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PROFESSIONAL HOME STUDY COURSE

TREATING TENSION HEADACHE

TRIGGER POINT THERAPY
MASTER COURSE

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NAT Pro Series:

TREATING HEADACHES

TRIGGER POINT THERAPY

Welcome

According to the World Health Organization[1], an estimated 47% of adults had a headache at least once within last year.

Headaches (cephalalgia) cause a 'world of pain' and disability, and sufferers are highly motivated to seek treatment or intervention; more often than not, they reach straight for the 'medicine bottle', but is that the best course of action? Chronic daily headaches (CDH) are thought to affect 4-5% of adults, and medication overuse is a huge issue for this group. Headaches of mechanical component are by far the most significant; they are well-documented with a substantial evidence base. The two most common types of mechanical headache are Tension Type Headaches (TTH), which affect an estimated 3% of the population, and Cervicogenic headache (CGH), which affects an estimated 0.4% - 2.5% of the population. These types of headache are often associated with stiff and tight neck joints and muscles.

Muscles are designed to move joints from A to B; that is their raison d'être. If a muscle can't excurse fully, it tends to become stiffer, tighter, and develop taut bands; in fact, over time, changes in the fibroblast activity make it become more fibrous – a bit like a ligament. Changes in neck mechanics over time can manifest as muscular knots or 'trigger points'. Trigger points can develop in muscles for a number of reasons. When present, they cause the host muscle to be shorter, tighter, and less efficient. Trigger points can also add to the cycle of increased input to the peripheral and central nervous system. Treating trigger points in neck muscles can have both immediate and long lasting effects for both TTH, CGH, and even migraines; it can also reduce the patient's dependency on medication.

This course is designed to help you understand mechanical headaches in more depth, and to offer you a clear hands-on pathway for 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 that 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

Introduction

Chronic Daily Headache (CDH) has a prevalence of 4-5% of adults. It affects males to females in a 1:2-3 ratio. These headaches often start as episodic but can 'transform' over time to CDH. Medication (rebound) induced headache is believed to play a role in up to 30% of CDH patients[2]. Interestingly, 50-80% of those treated in tertiary headache centers are recorded as having 'medication overuse'. Those with CDH often have a poorer quality of life than 'episodic headachers'. CDH can be either primary or secondary.

Whilst many people reach for the bottle, some realize that medications can mask underlying mechanical problems such as stiff and tense muscles, and look to us for long term help. For these reasons, manual therapy has become 'a popular choice' for patients with common and benign forms of headaches, such as Cervicogenic (CGH) and Tension Type (TTH), because these two conditions are often associated with mechanical neck pain.[3]

Symptoms and causes of headaches

A headache is defined as 'aching or pain in one or more areas of the head or neck'. Both the frequency and pain level can vary greatly. Depending on the classification, between 65-90 percent of all headaches are due to Tension Type Headaches (TTH)[4]. The remaining 10-35 percent are: migraines, neck based/Cervicogenic headaches (CGH), TMJ type, sinus, and cluster headaches. In terms of trigger point therapy, TTH & CGH are the most accessible and amenable to intervention, although some authorities recognize that trigger points may have an important role to play in relieving migraines.

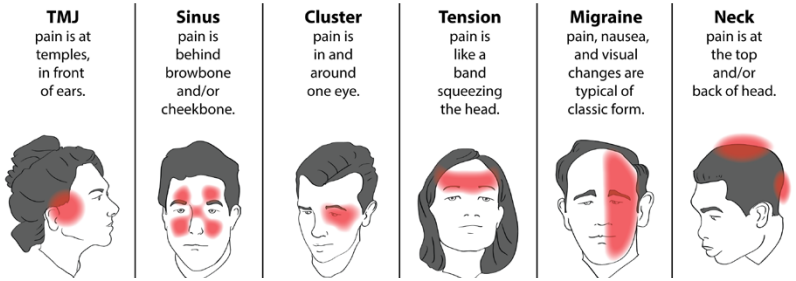
How are they classified?

Headaches are defined as primary or secondary. Primary headaches are benign, recurrent headaches not caused by underlying disease or structural problems. For example, migraine is a type of primary headache. While primary headaches may cause significant daily pain and disability, they are not dangerous. Secondary headaches are caused by an underlying disease, like an infection, head injury, vascular disorders, brain bleed, or tumors. Secondary headaches can be harmless or dangerous. Certain 'red flags' or warning signs indicate a secondary headache may be dangerous.

Here is a list of conditions that are known to cause a secondary headache:

- Sinus headache
- Giant cell arteritis (associated with polymyalgia rheumatica)
- Carotid artery dissection (in the neck)
- Vasculitis
- Headache associated with nonvascular intracranial disorders
- Neoplasm (tumors)
- Idiopathic intracranial hypertension
- Infection
- Post-traumatic headache
- Subdural hematoma
- Cervical spinal disorders (CGH)
- Temporomandibular joint (TMJ) dysfunction
- Headache caused by sleep disorders, such as obstructive sleep apnea.

As you can see, a headache can be a sign that something very wrong is happening inside, so we must always approach them with caution. Fortunately, most headaches are benign. Here is a table with characteristics of the most common types of headaches that should help deepen our understanding.



The International Headache Classification III (ICDH)

We will be following the standardized International Headache Classification III (ICDH) – a full classification can be found here. This classification is hierarchical, and you must decide how detailed you want to make your diagnosis. A diagnosis should be based around the main headache that clients present with (over the last year). When a patient receives more than one diagnosis, these should be listed in the order of importance to the patient. To receive a particular headache diagnosis, the patient must, in many cases, experience a minimum number of attacks of (or days with) that headache. When a patient is suspected of having more than one headache type or subtype, it is highly recommended that he or she fill out a 'headache diary' to record each headache episode.

Tension Type Headaches (TTH)

TTH are by far the most common type of chronic headache. People who experience migraines typically also have tension headaches in between their migraines; these are also known as transformed headaches.

Prevalence of TTH and CGH

	Cervicogenic headache	Tension-type headache
General population (%)	0.4 – 2.5 %	3 %
Headache clinics (%)	15 – 20 %	40%
Mean age	42.9 y/o (all ages are affected)	Onset any age, but most commonly during adolescence or young adulthood
Gender	4 x more prevalent in female (79.1 % ♀ and 20.9 % ♂)	88 % female and 69 % male
Other	CGH is a common symptom after neck trauma; 54 % –66 % of patients with whiplash-associated disorder	Chronic TTH commonly occur during periods of stress and emotional upset.
Intensity	Moderate to severe	Mild to moderate

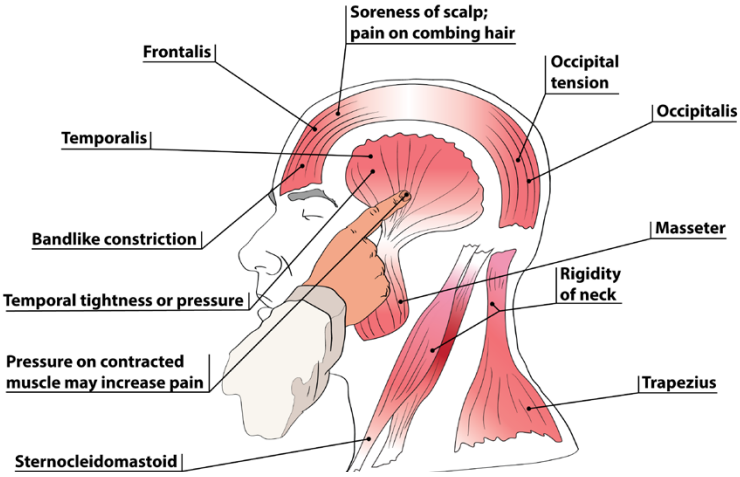
Symptoms of TTH

Tension headaches usually affect both sides of the head and last from thirty minutes to several days or more. They often have a characteristic tight-band or vise-like pain with a 'dull, steady aching' quality. Symptoms can vary in intensity from mild to moderate to severe; they may also affect sleep. They are not accompanied by the additional symptoms that traditionally distinguish migraine headaches such as light sensitivity (photophobia), flashes and patterns in the eyes (visual scotoma), and warning signs (prodromal). Tension headaches affect about 1.4 billion people (20.8% of the population), and are more common in women than men (23% to 18% respectively)[5].

TTH and trigger points

Muscular problems and tension are commonly associated with TTH, and trigger points within muscles may either be causative or may perpetuate TTH. The most commonly affected muscles are: trapezius, sternocleidomastoid, temporalis, masseter, and occipitofrontalis. There is also a strong association with postural issues such as the upper crossed pattern. The pain processing part of the central nervous system is almost certainly involved in TTH, as it shows up abnormal in scans. Trigger points often add to the misery of headaches because they are associated with peripheral and central sensitization (see later). Long-term inputs from trigger points may lead to a vicious cycle that converts periodic headaches into chronic tension headaches. In such cases, even if the original initiating factor is eliminated, the trigger point-central sensitization cycle can perpetuate or even worsen.

TTH are often aggravated by stress, anxiety, depression, fatigue, noise, and glare, but they can also be associated with neck arthritis or neck disc problem.



Seven major causes of TTH

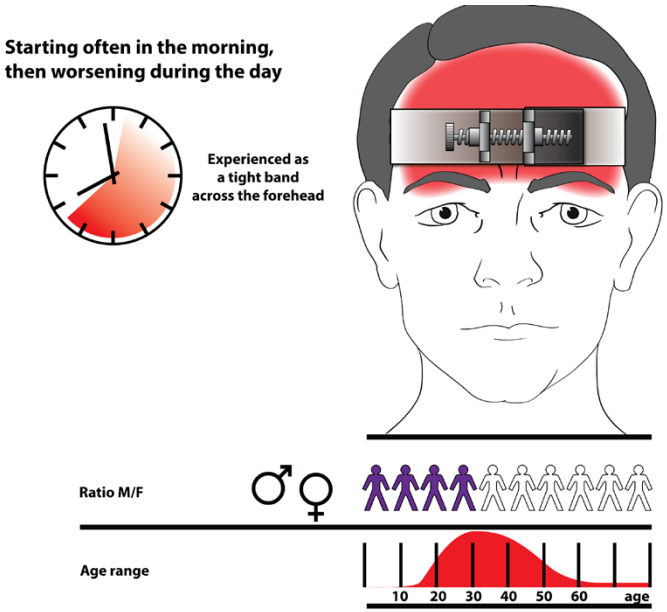
- Stress: usually occurs in the afternoon after long stressful work hours or after an exam
- Sleep deprivation
- Uncomfortable stressful position and/or bad posture
- Irregular meal time (hunger is reported in up to 50% of people)
- Eyestrain
- Tooth clenching (bruxism)
- Postural issues

Acute or Chronic

TTH headaches can be episodic or chronic. Episodic tension-type headaches are defined as tension-type headaches occurring fewer than 15 days a month, whereas chronic tension headaches occur 15 days or more a month for at least 6 months. Headaches can last from minutes to days, months, or even years, though a typical tension headache lasts 4–6 hours.

TTH fact file - (Vernon H, 2009)[6]

1. The most prevalent form of benign primary headache with a reported prevalence varying from 65-90%, depending on the classification, description, and severity of headache features.
2. The psychosocial impacts of TTH include disruptions of daily activities, quality of life & work, and are accompanied by considerable costs.
3. The International Headache Society (IHS) characterizes TTH as bilateral headaches of mild-to-moderate intensity that are experienced with an aching, tightening, or pressing quality of pain.
4. Headaches may last from 30 minutes to 7 days, are not accompanied by nausea or vomiting, and may have light sensitivity (photophobia) or sound sensitivity (phonophobia) but not both.
5. Headache frequency is classified as 'episodic' (<15 headaches per month) or 'chronic' (>15 per month).



6. Episodic TTH is by far the more prevalent category.
7. The chronic TTH patient has a higher frequency of both active and latent triggers points in the suboccipital mm.
8. The chronic TTH patient with active trigger points may have a greater headache intensity and frequency and forward head posture than those with latent trigger points.

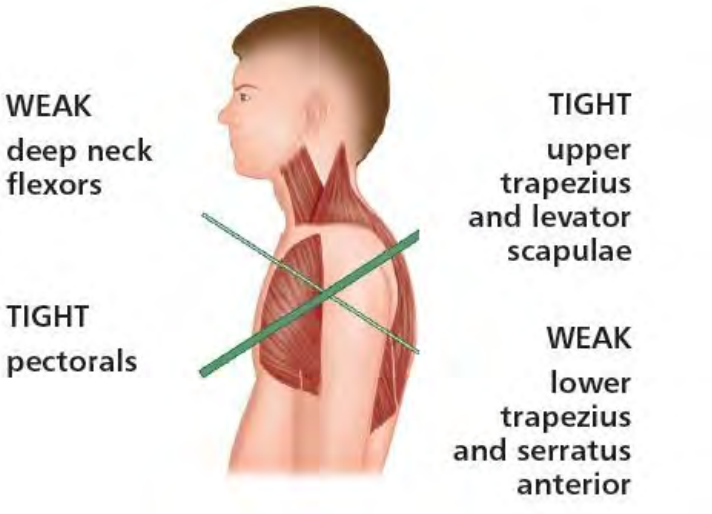
Posture and headaches

There are specific activities and maladaptive postures that serve as trigger point activators, either promoting 'new' trigger points to develop or causing latent ones to become active. These may cause a previously asymptomatic neck muscle to develop pain, especially the **sternocleidomastoid** (see later). Don't forget that the head is heavy, and any alteration in head or jaw posture will have an impact on the biomechanics. Furthermore, because the body is always trying to keep the eyes and ears level for balance, poor posture in any part of the body can lead to neck muscle compensation. The most common mechanical maladaptations are:

- Head-forward posture (upper crossed pattern: Janda)
- Round shoulders (upper crossed pattern: Janda)
- Head to one side—telephone posture
- Occupational/ergonomic stressors
- Slouched standing (lower crossed pattern: Janda)
- Slouched sitting (e.g. computer screen/ergonomics)
- Cross-legged sitting
- Habitual postures
- 'Sway-back' posture (lower crossed pattern: Janda)
- Driving position
- Scoliosis
- Joint hypermobility
- Lifting/carrying
- Primary short lower extremity (PSLE)

Two other key factors in neck pain are the mobility of the thoracic and cervical spine (Lewis 2014)[7]; a stiff thoracic spine may be compensated for by a hypermobile neck (cervical spine). Janda (1996)[8, 9, 10] identified 'the upper crossed pattern' with its tight and short anterior chest muscles with weak and overstretched posterior shoulder muscles.

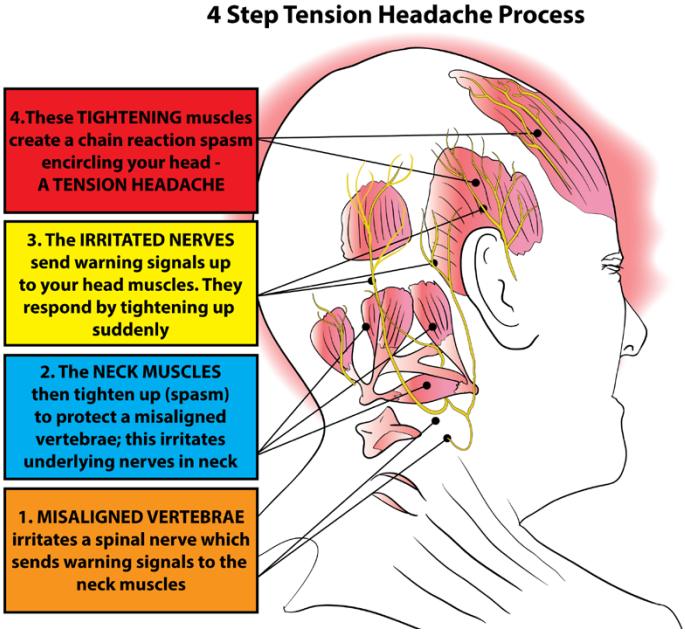
Janda's upper crossed pattern (1996)



Cervicogenic Headache (CGH) - (Antonaci et al., 2006)[11]

First proposed by Sjaastad et al. (1983), CGH is defined as 'referred pain perceived in any region of the head caused by a primary (nociceptive) source in the musculoskeletal tissues innervated by the cervical nerves'.[12] The source of the pain can be from any of the structures of the cervical spine[13]. These structures are innervated by the C1-C3 spinal nerves and include the[14]:

- Upper cervical synovial joints, ligaments
- Muscles of the subcranial spine (specifically **splenius cervicis**, **splenius and semispinalis capitis**, **obliquus capitis**, **multifidus**)
- Discs (C2-C3)
- Pain-sensitive dura mater (the covering of the spinal cord)



Typically, unilateral (hemicranial) CGH is characteristically provoked by neck movements, awkward head positions, and/or pressure on tender points in the neck. CGH can last from hours to days. The pain is often described either as 'dull' or 'piercing'. The underlying mechanisms, signs, symptoms, and treatment are still of debate. The most commonly accepted neurophysiological explanation is that it is due to the convergence of the upper cervical roots on the nucleus caudalis of the trigeminal tract. Most cases of CGH are caused by pathology in the upper cervical spine. Trigger points are part of the body's protect and defend mechanism (see later), and they may become permanently switched on if the underlying causes are not addressed. Anesthetic blocks can be used to confirm the diagnosis and determine the source of pain in the neck. Clearly, as in TTH, posture also has an important role to play.

Trigger points and CGH

There are several muscle trigger points worth exploring in CGH, and trigger point intervention can be hugely beneficial when combined with stretching, exercise, and self help. There is also some evidence supporting spinal manipulation and/or articulation/mobilization as part of manual therapy. The key muscles are **upper trapezius, splenius cervicis, splenius capitis, semispinalis capitis, obliquus capitis, multifidus and temporalis.**

CGH fact file - (J Am Osteopath Assoc., 2000)

1. Unilateral head or face pain without side-shift; the pain may occasionally be bilateral
2. Pain localized to the occipital, frontal, temporal, or orbital regions
3. Moderate to severe pain intensity
4. Intermittent attacks of pain lasting hours to days, constant pain, or constant pain with superimposed attacks of pain

5. Pain is generally deep and non-throbbing; throbbing may occur when migraine attacks are superimposed (transformational)
6. Restricted active and passive neck range of motion; neck stiffness
7. Head pain is triggered by neck movement, sustained or awkward neck postures; digital pressure to the suboccipital, C2, C3, or C4 regions or over the greater occipital nerve; Valsalva, cough, or sneeze might also trigger pain
8. Associated signs and symptoms can be similar to typical migraine accompaniments including:
    — nausea, vomiting
    — photophobia, phonophobia, dizziness
    — others include: ipsilateral blurred vision, lacrimation and conjunctival injection, or ipsilateral neck, shoulder, or arm pain

Migraine Headaches

Migraine is a neurological disease characterized by recurrent moderate to severe headaches. A migraine headache is described as a throbbing, pounding, or pulsating pain; it is often associated with specific and characteristic autonomic nervous system (ANS) symptoms. Typically, headache affects half of the head (hemicranial) but may occur on both sides or oscillate from side to side. Migraines last from 2 to 72 hours. The pain can be made worse by movement, coughing, straining, or flexing the head.

