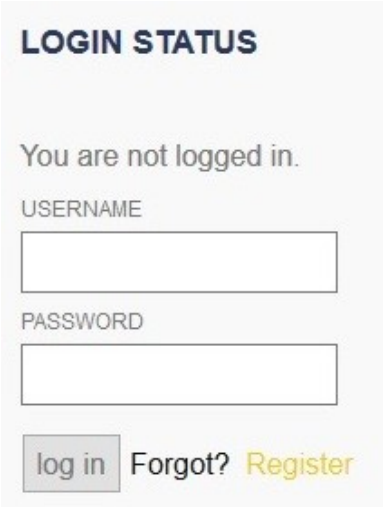
IMT1 PRE-READING

We appreciate your decision to advance your career in manual therapy with the Integrative Manual Therapy Series. Prior to each course module, there is a pre-reading component. The purpose of the pre-reading is to prepare you with background information so that the majority of the course time can be spent learning practical skills. As people attending this module have different educational backgrounds, for some this may be a lot of new material while for others it may be a review. An accompanying online multiple choice quiz based solely on the provided material should be attempted prior to participating on the course (see details below). The quiz is designed to ensure a minimum level of understanding so that all participants have an adequate knowledge base that can be built upon during the course. There is no set passing grade for this quiz but participants are encouraged to attempt it to achieve full accreditation for the module and it may also be necessary to unlock additional online media and to receive a certificate of completion following the course.

Click here (http://www.manualtherapyinstitute.com/quiz/imt1-pre-reading-quiz) to access the online quiz. This will bring you to the page with this message:

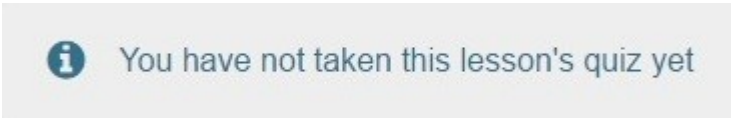
Please ignore this message and scroll down and in the right sided panel you will see the login status widget:



Please **login** with your username and password.

(If you've forgotten your username or password you can use the Forgot? link to reset it).

Having logged in, you will be brought to a screen that reads:



Then choose your answers and finish by clicking the green button:

When you've completed the quiz it will give you immediate feedback how you did and send you an email for your records.

While it is suggested that you take the course modules sequentially (in order), any module after IMT1 can be taken in any order. As the course time in each module is largely practical, you will be required to participate in the subjective and objective assessments and treatment techniques. For some people this may be difficult as we may cover some sensitive issues and regions of the body. Please let us know if at any time during the course you feel that it is inappropriate for you. Please also remember to bring loose fitting attire to make it easier to palpate.

Please be aware that image recording devices are not permitted during this course due to the above stated issues. We do permit audio recording for the majority of the course but you may be requested to turn it off during some sections of the course.

Finally, the course especially relates to chronic pain. If you do suffer with chronic pain, please email me prior to the course so we can include you in the course structure (and help you with your problem). Please also familarize yourself with the Consent To Participate In Manual Therapy Training Policy prior to the course. If you have any further questions please email me on haydn@manualtherapyinstitute.com

We thank you for your trust and commitment and we look forward to working with you during the series.

With regards,

Haydn Gambling

MODELS OF TREATMENT

There are many different models of treatment available to the practitioner. The main models covered in this series (and will be discussed in greater detail in the relevant module) include:

Mechanical Adaptive model

A biomechanical approach to the musculoskeletal system addresses the osseous skeleton as a series of building blocks stacked on top of one another connected by ligaments and fascia and moved by muscles. Alterations in alignment of the blocks changes the apposition of joints, length of associated ligaments and fascia and function and tone of connected muscles. Adaptation occurs within the system to maintain a dynamic functional balance. Treatment is directed at restoring optimal alignment and balance in the musculoskeletal system.

Myofascial model

The myofascial model looks at the interconnected web of fascia that links all cells and tissues within the body. The model looks at the fascia playing a role in the bodies structural integrity, fluid dynamic and immune response system. Central to this model is the concept of tensegrity that distributes force evenly throughout the body. Treatment is directed at balancing tension in the fascia throughout the body.

Fluid Dynamic model

The cardiopulmonary system is the main driving force behind the flow of blood, lymph and intercellular fluid. The diaphragms function to pump the fluids to deliver oxygen, eliminate waste and maintain the correct pH for cellular chemical reactions. Similarly, the craniosacral system is the mechanism driving the flow of cerebrospinal fluid that creates a healthy environment for the nervous system. Treatment is directed at altering structural adaptations and adverse tissue tension that is impeding optimal fluid motion.

Neuroendocrine model

The neurological system is central to all the treatment modalities explored in this course. It lies at the very core of the pain experience. The autonomic nervous system, consisting of the sympathetic, parasympathetic and enteric divisions, connects all the tissues of the body to the CNS and is vital to the maintenance of homeostasis. It communicates with the CNS via axons and neurotransmitters including hormones. Treatment is directed at changing autonomic imbalance that may have a significant effect on dysfunction and pain.

TREATMENT TECHNIQUES

The main treatment techniques addressing these models of treatment in this series (and discussed in greater detail during the relevant modules) include:

Strain and Counterstrain (SCS) (covered in all four modules)

An indirect technique to treat somatic dysfunction based on Korrs [1] facilitated segment concept. Korr proposed that disturbed afferent input into a spinal cord segment would facilitate the spinal interneurons and produce increased motor activity of segmentally innervated muscles. Jones [2] described his technique as an approach that relieved pain by the "reduction and arrest of the continuing inappropriate proprioceptor activity". This is accomplished by markedly shortening the facilitated muscle containing the malfunctioning golgi tendon organ by applying mild strain to its antagonists (counterstrain). Key to this concept is the identification of tender points relating to each specific somatic dysfunction and joint positioning to find its maximum position of ease (mobile point) to create a neurological change in the tissues. A recent evolution of the technique has seen SCS utilised for non-musculoskeletal dysfunction which has necessitated a new physiological paradigm proposed by Tuckey [3] who advocates a protective reflex within the fascia is often the origin of dysfunction (read on for a more detailed review).

Myofascial Release (MFR) (covered in detail in modules 3 & 4)

A direct or indirect technique treating somatic dysfunction specifically related to the fascia. It applies the principles of biomechanical loading of the fascia to increase or decrease tissue tension. A "release" is thought to result from tissue hysteresis and stimulation of the fascial mechanoreceptors that results in neuroreflexive inhibition of innervated tissue at the CNS level [4].

Visceral Manipulation (VM) (covered in detail in module 3)

A direct or indirect technique treating visceral dysfunction. In this concept the visceral organs are viewed to move on a physiological axis and are restrained by their connective tissuesmuch like joints in the musculoskeletal system. Similar to myofascial release, treatment by connective tissue hysteresis and stimulation of fascially located mechanoreceptors can create a neuroreflexive inhibition of hypertonic tissue maintaining a dysfunction. This results in improved mobility and functioning of the organs and in the musculoskeletal system connected to the viscera via the sympathetic nervous system [5] .

