically silent during postural work (Kippers 1984)[8]. This muscle group is designed to activate during extension from flexion – i.e. standing upright from bending forward. Because most patients with a spondylolisthesis have a permanently slightly bent forwards posture, there is increased load on erector spinae muscle fibres that can lead, over time, to trigger point formation.

Multifidus

Similarly, the multifidus muscle (which has a deeper and a more superficial arrangement), gluteus maximus, and gluteus medius also

demonstrate increased loading and can all develop trigger points for the same reasons.

Piriformis

The piriformis takes its origin from the lower part of the sacrum, but it also often gets involved with the protective spondylolisthesis pattern. When the piriformis gets tight it can squash the sciatic nerve, or even the blood vessels to the nerve (vaso nervorum) which can lead to (pseudo) sciatica.

Glutes and hamstrings

Along with the tight glutes and piriformis, the lower back muscles form a triangle of tight, spastic, and fatigued tissues. Postural changes also cause tension in the hamstring muscles, which also often manifest trigger points and can ache after exercise. Trigger points make their host muscles shorter, fatter, and less efficient. Also, over time and if untreated, trigger points can have a host of unwanted effects such as **peripheral and central sensitization**.

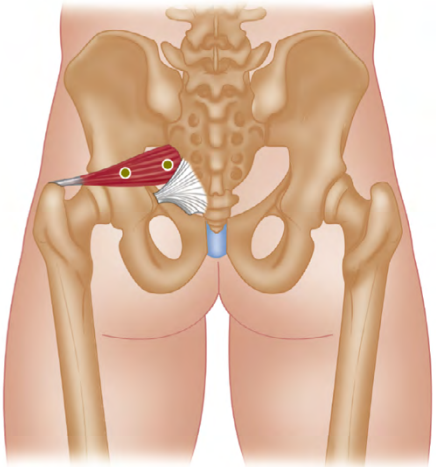
Protect and defend

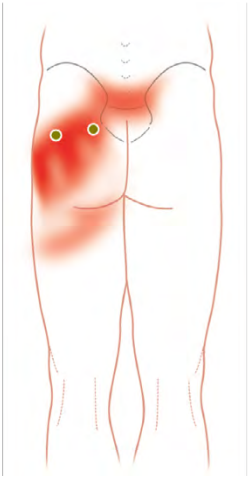
As you can see, the body employs a range of muscles as part of its protect and defend mechanism around the vertebral slippage. Activating these muscles can also lead to reciprocal inhibition effects on other muscles such as the core abdominal muscles, which may weaken.

Which muscles may be affected by spondylolisthesis?

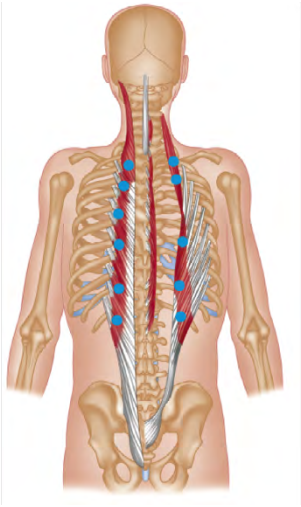
(The image on the right details the trigger point and pain map)

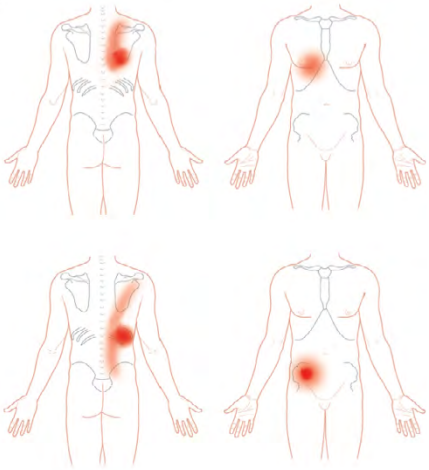
PIRIFORMIS



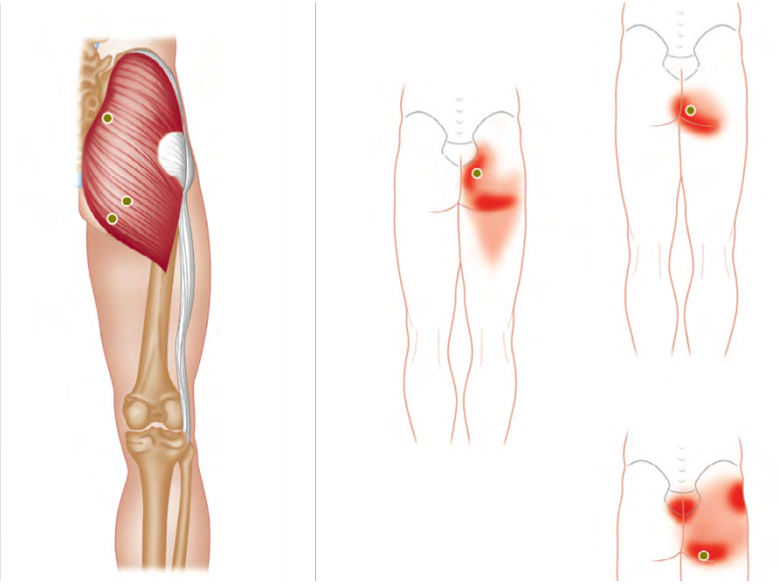


LUMBAR ERECTOR SPINAE

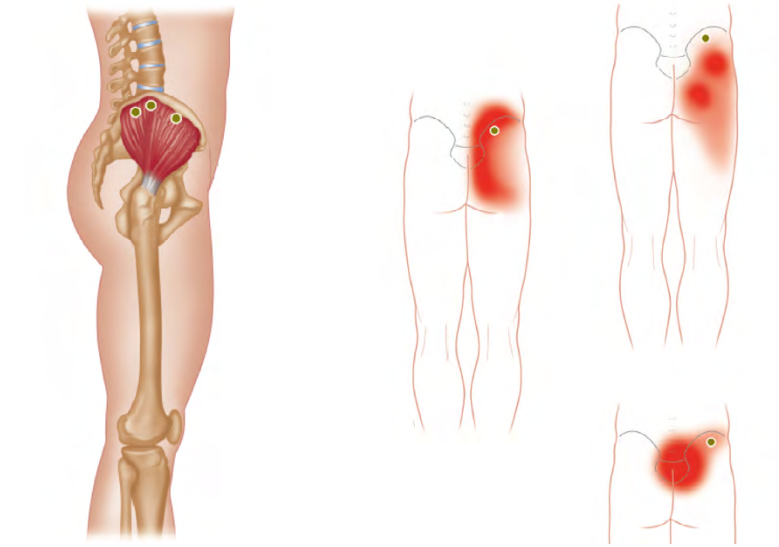




GLUTEUS MAXIMUS

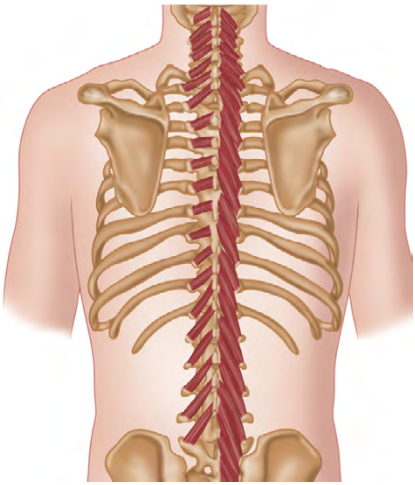


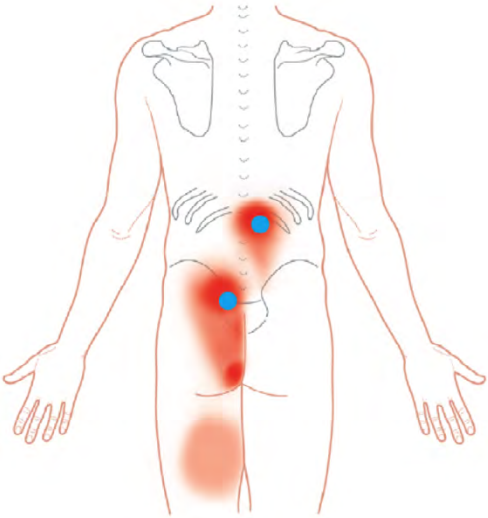
GLUTEUS MEDIUS



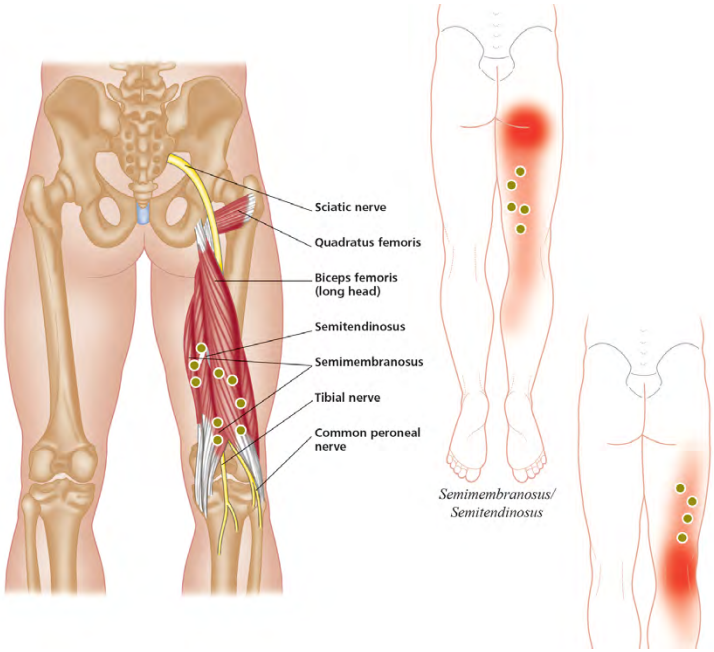
MULTIFIDUS

(The image below details the trigger point and pain map)





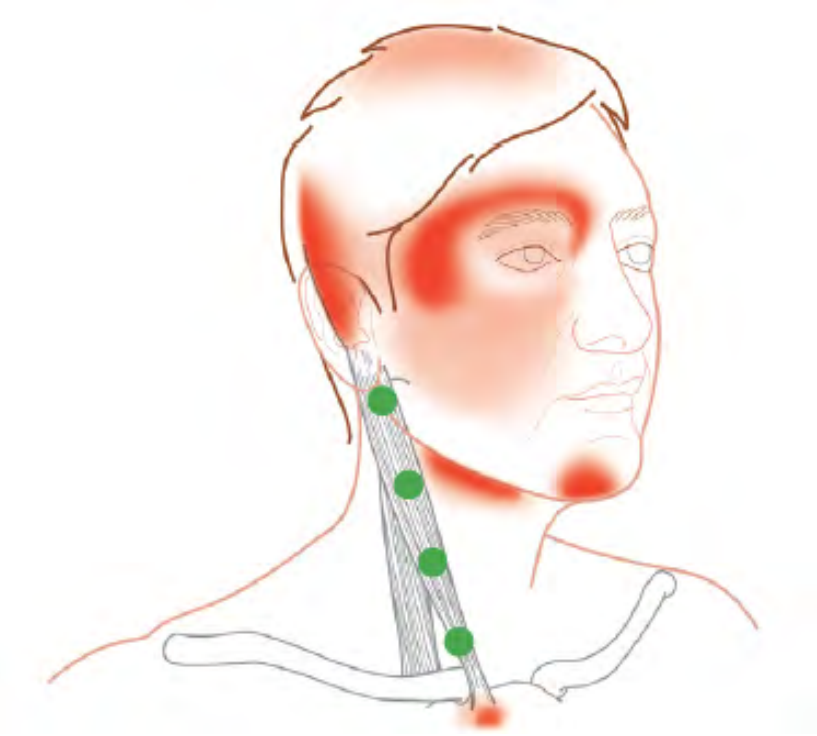
HAMSTRING


Sciatic nerve
Quadratus femoris
Biceps femoris (long head)
Semitendinosus
Semimembranosus
Tibial nerve
Common peroneal nerve
Semimembranosus/ Semitendinosus

TRIGGER POINTS 101

Coined in 1942 by Dr. Janet Travell to describe painful lumps or nodules felt within tight bands of muscle. Trigger points all seem to have the following characteristics:

- Pain, often extreme, is present at a discrete point.
- A nodule is embedded within a taut band in the muscle.
- Pressure reproduces the pain symptoms, with radiations in a specific and reproducible distribution (map).
- Pain cannot be explained by findings from a neurological examination.



