Chapter

Clinical Insights into the Importance of Scars and Scar Release in Paediatric Chronic Myofascial Pain

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"Scars have the strange power to remind us that the past is real".

Cormac McCarthy, All the Pretty Horses.

Abstract

Humans exhibit biotensegrity, whereby the whole body is a three-dimensional visco-elastic vehicle whatever position it adopts: bones form non-contact compression struts embedded in a networked and tensioned myofascial matrix; each part of the organism combines with the mechanical system to create an integrated functional movement unit and contributes to the stability of the whole system. When tissue at/below the dermis is breached by surgery/injury, healing leads to scar tissue formation. Scars can cause local and distant effects that are not purely cutaneous. Restriction of normal movement of underlying tissues from defective fascial sliding generates anomalous tension that affects the fascial continuum leading to distorted biomechanics, altered biotensegrity and chronic pain. Scars are common in children and significant contributors to chronic pain presentations. Scars can be released (soft tissue mobilization and/or needling) to sustainably improve pain, flexibility and range of motion. This chapter outlines the importance of skin and fascia in the biotensegrity model. Emphasis is placed on the fundamental need to assess scar history and scar characteristics to determine if scars should be treated as a component of multidisciplinary chronic pain management. Case studies outline some key clinical observations. Appropriately controlled research studies are required to fully demonstrate the highlighted benefits.

Keywords: chronic pain, paediatric pain, myofascial pain, scars, scar release, treatment approaches, fascia, biotensegrity

1. Introduction

1.1 Skin

> *"Our skin mediates the most important transactions of our lives, Skin is key to our biology, our sensory experience, our information gathering, and our relationships with others.....although rarely appreciated, it is one of the most remarkable and highly versatile parts of the human body."*
>
> *Nina Jablonski, Skin – A Natural History [1]*

The skin is the largest organ in the body. It provides a crucial interface between the body and its environment. It has very diverse functions, which include: a defensive barrier against toxic agents and micro-organisms, a multidimensional sensor between the body and the external environment, a protective shield from sunlight, a harnesser of sunlight to begin the process of Vitamin D production in the body, and host to a large part of our microbiome [1, 2]. The skin is thinnest at the eyelids and thickest on the soles of the feet. Skin consists of the epidermis and the dermis, which differ markedly in their structure and function.

The epidermis, the outermost layer is a stratified keratinizing epithelium, approximately 1 mm thick, which is in constant turnover. It maintains a shield against environmental toxins, resists water and protects against heat. The main types of cell found in the epidermis are the keratinocytes, but there are also melanocytes, Langerhans cells, T-lymphocytes and Merkel cells.

Basal keratinocytes are known to differentiate and flatten out to create the stratum corneum. The balance of proliferation and differentiation of the basal keratinocytes is essential for epidermal integrity and ongoing epidermal tissue renewal. Keratinocytes are in close physical contact with free sensory afferent nerve endings and therefore involved in nociceptive responses [3]. Keratinocytes also have some specialised functions, such as: synthesis of keratin, neuropeptides, neurotransmitters, endogenous opioids and autacoids; involvement in local inflammatory responses; as well as expression of multiple different receptors [3]. Tactile stimulation is known to induce peripheral oxytocin release from keratinocytes to mediate local, peripheral oxytocin mediated analgesia [4]. In addition, keratinocytes play a fundamental role in the pathogenesis of neuropathic pain and central sensitization [3, 5–8]. Keratinocytes play a significant role in skin tissue repair, but an abnormal balance of proliferation and differentiation of keratinocytes may lead to generation of pathological scars [9].

Melanocytes are responsible for the primary pigment colour of the skin and the production of melanin. Melanin content is the differentiator for skin colour. Lighter skin or depigmented skin has less melanin, associated with living in more northern climates, where the lighter skin can synthesise more Vitamin D from exposure to sunlight. Merkel corpuscles are skin mechanoreceptors, whereas Langerhans cells and the T-lymphocytes have immunomodulatory properties.

The dermis is a thick layer of connective tissue, which is elastic, pliable, and has substantial tensile strength. The dermis is split into the upper papillary dermis, and the lower reticular dermis. The reticular dermis is characterised by dense irregularly packed collagen, elastin, and reticulin, from which it derives its name. It will be seen later that the duration and intensity of the inflammatory process within the reticular dermis, following surgery or injury, has an impact in the development of pathological scars. The dermis houses other essential organs of the skin including hair follicles, sebaceous glands, endocrine glands and apocrine glands which are absent in scarred skin.

Whereas the primary cells of the epidermis are the keratinocytes, the primary cells of the dermis are the fibroblasts. The dermis gets its strength from a combination of collagen and elastin fibres. The fibroblast is a connective tissue cell that secretes collagen, elastin and other elements of the extracellular matrix. Collagen bundles in healthy normal skin are organised in a three-dimensional basket-weave pattern [10], whereas collagen bundles in scarred skin are organised in parallel to the epithelial surface [11]. Fibroblasts are critical in all phases of wound healing, wound contraction, and remodelling of scars. There are multiple distinct subpopulations of dermal fibroblasts. Fibroblasts with abnormal phenotypes (altered proliferation profile and different patterns of cytokine release) are associated with the development of pathological scars [12].

There are different receptors within the skin, which include: Meissner's corpuscles for light touch; Merkel's discs for constant pressure; Pacinian corpuscles for deep pressure or vibration sense; and Ruffini corpuscles for skin stretch and temperature sensation. The skin is also a primary site of small fibre nociceptive endings [13]. Underneath the dermis is the subcutaneous layer, which contains fat that insulates us to prevent heat loss, and fascial layers that give the skin mobility relative to the muscles, tendons and bones.

Adipose tissue has long been considered just a means of fat storage. However, it has important metabolic and endocrine functions. Regulation of whole-body energy metabolism occurs through its storage function in white, brown or beige adipocytes. Its endocrine function occurs through production of adipocytokines, including leptin and adiponectin. Neurons of the sympathetic nervous system innervate different fat deposits to create communication with adipocytes. Leptin is hormone secreted from white adipose tissue to alter the sympathetic outflow centrally. This leptin-dependent neuro-adipose tissue connection plays an important part in regulating lipolysis and thermogenesis [14]. Leptin also acts on the hypothalamus to regulate food intake [15]. Adiponectin, also a protein hormone produced by adipocytes, has organ protective functions that result from the binding of adiponectin to receptor sites on many organs to activate exosome formation and release for cellular haemostasis [16]. Adiponectin and adipocytes are key players in skin health and disease [17].

The skin is electrically active and contains many nerve elements for interaction with the nervous system [18], the autonomic nervous system [18, 19], and the locomotor apparatus [20]. There is continual nervous activity, in afferent and efferent mode, between the skin and central nervous system to maintain normal physiological and biomechanical homeostasis [18, 21]. There is an independent central emotional connection, principally between the anterior cingulate cortex and the skin, whereby a sympathetic electrical signal can be detected in the skin in response to viewing emotionally charged images [22].

1.2 Touch

Information from skin sensors is relayed via A-delta and C-tactile fibres to the spinal cord and from there to the thalamus, then to specific regions in the primary somatosensory cortex and further on to higher somatosensory areas. The somatosensory maps are not represented in direct proportion or alignment to the body. Certain regions are magnified in response to the density of mechanoreceptors in the associated skin; for example, fingers and lips. The brain receives converging input from numerous nerves innervating adjacent regions of the skin to enable recognition of a specific touch sensation. Neuroplastic adaptive changes occur in the somatosensory map related both to increased repetitive activity and to disuse. Although the primary somatosensory cortex responds appropriately to touch

information, the brain's higher centres are more strongly influenced by cognitive factors, such as expectations, context, and attention.

Wide dynamic range neurones integrate afferent pain and touch information. Others converge afferent viscero-somatic information into the spinal cord [23, 24]. Integration of these pathways can sometimes lead to sensory illusions. For example, referred pain is exemplified by shoulder pain from irritation of air under the diaphragm, or left arm pain in association with angina. Conversely, convergence of efferent sensory and sympathetic innervation to skin vasculature may explain sympathetic involvement in conditions such as complex regional pain syndrome and fibromyalgia [25, 26].

It is clear that the skin is a versatile and important organ, but it cannot be considered in isolation from its underlying fascia when considering the impact of scars.

1.3 Fascia

> *"The unsung hero of spine biomechanics."*
>
> *M. Driscoll [27]*

Fascia has long been thought of as the annoying tissue that has to be dissected off to get to the underlying anatomy. This traditional view of fascia has now been surpassed. The most up to date definition describes fascia as a sheath, a sheet or any number of dissectible aggregations of connective tissue that forms beneath the skin [28]. However, this definition is too simplistic, and it is the definition of the fascial system that highlights the importance of fascia in biomechanical regulation:

> *"The fascial system consists of the three-dimensional continuum of soft, collagen containing loose and dense fibrous connective tissues that permeate the body. It incorporates elements such as adipose tissue, adventitiae and neurovascular sheaths, aponeuroses, deep and superficial fasciae, epineurium, joint capsules, ligaments, membranes, meninges, myofascial expansions, periostea, retinacula, septa, tendons, visceral fasciae, and all the intramuscular and intermuscular connective tissues including endo-/peri-/epimysium. The fascial system surrounds, interweaves between, and interpenetrates all organs, muscles, bones and nerve fibres, endowing the body with a functional structure, and providing an environment that enables all body systems to operate in an integrated manner."*
>
> *Adstrum et al. [29], Stecco et al. [30] in Adstrum and Nicholson [28]*

Fascia is multi-layered and has both loose and hard fibrous connective tissue components. Loose fascia functions to help slide and glide between structures and dense fascia exerts a tensile strength in tissues like tendons. Fascia and muscle tissue have a complex interplay to achieve skeletal balance, co-ordination, posture, and locomotion.

Fascia contains cells (fibroblasts, fasciacytes, myofibroblasts, and telocytes), an **extracellular matrix (ECM)**, nerve elements (proprioceptors, interoceptors, and nociceptors), and a system of vascular micro-channels (the primo vascular system) [30, 31]. The fasciacytes produce hyaluronan in response to shear stresses [32]. Hyaluronan (formerly known as hyaluronic acid or hyaluronate) is a glycosaminoglycan of the ECM. Hyaluronan exists between fascia and muscle and promotes sliding and gliding between fascia, muscle, blood vessels, nerves, and lymph channels. It is one of the most important determinants of the viscoelastic properties of a tissue [32–35]. The fascial fibroblasts produce collagen in response to load and stretching. Both acute and chronic fascial loading stimulate collagen

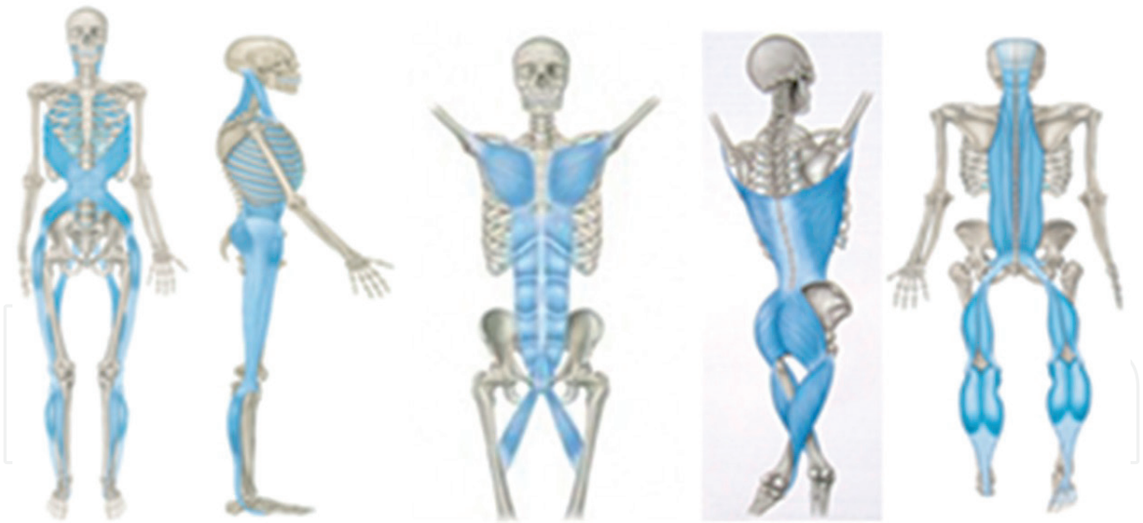

Figure 1.
Myofascial chains, from left to right: spiral line, lateral line, front functional line, back functional line, and superficial back line (adapted from Wilke et al. [36], with permission from Elsevier).

re-modelling [37]. Myofibroblasts within large sheets of fascia are considered to exert clinically significant change in fascial tension in response to sustained mechanical tension, cytokines, low pH, oxytocin, and other agents such as nitric oxide. The myofibroblasts' contractile properties may contribute to spasms, dysfunction, and pain [18, 38]. Telocytes are probably important in regeneration [39]. Fascial tissue has highly variable density, stiffness, humeral action, and metabolic activity, depending on its location and purpose [23].

