The Concise Book of Trigger Points

Exam Edition

Third Edition

Simeon Niel-Asher

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DISCLAIMER
The techniques offered in this book are not a substitute for proper therapy from a registered practitioner; although aches and pains from trigger points are common, there can sometimes be an underlying pathology. It is advisable to always seek a proper diagnosis from a qualified medical practitioner. You follow the treatment techniques in this book at your own risk.

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Dedication
To my wife, sons, mother, friends, family, and wonderful patients.

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Preface

Welcome to the start of an exciting journey. I know there are many books on trigger points out there and I would like to thank you for reading this one. I was asked to write the first edition of the *Concise Book of Trigger Points* in 2003 and am delighted that, since its publication, the book has been translated into over 20 languages and become a best seller worldwide. Now, over 10 years later, I have completely updated and revamped the content. I am pleased to share current research, evidence, and advanced techniques for manual therapy practitioners, as well as simple self-help protocols that can do at home.

Many of you reading this book are suffering needlessly from pain and disability. So much of this can be relieved quickly and efficiently with simple trigger point therapy. My hope and prayer for you is that you will find relief in the pages of this book.

Simeon Niel-Asher 2014
www.nielasher.com

Abbreviations

ACh	Acetylcholine
AIIS	Anterior inferior iliac spine
ANS	Autonomic/automatic nervous system
ASC	Anterior sagittal chain
ASIS	Anterior superior iliac spine
ATP	Adenosine triphosphate
BK	Bradykinin
CRHR	Contract and relax/hold and relax
CNS	Central nervous system
CNSP	Cortico neuro somatic programming
COPD	Chronic obstructive pulmonary disease
DAC	Deep anterior chain
DSM	Deep stroking massage
EMG	Electromyogram
ENT	Ear, nose, and throat
GCA	Giant cell arteritis (temporal arteritis)
GI	Gastrointestinal
GTO	Golgi tendon organ
HLA	Human leukocyte antigen
ICT	Ischemic compression technique
IMES	Intramuscular electrotherapy stimulation
IMS	Intramuscular stimulation
IT	Iliotibial
ITPH	Integrated trigger point hypothesis
LC	Lateral chain
LTR	Local twitch response
MEP	Motor endplate
MET	Muscle energy techniques
MLD	Manual lymphatic drainage
MT	Myotherapy
MTP	Myofascial trigger point
NAT	Niel-Asher technique
NLP	Neurolinguistic programming
NMDA	N-methyl-D-aspartate
NMT	Neuromuscular technique
OMT	Osteopathic manipulative medicine
PID	Pelvic inflammatory disease
PIR	Post-isometric relaxation
PMR	Polymodal receptor
PNF	Proprioceptive neuromuscular facilitation
PNS	Peripheral nervous system
POL	Posterior oblique link
PRT	Positional release technique
PSC	Posterior sagittal chain
PSLE	Primary short lower extremity
RI	Reciprocal inhibition
RSI	Repetitive strain injury
RTA	Road traffic accident
SCS	Strain–Counterstrain
SCM	Sternocleidomastoideus
SLE	Systemic lupus erythematosis
SNS	Sympathetic nervous system
SR	Sarcoplasmic reticulum
STP	Super trigger point
TCM	Traditional Chinese Medicine
TFL	Tensor fasciae latae
TMJ	Temporomandibular joint
TMJD	Temporomandibular joint disorder
TPR	Trigger point release

Introduction

About Me

I learned about osteopathy from my great-uncle Sidney Roseneil when I was 14; he was an osteopath, acupuncturist, and naturopath in the 1960s, a time of great change in modern medicine. The notion of the body being encouraged to heal itself resonated within me even then. Osteopathic manipulative medicine (OMT) emphasizes the innate ability of the body to heal itself and teaches techniques to bring out this "semi-automatic" response. The body has self-regulatory and self-healing mechanisms that still outmaneuver and outsmart modern medicine. Through my work as an osteopath I learned to feel and understand the powerful and pre-verbal "language of touch." When I was introduced to trigger points (TPs) in my second year of college I just knew that I had found something special. I spent the next two and a half years, along with a couple of friends every weekend, visiting, learning, and watching David Warren, D.O., "the master" at work.

