When properly developed, communicated and implemented, guidelines improve the quality of care that is provided and patient outcomes. Guidelines are intended to support the healthcare professional’s critical thinking skills and judgment in each case.

In addition to guiding safer, more effective and consistent outcomes, the authors can only dare to dream that these guidelines will also support better constructed MT research methodology and designs, ultimately leading to the inclusion of MT professionals in mainstream interprofessional healthcare.

**What Defines Traumatic Scarring?**

The American Psychological Association defines trauma as an emotional response to a terrible event like an accident, sexual assault or natural disaster (APA 2015).

Scar or *cicatrix*, derived from the Greek *eschara* – meaning scab – is the fibrous *replacement* tissue that is laid down following injury or disease (Farlex 2012).

Scars are not obligated to be problematic – where would we be without our body’s natural ability to heal itself following a wound? However, normal physiological processes can be altered in a variety of ways and, when altered, this constitutes what is termed pathophysiology.

Abnormal or *pathophysiological scars* can impact function within and beyond their physical borders and present considerations outside of the physical/physiological aspect of the scar tissue (Lewit & Olsanska 2004, Bouffard et al. 2008, Valouchová & Lewit 2012, Bordoni & Zanier 2014).

Problematic scarring following planned and unplanned trauma can be accompanied by serious physiological and psychological considerations and, as such, this brings us to traumatic scars as defined by the authors:

*pathophysiological scars further compounded by traumatic emotional sequelae and other comorbidities.*

Traumatic scars can occur as a result of accidents, acts of violence and other catastrophic events (e.g. disease, burn accident and surgery).

Over the last several decades, advancements in medical technology have led to improved surgical techniques and emergency care (Blakeney & Creson 2002). Simply put, more people are surviving injuries that would have been fatal 20 or 30 years ago, and an increase in survival rate means an increased need for professionals skilled in treating *people with scars*. Working successfully with traumatic scars requires expert navigation, not only of the physicality of the scar material but also inclusive of the whole clinical presentation. This book will provide the information needed to help guide the development of that expertise.

Given the complexity of traumatic scars, where such presentations and/or appropriate treatment fall outside of the MT profession’s scope of practice, suggestions will be provided for appropriate referral resources to support the patient’s best possible biopsychosocial outcomes.

It is the authors’ intention that the end product will comprehensively cover both the physiological/physical and empathetic elements of MT and in doing so honour both the art and science of the work. Like interprofessional collaboration, integration of art and science are essential for excellence in contemporary healthcare and achieving the best possible patient-focused outcomes for *people with scars*.

**Overview of Chapters**

Individual chapters will cover normal/abnormal scar formation; the role of various systems in wound healing; the impact of pathophysiological
scars and associated sequelae; the biochemical and emotional impact of trauma; communication skills (including interprofessional communication); assessment and treatment protocols; client/therapist self-care and shared experiences from therapists and people with scars.

In order to assist with research translation, throughout this book pathophysiological and clinical considerations will be interspersed as Boxes where it makes sense to do so.

References
responses, the internal to external and cell-to-cell linkage mechanism provides the means by which the organism can sense and respond to mechanical demand or forces placed upon it – monitoring and regulating ‘enough tension’ to ensure the shape and integrity of any given cell and the entire musculoskeletal/myofascial system.

Skin Structure and Function

In order to better understand the formation of scar tissue, we need to look at the marvelous organ that is our skin.

Along with the glands, hair and nails, skin makes up the integumentary system. On average, skin
Skin and fascia

constitutes 10% of human body mass. Skin acts as a barrier to the outside world and plays an important role in **homeostasis**. The skin, our outer layer of protection, is susceptible to infections, injuries, growths, rashes, cysts, boils, discoloration, burns, adhesions and **scars**.

**Skin Histology**

The skin comprises:

- **Epithelium**
- **Connective tissue (CT)**.

**Epithelium**

There are three basic types of epithelial tissue: squamous, cuboidal and columnar – arranged in either a one-layer (simple) or multilayer (stratified) configuration. Epithelium forms many glands and lines the cavities and surfaces of structures throughout the body (e.g. the **epidermis** consists of stratified squamous keratinizing epithelium) (Marieb et al. 2012).

**CT**

Considered a system, CT consists of several different types of cells (e.g. fibroblasts and adipocytes), protein fibers (elastin and collagen) surrounded by the gelatinous ECM (Schleip et al. 2012a, Andrade 2013).

CT is a continuous bodywide system that plays a well-identified role in integrating the functions of diverse cell types within each tissue it invests (e.g. skeletal muscle, tendon, bone, viscera (Langevin 2006)). CT is highly variable in its presentation. Various terms are used to describe CT typology, for example:

- Dense and loose are used to describe how dense, tightly or spread out the fibers are packaged within an array of tissue
- Regular, irregular, unidirectional, multidirectional, parallel ordered and woven are used to describe fiber orientation and configuration within a particular sheet, layer or area of tissue (Terminologia Histologica 2008).

**Clinical Consideration**

As CT is intimately associated with other tissues and organs it may influence the normal or pathological processes in a wide variety of organ systems (Findley et al. 2012).

**CT, fascia and the sliding mechanism**

One of the more recent discoveries in the world of fascia research is the sliding/gliding that occurs throughout the CT and fascial systems, which facilitates unimpeded, frictionless movement (McCombe et al. 2001, Guimberteau & Bakhach 2006, Stecco et al. 2008, Wang et al. 2009). Some suggest that sliding layers are interspersed between CT and fascial layers; however, Guimberteau suggests that rather than separated or superimposed layers there exists a singular, highly hydrated, tissue architecture which can maintain the necessary space between structures to facilitate optimal sliding and tissue excursion (Guimberteau & Bakhach 2006, Guimberteau 2012) – see Figure 2.2. Whether the presentation is layers between layers or a singular architecture, the sliding mechanism comprises loose CT – consisting of predominantly fine collagen strands, adipocytes and an abundance of HA.

Fascicular

**Fascicular fascia** augments continuity and force transmission, provides proprioceptive feedback and protection of nerve, blood vascular and lymph vessels.

**Clinical Consideration**

Perimysium plays a significant role in force transmission. Perimysium is commonly thicker in postural muscles, tends to display a higher concentration of MFBs and can adapt more readily to changes in mechanical tension. The driving force behind long-term, sustained contracture and pronounced muscular ‘stiffness’ could be explained in part by the presence of MFBs in fascia – in particular in the perimysium. Surgical reconstruction processes (e.g. distraction osteogenesis, psoas lengthening) are frequently accompanied by increased muscle stiffness, shown to correlate with a significant increase in perimysial thickness – a response to the (sudden) increased tissue stretch (Schleip et al. 2005, Huijing 2007).

**Clinical Consideration**

Muscle spindles are embedded in the endomysium. If the enveloping fascia is too rigid (e.g. fibrosis, contracture), this may alter the stretch of the muscle spindle and adversely affect its normal firing (Stecco 2004).

**Compression**

**Compression fascia** forms a pressurized compartment to augment vascular function (e.g. venous return) and enhances proprioception, muscular efficiency and coordination.

**Separating**

**Separating fascia** provides structural support, helps absorb shock, limits the spread of infection and reduces friction and augments movement between articulating structures and surfaces. The loose well-hydrated sliding layers also fall into this category.

**Functions of Fascia: Summary**

Fascia physiology in brief: as a component of the locomotor system, fascia performs essentially three mechanical functional roles: separation, connection and energy facilitation.