Associated symptoms may include nausea, vomiting, and sensitivity to light, sound, or smell. The pain is generally made worse by physical activity[15]. Up to one-third of people with migraine headaches perceive an aura: a transient visual, sensory, language, or motor disturbance that signals that the headache will soon occur (aura)[16]. A migraine without an aura (common migraine) may be preceded by mental fuzziness, mood changes, fatigue, and an unusual retention of fluids. Occasionally, an aura can occur with little or no headache following it.

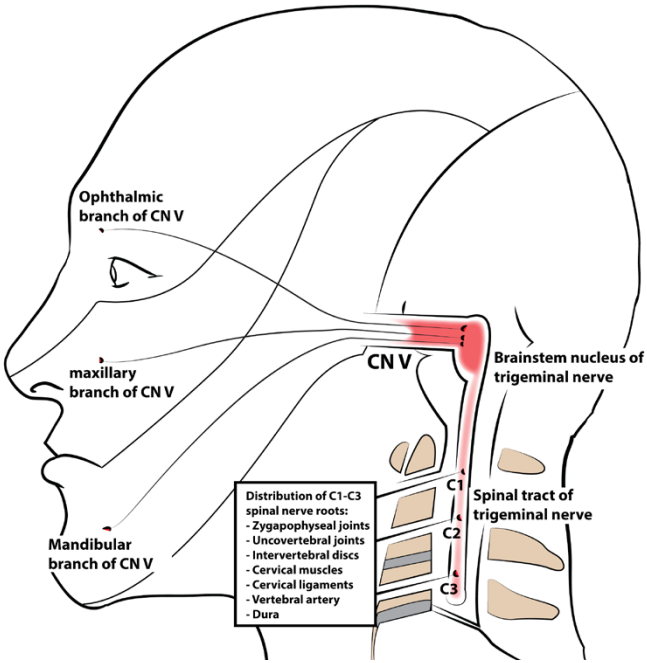
Causes of migraines

According to the 'migraine trust', the exact cause of migraine is not fully understood. Migraine has long been observed to run in families (about two-thirds of cases)[17] so it is thought that there is a genetic component in migraine. Recent research has identified genes for rare types of migraine. People who get migraines may have abnormal genes that control the functions of certain brain cells. Current research is focused around the notion that people who have migraines have a hypersensitive or 'hyper-excitable' cortex as a result of aberrant neurones in the trigeminal cortex of the brainstem[18]. The implications are that the sensitivity threshold is lower in this group than the normal population. This degree of sensitivity is possibly genetically determined, influencing the threshold for triggering attacks. The trigeminal nerve is long and has a cervical branch that loops all the way down to C3. En route the nerve puts out branches that supply the joints, discs, ligaments, and arteries. It has been suggested that any of the structures in these areas, as well as muscular trigger points in the region, may contribute to this 'input'.

Migraine symptoms are now thought to be due to abnormal changes in levels of substances that are naturally produced in the brain. Until fairly recently, it was a commonly held view that an alteration in chemical substances such as serotonin and other vasostimulatory neurotransmitters affected the blood vessels in the brain (*vascular system input*), causing them to become inflamed and swollen, resulting in migraine headaches. However, changes in blood vessels are now thought to be secondary to more important changes in brain chemistry.

Experts do know that people with migraines react to a variety of factors and events, called triggers. These triggers can vary from person to person and don't always lead to migraine. A combination of triggers — not a single thing or event — is more likely to set off an attack. A person's response to triggers also can vary from migraine to migraine.

Spinal Tract of the Trigeminal (V) Nerve Pathway



Frequently mentioned migraine triggers include:

- Lack of, or too much, sleep
- Skipped meals, getting hungry, or not eating enough
- Bright lights, loud noises, or strong odors
- Hormone changes during the menstrual cycle
- Stress and anxiety or relaxation after stress
- Some weather changes
- Alcohol
- Caffeine (too much or withdrawal)
- Changes of routine and travel

The four stages of migraine

Typically migraines go through four distinct phases:

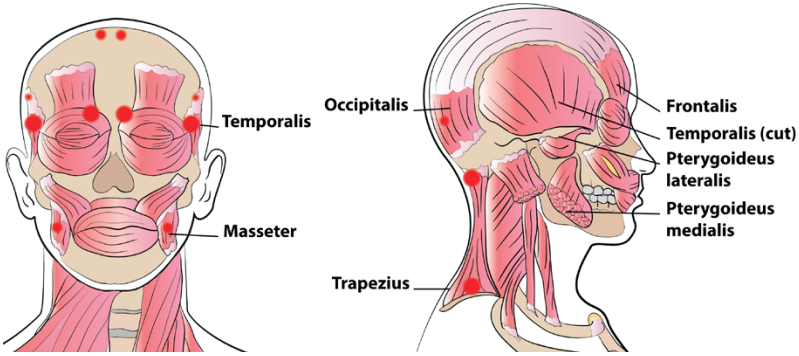
1. The prodrome, which occurs hours or days before the headache (60%).
2. The aura, which immediately precedes the headache: visual disturbance (99%), sensory effects (50%) lasting for about an hour.
3. The pain phase, also known as headache phase. Classically: throbbing with moderate to severe pain and aggravated by physical activity. Frequently associated with nausea and vomiting, photophobia, phonophobia, and sensitivity to smell. Swelling or tenderness of the scalp may occur, as can neck stiffness.
4. The postdrome, the effects experienced following the end of a migraine attack. The effects of migraine may persist for some days after the main headache has ended. The patient may feel tired or 'hung over' and have head pain, cognitive difficulties, gastrointestinal symptoms, mood changes, and weakness.

Trigger points and migraine headaches

A 2006 study by Calandre et al.[19] compared patients at a headache clinic who suffered from frequent migraines, with both non-clinic subjects suffering fewer migraine attacks and healthy control subjects who, at most, had infrequent tension headaches. The data was compelling and indicated that trigger points can lead to both peripheral and central sensitization as well as chronic tissue changes. These changes were directly proportional to the longevity and frequency of the migraine attacks.

They investigated the presence of trigger points and elicited referred pain in 98 migraine patients and in 32 healthy subjects. Trigger points were found in 93.9% of migraines and in 29% of the control group. The number of individual migraine trigger points varied from zero to 14, and was found to be related to both the frequency of migraine attacks and the duration

of the disease. About 74% of the total detected trigger points were found in **temporalis** and/or **suboccipital (obliquus capitis)** areas; other locations, such as orbicularis oculi and occipitofrontalis, were mainly found in patients showing more than four trigger points. Trigger point palpation provoked a migraine attack in 30.6% patients.

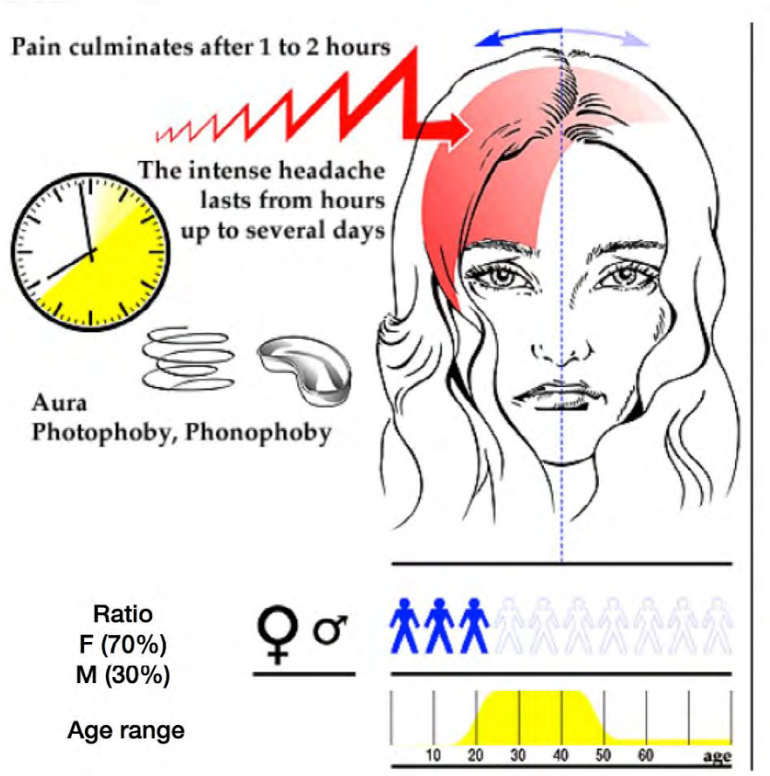


Migraines, TTH, transformed headaches, and trigger points

According to many neurologists, the most common form of headache seen in the clinic is 'chronic daily headache associated with muscle tension' (chronic TTH) **with periodic migrainous** features. This type of headache is often included in the category of migraine or **transformed migraine**, but is characterized by a background of daily headache with tender, tight muscle bands (trigger points) in the head, neck, and shoulder muscles, with pain referred to the head[20]. Trigger points are also thought to be 'input factors', as they make their host muscle shorter and less efficient and also have a role in both peripheral and central sensitization. This type of 'transformed headache' can often be reproduced by stimulating the trigger points that refer pain to the head (mainly the **temporalis, obliquus capitis, sternocleidomastoid** and **upper trapezius**).

Inactivation of the trigger points in these muscles can be an effective treatment in both the chronic and acute states. Furthermore, it has

been suggested that emotional stimuli may trigger the limbic system to increase muscle contractions and precipitate trigger point formation within muscles. It is theorized that the sum of the vascular system input plus the input from trigger points and emotional stimuli determines whether or not pain is a symptom, and if so, how intense the pain is. This could explain how some people can have trigger points or experience emotional duress without having headaches or migraines, while others get severe headaches. People who tend to have migraines and tension headaches that occur at the same time are likely to have a very strong input from emotional factors, or possibly abuse drugs[21].



Other common types of headache (there are many more):

Medication Overuse Headaches (MOH)

People who use acute pain-relief medicine more than two or three times a week or more than 10 days out of the month can set off a cycle called Medication Overuse Headaches (MOH). As each dose of medicine wears off, the pain comes back, leading them to take even more. This overuse causes the medicine to stop helping your pain and actually start causing headaches. MOH can occur with both over-the-counter and prescription pain-relief medicines. They can also occur whether patients take them for headache or for another type of pain. Trigger point intervention may help reduce drug dependency and could be woven into any headache care pathway.

Diagnostic criteria[22]:

- Intake on ≥10 days per month on a regular basis for ≥3 months (>15 days for simple analgesics)
- Headache has developed or markedly worsened during overuse
- Headache resolves or reverts to its previous pattern within 2 months after discontinuation of ergotamine

Applies to:

Ergotamine, triptan, analgesic, opioid, and combination medication-overuse headache

Cluster Headaches (CH)

CH is a neurological disorder characterized by recurrent, severe headaches on one side of the head, typically around the eye[23]. There are often accompanying ANS symptoms during the headache such as eye

watering, nasal congestion, swelling around the eye, and ptosis (drooping eye) typically confined to the side of the head with the pain.

Cluster headache belongs to a group of primary headache disorders, classified as the 'trigeminal autonomic cephalalgias' or (TACs). CH is named after the pattern of headache attacks, which tend to occur together (in clusters). Typically, sufferers experience repeated attacks of excruciatingly severe unilateral headache pain. CH attacks often occur periodically; spontaneous remissions may interrupt active periods of pain, though about 10–15% of chronic CH never remits. The cause of cluster headache has not been identified.

Trigger Points and Cluster Headaches

Note that the triggers and causes of CH are similar to some of the triggers and causes of migraines, and of trigger points. Oxygen deprivation of muscle cells may also play a role in causing cluster headaches; it has been suggested that deprivation also activates or 'switches on' trigger points. The role of trigger points in activating and perpetuating cluster headaches has not yet been studied, however, treating trigger points and eliminating perpetuating factors have been shown to help resolve this type of headache. Typically, the trigger points can be found in the **occipitofrontalis** and **orbicularis oculi** muscles.

Sinus Headache

Sinusitis is a common condition. It affects about between 10% and 30% of people each year in the United States and Europe[24]. Women are more often affected than men. Chronic sinusitis affects approximately 12.5% of people. Treatment of sinusitis in the United States results in more than 11 billion USD in costs. Headache/facial pain or pressure of a dull, constant, or aching sort over the affected sinuses is common with both acute and chronic stages of sinusitis. This pain is typically localized to the involved

sinus and may worsen when the affected person bends over or when lying down. Pain often starts on one side of the head and progresses to both sides. Common trigger points that may help relieve the symptoms can be found in: **zygomaticus, occipitofrontalis,** and **orbicularis oculi** (especially around the orbit).

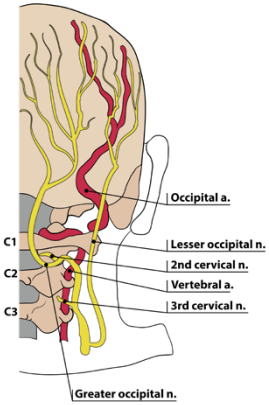
Comparing migraine, TTH, and cluster headaches

Symptom	Migraine	Tension Type	Cluster
Location	Unilateral in 60 to 70 percent; bifrontal or global in 30 percent	Bilateral	Always unilateral, usually begins around the eye or temple
Characteristics	Gradual in onset, crescendo pattern; pulsating; moderate or severe intensity; aggravated by routine physical activity	Pressure or tightness which waxes and wanes	Pain begins quickly, reaches a crescendo within minutes; pain is deep, continuous, excruciating, and explosive in quality
Patient Appearance	Patient prefers to rest in a dark, quiet room	Patient may remain active or may need to rest	Patient remains active
Duration	≥5 attacks lasting 4 to 72 hours	≥10 attacks lasting 30 minutes to 7 days	30 minutes to 3 hours
Associated Symptoms	≥2 of the following 4: • Unilateral • Pulsating • Moderate or severe intensity • Aggravation by routine physical activity ≥1 of the following: • Nausea and / or vomiting • Photophobia and phonophobia Not attributable to another disorder; can involve other senses or cause speech or motor deficits	≥2 of the following : • Bilateral • Not pulsating • Mild or moderate intensity • Not aggravated by routine physical activity • No nausea or vomiting • One or neither photophobia or phonophobia Not attributable to another disorder.	Ipsilateral lacrimation and redness of the eye; stuffy nose; rhinorrhea; pallor; sweating; Horner's syndrome; focal neurologic symptoms rare; sensitivity to alcohol

Greater Occipital Neuralgia (GON)

GON, also known as C2 neuralgia or (rarely) Arnold's neuralgia, is a medical condition characterized by chronic pain in the upper neck, back of the head, and behind the eyes. These areas correspond to the locations of the lesser and greater occipital nerves. The greater occipital nerve also has an artery that supplies blood that is wrapped around it - the occipital artery - that can contribute to the neuralgia. This condition is also sometimes characterized by diminished sensation in the affected area as well.

Occipital neuralgia is characterized by severe pain that begins in the upper neck and back of the head. This pain is typically unilateral, although it can be bilateral if both occipital nerves have been affected. Additionally, the pain may radiate forward toward the eye, as it follows the path of the occipital nerve(s). Sufferers sometimes report blurred vision as the pain radiates near or behind the eye. The pain (neuralgia) is commonly described as sharp, shooting, zapping, an electric shock, or stabbing. Bouts of pain are rarely consistent, but can occur frequently with some patients depending on the damage to the nerves. The amount of time the pain lasts typically varies each time the symptom appears; it may last a few seconds or be almost continuous. Occipital neuralgia can last for hours or for several days.



Symptoms of GON may include:

- Aching, burning, and throbbing pain that typically starts at the base of the head and radiates to the scalp
- Pain on one or both sides of the head
- Pain behind the eye
- Sensitivity to light
- Sensitivity to sound
- Slurred speech
- Pain when moving the neck
- Difficulty with balance and coordination
- Tender scalp
- Nausea and/or vomiting

Trigger Points for GON

Are often found in the **semispinalis capitis, upper trapezius,** and **multifidus**. The most common point to help GON is to place a finger on the spine of C2 and another on the mastoid process and imagine a line. The best trigger points can be found deep somewhere in the middle of this line – near **obliquus capitis** (see below); you may need to use your elbow to find them.

TMJ Disorder (TMJD) Headaches

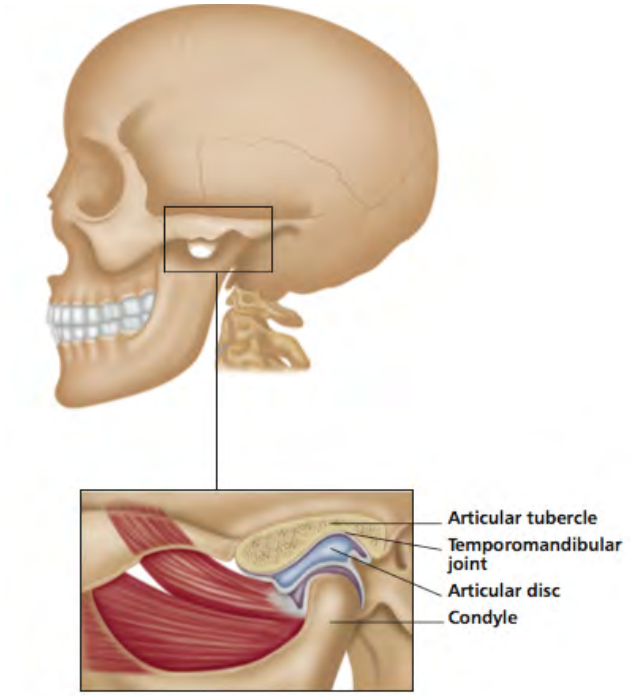
TMJ disorder is sometimes called the 'Great Impostor' because of its multiplicity of symptoms. Pain, stiffness, clicking, clunking or popping sounds, and aching in the jaw muscles characterize this debilitating problem. Classically the headache is strongly felt in the region of the ear.

TMJD is multifactorial, and the following list covers some of the common differential diagnostic criteria:

- 'Under,' 'over,' 'lateral' bite, or malocclusion
- Dislocation on yawning, popping, and/or crepitus
- Ear pain
- Headache
- Cervical spine disorders
- Type/shape of synovial joint; several anatomical variations occur
- Gum chewing
- Masticating food unilaterally
- Chronic dental problems
- Problems with wisdom teeth
- Tooth grinding; bruxism
- Clenching in response to stress/anxiety
- Depression and bipolar disorder
- Arthritis (osteo and rheumatoid)
- Ill-fitting dentures

TMJD often has a serious impact on quality of life and it has been known to affect the whole body if it becomes chronic. TMJD may be primary, as the result of anomalous jaw or bite formation, such as malocclusion or a variation in jaw joint anatomy; or it may be secondary to a variety of conditions, such as tooth clenching or grinding (bruxism). TMJD changes the jaw's mechanics and over time manifests in areas of tight bands primarily in: **masseter**, **temporalis**, **medial** and **lateral pterygoid** muscles. The secondary muscles are the **mylohyoid** and the **anterior digastricus**. Chronic trigger points in any of these muscles may lead to an increase in muscular stiffness, fatigue, and dysfunction. Symptoms may be unilateral and/or bilateral, and are rarely seen in the under-20

age group. Further, satellite trigger points may be located in the **upper trapezius**, **upper semispinalis capitis**, **suboccipitalis**, and **SCM**. Trigger point therapy can have a significant role to play in relieving both the headache and many of the TMJD symptoms.