Since graduating in 1992 I have been working as a busy osteopath, researcher, student, and teacher. For over 22 years I have been privileged to meet and help many thousands of patients. I have been blessed with many gifts, a great family, great friends, and a wonderful, international career. I have met amazing people and been a part of their healing journey. I have been flown all around the world and worked with pop stars, Hollywood actors and actresses, gurus, politicians, and Olympians. In 1999 I developed and pioneered an advanced trigger point technique called the *Niel-Asher technique* (NAT). All this because I learned and understood one of the best kept secrets in pain medicine—*trigger point therapy*.

About You

Acute and chronic pain are highly motivating signals. When we are in pain we are vulnerable and will often try anything that is suggested. You may have been to the doctor and had MRIs and blood tests only to be given medication and sent away or told there is nothing wrong with you! Or worse—it is all in your mind. You may have tried physical therapy, nutrition, diets, acupuncture, chiropractic, osteopathy, massage, Bowen technique, Pilates, and so on, all to no avail. In the information age we are all increasingly bombarded with a bewildering array of new drugs, fad diets, therapies, and therapists, all "selling their wares."

Trigger point therapy is the *real deal*: it works fast, it is low cost, it is reproducible, it is evidence based, and it is easy to master. So why do all doctors and manual therapists not practice or know about it? The truth is that in time they all will. Many practitioners use trigger points every day in their work one way or another, even if they do not know it. Learning how to use them properly increases the efficiency, speed, and efficacy of treatment.

About Pain

Muscular (myogenic) pain and dysfunction can arise from many factors, such as trauma, chronic postures, sports injuries, and systemic disease. Muscular pain is a key part of our protective and defense mechanism. Pain is a valuable alarm bell to tell us something is wrong.

Furthermore, trigger points have been implicated in a range of conditions and they can often mimic others. Conditions ranging from headache, earache, and toothache, to back pain, tennis elbow (lateral epicondylitis), and even dizziness often have a trigger point at their very core.

In this book you are going to learn how to identify the source of your pain and apply effective and simple home relief. For those therapists already working with trigger points, I hope you will find this guide concise, practical, clinically relevant, and useful. In Chapters 4 and 5 I have included advanced techniques, such as dry needling, spray and stretch, proprioceptive neuromuscular facilitation (PNF), advanced hands-on positional release techniques (PRTs), and basic NAT protocols.

About This Book

This book is designed in quick reference format to offer useful information about the trigger points relating to the main skeletal muscles, which are central to massage, bodywork, and physical therapy. The information about each muscle is presented in a uniform style throughout. An example is given in Figure 1, with the meaning of headings explained in bold (some muscles will have abbreviated versions of this).

X Marks the Spot

While I have included dots/markings in the regions of the most common trigger points, please note that these are not exact locations, but are approximations. A number of factors influence the exact location of any given trigger point. Myofascia is a continuum, and minor variations in, for example, anatomy, posture, or weight bearing will have an impact on the location and formation of trigger points. In the "real world," you may well find that the trigger point location varies slightly from the dots on the muscles in Chapters 7–12. Varying the direction, amplitude, and applicator force will also have an impact on locating the trigger point.