As the fascia envelops every structure within the body, it creates structural continuity that provides form and function to every tissue and organ. Fascia is one of the essential biological structures that combines with muscles, tendons and bones to create the tensioned and compressed parts that create the **biotensegrity** properties of the human body [40]. Fascia has active mechanical, proprioceptive, nociceptive, and biomechanical and biotensegral properties [30, 36, 41]. Fascia is essential for physiological and metabolic haemostasis as well as healing and repair [30, 31]. Fascia conveys mechanical forces directly or indirectly: directly through muscles at the attachments to bones [42]; or indirectly, as extramuscular fascial chains [43]. There is evidence to support the existence of five myofascial chains (**superficial back line**, **back functional line**, **front functional line**, **lateral line** and **spiral line**, see **Figure 1**) [36], which work within the biotensegrity model [44]. These chains have opposing chains mirrored on either side of the body to achieve balance within the musculoskeletal system. This emphasises the complex nature of human movement and why range of motion at one peripheral articulation is dependent on the positioning of the entire myofascial chain system of the body [45]. Like the skin, fascia is also innervated by the autonomic nervous system and afferent free nerve endings [23, 46] and can be considered as a sensory organ of human biomechanics [23, 46–48]. Fascial tissue homeostasis is a complex, inadequately elucidated, interaction which depends upon active communication between cellular components of the fascia and the related ECM [43, 49].

1.4 The extracellular matrix

> *"The Organ of Form"*
>
> *Varela and Frenk [50]*

The extracellular matrix (ECM) is defined by Grey's Anatomy as *"the extra cellular components of connective and supporting tissues. Essentially, it consists of a system of insoluble protein fibres, adhesive glycoproteins and soluble complexes composed of carbohydrate polymers linked to protein molecules (proteoglycans and glycosaminoglycans), which bind water. The ECM distributes the mechanical stresses on tissues and also provides the structural environment of the cells embedded in it, forming a framework to which they adhere and on which they can move. It provides a highly hydrated medium, through which metabolites, gases and nutrients can diffuse freely between cells and the blood vessels traversing it"* [51].

The ECM is present within every tissue of the body to provide fundamental physical scaffolding for the cellular constituents and orchestrate vital biochemical and biomechanical functions required for morphogenesis, differentiation, and homeostasis. The ECM is basically composed of water, salts, and macromolecules consisting of fibrous proteins (collagen, elastin), adhesive glycoproteins (laminin, fibronectin, tenascin, integrin), and carbohydrate polymers (proteoglycans and glycosaminoglycans). Collagen is the most abundant ECM fibrous protein. However, each individual tissue has a heterogeneous ECM composition providing highly variable but unique biochemical, protective, organisational and biomechanical ECM properties, which differ from one tissue to another. This composition is primarily dependant on that tissue's particular function [52]. The ECM is a highly dynamic structure, constantly being remodelled with its molecular components subject to a multitude of modifications.

Morphological organisation and physiological function of the ECM is orchestrated by binding growth factors and interacting with cell-surface receptors to elicit signal transduction and regulate gene transcription. Enzymes released from fascial cells degrade the ECM, such as cathepsins, heparinase, hyaluronidases, and metalloproteases, to maintain normal tissue turnover.

Matrix metalloproteinases (MMPs) are key in ECM remodelling as they degrade matrix components. MMPs are classified according to substrate specificity; for example, collagenases or gelatinases [52]. It will be seen later that the ratio of MMPs to MMP-inhibitors is of importance in the development of atrophic scars.

2. Scars

2.1 The healing of wounds and scar creation

Any breach of the dermal layer will result in a scar. When the dermis is breached by surgery or injury, a healing process occurs that leads to the formation of scar tissue [53]. There are two main types of healing: primary intention and secondary intention. Healing by primary intention occurs in wounds with dermal edges in close proximity; for example, a surgical incision. Healing by secondary intention occurs when there is unavoidable tissue loss with abnormal tension, infection or necrosis, creating a large deficit where the sides of the wound are not opposed or too big to heal by primary intention; therefore, healing must occur from the bottom of the wound upwards. For both primary and secondary intention healing, there are three overlapping stages to healing after haemostasis is achieved; inflammation, proliferation, and remodelling [54]. This extremely complex and co-ordinated process is guided by cytokines plus chemokines secreted by platelets, macrophages, endothelial cells, keratinocytes, fibroblasts, and adipocytes [2, 12, 17, 18, 55, 56].

Wound healing and scar maturation can take years to complete, but depend on the size and nature of the initial wound. During remodelling, type 3 collagen is replaced by a stronger type 1 collagen, but not in an ordered manner. Scar tissue

is therefore strong, but not as elastic or flexible as normal tissue [38, 54]. Normal scars first appear as a red line, but maturation results in a slightly broadened white line, in the same plane with the surrounding skin, known as a normotrophic scar. Normotrophic mature scars are less innervated than the surrounding normal skin [57, 58]. Scar tissue, therefore, has different properties to the tissue it replaced prior to injury. Skin healing is not just dependent on the size and site of the wound, but is also influenced by bacterial contamination, nutrition, comorbid medical conditions, as well as genetic and epigenetic factors [56, 57]. Once normotrophic scars are established, they are often forgotten and ignored unless they constitute a cosmetic embarrassment.

2.2 Scar morphology

If the healing process is interrupted by tissue disruption, infection or a comorbid disease process, then a pathological scar may occur, which may be atrophic, hypertrophic, tethered, or keloid in nature. Development of pathological scars may be related to genetics, as well as differences in the duration, and intensity of the inflammatory process in the reticular dermis [56, 59–61]. Excessive scar tissue movement, scar rubbing, scar scratching or abnormal tension related to surgery or injury may also induce an enhanced neurogenic inflammatory response [18, 62–65]. This may be an important consideration when reflecting on what constitutes a significant scar in terms of myofascial dysfunction later.

Atrophic scars are concave indentations resulting from tissue loss (including collagen) associated with relative shift in the ratio of MMPs to tissue inhibitors of MMPs that favours a lytic process and the development of an atrophic scar [66]. Chicken pox scars are atrophic, which can occur in up to 20% of children following chicken pox infection [67, 68]. Acne scars are a common problem in developed countries, occurring in 80% of 11–30 years old and 5% of adults aged over 30 years [69]. Acne scars are usually atrophic, subdivided into rolling, boxcar, or ice-pick. However, acne scars can also present in hypertrophic or keloid morphology. Hypertrophic scars and keloid scars have excess deposition of collagen. Hypertrophic scars form early within the time frame of injury and remain within the boundaries of the inciting injury. Keloid scars have excess thickened hyalinized collagen. Keloid scars may have a delayed onset of formation and extend beyond the boundaries of the original scar. Keloid scars are more likely to occur in dark-skinned individuals, between ages 10–30, at times of peak hormones (puberty, pregnancy), with a positive family history, and in association with hyperimmunoglobulinemia E, or blood type A [70].

2.3 Scar assessment

Clinical documentation of scar size, thickness, and treatment effects are usually performed with the use of serial photographs, scar scales (such as the **Vancouver Scar Scale, VSS**), and validated questionnaires, such as the **Patient and Observer Scar Assessment Scale (POSAS)** or Dermatology Life Quality Index (DLQI). There are few reliable, objective non-invasive tools to measure scar characteristics. Ultrasonography and high-definition optical coherence tomography (HD-OCT) have also been reported for objective measurement of scars [71–73]. Scar pliability (mechanical property of the skin's stiffness, extensibility, and adherence) is measured using adheremeters, cutometers, and ultrasonography [74]. Viscoelastic properties of scars and connective tissue around scars are measured using sonoelastography [75–78]. Scar colour can be assessed using narrow-band reflectance spectrophotometry colour analysis or Tristimulus colour systems [79], and scar perfusion can be measured with Laser Doppler Flowmetry [79].

2.4 Scar management

Scar optimization and mitigation are key principles that are vitally important in medical practice, but beyond the scope of this article. Readers are directed to algorithms of management that exist for both the adult and paediatric populations [70, 80–83].

2.5 Asymptomatic or symptomatic scars

Diverse symptoms can occur in up to 70% of patients with scars [74]. Symptoms can occur at random timing and even at long intervals after wound healing. Some of these symptoms are highlighted in **Figure 2**.

Scars may be intrinsically painful or play a role in pain located at an anatomically distant site. Scars may present as painful or itchy at the site of the healed scar. One potential cause of intrinsic pain in a healed scar is a neuroma, derived from a regenerating nerve trapped in the fibrotic scar tissue. This is usually associated with numbness in the innervated area of the injured nerve and a positive percussion test. In the absence of a neuroma, the estimated prevalence of painful scars is 2%, but increases in the burn population to 30–68% [57]. Intrinsic scar pain occurs in 7–18% of patients following lower segment Caesarean section [84–86]. Pathological and intrinsically painful scars have been shown to have an in imbalance between non-peptidergic unmyelinated c-fibres and peptidergic c-fibres, but not necessarily in total nerve fibre density [57]. There is also an increased abundance of neuropeptides, and nociceptors in pathological scars, especially hypertrophic scars [18, 87].

It is clear that what is happening underneath a scar is critical to whether it subsequently creates symptoms. Scar tissue can adhere to fascia, underlying muscles, organs, blood vessels and nerves, and recognition has been given to scars as an etiological factor in post-surgical visceral dysfunction [88, 89], nerve entrapment syndromes [90], and symptoms related to obvious contractures. However, very little is documented for the role of scars in locomotor dysfunction [91–95] or chronic myofascial pain [38, 95–97].