Examination, History and Testing – 'S.O.A.P.'

SUBJECTIVE	*Onset of Pain* *Palliative, Provocative factors* *Quality of the pain* *Radiation and localisation* *Severity, Stimulating/ Relieving* *Timing*
OBJECTIVE	*Appearance, Asymmetry* *Range of Motion* *Touch, Tissue Texture* *Neurological*
ASSESSMENT	*DDx, Diagnostic Tests* *Discussion*
PLAN	*Diagnostic* *Therapeutic* *drug therapy* *manual therapy* *intervention*

Clinical History

A thorough case history is hugely important for formulating a diagnosis/ hypothesis. Importantly, we need it to help us rule out any more serious pathology. Whilst we need to know about the general wellbeing, this case history and examination should be more headache-focused. We also need to bear in mind that trigger points often cause pain in areas remote from their location.

When did it start? How often does it occur? Can you point to a specific area? Is the neck stiff? Is there aura? Photophobia? Phonophobia? Scotoma? Prodrome? Postdrome? What are the aggravating, relieving, and non-affecting factors?

Is there a past history of similar episodes? Is this episode the same or is it different? Is this episode worse? Is there a family history? Has the patient ever had investigations or been given a diagnosis?

Is the patient a smoker? Are there social, family, or psychological factors that prolong or amplify symptoms? How is their general health? How is their level of fitness?

You should run through a body system checklist and ask about medication to make sure there are no **red flags**. Once you have a good history, you should move on to examination and/or clinical testing.

Examination

A thorough neck movement evaluation should be performed. Get the patient to stand and then sit – start to note down any anomalies. How is the posture? Is there a head forward (upper crossed pattern) or slouch (lower crossed pattern)? Is there reddening of the skin, any trophic changes (might indicate an underlying pathology)? How is the thoracic spine mobility? Remember that problems elsewhere in the body will have an impact on proper neck functioning.

Movement

It is really important to watch the patient moving; there are so many subtle clues that give away information as to the tissues causing symptoms. Have a look at the thoracic spine; is it moving as individual segments or in blocks? Is the patient avoiding problems elsewhere in the body?

The Neck

Check neck mobility thoroughly in flexion, extension, rotation, and side-bending; is it stuck? Is it held to one side?

If you see that the occiput and **C1/2** are hyperextended with an increased lordosis/craniocervical angle – that might be a clue indicating CGH.

Remember that forward head posture and TTH (upper crossed) - tends towards tension-type headache. Faulty head postures can result in shortening of the posterior cervical extensor muscles (**suboccipitals, semispinalis**, **splenii**, and **upper trapezius**) and active trigger points. In chronic TTH, make sure you examine the **SCM**, **suboccipitals,** and **scaleneii**.

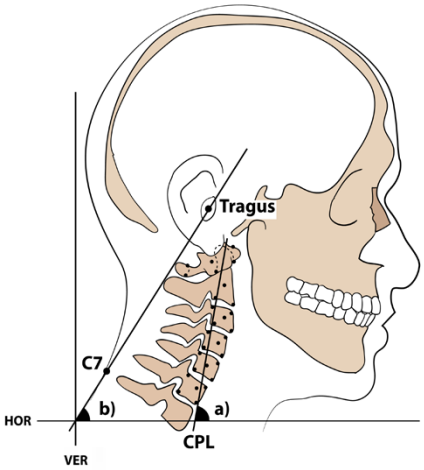


Fig. 4. Schematic diagram of the Cervical Position Line (CPL) proposed by Visscher et al. (2002), its reference points (·), and the angle with the horizontal plane (a); and a schematic diagram of the line between the tragus of the ear and the tip of the seventh cervical spinous process (C7) and its angle with the horizontal (b). x = mathematical centre of a vertebra. (Extracted from Visscher et al. 2002)

Palpation

The next step is to feel the tissues; palpation is part science, part art. Always try to have warm hands and short nails. Be relaxed and focus your attention on the anatomy. Move slowly from superficial tissues to deeper ones. Remember, if it feels nice for your hands, it feels nice for the patient!

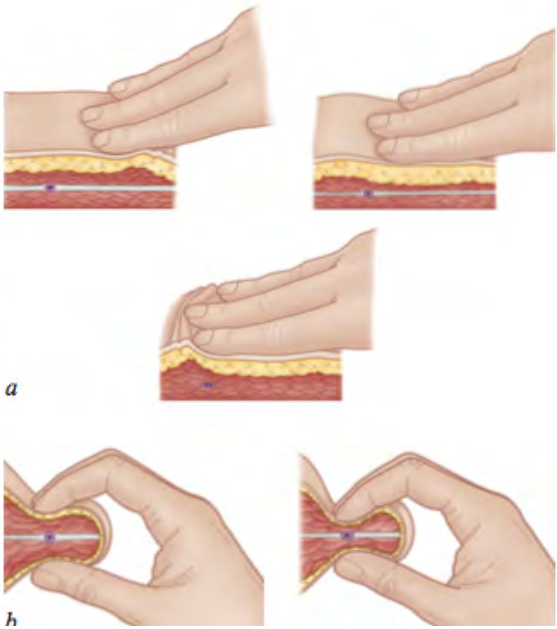
A thorough understanding of the anatomy is really helpful when it comes to trigger point work; we need to locate the taut bands in any specific muscles that might be involved. It can be very helpful to ask the patient to point to the pain; it helps to involve and empower them. Feel the local area; are there any tender spots? We cannot emphasize how important it is to reproduce the patient's symptoms; that is part of the 'magic' of trigger points. Holding a trigger point for more than 5 seconds produces a blush of pain in the distribution of a specific trigger point pain map. If you can reproduce the patient's symptoms, it has profound effects. It validates the patient's experience, tells them you know what they are doing, and actually starts the healing process.

It is also **important** to remember that one of the defining characteristics of trigger points is that pain may be felt remotely from where the trigger point is located. For this reason, we would advise you to study the trigger point maps found a little later on in the course before you start palpation.

Have a pad to note down any tender points or taut bands. Start with the patient sitting and feel the **upper trapezius**, **levator scapulae**, **SCM**. Ask the patient to lie on their back (supine) and feel the splenii, suboccipitals, temporalis, masseter, orbicularis oculi and the occipitofrontalis.

Remember, you are looking to feel for a taut band of muscle; the trigger point is somewhere in this taut band. Feel the direction of the muscle fibers; this will guide you to which muscles are affected. The two best techniques for feeling trigger points are cross fiber strumming (a) with a

flat fingered hold (sometimes you might see a muscle twitch as you strum it); and a pincer grip (b). The type of palpation you choose will depend on where the muscle is located.



Evaluation	Suggested Diagnosis
Clinical History	
• Recent onset, recent change, progressive symptoms of headache	Possible secondary headache
• Fever, weight loss, cancer history	Possible systemic illness, secondary headache
• Daily headache with occasional migraine-like flairs	Chronic migraine
• Daily headache without migraine-like flairs	Chronic tension type headache (TTH)
• Headache started 'out of the blue'	New daily persistent headache
• Near daily use of medications	Medication overuse headache (MOH)
• Severe headache <4 hours unilateral, tearing, rhinitis, clustering	Cluster headache
• History of cervical trauma, headache triggered by cervical movement	Cervicogenic headache (CGH)
• Obese, fertile woman, transient visual symptoms	Idiopathic intracranial hypertension
• Anxiety or depression	Comorbidity (other factors)
Physical Examination	Intracranial mass, idiopathic intracranial
• Papilledema	Hypertension
• Any neurological abnormality	Possible secondary headache
• Restricted or painful cervical motion or temporomandibular (TMJ) motion	Cervicogenic headache, Temporomandibular (TMJ) dysfunction
Laboratory investigation	
• Anemia, liver, and thyroid function abnormalities	Evaluate and treat underlying condition

Differential diagnosis - What else could it be?

Differential diagnosis is an essential tool to assess the risk of any problems when they come to the office for treatment. Early detection and treatment can facilitate effective recovery, plus we don't want to hurt someone who comes to us in pain.

We highly recommend using the pathological sieve **CDFIMNRT** to sift through the potential alternatives when it comes to a headache; as we have said, rarely, they can be the result of a more serious underlying condition. Remember too, that it is possible to have two or more conditions happening at the same time. This sieve is a quick and easy format that you can apply to all complaints. Don't forget to add **'emotional'** to this list.

PATHOLOGICAL SIEVE	
HEADACHES	
CONGENITAL	Vascular malformation, Syringomyelia, Intracranial anomaly
DEGENERATIVE	Cervical issues, arthritis/disc C3/4/5/6/7, shoulder issues, Carotid artery dissection (in the neck), Vertebral artery disease (VAD)
FUNCTIONAL	Sinus headache, Idiopathic intracranial hypertension, Post-sex Headache, Swimming goggle headache, Post-exercise headache, Cervical spinal disorders (CGH), Temporomandibular joint (TMJ) dysfunction, sleep disorders such as obstructive sleep apnea, COPD Headache. Hypertension (raised blood pressure)
INFECTIVE	Abscess, Shingles, Vasculitis, Respiratory, Meningitis

METABOLIC	Hormonal: Hyperparathyroidism, Pituitary disease related, menopause. Inflammatory arthritis (HLAB27), Psoriatic Arthropathy, Polymyalgia Rheumatica - Giant cell arteritis (GCA).
NEOPLASTIC	Most commonly secondary metastases (brain), Glioma of the optic nerve, Neuromas of the inner ear. Pheochromocytoma of the adrenal glands can cause flushing and headache
RETICULO-ENDOTHELIAL	Neuropathy: e.g., Mononeuropathy multiplex Systemic Lupus Erythematosus - (SLE), Multiple myeloma, Paget's disease
TRAUMATIC	Whiplash associated disorder, Post-traumatic headache, Subdural hematoma

We advise you to follow the **SNOOP** criteria if you are worried about the headache[25]:

• **S**ystemic symptoms (fever, weight loss) or secondary headache risk factors (HIV, systemic cancer)

• **N**eurologic symptoms or abnormal signs (confusion, impaired alertness, or consciousness)

• **O**nset: sudden, abrupt, or split-second

• **O**lder: new onset and progressive headache, especially in middle-age >50 (giant cell arteritis - GCA)

• **P**revious headache history or headache progression: first headache or different (change in attack frequency, severity, or clinical features)

Patients who have a sudden or abrupt headache that peaks in seconds or minutes require careful assessment to exclude causes such as

subarachnoid hemorrhage (SAH), venous sinus thrombosis, arterial dissection, or raised intracranial pressure.

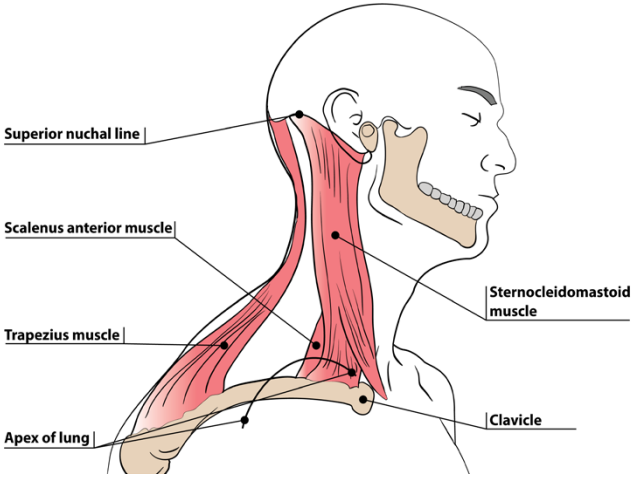
Beliefs, attitudes and emotions

Beliefs and attitudes about pain have a role to play in all types of pain. Sometimes even if the pain improves, the patient becomes 'hyper' symptom focused and is unable to recover fully. Pain behavior is complex; it is influenced by many factors as diverse as anger, depression, and hopelessness, to childhood and family pain behavior. Other factors such as family problems, work problems, litigation issues, or even diagnostic and treatment issues may also have a role to play. We need to keep all of these factors in mind as we approach treatment.

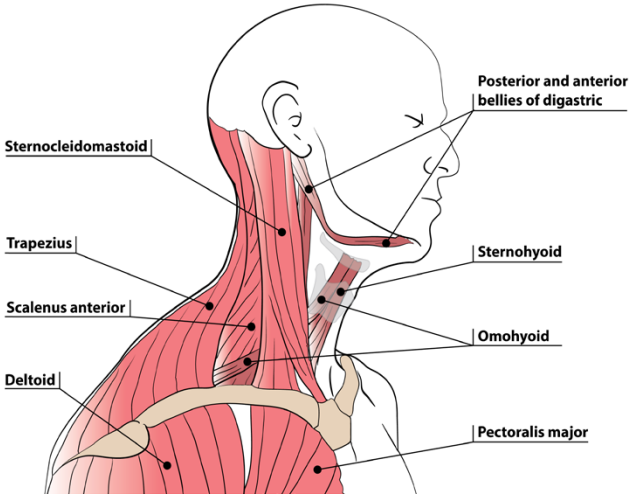
The Main Muscles

Surface Anatomy

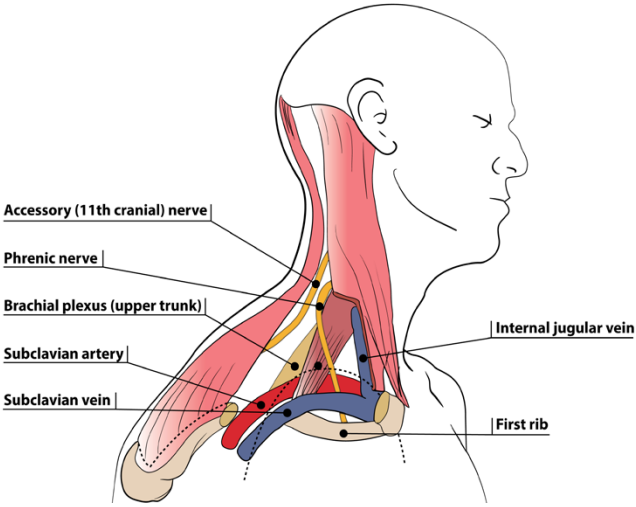
Posterior triangle of the neck



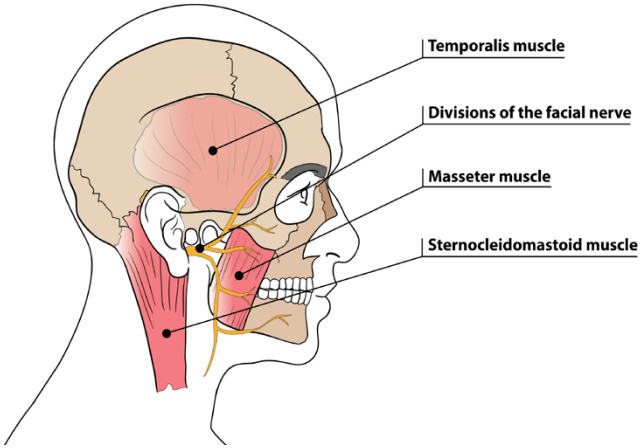
Lateral aspect of the neck: muscles



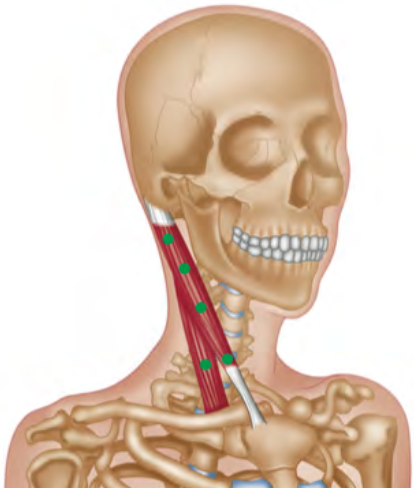
Subclavian vessels and branchial plexus

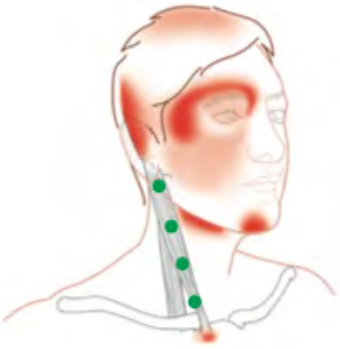


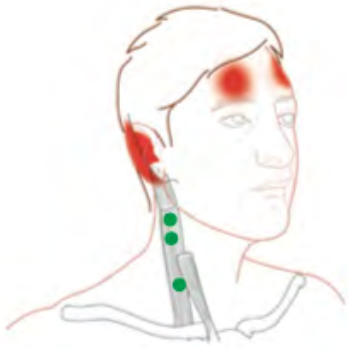
Lateral aspect of the head: soft tissues



Sternocleidomastoid muscle and pain map





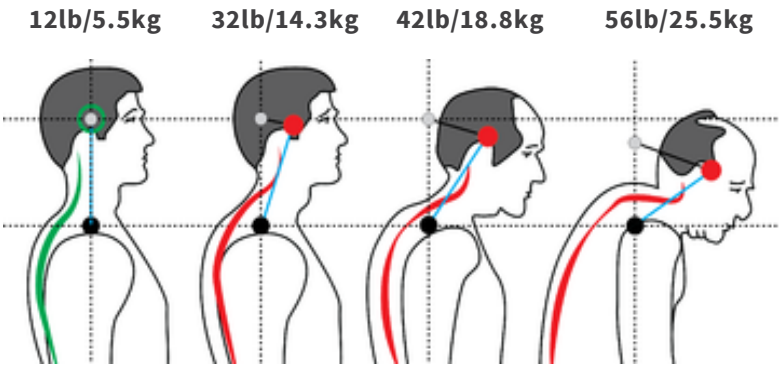


Sternocleidomastoid (SCM) & SCM Syndrome

This syndrome includes neck stiffness with decreased rotation along with the following: nausea/tinnitus/vertigo/torticollis (spasm in the neck). Remember to check for trigger/tender points in the SCM and upper trapezius. SCM syndrome is often associated with 'Upper Crossed Pattern', neck aging, or after a trauma such as a whiplash; it is also prevalent in certain occupations (such as violinists).

Remember the head is heavy. In order to maintain balance, the eyes and ears need to be kept level. As we age, and often due to spinal degeneration, we frequently see the head flexing forward. This is a particularly difficult scenario as we are dealing with an older person with less muscle tone AND chronically tightened SCM's. Over time, a tight SCM with trigger points may well lead to headaches, disequilibrium, or balance issues (see below).