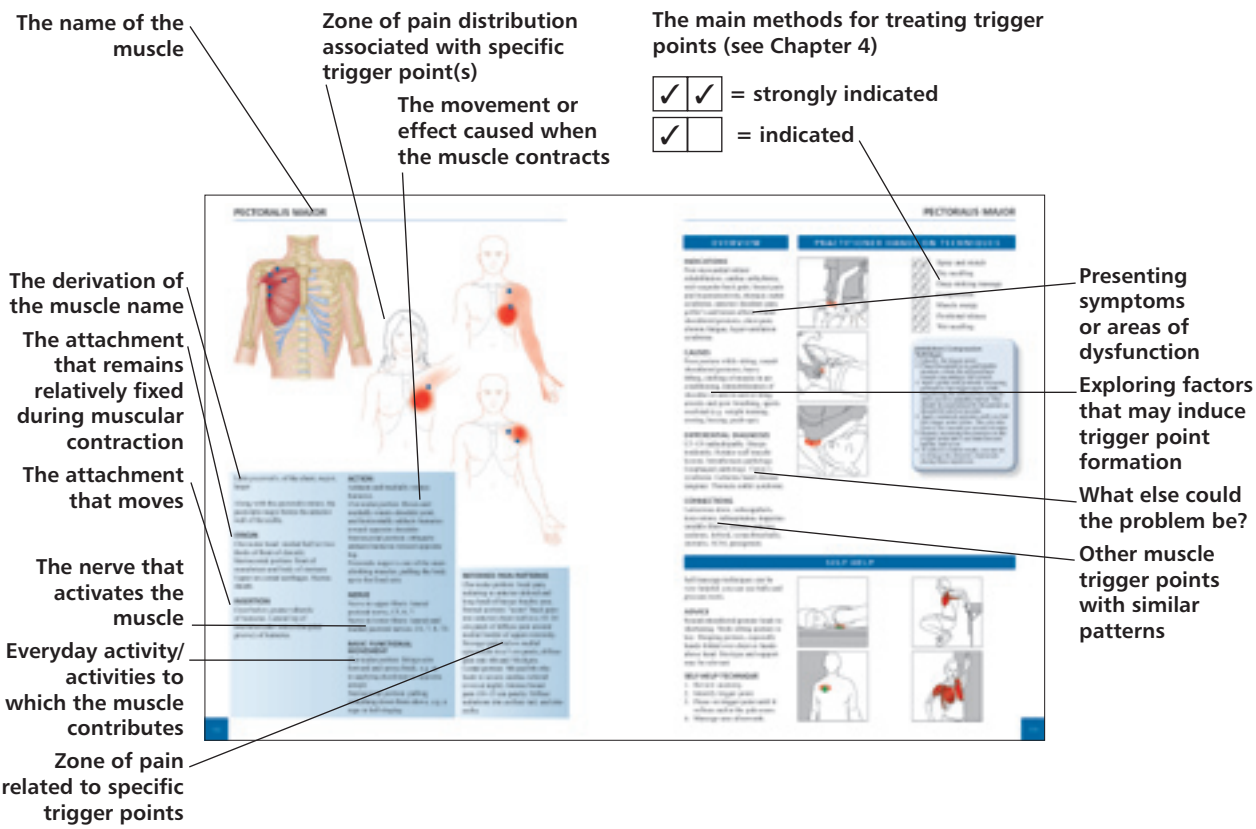

Figure 1: Sample page layout for a muscle.

A Note About Peripheral Nerve Supply

The nervous system comprises:

- The central nervous system (CNS)—i.e. the brain and spinal cord.
- The peripheral nervous system, including the autonomic nervous system—i.e. all neural structures outside the brain and spinal cord.

The peripheral nervous system consists of 12 pairs of cranial nerves and 31 pairs of spinal nerves (with their subsequent branches). The spinal nerves are numbered according to the level of the spinal cord from which they arise (the level is known as the *spinal segment*).

The relevant peripheral nerve supply is listed with each muscle presented in this book, for those who need to know. However, information about the spinal segment* (Figure 2) from which the nerve fibers emanate often differs between the various sources. This is because it is extremely difficult for anatomists to trace the route of an individual nerve fiber through the intertwining maze of other nerve fibers as it passes through its plexus (plexus = a network of nerves: from the Latin word meaning "braid"). Therefore, such information has been derived mainly from empirical clinical observation, rather than through dissection of the body.