- Trigger points develop in the muscle belly; so multipennate (several heads) muscles such as the deltoid or serratus anterior may have several trigger points at once.
- They are the result of overstimulation of the muscle motor end plates, which become sticky and permanently 'switched-on' – this is the lump that we feel.
- They are often embedded in the muscles remotely from where the pain is felt.
- They make the host muscle shorter and fatter and reduce its efficiency: this can lead to pressure on nerves and blood vessels.
- Reduced efficiency = increased risk of injury

How do trigger points develop?

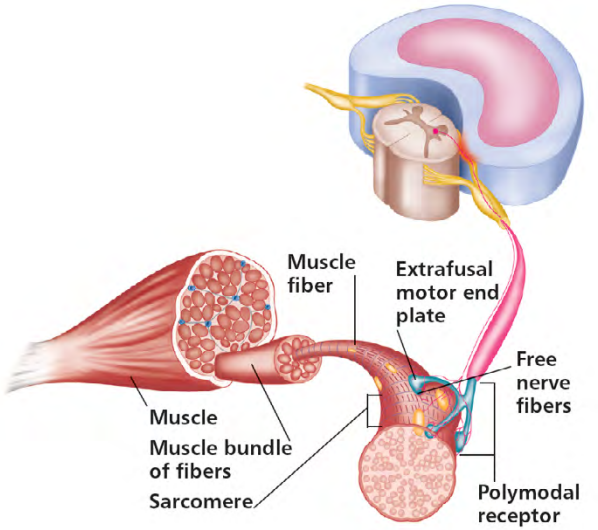
Several possible trigger point mechanisms (Dommerholt 2006)[9]:

- Low-level muscle contractions
- Uneven intramuscular pressure distribution
- Direct trauma
- Unaccustomed eccentric contractions
- Eccentric contractions in unconditioned muscle
- Maximal or submaximal concentric contractions

Pathophysiology of trigger points

The Integrated Trigger Point Hypothesis (ITPH) is the current theory/ working hypothesis: it explains most of the trigger point phenomena, and is based on the best electrodiagnostic and histopathological evidence to date. First introduced by Travell and Simons in 1981 as the 'energy crisis theory' (Simons et al. 1998) [10], the ITPH has been expanded over the years by many others in the field.

Trigger points manifest in the region where sarcomeres and extrafusal motor endplates become overactive. Microscopy has demonstrated that actin and myosin myofilaments (sitting within a taut band) stop sliding over one another and get stuck. Reitinger et al. (1996)[11] reported "pathological alterations" in mitochondria within these myofilaments, as well as an increase in the width of 'A bands' and a decrease in the width of 'I bands'. The affected sarcomere(s) becomes permanently "switched on", leading to a contraction and "wind-up". The swollen, contracted actin and myosin filaments may actually get stuck in the 'Z band' because of the gel-like titin molecules ratcheting the fibers in place and preventing detachment (Dommerholt et al. 2006).



Recent electrophysiological investigations have revealed that the electrical activity of 'active trigger points' arises from dysfunctional extrafusal motor endplate zones rather than from (as previously thought) muscle spindles. Electrical discharge frequencies of 10–1000 times the normal have been demonstrated in the 'endplate zone' in horses, rabbits, and humans (Simons et al. 2002, Dommerholt et al. 2006).

Histological investigation indicates abnormal calcium and ACh levels, and a shortage of ATP in the vicinity of the trigger point. It is worth noting that Grinnel et al. (2003)[12] demonstrated that stretching and/or hypertonicity of muscles causes a pulling of integrin protein peptides at the motor nerve terminal, triggering excessive ACh release without the need for calcium. Other abnormal chemicals present in the milieu of 'active' trigger points include (Shah et al. 2003)[13]:

- Prostaglandins
- Substance P
- Cytokines
- Bradykinin (BK)
- Hydrogen (H+)
- Calcitonin gene-related peptide (CGRP)
- Tumor necrosis factor (TNF- α)
- Interleukins IL-1 beta, IL-6, and IL-8
- Serotonin
- Norepinephrine

These chemicals have many interactions and are part of various feedback loops. For instance, bradykinin is known to activate and sensitize muscle pain fibers (nociceptors). This may help to explain some of the inflammatory hyperalgesia, tenderness, pain, and lowered pain thresholds seen in patients with chronic trigger points.

Vicious cycle of energy crisis

Sustained dysfunction and sarcomere contraction leads to local intracellular and extracellular chemical changes including:

- Localized ischemia/hypoxia
- Increased metabolic needs
- Increased energy (required to sustain contraction)

- Failed reuptake of calcium ions into the sarcoplasmic reticulum
- Localized inflammation (to facilitate repair)
- Compression or watershed effect on local vessels
- Energy crisis
- Production of inflammatory agents (which sensitize local autonomic and nociceptive (pain) fibers)

If this situation is allowed to continue over a significant period of time, the above changes lead to a vicious cycle. Calcium is unable to be taken into the actin and myosin myofilaments, leading to sarcomere 'failure'.

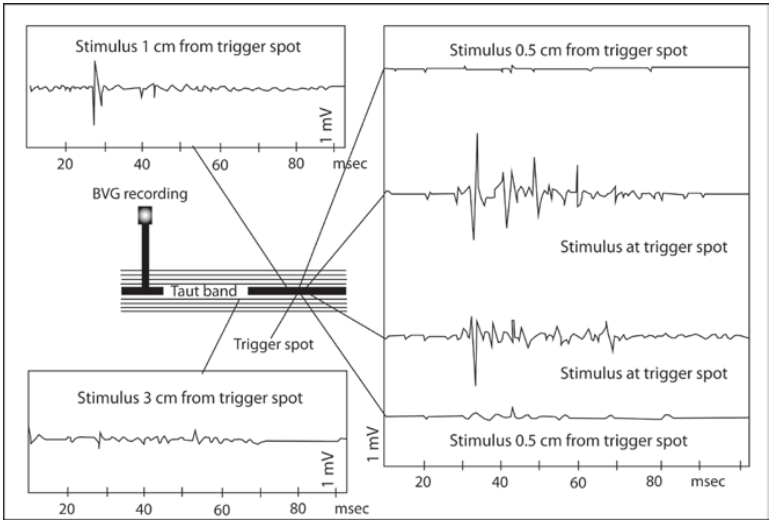
Bengtsson et al. (1986)[14], Hong (1996)[15], and Simons et al. (1998) have all proposed variations of the energy crisis theory. This theory suggests that the body attempts to resolve sarcomere and endplate failure (outlined above) by changing the blood supply to the sarcomere (vasodilation). One further result of this anomalous situation is the migration of localized acute and chronic inflammatory cells. Inflammation is a cascade: this cascade mechanism starts to occur around the dysfunctional sarcomere. Inflammation brings with it sensitizing substances, such as bradykinin and substance P, a peptide present in nerve cells, which not only increases the contractions of gastrointestinal smooth muscle, but also causes vasodilation. This has the effect of stimulating both local (small) pain fibers and local autonomic fibers, which in turn leads to increased ACh production, and hence a vicious cycle.

Eventually, the brain sends a signal to the muscle in which the trigger point manifests to cause it to rest. This leads to hypertonia, weakness, shortening, and fibrosis (muscle stiffness) of the muscle, along with reflex inhibition of other muscle groups. Under microscopy, these fibers have been described as 'ragged red'. Treatment is thus aimed at interfering with and attenuating this vicious cycle. Other theories include radiculopathic theory (Gunn 199 7)[16], (Quintner & Cohen 1994, 2015)[17] and polymodal theory (Kawakita et al. 2002)[18].

Evidence for trigger points

Over the past decade, studies of imaged trigger points[19, 20] have shown through fMRI scanning[21] that trigger point activation results in CNS activation, demonstrated electrophysiological activity at the trigger point[22], and revealed that biochemical changes occur in the trigger point zone[23]. Further studies have shown that manipulation of the trigger point modulates muscle function[24] and induces local and referred pain[25].

Here's a picture of the twitch response, stimulated in a rabbit gastrocnemius muscle. Notice the increase (spike) in electrical activity as the trigger point is stimulated.



Local twitch response (LTR) in a rabbit tender spot. LTRs are elicited only when the needle is placed accurately within the trigger spot. (Adapted from: Hong 1996.)

For more information refer to the book ' Muscle Pain' by Mense[26].

BEYOND THE TRIGGER POINT

Identifying and treating myofascial trigger points can be uniquely effective therapeutically; trigger points, however, rarely develop in isolation and may return if the underlying cause is not identified and addressed. Long-standing trigger points may lead to secondary (and even tertiary) changes in the nervous system (sensitization), and to trigger point formation elsewhere, remote from the original problem. While trigger points may develop as a result of trauma, injury, or overuse, there may be other mechanisms at play.

The fact that trigger points are so ubiquitous in the population as a whole (from babies to the elderly), needs to be explored. So far, models have focused on the 'where' and 'how' but not the 'why'. Our mechanical systems are imbued with self-awareness, self-healing, and self-regulation, so what is the body trying to achieve, and why? We believe it will help us to stand back and think about the 'why' by exploring some other relevant models.

Protection

We are born with a number of protective mechanisms pre-wired into our nervous system. When we touch something hot, we quickly withdraw our hand; when we smell something unpleasant, we turn or move away. As a rule, the body reacts to noxious stimuli by 'switching off' or pulling away from the stressor. Mechanical pain is relayed back to the brain via a number of mechanoreceptors: the brain responds by initiating movements for maximal efficiency. Muscle groups are then arranged hierarchically into functional units of agonist, antagonist, fixator, and synergist.

In spondylolisthesis, 'switch-off' mechanisms work to avoid the noxious stimuli. We are forced to recruit synergists, fixators, and agonists, often in a less efficient manner, to perform our daily tasks. This is fine in the

short term, but over time it can lead to neuroplastic changes in the spinal cord and brain (sensitization). These mechanisms often include reflexes maintained locally in the spinal cord, and centrally in the brain.

Muscular conflict can be palpated around a region of pain as a result of these protective mechanisms. It is worth noting that as humans, we often 'push through' these barriers to carry on with our complex lives.

This 'switching-off' mechanism is universal throughout the body. On a cellular level, the 'switch-off' phenomenon has been observed in a diverse range of diseases and conditions. In cancer, for example, some of the latest ideas center around the fields of the 'immune-neural cortex' and 'immune oncology'. In these fields, the cancer cells have been observed to suppress or 'switch off' our immune surveillance mechanisms by creating an immunosuppressive microenvironment around them: they fool our 'immune checkpoints' and self-tolerance systems. Chronic viral infections, such as hepatitis, have a similar effect on the immune system. The latest HIV research, for example, suggests that the virus acts as a chronic noxious stimulus: this not only fools the immune surveillance mechanisms into 'switching off' but, over time, also makes T-cells both hyperactive and unresponsive (or silent) at the same time. The immune and nervous systems operate as a continuum. In the musculoskeletal system we are able to observe both the 'switching-off' and the hyperactivation in the peripheral (spinal cord) and the somatosensory and motor cortices.