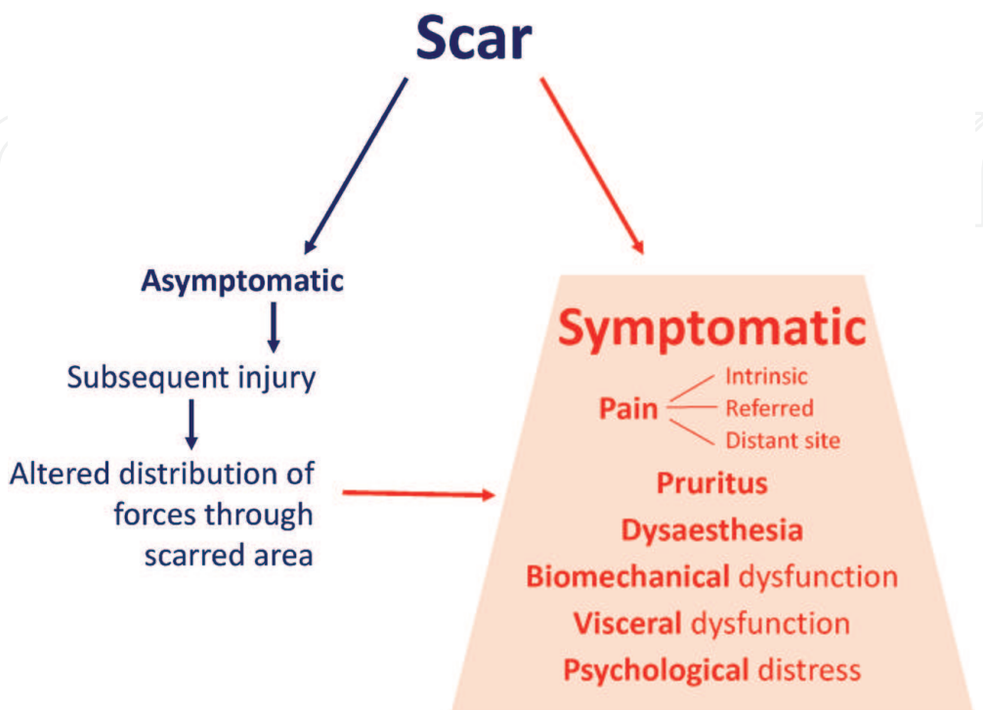

Figure 2.
Symptoms related to scars.

In patients asked to move actively, electrical activity from a scarred area is higher than that from normal tissue in the same patient doing the same movement [93]. Mechanoreceptors and mechanosensitive nociceptors in scarred areas may sense an alteration in tension from normal and send non-physiological signals creating a pathological reflex arc [18], inducing neurogenic inflammation and worsening scar formation and pain [18, 63].

2.6 How scars impact fascia, muscles and movement

> *"Any skin injury, like a scar, even if small, affects the organism and constitutes an active obstacle to its normal functioning"*
>
> *Alvira-Lechuz et al. [98]*

Scars can limit normal movement and the flexibility of skin, the underlying fascia, and its associated muscles. It is clear that any scar that restricts fascial or muscular movement will also have an impact on whole body movement related to the myofascial chains discussed in the fascia section above [18, 99]. The fascia is recognised as a significant contributor to chronic pain [100–102]. Other factors that may play a role include alterations in fascial density. Changes in hyaluronan may lead to fascial densification that restricts fascial sliding and gliding, aggravating fascial dysfunction associated with musculoskeletal disorders and chronic pain [32, 103–109]. Injury reduces the flexibility of fascia; for example, a compound fracture may be associated with periosteal tethering to the skin during healing. The subsequent defective fascial sliding generates anomalous tension, which affects the fascial continuum leading to musculoskeletal pathology, pain and progressive immobility [18, 32, 103, 110–112]. Fascial injury also leads to fascial dysfunction as well as significant loss of sports performance [18, 21, 113, 114]. Symptoms may not necessarily just occur at the site of injury, but also at a distant site related to referred pain and impacts through myofascial chains. Reduced fascial mobility, related to movement restriction secondary to injury, worsens over time, but may persist even when movement is subsequently restored [110]. This was demonstrated with ultrasound imaging of adults with chronic low back pain with increased thickness and reduced mobility of the thoracolumbar fascia [102, 110, 115]. Sonoelastography has been used to measure viscoelastic properties of the connective tissue around scars: it superimposes a parametric image, in a range of colours, over the ultrasound anatomical scan image to semi-quantitatively correlate images with that tissue's elasticity. Sonoelastography has been used to show the changes in post-surgical scars following manual therapy [75, 76].

Fascial trauma or injury may also irritate primary afferent nociceptive fibres leading to spinal cord wind up and central sensitization as another mechanism of ongoing pain [35, 96].

Myofascial pain is characterised by the presence of myofascial trigger points. Myofascial trigger points develop in response to many different insults such as: trauma, injury, surgery, repetitive microtrauma, poor posture, muscle overuse or overload. Myofascial trigger points that cause pain can originate in scars, skeletal muscle, and/or fascia. **myoActivation®** [116] is an innovative structured assessment and therapeutic process targeting release of fascia (including scars) and muscles in sustained contraction.

myoActivation is unique in focusing on aspects of the immediate and past trauma history (often overlooked in a classical medical history) and relating this to the current chronic pain presentation. The **timeline of lifetime trauma (TiLT)** inventory explores all injuries, especially those occurring at a young age, mechanisms of injuries, healing processes, and the perceived physical impact of

Features in the past medical history and timeline of lifetime trauma (TiLT) inventory	Scars incurred at a young age, especially the first or earliest scar in childhood
	Ongoing pain occurring in the immediate timeframe of a surgical procedure which is unexplained and resistant to other forms of therapy
	History of poor wound healing, wound infection or wound dehiscence
	Painful/itchy scars
	Scars incurred at a time of intense emotional turmoil or trauma.
	History of mood disorders may indicate any chin scars will be significant
	History of anxiety may indicate any chest wall scars will be significant
	History of "brain fog" (confused, disorganised, find it hard to focus or put thoughts into words) may indicate a face/chin/sternal or midline abdominal scars will be significant
	History of injury in the presence of an asymptomatic scar (injury may be months or years after scar acquisition), which results in pain in the region of this previously asymptomatic scar
Type of scars	Scars related to chicken pox infection, surgery and especially surgical drain scars, burns and animal (or human) bites
Site of scar	Over bony prominences, e.g. anterior chest, in feet, scar in region of main pain complaint, on the same side as a unilateral pain complaint
	At sites of periosteal tethering
Scar characteristics	Painful scars, purple discolouration, widened scar, palpable densities within or around the scar, scar dysesthesia, adherent/tethered scars
myoActivation examination BASE test [116]	If scar is situated in region of most painful or restricted BASE test on myoActivation examination, even if it looks like a normal scar and/or reported as asymptomatic
Response to dry needling release	Enhanced "biting" sensation when scar is released with dry needling even with appropriate use of topical anaesthesia and/or vapo-coolant spray
	Reported pain decreased or moved immediately following scar release
	Improvement in ROM and flexibility immediately following scar release
	Decreased irritability of other palpable pain points in region of and distant to area of scar release
Size of scar	Size of scar does **not** seem to play a factor as tiny epidural needle scars or chicken pox scars can be as significant as large surgical wounds.
Less significant scars	Some scars seem to be less significant in terms of myofascial dysfunction and chronic pain, e.g.
	• Superficial self-harm scars
	• Tattoos (although they may be hiding another significant scar)
	• Stretch marks

Table 1.
Clinical factors indicating a scar may have potential for myofascial dysfunction and chronic pain.

those injuries to that person. Assessment involves the use of a series of **systematic movement tests (BASE tests)** [116], postural observations and examination of tissues to find palpable pain points and determine if there are important myofascial sources of perceived pain. A key principle in myoActivation is that the site of perceived pain is often not the true source of pain. The most painful or restricted movement on BASE tests identifies the most important tissues to treat first. Careful inspection and palpation of these tissues localises the myofascial source of pain. Treatment entails refined trigger point injections to restore anatomic integrity to injured tissues. Fine gauge hypodermic needles are inserted into trigger points that compromise function of muscle, ligament, tendon, subcutaneous fascia, scar tissue, and the peripheral nerves of the skin. After each individual myofascial area is treated, BASE movement tests are repeated to demonstrate immediate change and direct the clinician to the next most important target area. Several cycles occur during each myoActivation session to unravel multiple sources of myofascial dysfunction in a structured way, to help resolve chronic myofascial pain. Immediate treatment responses occur, which include reduction in pain, increased flexibility, and improved fluidity of movement. This process often reduces or resolves pain sufficiently to enable the subsequent weaning of prescription and over-the-counter analgesia medications [117]. myoActivation is not effective in all patients because pain perception is a complex biopsychosocial phenomenon, but determination of those who will respond rests with the TiLT inventory, clinical history and the series of BASE tests on examination.

Clinical experience with myoActivation [116] reveals that a scar can play a very significant role in chronic myofascial dysfunction and pain even if that scar has the appearance of a normotrophic scar and is reported to be asymptomatic. An ankle scar may alter the gait dynamics through maldistribution of myofascial loads [18] and may also present with ongoing pain at a distant site on the ipsilateral side. Patients with scars in the abdominal region, such as a Caesarean section scar or an abdominoplasty scar, may present with low lumbar back pain related to impaired mobility of the soft tissues of the abdomen, which then puts stretch on the lumbar muscles, thoracolumbar fascia, and the posterior myofascial chain [92, 93, 112]. Scars may also have an impact on the distribution of forces that pass through the body at the time of an injury, such as during a motor vehicle accident [18], causing a previously asymptomatic scar to be the inciting event for myofascial impairment and pain.

2.7 Significance for myofascial dysfunction and chronic pain

Classic scar assessment techniques such as use of adheremeters, cutometers, ultrasonography, and rating scales (POSAS or VSS) [74] were not designed to determine a scar's relevance to myofascial dysfunction and pain. A scar may be significant in terms of myofascial dysfunction and chronic pain, but not be characterised as a pathological scar in current "classical" medical terminology. Clinical experience with myoActivation therapy has found some features, outlined in **Table 1**, that indicate a scar may cause significant myofascial dysfunction and/or chronic pain. These observations will have to be researched to determine which ones are the more common and substantial factors in myofascial pain and dysfunction.

3. Scar release

Non-surgical scar release can be achieved with soft tissue mobilisation techniques, subcision or dry needling [84, 93, 118–121]. Subcision, or microneedling, also known as percutaneous collagen induction therapy, is a minimally invasive

minor surgical procedure used for treating depressed cutaneous scars and wrinkles. A simple hypodermic needle or a dermaroller are the tools used to effect subcision [122]. Subcision was first described in 1995 [123]. It is a safe and effective microneedling technique used as an aesthetic treatment for several different dermatological conditions including scars, rhytids, and striae [122, 124, 125]. Histological changes have been demonstrated with this technique [126]. It is hypothesised that microneedling stimulates the body's own regenerative mechanisms through collagen bundle break down and new collagen formation (neocollagenesis), stimulation of the release of platelet and neutrophil derived growth factors and cytokines (FGF, TGFα, TGFβ, VEGF, FGF-76, EGF, platelet derived growth factor, connective tissue growth factor, and connective tissue activating protein), resulting in increased production of collagen, elastin and glycosaminoglycans [122, 127–132]. Up-regulation of TGF-β3 (which prevents aberrant scarring) in excess of TGF-β1 and TGF-β2 may be responsible for the benefits seen with microneedling [129, 133].

The other physiological effects of dry needling are not yet fully elucidated, but may include local, hormonal, neuronal, and placebo effects [134]. Neuronal effects include: modulation of the peripheral nervous system; gate control mechanisms; and central pain modulation, such as activation of the diffuse noxious inhibitory control systems [135]. Dry-needling stimulation of skin nociceptive nerve fibres may release endogenous opioids to activate enkephalinergic inhibitory dorsal horn interneurons and oxytocin to mediate peripheral inhibition of c-fibre discharge [4].

For the release of scars, the myoActivation needling technique involves the sequential insertion of 30 g hollow bore needle in the line of the scar and in any areas of densification around the scar performing multiple perforations approximately 3 mm apart. Clinical experience reveals that a patient report of a "biting sensation" with needling is characteristic of a myofascially significant scar. This biting occurs even if the scar has been treated with appropriately timed topical anaesthetic or pre-needling vapocoolant spray.