In order to give the most accurate information possible, I have duplicated the method devised by Florence Peterson Kendall and Elizabeth Kendall McCreary. Kendall & McCreary (1983) integrated information from six well-known anatomy reference texts, namely those written by Cunningham, deJong, Foerster & Bumke, Gray, Haymaker & Woodhall, and Spalteholz. Following the same procedure, and then cross-matching the results with those of Kendall & McCreary, the following system of emphasizing the most important nerve roots for each muscle has been adopted in this book.

Let us take the supinator muscle as our example, which is supplied by the deep radial nerve, C5, **6**, (7). The relevant spinal segment is indicated by the letter [C] and the numbers [5, **6**, (7)]. Bold numbers [e.g. **6**] indicate that most (at least five) of the sources agree. Numbers that are not bold [e.g. 5] reflect agreement by three of four sources. Numbers not in bold and in parentheses [e.g. (7)] reflect agreement by two sources only, or that more than two sources specifically regarded it as a very minimal supply. If a spinal segment was mentioned by only one source, it was disregarded. Hence, bold type indicates the major innervation; not bold indicates the minor innervation; and numbers in parentheses suggest possible or infrequent innervation.

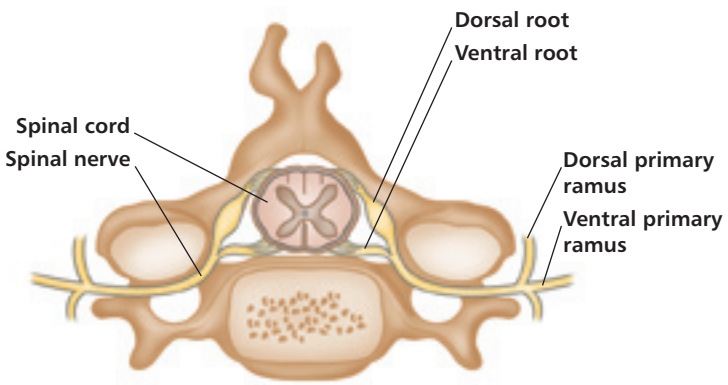

Figure 2: A spinal segment, showing the nerve roots combining to form a spinal nerve, which then divides into ventral and dorsal rami.

** A spinal segment is the part of the spinal cord that gives rise to each pair of spinal nerves (a pair consists of one nerve for the left side and one for the right side of the body). Each spinal nerve contains motor and sensory fibers. Soon after the spinal nerve exits through the foramen (the opening between adjacent vertebrae), it divides into a dorsal primary ramus (directed posteriorly) and a ventral primary ramus (directed laterally or anteriorly). Fibers from the dorsal rami innervate the skin and extensor muscles of the neck and trunk. The ventral rami supply the limbs, plus the sides and front of the trunk.*

1 Healing Yourself through Self-Help Techniques

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A True Story

Let us begin with a true story about John, who, according to his mother, was a "very, very sick little boy"; he nearly died of scarlet fever just before his third birthday. By the age of five he had developed whooping cough and chicken pox, and he was left with shaky health. In his teens, even though he played lots of sports and tried to stay healthy, John developed digestive problems; at age 14 he weighed 95 pounds. He was (eventually) diagnosed with colitis and celiac disease. John also suffered from back pain. At age 17 his father was so concerned that he sent John to the Mayo clinic in Rochester, Minnesota, where he was eventually diagnosed with Addison's disease of the adrenal glands (hypothyroidism).

In the course of time, John developed muscular pain. His problems started after a spinal accident during military service, as a result of which John underwent major back surgery. This was only partially successful, so he was treated with drugs and a back brace, but his pain got worse and worse; according to his brother it was a "constant source of difficulty." As time went on, he could not touch his toes or even do up his shoelaces. Sometimes he had to use crutches, and he was on constant medication. This medicine helped him temporarily but also left him with unwanted side effects, such as depression, osteoporosis, chronic constant muscular pain, and muscle spasms.