Pain is a big stimulus

With regard to myofascial trigger points, the stressor is acute or chronic pain, either in a joint or in the myofascial matrix. In both cases, the body 'switches off' around the stimulus; this switching is maintained both locally and centrally. The phenomenon is observable in muscles around a fracture site, a slipped disc, or for example, a frozen shoulder. Painful

stimuli are often mediated by inflammation and its noxious exudates, which is part of a well-demonstrated cascade. When our feedback mechanisms are altered, the brain is forced to adapt and compensate. Pain is a highly motivating symptom for the nervous system: it is our alarm bell that something is wrong.

Research into central sensitization has introduced the concept of polymodal receptors. Kawakita et al. (2002) suggested that these "sensitized neural structures" may be proto-trigger points, or "trigger points in situ." In this scenario, the brain switches on "trigger points on demand" where needed as part of the myofascial protective mechanism.

Trigger points on demand—TODs

Ever heard of 'Video on Demand'? Because trigger points make the host muscles weak, they are a useful mechanism for rapidly switching off muscle power around an injury. This is essential if, for example, there is a fracture: it is an important part of our defense, protect, and repair mechanisms. The nervous system uses myofascial trigger points as part of its feedback vocabulary to accomplish this. This may also help to explain the local and rapid neurogenic responses in the muscles to acute injury or fracture.

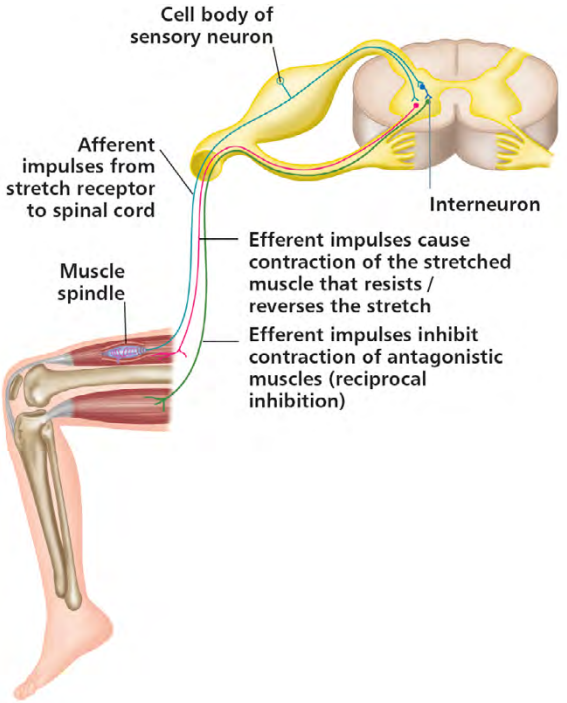
Reciprocal inhibition

Reciprocal inhibition is an important reflex within our nervous system, and has a major role in the control of voluntary movement. It describes the 'automatic' process that occurs when muscles on one side of a joint relax to accommodate contraction on the other side of that joint. Joints are controlled primarily by opposing sets of muscles, extensors, and flexors, which must work in synchrony for smooth movement.

When a muscle spindle is stretched and the stretch reflex is activated, the opposing muscle group must be inhibited to prevent it from working

against the resulting contraction of the homonymous muscle. This inhibition is accomplished by the actions of an inhibitory interneuron in the spinal cord.

The primary (Ia) afferent fiber of the muscle spindle bifurcates in the spinal cord. One branch innervates the alpha motor neuron that causes the homonymous muscle to contract, producing the reflex. The other branch innervates the inhibitory interneuron, which in turn innervates the alpha motor neuron that synapses onto the opposing muscle. Because the interneuron is inhibitory, it prevents the opposing alpha motor neuron from firing, thereby reducing the contraction of the opposing muscle. This is a part of our protective mechanism; without this reciprocal inhibition, both groups of muscles might contract simultaneously and work against each other.



The implications for this are clear: not only do trigger points interfere with host muscle efficiency, but they also have a reciprocal effect on antagonist muscles. This effect is increased with the chronicity of the condition and needs to be recognized and addressed during treatment. This reflex also offers the therapist the opportunity to treat acute myofascial trigger points via their antagonist.

Holding patterns

This phrase is often used in relation to planes as they wait in a queue to land, but it also neatly encapsulates the way we see a patient when they present in the therapeutic setting. Patients may come with acute or chronic symptoms, but whatever the origin, the body's myofascial framework adapts and changes in a protective 'holding pattern'. Over time the 'normal' muscle functioning fails, often resulting in multiple trigger point formation. The longer a problem persists, the more rigid these patterns may become. Chains of sarcomeres fail and chronic recalcitrant trigger points form. Peripheral and central sensitization play a role in maintaining this holding pattern, but so does the adapted myofascial infrastructure.

It is important, therefore, to see trigger points in context:

What is the body trying to achieve? Why has its tolerance/compensation broken down? Where and what is the central or core issue? We encourage you to think like a detective: find the 'tissues that are causing the symptoms' and then reflect and observe how the body has adapted over time to compensate. This requires a holistic view of the patient's body, organs, bones, and supporting tissues, as well as their posture, nutrition, occupation, psychological state, and general wellbeing.

Peripheral and central sensitization

Pain is a complex area of medicine, and current research has thrown up a number of discoveries relevant to trigger point manifestation and perpetuation. Pain systems need to be sensitive enough to detect potentially harmful stimuli. But in the case of trigger points, these systems eventually become too sensitive, causing us pain with no benefit. Hypersensitivity arises because our pain pathways actually increase in sensitivity when they relay pain messages, and with regard to MTPs, the mechanisms of this sensitization are now coming to light.

Peripheral sensitization

Within 48 hours of developing and if untreated, MTPs cause inflammation, chronic facilitation, and changes in feedback from the host muscle. Physiologically, there is a drop in the excitation threshold of polymodal nociceptors (discussed above) so that even normally innocuous, light stimuli activate them. After sensitization of 'pain fibers', stimuli that as a rule are non-painful can cause pain (Schaible 2006)[27]; in addition, mechanosensitive nerve fibers can become mechano-sensitive. "This recruitment of silent nociceptors adds significantly to the nociceptive input to the spinal cord. Resting discharges may be induced or increased in nociceptors" (Schaible 2006). This occurs because of chronic active trigger points providing a continuous afferent barrage into the spinal cord.

The suspected mechanism is:

- Substance P, released from nociceptor terminals, carries nociceptive signals for central processing, and alters local microcirculation and vessel permeability, leading to local edema, activating both mechanoreceptors and nociceptors, with subsequent increased tenderness and pain.

- Persistent activation with these algogenic substances leads to changes in nociceptor responsiveness both peripherally and centrally.

It has been shown that up to 50% of muscle nerves may be made up of nociceptors, and that nociceptors also innervate the connective tissue surrounding muscle. This could account for the severity of pain and exquisite tenderness found in muscles on palpation. Persistent activation of nociceptors leads to peripheral sensitization whereby primary afferent nociceptors exhibit an enhanced responsiveness to natural stimuli.

Central sensitization (spinal hyperexcitability)

In the course of time, the peripheral changes move deeper into the nervous system, and the pattern becomes established centrally. The superficial, the deep, and the ventral spinal cord show pronounced changes in their response properties (Schaible 2006). This is a form of neuroplasticity: after sensitization, an increased percentage of neurons in a segment respond to stimulation of an inflamed tissue. The sensitivity of the spinal cord neurons becomes enhanced, so that an input that was previously subthreshold may now be sufficient to activate the neurons. This effect is magnified up and down the spinal cord over several segmental levels both caudally and cephalically, which may lead to lowered activation thresholds for other MTPs.

The implications of this are profound: it may well be that a chronic trigger point in one area may sensitize levels of the spinal cord above and below the input level. Over time this may lead to a type of neuroplastic change in the CNS. This will decrease the pain threshold in other regions remote from the original source, and possibly lower the threshold potential for other trigger points within the pain map. Central sensitization can persist for weeks, months, and even years, depending on the chronicity of the stimulus.

The suspected mechanism is:

- Repetitive stimulation of primary afferent nociceptors leading to a progressive increase in action potential discharge—a phenomenon called wind-up, which may lead to a 20-fold increase in neuronal sensitivity.
- The result is an increase in intensity of pain and sensitization of neurones in the dorsal horn of the spinal cord because of the activation of N-methyl-D-aspartate (NMDA) receptors — central sensitization.
- Sensory neurones from the dorsal root ganglia become sensitized to mechanical stimuli, so that only mildly painful stimuli become more painful — mechanical hyperalgesia.
- Sustained nociceptive input from active trigger points may not only sensitize dorsal horn neurons, leading to hyperalgesia and allodynia, but also generate expanded referred pain regions.

Potential mechanisms for this phenomenon are the activation of previously redundant synapses at the dorsal horn, and the sprouting of new spinal terminals that broaden synaptic contacts at the dorsal horn, which may explain the referred pain seen with active trigger points.

Both peripheral and central sensitization can have serious unwanted effects; therefore, the advice is to interfere with this process as soon as possible. The good news is that myofascial trigger point release (including dry needling techniques) has been reliably demonstrated to reduce these effects (Mense S, Gerwin RD, Eds. Muscle Pain: Understanding the Mechanisms. Heidelberg: Springer; 2010.).

TREATMENT

TRIGGER POINT TECHNIQUES

TPT includes a range of hands-on pressure-point techniques, two of which that we are going to use are explained here:

- Deep Stroking Massage (DSM) where the sore/trigger point is gently massaged rhythmically to and fro to stimulate inner repair
- Inhibition Compression Technique (ICT) which uses sustained pressure on the sore/trigger point until it releases.

Both DSM and ICT are very safe and effective, but can leave some soreness for a few minutes to hours afterwards. They may very occasionally leave bruising if performed overzealously or if you are on certain medication (especially blood thinners).

How do I know it is a trigger point?