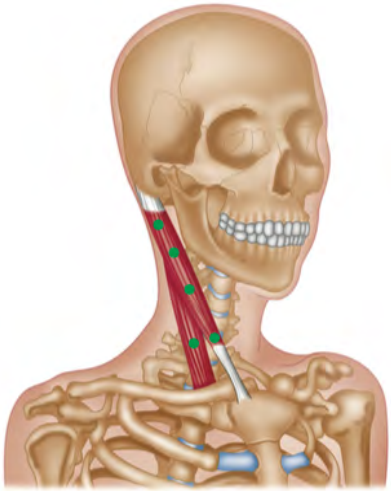
'Relative' head weight according to posture – SCM

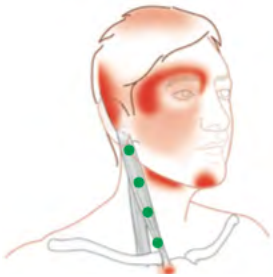


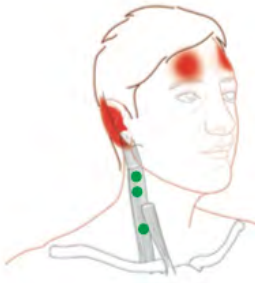
SCM & Vertigo

Travell and Simons have suggested another role for the sternocleidomastoid, that of spatial awareness and balance. This makes sense when we consider the changes in SCM tone and trigger point formation that occur secondary to altered postural dynamics of the head (head forward/upper crossed pattern). As we have already seen, our body attempts to keep our ears and eyes level as far as possible to help maintain balance. The two heads of SCM seem to have differing roles and a trigger point in either of them can create a host of unwanted effects.

Sternal head of SCM: Eye issues such as blurred vision and/or ptosis, sinus headaches

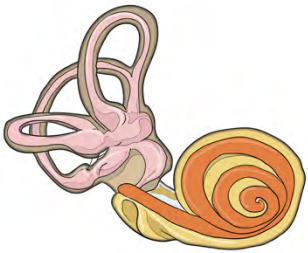






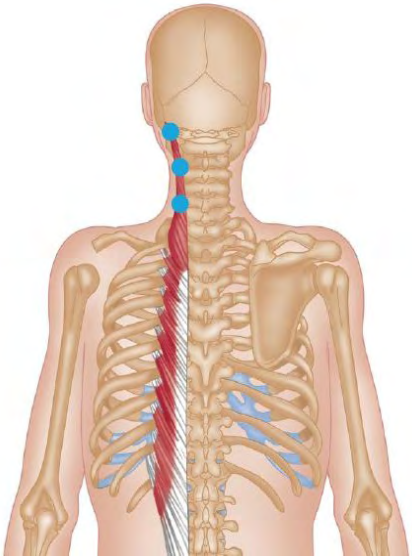
Clavicular Head of SCM: forehead pain, ear pain, dizziness, night time (paroxysmal) dry cough

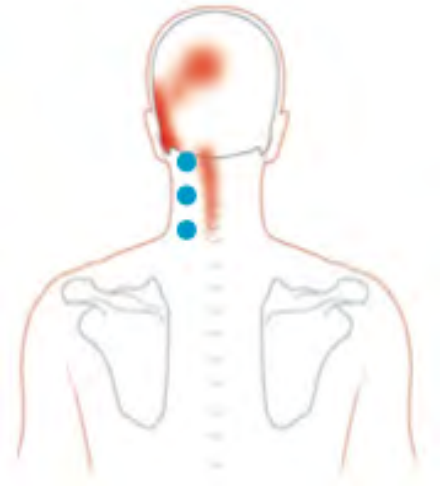
- Concomitant Symptoms
    - Postural dizziness
    - Vertigo
    - Dysequilibrium
- Sudden fall when bending
- Loss of balance (Ataxia)
    - Exaggerated postural responses (looking up or down)
    - Benign postural vertigo
- Nausea (vomiting rarely)



Longissimus Capitis

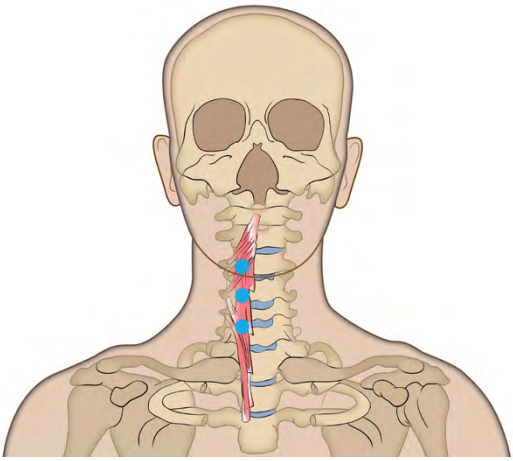
Trigger Point and Pain Map

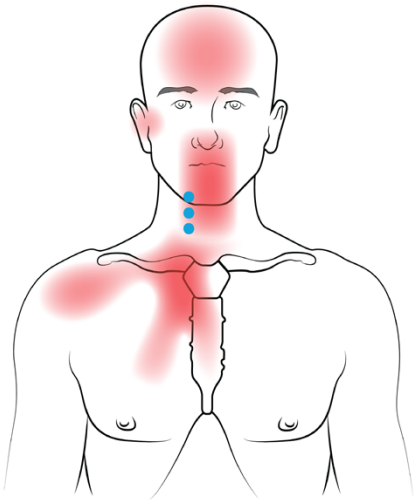




Longus Colli (The psoas of the neck)

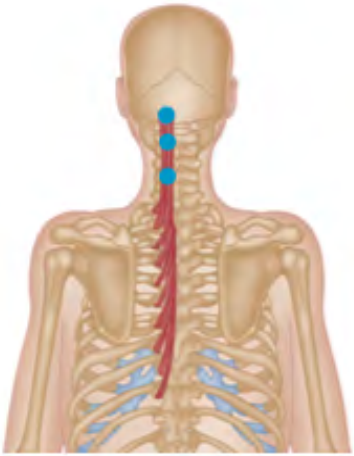
Trigger Points and Pain Map

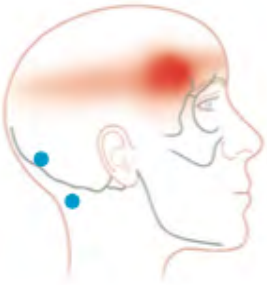


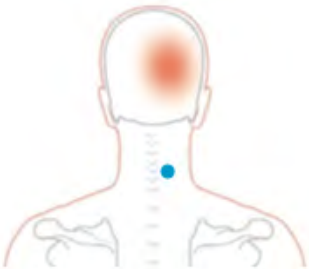


Semispinalis Capitis and Cervicis

Trigger Points and Pain Map

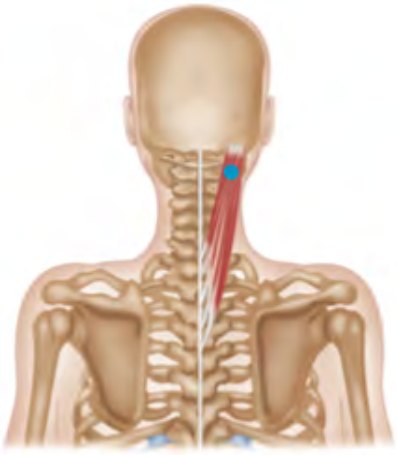


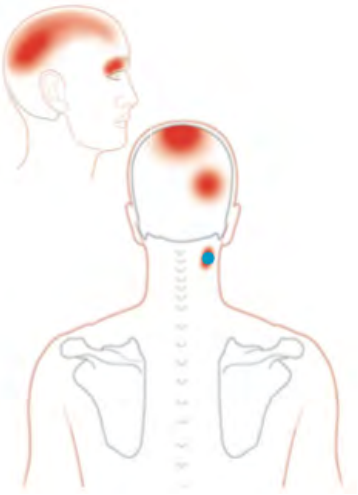




Semispinalis Capitis

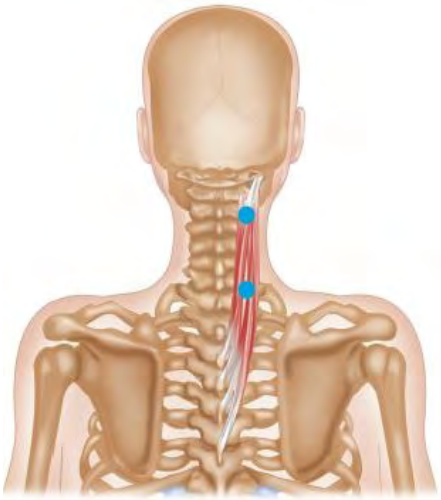
Trigger Points and Pain Map

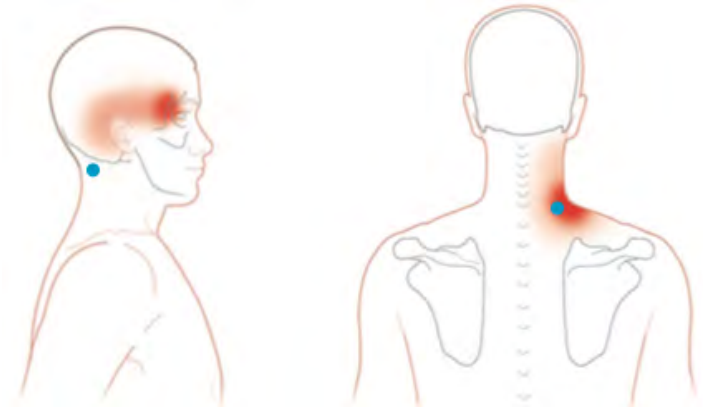




Splenius Cervicis

Trigger Points and Pain Map

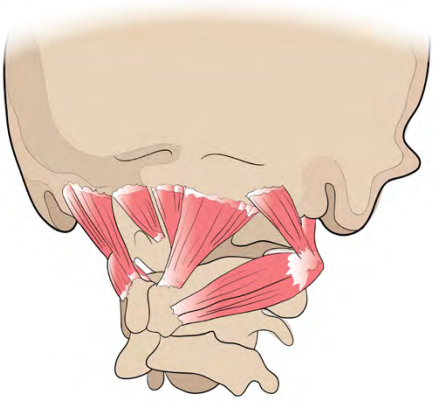


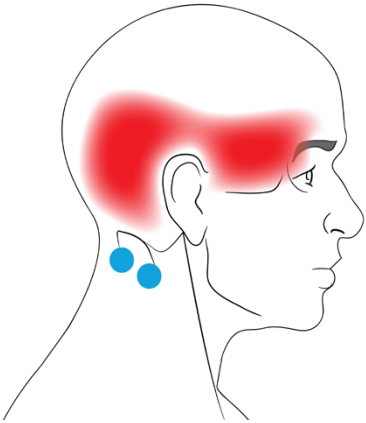


The suboccipital muscles – obliquus capitis

Trigger points and pain map

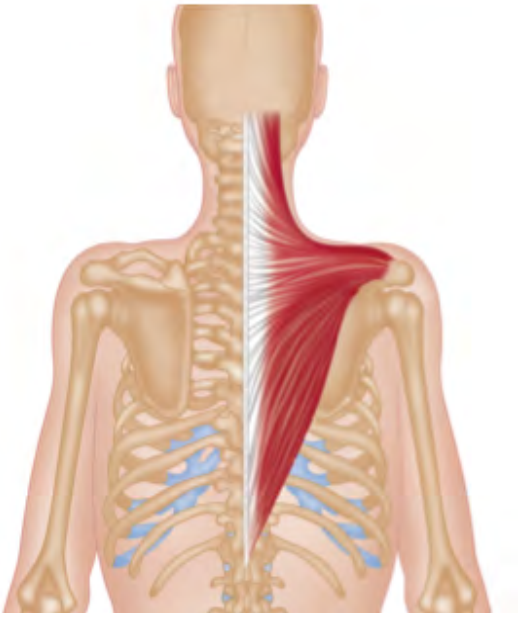
The reason we look at the obliquus capitis is that this group of muscles is really deep, and it is the easiest to palpate and to change with trigger point intervention.

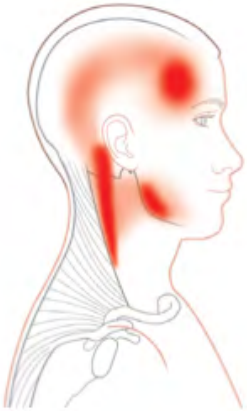




Upper Trapezius Muscle

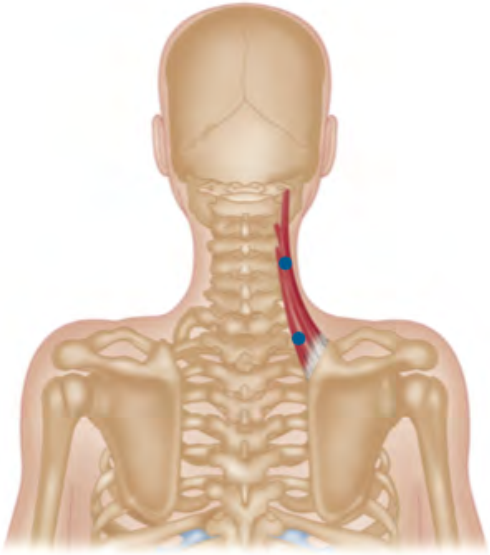
Pain Map

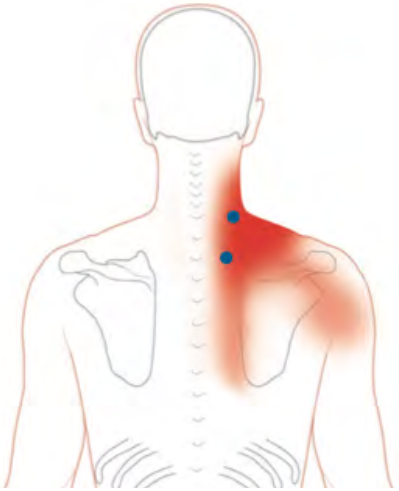




Levator Scapulae

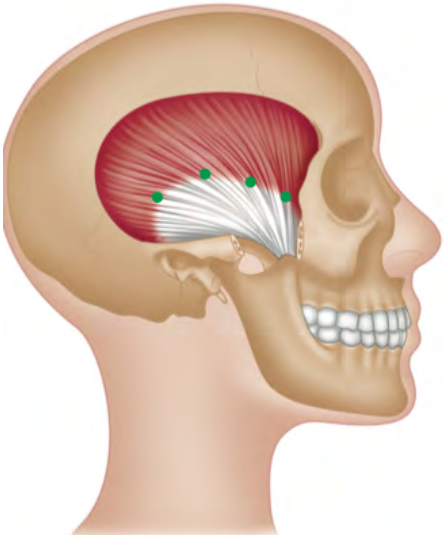
Trigger Points and Pain Map

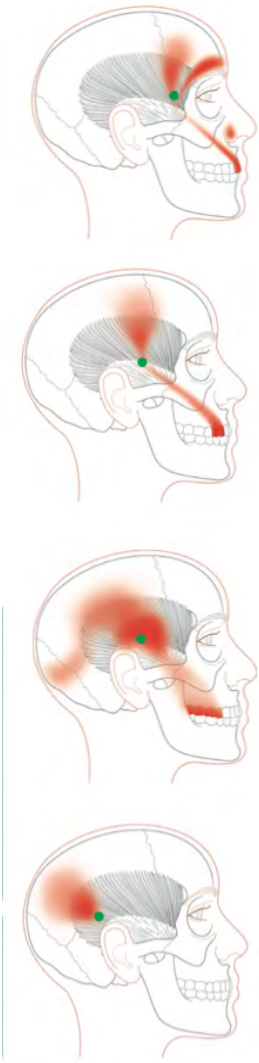




Temporalis

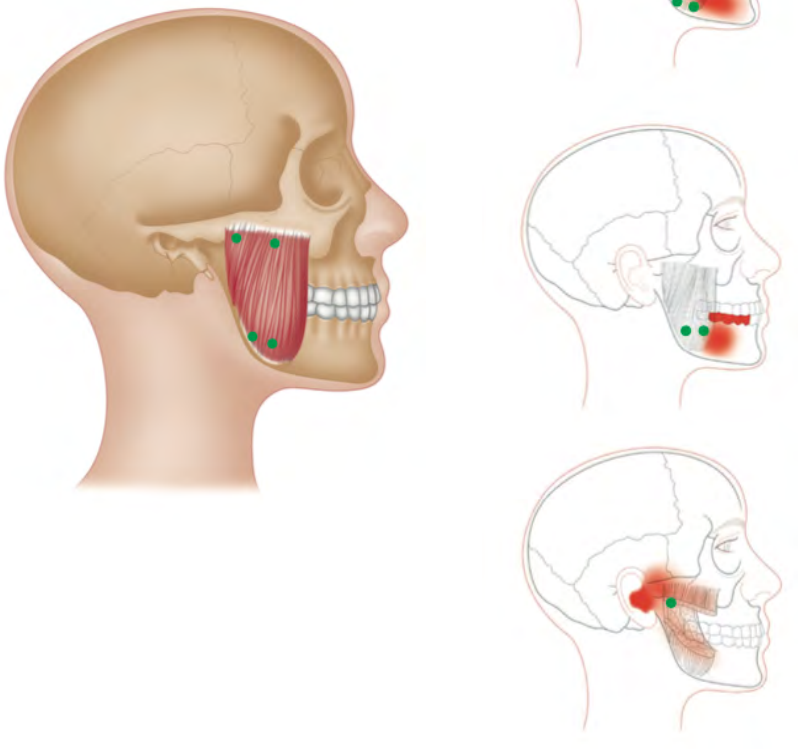
Trigger Points and Pain Map





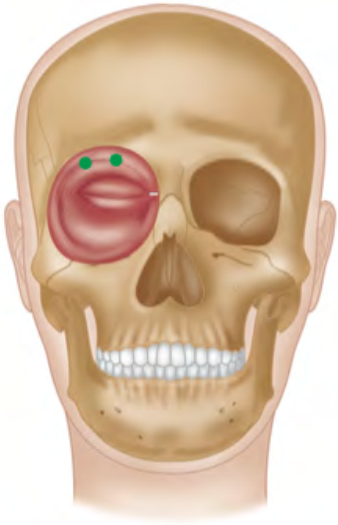
Masseter

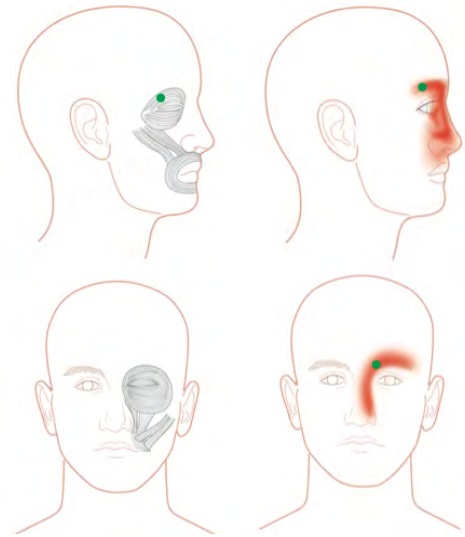
Trigger Points and Pain Map



Orbicularis Oculi

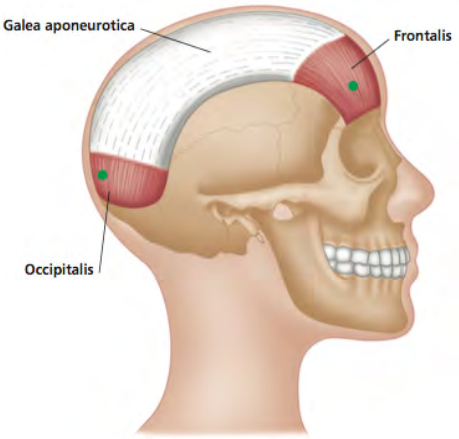
Trigger Points and Pain Map

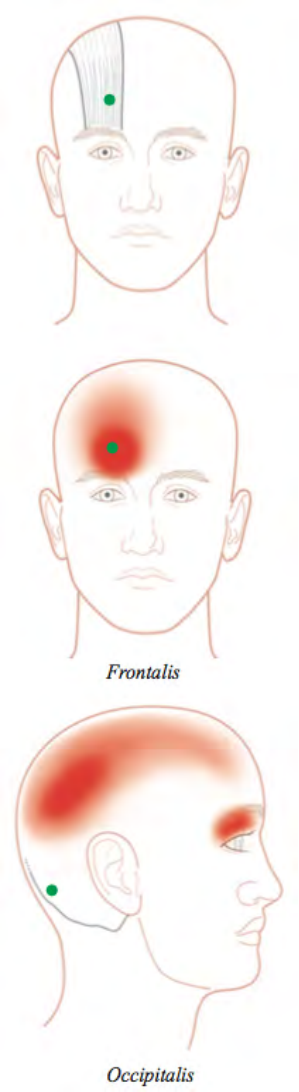




Occipitofrontalis

Trigger Points and Pain Map





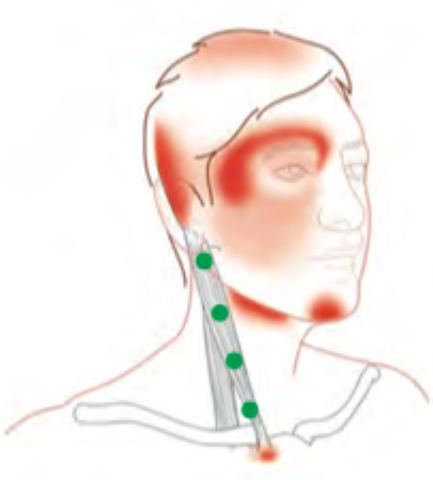
Trigger Points 101

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 exquisite, 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.