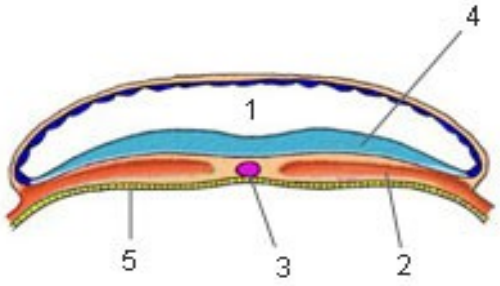
Cranial (Craniosacral) Techniques (CST) (covered in detail in module 4)

A direct or indirect technique treating dysfunction in the craniosacral system. Based on the concept that the cranium is a mobile structure that moves in a rhythmic way to pump cerebrospinal fluid which helps to provide a healthy environment for the nervous system [6]. Dysfunction in this motion has effects on the neuroendocrine, visceral and musculoskeletal systems. Treatment is directed at identifying dysfunctions and treating them with manual techniques.

EMBRYOLOGY

The purpose of including embyology in the prereading is to remind participants that we start from related cell layers and we maintain those relationships throughout life. During the third week of embryonic development the process of gastrulation converts the bilaminar disc into a trilaminar germ disc consisting of ectoderm, intraembryonic mesoderm and endoderm. The midline (axial) mesoderm begins the formation of the notochord while the remaining mesoderm forms the paraxial, intermediate and lateral plate mesoderm [7], [8].

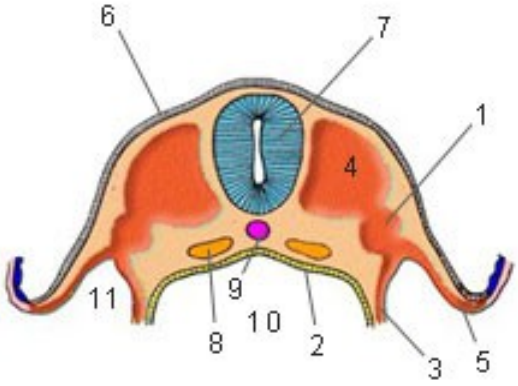
Figure 1 Trilaminar Disc



1 . Amniotic cavity
2 . Mesoderm
3 . Notochord
4 . Ectoderm
5 . Endoderm

Adapted from reference [8]

Figure 2 Intraembryonic mesoderm



1. Intermediate mesoderm
2. Endoderm
3. Visceral mesoderm
4. Paraxial mesoderm
5. Somatic mesoderm
6. Surface ectoderm
7. Neural tube
8. Dorsal aorta
9. Notochord
10. Yolk sac
11. Intraembryonic coelom

Adaped from reference [8]

The **paraxial** mesoderm begins to form somitomeres which will later divide into sclerotomes, myotomes and dermatomes. These elements will give rise to the axial skeleton, skeletal musculature and dermis.

The **intermediate** mesoderm connects the other mesoderm and forms the urogenital ridge which will be involved in the formation of the gonads and kidneys.

The **lateral** mesoderm is split by the intraembryonic coelom into the somatic mesoderm (somatopleure) and the visceral mesoderm (splanchnopleure). The somatic mesoderm will form the appendicular skeleton while the visceral mesoderm will form the visceral organs.

Ectoderm layer gives rise to:

- the central nervous system
- the peripheral nervous system
- the sensory epithelium of the ear, nose and eye
- the epidermis, hair and nails
- the subcutaneous, mammary and pituitary gland
- the enamel of teeth

Neural crest cells give rise to the cells of the ganglia and ensheathing cells of the peripheral nervous system, pigment cells of the dermis, muscles, connective tissue and bone of the branchial arches, suprarenal medulla and meninges.

Endoderm layer gives rise to the epithelial lining of the:

- gastrointestinal system
- respiratory system
- urinary bladder and urethra
- tympanic cavity and auditory tube
- the parenchyme of the tonsils, thyroid, parathyroid, thymus, liver and pancreas

PHYSIOLOGY

The purpose of including physiology in the pre-reading is to remind participants of the inter-connectiveness of the nervous system with particular reference to pain perception.

SENSORY RECEPTORS

The central nervous system is kept continually informed of the ever changing external and internal environment of the body. Receptors detect and transmit sensory information from the periphery to the CNS via afferent nerve fibres of the peripheral nervous system. The receptors report on a wide variety information including changes in temperature, pressure, touch, sound, light, taste, smell, body and limb movements, blood pressure and chemistry and nociception.

ION CHANNELS

Ion channels are protein subunits embedded in receptor membranes. They are gated and can be either open or closed. Channels open in response to various stimuli to allow the passage of ions. Some of the ions that are able to cross the membrane are sodium (Na^+), Potassium (K^+), Chloride (Cl^-) and Calcium (Ca^{2+}). The flow of ions changes the charge within the receptor and its associated neurone generating an action potential that is transmitted towards the CNS. Most ion channels are opened by the presence of chemicals (ligand gated) particularly those chemical associated with inflammation. Ion channels will also open to electrical current (voltage gated) and stretch or pressure (mechanically gated). Ion channels are in a constant state of insertion and degradation [9]. This may allow a self regulatory mechanism that can increase the numbers of certain types of ion channels to increase the sensitivity of the system.

NOCICEPTION

Receptors which respond primarily to injurious or painful stimulation are called **nociceptors**. Within this category are four subgroups: mechanonociceptors, mechano-heat nociceptors, mechano-cold nociceptors, and poly modal nociceptors [10]. The receptors transmit information to the CNS via type A delta (myelinated) and type C (unmyelinated) afferent fibres. Nociceptors can be found in the skin, subcutaneous and adipose tissue, viscera, walls of intramuscular arteries and blood vessels supplying the spinal joints, sacroiliac joints and vertebral cancellous bone, walls of epidural and paravertebral veins, fibrous capsules of apophyseal and sacroiliac joints, spinal ligaments, periosteum covering vertebral bodies and arches (and attached fascia, tendons and aponeurosis), the outer third of the annulus fibrosis, dura mater and epidural fibro-adipose tissue [11], [12], .

Nociceptors in the Skin [10]

Cutaneous mechanonociceptors are associated with type A delta fibers and respond to high shearing force. Cutaneous mechano-heat nociceptors respond to noxious levels of mechanical stimulation and heat in excess of 43°C. They are associated with type A delta fibers. Cutaneous mechano-cold nociceptors are associated with type C fibers. They are particularly adept at responding to noxious levels of mechanical stimulation and temperatures below 10°. Polymodal nociceptors respond to noxious levels of mechanical, heat, and chemical stimulation and are associated with type C fibers.

Nociceptors in Muscle, Joints, Viscera and Fascia [10], [13]

Muscle Nociceptors responding to strong pressure and excessive muscle stretch are associated with A delta fibers. Nociceptors responding to pressure, temperature extremes, and anoxia are associated with type C fibers.

Joint Nociceptors responding to joint overextension are associated with type A delta fibres .

Viscera Nociceptors are probably not located in the parenchyma of the internal organs themselves but are found in the peritoneal surfaces and ligaments, pleural membranes, dura mater, and the walls of blood vessels. They are associated with type C fibres.

Fascia is embedded with type III and IV peripheral neurones (free nerve endings) which have both nociceptive and mechanoreceptive capabilities. As these fascial receptors are embedded in all structures, they are most likely associated with type A and C fibres.

THE SENSORY PATHWAYS

The sensory pathways from the periphery to the CNS can be delineated into five categories. With relation to nociception, the first three may be most important.