Janet and John

Finally, when John was in his late 30s, a friend introduced him to the "controversial but brilliant" MD Dr. Janet Travell, who was pioneering a new type of treatment called *myofascial trigger point therapy*. She treated him regularly and also recommended him heel lifts and a rocking chair to ease his pain. After only a few weeks John started getting better: for the first time in his life he was able to manage and reduce his pain. In fact, her treatments were so "profoundly successful" that she helped John to achieve and sustain his wonderful career—a career that changed the world!

John finally found the relief from his pain that had eluded the most eminent of doctors; his problems were mechanical—his muscles had developed trigger points. Dr. Travell's treatment was 'natural', mechanical, and simple; she had found a way to release hidden pain-codes locked within his muscular system. John publically acknowledged Dr. Travell's work and soon after he became the President of the United States, he appointed Janet as his "Personal Physician", the first woman and one of the few civilians to hold that post. Dr. Travell continued to explore and develop her theories and the science behind trigger points until her death in 1997 at the age of 95. Over time her legacy has been extensively researched, expanded and validated. Now it is time for you to benefit from these simple but powerful techniques.

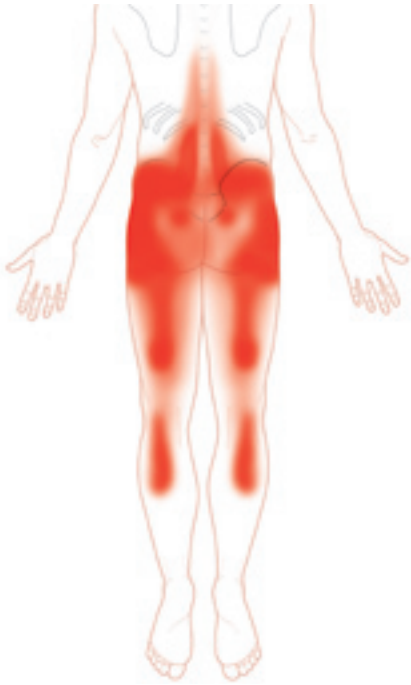

Figure 1.1: John F. Kennedy's pain map (suggested). Erector spinae bilateral lower, gluteus maximus, minimus and medius both sides; tensor fasciae latae both sides; gastrocnemius both sides.

Figure 1.2: Photograph of Janet Travell and John F. Kennedy; her most famous success story. http://www.janettravellmd.org

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A Few Words Before You Start

There are many reasons why you might have trigger points, so it is important to consider your trigger point pain in the context of the rest of your body. It must be stressed that the techniques offered in this book are not a substitute for therapy from a qualified practitioner; although aches and pains from trigger points are common, there can sometimes be an underlying pathology. *It is advisable to always seek a proper diagnosis from a qualified medical practitioner or experienced manual therapist.*

Acute and Chronic Pain

Authorities estimate that in 75–95% of muscular pain cases, myofascial trigger points are a primary cause! Therefore there is a high probability that understanding what trigger points are, and learning how to "switch them off," will help you overcome your pain.

Trigger points may arise for many different reasons; some of the most common factors to be aware of are:

- Head-forward posture (upper crossed pattern)
- Round shoulders (upper crossed pattern)
- Head to one side—telephone posture
- Occupational/ergonomic stressors
- Slouched standing (lower crossed pattern)
- Slouched sitting (e.g. computer screen/ ergonomics)
- Cross-legged sitting
- Habitual postures and/or habits
- Driving position
- Scoliosis
- Joint hypermobility
- Lifting/carrying
- TMJ syndrome
- Whiplash
- Primary short lower extremity (PSLE)
- Repetitive activity or sport
- Chronic vitamin and/or mineral deficiency
- Iron deficiency and hypothyroidism
- Medication induced (iatrogenic)

With any long-standing or chronic pain, there will be compensations and adaptations in a range of muscles locally and even remotely from the pain area.