**Fascia as a tissue of separation:** creates space which serves as an interface for ease of sliding and gliding of structures and tissues in relation to one another. Spatial separation supports unimpeded motion and motion ensures the healthy functioning of tissues and structures and the sliding–gliding interface.

**Fascia as a tissue of connection:** links together various anatomical components (bones, joints, muscles etc.) thereby creating an architecture that augments the transfer of forces across a broader array of tissues/structures. Connection enhances strength, stability and efficient coordinated movement. Bodywide perceptive and functional continuity affords linked tissues the ability to function and, potentially, dysfunction together.

**Fascia as an energy facilitator:** healthy, springy or crimped collagen is a potential energy generator or storehouse rather than an energy consumer, such as muscle. When stretched, collagen will rebound, augmenting propulsion (especially flatter, sheet-like fascia and tendons, e.g. Achilles tendon and Achilles aponeurosis) (Schleip & Müller 2013). Ultrasound-based measurements indicate that fascial tissues are commonly used for a dynamic energy storage (catapult action) during oscillatory movements, such as walking, hopping or running, and during such movements the supporting skeletal muscles contract more isometrically while the loaded fascial elements lengthen and shorten like elastic springs (Fukunaga et al. 2002). The rebound/catapult potential of fascia decreases the need
for muscular energy – resulting in greater efficiency and endurance.

**Myokinetic/Myofascial Chains and Meridians**

The continuity seen throughout the fascial system is a key functional characteristic. It is agreed upon by many that dysfunction (scarring, densification, fibrosis) anywhere along the chain or meridian can impact function in other regions.

When challenged by stretch or movement dense and/or restricted fascia may alter proprioceptive afferent signals that lead to eventual abnormal biomechanics, aberrant movement patterns, muscle compensation, joint distress and pain (Bouffard et al. 2008).

In the absence of normal physiological elasticity, receptors embedded within the fascia may also be in an active state even at rest. Any further stretching, even that produced by normal muscular contraction, could cause excessive stimulation with consequent propagation of nociceptive afferents. Furthermore, over a certain threshold (i.e. consistent stimulus over time), all receptors can potentially become algoreceptors (pain receptors) in response to consequent propagation of nociceptive signals (Ryan 2011).

Chain/meridian clinical considerations will be covered in greater detail in Chapter 9.

**Clinical Consideration (Cont.)**

In the field of MT we commonly encounter fascial changes associated with acute injury and trauma, chronic strain (physical or emotional), immobilization and the spectrum of subsequent sequelae (e.g. adhesions, fibrosis, myofascial trigger points, diminished gliding within the sliding layers, neural and circulatory consequences).

**Clinical Consideration**

According to Lewit and Olsanska (2004), scars may contribute to the formation of myofascial trigger points in adjacent tissues along with the potential for pain in other regions (e.g. low back pain associated with appendectomy).

As the impact of a scar can reach far beyond its physical borders, in addition to the degree or depth of the scar – from a MT perspective – it is also important to consider how a scar in one region of the body can impact functioning in distant (seemingly unrelated) tissues or areas.

According to Bordoni and Zanier (2014):

*Every element or cell in the human body produces substances that communicate and respond in an autocrine or paracrine mode, consequently affecting organs and structures that are seemingly far from each other. This applies to the skin and subcutaneous fasciae. When the integrity of the skin has been altered, or when its healing process is disturbed, it becomes a source of symptoms that are not merely cutaneous. Additionally, the subcutaneous fasciae is altered when there is...*
Clinical Consideration

Lymph is the new blood

Readily visible, blood and its associated vessels have been studied for centuries, garnering much attention including detailed, shiny color plates in health science textbooks. However, a recent stunning discovery challenges current textbooks, calling for more in-depth study and an extreme make-over for anatomy and physiology editions.

Generally presumed by manual lymph therapists but unverified, researchers have now discovered that the meningeal linings of brain have a lymphatic vessel network that connects to the systemic lymphatic system. Until now, lymphatic vessels in the central nervous system (CNS) have remained elusive (i.e. no shiny color plates), but the development of better imaging methods has changed all that (Aspelund et al. 2015, Louveau et al. 2015, University of Helsinki 2015, University of Virginia Health System 2015).

Vessels displaying all the molecular hallmarks of lymphatic vessels have been discovered and mapped, running parallel to the dural sinuses, arteries, veins and cranial nerves. It is suggested that they serve as a direct clearance route for the brain and cerebrospinal fluid macromolecules out of the skull and into the deep cervical lymph nodes (Aspelund et al. 2015, Louveau et al. 2015, University of Helsinki 2015, University of Virginia Health System 2015).

As is known, the lymph system clears fluids and macromolecules and plays an important role in immune function. Previously, the CNS has been considered devoid of lymphatic vasculature, leaving immunologists puzzled as to how lymphocytes access and exit the brain (Aspelund et al. 2015, Louveau et al. 2015, University of Helsinki 2015, University of Virginia Health System 2015).

The extensive lymph network found in the brain changes our understanding of how the brain is cleared of excess fluid and calls for the rethinking of brain disease etiology and treatment approaches. This discovery has also raised several new questions concerning some fundamental brain functions and changes how we look at the relationship between the CNS and immune system (Aspelund et al 2015, Louveau et al. 2015, University of Helsinki 2015, University of Virginia Health System 2015).

Researchers find it highly possible that brain–lymph connection will prove important in neuro-immunological diseases as well as in diseases characterized by the pathological accumulation of misfolded proteins or fluid into the brain parenchyma (e.g. Alzheimer’s disease, MS and autism). In Alzheimer’s for example, there are accumulations of big protein chunks in the brain and it has been suggested that these chunks may be accumulating because they’re not being efficiently removed by the CNS lymph system (Louveau et al. 2015).

Given the lymph and immune systems role in wound healing, one can surmise that future work in this area may reveal significant considerations for scar management.

Fig 3.5

Brain lymphatic vessels.

University of Virginia Health System 2015).
and armpit. It sometimes strikes individuals who have had coronary artery bypasses using a saphenous vein from the leg: the removal of this vein is accompanied by removal of related structures of the lymphatic system, lowering immunity to infection.

Acute lymphangitis is a bacterial infection in the lymphatic vessels, which is characterized by painful, red streaks below the skin surface. This is a potentially serious infection that can rapidly spread to the bloodstream and be fatal.

Lymph nodes are filters that can catch malignant tumor cells or infectious organisms. When they do, lymph nodes increase in size and are easily felt.
While lymph nodes are the most common cause of a lump or a bump in the neck, there are other, much less common causes, e.g. cysts from abnormalities of fetal development or thyroid gland enlargement.

**Wound Healing**

As with the blood vascular system, successful tissue repair requires the regrowth and reconnection of lymphatic structures. In the early stages of wound healing, the formation of lymphatic vessels in circumferential wounds helps bridge the margins of a newly forming scar (Bellman & Oden 1958, Oliver & Detmar 2002).

In full-thickness skin wounds angiogenesis in newly formed **granulation tissue** largely dominates the delayed and comparatively less pronounced formation of new lymphatic vessels (**lymphangiogenesis**) (Paavonen et al. 2000, Oliver & Detmar 2002).

During tissue repair, lymphatic vessels reconnect with lymphatic vessels – not with blood vessels – and cultured lymphatic endothelial cells remain separate from blood vascular endothelial cells during tube formation in vitro (Kriehuber et al. 2001).