The release of scars with micro-needling techniques has been shown to produce relief of chronic pain [116, 136]. Currently, the immediate relief of chronic pain following needling of surgical scars is limited to case reports [116, 120, 136], and to date there is insufficient evidence to advise on the right time to treat scars after injury or surgery. It has been suggested that the skin can keep a memory of trauma [118, 137]. It is clinically important to consider this and be cognizant of events which were associated with creation of a scar. Scars inflicted at a time of severe emotional distress or at the time of a traumatic event can be associated with flashbacks or emotional release at the time of, or a number of hours after, treatment whether the therapy was with a needling procedure or manual manipulation [138].

3.1 Effects of scar release

As all structures of the human body are intricately connected through skin and the myofascial system, scar and myofascial release (often at a distant site) can result in immediate improvement of pain, flexibility and range of motion [116] (**Table 2**), but it is imperative that this is combined in a multidisciplinary approach to address the whole biopsychosocial aspects of pain especially in the paediatric population [136]. It is absolutely essential to prevent needle-related pain in paediatric patients; the practitioner should employ a variety of non-pharmacological techniques including distraction, breathing techniques, music, virtual reality or mobile devices. Pharmacological modalities can be added when non-pharmacological methods are considered insufficient to address the patient's anxiety and needle-related pain. Topical anaesthetic cream can be applied to target sites (especially scars) an

Immediate effects	Decreased pain or pain which has shifted to a new location
	Increased flexibility and range of movement in myoActivation BASE tests
	Altered weight distribution on feet
	Emotional release
	Reports of feeling lighter or "walking on a cloud"
	Sympathetic response
	Vasovagal response
	Bruising
	Post treatment pain at site of needle insertions lasting minutes to hours
	Exacerbation of muscle spasms (requires additional needle insertions to resolve)
	Adverse events (Injury to adjacent structures e.g. pneumothorax when needling around lung fields)
Long-term effects	Improved pain
	Improved flexibility
	Improved mood
	Improved sleep
	Maturation to a normotrophic scars

Table 2.
Effects of scar release.

Absolute	New scars in process of normal healing and remodelling
	At sites of active infection
Relative	Near indwelling metalwork/hardware to minimise risk of hardware infection.
	Near indwelling mesh to minimise risk of infection.
	Where it is considered to be too painful (especially foot scars)
	When patient has needle aversion or needle phobia

Table 3.
Relative and absolute contra-indications to needling scars.

hour before the appointment to minimise needle pain. Oral benzodiazepines can be useful for anxiolysis. If the above methods are not adequate, IV sedation with appropriate anaesthetic monitoring and care may have to be utilised.

Over and above these considerations there are some relative and absolute contra-indications to needling of scars (**Table 3**).

4. Case based discussion of important points

Chronic myofascial pain is common in the paediatric population and its prevalence has increased since the 1980s [139]. Musculoskeletal pain is a common cause of pain in adolescents, with incidence ranging from 30 to 65% [140–144], and is a leading cause of years lived with disability among children and adolescents [145]; 4% of all primary care consults represent musculoskeletal issues in children aged up to 15. Between 4 and 40% of adolescents report limb pain and 14–24% complain of

low back pain [146]. The prevalence of paediatric chronic myofascial pain increases with increasing age [139, 147–150] and is more common in females [146, 151]. Young children are not immune with a 10% prevalence of musculoskeletal pain in 6-year-old children. A staggering one-third of this six-year-old population has chronic musculoskeletal pain and 44.6% of them report that pain is multisite in nature [152].

Despite its prevalence, reported musculoskeletal pain is often under-diagnosed in adolescents [153, 154]. Healthcare providers may be unfamiliar or not trained to diagnose chronic pain, muscle trigger points, palpable pain points, and fascia in tension [155–158]. Presenting symptoms such as neck pain, shoulder pain, abdominal pain and headaches do not immediately direct a physician to look for a myofascial component to pain. Myofascial pain can also imitate other pathologies; for example, a trigger point in the quadratus lumborum muscle can mimic the symptoms of appendicitis on the ipsilateral side, or fascial tension in the peri-coccygeal soft tissues may present as neck pain. System, practice and time pressures may also limit the ability to undertake a full history and physical examination [159, 160].

Experience dictates that dysfunctions in muscles, fascia and scars are common in the paediatric population and are significant contributors to paediatric chronic pain [116, 136]. The following cases highlight the importance of scars in paediatric myofascial pain presentations. It must be emphasised that scars are not treated in isolation, but as one element of myoActivation, which is one component of multidisciplinary care.

The children described in these case studies were referred to the **Complex Pain Service (CPS)** at BC Children's Hospital in Vancouver, Canada, and were treated within a program of multidisciplinary care based on the "**3P**" principle (*P***harmacology,** *P***hysiotherapy,** *P***sychology**). The data for these case studies were collected with the approval of the University of British Columba/Children's and Women's Research Ethics Board (H20-01862).

4.1 Case 1: standard myoActivation assessment finding unreported scars

A 13-year-old, 54 kg female was referred to the CPS for management of multisite chronic pain of 3 years duration with a diagnosis of "pain syndrome" focused mainly on the left knee, but also affecting the low back and neck. The low back pain of 3 years was reported to be secondary to a fall 3 years previously. She had been involved in a motor vehicle accident (MVA) 2 years after that when she was struck on the left side whilst walking across a road. She was admitted by ambulance to the local emergency room (ER) and discharged later that day with a diagnosis of bruising to her left knee and ribs after normal imaging. She initially mobilised with crutches and gradually regained physical functioning, but with ongoing pain.

She described her current pain as dull and achy with stabbing pain elements. Pain was aggravated by walking long distances or any physical activities or exercise. She stated that she had some irritability and sadness related to her pain. She complained about some inability to concentrate due to pain and had been absent from school for 21 days in the previous 3 months. She reported that pain also affected her ability to fall and stay asleep. Her TiLT inventory revealed no other injuries. She reported that she had no scars. She had been using acetaminophen and ibuprofen or naproxen for analgesia as required. She was otherwise healthy and her family history was unremarkable.

She was assessed by her orthopaedic surgeon and family doctor. Previous classical examination, imaging (X-rays and MRI) and bloodwork revealed no remedial cause of her pain.

myoActivation examination revealed unreported scars on the left knee and the right upper back, fascia in tension and multiple muscles in sustained contraction.

A myofascial component to her pain was diagnosed. She was enrolled in the 3P care plan. She was started on Magnesium Bisglycinate, Vit K2 and Vit D (MgBis/K2/D3). Written information about myoActivation was given to the family. One month later, there was a reduction in pain with improved fluidity of movements. Her family also reported improved mood and sleep.

4.1.1 myoActivation session 1

It was deemed appropriate to proceed with myoActivation at this one month follow up based on her stoical character. Written consent was obtained. Distraction techniques, vapocoolant spray and topical anaesthetic for scars were used to minimise procedural pain. All needling was done using sterile technique.

The right thoracic paraspinal, right gluteus medius, right gluteus maximus and left iliopsoas were sequentially activated based on the repeat cycles of assessment to determine the most restrictive and painful BASE tests [116]. The three left knee scars and upper back scar were in regions of worst BASE tests so they were considered significant and released. Scar release consisted of sequential perforation needling of the scar using a 30 g hollow bore needle. After this first myoActivation session the patient experienced immediate improvement in pain and flexibility.

4.1.2 myoActivation session 2

Five days after her first myoActivation session the patient reported that her low back pain no longer bothered her. She reported that the left knee pain felt less tight and tense and she was able to move "more freely", but going up and down stairs was still a problem. The left lumbar paraspinal, left rectus femoris, left vastus lateralis and left pubic fascia were sequentially activated based on the repeat cycles of assessment to determine the most restrictive and painful BASE tests [116].

4.1.3 myoActivation session 3

At the next myoActivation session, 1 day later, the patient reported that her pain had moved to the medial aspect of her left knee overnight. Repeat myoActivation examination revealed marked improvement in all core BASE tests with no pain except mild limitation of squats with arms down and arms up. The left vastus medialis was activated with improvement in squat with the arms down. A left shin scar was noted and released with improvement in squats with the arms up. The patient reported no pain at the end of this session.

4.1.4 myoActivation session 4

One month later, the patient again reported that her low back pain no longer bothered her. She reported that the left knee pain returned on stopping MgBis/K2/D3, so she restarted them. Based on the repeat cycles of assessment to determine the most restrictive and painful BASE tests [116], the right lumbar paraspinal, right hamstring, left gluteus medius and left iliopsoas were sequentially activated. Based on core BASE tests, the left knee scars were also re-released.

Four months after initial assessment, she only had minimal left knee pain with long walks, was sleeping, and was attending school full time with no absences. She required no prescribed or over-the-counter analgesia medications. On examination, she had normal movements in all core BASE tests with no pain. She was discharged from CPS care and advised to slowly wean MgBis/K2/D3.

4.1.5 Key points

- During examination, it is important to view as much skin as possible to find scars that patients do not remember or acknowledge are present.
- Scar found in the region of most restrictive or painful myoActivation BASE test should be released even if they are small, look normotrophic and are reported as asymptomatic.
- Scars may have to be re-released on subsequent myoActivation sessions.
- myoActivation is unique in enabling the unravelling of multiple contributors to pain, as in this case with low back and left knee pains.
- The patient-physician relationship is enhanced by hands-on examination and demonstration of change with sequential examinations.
- In children and adolescents, it is important to utilise techniques to minimise procedural needling pain.
- As a component of multidisciplinary care, myoActivation enhances recovery, even in patients with a history of years in pain.

4.2 Case 2: non-verbal patient

An 18-year old, 46 kg, non-verbal, wheelchair bound male with cerebral palsy (Gross Motor Function Classification System V), global developmental delay and seizures, was referred to the CPS. His paediatrician requested that the CPS determine if a series of myoActivation sessions might help with ongoing management of worsening bilateral leg spasms and pain. The patient had multi-physician paediatric care of his condition and was optimised on multiple medications (analgesic, anti-epileptic, and antispasmodic).

His carer believed that his pain was focused to his legs and was worsened with touching his legs or changing his diaper. It would take him 30–45 minutes to settle and seem calm after diaper changes or turns. Benzodiazepine rescue medication was used to help with spasms at these times. There was no specific pain pattern, with the pain occurring daily and restricting ability to move him and change diapers. There were no changes in skin colour or temperature and no oedema. Each night he is moved every 2 hours as he is unable to move himself; however, his carer felt that sleep was further disturbed by pain. He could not tolerate lying prone.

Multiple surgeries in the past included: posterior spinal instrumentation and fusion T3-Pelvis; right femoral head excision and subtrochanteric valgus osteotomy; circumferential release of capsule right hip; right and left pelvic osteotomy; left femoral osteotomy; and bilateral leg soft tissue releases and Botox injections. His imaging confirmed normal healing and no complications related to his multiple surgeries.

A complete myoActivation assessment examination could not be performed given his non-verbal and wheelchair-bound status. Lying flat multiple bilateral scars were noted from the listed surgeries. His right leg was shortened and externally rotated. There was a torso shift to the right and the pelvis was lower on the right. The right knee was hyper-flexed. The left iliopsoas, rectus femoris and vastus medialis appeared to be in sustained contraction. He was started on MgBis/K2/D3 supplements. One month after initial assessment, scar release was performed

under anaesthesia to release all the right-sided leg scars. Scar release consisted of sequential perforation needling of the scar using a 30 g hollow bore needle, using sterile technique.

Two months after initial assessment, myoActivation scar release was done to release all the left sided leg scars and activate the left iliopsoas, left rectus femoris and left vastus medialis under anaesthesia.

At follow up 1 month later, 3 months after initial assessment, his caregiver reported that he was much happier with no further episodes of crying out in pain with movement or diaper changes. He was also able to lie on his front without any issues.

4.2.1 Key points

- It is difficult to assess pain in a non-verbal patient.