About Trigger Points

- 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 spindle, which becomes 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?

There are several possible trigger point mechanisms[26]:

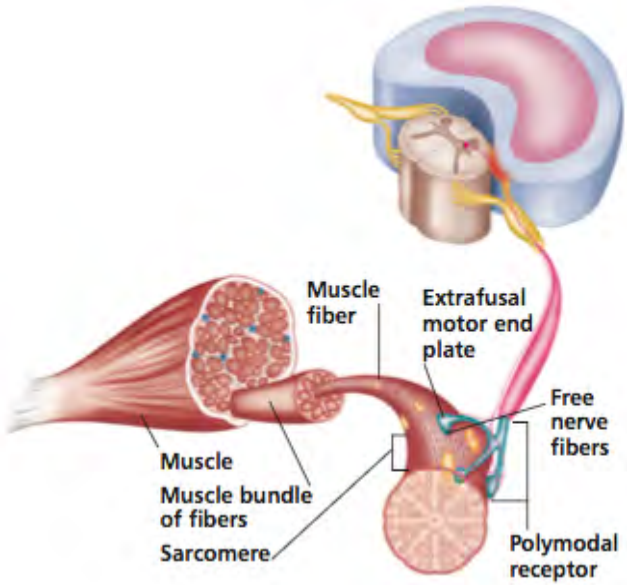
- Low-level muscle contractions
- Uneven intramuscular pressure distribution
- Direct trauma
- Unaccustomed eccentric contractions
- Eccentric contractions in unconditioned muscle
- Maximal or submaximal concentric contractions

Pathophysiology of trigger points

The Integrated Trigger Point Hypothesis 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'[27], 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[28]. Others have 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 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)[29] 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[30]:

- **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)[31], Hong (1996)[32], 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.

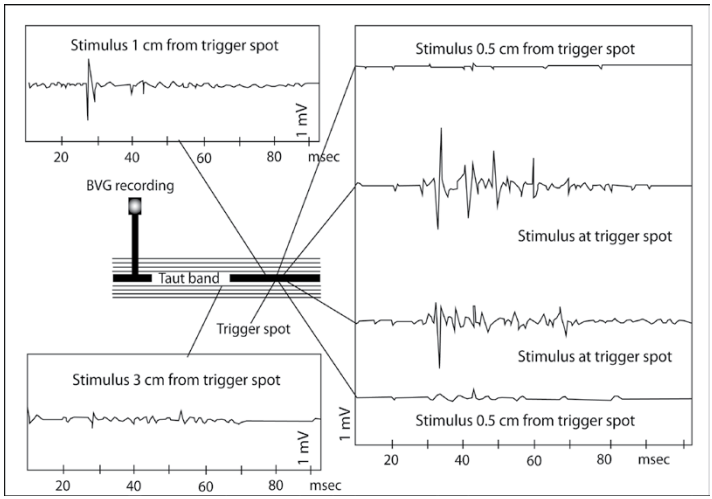
Eventually, the brain sends a signal to the muscle in which the trigger point manifests to cause it to rest. This leads to hypertonia, weakness, shortening, and fibrosis (muscle stiffness) of the muscle, along with reflex inhibition of other muscle groups. Under microscopy, these fibers have been described as 'ragged red.' Treatment is thus aimed at interfering with and attenuating this vicious cycle.

Other theories include radiculopathic theory[33, 34] and polymodal theory[35].

Evidence for trigger points

Studies over the past decade have imaged trigger points[36, 37], shown that their activation results in CNS activation through fMRI scanning[38], demonstrated electrophysiological activity at the trigger point[39], and have shown biochemical changes in the trigger point zone[40]. Further studies have shown that manipulation of the trigger point modulates muscle function[41], and induces local and referred pain[42].

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 see the book 'Muscle Pain' by Mense[43]

Trigger point classification

Trigger points are described—according to location, tenderness, and chronicity—as: **central** (or primary), **satellite** (or secondary), **attachment**, **diffuse**, **inactive** (or **latent**), and **active**.

Central (or primary) trigger points

These are the most well-established and 'florid' points when they are active, and are usually what people refer to when they talk about trigger points. Central trigger points always exist in the center of the muscle belly, where the motor endplate enters the muscle.

Note: Muscle shape and fiber arrangement is of importance in this regard. For example, in multipennate muscles (such as the deltoid), there may be several central points. Also, if muscle fibers run diagonally, this may lead to variations in trigger point location.

Satellite (or secondary) trigger points

Trigger points may be 'created' as a response to the central trigger point in neighboring muscles that lie within the referred pain zone. In such cases, the primary trigger point is still the key to therapeutic intervention: the satellite trigger points often resolve once the primary point has been effectively rendered inactive. As a corollary, it is also true that satellite points may prove resistant to treatment until the primary central focus is weakened; such is often the case in the paraspinal and/or abdominal muscles.

Attachment trigger points

Myofascia is a continuum. It has been noted that the area where the tendon inserts into the bone (tendo-osseous junction) is often 'exquisitely' tender (Simons et al. 1998; Davies 2004). This may well be

the result of the existing forces travelling across these regions. It has also been suggested by the same authors that this may result from an associated chronic, active myofascial trigger point. This is because the tenderness has been demonstrated to reduce once the primary central trigger point has been treated; in such cases, the point is described as an attachment trigger point. Furthermore, it has been suggested that if a chronic situation occurs where the primary and attachment trigger points remain untreated, 'degenerative changes' within the joint may be precipitated and accelerated (Simons et al. 1998).

Diffuse trigger points

Trigger points can sometimes occur where multiple satellite trigger points exist secondary to multiple central trigger points. This is often the case when there is a severe postural deformity, such as a scoliosis, and an entire quadrant of the body is involved. In this scenario, the secondary points are said to be diffuse. These diffuse trigger points often develop along lines of altered stress and/or strain patterns.

Inactive (or latent) trigger points

This applies to lumps and nodules that feel like trigger points. These can develop anywhere in the body and are often secondary. However, these trigger points are not painful, and do not elicit a referred pain pathway. The presence of inactive trigger points within muscles may lead to increased muscular stiffness. It has been suggested that these points are more common in those who live a sedentary lifestyle (Starlanyl & Copeland 2001). It is worth noting that these points may reactivate if the central or primary trigger point is (re)stimulated; reactivation may also occur following trauma and injury. Latent trigger points may have associated autonomic symptoms with pain, and their presence results in a limited range of motion, muscle fatigability, and muscle weakness as in the active[44, 45] presentation.

Active trigger points

This can apply to central and satellite trigger points. A variety of stimulants, such as forcing muscular activity through pain, can activate an inactive trigger point. This situation is common when activity is increased after a road traffic accident (RTA), where multiple and diffuse trigger points may have developed. The term denotes that the trigger point is both tender to palpation and elicits a referred pain pattern.

Ligamentous trigger points

There is some evidence that ligaments may develop trigger points[46] but the relationships are not clear. The sacrotuberous and sacrospinous ligaments can refer pain down to the heel, and the iliolumbar ligament can refer pain down to the groin and even into the testicles or vagina (Hacket 1958). Trigger points in the sacrotuberous ligaments can have profound effects on low back, **lumbar erector spinae,** and pelvic pain[47], and may also be associated with backache, neck pain, and even vocal dystonia[48]. As well as stabilizing structures, ligaments have strong proprioceptive functions (Varga et al. 2008). Working on ligament trigger points therapeutically can be clinically useful as part of the neuroplastic model (explored later). Trigger points may manifest in the Anterior Longitudinal Ligament (ALL) of the spine (e.g. after whiplash) which may result in neck instability[49]. The Fibular Collateral Ligament (FCL) has a similar referred pain pattern to the **vastus lateralis,** and trigger points in the ligamentum patellae are profoundly useful for treating knee pain syndromes.

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? I 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 prewired 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 then responds by initiating movements for maximal efficiency. Muscle groups are then arranged hierarchically into functional units of agonist, antagonist, fixator, and synergist.

When a structure is damaged or strained in the neck or spine, 'switch-off' mechanisms kick in to avoid further 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 also, over time makes T-cells simultaneously hyperactive and unresponsive (or silent). 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 S1 and motor M1 cortices.

Holding patterns

A few years ago, we were stuck in an airplane for almost an hour, circling around Heathrow Airport, waiting for a 'landing window.' The Captain informed us that we were in a holding pattern and should be landing shortly.

We have thought a lot about this phrase ever since. For us, it 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.

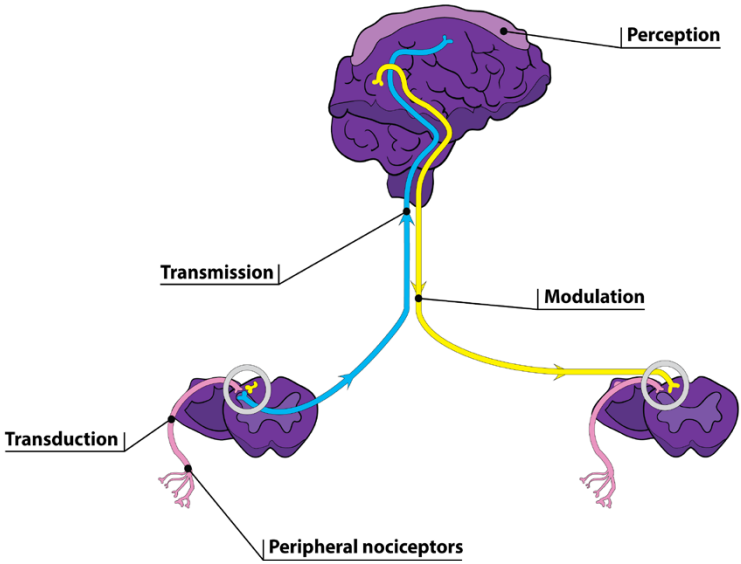
Stress and tension holding pattern

It is widely recognized that stress and tension have a large role to play in headaches. The trapezius muscle is especially well known to manifest tension. It is one of the only muscles to receive innervation directly from the brain via the spinal accessory (XI cranial) nerve. Some authorities talk of an 'emotional dumping' effect when there are high levels of emotional stress that seem to overload in the brain, causing it to dump tension in the Traps (an emotional holding pattern). Certainly this is something we see clinically; there is also some published evidence to support this. According to Wahlström, high emotional stress (is) associated with higher muscle activity in the trapezius muscle[50]. Furthermore, stress and tension combined with poor work ergonomics can be potentially disastrous.

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 are 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)[51] 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.

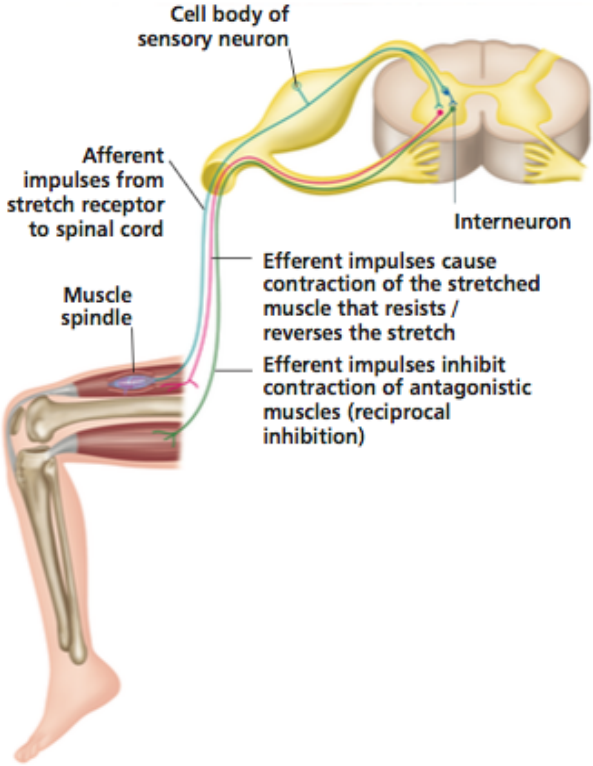
Reciprocal inhibition

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.

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; with regard to myofascial trigger points, 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[52]); in addition, mechano-insensitive 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 pain-inducing (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; the advice, therefore, is to interfere with this process as soon as possible. The good news is that myofascial trigger point release (and dry needling techniques) has been reliably demonstrated to reduce these effects (Mense S, Gerwin RD. *Muscle Pain*: *Understanding the Mechanisms*. Springer: Heidelberg, 2010).

Treatment

Treatment might include a combination of:

- (Maitland's) Spinal mobilization
- Deep Stroking Massage (DSM)
- Inhibition Compression Technique (ICT)
- Stretch and spray
- Muscle Energy Technique (MET)
- NAT
- Exercise
- Stretching

Mobilization

According to the pioneering work of Geoffrey Maitland, headaches and neck stiffness can be the result of 'alignment' issues with the spinal joints. We have already seen how the upper vertebrae of the neck are involved in CGH, TTH, and migraines. Although this is an **advanced technique** often used by Osteopaths, Chiropractors, and/or physical therapists, it is worth mentioning here, as it really helps to improve neck mobility and reduce headaches when combined with a trigger point therapy routine. Maitland maintains that each spinal joint has a different movement arc in a different direction to other surrounding joints. Each joint should be assessed according to these movements, which are:

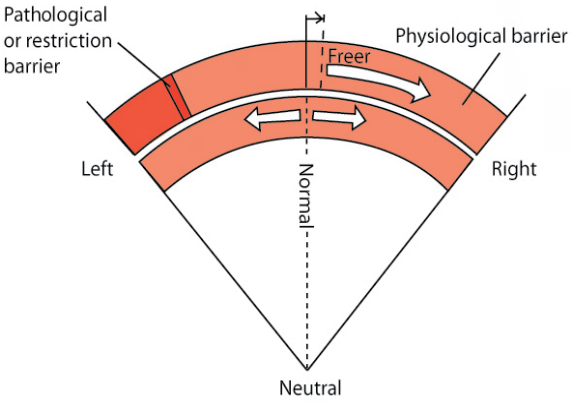
- A-P (Anteroposterior)
- P-A (Posteroanterior)
- Longitudinal caudad
- Longitudinal cephalad
- Joint distraction
- Medial glide

• Lateral glide

The idea is to assess the spinal joint mobility and then apply gentle corrective forces or 'glides' to normalize these misalignments. These forces are applied in various 'grades'. **As we have already said, this type of work is advanced and should NOT be attempted without proper training and insurance.**

Maitland's grades of corrective movement:

- Grade I: Small amplitude movement performed at the beginning of the range.
- Grade II: Large amplitude movement performed within the range but not reaching the limit of the range.
- Grade III: Large amplitude movement performed up to the limit of the range.
- Grade IV: Small amplitude movement performed at the limit of the range.



Deep Stroking Massage (DSM) and Inhibition Compression Technique (ICT)

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

How do I know it is a trigger point?

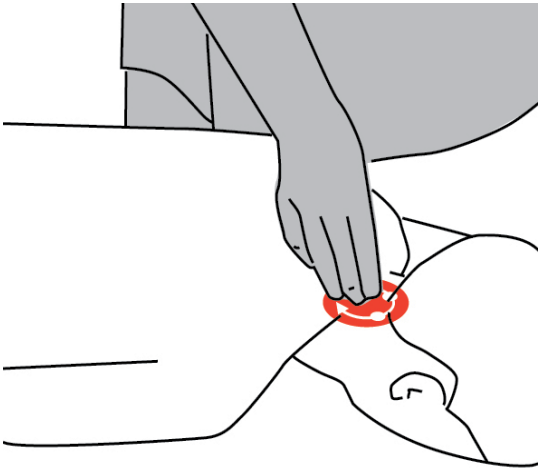
You are looking for:

- 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 - 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.

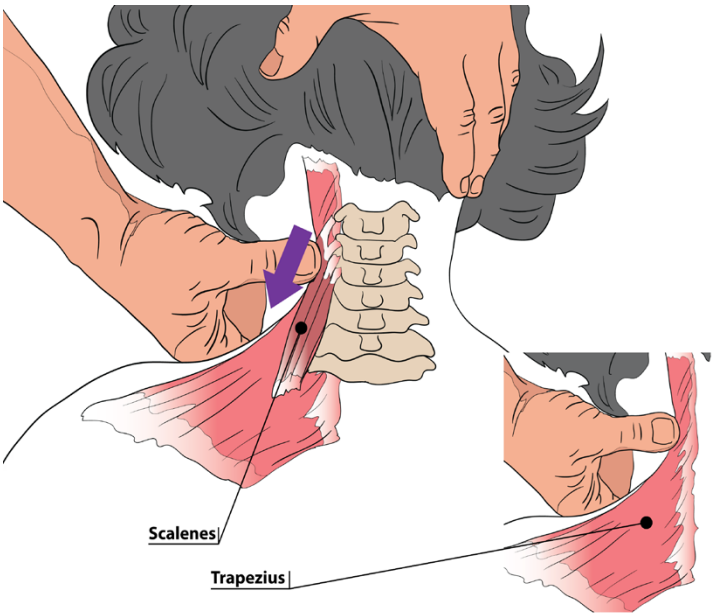
Example – ICT to the posterior cervical muscles



Technique – Deep Stroking Massage : (DSM)

- Identify the trigger point by having a look at the illustrations, and then feel for the taut muscle or band
- Lubricate the skin with oil, cream, or lotion
- Identify and locate the tender/trigger point or taut band
- In one direction only 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.