It is known that in adults lymphangiogenesis can occur by outgrowth from pre-existing lymph vessels (Clark & Clark 1932, Paavonen et al. 2000) but it is unclear if during tissue repair lymphangiogenesis involves progenitor cells, as in angiogenesis.

**Impact of Trauma and Pathophysiological Scars on the Lymphatic System**

Trauma and scars can disrupt the network of lymph capillaries, which can hinder fluid drainage and negatively affect the healing process.

The development of scar tissue that hinders the flow of lymph can have various causes:

- Surgical procedures that interrupt normal lymphatic function, such as surgery for cancer in the breast or groin areas, may prevent lymph flowing naturally through its system (Zuther 2011)
- Radiation therapy can damage an otherwise healthy lymphatic system by causing scar tissue to form, subsequently interrupting the normal flow of lymph (Zuther 2011)
- Traumatic scars may affect the lymphatic system, damaging the normal flow of lymph.

**Clinical Consideration**

Four continuous minutes of abdominal lymph pumping (1 pump/sec) has been shown to increase immune cell release from mesenteric lymph nodes. Specialized T cells primed in the gut are faster-acting/superior-performing immune cells. Recent studies suggest that abdominal lymphatic pumping may inhibit solid tumor development and bacterial pneumonia (Hodge et al. 2010, 2013).

**Pathophysiological Consideration**

Abdominal scarring may interfere with the effectiveness of local lymph pump techniques (Hodge et al. 2010, 2013).

In addition, repeated episodes of infection can cause progressive closure of the lymphatic system, thus worsening the condition.

If the transport pathway becomes congested, blocked, damaged or severed, fluids can accumulate in the surrounding tissues leading to edema and fibrosis. Eventually cell pathology may occur. If the lymph system is incapacitated in any way (e.g. burns, ulceration, chronic inflammation, hematoma), wound healing processes...
can be impaired (e.g. transport of damaged cells and inflammatory byproducts away from the injury site). Healing and recovery can be assisted by supporting healthy lymphatic function.

**Lymphatic Inadequacy**

Inadequacy in the lymphatic system occurs if the lymphatic load is more than the transport capacity can handle. When lymphatic tissues or lymph nodes have been damaged, destroyed or removed, lymph cannot drain normally from the affected area. When this happens excess lymph accumulates and results in the swelling that is characteristic of edema and lymphedema (Zuther 2011).

**Clinical Consideration**

Compression bandages promote reduction in capillary hypertension and lymphatic load – creating a massaging effect to influence venous and lymphatic hemodynamics resulting in reduction of edema (Földi et al. 2005, Williams 2005).

There are three types of inadequacies which may result in lymphedema or edema: dynamic, mechanical and combined (Zuther 2011).

**Dynamic**

Dynamic inadequacy occurs when the active and passive edema protective measures are depleted and results in edema.

**Mechanical**

Mechanical inadequacy occurs when the transport capacity of the lymphatic load slows due to functional or organic causes, such as surgery, radiation, and trauma, or to a response from certain drugs or toxins. Due to the pressure exerted and leakage of proteins into the lymphatic wall structure, fibrosis may occur.

The stagnation of water, proteins, waste products and cell debris in the tissue can cause damage to the tissue. It also reduces the ability of the immune response due to the lack of circulation of the macrophages and lymphocytes. This can lead to a high incidence of infections such as cellulitis (Zuther 2011).

**Combined**

Combined inadequacy results from the lymphatic load being slowed due to the stagnation of the transport capacity. In other words, the system is very sluggish and not moving the fluid at a normal rate. When this occurs, the combination can lead to necrosis (severe tissue damage) and chronic inflammation (Zuther 2011).

**Edema**

Edema – the medical term for swelling – is considered to be a symptom. Edema can occur as a natural response of the body to injury or insult. Increased fluid in the affected area can be beneficial by assisting the delivery of immune cells to the area to fight an infection and assist with debris cleanup.

Edema can be isolated to a small area (localized) or can affect the entire body (generalized).

Pregnancy (preeclampsia or toxemia), tissue trauma, infections, medications and various conditions can result in edema; for example, venous insufficiency, obstruction of flow (e.g. thrombosis, tumor), low albumin, allergic reactions, congestive heart failure, liver and kidney disease, and critical illness (WebMD 2015).

Medications known to result in lower extremity edema include non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids and some medications used for hormone replacement, diabetes, depression and high blood pressure (WebMD 2015).

Discussion of the role of edema in critical illness, such as a burn or surgery, will be discussed in Chapter 5.
Clinical Consideration

Although muscle spindles (stretch receptors) have traditionally been associated with muscle tissue, more recent research indicates that these receptors are embedded in myofascial tissue. Muscle spindle response to detectable stretch can be altered if myofascia (e.g. endo or perimysium) is too rigid. In such cases, normal firing of the muscle spindles can be altered (Stecco 2004).

Clinical Consideration (Cont.)

(Jones 1944) or myokinetic linkage system (Stecco 2004). For example, such is seen in the thoracolumbar region during locomotion and load lifting (Jones 1944, Vleeming et al. 1995, Graceovetsky 2007).

Clinical Consideration

Retinaculae in particular display a greater density of nerve receptors than that typically seen in muscle bellies or some other soft tissues (Stecco et al. 2010). It is suggested that fascia plays an integrating function as a proprioceptive organ and that it can coordinate the action of different muscles by acting as a common 'ectoskeleton'.

Example 1

According to Cottingham (1985), Golgi receptors are stimulated during soft tissue manipulation, Hatha yoga postures and slow active stretching resulting in a lower firing rate of specific alpha-motor neurons,
Clinical Consideration (Cont.)

Example
Hypersensitive nerves (associated with muscle) are prone to spontaneous electrical impulses that trigger false pain signals or provoke involuntary muscular activity such as increased tone or tension (Culp & Ochoa 1982). In turn, this increase in tone/tension can subsequently increase the risk of secondary injury (e.g. sprain/strain, tendinopathies, MTrPs).

Clinical Consideration
Hypersensitive afferents can display other forms of aberrant behavior.

Example 1
Hypersensitized nerve fibers become receptive to chemical transmitters all along their length rather than just at their receptor endings (Thesleff & Sellin 1980).

Example 2
Hypersensitized nerves are prone to accept contacts from other types of nerves including autonomic and sensory nerve fibers (Thesleff & Sellin 1980). Impaired communication between sensory and autonomic nerves may contribute to complex regional pain syndrome.

Pathophysiological Consideration
Pain experts suggest that pain due to peripheral or central neural damage or aberration (neuropathic pain) may differ in clinical features and responsiveness to pharmacological therapy, from pain caused by activation of primary afferents in somatic or visceral tissues (nociceptive pain).

Clinical Consideration
Neuropathic pain may be resistant or less responsive to opioid therapy, suggesting the necessity of using alternative analgesics to achieve pain relief in patients with neuropathic pain (Wilkie et al. 2001). Additionally, there is growing concern over the detrimental side effects, including opioid-induced hyperalgesia. Although this family of drugs provides analgesic and antihyperalgesic effects initially, subsequently they are associated with the expression of hyperalgesia, suggesting that opioids can activate both pain inhibitory and pain facilitatory systems. Paradoxically, opioid therapy aiming at alleviating pain may render patients more sensitive to pain and potentially may aggravate their pre-existing pain (Angst & Clark 2006).