Trigger points can be active (painful) or inactive (latent); they can also manifest in secondary muscles or as satellites in and around the vicinity of the primary pain. They can mimic angina, bursitis, prostatitis, appendicitis, cystitis, arthritis, esophagitis, carpal tunnel syndrome, pelvic inflammatory disease, diverticulosis, costochondritis, sciatica, and pain from a heart or gall bladder attack.

Trigger Points 101

The term *trigger point* was coined in 1942 by Dr. Janet Travell to describe painful lumps or nodules felt within tight bands of muscle. Trigger points all seem to have the following characteristics:

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

One of the most important features of trigger points is that they may be embedded in the muscles remotely from where the pain is felt. It is partly for this reason that so many therapies fail to help. More often than not, therapists and doctors tend to look at the place that hurts rather than find the source of the pain. A trigger point makes its host muscle shorter and fatter and reduces its efficiency: this can lead to pressure on nerves and blood vessels. Understanding trigger points and their maps will help guide you toward finding the source of your pain.

What are the physical characteristics of trigger points?

Our language for describing sensation is not highly sophisticated: unfortunately we have not yet evolved a suitable vocabulary to classify what we feel with our hands. With this in mind I will attempt to classify what trigger points feel like:

- Small nodules the size of a pinhead
- Pea-sized nodules
- Large lumps
- Several large lumps next to each other
- Tender spots embedded in taut bands of semi-hard muscle that feels like a cord
- Rope-like bands lying next to each other like partially cooked spaghetti
- Skin over a trigger point slightly warmer than the surrounding skin (due to increased metabolic/autonomic activity)

Myofascia

Imagine you are an orange. Your skin is (superficial) fascia embedded with hairs and receptors; the white tough pith beneath the skin is fascia; the bags that surround each segment are (deep) fascia; and, if you look really closely, the juice of the orange is held in even smaller fascial bags. We are all similar to some extent: our fascia is ubiquitous—it wraps and supports organs, bones, and tendons. Where it wraps muscles, it is known as *myofascia*. Fascia is a living tissue and has memory; it also helps transport and move chemical and other substances around the body. When we refer to "myofascial trigger points," we are talking about a trigger point in a specific muscle *and* its fascial wrapping. Myofascia connects many of the areas of the body together, which is why it is sometimes referred to as *connective tissue*.

What is trigger point therapy?

Trigger point therapy covers a range of techniques aimed at deactivating these painful knots. Many approaches are practical and "hands-on"; they can be performed at home with a partner or on your own with trigger point "tools." Combined with some simple lifestyle changes, trigger point therapy can yield dramatic, immediate, and sustainable results. The goals of this therapy are simple:

- To identify the correct trigger point(s)
- To pinpoint how or why they manifested
- To use appropriate techniques to deactivate the point(s)
- To develop strategies to prevent them returning

Pressing on trigger points:

- numbs and reduces pain in the treated area and in the area of the percieved pain;
- attenuates the pain feedback pathways;
- breaks the vicious cycle of pain and spasm;
- stretches tight structures, which will have an indirect effect on other tissues;
- opens out the plastic-wrap-like myofascial bag surrounding, investing, and supporting the muscles;
- stimulates the blood supply, to clear away debris and toxins;
- increases the release of powerful pain-killing agents called *endorphins*;
- affects the autonomic/automatic nervous system.

What is a referred pain map?

Trigger point referred pain is not the same as the referred shoulder pain of appendicitis or the jaw/arm pain associated with a heart attack! When you hold a painful trigger point for 5–6 seconds, part or all of the map should activate: this should reproduce your symptoms (often remotely from the area pressed).