You are looking for:

- Stiffness in the affected muscle
- Spot tenderness (exquisite pain)
- A palpable taut/tight nodule or band
- Presence of referred pain (as indicated on the trigger point map showing you where you should feel pain when pressed)
- Reproduction of your symptoms (accurate)
- The affected area may be moister or warmer (or colder) than the surrounding tissues, and may feel a little like sandpaper

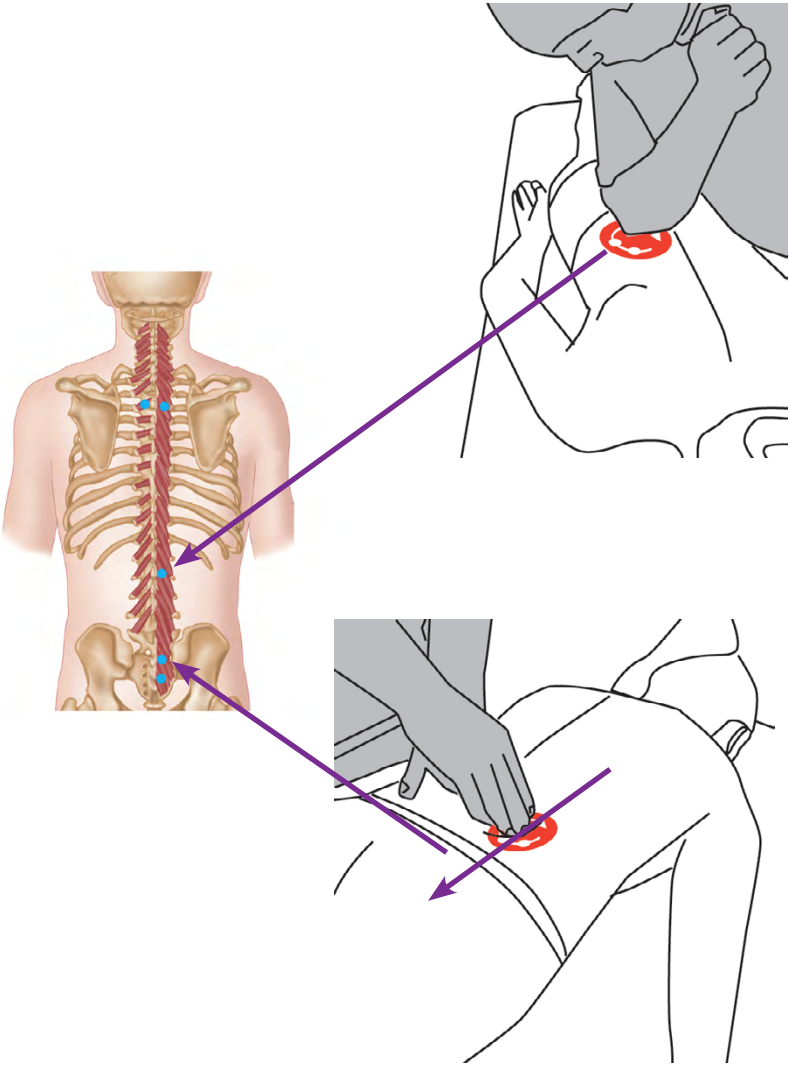
Technique – Deep Stroking Massage (DSM):

1. Identify the trigger point by having a look at the illustrations and then feel for the taut muscle or band
2. Lubricate the skin with oil, cream or lotion
3. Identify and locate the tender/trigger point or taut band
4. Work in one direction only —from the waist level towards the buttocks —don't forget to go right down to the sacrum; perform slow, stroking, rhythmic massage using your thumb/elbow/trigger point tool on the taut band, and reinforce with your other hand. It should feel a bit like squeezing toothpaste from a tube. This should be experienced as discomfort and not as pain. Come away and repeat three times

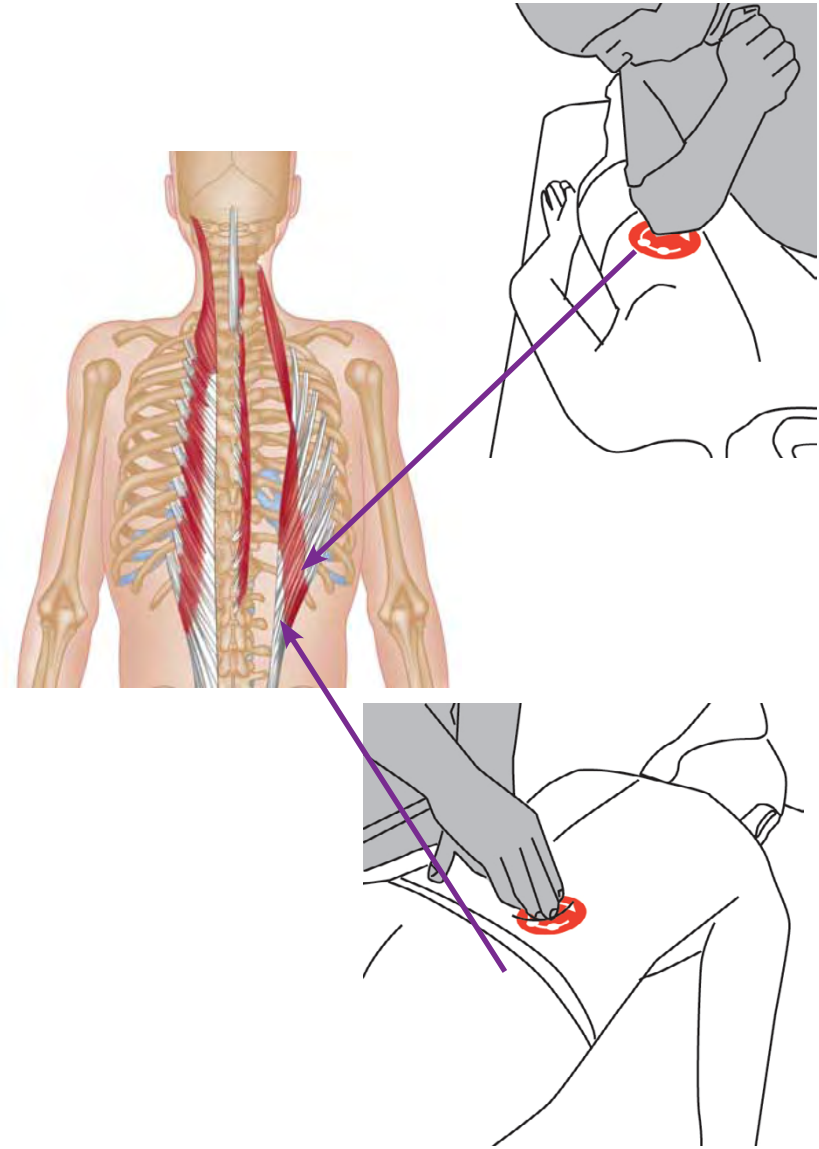
Technique - Inhibition Compression Technique (ICT)

1. Identify the tender/trigger point you wish to work on.
2. Place the host muscle in a comfortable position, where it is relaxed and can undergo full stretch.
3. Apply gentle, gradually increasing pressure to the tender point until you feel resistance. This should be experienced as discomfort and not as pain. You should feel some radiation of the pain.
4. Apply sustained pressure until you feel the tender point yield and soften. This can take from a few seconds to several minutes.
5. Steps 3-4 can be repeated, gradually increasing the pressure on the tender/trigger point until it has fully yielded.
6. To achieve a better result, you can try to change the direction of pressure during these repetitions.
7. At the end of each self help 'treatment', massage the area with some cream, oil or lotion in the direction of the muscle. You can also apply warmth or a heat rub afterwards.

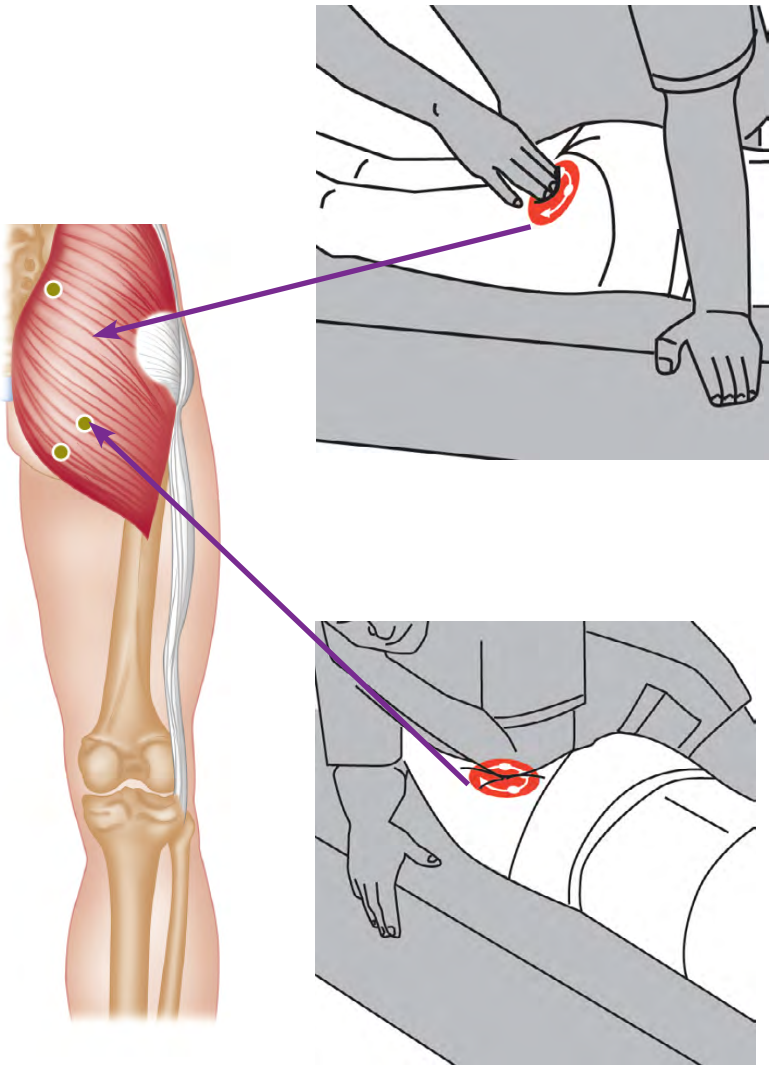
Trigger point therapy – Multifidus ICT & DSM



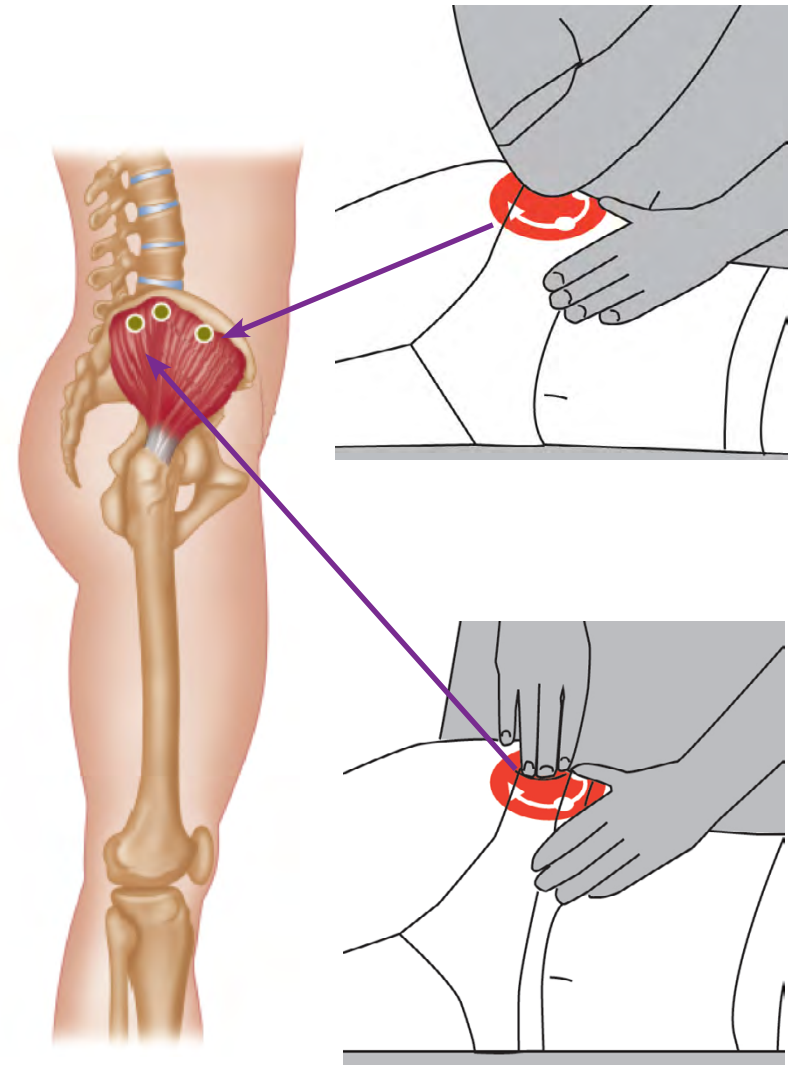
Trigger point therapy – Erector spinae ICT & DSM