Clinical Consideration
MT has been found to be an effective and safe adjuvant therapy for the relief of acute postoperative pain in patients undergoing major operations. Patients reported markedly less intense and less unpleasant pain and less anxiety than patients who received standard pain medication or individual attention but no MT. The day after surgery, some patients reported that massage delivered about as much pain relief as a dose from a morphine drip. It is suggested that MT works by creating
Wound Healing

The NS plays an important role in mediating normal wound healing via the involvement of various neuropeptides and growth factors. Noxious stimuli causes nerves to release neuropeptides such as substance P and CGRP and certain growth factors (e.g. NGF). Along with healthy scar formation, in order to re-establish normal functioning post-injury, innervation of the injured tissue must also be re-established.

Wounds initially display hyperinnervation, similar to hypervascularization seen in early wound healing. With normal wound healing, nerve density will normalize with scar maturation. Although densities may return to normal, normal responsiveness is not always re-established, as nerve end organs cannot regenerate and therefore sensory deficit or aberrancies may occur. In spite of the clinical significance of abnormal innervation in scars, the NS has been largely ignored in the pathophysiology of scars.

The role of the NS in wound healing, normal and abnormal, is covered in greater detail in Chapter 5.

Impact of Trauma and Pathophysiological Scars on the NS

As nerves traverse throughout the body they track between and pass through various tissues and structures. The greater proportion of our larger nerves track in the superficial fascia and smaller nerves in the clefts created between perimysial bundles.

Irritation or compression at any point along the complex network of the NS can evoke changes not only locally but also in distant regions and can elicit autonomic disturbances.

Excessive scarring in the layers of tissue a nerve is travelling through can impede all manner of NS functioning (e.g. nerve and electromagnetic conduction, local and systemic responsiveness, sensitization, intraneural blood and nerve supply).

Additionally, injured or distressed somatic tissues adjacent to nerve structures release inflammatory substances that can chemically irritate neural elements. This suggests that a nerve does not have to be entrapped in scar tissue, it can also be impacted by scarring in neighboring tissue.

Pressure and compression constitute the most common forms of nerve injury as complete severance is rare. While nerve injury grading classification systems, such as Seddon (1943) and Sunderland (1952), describe the severity of neural injury (neuropraxia, axonotmesis, neurotmesis) and consider negative symptomology (e.g. numbness and weakness), they do not include considerations for positive symptomology like neuropathic pain.
• Display textural variations (e.g. rough, lumpy, dimpled, puckered)
• Tend to exhibit compromised elasticity and mobility
• May display altered or abnormal neuro-functioning (hypersensitivity, hyposensitivity, increased electrical activity with movement) (Bordoni & Zanier 2014, Valouchová & Lewit 2009)
• Some mature scars may be pale due to limited blood supply or appear reddish due to hypervascularization that lasts beyond the ‘normal’ time frame (i.e. early stage of wound healing).

**Types of Pathophysiological Scars**

In order to assist with this next section, a few key terms are briefly described in Table 5.2.

The type of tissue damage and severity of the injury factor significantly in forming the proper protocol for long-term recovery.
and rehabilitation. Being able to identify the differences in the types of scar tissue will help to safely and effectively deliver care and develop more precise long-term protocols for clients.

Traumatic scars will be explored in greater detail in subsequent chapters where discussion will center around specifics on scarring from burns, surgery (e.g. mastectomy) and other types of traumatic events. Special consideration will be given to each type of scar presentation along with specific treatment protocols.

A few key terms and an overview of the various types of pathophysiological scars are briefly

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition and other information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scar</td>
<td>Commonly used in reference to the ‘normal’ end product – describing a mark left in the skin, fascia, muscle (or other tissue) and organs as a result of healing of a wound, sore or injury</td>
</tr>
<tr>
<td>Adhesion</td>
<td>Commonly used to describe problematic areas of profuse or abnormal scarring occurring in organs and tissues. Adhesions can result in pain/dysfunction</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>Replacement of the normal structural elements of tissue by excess accumulation of dysfunctional fibrotic tissue (Rodriguez &amp; del Rio 2013). A process driven by excessive or sustained production of TGF-ß1 and consequent superfluous MFB activity (Smith et al. 2007, Karalaki et al. 2009, Fourie 2012). When used in reference to the myofascial/musculoskeletal system this term is commonly used to describe what occurs as a result of onerous tension or mechanical strain associated with poor remodeling following injury; culminating in superfluous, maladaptive, poorly constructed collagen (e.g. aberrant fiber/bundle arrangement, increased incidence of pathological cross-links and reduced elastic-malleability) (Henry &amp; Garner 2003, Diegelmann &amp; Evans 2004). It is a reasonable conclusion that prevention or resolution of fibrosis can diminish the occurrence of common sequelae and comorbidities</td>
</tr>
</tbody>
</table>

**Pathophysiological consideration**


**Pathophysiological consideration**

Fibrosis is commonly considered to be a by-product of an abnormal or chronic inflammatory response as a result of cumulative or overuse-type trauma, immobilization and poor wound healing. When not effectively managed, fibrosis can result in a milieu of functional deficits. The fall-out from fibrosis include the following and associated sequelae: increased risk of recurrent injury, muscle contraction dysfunction, altered force transmission, impaired tissue; slide/glide and stretch, neural, circulatory and lymphatic compression, antalgia, pain and pain translation (i.e. shift from acute to chronic pain) (Simons, Travell & Simons 1999, Gabbiani 2003, Shah et al. 2005, 2008, Hinz 2007, Grinell 2008, Chiquet et al. 2009, Lowe 2009, Ciciliot & Schiaffino 2010).

**Clinical consideration**

Mechanical disruption of fibrotic tissue increases the pliability of scars (Bhadal et al. 2008, Chan et al. 2010, Cho et al. 2014).
As noted by Barbe at the FRC III in 2012, immobilization may result in correlating grey matter shrinkage and subsequently there is a diminished or lost ability for movement stimulus and/or coordination. Nerve regions that were originally devoted to sensing, initiating and controlling movement lack activity/stimulation when the region is immobilized, resulting in re-allocation of these nerves to other functions and/or actual shrinkage of grey matter (Granert et al. 2011).

Clinical Consideration

Here we see three mechanisms contributing to move it or lose it: fibrosed/stuck (can’t move); it hurts (avoid moving or alter movement pattern); and where the neural network that governs movement is affected (can’t generate, control or coordinate movement).

The Nervous System: Wound Healing And Pathophysiological Scars

As noted in Chapter 4, the NS plays an important role in wound healing. Noxious stimuli triggers the release of neuropeptides such as substance P and calcitonin gene-related peptide (CGRP) and certain growth factors such as NGF. Neuropeptides

Figure 5.4
The fall-out associated with immobilization.
Burns, mastectomies and other traumatic injuries can involve significant penetration of the skin layers, sometimes penetrating deeper layers of tissue down to bone. Extensive tissue damage due to trauma (planned or unplanned) can compromise sensation, proprioception, circulation, lymphatic drainage, thermoregulation and dermal excretory capacities (Fitch 2005).

As discussed in previous chapters, scar tissue can affect the body in a variety of ways. Fibrotic scars and scars that are bound to the underlying tissues, organs or skeletal structures can restrict movement and organ motility (Fitch 2005, Bove & Chapelle 2012). In addition to physical disability and dysfunction, traumatic scars can also result in a spectrum of psychosocial sequelae, which will be covered in greater detail in Chapter 7.

The depth and extent of the scar strongly influence the consequent sequelae. Extensive tissue damage requires additional recovery time and increases the potential for complications, including excessive scarring and successive surgeries (Kania 2012).