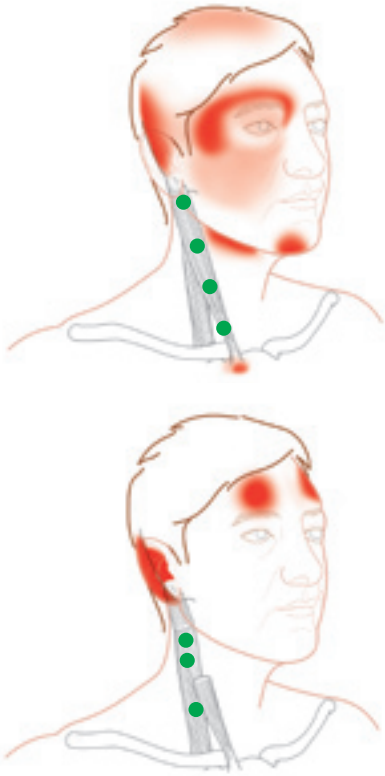

Figure 1.3: The SCM referred pain patterns.

What is the autonomic nervous system (ANS)?
Our ANS is concerned with our vegetative functions, such as sweating, digesting, and breathing. Trigger points can cause or contribute to many perplexing ANS symptoms, including sweating, skin blanching, coldness, gooseflesh, redness, excessive sweating, dizziness, dysmenorrhea, toiletry dysfunction, earache, stuffiness, and difficulty breathing.

Self-Treatment

Simply understanding what trigger points are and how they may be the cause of your pain is therapeutic. Reproducing your pain in the therapeutic context validates that you are not "crazy" and gives you a powerful self-help tool. I believe that it is essential to empower my patients to get better on their own, and that "knowledge is power." Please study the muscles, understand the process of treatment, and orientate yourself before you start.

Self-treatment will help you to understand, manage, and control your pain on your own, at home, and without a therapist. Once you get used to working with trigger points, you may even find your friends, relatives, and neighbors all want treatment. Who knows, you may even become a therapist yourself one day!

Throughout this book I have indicated the most effective self-help techniques and stretches based on my many years of practice.

What equipment do I need?
You should use a bed (or a couch), although sometimes a table with some padding will suffice. You will need some cream or lotion for the stroking massage technique. You might want some pressure "tools" to save your fingers and hands.

How do I know it is a trigger point?
You are looking for:

- Stiffness in the affected/host muscle
- Spot tenderness (exquisite pain)
- A palpable taut nodule or band
- Presence of referred pain
- Reproduction of the symptoms (accurate)
- Possible loss of skin elasticity in the region of the trigger point

The affected area may be moister or warmer (or colder) than the surrounding tissues, and may feel a little like sandpaper.

What bits of my hands should I feel them with? (see Figure 4.1 in Chapter 4.)

- Finger pads: remember to cut your fingernails (shorter is better).
- Flat fingers: use the fingertips to slide around the skin across muscle fibers.
- Pincer: pinch or grip the belly of the muscle between the thumb and the other fingers, rolling muscle fibers back and forth.
- Flat-hand palpation: useful in the abdominal region (viscera).
- Elbow: allows a stronger and shorter lever, which can be a distinct advantage.

How do I press/self-treat a trigger point?
For those of you who have worked with trigger points before, this concept will be very familiar. For the rest of you, there are two very simple, safe and effective techniques: (1) ischemic compression technique (ICT), and (2) deep stroking massage (DSM).

How much pressure do I use?
This is something that comes with experience, but as a rule of thumb the more painful the tissue, the slower and deeper the pressure. In all cases, the key words are "work slowly," "sensitively," and "thoroughly." Deep stroking massage should feel a bit like gently squeezing toothpaste out of a tube.

Another factor that determines the amount of force required to make a change is the muscle type (phasic type I/tonic type II fibers) and your morphology. This

will affect the depth of treatment. If you are "stocky," you should expect to have to work quite vigorously, especially into the postural muscles. If you are slight, you will not need to use as much force to cause a change in the tissues (see Chapter 2).