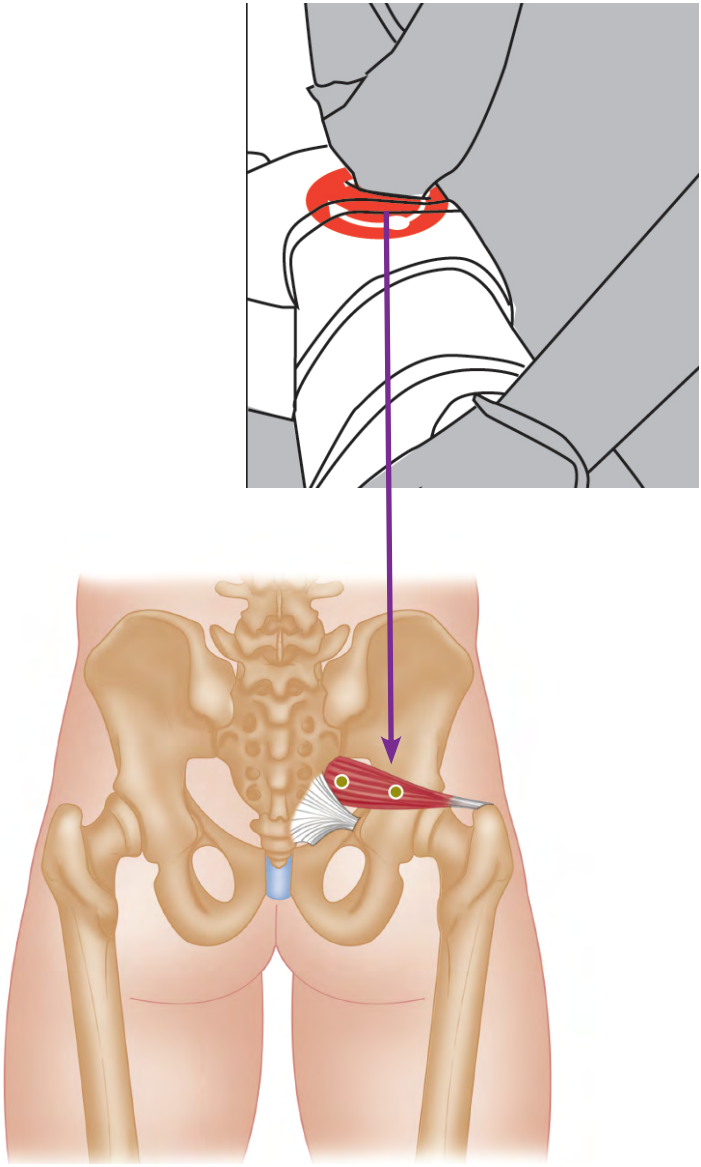
Trigger point therapy – Gluteus maximus ICT & DSM



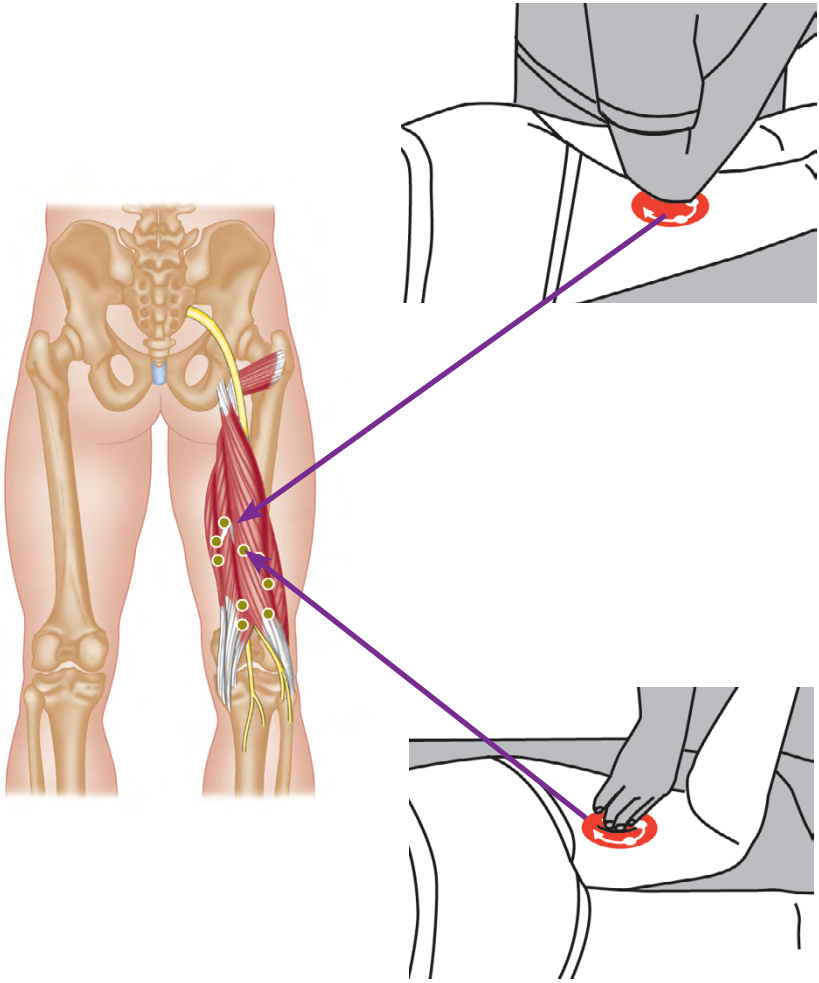
Trigger point therapy – Gluteus medius ICT & DSM



Trigger point therapy – Piriformis ICT & DSM



Trigger point therapy – Hamstring ICT & DSM



NAT – ALGORITHM

UTILIZING THE NEUROPHYSIOLOGY BEHIND MYOFASCIAL TRIGGER POINTS IN A NOVEL AND DELIBERATE WAY

NAT is an advanced trigger point technique — advanced, because it utilizes the neurophysiology behind myofascial trigger points in a novel and deliberate way. The technique uses deep stroking massage and compression/inhibition techniques blended together in a choreographed algorithm. In NAT, trigger points are regarded not as the familiar knots of muscular dysfunction, but as 'inputs' to the spinal cord and central nervous system. This is because trigger points are exquisitely painful, and stimulating the pain pathways has many profound effects on the nervous system, including the attenuation of peripheral and central sensitization.

During most hands-on treatments, a haphazard stream of various mechanoreceptors are stimulated. In NAT, the number of inputs is reduced to an absolute and purposeful minimum. Inputs are made via the trigger points in repeatable sequences, which always include the manipulation of STPs (Super Trigger Points) as well as trigger points in agonists and antagonists. Part of the NAT input sequence is performed three times: repeating something three times (either verbally or somatically) seems to help the nervous system 'get the point'. Stroking massage is performed in 'one direction' only, and compression techniques are performed to the point of pain (and are sometimes held for up to 10 minutes).

NAT was originally developed in 1999 to treat frozen shoulder syndrome (adhesive capsulitis). The shoulder is one of the most complex regions in the body, involving four joints and eighteen muscles. A frozen shoulder is considered by many to manifest the worst components of all other shoulder problems put together. It is in many ways an enigma, and like other enigmas, solving the puzzle helps us gain many truths and insights of the inner workings of the nervous system. We observed that specific

tender/trigger points seemed to be present in the same distribution in every single patient. To our surprise, by manipulating these points in a sequence, we discovered that our patients' long-standing frozen shoulders seemed to melt away in as few as one or two treatments. This rapid 'defrosting' could only be explained by a neurological process which led to the current theoretical model. Many thousands of patients have now been successfully treated with NAT. It is evidence-based, and is now used to treat a wide range of musculoskeletal conditions by over 40,000 therapists worldwide.

NAT theory

Intentionally stimulating mechanoreceptors embedded within and around the trigger points (and joints) generates a novel 'neural signature', which affects the spinal cord and the somatic cortices. NAT deliberately utilizes some of the automatic reflexes associated with trigger points, including:

- Co-ordination
- Reciprocal inhibition
- Post-isometric relaxation
- Post-activation depression
- Pure facilitation
- Co-facilitation
- Autonomic (ANS) responses
- 'Pain gate'
- Spinal cord reflex responses
- Neuroplasticity

The nervous system responds to these input sequences by releasing the 'holding pattern', normalizing motor unit output, and improving co-coordination. Clinically, after each NAT session, patients describe

a sense of joints being 'oiled inside' or feeling that 'normal' muscular control has been regained. With regard to somatic dysfunction, NAT is readily used to reinvigorate and release protective joint postures (such as with spondylolisthesis) and/or treat protective spasm around joint problems (such as an arthritic hip).

Furthermore, NAT sequences seem to tone or reactivate the muscles around the joint; as an automatic response, there is also an increase in strength and power. This is one of the reasons why NAT was successfully used by physical therapists for members of the Canadian and Australian teams in the 2012 London Olympics. Evidence for this phenomenon has been supported by research at Addenbrooke's Hospital in Cambridge, UK (Weis et al. 2003)[28]. Patients with long-standing shoulder pain and weakness treated with NAT demonstrated a significant improvement in active range of motion ($P<0.002$) and in strength and power ($P<0.046$) over and above standard physical therapy and a hands-on placebo, even though no exercises were given to the NAT group.

NAT Algorithm for spondylolisthesis

The sequence and the depth of pressure are key to getting the most effective results. In terms of depth, the deep stroking massage should be experienced like squeezing toothpaste out of a tube. Slow and luxurious stroking in one direction only — this leads to a type of neurological stimulation of the muscles. It is important to visualize the muscles you are working on; see the fibers —how do they feel? What is the fiber direction? You may well note the spondylolisthesis step or anomaly; as you get near the lower lumbar spine, you can pause on it gently and then finish the stroke all the way to the buttock. You can use your hands, fingers, or even gently and respectfully with your elbow.

Rather than simply generally massaging the area, NAT involves a deliberate algorithm of trigger point stimulation. This may be a little different to the way you normally treat, but give it a go; it works. Trigger points are to be thought of as INPUTS to the nervous system rather than just painful knots. We utilize the pain to change the neurology and feedback from the tissues which, in turn, alters and attenuates sensation.

NAT and trigger point therapy has proven successful in the vast majority of patients for treating and managing spondylolisthesis, and we sincerely hope you will find it a valuable addition.

What creams or lotions can I use?

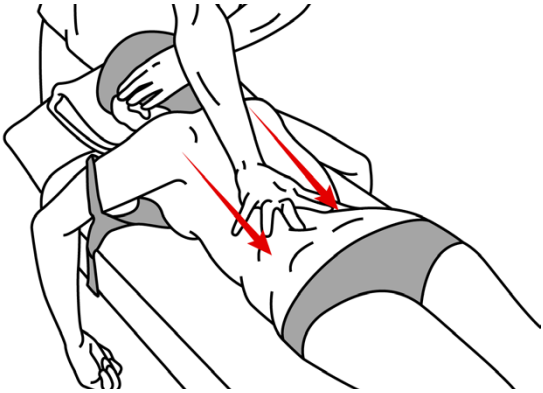
In general, it is better to avoid oils, as they may cause you to slide off from the pressure points once you have found them. We use plain blue Nivea Creme. Alternatively, arnica cream or plain aqueous cream mixed with some vitamin E oil (with a wooden spoon) may be sufficient. Petroleum gel, talcum powder, or massage oil may also be used if you have a lanolin allergy.