The aim of this chapter is to provide more in-depth understanding of the types of pathophysiological scars and scars seen in conjunction with specific types of trauma.

**Excessive Scarring**

Excessive scarring was first described in the Smith papyrus around 1700 BC (Berman & Bieley 1995). A few millennia later, Mancini & Quaife (1962) and Peacock et al. (1970) differentiated excessive scarring into hypertrophic and keloid scar formation and defined their distinguishing characteristics: both scar types rise above skin level, but while hypertrophic scars do not extend beyond the initial site of injury, keloids typically project beyond the original wound margins (Gauglitz et al. 2011).

Excessive or pathophysiological scars form as a result of prolongations or aberrations of physiologic wound healing and may develop following any injury to the deep dermis, including burn injury, lacerations, abrasions, surgery, piercings and vaccinations. Wound healing in pathophysiological scars is characterized by a prolonged and stronger inflammation phase with inappropriately released cytokines followed by a subsequent delay in the healing response (Huang et al. 2013). Excessive scarring resulting in pain, pruritus, adherences and contractures can affect quality of life, physically, physiologically and psychologically (Gauglitz et al. 2011). Scar depth can have a negative impact on the healing process and, subsequently, impact the functions of the dermal layer and deeper layers of tissue (e.g. muscle, fascia).

Each year in industrialized nations, 100 million people develop scars as a result of 55 million elective operations and 25 million operations due to trauma (Gauglitz et al. 2011). From 55% to 100% of surgical patients will experience postsurgical scar/adhesion complications that may not become evident until months or years later (Diamond 2012).

Although there is extensive research on the pathophysiologic process of wound healing and
scar formation, there is still no consensus on the best treatment strategy for preventing and reducing the issues associated with pathophysiological scars (Van der Veer et al. 2009, Cho et al. 2014).

Clinical Consideration

Postsurgical scar complications, including chronic pain, can occur as a result of incisions (even less invasive ‘keyhole’ incisions), cauterization, suturing or fusing together of segments that are normally meant to be separate from one another (e.g. spinal fusion) (Lee et al. 2009, Fourie 2012). The side-effects of peritoneal adhesions have been reported to occur in 90–100% of cases following surgery. With abdominal surgeries, organ function can be compromised (e.g. impaired motility, obstructions, infertility, pelvic pain). Detection and diagnosis are hindered by lack of identifiable biomarkers, poor imaging methods or the onset of other health issues masking the underlying scar/adhesion sequelae. Re-operating to remove the offending scar tissue appears to be a case of fighting fire with fire – with questionable productive outcomes (Diamond 2012). The most recent cost estimate of morbidity associated with abdominal adhesions is $5 billion in the US alone (Bove & Chapelle 2012). It is evident that prevention and appropriate early intervention are the most cost-effective and patient-considerate approaches.

A Deeper Look at Hypertrophic and Keloid Scars

Chapter 5 provided a brief overview of the various types of pathophysiological scars and how scars form. To further the understanding of the healing process and the impact of traumatic scars, this next section will provide a more in-depth look at hypertrophic and keloid scars – see Table. 6.1 for a comparative summary of hypertrophic and keloid scars.

Wound healing is an intricate biological process involving overlapping phases. When this process is disrupted or altered, abnormalities in scarring appear. Both hypertrophic and keloid scars are a result of disruption in the fundamental processes of wound healing. Race, age, genetic predisposition, hormone levels, atopy/hypersensitivity and immunologic responses, type of injury, wound size/depth, anatomic region, and local mechanical tension all factor into wound healing process and subsequent nature of the scar (Niessen et al. 1999, Cho et al. 2014).

Multiple studies on hypertrophic and keloid scar formation have been conducted over several decades, leading to many therapeutic strategies to prevent or attenuate excessive scar formation. Most therapeutic approaches remain clinically unsatisfactory, however, with an agreement that there is a meager understanding of the complex mechanisms underlying the processes of scarring and wound contraction and fibroproliferative disorders in general (Gauglitz et al. 2011, Rabello et al. 2014).

Scar pathogenesis involves cellular and extracellular matrix (ECM) components in both the epidermal and dermal layers that are regulated by a wide array of interfering factors in the inflammation, proliferation, and remodeling stages of healing (Huang et al. 2013).

Hypertrophic scarring after deep or partial-thickness wounds is common. A review of the literature on the prevalence of hypertrophic scarring found that females, children, young adults, and people with darker, more pigmented skin are particularly at risk and, in this subpopulation, the prevalence is up to 75% (Engrav et al. 2007). Hypertrophic scars are morphologically characterized by (Linares 1996, Cho et al. 2014):

- Abnormal collagen
- Reduced elastin
discovered around the lymphatic limb (Dylke et al. 2013).

**Auxillary Web Syndrome or Cording**

Axillary web syndrome (AWS), also known as ‘cording’ in the postsurgical breast cancer patient, is characterized by painful cording or strings of hardened lymph tissue in the axilla of the affected side. It affects functioning by causing pain and restriction in arm ROM, especially abduction. Alexander Moskovitz refers to the syndrome as: ‘axillary pain radiating down the ipsilateral arm, shoulder ROM limitation, and an axillary web of tissue attempts abduction of the arm.’ It appears that the axillary lymph node dissection of the breast procedure is the trigger to the lymphatic disruption that causes AWS (Bock 2013).

AWS may manifest with one large cord or several distinct, smaller cords running down the arm. These cords usually start near the site of any scarring in the underarm region and extend down the inner arm to the inside of the elbow. Sometimes they can continue all the way down to the palm of the hand. In some people, cording can extend down the chest wall instead of, or in addition to, the inner arm (BreastCancer.org 2015b).

The anatomy of the axilla bears review for a full understanding of AWS. The axilla compartment is home to a fascial sheath that contains neurovascular bundles, 20–30 lymph nodes, and is bounded superiorly by the head of the humerus, covered by the coracobrachialis and the short head of the biceps. The axilla is bound anteriorly by the pectoralis major; posteriorly by the subscapularis, latissimus dorsi and teres major; and inferiorly by the ribcage which is covered by the serratus anterior (Calais-Germain 2007).

The fasciae that envelopes the regional muscles is continuous with the brachial fascia, which plays a significant functional role (Stecco et al. 2008); for example, the fascia of the clavicular head of the pectoralis major displays a thickening of collagen fibers that extend into the anterior brachial fascia, surrounding the biceps and thereby creating a continuous functional link (i.e. myokinetic chain/myofascial meridian). Additionally, the fascia from the sterna and costal heads are continuous with the axillary fascia and the medial brachial fascia (Fourie 2008).

In addition to musculature and fascia, five identifiable groups of lymph nodes are found in the axilla. These groups reside in the fatty CT of the axilla or are arranged around the blood vessels such as the lateral thoracic and subscapular arteries and the axillary vein. These nodes drain the lymphatic vessels of the ipsilateral upper quadrant (both anterior and posterior), the ipsilateral mammary gland and the ipsilateral extremity (Zuther 2011). Seventy-five percent of the lymph from the breast and areolar plexus drains into the anterior pectoral group of lymph nodes before they move to the central nodes.

When any of the lymph nodes are removed, the flow and pathway of lymphatic fluid is disrupted and fibrosis may occur contributing to the development of AWS.

As scar formation is the body’s mechanism for restoring tissue integrity, all wounds are subject to the repair process. For internal tissue injuries and inflammation involving fascia, scarring and adhesions contribute to the occurrence of tissue rigidity, abnormal movement patterns and pain.