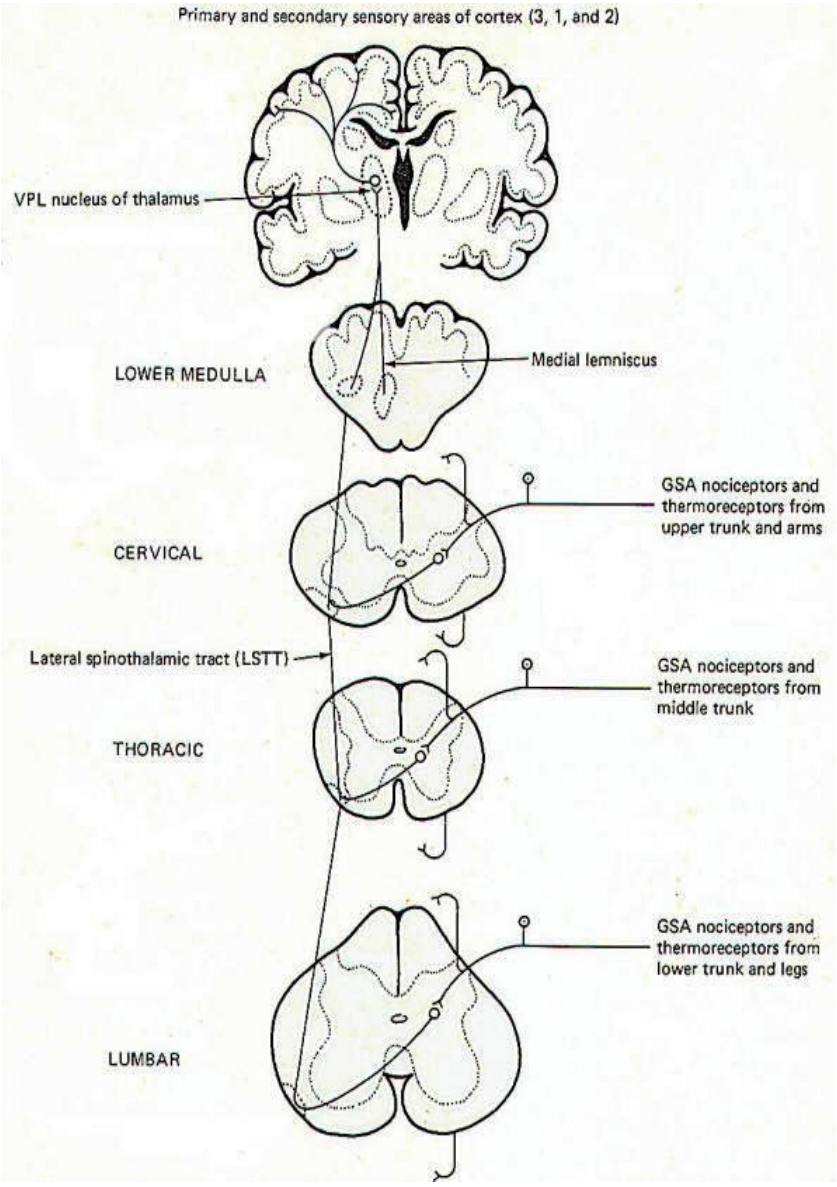
- General Somatic Afferents from the body
- General Somatic Afferents from the face
- General Visceral Afferents
- Special Somatic Afferents (hearing, balance and vision)
- Special Visceral Afferents (taste and smell)

GENERAL SOMATIC AFFERENT (GSA) PATHWAYS FROM THE BODY

Pain from general somatic nociceptors is conducted over small-diameter (type A delta and type C) GSA fibers of the spinal nerves into the posterior horn of the spinal cord gray matter. These are monopolar neurons with cell bodies in the posterior root ganglia. After entering the cord, the fibers pass up or down in the dorsolateral tract, before finally synapsing in laminae III and IV.

Second-order neurons from these synapses cross over to the opposite side of the cord in the anterior white commissure, where they turn upward as the lateral spinothalamic tract (LSTT). At higher pontine levels this tract comes to lie close to the medial lemniscus, with which it travels to the ventral posterior lateral nucleus (VPL) of the thalamus. Some fibers of this tract don't enter the thalamus but end instead in the brainstem reticular formation. After synapsing in the thalamus, third-order neurons enter the posterior third of the internal capsule, pass through the corona radiata, and terminate in the primary and secondary sensory areas of the parietal lobe cortex.

FIGURE 3: GSA BODY PATHWAYS – image from [14]



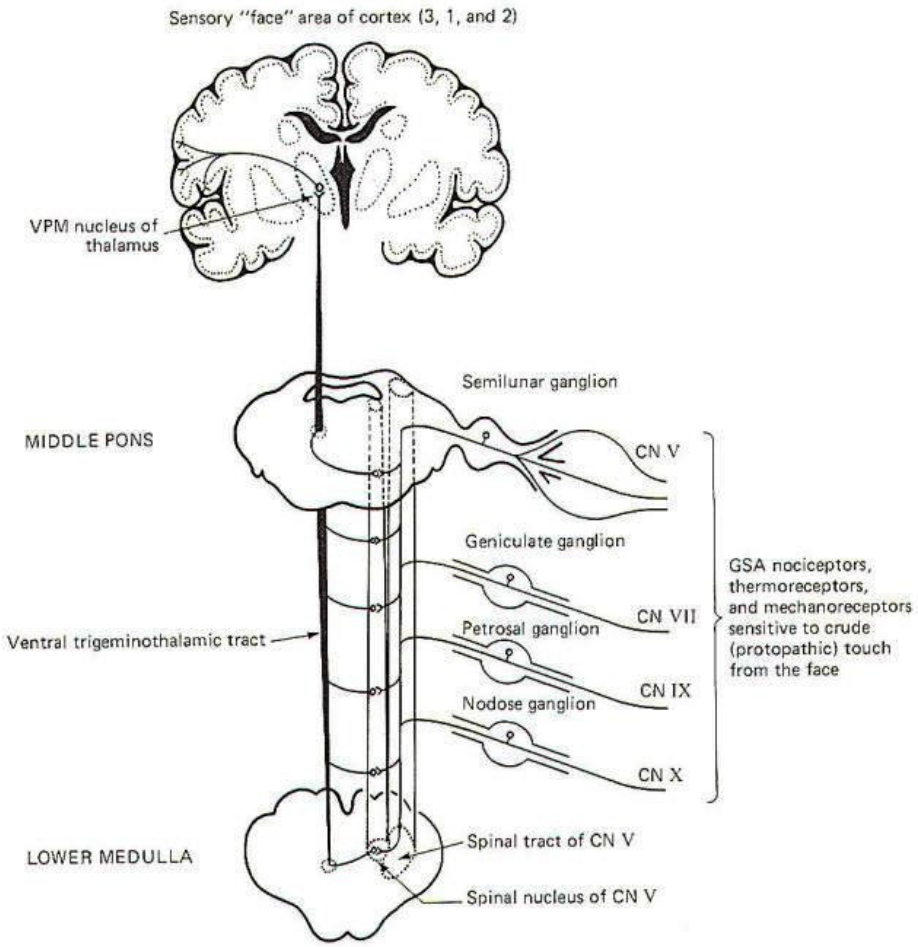
FAST AND SLOW PAIN [14]

Fast pain, often called sharp or pricking pain, is usually conducted to the CNS over type A delta fibers passing through the lateral spinothalamic tract and going directly to the VPL of the thalamus on the contralateral side. From here third-order fibers project to the cerebral cortex where they are somatotopically organized and sharply localized. Slow pain, often called burning pain, is conducted to the CNS over smaller-diameter type C fibers. After entering the cord these fibers stimulate lateral spinothalamic tract neurons which send collaterals into the brainstem reticular formation. Fibers from the reticular formation diffusely project to the thalamus and hypothalamus perhaps giving rise to the emotional component of pain. Pain signals following this route are poorly localized.