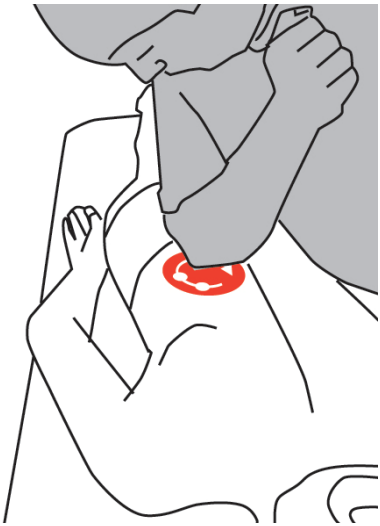
How often should I treat?

Stabilizing and helping the patient come out of the acute phase usually takes 3-5 sessions. These should be performed between 5-10 days apart. After this, we usually recommend maintenance sessions anywhere from 6-12 weeks apart. The exact frequency of these visits will vary from patient to patient, but as a rule – they should come back when they start to feel those telltale symptoms. This also means you will have time to build a relationship with your patient and can advise them on their general health, well-being, and lifestyle factors.

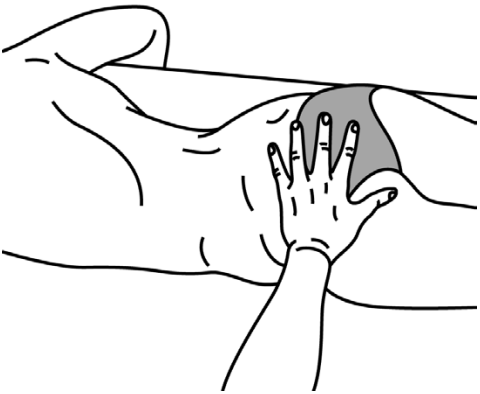
Below we introduce the treatment for spondylolisthesis, followed by recommended stretches and exercises for your patient to do themselves to aid the healing process.

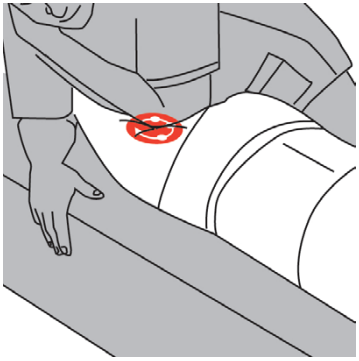
Step 1: one sided (prone ipsilateral) —erector spinae —deep stroking massage. Work 3 times in 1 direction only, working down the spine towards the coccyx. Here we start by working down the center of the back for a few strokes just to get a feel of the tissues. After introducing your hands with slow, rhythmic glide, start focusing on **one side only**.





Step 2: same side (prone – ipsilateral) —gluteus inhibition compression technique. Hold the point until it fatigues, then move around the point to check if there are other gluteal trigger points. Approach the trigger points with respect, building the pressure up slowly, holding it until you feel the tissues yield, and then slowly come away – you should hold the points for up to 4 minutes.

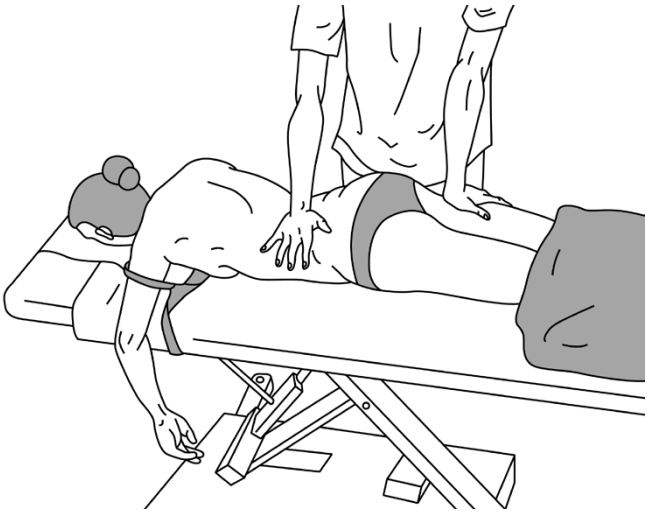




MOVE TO THE OTHER SIDE

SLOWLY REPEAT 3 TIMES

Step 3: (prone) End the session with a 'cool down' move using gentle leaning/compression technique on the opposite lumbar flank and hamstring, balancing your weight between the two of them and then pushing until you feel equal resistance. Come away really slowly, then repeat rhythmically 3-4 times, changing hand positions. As you move up the back, move down the leg. Don't forget to change sides.

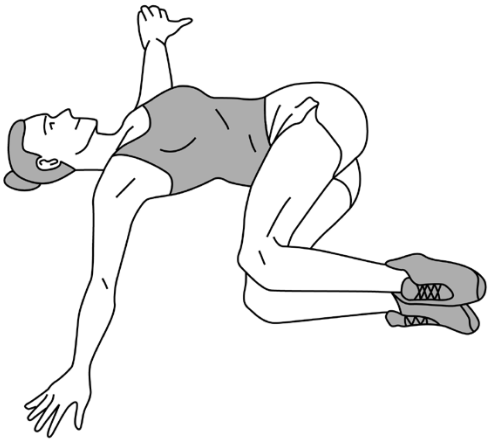


Remember

NAT is about stimulating a neural sequence rather than making sure you have stretched out every single muscle in pain. In the case of spondylolisthesis, it's not a good idea to completely relax all the muscles —they are doing the important job of holding and protecting the spine. We are aiming to use the trigger points as feedback inputs to change the relationship between the brain and the holding pattern around the spondylolisthesis.

STRETCHING

Stretch 1: Two Knee Twist



Adopt position as shown

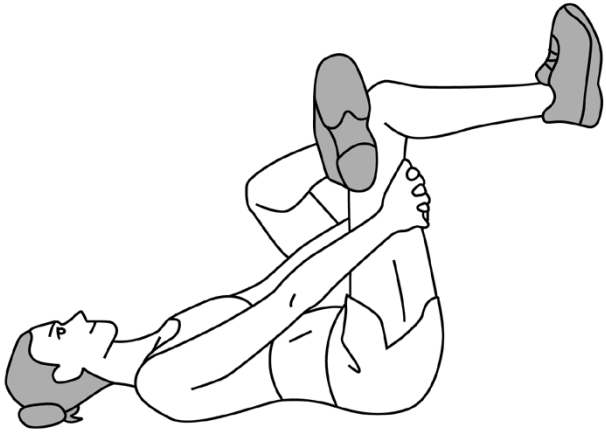
Technique

- Lying on your back, bend your knees into your chest and bring your arms out as a T
- As you exhale, lower your knees to the ground on the right
- Keep both shoulders pressed down firmly
- If the left shoulder lifts, lower your knees further away from the right arm
- Hold for 1-2 minutes each side

How Often?

3 times each side, twice daily

Stretch 2:



Adopt position as shown

Technique

- Lying on your back, bend both knees with your feet flat on the ground
- Bend the right knee like a figure four, with the outer left ankle to the right thigh
- Lift the left foot into the air, bringing the left calf parallel to the ground
- Thread your right hand between the opening of the legs, and interlace your hands behind your left thigh
- Hold 2-3 minutes and then repeat on the other side

How Often?

Twice Daily

Stretch 3:

Adopt position as shown

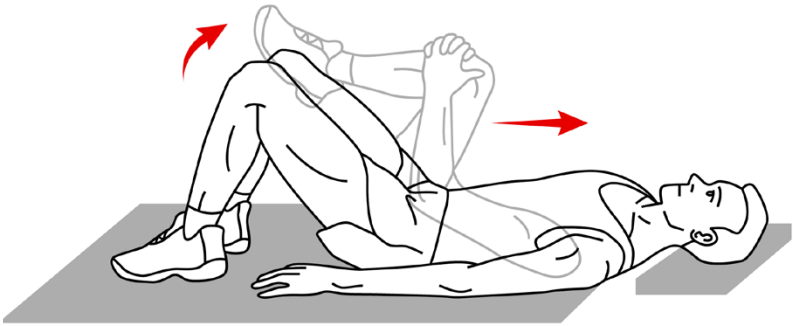
Technique

- As soon as you get up in the morning you should lie prone —on your stomach
- By getting into this position, your lower back curves/arches
- The increased arch pushes on the disk helping to bring the nucleus forward into the correct position

How Often?

Hold for 1 min, twice daily

Stretch 4: Knee to chest stretch



Adopt position as shown

Technique

Start position:

- Lie on your back on a mat or carpet
- Place a small flat cushion or book under your head
- Bend your knees, and keep your feet straight and hip-width part
- Keep your upper body relaxed and your chin gently tucked in

Action:

- Bend one knee up towards your chest and grasp your knee with both hands
- Slowly increase this stretch as comfort allows
- Hold for 20-30 seconds with controlled deep breaths

Tips:

- Do not tense up through the neck, chest, or shoulders
- Only stretch as far as is comfortable

Variation: Grasp both knees and press into chest

How Often?

Repeat 3 times, alternating legs, twice daily

Stretch 5: Sciatic mobilizing stretch

Adopt position as shown

Technique

Start position:

- Lie on your back
- Place a small flat cushion or book under your head
- Bend your knees, and keep your feet straight and hip-width apart
- Keep your upper body relaxed and your chin gently tucked in

Action:

- Bend one knee up towards your chest and grasp your hamstring with both hands below the knee
- Slowly straighten the knee while bringing your foot towards you
- Hold for 20-30 seconds, taking deep breaths
- Bend the knee and return to the starting position

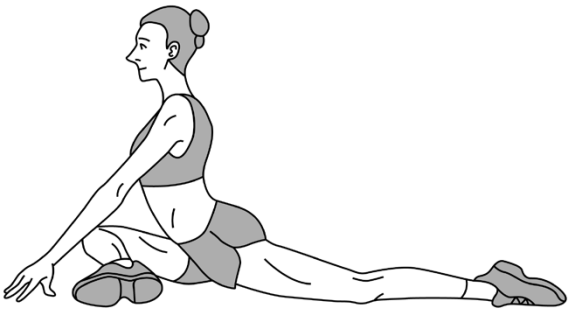
Tips:

- Don’t press your low back down into the floor as you stretch
- Only stretch as far as is comfortable, and stop immediately if you feel any pain, numbness, or tingling

How Often?

Repeat 2 or 3 times, alternating legs, twice daily

Stretch 6: The pigeon (Yoga stretch)



Adopt position as shown

Technique

- Begin in a table top position. Exhale; slide your right knee forward so that the femur (thigh bone) and knee are directly in front of its hip socket. Comfortably align the right heel over towards the left hip without letting the knee slide to the side
- Check the back leg and make sure you are on the top of the knee and thigh, your foot is laying flat, and the leg is in line with its own hip socket
- Inhale and begin to draw your torso upright. Draw your attention to the pelvis, and notice if your ASIS (anterior superior iliac spine 'insert em dash' aka "knobbly points on the front of the pelvis") are dropping forward and your lower back is arched
- Activate your pelvic floor to adjust the pelvis from a largely forward tilted position to a more upright position, this will take the load out of the lower back and also direct the stretch more fully across the front inner groin

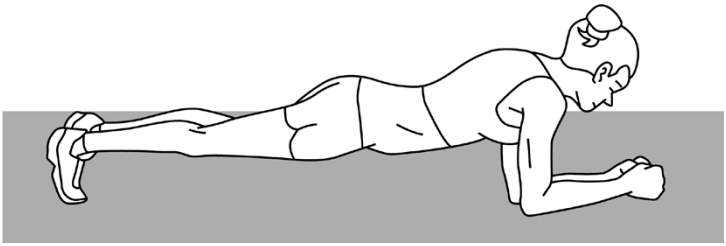
- Remain there for ten slow steady breaths, directing the release into the area of concern. Remember to keep the shoulders relaxed, the abdomen active, and the outside of the back leg's hip moving forward, while at the same time feeling the hip of the front leg moving back

How Often?