Example DSM for the Upper Trapezius



Spray and stretch

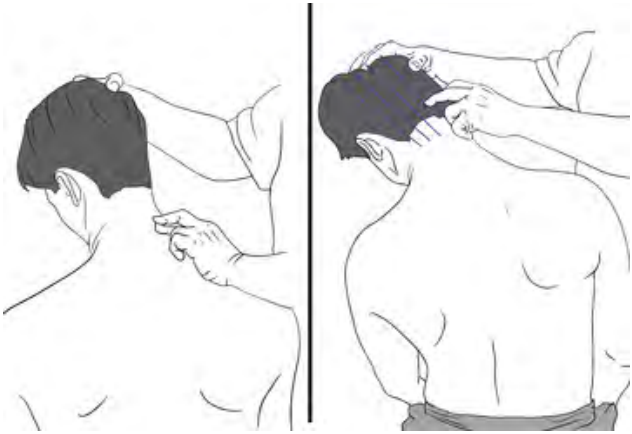
According to the pioneers of trigger point therapy, spray and stretch is the 'single most effective noninvasive method' for deactivating trigger points. The basic spray and stretch technique is quite straightforward, as it does not require the same precise localization of trigger points as for ICT. You need only locate and identify the affected/host muscle to release its fibers. Apply a cold spray, and then stretch the muscle. This is especially good for the **upper trapezius** and **splenius cervicis** muscles. There are several great cold sprays on the market.

Procedure

1. Identify the taut band, then **spray**: The spray is aimed out of the inverted bottle nozzle at 30 degrees to the skin, in a fine jet over a distance of about 20–50 cm (do not aim at a single spot).

2. **Stretch**: This is the therapeutic component of the technique. While two to three sweeps of spray are applied to the affected/host muscle, the muscle is gently extended to its full stretch length.

Example – spleneus cervicis spray and stretch



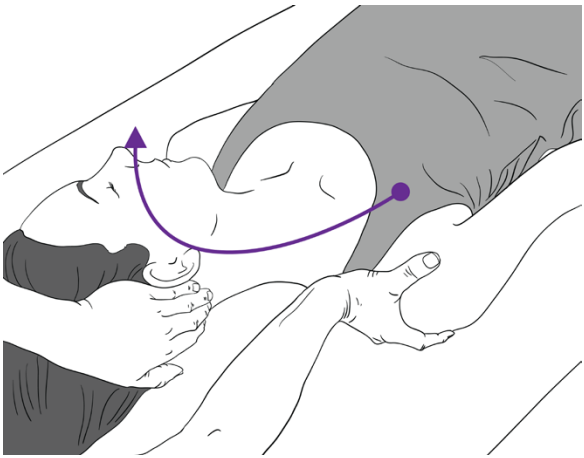
Isometric contraction technique: Muscle Energy Technique – MET

This technique is hugely effective for the upper trapezius. Remember we talked about the concept of an over-stimulated motor endplate? Utilizing contraction and relaxation while fixing through the trigger point may well 'normalize' the sarcomere length. It is believed that MET sets in place a cascade, releasing the affected actin and myosin, and reducing the energy crisis.

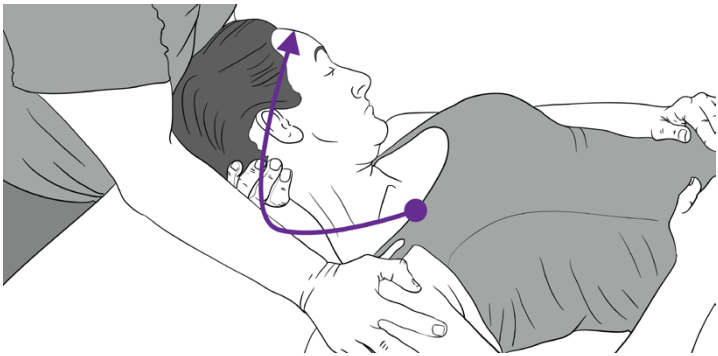
Procedure

1. Hold or fix through the shoulder and neck either a) in side bending or b) side bending with slight rotation.
2. Ask the patient to actively contract the muscle with 30-40% resistance for a slow count of ten whilst you hold back against them, **keeping the neck in the same position**.
3. Ask the patient to relax.
4. Actively and passively stretch the muscle a) in side bending, b) in rotation and side bending.

Upper trapezius MET



SCM and lateral neck flexors MET



Common Headache Patterns and Associated Trigger Points

1. Sternocleidomastoid
Splenius capitis

2. Trapezius
Sternocleidomastoid
Posterior neck
Occipitalis
Digastric
Temporalis

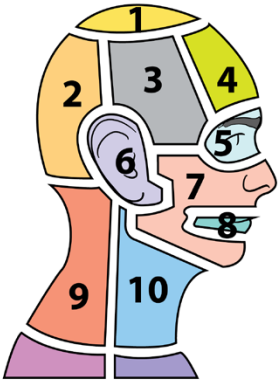
3. Trapezius
Sternocleidomastoid
Temporalis
Posterior neck

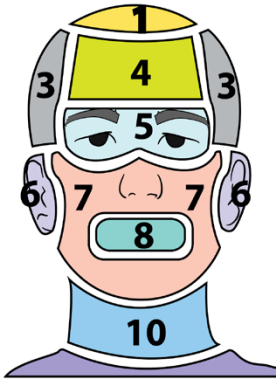
4. Sternocleidomastoid
Semispinalis capitis
Facial / Scalp

5. Sternocleidomastoid
Temporalis
Posterior neck
Masseter
Facial / scalp
Trapezius

6. Lateral pterygoid
Masseter
Sternocleidomastoid
Medial pterygoid

7. Sternocleidomastoid
Masseter
Lateral pterygoid
Trapezius
Digastric
Medial pterygoid
Facial / Scalp

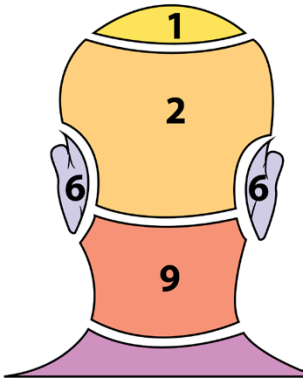




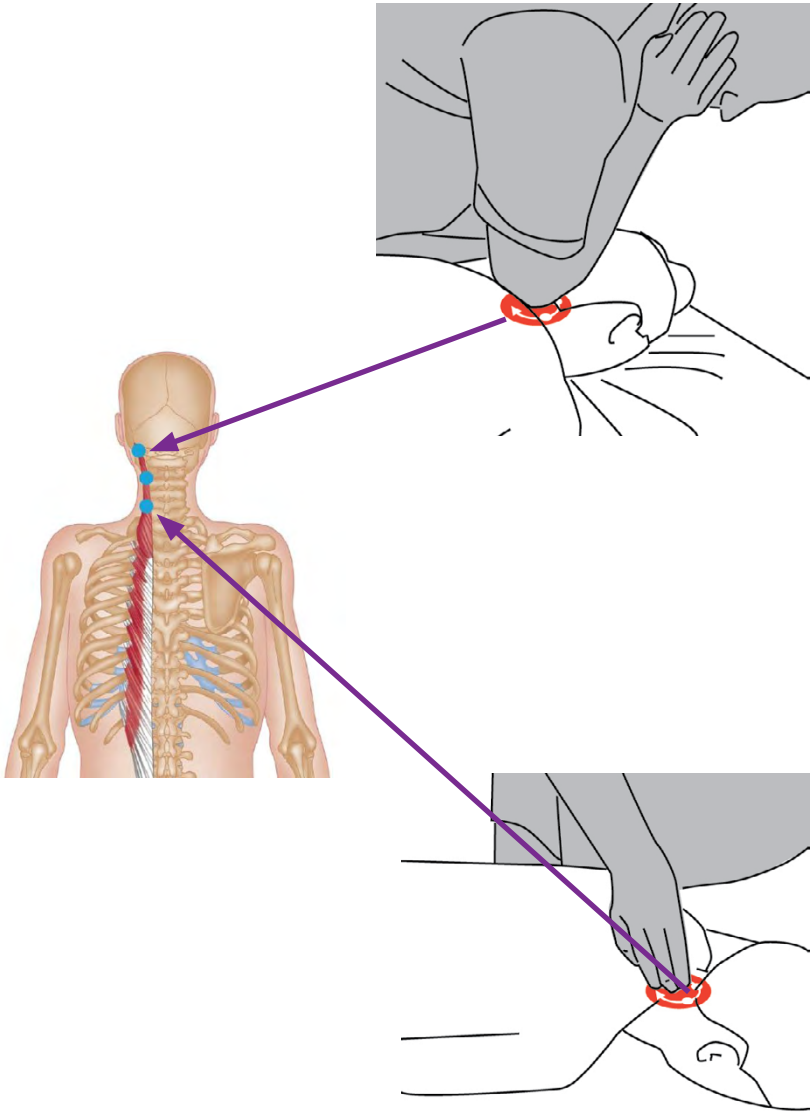
8. Temporalis
Masseter
Digastric

9. Trapezius
Cervical multifidi
Splenius cervicis
Levator scapulae
Infraspinatus

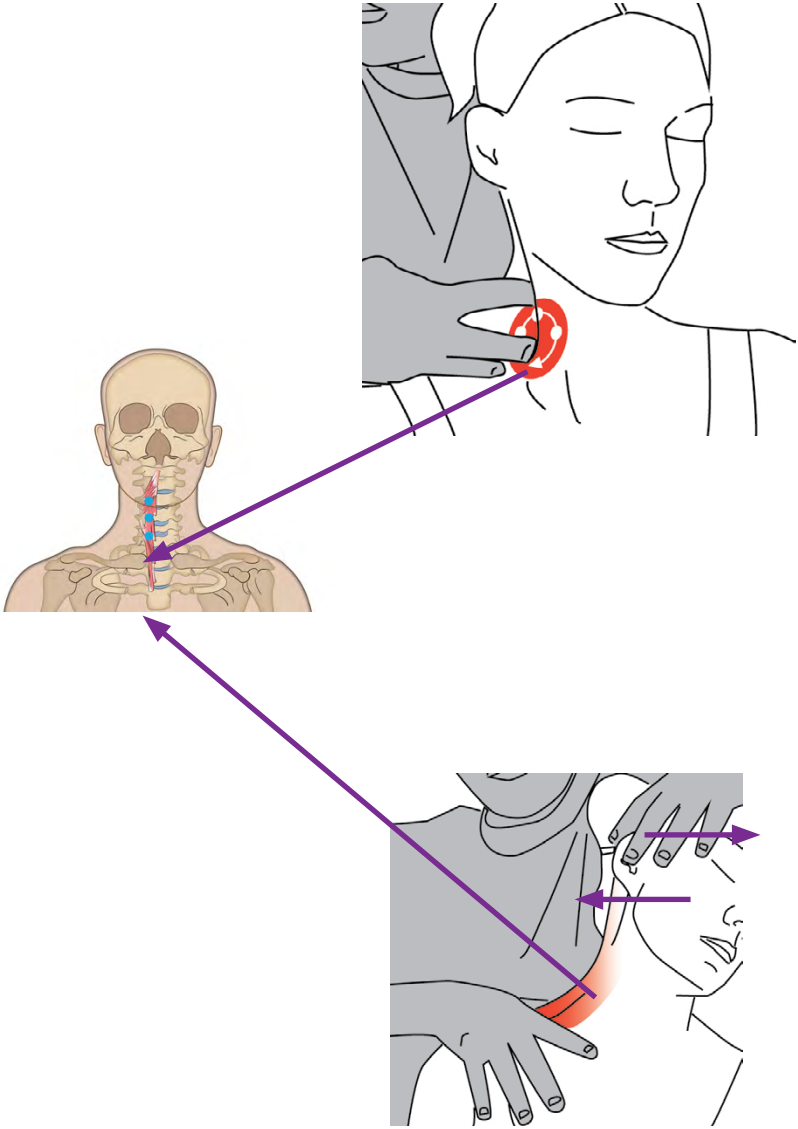
10. Sternocleidomastoid
Digastric
Medial pterygoid



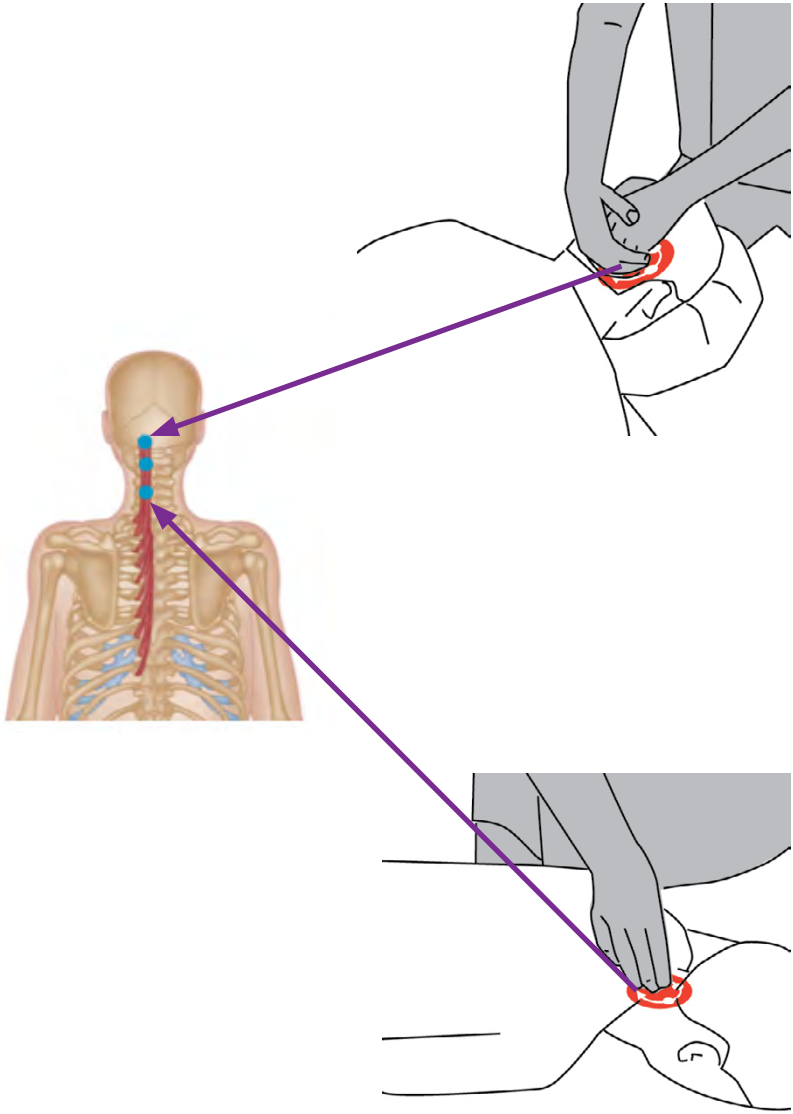
Longissimus Capitis - Treatment



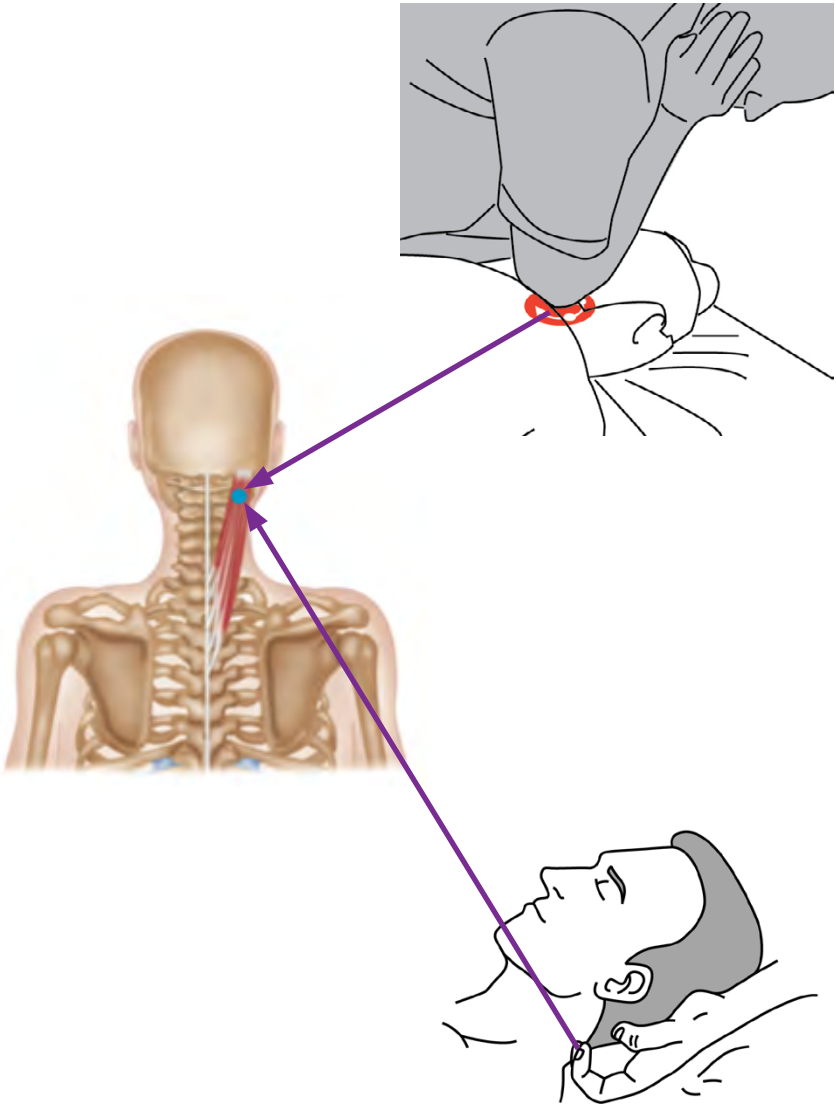
Longus Colli (The psoas of the neck) – Treatment