Issues after breast cancer surgery that result in AWS may arise from the maturing process of CT and the prolonged inflammatory phase of wound healing. The CT maturation could result in either a dense, non-pliable scar or a pliable mobile scar. A prolonged inflammatory phase results in hypertrophic scarring and increased fibrosis in the damaged area (Fourie 2008).
Clinical Consideration

It has been demonstrated – via ultrasound imaging – that skin rolling can modify collagen density (Pohl 2010).

Scar tissue can develop up to 6 months after treatment is complete. The skin may become hard, thick or feel bound in the areas that have been irradiated; surgical sites can also be aggravated (MacDonald 2003).

Scarring from radiation or surgery disturbs the lymphatic flow and, if not addressed, can create an environment for fibrosis. Add to this the fact that lymphatic pathways do not re-establish themselves across scars and you could draw the conclusion that un-drained lymphatic fluid contributes to the pathogenesis of the raised and swollen tissues abutting a scar (Warren & Slavin 2007).

Although no protocols exist, several excellent case studies indicate that a combination of manual lymph drainage techniques, myofascial release, snapping or popping of the cords and ROM exercises with passive stretching can bring about productive outcomes (Bock, 2013).

Breast Cancer-Related Neuropathy

Chemotherapy-associated peripheral neuropathy is the most common cause of breast cancer-related neuropathy. Chemotherapy medications travel throughout the body, where they can cause damage to the nerves (BreastCancer.org. 2015c).

Certain chemotherapy medications can cause neuropathy. Chemotherapy-associated neuropathy may begin as soon as treatment starts, and it may worsen as treatment continues. Usually it begins in the toes, but it can expand to include the legs, arms, and hands. The most common symptoms include:

- Pain, tingling, burning, weakness, tickling, or numbness in arms, hands, legs, and feet
- Sudden, sharp, stabbing, or shocking pain sensations
- Loss of touch sensation
- Clumsiness
- Trouble using hands to pick up objects or fasten clothing.

Other possible symptoms include:

- Balance problems and difficulty walking
- Hearing loss
- Jaw pain
- Constipation
- Changes in sensitivity to temperature
- Decreased reflexes
- Trouble swallowing
- Trouble passing urine
- Blood pressure changes.

Other treatments for breast cancer can cause neuropathy as well. Surgery and radiation therapy cause damage to nerve plexuses in the chest and axillary areas that include the brachial and cervical plexuses.

Nerve plexuses serve the motor and sensory needs of the limb (Marieb 2003). To fully understand the implications of damage or impingement to these plexus, we need to take a look at how they weave through the area affected by the surgery (Fig. 6.2).

The brachial plexus is shaped by the anterior branches of the last four cervical and first thoracic nerve before they begin their peripheral distribution. The brachial plexus traverses the subclavian triangle where it rests on the posterior scalene. The brachial plexus is covered by the omohyoid muscle and the middle and deep
cervical aponeuroses. It is separated by the subclavian muscle behind the clavicle. Its rests on the first rib and the superior digitation of the serratus anterior. It is behind the pectoral muscles, anterior to the subscapularis tendon and flanked by the two scalenes. The subclavian artery is found at the lower section of the plexus, slightly anterior to it (Croibier 1999) – see Figure 6.3.

The cervical plexus is a string of anastomoses formed by the anterior branches of the first four cervical nerves before they divide. The anterior branches are lodged in the groove that is formed by the superior surface of the transverse processes, and pass between the two intertransverse muscles behind the vertebral artery.

The cervical plexus is situated behind the posterior edge of the sternocleidomastoid, deep to the internal jugular vein, internal carotid artery and the vagus nerve. It anastomoses with the hypoglossal, vagal and sympathetic nerves (Croibier 1999).

Trauma to these plexuses can lead to neuropathic symptoms such as pain, numbness, tingling, and/or increased sensitivity in those areas. Targeted drug therapies (Perjeta® – generic name: pertuzumab – and Kadcyla® – generic name:
Stress

Day in and day out, our hands feel it – STRESS. Tissue so tightly wound and densely compressed, we pause for a moment and contemplate … is this bone or is this flesh???

Cathy Ryan RMT

Lazarus and Folkman (1984) define stress as: ‘a relationship between the person and the environment that is appraised by the person as taxing or exceeding his/her resources and endangering his/her wellbeing’. Appraisal and coping are key to this definition and lead to the subjective experience of stress. And generally speaking, the degree of perceived threat (appraisal) influences the magnitude of the stress response.

Stress response is psychobiologically complex, involving the individual’s appraisal of the situation, coping skills/behaviors and the resources the individual has available to draw on. Resources include both extrinsic (e.g. social support) and intrinsic in the form of the functioning of the involved systems. When an individual can no longer cope with stressful situations, affective, behavioral, and physiological changes result (Cohen et al. 1997, Lucas 2011).

Whether you are human, cat, dog or mouse, experiencing stress is simply a part of life. But neither stress nor stress response are inherently harmful and certainly stress response does have its time and place, such as when we need to run fast or leap far to avoid some calamity. Therefore, it is important to differentiate between acute stress, a beneficial adaptation response and chronic stress, which can prove detrimental.

Stress Response and Stress Hormones

Stress response encompasses the hormonal and metabolic changes that are activated by real or perceived danger, injury or trauma (Desborough 2000). Human stress response is innately intended to enhance coping, adaptation and chances of survival. Stress response is rooted in the capacity for rapid recognition of potentially harmful stimuli and the ability to mobilize a defense/stress response. Mobilization of stress response is adaptive and resilient and normally terminates as soon as the danger has passed (Friedman 2015).

Stress Adaptation Response

A vast array of potential stress responses exist. In any given circumstance one may run like crazy, fight when cornered, stand perfectly still so as not to be seen or gather with others; commonly referred to as fight, flight and tend/befriend.

The phrase fight or flight was coined by Cannon in the 1920s to describe the typical behaviors that occur in the context of perceived threat. A freeze response, or tonic immobility, may occur in some threatening situations (Gallup 1977, Barlow 2002). Tend and befriend refers to coping with stress through social or group support (i.e. befriending) and providing or receiving protection, nurturing or emotional support (i.e. tending to others or being tended to). Social isolation significantly enhances risk of mortality, whereas securing social support results in beneficial health outcomes, including reduced risk of illness and death (Cohen & Willis 1985).

Fight, flight or freeze are recognized as the initial stage of stress response adaptation. Fight may manifest not only as a physical exchange but also as vocally aggressive or argumentative behavior. Flight can occur as escaping in either a sensory way (e.g. social withdrawal, substance abuse or television viewing – Friedman & Silver 2007) or a physical way (e.g. running away from something perceived as threatening or toward something that is needed or feels safe).

Freeze response may occur when fleeing or aggressive responses are perceived to likely be
Prolonged stress response has been shown to suppress the immune system, disturb diurnal rhythm, stimulate or sustain obesity, adversely impact the body's pH balance and increase the incidence of chronic myofascial tension.

Stress and the myofascial system; psychological distress and anxiety have clearly been identified as a source of unnecessary muscular tension. Unnecessary muscle tension being; the confusing intermediate between a non-voluntary muscle contraction (spasm) and viscoelastic tension showing no EMG activity (Simon & Mense 2007). According to Chaitow (2013):

…the shortened fibers of the soft tissues may be the result of a combination of structural anomalies, trauma, and/or physical or emotional stress, and are always influenced by underlying nutritional and behavioral elements. Some of these shortened fibers and tender spots (i.e. trigger points) may be the source of reflex symptoms and pain. All such soft tissue dysfunctions respond to manual pressure in the form of modalities like massage therapy.