30-50 secs per side, twice daily

EXERCISES

Exercise 1: Plank



Adopt position as shown

Technique:

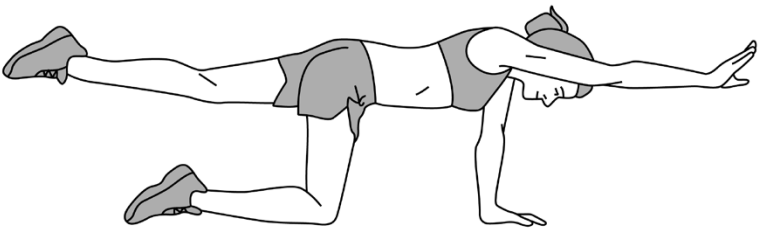
The basic plank exercise, also called a hover, is the starting place if you want to improve your core strength and stability.

- Begin in the plank position with your forearms and toes on the floor
- Keep your torso straight and rigid, and your body in a straight line from ears to toes with no sagging or bending
- Your head is relaxed and you should be looking at the floor
- Hold this position for 10 seconds to start
- Over time, work up to 30, 45 or 60 seconds

How Often?

Repeat 4 times, twice daily

Exercise 2: Birddog



Adopt position as shown

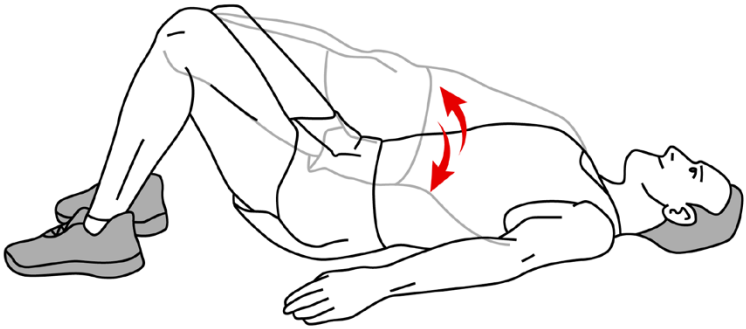
Technique:

- Begin on all fours, hands directly under your shoulders and knees directly under your hips
- Keep head aligned with spine (to help avoid tilting head, look at floor)
- Keep buttocks and abdomen tight. Do not arch the back
- Lift one arm up and forward until it is level with torso; simultaneously lift the opposite leg in the same manner
- Keep arm, spine, and opposite leg aligned as if they are forming a tabletop
- Balance yourself for 10-15 seconds then slowly return to starting position
- Switch sides and repeat
- Remember to breathe

How Often?

Do 10 repetitions each side, twice daily

Exercise 3: Bridge



Adopt position as shown

Technique:

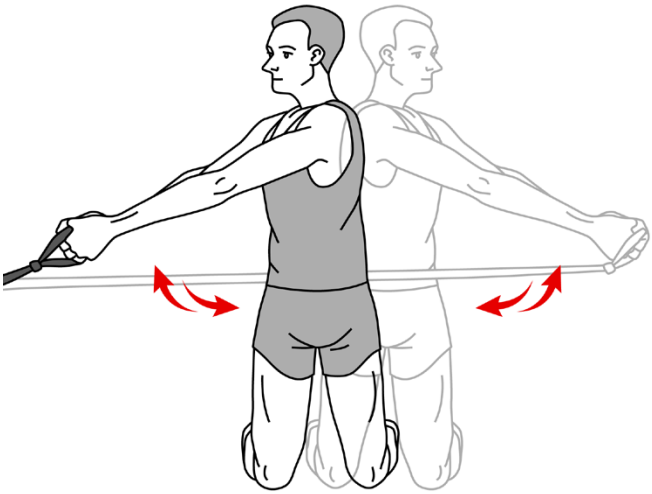
To improve core strength of several muscles in combination, try a bridge

- Lie on your back with your knees bent
- Keep your back in a neutral position, not arched and not pressed into the floor. Avoid tilting your hips
- Tighten your abdominal muscles
- Raise your hips off the floor until your hips are aligned with your knees and shoulders
- Hold for 20 to 30 seconds
- Return to the start position and repeat

How Often?

30 times, twice daily

Exercise 4:



Adopt position as shown

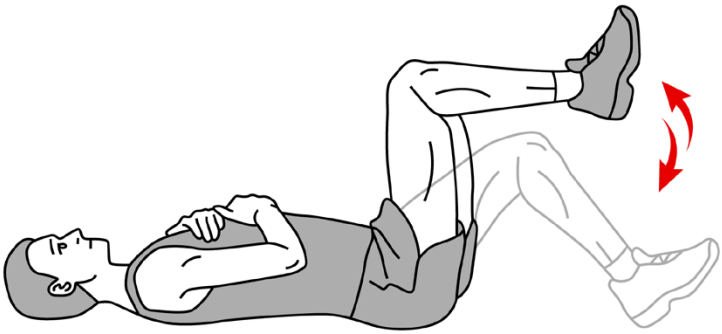
Technique:

- Begin this exercise kneeling or standing with your back straight, holding the resistance band as demonstrated. (Band should be attached to something fixed at waist height)
- Slowly rotate your body, keeping your arms and back straight
- Perform 3 sets of 10 repetitions as far as possible, provided it is pain free

How Often?

Repeat on opposite side, twice daily

Exercise 5: Heel taps



Adopt position as shown

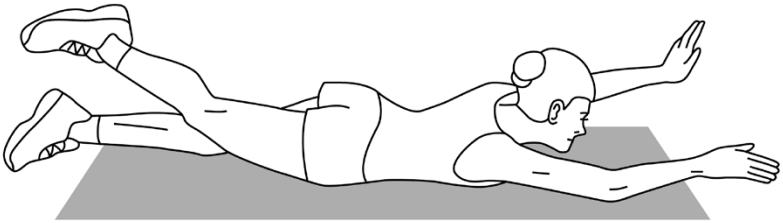
Technique

- Begin this Pilates exercise lying on your back in neutral spine with your hands by your side and your hips and knees bent to 90 degrees as demonstrated
- Maintain activation of your deep stomach muscles and pelvic floor muscles throughout the exercise
- Slowly lower one leg until your heel touches the ground and then return to the starting position
- Keep your spine and pelvis completely still and breathe normally

How Often?

Perform 30 times alternating between legs, twice daily

Exercise 6: Alternating leg and arm raises (Superman)



Adopt position as shown

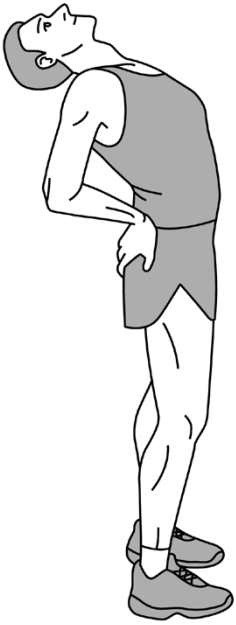
Technique

- Lie on stomach, arms stretched out past your head, with palms and forehead on the floor
- Tighten abdominal muscles
- Lift one arm (as you raise your head and shoulders) and the opposite leg at the same time, stretching them away from each other
- Hold for 5 seconds and then switch sides

How Often?

Repeat 5 - 10 times each side, twice daily

Exercise 9: Standing extension



Adopt position as shown

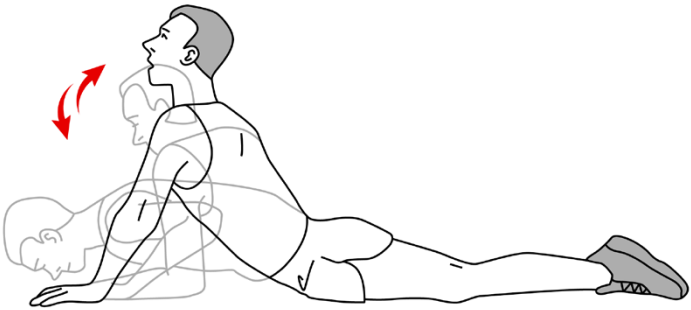
Technique:

- Stand straight, hands behind your hips, with your fingers pointing down
- Push your hands into your pelvis so that your lower back arches
- Don’t use your lower back muscles

How Often?

These exercises can be done 6-8 times, 2-3 times per day

Exercise 10: McKenzie extension



Adopt position as shown

Technique:

Start position:

- Lie on your stomach and prop yourself on your elbows, lengthening your spine
- Keep your shoulders back and neck long

Action:

- Keeping your neck long, arch your back up by pushing down on your hands
- You should feel a gentle stretch in the stomach muscles as you arch backwards
- Breathe and hold for 5 to 10 seconds
- Return to the starting position

How Often?

Repeat 3 times, twice daily

Lifestyle changes to discuss with patient

- Reduce the amount of long period sitting – stand up during the course of the day, walk around, stretch, and then resume working
- Don't rest excessively – keep moving
- Sit on an ergonomically correct chair for office use
- Sit with correct posture
- Place a small pillow in the curve of the lower back – pillows can be purchased at orthopedics stores that are specific to lower back support
- Sleep with a pillow under knees when sleeping on back
- Sleep with a pillow between legs whilst sleeping on side – preventing pelvic rotation
- Weight loss
- Stress management – stress can cause muscle tension, which in turn can cause back pain – this can be done through counseling, yoga, breathing techniques
- Avoid standing for long periods
- Modify your environment – adjust mattress to sleep on a softer mattress, prevent sleeping on stomach, reduce amount of time walking in high heel shoes, use softer chair
- Quit smoking – smoking contributes to the degeneration of spinal disks
- Discontinue with any activities that aggravate symptoms such as bending over, heavy lifting, and any quick twisting or jerking motions. Avoid standing or sitting (e.g. driving) for extended periods as it will increase strain to the spine and aggravate disc pain. At home, keep away from overstuffed and low furniture, because it is difficult to stand back up after sitting in them

Recommended sports

- Swim, swim, swim – an excellent way to look after your back and strengthen your core muscles without putting too much strain on the back
- Pilates and Yoga
- Feldenkrais
- TRX training
- Crosstraining

Not recommended sports/activities

- Avoid mountain biking
- Excessive weight lifting
- Golf
- Running on hard or uneven surfaces
- Tennis
- Skiing

Diet

Studies have demonstrated that underlying health issues – such as folic acid, iron, vitamin, and/or mineral deficiency – may both contribute to and perpetuate trigger point activity.

Other factors such as fatty foods and exposure to free radicals may also have a detrimental effect on our soft tissues. Supplements – for example, omega-3, zinc, magnesium, iron, and vitamins K, B12, and C, as well as folic acid – may help to speed up your recovery.

Physical therapy

Most patients with low-grade isthmic spondylolisthesis and degenerative spondylolisthesis can be treated conservatively. If an isthmic lesion is acute, the patient should be restricted from provocative activities or sports until they are asymptomatic. Physical therapy is an integral part of the patient's rehabilitation process. The most accepted protocol includes activity and exercise that reduces extension stress.

The goals of exercise are to improve abdominal strength and increase flexibility. Since tight hamstrings are almost always part of the clinical picture, appropriate hamstring stretching is important. Instruction in pelvic tilt exercises may help reduce any postural component causing increased lumbar lordosis. Myofascial release plays a role as well in reducing pain from the surrounding soft tissues.

If conservative treatment is indicated for congenital spondylolisthesis, the above principles apply. Adequate work up must be completed for pathologic causes of spondylolisthesis prior to treating with conservative means. Traumatic spondylolisthesis most often requires surgical stabilization.