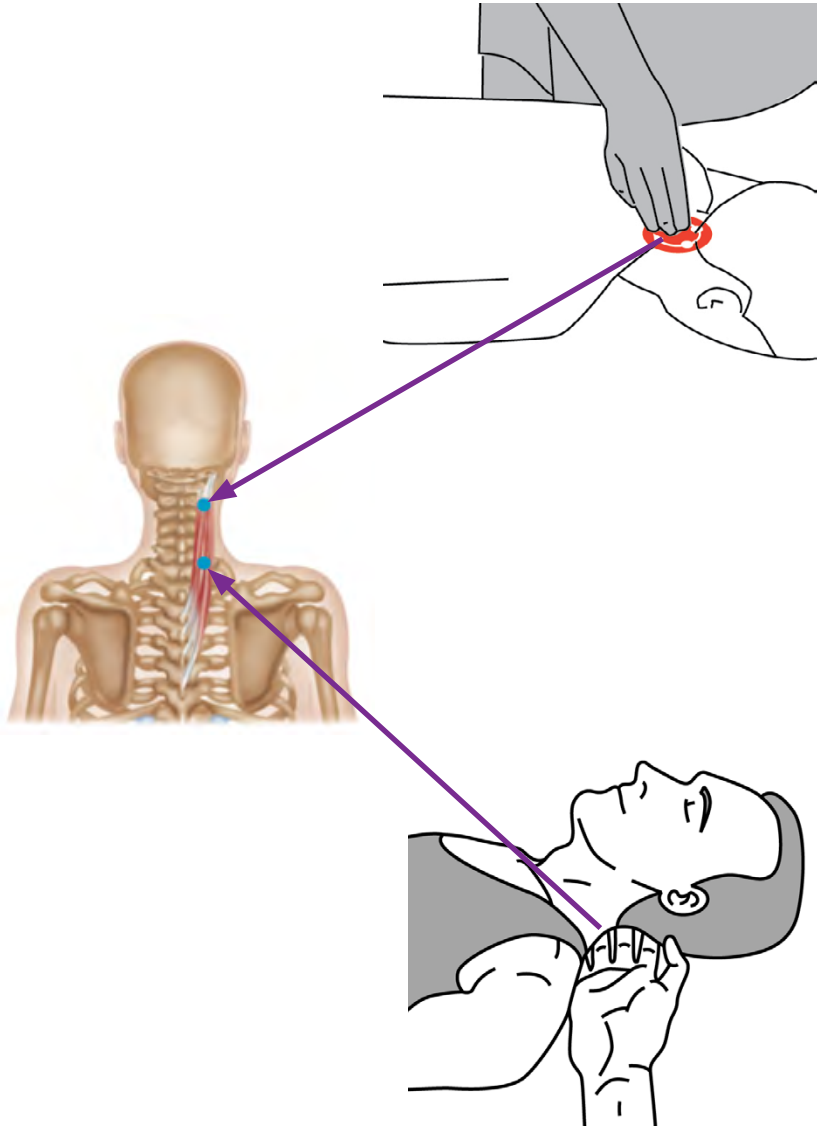
Semispinalis Capitis and Cervicis - Treatment



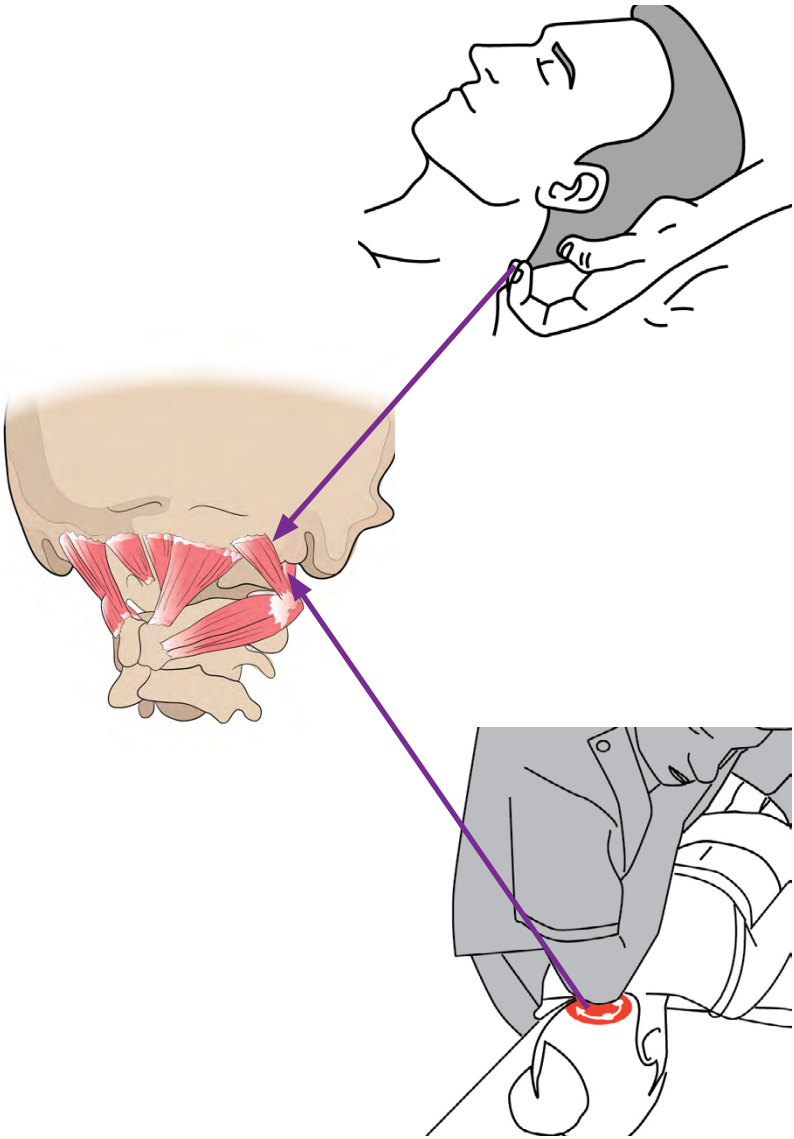
Semispinalis Capitis - Treatment



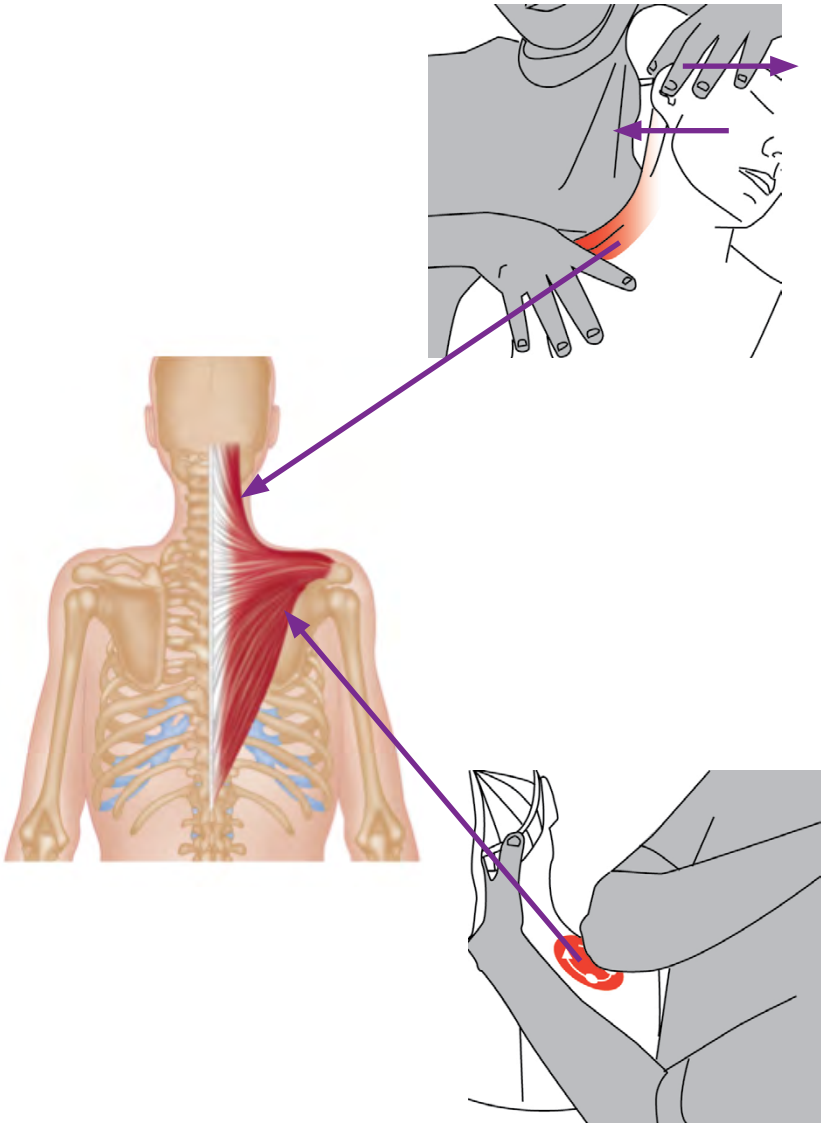
Splenius Cervicis - Treatment



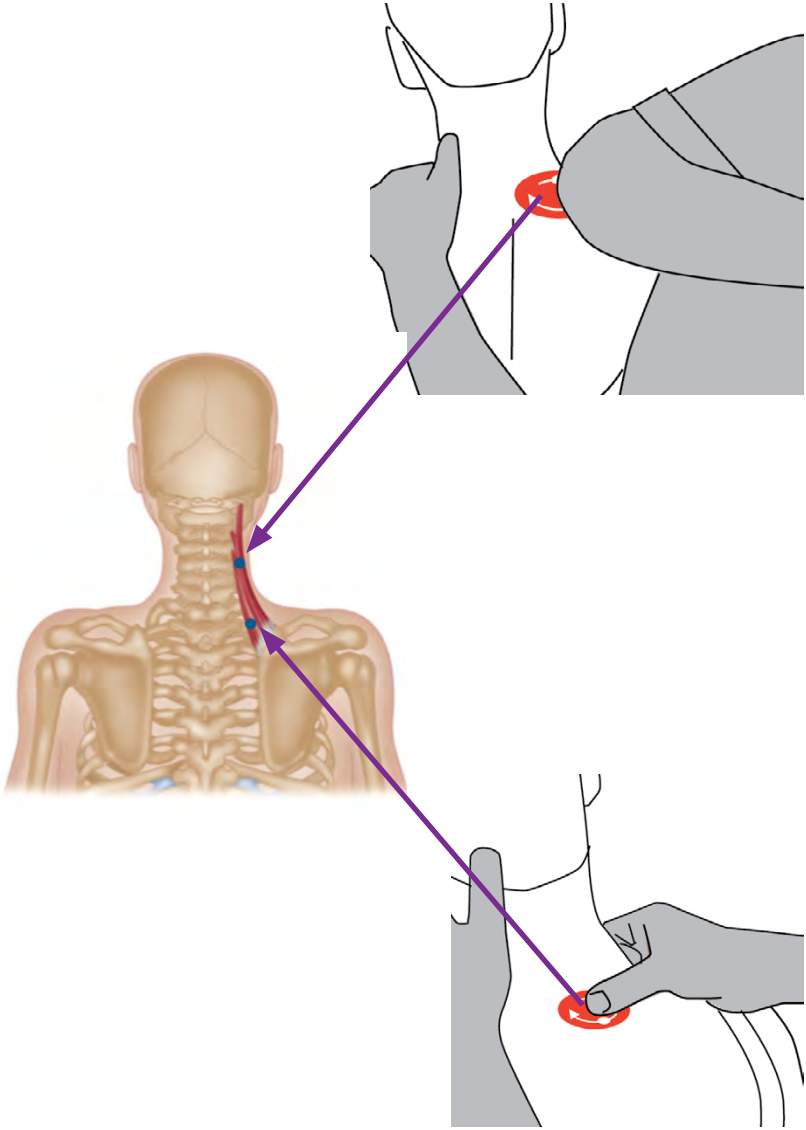
The Suboccipital Muscles – Obliquus Capitis - Treatment