GENERAL SOMATIC AFFERENT (GSA) PATHWAYS FROM THE FACE [14]

General somatic nociceptors from the face conduct signals to the brainstem over GSA fibers of cranial nerves V, VII, IX, and X. These monopolar neurons have cell bodies in the semilunar, geniculate, petrosal, and nodose ganglia respectively. The neurons enter the spinal tract of lamina V, where they descend through the brainstem for a short distance before terminating in the spinal nucleus of V. Second-order neurons then cross over the opposite side of the brainstem to enter the ventral trigeminothalamic tract, where they ascend to the VPM of the thalamus. Finally, third-order neurons project to the "face" area of the cerebral cortex.

FIGURE 4: GSA FACE PATHWAYS – image from [14]

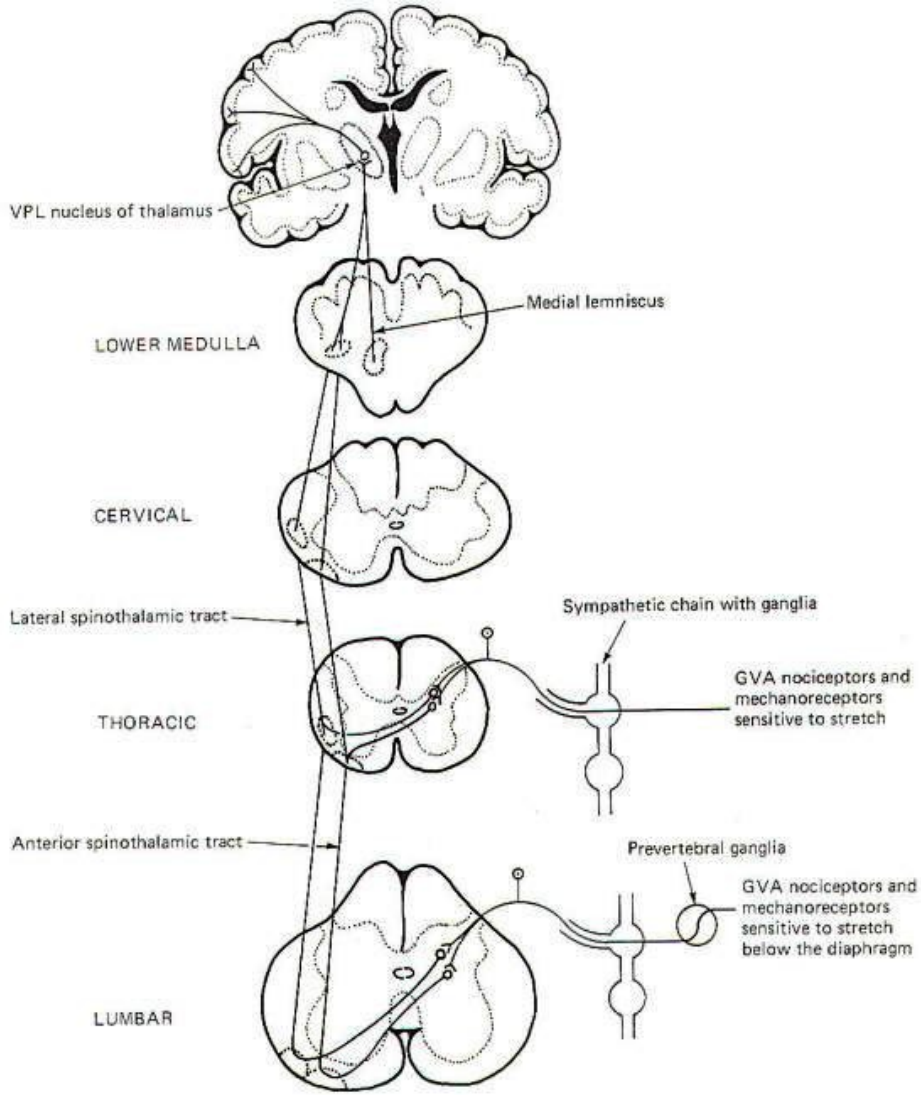


GENERAL VISCERAL AFFERENT (GVA) PATHWAYS [14]

Visceral nociceptors are located in peritoneal surfaces, pleural membranes, the dura mater, walls of arteries, and the walls of the GI tube. Nociceptors in the walls of the GI tube are particularly sensitive to stretch and overdistension.

General visceral nociceptors conduct signals into the spinal cord over the monopolar neurons of the posterior root ganglia and terminate in laminae III and IV of the posterior horn. Their peripheral processes reach the visceral receptors via the gray rami communicantes and ganglia of the sympathetic chain. Second-order neurons from the posterior horn cross in the anterior white commissure and ascend to the thalamus in the anterior and lateral spinothalamic tracts, Projections from the VPL of the thalamus relay signals to the sensory cortex.

FIGURE 5: GVA PATHWAYS – image from [14]



The localization of visceral pain is relatively poor as visceral pain is often referred by the brain to some area on the surface of the body. The mechanism for referred visceral pain may result from the close proximity in the posterior horn of the GVA pain fibers and GSA fibers from the body surface. This is supported by the fact that pain from a visceral origin is referred to a dermatome with which it shares the same posterior root.

FIGURE 6 : VISCERAL PAIN PATHWAYS – image from [14]