- Children and adolescents with developmental delay may not be able to participate in a structured myoActivation examination; therefore, the pain physician will have to rely on parent or carer observations (most painful movements or triggers of pain, most comfortable positions when awake and during sleep).

- In a non-verbal patient, clinical experience and acumen determine if there is a myofascial component to the patient's pain and help direct therapy to the most likely sources of myofascial pain.

- As in developmentally normal children, techniques to minimise procedural needling pain are essential, as in this case where multiple scars were released utilising procedural sedation.

4.3 Case 3: patient requiring general anaesthetic for release of foot scar, distant from pain site

A 14-year old, 57 kg male was referred to the CPS for management of chronic left shoulder pain of 7 months duration. His pain was severe, graded as 8/10 on a visual analogue scale, localised to the left shoulder with no radiation. There was no inciting event to his pain. The pain was exacerbated by any movement of his shoulder. He had mild left sided hemiplegia secondary to removal of a thalamic astrocytoma at age 6. He had undergone a left ankle tendon transfer at age 13. He reported that pain significantly affected his ability to fall and stay asleep. His TiLT inventory revealed he had had a fractured left ankle, but no MVAs. He had scars related to the above surgeries as well as scars from his right sided venous access device, which was inserted at the time of diagnosis of his astrocytoma and removed a year later upon successful treatment of his oncological presentation. He was otherwise healthy, and his family history was unremarkable.

He was assessed by his orthopaedic surgeon, neurologist and oncologist. Previous classical examination, imaging (X-rays, CT and MRI) and bloodwork revealed no remedial cause of his pain and no recurrence of his astrocytoma. Prior to referral to CPS, he was already integrated with regular physiotherapy and intermittent massage therapy. Medications at initial assessment were as required tramadol, acetaminophen, and ibuprofen. myoActivation examination revealed a myofascial component to his pain. In view of distressing pain symptoms, it was agreed that myoActivation should be performed at the initial assessment to help relieve his pain. Written consent was obtained and myoActivation information given to the family. Distraction techniques and vapocoolant spray were used to minimise procedural pain.

The left external oblique, left subscapularis, left platysma were released based on structured regional tests for the shoulder [116]. After this first myoActivation session, the patient experienced immediate improvement in range of motion of the left shoulder and decreased pain. Eight days after this myoActivation session the patient reported that his left shoulder pain was "way better than before" and his mum reported that he was complaining less about his shoulder and was able to sleep better. He had some ongoing left shoulder pain. Repeat myoActivation was performed at one-month intervals using BASE tests and regional test for shoulder function. On the fourth and final myoActivation session his left foot scars were released. As foot scars can be painful, even with appropriately applied topical anaesthetic and vapocoolant spray, it was agreed to release these scars with procedural sedation using standard anaesthetic monitoring and care.

At the time of discharge, 3 months after initial assessment, he had no pain, he was able to function physically within the restrictions of his existing hemiplegia, but with no pain, and was attending school full time. He was discharged from CPS care and advised to slowly wean MgBis/K2/D3. Fourteen months after discharge he remains pain free.

4.3.1 Key points

- In the presentation of severe ongoing pain, it is sometimes important to address regional tests, like the shoulder in this case before addressing potential myofascial triggers in BASE tests.

- For some children and adolescents, there is a need for a general anaesthetic to perform needling of scars or trigger points: for example, in anxiety/mood disorder, young age, non-verbal patients, or where it will be considered too painful (e.g., foot scars) as in this case.

- Distant site same-side scars can be clinically important with respect to the tension they create through myofascial chains and biotensegrity principles described above. For example, left foot scar release to resolve left shoulder pain as outlined in this case.

4.4 Case 4: standard myoActivation assessment and treatment of multisite pain

A 15-year-old, 57 kg female was referred to the CPS by her orthopaedic physician for management of right knee pain of 3 years duration with a diagnosis of "possible tendonitis or tendinosis of medial hamstring" with normal blood work and imaging (X-rays and MRI). Her main hobby was horse riding and she had sustained multiple injuries related to her recreational activities. She related her knee pain to one of these injuries, 3 years ago, when she was bucked off her horse and then the horse stood on her right knee. Immediately after this injury, she attended the local ER and was discharged after normal imaging. She initially mobilised with crutches and gradually regained physical functioning over the course of the subsequent 2 months.

She described her current pain as dull and achy with stabbing pain elements radiating up her leg and down into her calf. Pain was aggravated by running, horse riding, doing squats, or any other physical activities or exercise. She had to wear a knee brace and only used the left stirrup when riding her horse as use of the right stirrup aggravated her right knee pain. She reported no effect on mood related to her pain. She had not been absent from school in the previous 3 months. She reported that pain sometimes affected her ability to fall asleep. Her TiLT inventory revealed

multiple horse-riding related injuries. She reported that she had scars from a recent laparoscopic appendicectomy and scars on her right knee from a wire penetrating injury when she was aged 6. She had been using over-the-counter simple analgesia as required. She was otherwise healthy and her family history was unremarkable.

myoActivation examination revealed unreported scars on the right knee and the abdomen, fascia in tension, and multiple muscles in sustained contraction. A myofascial component to her pain was diagnosed. She was enrolled in the 3P care plan. She was started on MgBis/K2/D3. Written information about myoActivation was given to the family.

4.4.1 myoActivation session 1

Five weeks later she reported feeling better and stronger, but no real change in her knee pain. It was deemed appropriate to proceed with myoActivation at this time. Written consent was obtained. Distraction techniques, vapocoolant spray and topical anaesthetic for scars were used to minimise procedural pain. All needling was done using sterile technique.

The right thoracic paraspinal, and right gluteus medius were sequentially activated based on the repeat cycles of assessment to determine the most restrictive and painful BASE tests [116]. The three appendicectomy scars and right knee scar were in regions of worst BASE tests so they were considered significant and released. After this first myoActivation session, the patient experienced immediate improvement in pain and range of motion in all BASE tests.

4.4.2 myoActivation session 2

Three weeks after her first myoActivation session, the patient reported that her right knee pain had moved down to the inner aspect of the right calf. The pain felt was less than on initial assessment. She reported that she could sit in saddle, horse riding for longer, but found eversion of her ankles painful. The left iliopsoas and left rectus abdominis were sequentially activated based on the repeat cycles of assessment to determine the most restrictive and painful BASE tests [116]. The three appendicectomy scars were again in regions of worst BASE tests so they were considered significant and re-released. After this myoActivation session, the patient experienced immediate improvement in pain and range of motion in all BASE tests.

Three months after initial assessment, she reported she could move better, had less pain, was horse riding for much longer with both feet in the stirrups. She required no over-the-counter analgesia medications. On examination, she had normal movements in all core BASE tests with no pain. She was discharged from CPS care and advised to slowly wean MgBis/K2/D3.

4.4.3 Key points

- The site of pain may not be the true source of pain. The right hamstrings were never activated, even though they were the initial site of pain.

- Release of a scar from the youngest age in the region of most restrictive or painful myoActivation BASE test can make clinically substantial change, as was the case for this girl.

- Scars acquired after onset of pain may also play an important role in a myofascial pain presentation.

- This case again illustrates that myoActivation, as a component of multidisciplinary care, enhances recovery even in patients with a history of years in pain and multiple injuries.

4.5 Case 5: standard myoActivation assessment and treatment of chronic multi-site pain secondary to trauma related pelvic and femoral fractures

A 16-year old, 57 kg, female was referred to the CPS for management of multisite pain related to injuries sustained when hit by a car whilst crossing a road. She was referred by her orthopaedic surgeon, 1 year following injury, when ongoing recovery of physical functioning was hampered by multisite pain. She had been fit and healthy prior to this accident.

She was a pedestrian who was struck on the right side and knocked down sustaining multiple pelvic ring fractures and a left femoral fracture. She sustained no other injuries. She required external fixators and an intramedullary femoral rod to stabilise her fractures and control blood loss at the time of admission. She was an inpatient in hospital for a month and then transferred for inpatient rehabilitation at a local rehabilitation centre. The external fixator was removed 2 months after application. She was discharged from the rehabilitation centre 5 months after the date of her presenting injury. She continued with ongoing outpatient physiotherapy, kinesiology, daily exercises and regular counselling but had recently stopped seeing a physiotherapist due to lack of financial support. Imaging confirmed normal healing and no complications related to the surgical sites.

The pain was multisite, affecting the upper, mid and low back, left knee, bilateral thighs, and bilateral ankles with no radiation and no motor or sensory deficits. Her pain was variable and intermittent, but aggravated by exercise. She had been absent from school for 5 days in the previous 3 months due to pain. She reported that pain also affected her ability to fall and stay asleep. Her lifetime trauma history revealed no other injuries. She took ibuprofen as required. She was otherwise healthy and her family history was unremarkable.

myoActivation examination revealed a myofascial component to her pain. Scars were noted from the external fixator and at the site of the left intramedullary nail. There were also abrasion scars from the injury on the left thigh and hip. She was enrolled in the 3P care plan and was started on MgBis/K2/D3. Written myoActivation information was given to the family.

One month later, there was a reduction in pain with improved fluidity of movements just with the addition of MgBis/K2/D3 and re-engagement with physiotherapy. It was deemed appropriate to proceed with myoActivation at this one-month follow-up visit, based on her very stoical character. Written consent was obtained. Distraction techniques and vapocoolant spray were used to minimise procedural pain. Fascial densification, muscles in sustained contraction and the scars in areas of most significant BASE tests [116] were released. Release of the surgical scars and the scars related to the injury made a clinically significant change in pain and range of motion during these sessions.

With such significant injuries and multiple areas required to be treated, she required nine myoActivation session in the course of the following year whilst integrated with the CPS. At the time of discharge, 16 months after initial assessment, she was able to function physically with no restrictions and minimal pain. She was sleeping well and successfully graduated from school with good marks. She noted that her scars were paler, flatter and less obvious than at the start of therapy. She was discharged from CPS care and advised to slowly wean MgBis/K2/D3.

4.5.1 Key points

- Patient reported change in function, just related to institution of the 3P approach, massage, and MgBis/K2/D3, helps to confirm a myofascial component to pain.

- This approach, with the addition of myoActivation, can have a substantial impact even in the presence of such devastating previous injuries.

- Release of the surgical scars and the scars related to the injury made clinically significant change for this patient.

The cases cited above have provided a view of how scars have made a substantial impact in pain presentation. Scar release (using a needling technique) helped make meaningful change for these patients, often when all other avenues of classic medical care for pain aetiology and resolution had been exhausted. Not only is this rewarding for the patients and their families, but also the CPS team who are helping them in their journey to recovery.

5. Conclusion

Humans exhibit biotensegrity, where each individual part of the organism combines with the mechanical system to create an integrated functional movement unit. All structures of the human body are intricately connected through skin and the myofascial system, they are in a continual process of change dependant on and adapting to the forces acting on and within them. When tissue is breached by surgery or injury, the healing process leads to the formation of scar tissue. Scars can limit normal movement of underlying and remote tissues. Defective fascial sliding, secondary to scars, generates anomalous tension that affects the fascial continuum and may lead to distorted biomechanics and chronic pain.

Scars are common in the paediatric population and are significant contributory factors to chronic pain. Many years, even decades, may pass between scar acquisition and the development of biomechanical dysfunction or myofascial pain. A subsequent trauma may be the inciting event as force transmission throughout the body is changed by a scar. Hence, it is important to assess the TiLT inventory, and characteristics of all scars, even when they appear to look "normal".

There are many characteristics of a scar that disrupt the myofascial system, which have been highlighted in the cases discussed. When scars are deemed significant, scar release should be considered as one component in a multidisciplinary approach to address the whole biopsychosocial aspects of chronic pain [136]. Scar and myofascial release, with soft tissue mobilisation or needling techniques, can result in immediate and sustainable improvements in pain, flexibility, and range of motion [116]. When contemplating scar release, consideration should be given to minimising procedural pain and to ensure support for any emotional reactions that may occur immediately or within the hours following scar release.