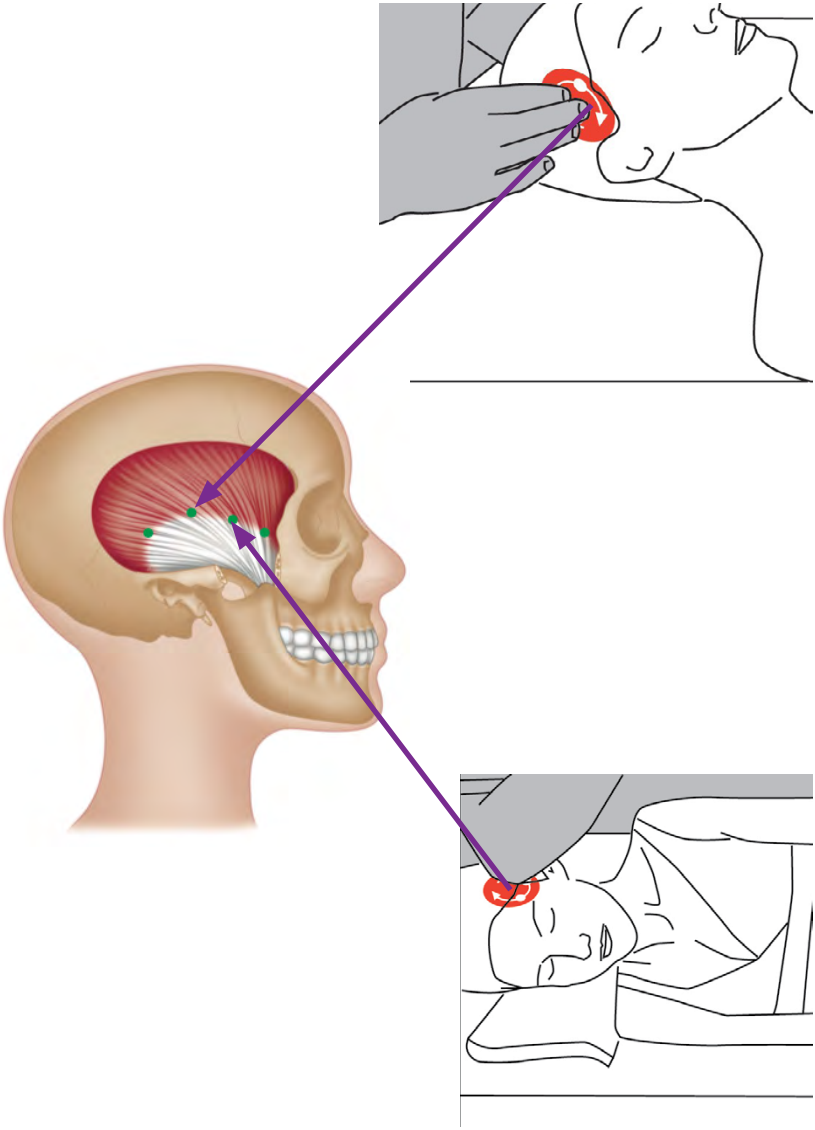
Upper Trapezius - Treatment



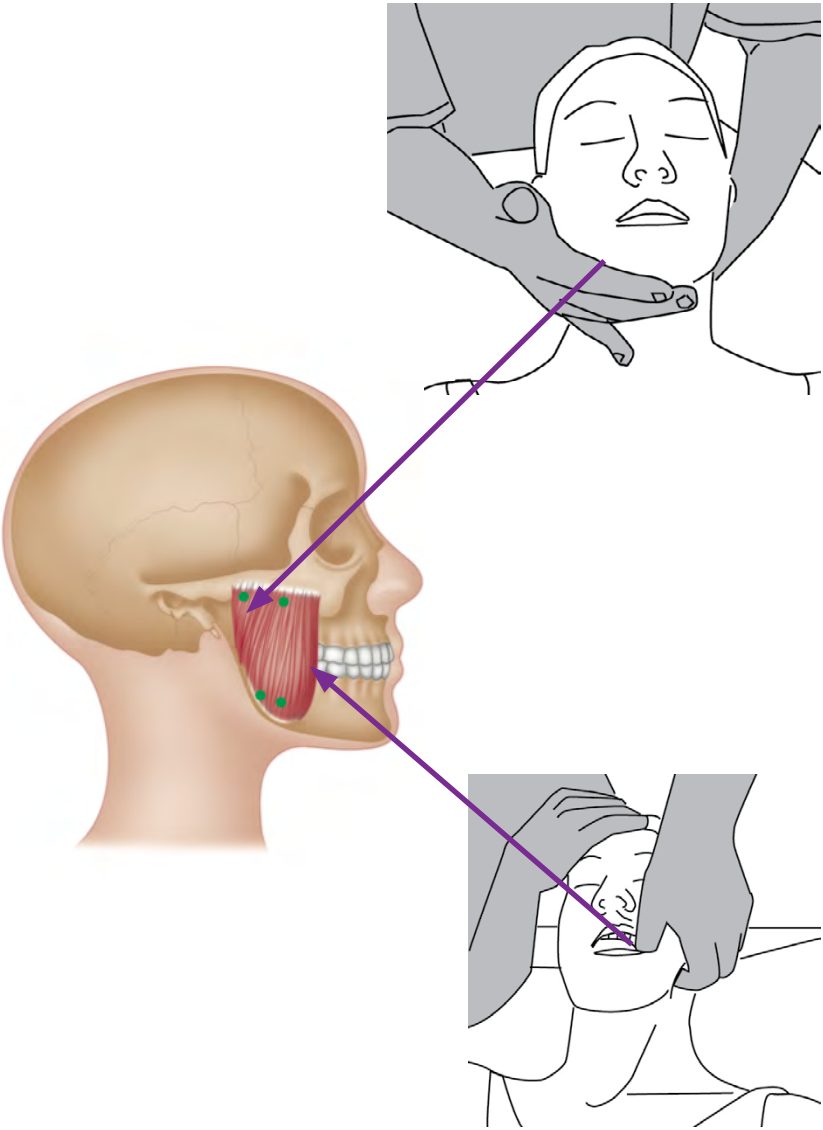
Levator Scapulae - Treatment



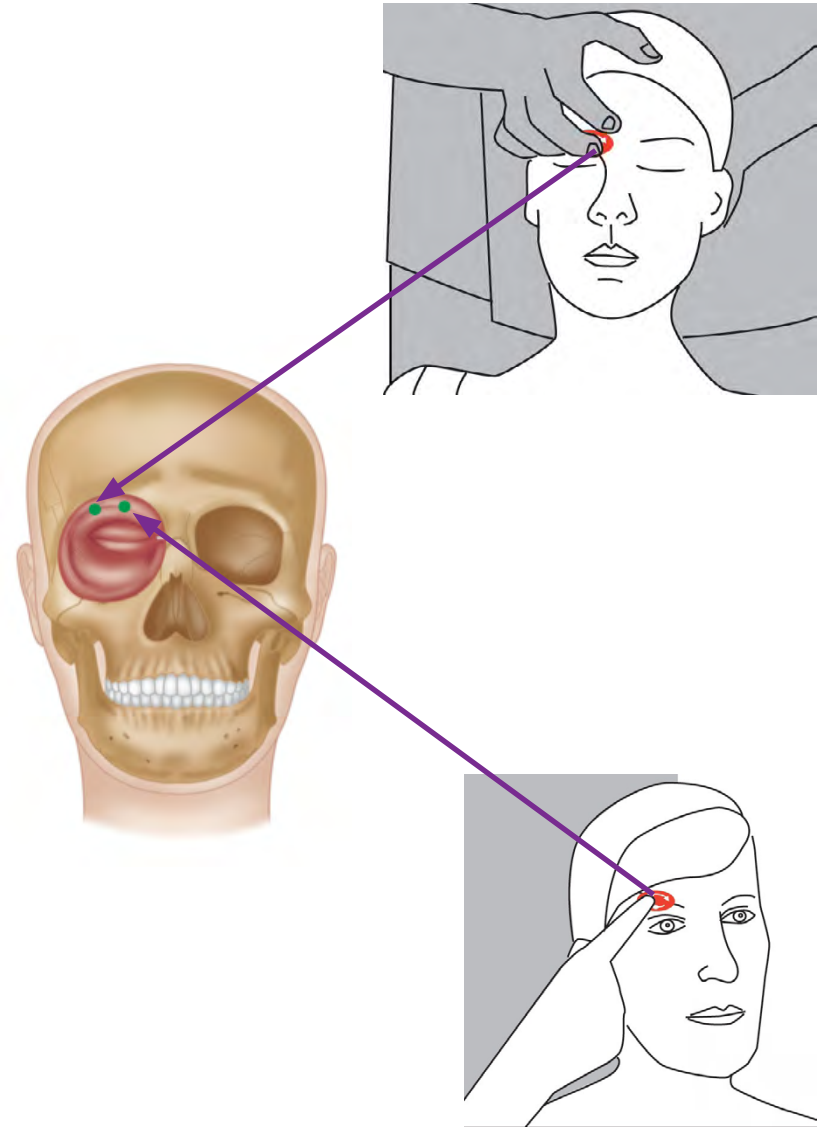
Temporalis - Treatment



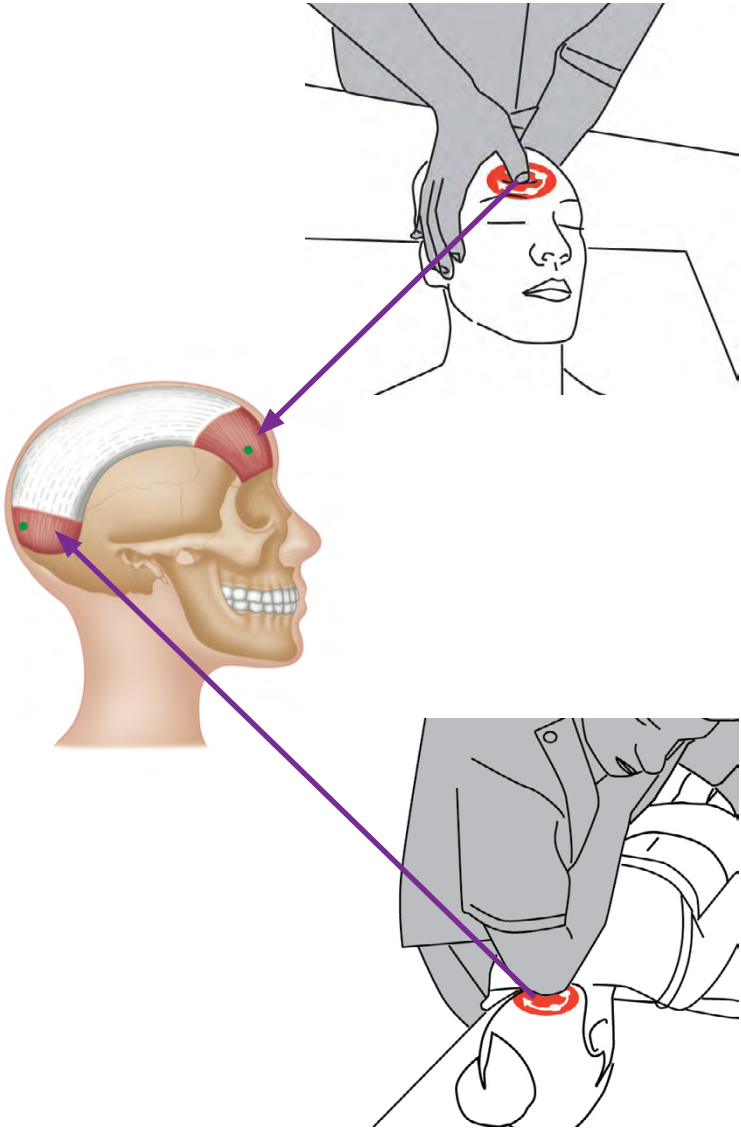
Masseter - Treatment



Orbicularis Oculi - Treatment



Occipitofrontalis - Treatment



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, 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-coordination
- 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

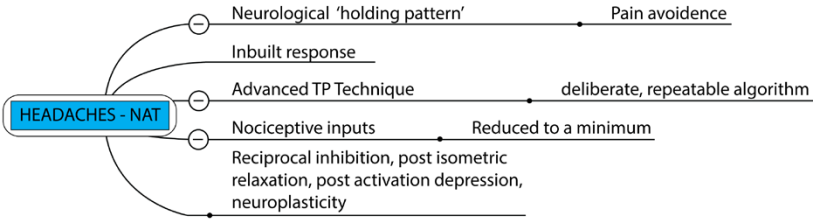
teams in the 2012 London Olympics. Evidence for this phenomenon has been supported by research at Addenbrooke's Hospital in Cambridge, UK (Weis et al. 2003)[53]. Patients with long-standing shoulder pain and weakness treated with NAT demonstrated a significant improvement in active range of motion ($P<0.002$) 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 headaches

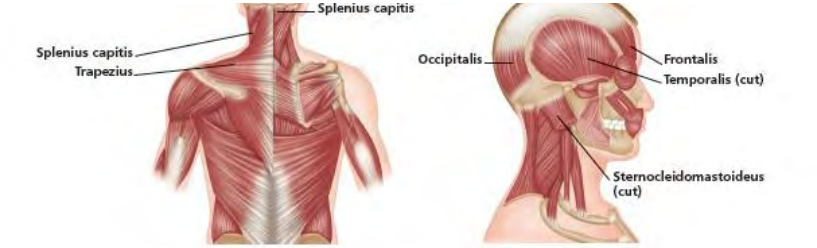
The sequence and the depth of pressure are key to getting the most effective results. In terms of depth, the deep stroking massage should be experienced like squeezing toothpaste out of a tube, slow and luxurious stroking in one direction only; this leads to a type of neurological stimulation of the 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 taut bands in the **splenius** as you get near the upper cervical spine; you can pause on it gently and then finish the stroke all the way to the skull. You can use your hands, fingers, or even gently and respectfully, 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 headache, and we sincerely hope you will find it a valuable addition to your existing approaches.



STEP 1 Study the anatomy and direction of the muscle fibers.



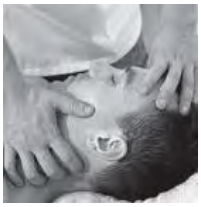
STEP 2

Sitting ICT to SCM (delicately find and press on trigger points). Head should be in a nodding forward position, and rotated toward the side of the pressure. Remember that there are a lot of delicate blood vessels and structures in this area of the neck.

Sitting ICT to trigger points within upper trapezius.

STEP 3 Massage area generously.

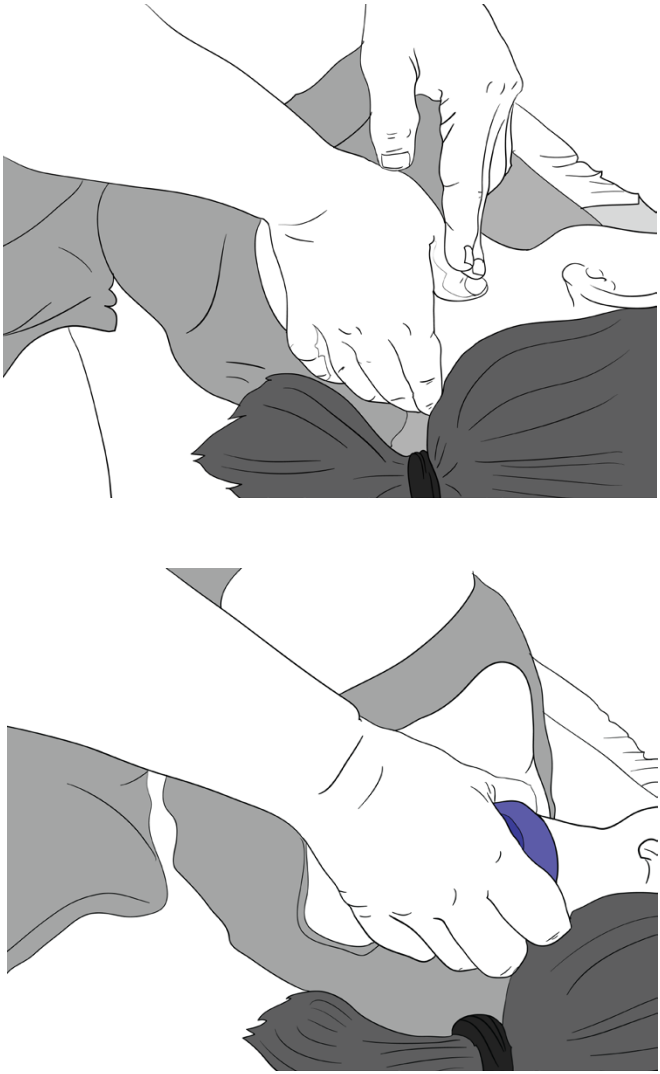
STEP 4 Supine ICT to:
cervical erector spinae and temporalis.



STEP 5
With patient still supine, finish with ICT on posterior points of epicranius (occipitofrontalis).

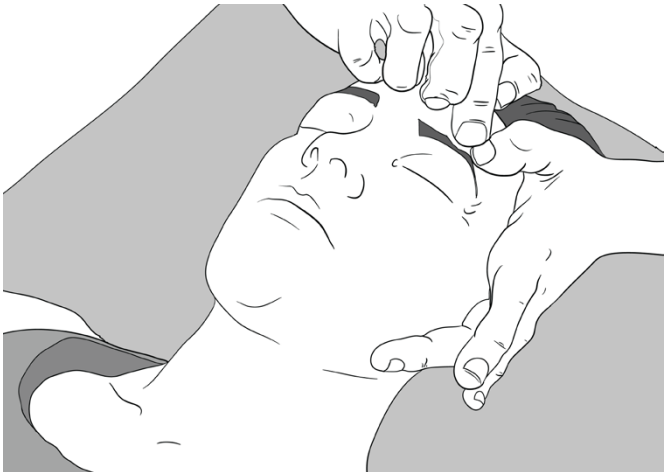
NAT side-lying modification for CGH

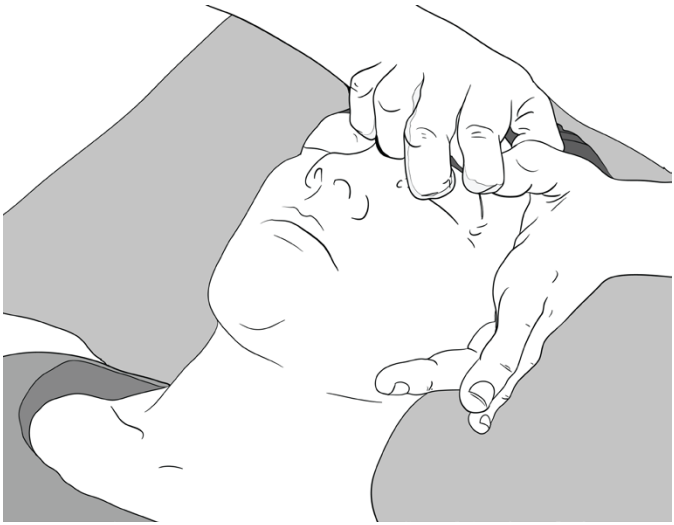
Adding ICT to the articular pillar of the neck – **splenius capitis and cervicis** and holding to wait for a change. Alternatively, you can use a pressure tool or even an elbow.



NAT modification for migraine and sinus headaches

Add ICT to orbicularis oculi





Advice and Exercises

Exercise and headaches

Regular exercise can reduce the frequency and intensity of headaches and migraines. When one exercises, the body releases endorphins, which are the body's natural painkillers. Exercise reduces stress and helps individuals to sleep at night. Stress and inadequate sleep are two migraine triggers. However, it is worth noting that some people may get headaches or migraines when they exercise. One possible reason for this is that a part of the physical reaction may be the elevation of blood pressure. This is not a reason to avoid exercise. Instead, headache and migraine patients need a plan for preventing headaches or migraines when they exercise; this includes warming up and advice on eating an hour or so before exercise, as well as adequate hydration.

The President's Council on Physical Fitness recommends a minimum of 150 minutes of exercise per week. This can be broken down into five sessions of 30 minutes. Alternatively, this goal can be achieved by recommending 10 minutes of continuous exercise for three times per day.

Ideally, an exercise program should include elements designed to improve each of these components:

- Cardio-respiratory endurance
- Muscular strength and endurance
- Flexibility

However...

Trigger points make their host muscles shorter, fatter, and less efficient. Exercise can sometimes be counterproductive if they are part of a holding pattern. We would therefore recommend that patients refrain from any fitness program that requires the muscles to work harder. Asking the

muscles to work harder will increase the potential to develop more trigger points and pain. Once the pain or holding pattern has gone, it's definitely time to start exercising again.

Stretching and trigger points

A muscle fiber that has been contracted due to trigger points has 'muscle memory' and will want to return to the contracted condition if it is not 'retrained'. To retrain the muscle, it is advisable to recommend to your patient that frequent stretching (up to 6 times daily) is beneficial, starting after the first treatment. In trigger point release therapy, the goal of stretching is to retrain the muscle that was treated. Participating in the home stretch program is **50%** of this therapy's success. Stretching has also been demonstrated to have a 'neuroplastic' effect, which may also help the brain move the muscles back towards homeostasis.

STRETCHING

Stretch 1: Butterfly

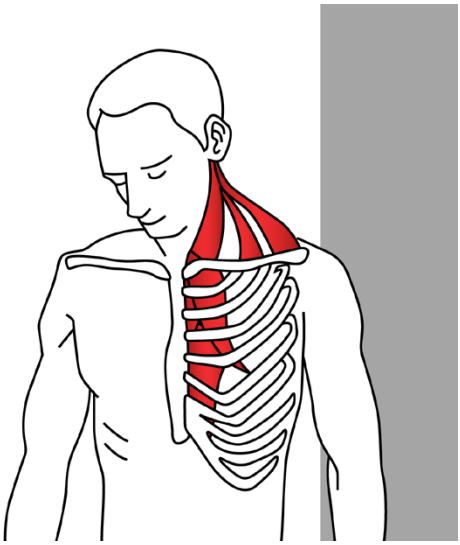
Technique

- The patient should place their hands on the side of their head
- Elbows facing forward
- Push their elbows backward - towards the wall (or bed if they do this laying down)
- Go as far as they can to get a nice, easy stretch (may get a few tingles...which is OK)
- Return elbows forward again. Do not hold

How Often?

30 times, daily

Stretch 2:



Technique

- The patient should assume the correct postural position
- Start with their head centered and gently bring down their right ear towards their right shoulder
- At this point, slightly rotate the neck to the side
- A normal stretch of the muscles on the side of the neck may be experienced. However, the exercise should be pain free

How Often?

Hold for 30 to 50 seconds 3 times on each side, twice daily

Stretch 3:

Technique

- The patient should turn their head 45 degrees to the right
- Reach over the top of their head with their right hand and cradle the back of their head with their fingers
- Gently pull their head toward their right shoulder, and hold for a count of 20-30
- Repeat on the left side using their left hand

How Often?

Repeat 3 times each side, twice daily

Stretch 4:

Technique

- The patient should position their head in a normal posture
- Face forward with their neck in a neutral position
- Gently drop their head back and look towards the ceiling

How Often?

Hold for 20 to 50 seconds and repeat 3 times with breaks in between to prevent dizziness, twice daily

EXERCISES

Exercise 1: Isometric neck strengthening

Technique

- The patient should assume the correct postural position and gently raise the back of their head
- Place their hand on their right cheek
- Without moving their head, turn their eyes to the right and gently push their head into their hands as if to look over their shoulder
- While performing this exercise no actual movement occurs
- Do the exercises smoothly and gently and try using only 10-20% of their maximal effort

How Often?

Hold for 5 seconds and repeat 10 times on each side, twice daily

Exercise 2: Chin tucks

Technique

- The patient should sit up straight in a chair. Sit to the front, not against the back of the chair
- Sitting up will help to strengthen the muscles needed for good posture
- Think of lifting the crown of their head to the ceiling
- Be careful not to tip the head back
- Their chin should be parallel to the floor
- Without tipping the head in any direction, pull their chin and head straight back
- They will feel a stretch in the back of their neck

How Often?

8 to 12 repetitions, 3 times, twice daily

Exercise 3: Rhomboid strengthening

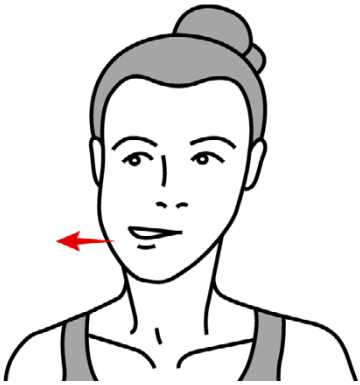
Technique

- Begin by having the patient lie down on their stomach with their arms by their side
- The patient should tighten their rhomboids by squeezing their shoulder blades together
- Slowly lift their arms and chest off the ground, keeping their neck straight
- Hold for 2 seconds at the top of the movement and then slowly return to the starting position
- The exercise may be performed with palms facing up or down

How Often?

Perform 3 sets of 10 repetitions, once a day, provided the exercise is pain free

Exercise 4:



Passive exercises should be done without causing pain. These exercises particularly help to maintain good blood flow to the muscle and joint. This in turn prevents the muscle from being stiff and disused.

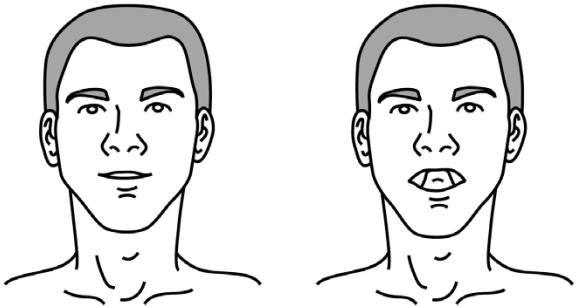
Technique

- The patient should stand in front of a mirror with head facing forward
- Gently slide their jaw from left to right keeping the movement controlled and smooth

How Often?

Repeat 10 times , six times a day

Exercise 5:



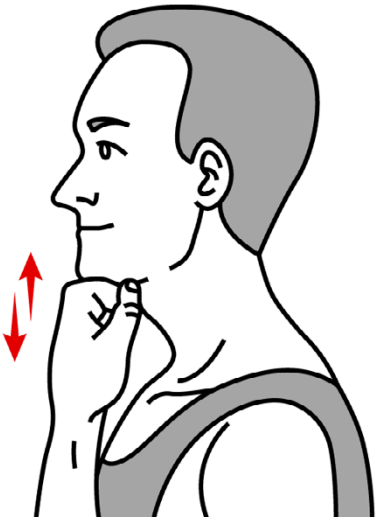
Technique

- The patient should place their tongue on the top of their palate
- Keeping the tongue on the palate, proceed to opening and closing the jaw
- Whilst doing the movements keep tongue placed on the palate/roof of the mouth
- Make sure that they are sitting up straight in the correct posture
- Prevent slouching and keep their stomach muscles tight

How Often?

Repeat 10 times, 3 times a day

Exercise 6:



Technique

- The patient should make a fist with one hand, rest their chin on their fist
- Slowly open their jaw and with their fist provide counter pressure in order to prevent jaw from opening
- The isometric muscle contraction should provide relaxation for the jaw bone (mandible)
- Do not allow the jaw to click during this exercise

How Often?

Hold for ten seconds, repeat ten times 3 times a day

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. I 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 out of the acute phase usually takes 3-5 sessions which should be performed between five to 10 days apart. After this, we usually recommend maintenance sessions anywhere from 2-12 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, wellbeing, and lifestyle factors.

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