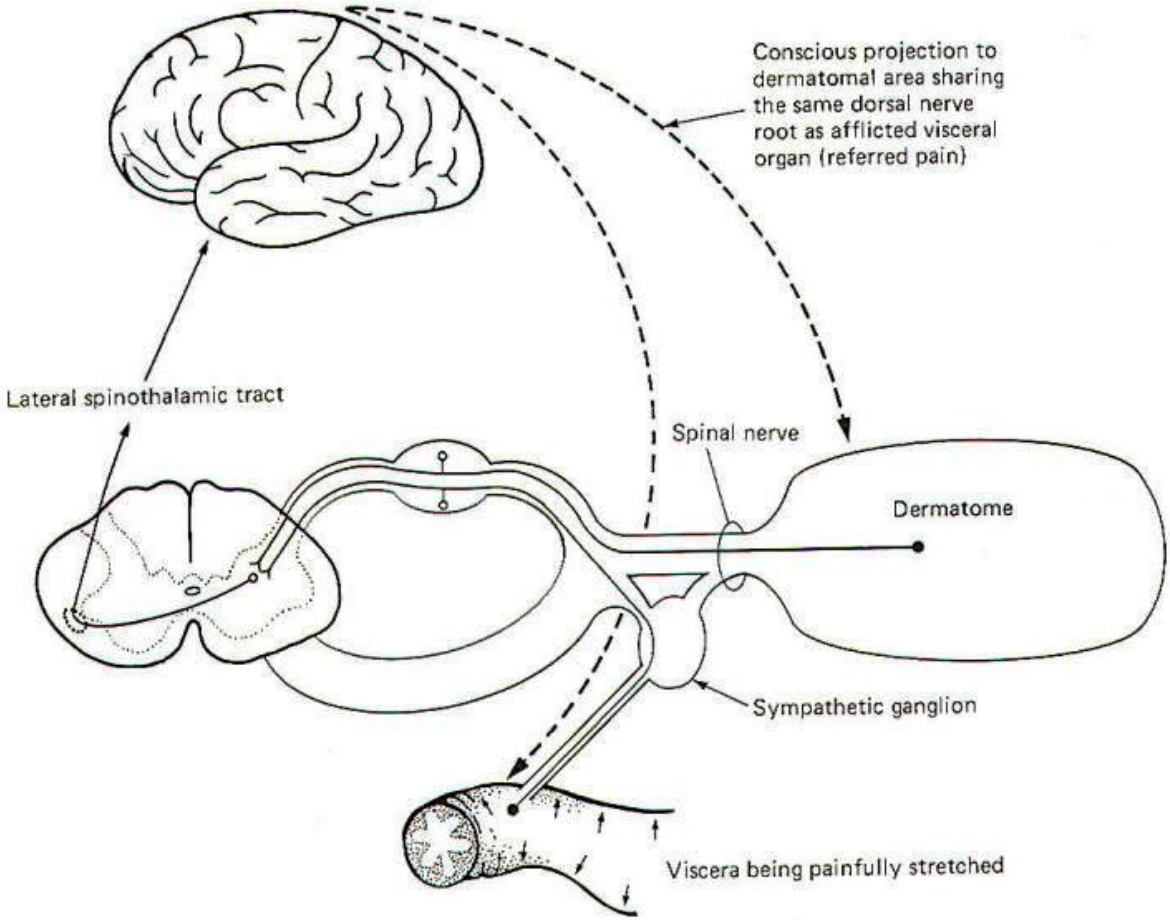


FIGURE 7 : MUSCULOKELETAL PAIN PATHWAYS – image from [15]

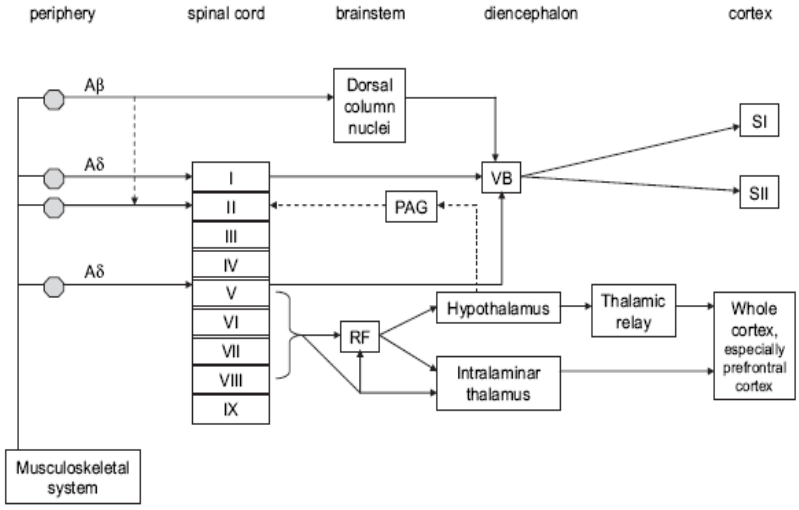
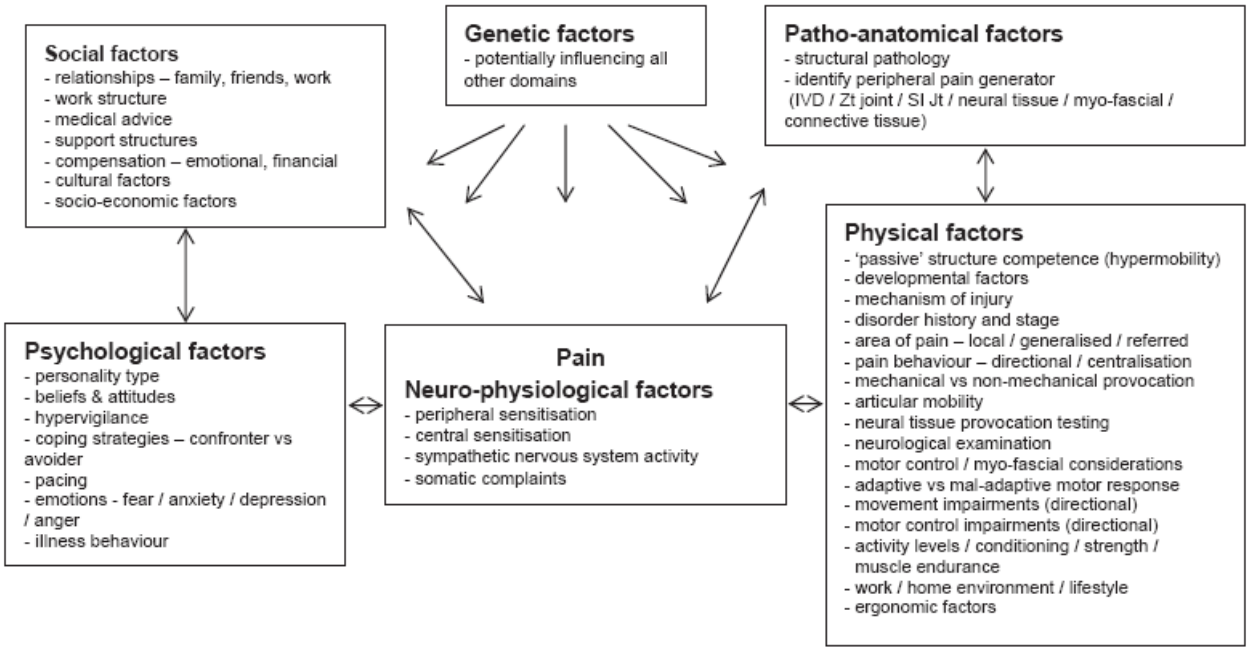


Fig. 1. Simplified block diagram displaying nociceptive processing in the nervous system. Input from the musculoskeletal system is transferred through Aδ and C nociceptive fibres and low threshold Aβ fibres. Dashed lines represent inhibiting (mainly descending) pathways, the ultimate effect of which is inhibitory in lamina II of the spinal cord. Numbers I–IX represent the corresponding laminae of the spinal cord. RF, reticular formation; PAG, periaqueductal grey matter; VB, ventrobasal nuclear complex of the thalamus; SI, primary somatosensory cortex; SII, secondary somatosensory cortex. Modified from: Wells et al. (1996, Fig. 8.3).

THE BIOPSYCHOSOCIAL PAIN MODEL

The biopsychosocial pain model is gaining recognition for its application in physiotherapy [16]. The model considers the body, mind and environment as inter-related factors contributing to the experience of pain. Much of this course is directed at the biological component of this model seeking to understand how the cause of pain stems from dysfunction in an individual's body. The psychological component such as emotions, beliefs and attitudes and the social component such as socioeconomic status, culture and relationships need to considered and will be screened for in the subjective assessment. Where there are many psychosocial components to your patients pain experience, other forms of management such as cognitive behavioural therapy may need to be implemented in addition to manual therapy [17]

FIGURE 8 BIOPSYCHOSOCIAL PAIN MODEL – image from [18].



BIOLOGICAL MECHANISMS OF PAIN

Pain can be defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage ". "Pain is an abnormal affective state, an emotional disturbance, that is called into being by the development of mechanical and/or chemical changes in the tissues of the body whose nature and magnitude are such that they give rise to activity in afferent systems within the neuroaxis that are normally quiescent" [19].

A popular definition of *chronic pain*, involving no arbitrarily fixed durations is "pain that extends beyond the expected period of healing" [20].

Pain can be induced through injury of either neural or non-neural tissues. Pain caused by non-neural tissue injury such as joint, muscle or visceral pathology, is referred to as nociceptive pain. Pain caused by neural tissue injury is referred to as neuropathic pain. Psychologic mechanisms can also be involved in the promotion of pain. Portenoy [21] has suggested that pain syndromes are either nociceptive, neuropathic or psychogenic in nature, or a combination of the three.