Clearly MT fulfils a number of the strategies for stress support (e.g. tend and befriend, providing a safe and calming environment) and is a means by which to safely and effectively address the adverse impact of prolonged stress response on the soft tissues.

Pathophysiological Consideration (Cont.)

energy following a threat are also innate mechanisms for restoring autonomic balance. If behavioral conditioning or circumstances prevents or interferes with our innate restoration mechanisms, our ability to return to a normal state of autonomic nervous system (ANS) functionality is impacted (Payne et al. 2015).

Clinical Consideration

Prolonged stress response has been shown to suppress the immune system, disturb diurnal rhythm, stimulate or sustain obesity, adversely impact the body's pH balance and increase the incidence of chronic myofascial tension.

Psychological Stress and Wound Healing

Studies over the last 30 years have shown that the effects of psychological stress on healing are moderate to large, resulting in poor surgical outcomes and poor wound healing associated with other forms of trauma (Padgett & Glaser 2003, Lusk & Lash 2005, Starkweather 2007, Von Ah & Kang 2007, Rosenberger et al. 2009, Lucas 2011, Broadbent & Koschwanez 2012).

Substantial data suggest that psychological stress and the subsequent immune system disruption can negatively impact wound healing, both directly and indirectly, with the most prominent impact occurring due to the effects of stress on cellular immunity.

Cellular immunity plays an important role in wound healing through the production and regulation of pro- and anti-inflammatory cytokines, which mediate many of the complex intricacies of wound healing. Dysregulation of various cytokines disrupts normal wound healing leading to delayed or improper healing, increased healing time, increased risk of infection, prolonged edema and wound complications, such as pathophysiological scars (Glaser et al. 1999, Broadbent et al. 2003, Ebrecht et al. 2003, Lucas 2011).

Psychological considerations, such as distress, depression and anxiety, have also been shown to slow wound healing. Patients reporting greater than average symptoms of depression or anxiety were four times more likely to be categorized as slow healers compared with patients reporting less distress. Heightened distress, associated with unhealthy behaviors, such as smoking, substance abuse, poor nutrition and alteration of normal...
Professional Ethics

The purpose of practicing our profession ethically is to promote and maintain the welfare of the client. Through their behavior, professionals can comply both with the law and with professional codes. If compliance with the law is the only motivation in ethical behavior, the person is said to be practicing mandatory ethics. If, however, the professional strives for the highest possible benefit and welfare for the client, he or she behaves with aspirational ethics (Corey et al. 2006, Fritz 2013), see Box 8.2.

Box 8.2

Eight principles that guide professional ethical behavior (adapted from Fritz 2013):

- Respect (esteem and regard for clients, other professionals, and oneself)
- Client autonomy and self-determination (the right to decide and the right to sufficient information to make the decision)
- Veracity (the right to the objective truth)
- Proportionality (benefit must outweigh the burden of treatment)
- Non-maleficence (the profession shall do no harm and prevent harm from happening)
- Beneficence (treatment should contribute to the client’s well-being)
- Confidentiality (respect for privacy of information)
- Justice and non-judgement (ensures equality among clients).

A traumatic scar injury can have multiple physical, neurological and cognitive consequences (Grigorovich et al. 2013). Many traumatic scar clients bring experiences of multiple surgeries, doctors’ appointments and physiotherapy visits before they walk through your clinic door. Or other consultations may be concurrent; the client may have just arrived at your door after such a visit.

With this understanding, it is imperative to gather a more complete picture of the client’s needs prior to each session. Pay attention to their mood and physical condition as they step through your clinic door. These are clues to open up dialogue about the pain they are experiencing and how to proceed in the session; for example, if the client

Ethical therapist behavior and clear communication are essential for a productive therapeutic relationship. The technical and interpersonal aspects of care are symbiotic. Touch demands intimate human contact, interaction and response. The therapist’s interactions and responses to the client are of parallel importance to skillful assessment and treatment (Fitch 2005).

A few rules in the professional setting are absolutes: a professional does not breach sexual boundaries with a client; clients are to be referred when the skills required are out of the scope of practice or training of the professional; all care must focus on giving help and avoiding harm; and clients are to be given complete information about the treatment (Fritz 2013).

Communication

Ethical and professional dilemmas tend to occur as a result of ineffective or mis-communication. Without a direct communication approach, ethical dilemmas tend to escalate and both parties suffer in the process. To make ethical decisions and resolve ethical dilemmas, we must communicate effectively. Good communication skills are required to retrieve information, maintain charting and client records, and provide information effectively so that the client can give informed consent (Fritz 2013).

A traumatic scar injury can have multiple physical, neurological and cognitive consequences (Grigorovich et al. 2013). Many traumatic scar clients bring experiences of multiple surgeries, doctors’ appointments and physiotherapy visits before they walk through your clinic door. Or other consultations may be concurrent; the client may have just arrived at your door after such a visit.

With this understanding, it is imperative to gather a more complete picture of the client’s needs prior to each session. Pay attention to their mood and physical condition as they step through your clinic door. These are clues to open up dialogue about the pain they are experiencing and how to proceed in the session; for example, if the client
presents tense and displays a more deliberate gait or holding pattern, ask about their day. Knowledge about their day-to-day life and prior experiences leading up to their session may indicate a need for changes in pressure and depth.

It is important to actively listen and empathically respond to the client as a whole person, not just the area of injury or symptom and create an appropriate set of protocols for each treatment session (Fitch 2014).

**Effective Listening and Empathetic Response**

Effective listening involves the development of focusing skills. You cannot listen effectively if you are distracted in any way (e.g. planning or preparing your response). Reflective listening involves restating the information to indicate that you have received and understood the message. Active listening may clarify a feeling attached to the message but does not add to or change the message (Fritz 2013).

Active listening is an important part of the therapeutic process. Listening carefully to the clients responses and making the client feel they are heard enables them to describe their situation more fully (Fitch 2014). Using your active listening and observation skills to validate client discomfort helps to build the therapeutic relationship.

Supportive, active listening when taking a case history (e.g. being non-judgmental and maintaining good eye contact) and asking open-ended questions, enables your client to provide responses in their own words.

There are some key concepts to the therapeutic listening relationship. When engaging a client during the intake, truly listen to the answer and the body language in which the statement is presented. Asking questions concerning the client’s history can sometimes feel uncomfortable and intrusive to the therapist and the client; however, asking direct questions is necessary for client safety. Active listening skills along with attention to the responses will give the therapist the information needed to formulate the proper protocol for the treatment session (Fitch 2014).

As massage therapists, we are visual and palpatory observers of the body. We may see scars that no-one in the client’s family or friends have seen and we may touch scars that no-one else, including the individual themselves, have touched. Traumatic scars carry their story within the tissue and mechanics of the body. Flexibility, comfort, edema and movement can play a significant role in the quality of life of the client (Fitch 2014).

Clinical experience has shown that gaining knowledge about the traumatic scar provides the therapist with important data, which helps shape protocol and enhance follow-up questions during each intake prior to the beginning of the session.