Research is required to ascertain the exact characteristics of a scar that determine whether it will have a significant contribution to myofascial dysfunction and chronic pain. The cases presented above illustrate that these investigations must recognise the multiple and complex biopsychosocial factors that contribute to a myofascial chronic pain presentation.

Whilst definitive answers are awaited, we need to think beyond scars as just being innocuous mementos of the past. Clinical experience indicates that they may

be restrictive barriers, exerting pervasive biomechanical and nociceptive effects in the present. Left untreated, that "*strange power" of a scar* may go on to have substantial impact in the future as well.

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Conflict of interest

The authors have no conflicts of interest to declare.

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References

[1] Jablonski NG. Skin: A Natural History. Berkeley, CA: University of California Press; 2006

[2] Virador GM, de Marcos L, Virador VM. Skin wound healing: Refractory wounds and novel solutions. In: Turksen K, editor. Skin Stem Cells: Methods and Protocols. Totowa, NJ: Humana Press; 2018. pp. 221-241

[3] Keppel Hesselink J, Kopsky D, Bhaskar A. Skin matters! The role of keratinocytes in nociception: A rational argument for the development of topical analgesics. Journal of Pain Research. 2016;**10**:1-8

[4] González-Hernández A, Manzano-García A, Martínez-Lorenzana G, Tello-García IA, Carranza M, Arámburo C, et al. Peripheral oxytocin receptors inhibit the nociceptive input signal to spinal dorsal horn wide-dynamic-range neurons. Pain. 2017;**158**(11):2117-2128

[5] Cunha TM, Verri WA, Silva JS, Poole S, Cunha FQ, Ferreira SH. A cascade of cytokines mediates mechanical inflammatory hypernociception in mice. Proceedings of the National Academy of Sciences. 2005;**102**(5):1755-1760

[6] Manjavachi MN, Costa R, Quintão NL, Calixto JB. The role of keratinocyte-derived chemokine (KC) on hyperalgesia caused by peripheral nerve injury in mice. Neuropharmacology. 2014;**79**:17-27

[7] Pang Z, Sakamoto T, Tiwari V, Kim Y-S, Yang F, Dong X, et al. Selective keratinocyte stimulation is sufficient to evoke nociception in mice. Pain. 2015;**156**(4):656-665

[8] Drummond PD, Dawson LF, Finch PM, Drummond ES, Wood FM, Fear MW. Up-regulation of cutaneous α1-adrenoceptors after a burn. Burns. 2015;**41**(6):1227-1234

[9] Yang S, Sun Y, Geng Z, Ma K, Sun X, Fu X. Abnormalities in the basement membrane structure promote basal keratinocytes in the epidermis of hypertrophic scars to adopt a proliferative phenotype. International Journal of Molecular Medicine. 2016;**37**(5):1263-1273

[10] de Vries HJC, Enomoto DNH, van Marle J, van Zuijlen PPM, Mekkes JR, Bos JD. Dermal organization in Scleroderma: The fast Fourier transform and the laser scatter method objectify fibrosis in nonlesional as well as lesional skin. Laboratory Investigation. 2000;**80**(8):1281-1289

[11] Verhaegen PDHM, van Zuijlen PPM, Pennings NM, van Marle J, Niessen FB, van der Horst CMAM, et al. Differences in collagen architecture between keloid, hypertrophic scar, normotrophic scar, and normal skin: An objective histopathological analysis. Wound Repair and Regeneration. 2009;**17**(5):649-656

[12] des Jardins-Park HE, Foster DS, Longaker MT. Fibroblasts and wound healing: An update. Regenerative Medicine. 2018;**13**(5):491-495

[13] Dalsgaard CJ, Rydh M, Haegerstrand A. Cutaneous innervation in man visualized with protein gene product 9.5 (PGP 9.5) antibodies. Histochemistry. 1989;**92**(5):385-390

[14] Caron A, Lee S, Elmquist JK, Gautron L. Leptin and brain–adipose crosstalks. Nature Reviews. Neuroscience. 2018;**19**(3):153-165

[15] Kwon O, Kim KW, Kim M-S. Leptin signalling pathways in hypothalamic neurons. Cellular and Molecular Life Sciences. 2016;**73**(7):1457-1477

[16] Maeda N, Funahashi T, Matsuzawa Y, Shimomura I. Adiponectin, a unique adipocyte-derived factor beyond hormones. Atherosclerosis. 2020;**292**:1-9

[17] Rivera-Gonzalez G, Shook B, Horsley V. Adipocytes in skin health and disease. Cold Spring Harbor Perspectives in Medicine. 2014;**4**(3):a015271-a015271

[18] Bordoni B, Zanier E. Skin, fascias, and scars: Symptoms and systemic connections. Journal of Multidisciplinary Healthcare. 2013;**7**:11-24

[19] Di Meglio P, Perera GK, Nestle FO. The multitasking organ: Recent insights into skin immune function. Immunity. 2011;**35**(6):857-869

[20] Jang SH, Seo JP, Ahn SH, Lee MY. Comparison of cortical activation patterns by somatosensory stimulation on the palm and dorsum of the hand. Somatosensory & Motor Research. 2013;**30**(3):109-113

[21] Mondelli M, Aretini A, Ballerini M, Vecchiarelli B, Rossi A. Sympathetic skin response. Glabella stimulation may be more useful than peripheral nerve stimulation in clinical practice. Autonomic Neuroscience. 2011;**164**(1-2):101-104

[22] Henderson LA, Stathis A, James C, Brown R, McDonald S, Macefield VG. Real-time imaging of cortical areas involved in the generation of increases in skin sympathetic nerve activity when viewing emotionally charged images. NeuroImage. 2012;**62**(1):30-40

[23] Bordoni B, Zanier E. Clinical and symptomatological reflections: The fascial system. Journal of Multidisciplinary Healthcare. 2014;**7**: 401-411

[24] Miranda A, Peles S, Rudolph C, Shaker R, Sengupta JN. Altered visceral sensation in response to somatic pain in the rat☆. Gastroenterology. 2004;**126**(4):1082-1089

[25] Albrecht PJ, Hou Q , Argoff CE, Storey JR, Wymer JP, Rice FL. Excessive Peptidergic sensory innervation of cutaneous arteriole-venule shunts (AVS) in the palmar glabrous skin of fibromyalgia patients: Implications for widespread deep tissue pain and fatigue. Pain Medicine. 2013;**14**(6):895-915

[26] Peppin JF, Albrecht PJ, Argoff C, Gustorff B, Pappagallo M, Rice FL, et al. Skin matters: A review of topical treatments for chronic pain. Part one: Skin physiology and delivery systems. Pain and therapy. 2015;**4**(1):17-32

[27] Driscoll M. Fascia – The unsung hero of spine biomechanics. Journal of Bodywork and Movement Therapies. 2018;**22**(1):90-91

[28] Adstrum S, Nicholson H. A history of fascia. Clinical Anatomy. 2019;**32**(7):862-870

[29] Adstrum S, Hedley G, Schleip R, Stecco C, Yucesoy CA. Defining the fascial system. Journal of Bodywork and Movement Therapies. 2017;**21**(1):173-177

[30] Stecco C, Adstrum S, Hedley G, Schleip R, Yucesoy CA. Update on fascial nomenclature. Journal of Bodywork and Movement Therapies. 2018;**22**(2):354

[31] Avila Gonzalez CA, Driscoll M, Schleip R, Wearing S, Jacobson E, Findley T, et al. Frontiers in fascia research. Journal of Bodywork and Movement Therapies. 2018;**22**(4): 873-880

[32] Stecco C, Fede C, Macchi V, Porzionato A, Petrelli L, Biz C, et al. The fasciacytes: A new cell devoted to fascial gliding regulation. Clinical Anatomy. 2018;**31**(5):667-676

[33] Hughes EJ, McDermott K, Funk MF. Evaluation of hyaluronan content in areas of densification compared to adjacent areas of fascia. Journal of

Bodywork and Movement Therapies. 2019;**23**(2):324-328

[34] Stecco C, Porzionato A, Lancerotto L, Stecco A, Macchi V, Ann Day J, et al. Histological study of the deep fasciae of the limbs. Journal of Bodywork and Movement Therapies. 2008;**12**(3):225-230

[35] Stecco C, Stern R, Porzionato A, Macchi V, Masiero S, Stecco A, et al. Hyaluronan within fascia in the etiology of myofascial pain. Surgical and Radiologic Anatomy. 2011;**33**(10):891-896

[36] Wilke J, Krause F, Vogt L, Banzer W. What is evidence-based about myofascial chains: A systematic review. Archives of Physical Medicine and Rehabilitation. 2016;**97**(3):454-461

[37] Kjaer M, Langberg H, Heinemeier K, Bayer ML, Hansen M, Holm L, et al. From mechanical loading to collagen synthesis, structural changes and function in human tendon. Scandinavian Journal of Medicine & Science in Sports. 2009;**19**(4):500-510

[38] Myers B. Wound Management: Principles and Practices. 3rd ed. Pearson: London, UK; 2012

[39] Bei Y, Wang F, Yang C, Xiao J. Telocytes in regenerative medicine. Journal of Cellular and Molecular Medicine. 2015;**19**(7):1441-1454

[40] Scarr G. Biotensegrity: The Structural Basis of Life. Pencaitland, UK: Handspring Publishing Limited; 2014

[41] Krause F, Wilke J, Vogt L, Banzer W. Intermuscular force transmission along myofascial chains: A systematic review. Journal of Anatomy. 2016;**228**(6):910-918

[42] Tidball JG. Force transmission across muscle cell membranes. Journal of Biomechanics. 1991;**24**:43-52

[43] Zügel M, Maganaris CN, Wilke J, Jurkat-Rott K, Klingler W, Wearing SC, et al. Fascial tissue research in sports medicine: From molecules to tissue adaptation, injury and diagnostics: Consensus statement. British Journal of Sports Medicine. 2018;**52**(23):1497-1497

[44] Dischiavi SL, Wright AA, Hegedus EJ, Bleakley CM. Biotensegrity and myofascial chains: A global approach to an integrated kinetic chain. Medical Hypotheses. 2018;**110**:90-96

[45] Tak I, Glasgow P, Langhout R, Weir A, Kerkhoffs G, Agricola R. Hip range of motion is lower in professional soccer players with hip and groin symptoms or previous injuries, independent of cam deformities. The American Journal of Sports Medicine. 2016;**44**(3):682-688

[46] Tozzi P. Selected fascial aspects of osteopathic practice. Journal of Bodywork and Movement Therapies. 2012;**16**(4):503-519

[47] Stecco C, Gagey O, Belloni A, Pozzuoli A, Porzionato A, Macchi V, et al. Anatomy of the deep fascia of the upper limb. Second part: Study of innervation. Morphologie. 2007;**91**(292):38-43

[48] van der Wal J. The architecture of the connective tissue in the musculoskeletal system-an often overlooked functional parameter as to proprioception in the locomotor apparatus. International Journal of Therapeutic Massage & Bodywork: Research, Education, & Practice. 2009;**2**(4):9-23

[49] Bordoni B, Myers T. A review of the theoretical Fascial models: Biotensegrity, fascintegrity, and myofascial chains. Cureus. 2020;**12**(2):e7092

[50] Varela F, Frenk S. The organ of form: Towards a theory of biological shape. Journal of Social and Biological Systems. 1987;**10**(1):73-83

[51] Susan S, editor. Gray's Anatomy: The Anatomical Basis of Clinical Practice. 41st ed. Amsterdam, The Netherlands: Elsevier; 2015