The pathobiological process of tissue injury and repair is a well known concept where injured tissues go through a phase of inflammation, cell proliferation and repair and then remodelling [22]. During the inflammatory phase the nociceptors are stimulated sending information to the CNS to be perceived as pain. This simple tissue model does not however explain why pain often persists after the healing process has completed, nor why compressed and irritated nerve roots identified on autopsy created no pain why the individual was alive. The phenomenon of phantom limb pain certainly must have a mechanism that doesn't involve the nociceptors.

It is proposed that in addition to the **tissue** origin of pain, pain mechanisms can be related to the **input** into the nervous system, the **processing** in the nervous system and the **output** from the nervous system [23].

The **tissue** mechanism of pain is related to the inflammation and remodelling cycle.

The **input** mechanism is related to nociceptive pain through the peripheral neurogenic pathway.

The **processing** mechanism relates to CNS sensory and psychological processing.

The **output** mechanism involves the ANS, endocrine, immune, motor and pain control pathways.

TISSUE MECHANISMS OF PAIN

With tissue injury, nociceptors in the tissues may be excited by noxious mechanical and chemical stimuli. Tissue injury disrupts the integrity of local structures creating noxious mechanical irritation and permitting the release of various chemical mediators of inflammation and nociception such as histamine, serotonin, prostaglandin and bradykinin. Many of these chemicals can be capable of stimulating and sensitising tissue nociceptors and are often described as the "inflammatory soup" [24].

The mechanism by which the chemical mediators sensitise nociceptors may occur through the interaction of the chemical mediators with ion channels. This process may result in the entry of sodium or calcium ions and the exit of potassium ions creating an action potential spike which depolarises the associated neurone.

Sensitisation can be defined as the lowering of nociceptor thresholds. Normally, nociceptors have very high thresholds of activation. There are even some (known as silent nociceptors) that never fire unless in the presence of inflammation [25] Sensitised nociceptors can be stimulated by innocuous stimuli such as touch and normal movements or may discharge spontaneously [26]. Inflammation may lower the threshold of firing and increases the rate of firing in what is defined as primary hyperalgesia. In addition, intracellular second messenger systems may be activated creating more ion channels to be inserted into the terminals creating greater sensitisation [27] **.** Pain may also result from tissue ischemia resulting in acidic extracellular fluids associated with an increase in hydrogen ions and protons and activation of acidic sensitive ion channels.

INPUT MECHANISMS OF PAIN

There are thought to be 2-4 times as many C fibres as A delta fibres. Both fibre types have an orthodromic (afferent to the spinal cord) and antidromic (efferent to the tissues) function. The efferent function is thought to transmit neurotransmitters and neuromodulators into the peripheral tissues. This may have a vasoactive effect making capillaries leakier with more plasma extravasation and swelling in a process known as neurogenic inflammation. It may have a healing effect bringing immune cells to the site of injury but as they degranulate and release histamine and serotonin it may add further to the inflammatory soup.

The antidromic impulse can be generated from the spinal cord, from injury along the nerve or from the dorsal root ganglia [10]. When injured, a segment of the peripheral nerve may develop the ability to generate its own impulses in what is known as abnormal impulse generating sites (AIGS). Myelin usually prevents the insertion of additional ions channels. Where there is no myelin such as the dorsal root ganglia (DRG), additional ion channels may be created which increases the sensitivity of the neurone. The DRG is vulnerable to chemical and mechanical irritation from nearby inflammation and is particularly sensitised by adrenalin released as part of the sympathetic nervous system response to stress [28]. Nociceptors are also thought to have adrenergic receptors which may become upregulated during sensitisation [10].

PROCESSING MECHANISMS OF PAIN

This mechanism of pain is often described as central sensitisation. This concept proposes that the CNS sensitivity or activation threshold is set lower and therefore stimuli that would not normally access central neurones can now have an effect. It is thought not just to be a sensitivity to physical stimuli but to psychological inputs as well. There are thought to be three ways that central sensitisation can be manifested in the CNS neurones – the firing threshold is reduced, there is an increase in the responsiveness of the neurones and the receptive field of the neurone will increase [29].

The dorsal horn (or medullary horn in the case of facial input) may operate in four modes: normal, suppressed, sensitised and reorganised.

In the **normal** state, innocuous inputs such as touch, pressure and warmth will be perceived as that stimulus. The CNS can define and extract each input including that which is noxious. Input will equal output and the key transmission chemicals are Glutamate (activating an AMPA receptor) and substance P (activating a neurokinin 1 receptor).

In the **suppressed** state, some inputs that you would expect to hurt, don't seems to hurt. This may enable a severely injured person to survive until they can get the damage treated. Some of the suppression may come from reduction of peripheral inputs. Most suppression comes from the endogenous pain control mechanisms in the brain and spinal cord that release inhibitory chemical such as GABA, enkephalin and serotonin into the dorsal horn synapsis.

The descending pain control systems include the periaqueductal grey (PAG) and rostal ventromedial medulla (RVM) pathways which can exert a facilitatory or inhibitatory effect at the dorsal horn via transmission of opiods, serotonin and noradrenalin. As these pathways have connections with higher brain centres they may be affected by the individuals past experience and context of pain.

In the **sensitised** state , a barrage of impulses from an area of inflammation or acidosis or from an AIGS release excitatory chemicals such as glutamate, aspartate or substance P into the synapsis. Glutamate activates AMPA and NMDA receptors (closed in the normal state) allowing calcium ions to cross the membrane which can activate phyosphorylation and transcription factors to keep the channels open longer and allow more channels to be synthesized. With a leaky membrane and excitator chemicals in the synapsis, other chemical such as nitric oxide and prostoglandins further sensitise the neurone to depolarise. As the CNS has a lower activation threshold, inputs such as light touch and postural tone feedback can evoke pain and noxious input is magnified. This process of sensitisation should return to a normal state as the afferent input and central modulation returns to normal but in some patients this state persists in what is sometimes called secondary hyperalgesia.

In the **reorganised** state, there is a persistent peripheral driver that maintains the sensitised state. This may be persistent inflammation or acidosis in the tissues, nociceptors, the dorsal horn or dorsal root ganglia. It may result from ion channel changes or AIGS. It may also occur from dieback of C fibres that are replaced by resprouting A Beta fibres into lamina 2 [30] . Hence it becomes even more likely that non-noxious stimuli can be perceived as pain. In the reorganised state the persistently upregulated dorsal horn will cause signalling changes in the brain particularly the limbic and reticular system, the hippocampus and the cortex.

More than one area of the brain is involved in the pain experience simultaneously in what is termed "ignition nodes". These parts link up electrically and chemically in what is termed a neurotag or neuromatrix [31].