**Clinical Consideration**

Never underestimate the far-reaching, therapeutic value of attentive and compassionate listening. A critical turning point in a client’s healing journey can occur when he/she feels as if their story has been heard. The authors have experienced numerous times, over decades of practice, the client (and sometimes therapist) reduced to tears when the client discloses; ‘you are the first care provider to take the time to, really, listen to my story’. In that moment something within the client shifts, hope is sparked and where there is hope, change begins to unfold. Herein lies one of the unique aspects of a MT practice, the luxury of time. Our clinical structure differs from many forms of healthcare in that appointments are typically an hour long, providing the opportunity for clients to more thoroughly share the complexities of their experience. This, in combination with therapeutic touch, can impact the client in significant ways beyond the physical/functional value of the work.
This is not to imply that we do not address the abdominal region; as a region it is addressed, but the work is directed to the skin, fascia, muscles, vessels and nerves rather than, specifically, the adherences between viscera and articulating tissues and structures.

**Clinical Consideration**

One important note on postsurgical abdominal and visceral adhesions is made by Chapelle. In personal email correspondence, in her research with Bove, she indicated that:

... no specific time course for intervention has been clearly identified; however, it appears that adhesion formation occurs 6–12 hours after surgery. Early manual mobilization is a hypothesis of prevention – keep things moving to avoid adherence and subsequent complications. Mostly, once established, adhesions are difficult to affect.

Seems the old adage applies – an ounce (manual mobilization) of prevention outweighs a pound (subsequent surgical lysing) of cure. One can reasonably surmise that ‘keeping things moving’ as a means to prevent adherences can also apply to other tissues and the sliding layers between tissues and structures.

As massage therapists we are not called upon, nor is it within our scope of practice, to treat medical conditions and illnesses (e.g. diabetes, cancer) or acute critical trauma. Our primary role is to provide treatment that is designed to facilitate the wound-healing process or elicit a desired change in consequent complications in the form of impairments, such as any loss in body function or abnormal body structures that occur as a result of a medical condition or trauma (Andrade & Clifford 2008, Andrade 2013, Dryden & Moyer 2012).

Specific to this book are impairments associated with pathophysiological scars, such as adherences, contractures, fibrosis, postural and movement adaptations, edema, pain, anxiety, sympathetic nervous system (SNS)-hyperarousal, disturbed sleep and altered or impaired body awareness, which are representative of the realm of considerations that accompany the aftermath of trauma and poor wound healing outcomes.

**Clinical Consideration**

Reduction of anxiety and musculoskeletal pain are among the most established outcomes for massage (Moyer et al. 2009).

**Clinical Consideration**

In a cancer patient study it was determined that while both healing touch and massage lowered anxiety and pain, massage also reduced the need for pain medicine (Post-White et al. 2003).

**Clinical Consideration**

MT has been found to be effective for reducing burn-related depression, pain, pruritus and state anxiety, and positively impacting scar characteristics such as thickness, melanin deposition, erythema, transdermal water loss, and elasticity/tissue mobility (Eti et al. 2006, Roh et al. 2007, Goutos et al. 2009, Gürol et al. 2010, Cho et al. 2014).
pathway is cleared so excess fluid can drain into the terminus without becoming congested.

**Pumping**

Pumping technique is mainly used on the extremities and continues the use of the circle-shaped stretching of the skin. The MT may use their entire palm flat on the skin surface and may use one or both hands to achieve the result. The hand is in a palmar flexion with ulnar deviation and transitions into radial deviation and wrist extension at the end of the stroke (see Fig. 9.3).

**Figure 9.3**

**Pumping:** clearing the extremities (right arm). Pumping is mainly used on the extremities and also involves circle-shaped stretching of the skin and SF. The MT may use their entire palm, flat on the skin surface and may use one or both hands to achieve the result. The hand is in wrist flexion with ulnar deviation and transitions into radial deviation and wrist extension at the end of the stroke. All strokes begin proximal to the terminus and move distal. (A) Directional movements of the hand/wrist; (B–D) arrows indicate the direction of lymph flow.
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tissue layers. Like J-strokes, gentle circles can be used in the earlier stages of healing, once the scar is stable (i.e. no risk of dehiscence). Typically, pressure grades 1–4 (see Box 9.8) are used when applying this technique. This technique is

Figure 9.15
‘Cs’. Begin as noted in Fig. 9.10 – grasp the bulk of tissue in an approximation-compression manner. Simultaneously apply pressure into the tissue with the thumbs and deviate the wrists in an ulnar direction (curved arrows), bending the tissue into the letter ‘C’ until barrier/bind is felt.

Figure 9.16
‘Ss’. Begin by grasping the tissue in an approximation-compression manner, then apply pressure into the tissue with the thumbs and fingers in opposing directions, bending the tissue into the letter ‘S’ (begin at pressure grade 1–3 (see Box 9.8), and slowly increase until barrier/bind is felt).

Figure 9.17
J-stroke. Begin at one end of the scar (X), apply compression (pressure grading can vary depending upon the stage of healing and client comfort) then slowly glide in to the scar margin and circularly glide back away from the scar (white arrow), making a letter ‘J’. This technique can be applied along the entire length of the scar (yellow arrow). (B) J-stroke – compression, tension and bending. As noted in (A), glide into the scar margin, bending the scar tissue (white line) and then circularly glide back.
training you have received in trauma-related care (Mathieu 2012).

When overtaxed by the nature of our work we may begin to show symptoms that are similar to our traumatized clients: difficulty concentrating, intrusive imagery, feeling discouraged about the world, hopelessness, exhaustion, irritability, high attrition and negative outcomes (dispiritedness, cynical outlook, boundary violations) (Mathieu 2012).

**Indirect Trauma**

Every scar has a story and there is a high likelihood of massage therapists bearing witness to a client’s story. Their stories can be horrific and the recounting of them may have lasting impact on both the client and therapist.

Indirect trauma is the cumulative response to working with many trauma survivors over time. The indicators for indirect trauma resemble those of direct trauma (e.g. intrusive imagery, SNS stimulation, anxiety and feeling overwhelmed) and can impact the therapist’s personal and professional relationships (ISTSS 2015).

As is the case with trauma survivors, indirect trauma will look and feel different for each person. Some of the characteristics that may contribute to indirect trauma are the therapist’s personal history, usual ways of coping with challenge and distress, and current life circumstances (e.g., other stressors, lack of support network) (ISTSS 2015).

**Prevention**

Ways of coping and current life circumstances can contribute to a greater likelihood of compassion fatigue and indirect trauma. So it goes without saying that taking measures to minimize stressors, develop effective stress coping skills and build solid, personal and professional, support networks are important preventative measures.

The therapist’s way of working with survivors may contribute to or diminish the incidence of compassion fatigue and indirect trauma. For example, managing boundaries effectively can help protect the therapist from compassion fatigue indirect trauma. The importance of professional boundaries for the sake of client safety and therapist well-being cannot be emphasized enough.

**Care for the care provider**

It is important to be able to recognize indicators of compassion fatigue indirect trauma, as awareness is the first step toward change.

Any professional working with trauma survivors can benefit from identifying specific difficulties, assessing the contributing factors, targeting specific steps to take, and getting support from friends or colleagues in taking those steps (ISTSS 2015).

Compassion fatigue and indirect trauma can be addressed by attending to basic self care: work-life balance, ensuring quality time for play and rest. Healthy nutritional practices and regular exercise are also essential.

Additionally, massage therapists can benefit from appropriate professional training, connection with their colleagues, ongoing consultation for their work, and a place to talk about their experience of indirect trauma and, when necessary, professional assistance.

Finally, it is essential to embrace or restore meaning and hope. Each individual must find ways to connect with whatever in life is meaningful and gives purpose for that person (ISTSS 2015).

**References**