SURGERY

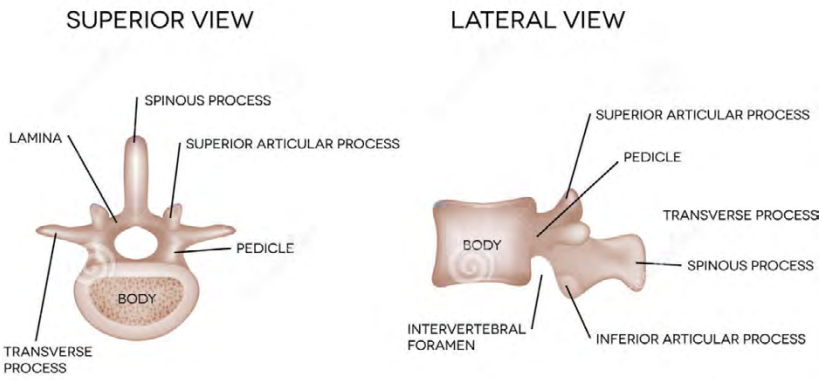
Whilst the majority of patients will respond to conservative management, surgery might be necessary if the vertebra continues to slip, or if the pain is not relieved by conservative treatment and begins to interfere with daily activities. We believe it is important to have a good understanding of the surgical option so we can support, reassure, and inform patients if surgery is needed. It may well be they come back to you after surgery for more therapy.

The main goals of surgery for spondylolisthesis are:

1) to relieve the pain associated with an irritated nerve

2) to stabilize the spine where the vertebra has slipped out of place

3) to increase the person's ability to function



The main surgical techniques used are (Longo 2014)[29]:

1) Laminectomy

When the vertebra slips forward, the nearby nerves that exit the spine can become pinched or irritated. In addition, the size of the spinal canal in the problem area shrinks, placing pressure on the nerves inside the canal. The goal is to remove the lamina and release pressure on the nerves.

i. Traditional open lumbar laminectomy :

- The two laminae and spinous process of a vertebra are removed to relieve excess pressure on the spinal nerves.

ii. METRx Minimally Invasive Hemilaminectomy:

- It involves removing part of one of the two laminae on a vertebra to relieve excess pressure on the spinal nerve(s).

2) Fusion

A spinal fusion is normally done immediately after laminectomy for spondylolisthesis. It is designed to fuse the two vertebrae into one bone and stop the slippage from worsening. The fusion is used to lock the vertebrae in place and stop movement between the vertebrae.

i. Traditional fusion

- The vertebrae are affixed to one another using surgical instrumentation.
- Bone graft is then placed between the vertebrae allowing them to 'fuse' together over time.
- The idea behind this is that the fusion stabilizes the painful joint segment and relieves pressure from the painful spinal nerves.

ii. Minimally invasive surgical spine fusion

- Posterolateral fusion (PLF) is the grandfather of fusion technique as it was developed just over 100 years ago.
- In a posterior approach to lumbar fusion, the surgeon makes an incision down the middle of the lower back.
- One of the criticisms of PLF is that it involves an extensive dissection (the stripping of muscle and fascia off the bone) of the adjacent transverse processes, facet(s), and sometimes lamina.
- After the decompression, the surgeon will place graft material along the sides of the vertebrae to stimulate bone growth.

- Titanium screws and rods are often used to provide immediate stability to the spine until a solid fusion has been achieved.

iii. Posterior Lumbar Interbody Fusion (PLIF):

- In this procedure, the problem vertebrae are fused from the anterior (front) and posterior (back).
- The surgeon works from the back of the spine and removes the disc between the problem vertebrae.
- Bone graft material is inserted from the back of the spine into the space between the two vertebrae where the disc was removed (the *interbody* space)
- Transpedicular instrumentation is attached to stabilize the motion segment while fusion occurs.

Risks of surgery

- Implant failure
- Pseudoarthrosis
- Non-union
- Foot drop
- Spinal compression
- Acute bowel ischaemia

REFERENCES

[1] Amundson G, et al. (1992). Spondylolisthesis. In: RH Rothman & FA Simeone (Eds.), The Spine. (3rd ed., Vol.1, pp. 913-969). Philadelphia: WB Saunders.

[2] Wiltse LL. Spondylolisthesis: Classification and Etiology. In: Symposium on the Spine, American Academy of Orthop Surgeons. St. Louis: CV Mosby, 1969: 143– 66.

[3] Oh JY, Liang S, Louange D, Rahmat R, Hee HT, Kumar VP. "Paradoxical motion in L5-S1 adult spondylolytic spondylolisthesis." Eur Spine J. 2012 Feb;21(2):262-7. Epub 2011 Jun 15.

[4] Froese, BF. (Updated 2018, July 2). Lumbar Spondylolysis and Spondylolisthesis. Retrived from https://emedicine.medscape.com/article/310235-overview

[5] Bono CM. "Low-back pain in athletes." J Bone Joint Surg Am. 2004 Feb;86-A(2):382-96.

[6] Simper LB. "Spondylolysis in Eskimo skeletons." Acta Orthop Scand. 1986 Feb;57(1):78-80.

[7] Wynne-Davies R, Scott JH. "Inheritance and spondylolisthesis: a radiographic family survey." J Bone Joint Surg Br. 1979 Aug;61-B(3):301-5

[8] Kippers V , Parker AW. "Posture related to myoelectric silence of erectores spinae during trunk flexion." Spine. 01 Oct 1984, 9(7):740-745.

[9] Dommerholt J, Bron C, Franssen J. "Myofascial trigger points: an evidence-informed review." J Man Manip Ther. 2006;14(4):206-21.

[10] Travell JG & Simons DG. Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol. 2. Baltimore, MD: Lippincott Williams & Wilkins, 1992.

[11] Reitinger A, Radner H, Tilscher H, Hanna M, Windisch A, Feigl W. "Morphologische Untersuchung and Triggerpunkten." Manuelle Medizin. 1996;34:256-262.

[12] Grinnell AD, Chen BM, Kashani A, Lin J, et al. "The role of integrins in the modulation of neurotransmitter release from motor nerve terminals by stretch and hypertonicity." J Neurocytol. 2003 Jun-Sept;32(5-8):489-503.

[13] Shah JP, Phillips TM, Danoff JV, & Gerber LH. "An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle." J Appl Physiol. (1985). 2005 Nov;99(5):1977-84.

[14] Bengtsson A, Henriksson K, & Larsson J. "Reduced high energy phosphate levels in the painful muscles of patients with primary fibromyalgia." Arthritis Rheum. 1986 Jul;29(7);817-21.

[15] Hong CZ. "Pathophysiology of the myofascial trigger point." J Formos Med Assoc. 1996 Feb;95(2):93-104.

[16] Gunn C. "Radiculopathic pain: diagnosis and treatment of segmental irritation or sensitisation." J Musculoskelet Pain. 1997;5:199-134.

[17] Quintner J, Cohen M. "Referred pain of peripheral nerve origin: an alternative to the 'myofascial pain' construct." Clin J Pain. 1994 Sep;10(3):243-51.

[18] Kawakita K, Itoh K, Okada K. "The polymodal receptor hypothesis of acupuncture and moxibustion, and its rational explanation of acupuncture points." International Congress Series. 2003;1238:63-68.

[19] Chen Q, Bensamoun S, Basford JR, Thompson JM, An KN. "Identification and quantification of myofascial taut bands with magnetic resonance elastography." Arch Phys Med Rehabil. 2007 Dec;88(12):1658-61.

[20] Sikdar S, Shah JP, Gilliams E, et al. “Assessment of myofascial trigger points (MTrPs): a new application of ultrasound imaging and vibration sonoelastography.” Conf Proc IEEE Eng Med Biol Soc. 2008;2008;5585-8.

[21] Niddam DM, Chan RC, Lee SH, et al. “Central modulation of pain evoked from myofascial trigger point.” Clin J Pain. 2007 Jun;23(5):440-8.

[22] Hong CZ, Simons DG. “Pathophysiology and electrophysiologic mechanisms of myofascial trigger points.” Arch Phys Med Rehabil. 1998 Jul;79(7):863-72.

[23] Shah JP, Phillips TM, Danoff JV, et al. “An in vitro microanalytical technique for measuring the local biochemical milieu of human skeletal muscle.” J Appl Physiol (1985). 2005 Nov;99(5):1977-84.

[24] Lucas KR, Rich PA, Polus BI. “Muscle activation patterns in the scapular positioning muscles during loaded scapular plane elevation: the effects of Latent Myofascial Trigger Points.” Clin Biomec. 2010 Oct;25(8):765-70.

[25] 3 references:

a) Wang C, Ge HY, Ibarra JM, et al. “Spatial pain propagation over time following painful glutamate activation of latent myofascial trigger points in humans.” J Pain. 2012 Jun;13(6):537-45.

b) Xu YM, Ge HY, Arendt-Nielsen L. “ Sustained nociceptive mechanical stimulation of latent myofascial trigger point induces central sensitization in healthy subjects.” J Pain. 2010 Dec;11(12):1348-55.

c) Affaitati G, Constantini R, Fabrizio A, Lapenna D, Tafuri E, Giamberardino MA. “Effects of treatment of peripheral pain generators in fibromyalgia patients.” Eur J Pain. 2011 Jan;15(1)61-9.

[26] Mense S, Gerwin RD. Muscle Pain: Understanding the Mechanisms. Heidelberg: Springer, 2010.

[27] Schaible H. “Peripheral and central mechanisms of pain generation.” Handb Exp Pharmacol. 2007;(177):3-28.

[28] Weis JT, Niel-Asher S, Latham M., et al. “A pilot randomised placebo controlled trial of physiotherapy and osteopathic treatment for frozen shoulder.” Rheumatol. 2003;42(1):146.

[29] Longo UG, Loppini M, Romeo G, Maffulli N, Denaro V. “Evidence-based surgical management of spondylolisthesis: reduction or arthrodesis in situ.” J Bone Joint Surg Am. 2014 Jan 1;96(1):53-8.

Testimonials

Michael Coffee, Licensed Massage Therapist, Colorado

NAT is amazing. I've used it, tested it, and approve this technique.

Darlajean M Griffiths, Licensed Massage Therapist, Florida

I am using NAT trigger point therapy protocols in my practice. Amazing results consistently. Not a paid spokesperson but a massage therapist of 26 years. No other treatment comes close to these results.

Anne Szumanski, PT, Philadelphia USA

Your trigger point method continues to prove successful, time and again. What continues to amaze me is the number of problems – that can be addressed and alleviated with the use of NAT. The other noteworthy point for me is the 'cosmic oddity' of so many shoulder ailments appearing in my office. Thank you so much again and again for sharing your talent.

Debbie Smith, Osteopath, United Kingdom

Having used NAT techniques for the past 8 years, I can vouch for their effectiveness. NAT far exceeds the results achieved with standard osteopathic techniques when treating complex shoulder conditions.

Gillian Lonsdale, Osteopath DO, ND, MRN, United Kingdom

I am writing to give you feedback on NAT techniques as I've been using them with our patients over the past two years. In short, NAT has revolutionised our treatment of patients at our clinic, it's so rewarding to have my patients getting better much faster and seeing them freed from painful and debilitating problems as full resolution arrives.

Will Brightman, Chiropractor, United States

So far we are having great success with your trigger point techniques. Thank you for your support.

Niel Asher Healthcare

New York
112 W. 34th Street
18th Floor
New York
NY 10120

London
Belsize Health Clinic
16 England's Lane
London
NW3 4TG

www.nielasher.com

NielAsher.

Advanced Trigger Point Techniques