FIGURE 9 NEUROMATRIX – image from [32]

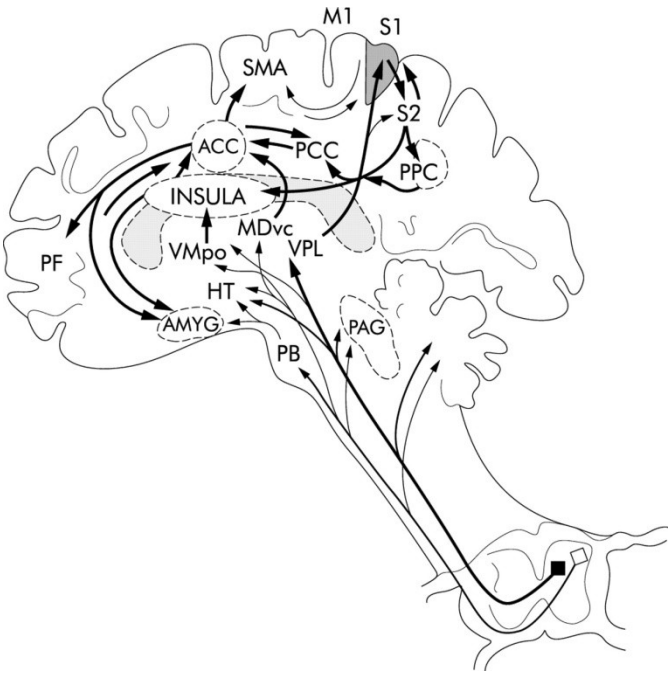
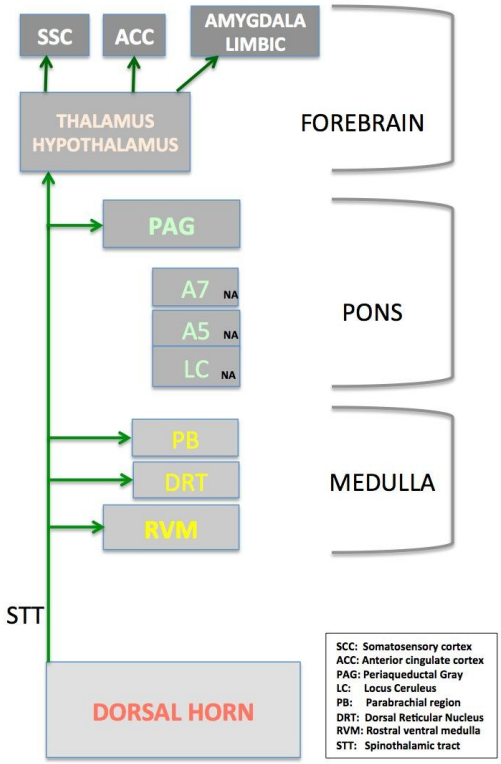


FIGURE 10 NEUROMATRIX – image from [33]



Just like the rest of the nervous system, the brain may change and adapt by manufacturing more sensors in the ignition zones. In the cortex, there may be 'smudging' of the neurotag – brain areas normally devoted to different body parts or different functions begin to overlap and areas of repeated use get larger [34]. The more chronic the pain becomes, the more advanced the brain changes may become [35]. In this state, even negative thoughts may have the potential to activate the neurotag and may lead to changes in the autonomic, endocrine, immune and motor (output) systems which may combine to perpetuate the neurotag.

OUTPUT MECHANISMS OF PAIN

Pain and stress may activate three key output circuits – the hypothalamus-pituitary- adrenal axis (HPA), the sympathoadrenal axis (SA) and the sympathetic neural axis. Each axis is thought to have its control components located in the brainstem.

In times of stress and pain the adrenal medulla secretes adrenaline and noradrenaline (SA axis) and cortisol from the adrenal cortex (HPA axis). Cortisol secretions are activated by the adrenocorticotrophic hormones (ACTH) secreted by the anterior pituitary gland. ACTH and cortisol secretions are higher in the morning and lower in the evening (circadian rhythm) but may be increased by stressors such as pain and thoughts and feelings. This stimulates the production of corticotrophin releasing hormones (CRH) from the hypothalamus which activates pituitary release of ACTH. A feedback loop is in place as the hypothalamus also monitors blood cortisol levels.

Cortisol maintains cardiovascular and metabolic homeostasis by stimulating protein catabolism and glycogen synthesis – vital energy systems for dealing with emergencies. It can cross the blood brain barrier to change mood and affect the immune response via cytokinine production [36]. In times of emergency, cortisol shuts down non essential systems such as the digestive, immune and reproductive system and enhances the cardiovascular, nervous and musculoskeletal systems. Chronic stress or pain is thought to increases cortisol levels which may lead to immunosuppression, cardiovascular disease, depression and slow tissue healing [37]. Adrenaline (epinepherine) secretion is more associated with mental stress while noradrenalin (norepinepherine) secretion is linked more with physical activity and blood pressure regulation. Both prepare us for action and like cortisol enhance the survival systems while turning down the non-essential systems. As part of the sympathetic nervous system (SNS), these catecholemines could contribute to the sensitivity of the nervous system and inflamed tissues leading to pain. It may do this by contributing to the inflammatory soup and by adrenoceptor upregulation in the DRG and AIGS. While the SNS is thought to liberate energy and inflammation, the parasympathetic system aids digestion, energy storage and tissue repair. It appears to act as a balancing mechanism for the SNS.

Linked into the HPA/SA system is the immune system via messenger molecules known as cytokines. Some are believed to increase inflammation (interleukin 1 and 6 and tissue necrosis factor alfa) while others are thought to reduce it (interleukin 4, 10 and 13) [38]. The immune system is thought to be regulated by the central and peripheral nervous system. Peripheral nerve responses such as the release of substance P and central activation from the HPA and SA axis, the vagus nerve or glia of the brain or spinal cord may activate pro-inflammatory cytokines.

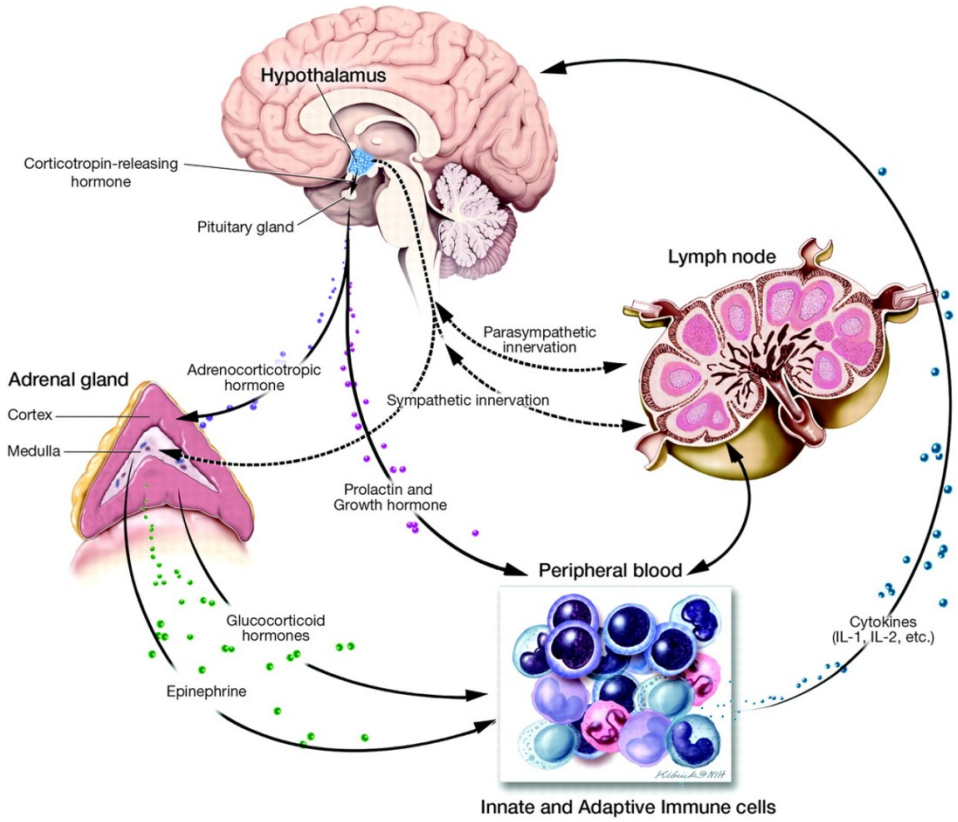


Figure 11 HPA Axis

image from [39]

The motor (muscular) system can be considered as an output system that responds to pain and stress by spasm, weakness, imbalance and loss of range of motion. With activation by the SNS, muscles can become acidic and inflamed and contribute to peripheral pain. Muscles may also become shortened and weak creating inefficient motor patterns which may further contribute to the pain cycle [39].