[52] Karamanos NK, Theocharis AD, Neill T, Iozzo RV. Matrix modeling and remodeling: A biological interplay regulating tissue homeostasis and diseases. Matrix Biology. 2019;**75-76**:1-11

[53] Gantwerker EA, Hom DB. Skin: Histology and physiology of wound healing. Clinics in Plastic Surgery. 2012;**39**(1):85-97

[54] Bran GM, Goessler UR, Hormann K, Riedel F, Sadick H. Keloids: Current concepts of pathogenesis (review). International Journal of Molecular Medicine. 2009;**24**(3):283-293

[55] Lichtman MK, Otero-Vinas M, Falanga V. Transforming growth factor beta (TGF-β) isoforms in wound healing and fibrosis. Wound Repair and Regeneration. 2016;**24**(2):215-222

[56] Sorg H, Tilkorn DJ, Hager S, Hauser J, Mirastschijski U. Skin wound healing: An update on the current knowledge and concepts. European Surgical Research. 2017;**58**(1-2):81-94

[57] Bijlard E, Uiterwaal L, Kouwenberg CAE, Mureau MAM, Hovius SER, Huygen FJPM. A systematic review on the prevalence, etiology, and pathophysiology of intrinsic pain in dermal scar tissue. Pain Physician. 2017;**20**(2):1-13

[58] Zhang L, Laato M. Innervation of normal and hypertrophic human scars and experimental wounds in the rat. Annales Chirurgiae et Gynaecologiae. 2001;**90**(Suppl 215):29-32

[59] Ogawa R. Keloid and hypertrophic scars are the result of chronic inflammation in the reticular dermis. International Journal of Molecular Sciences. 2017;**18**(3):606

[60] Karkampouna S, Kreulen M, Obdeijn MC, Kloen P, Dorjée AL, Rivellese F, et al. Connective tissue degeneration: Mechanisms of palmar fascia degeneration (Dupuytren's disease). Current Molecular Biology Reports. 2016;**2**(3):133-140

[61] Roseborough I, Grevious M, Lee R. Prevention and treatment of excessive dermal scarring. Journal of the National Medical Association. 2004;**96**(1):108-116

[62] Akaishi S, Ogawa R, Hyakusoku H. Keloid and hypertrophic scar: Neurogenic inflammation hypotheses. Medical Hypotheses. 2008;**71**(1):32-38

[63] Ogawa R. Mechanobiology of scarring. Wound Repair and Regeneration. 2011;**19**:s2-s9

[64] Miyamoto J, Nagasao T, Miyamoto S, Nakajima T. Biomechanical analysis of stresses occurring in vertical and transverse scars on the lower leg. Plastic and Reconstructive Surgery. 2009;**124**(6):1974-1979

[65] Wolfram D, Tzankov A, Pülzl P, Piza-Katzer H. Hypertrophic scars and keloids—A review of their pathophysiology, risk factors, and therapeutic management. Dermatologic Surgery. 2009;**35**(2):171-181

[66] Midwood KS, Williams LV, Schwarzbauer JE. Tissue repair and the dynamics of the extracellular matrix. The International Journal of Biochemistry & Cell Biology. 2004;**36**(6):1031-1037

[67] el Hayderi L, Nikkels-Tassoudji N, Nikkels AF. Incidence of and risk factors for cutaneous scarring after herpes zoster. American Journal of Clinical Dermatology. 2018;**19**(6):893-897

[68] Leung AKC, Kao CP, Sauve RS. Scarring resulting from chickenpox. Pediatric Dermatology. 2001;**18**(5): 378-380

[69] Connolly D, Vu HL, Mariwalla K, Saedi N. Acne scarring-pathogenesis, evaluation, and treatment options. The Journal of Clinical and Aesthetic Dermatology. 2017;**10**(9):12-23

[70] Krakowski AC, Totri CR, Donelan MB, Shumaker PR. Scar Management in the pediatric and adolescent populations. Pediatrics. 2016;**137**(2):e20142065-e20142065

[71] Reinholz M, Schwaiger H, Poetschke J, Epple A, Ruzicka T, Von Braunmühl T, et al. Objective and subjective treatment evaluation of scars using optical coherence tomography, sonography, photography, and standardised questionnaires. European Journal of Dermatology. 2016;**26**(6):599-608

[72] Cheng W, Saing H, Zhou H, Han Y, Peh W, Tam PKH. Ultrasound assessment of scald scars in Asian children receiving pressure garment therapy. Journal of Pediatric Surgery. 2001;**36**(3):466-469

[73] Simons M, Kee EG, Kimble R, Tyack Z. Ultrasound is a reproducible and valid tool for measuring scar height in children with burn scars: A cross-sectional study of the psychometric properties and utility of the ultrasound and 3D camera. Burns. 2017;**43**(5):993-1001

[74] Ferriero G, Di Carlo S, Ferriero A, Salgovic L, Bravini E, Sartorio F, et al. Post-surgical scar assessment in rehabilitation: A systematic review. Physical Therapy Rehabilitation. 2015;**2**(1):2

[75] Klingler W, Velders M, Hoppe K, Pedro M, Schleip R. Clinical relevance of fascial tissue and dysfunctions. Current Pain and Headache Reports. 2014;**18**(8):439

[76] Martínez Rodríguez R, Galán del Río F. Mechanistic basis of manual therapy in myofascial injuries. Sonoelastographic evolution control. Journal of Bodywork and Movement Therapies. 2013;**17**(2):221-234

[77] Guo R, Xiang X, Wang L, Zhu B, Cheng S, Qiu L. Quantitative assessment of keloids using ultrasound shear wave elastography. Ultrasound in Medicine & Biology. 2020;**46**(5):1169-1178

[78] DeJong HM, Abbott S, Zelesco M, Kennedy BF, Ziman MR, Wood FM. The validity and reliability of using ultrasound elastography to measure cutaneous stiffness, a systematic review. International Journal of Burns and Trauma. 2017;**7**(7):124-141

[79] Perry DM, McGrouther DA, Bayat A. Current tools for noninvasive objective assessment of skin scars. Plastic and Reconstructive Surgery. 2010;**126**(3):912-923

[80] Gold M, Berman B, Clementoni M, Gauglitz G, Nahai F, Murcia C. Updated international clinical recommendations on scar management: Part 1--evaluating the evidence. Dermatologic Surgery. 2014;**40**(8):817-824

[81] Gold MH, McGuire M, Mustoe TA, Pusic A, Sachdev M, Waibel J, et al. Updated international clinical recommendations on scar management: Part 2--algorithms for scar prevention and treatment. Dermatologic Surgery. 2014;**40**(8):825-831

[82] Arno AI, Gauglitz GG, Barret JP, Jeschke MG. Up-to-date approach to manage keloids and hypertrophic scars: A useful guide. Burns. 2014;**40**(7):1255-1266

[83] Arno AI, Gauglitz GG, Barret JP, Jeschke MG. New molecular medicine-based scar management strategies. Burns. 2014;**40**(4):539-551

[84] Wasserman JB, Steele-Thornborrow JL, Yuen JS, Halkiotis M, Riggins EM.

Chronic caesarian section scar pain treated with fascial scar release techniques: A case series. Journal of Bodywork and Movement Therapies. 2016;**20**(4):906-913

[85] Nikolajsen L, Sørensen HC, Jensen TS, Kehlet H. Chronic pain following caesarean section. Acta Anaesthesiologica Scandinavica. 2004;**48**(1):111-116

[86] de Brito Cançado TO, Omais M, Ashmawi HA, Torres MLA. Chronic pain after cesarean section. Influence of anesthetic/surgical technique and postoperative analgesia. Brazilian Journal of Anesthesiology. 2012;**62**(6):762-774

[87] Scott JR, Muangman P, Gibran NS. Making sense of hypertrophic scar: A role for nerves. Wound Repair and Regeneration. 2007;**15**(Suppl 1): S27-S31

[88] Shayani V, Siegert C, Favia P. The role of laparoscopic adhesiolysis in the treatment of patients with chronic abdominal pain or recurrent bowel obstruction. JSLS. 2002;**6**(2):111-114

[89] Kindermann G, Debus-Thiede G. 18 postoperative urological complications after radical surgery for cervical cancer. Baillière's Clinical Obstetrics and Gynaecology. 1988;**2**(4):933-941

[90] Isla A, Alvarez F. Fibrosis epidural espinal postdiscectomía lumbar y barrera antiadhesiva. Neurocirugia. 2001;**12**(5):439-446

[91] Lewit K, Olsanska S. Clinical importance of active scars: Abnormal scars as a cause of myofascial pain. Journal of Manipulative and Physiological Therapeutics. 2004;**27**(6):399-402

[92] Kobesova A, Morris CE, Lewit K, Safarova M. Twenty-year-old pathogenic "active" postsurgical scar: A case study of a patient with persistent right lower quadrant pain. Journal of Manipulative and Physiological Therapeutics. 2007;**30**(3):234-238

[93] Valouchová P, Lewit K. Surface electromyography of abdominal and back muscles in patients with active scars. Journal of Bodywork and Movement Therapies. 2009;**13**(3):262-267

[94] Wasserman JB, Copeland M, Upp M, Abraham K. Effect of soft tissue mobilization techniques on adhesion-related pain and function in the abdomen: A systematic review. Journal of Bodywork and Movement Therapies. 2019;**23**(2):262-269

[95] Serra-Añó P, Inglés M, Bou-Catalá C, Iraola-Lliso A, Espí-López GV. Effectiveness of myofascial release after breast cancer surgery in women undergoing conservative surgery and radiotherapy: A randomized controlled trial. Supportive Care in Cancer. 2019;**27**(7): 2633-2641

[96] McKay RMTE. Assessing the effectiveness of massage therapy for bilateral cleft lip reconstruction scars. The International Journal of Therapeutic Massage & Bodywork: Research, Education, & Practice. 2014;**7**(2):3-9

[97] Myers TW. Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists. 2nd ed. London, UK: Churchill Livingstone; 2008

[98] Alvira-Lechuz J, Espiau MR, Alvira-Lechuz E. Treatment of the scar after arthroscopic surgery on a knee. Journal of Bodywork and Movement Therapies. 2017;**21**(2):328-333

[99] Benjamin M. The fascia of the limbs and back--a review. Journal of Anatomy. 2009;**214**(1):1-18

[100] Stecco A, Stern R, Fantoni I, De Caro R, Stecco C. Fascial disorders:

Implications for treatment. PM & R: The Journal of Injury, Function, and Rehabilitation. 2016;**8**(2):161-168

[101] Bednar DA, Orr FW, Simon GT. Observations on the pathomorphology of the thoracolumbar fascia in chronic mechanical back pain. A microscopic study. Spine (Phila Pa 1976). 1995; **20**(10):1161-1164

[102] Langevin HM, Fox JR, Koptiuch C, Badger GJ, Greenan-Naumann AC, Bouffard NA, et al. Reduced thoracolumbar fascia shear strain in human chronic low back pain. BMC Musculoskeletal Disorders. 2011;**12**:203

[103] Chaitow L. Somatic dysfunction and fascia's gliding-potential. Journal of Bodywork and Movement Therapies. 2014;**18**(1):1-3

[104] Wilke J, Schleip R, Yucesoy CA, Banzer W. Not merely a protective packing organ? A review of fascia and its force transmission capacity. Journal of Applied Physiology. 2018;**124**(1):234-244

[105] Schleip R, Zorn A, Klingler W. Biomechanical properties of fascial tissues and their role as pain generators. The Journal of Musculoskeletal Pain. 2010;**18**(4):393-395

[106] Kwong EH, Findley TW. Fascia–current knowledge and future directions in physiatry: Narrative review. Journal of Rehabilitation Research and Development. 2014;**51**(6):875-884

[107] Gibson W, Arendt-Nielsen L, Taguchi T, Mizumura K, Graven-Nielsen T. Increased pain from muscle fascia following eccentric exercise: Animal and human findings. Experimental Brain Research. 2009;**194**(2):299-308

[108] Ajimsha MS, Daniel B, Chithra S. Effectiveness of myofascial release in the management of chronic low back pain in nursing professionals. Journal of Bodywork and Movement Therapies. 2014;**18**(2):273-281

[109] Day JA, Stecco C, Stecco A. Application of fascial manipulation© technique in chronic shoulder pain—Anatomical basis and clinical implications. Journal of Bodywork and Movement Therapies. 2009;**13**(2):128-135

[110] Langevin HM, Bishop J, Maple R, Badger GJ, Fox JR. Effect of stretching on thoracolumbar fascia injury and movement restriction in a porcine model. American Journal of Physical Medicine & Rehabilitation. 2018;**97**(3):187-191

[111] Henry R, Cahill CM, Wood G, Hroch J, Wilson R, Cupido T, et al. Myofascial pain in patients waitlisted for total knee arthroplasty. Pain Research & Management. 2012;**17**(5):321-327

[112] Earl J. A small scale study to establish if an abdominal scar is a factor in lower back pain [dissertation]. 2015. Available from: https://jackieearl.co.uk/images/Dissertation_Final_Copy.pdf [Accessed: 12 June 2020]

[113] Willard FH, Vleeming A, Schuenke MD, Danneels L, Schleip R. The thoracolumbar fascia: Anatomy, function and clinical considerations. Journal of Anatomy. 2012;**221**(6):507-536

[114] Ljungqvist A, Schwellnus MP, Bachl N, Collins M, Cook J, Khan KM, et al. International Olympic Committee consensus statement: Molecular basis of connective tissue and muscle injuries in sport. Clinics in Sports Medicine. 2008;**27**(1):231-239

[115] Langevin HM, Stevens-Tuttle D, Fox JR, Badger GJ, Bouffard NA, Krag MH, et al. Ultrasound evidence of altered lumbar connective tissue structure in human subjects with chronic low back pain. BMC Musculoskeletal Disorders. 2009;**10**(1):151

[116] Lauder G, West N, Siren G. myoActivation: A structured process for chronic pain resolution. In: Cascella M, editor. From Conventional to Innovative Approaches for Pain Treatment. London: IntechOpen; 2019. DOI: 10.5772/intechopen.84377

[117] Miazga S, West N, Ng M, Lauder G. myoActivation, a structured system for assessment and management of chronic pain: A retrospective review of pediatric cases. In: University of British Columbia Department of Anesthesiology, Pharmacology & Therapeutics 14th Annual Research Day. Canada: Vancouver; 2020

[118] Minasny B. Understanding the process of fascial unwinding. International Journal of Therapeutic Massage & Bodywork: Research, Education, & Practice. 2009;**2**(3):10-17

[119] Wasserman JB, Abraham K, Massery M, Chu J, Farrow A, Marcoux BC. Soft tissue mobilization techniques are effective in treating chronic pain following cesarean section. Journal of Women's Health Physical Therapy. 2018;**42**(3):111-119

[120] Chung MK, LaRiccia PJ. Successful integrative medicine assessment and treatment of chronic pain associated with breast surgery: A report of 3 cases. Holistic Nursing Practice. 2017;**31**(1):21-29

[121] Costa IMC, Costa MC. Microneedling for varicella scars in a dark-skinned teenager. Dermatologic Surgery. 2014;**40**(3):333-334

[122] Alster TS, Graham PM. Microneedling: A review and practical guide. Dermatologic Surgery. 2018;**44**(3):397-404

[123] Orentreich DS, Orentreich N. Subcutaneous incisionless (subcision) surgery for the correction of depressed scars and wrinkles. Dermatologic Surgery. 1995;**21**(6):543-549

[124] Ramaut L, Hoeksema H, Pirayesh A, Stillaert F, Monstrey S. Microneedling: Where do we stand now? A systematic review of the literature. Journal of Plastic, Reconstructive & Aesthetic Surgery. 2018;**71**(1):1-14

[125] Hou A, Cohen B, Haimovic A, Elbuluk N. Microneedling: A comprehensive review. Dermatologic Surgery. 2017;**43**(3):321-339

[126] Schwarz M, Laaff H. A prospective controlled assessment of microneedling with the dermaroller device. Plastic and Reconstructive Surgery. 2011;**127**(6):146e-148e

[127] Nair P, Arora T. Microneedling using dermaroller a means of collagen induction therapy. Gujarat Medical Journal. 2014;**69**(1):24-27

[128] Aust MC, Fernandes D, Kolokythas P, Kaplan HM, Vogt PM. Percutaneous collagen induction therapy: An alternative treatment for scars, wrinkles, and skin laxity. Plastic and Reconstructive Surgery. 2008;**121**(4):1421-1429

[129] Aust MC, Knobloch K, Reimers K, Redeker J, Ipaktchi R, Altintas MA, et al. Percutaneous collagen induction therapy: An alternative treatment for burn scars. Burns. 2010;**36**(6):836-843

[130] Aust MC, Reimers K, Kaplan HM, Stahl F, Repenning C, Scheper T, et al. Percutaneous collagen induction–regeneration in place of cicatrisation? Journal of Plastic, Reconstructive & Aesthetic Surgery. 2011;**64**(1):97-107

[131] Doddaballapur S. Microneedling with dermaroller. Journal of Cutaneous and Aesthetic Surgery. 2009;**2**(2):110

[132] Fabbrocini G, Fardella N, Monfrecola A, Proietti I, Innocenzi D. Acne scarring treatment using skin needling. Clinical and Experimental Dermatology. 2009;**34**(8):874-879

[133] Bonati LM, Epstein GK, Strugar TL. Microneedling in all skin types: A review. Journal of Drugs in Dermatology. 2017;**16**(4):308-313

[134] Cagnie B, Dewitte V, Barbe T, Timmermans F, Delrue N, Meeus M. Physiologic effects of dry needling. Current Pain and Headache Reports. 2013;**17**(8):348

[135] Liu X, Zhu B, Zhang SX. Relationship between electroacupuncture analgesia and descending pain inhibitory mechanism of nucleus raphe magnus. Pain. 1986;**24**(3):383-396

[136] Lauder GR, Huang J, West NC. Unrecognized myofascial components of pediatric complex pain: myoActivation, a structured solution for assessment and management. Current Trends in Medicine. 2019;**1**(1):1-16

[137] Tozzi P. Does fascia hold memories? Journal of Bodywork and Movement Therapies. 2014;**18**(2):259-265

[138] Rothschild B. The Body Remembers: The Psychophysiology of Trauma and Trauma Treatment. New York: W. W. Norton & Company; 2000

[139] Hakala P, Rimpelä A, Salminen JJ, Virtanen SM, Rimpelä M. Back, neck, and shoulder pain in Finnish adolescents: National cross sectional surveys. BMJ. 2002;**325**(7367):743

[140] Sperotto F, Brachi S, Vittadello F, Zulian F. Musculoskeletal pain in schoolchildren across puberty: A 3-year follow-up study. Pediatric Rheumatology Online Journal. 2015;**13**:16

[141] Huguet A, Tougas ME, Hayden J, McGrath PJ, Stinson JN, Chambers CT. Systematic review with meta-analysis of childhood and adolescent risk and prognostic factors for musculoskeletal pain. Pain. 2016;**157**(12):2640-2656

[142] Silva GRR, Pitangui ACR, Xavier MKA, Correia-Júnior MAV, De Araújo RC. Prevalence of musculoskeletal pain in adolescents and association with computer and videogame use. Jornal de Pediatria. 2016;**92**(2):188-196

[143] Queiroz LB, Lourenço B, Silva LEV, Lourenço DMR, Silva CA. Musculoskeletal pain and musculoskeletal syndromes in adolescents are related to electronic devices. Jornal de Pediatria. 2018;**94**(6): 673-679

[144] Leino-Arjas P, Rajaleid K, Mekuria G, Nummi T, Virtanen P, Hammarström A. Trajectories of musculoskeletal pain from adolescence to middle age: The role of early depressive symptoms, a 27-year follow-up of the northern Swedish cohort. Pain. 2018;**159**(1):67-74

[145] Kyu HH, Pinho C, Wagner JA, Brown JC, Bertozzi-Villa A, Charlson FJ, et al. Global and national burden of diseases and injuries among children and adolescents between 1990 and 2013. JAMA Pediatrics. 2016;**170**(3):267

[146] King S, Chambers CT, Huguet A, MacNevin RC, McGrath PJ, Parker L, et al. The epidemiology of chronic pain in children and adolescents revisited: A systematic review. Pain. 2011;**152**(12):2729-2738

[147] Jones GT, Macfarlane GJ. Epidemiology of low back pain in children and adolescents. Archives of Disease in Childhood. 2005;**90**(3):312-316

[148] Salminen JJ. The adolescent back. A field survey of 370 Finnish schoolchildren. Acta Paediatrica Scandinavica. Supplement. 1984;**315**: 1-122

[149] Oksanen AM, Laimi K, Löyttyniemi E, Kunttu K. Trends of

weekly musculoskeletal pain from 2000 to 2012: National study of Finnish university students. European Journal of Pain. 2014;**18**(9):1316-1322

[150] Mikkelsson M, El-Metwally A, Kautiainen H, Auvinen A, Macfarlane GJ, Salminen JJ. Onset, prognosis and risk factors for widespread pain in schoolchildren: A prospective 4-year follow-up study. Pain. 2008;**138**(3): 681-687

[151] Gobina I, Villberg J, Välimaa R, Tynjälä J, Whitehead R, Cosma A, et al. Prevalence of self-reported chronic pain among adolescents: Evidence from 42 countries and regions. European Journal of Pain. 2019;**23**(2):316-326

[152] van den Heuvel MM, Jansen PW, Bindels PJE, Bierma-Zeinstra SMA, van Middelkoop M. Musculoskeletal pain in 6-year-old children. Pain. 2020;**161**(6):1278-1285

[153] Friedrichsdorf SJ, Giordano J, Desai Dakoji K, Warmuth A, Daughtry C, Schulz CA. Chronic pain in children and adolescents: Diagnosis and treatment of primary pain disorders in head, abdomen, muscles and joints. Children. 2016;**3**(4):pii: E42

[154] Lee YJ, Kim S-H, Chung SW, Lee Y-K, Koo K-H. Causes of chronic hip pain undiagnosed or misdiagnosed by primary physicians in young adult patients: A retrospective descriptive study. Journal of Korean Medical Science. 2018;**33**(52):e339

[155] Feinberg BI, Feinberg RA. Persistent pain after total knee arthroplasty: Treatment with manual therapy and trigger point injections. The Journal of Musculoskeletal Pain. 1998;**6**(4):85-95

[156] Ingber RS. Iliopsoas myofascial dysfunction: A treatable cause of "failed" low back syndrome. Archives of Physical Medicine and Rehabilitation. 1989;**70**(5):382-386

[157] Tay A, Chua K, Chan K-F. Upper quarter myofascial pain syndrome in Singapore: Characteristics and treatment. The Journal of Musculoskeletal Pain. 2000;**8**(4):49-56

[158] Vadivelu N, Mitra S, Hines RL. Undergraduate medical education on pain management across the globe. Virtual Mentoring. 2013;**15**(5):421-427

[159] Canadian Medical Protective Association. What happened to the physical exam? [Internet]. 2019. Available from: https://www.cmpa-acpm.ca/en/advice-publications/browse-articles/2019/what-happened-to-the-physical-exam [Accessed: 26 July 2019]

[160] Clark BW, Derakhshan A, Desai SV. Diagnostic errors and the bedside clinical examination. The Medical Clinics of North America. 2018;**102**(3):453-464