RELIABILITY AND VALIDITY OF TESTING IN MANUAL THERAPIES

One of the key concepts in osteopathic manual therapy is palpatory diagnosis. There are four general types of palpatory diagnostic tests which include differentiation of tissue texture, evaluation of static landmark positional asymmetry, evaluation of motion asymmetry and assessment of tenderness [40].

A systematic review reported that palpatory tests assessing tenderness have consistently shown at least moderate interexaminer reliability while palpatory tests of landmark positional asymmetry, motion asymmetry and tissue texture have consistently shown poor interexaminer reliability [41]. Acceptable kappa values after consensus training was achieved only for tissue texture and tenderness (K=0.68) [42].

One recent review of common palpatory landmarks found that the inter-examiner reliability was poor [43] Intraexaminer reliability was slightly higher but both intra and inter examiner reliability may be increased substantially with training [44], [45].

STRAIN AND COUNTERSTRAIN TECHNIQUE (SCS)

Counterstrain (SCS) is defined as a passive positional technique aimed at relieving pain and dysfunction through indirect manual manipulation [46]. In contrast to most forms of manual therapy, treatment is applied away from a restrictive barrier making it a very gentle approach.

Counterstrain technique was first accidently discovered (in 1955) and later described by osteopath Lawrence Jones in 1964 [47]. SCS is based on the palpation of tender points (TP) used to both diagnose and treat pain and dysfunction throughout the body. Unlike myofascial trigger points (MTrPs) described by Shah [48], SCS TPs are not felt as ropy bands nor do they exhibit a twitch response or refer pain. TPs have been described as exquisitely tender upon palpation, small (<1 cm), round, edematous, and found in muscle, tendon, ligament, or fascial tissues [49]. Unlike MTrPs, SCS treatment is not directed at the TPs - they merely aid in diagnosis and in finding the ideal treatment position. There has been no research into the biological milieu of TPs but it has been found that symptomatic patients have lower electrical detection and pain thresholds for TPs than controls [50]. SCS TPs are very common – a large study that grouped TPs by region found half of the groups were positive for TPs in 50% or more of participants. It also found a higher prevalence of TPs in the anterior cervical and upper rib groups (80- 90%) and in participants with history of pain or trauma [51].

TECHNIQUE AND EVIDENCE BASE

SCS treatment begins with identifying and gently monitoring a TP while positioning the patient to reduce the tissue tension and palpation tenderness of the TP. The position of comfort is typically obtained by shortening tissues around the TP. Full release of the TP occurs after 90 seconds, after which the practitioner slowly returns the patient to their neutral resting position. The TP is reassessed with relief of the TP tenderness the desired outcome [52]. There are numerous studies of varying quality which find SCS does decrease TP tenderness. This has been supported by a systematic review [53]. However there are limited SCS studies showing positive clinical outcome measures. Positive outcomes include lasting pain relief [54] [55], improvements in range of motion [56] and in strength [57]. Recent research has also found SCS to be effective for headaches [58], restless leg syndrome [59] and chronic ankle instability [60]. Like most manual therapy techniques, more quality research is needed to determine the effectiveness of SCS.

DIAGNOSTIC VALIDITY

There are three specific components to diagnosis in SCS. The practitioner first palpates gently for tissue texture changes described as tense, ropey and boggy which represents muscle hypertonicity and oedema. Second, the practitioner palpates the altered tissue for specific TePs which elicit exquisite local pain and indicate a specific somatic dysfunction. Thirdly the practitioner may evaluate the involved level for impairment in the amplitude and quality of joint motion [61].

Tissue texture and tenderness have acceptable diagnostic reliability following training [42]. Fryer correlated palpated tissue texture changes with alterations in muscle activity detected by EMG which he later concluded was from tissue fluid from inflammatory mediators [62]. One study found that SCS assessment (tissue tension, tenderpoint and joint motion) was more reliable than traditional osteopathic palpation (tissue tension, joint capsule tenderness and joint motion) in symptomatic neck pain patients [63]. An unpublished study found that there was good inter-examiner reliability between novice and expert SCS practitioners suggesting that diagnostic TePs were accurately reproducable [64].

PROPOSED PHYSIOLOGICAL MECHANISMS

When Jones first discovered SCS, he had no explanation of its physiological mechanism until the emergence of the proprioceptive theory [1]. This theory implies that a rapid stretching injury stimulates a muscle spindle reflex to resist further stretching and also facilitates the antagonists muscle spindles. This results in a neuromuscular imbalance perpetuated by opposing muscle spasms each unable to release due to ongoing muscle spindle excitation. The theory also suggests that spindle activity and reflexive muscle contraction decreases upon shortening. By passively shortening the dysfunctional agonist muscle, SCS allows normal muscle spindle activity to return. Once agonist muscle spindle activity is reset, antagonist muscle spindle activity can also return to resting state, relieving aberrant neuromuscular activity and restoring normal function. Jones described the TP as a sensory manifestation of this neuromuscular imbalance – the centre of the point is neurologic while it's surrounded by the resultant inflammatory oedema. There is little recent evidence to support the proprioceptor theory although one study found the achilles tendon stretch reflex diminshed after SCS (while the H-reflex remained unchanged) suggesting that SCS may alter the stretch reflex by altering muscle spindle activity [55].

EVOLUTION OF SCS

Over decades, Jones discovered 56 musculoskeletal TPs and towards the end of his career discovered a further 9 TPs related to the bones and sutures of the cranium. As most of these later TPs were not influenced directly by skeletal muscle, he felt that the proprioceptive theory model needed reconsideration. Jones's students, American physical therapists Randall Kusunose and Brian Tuckey not only expanded on his original work (increasing the number of musculoskeletal and cranial TPs to over 280) but evolved the technique to include over 400 "fascial" TPs. These TPs were related to the fascia surrounding the visceral organs, blood and lymphatic vessels, central and peripheral nervous system and within the joint capsules and ligaments.

Our knowledge of fascia has advanced dramatically over the past two decades. We now know that fascia contains contractile smooth muscle fibres and myofibroblasts that are able to contract with enough force to influence the musculoskeletal system [65], [66], [67]. We also know that fascia is embedded with type III and IV peripheral neurones (free nerve endings) which have both nociceptive and mechanoreceptive capabilities. These neurones also travel multiple segments of the spinal cord, release inflammatory chemicals when irritated and link to the autonomic nervous system. They can therefore trigger both "nocifensive" and "nociautonomic" reflexes [13], [68]. These reflexes recruit skeletal muscle and autonomic processes to protect the pain sensitive free nerve endings in the fascia. Additionally the stimulation threshold is lowered through central sensitization in the spinal cord and excretion of inflammatory cytokines in a positive feedback loop [69]. While musculoskeletal TPs are typically treated by folding the body over the point, fascial TPs are generally treated by manually gliding the restricted fascia in the direction of ease. Tuckey [70] suggests the physiological mechanisms behind fascial SCS includes the decompression of the free nerve endings which silence the nocifensive/ nociautonomic reflexes and opens the local fascial venous and lymphatic channels draining inflammatory chemicals from the region. SCS TP's may not only contribute significantly to central sensitization leading to chronic pain states but influence fluid dynamics creating swelling and effect autonomic functions such as digestion and immunity.

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