Which direction should the pressure/force be applied?
It is desirable to apply steady, deep, direct pressure to the nodule or pea-like trigger point. I have tried to represent this by the idea of a *hot zone*. The *heart* of the trigger point is located somewhere in this zone. You want to find the direction of pressure that, where possible, exactly reproduces the pain. It often amazes me that a slight change in the direction of the pressure can cause a totally different pain elsewhere. You will feel when you are "there."

Figure 1.4: Hot zones.

How do I know when I have done enough pressing?
Hold the trigger point for 6 seconds:

- If the pain diminishes rapidly, stay with it until the trigger point softens or evaporates beneath your pressure.
- If the pain stays the same or gets worse, come away for 15 seconds and then try again.
- Repeat 3 times if necessary.
- If the trigger point still does not deactivate after the third repetition, note it down as it may be a secondary or satellite point.

What do I do after I have come away from the point?
Follow all deep work with a gentle generalized effleurage massage. The area where you did the deep work may still be tender, but do not avoid it. This will help to dispel pain-inducing toxins from the area and stimulate the repair of the fascia.

Are the trigger points and referred pain patterns the same for everyone?
Generally yes, but they can sometimes move around depending on your size, shape, weight, etc. These factors will change the fat/muscle ratio and skew the position of the trigger points. They will also have an effect on the planes of the fascia, and hence the location of the trigger points. Similarly, scar tissue or keloid may cause a deviation in the myofascial strain pattern and hence the location of the trigger point.

What about the type and orientation of muscle fiber?
Depending on where they are in the body and the job they have to do, muscle fibers are arranged into various structures (see Figure 2.4 in Chapter 2). This allows the muscles to generate either more force or a more specific force. Locating a central trigger point will vary therefore according to the arrangement of muscle fibers within any given muscle. In the multipennate fiber arrangement, for example, several trigger points may exist in the middle of each of the functional components.

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

What is the frequency of treatment?
In my experience, for self-help hands-on treatments you should perform these sessions gently no more than once a day and preferably three to four days apart. Balls, rollers, or hooks may be used for up to 10 minutes per session and up to six times a day.

Tools

While fingers, elbows, and thumbs still remain the most readily utilized instruments for treatment, a variety of self-help tools have been developed for manipulating trigger points, including:

- Balls
- Canes
- Knobs
- TOLA System
- Rollers (foam)

Each of these tools has a different treatment effect. In general they are designed either to put pressure on a specific trigger point or to stretch out the muscles after treatment. There are many tools on the market and each has its plusses and minuses.

Tools such as balls and the knobble can be used instead of your hands and elbows to amplify pressure and reduce stress on your fingers. Other tools, such as the Theracane and the TOLA System, allow you to reach hard-to-access points.

Tools can be used standing, sitting, lying, or side lying. It is easy to overstimulate an active trigger point, so pressure should be applied slowly and gently until it is "just right." You should hold the point until it softens or the pain yields. It is OK to use pressure tools up to six times a day, depending on how chronic the problem is.

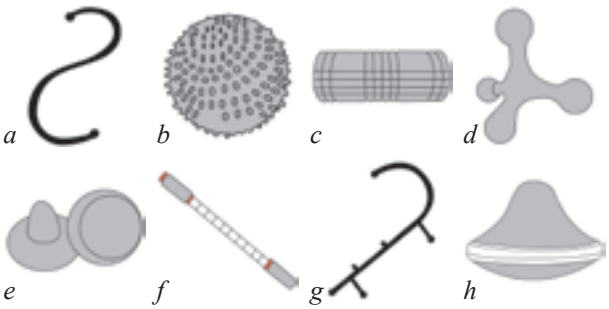

Figure 1.5: Self-help tools for manipulating trigger points, a) backnobber, b) ball, c) foam roller, d) four, e) knobble, f) one, g) theracane, h) tola.

For more information please visit www.nielasher.com.

How often should I treat a trigger point with balls or hooks?
This depends on how acute or chronic the problem is. For a chronic trigger point, you can work the area up to six times a day: persistence pays off. An acute problem may require less work than a chronic one. If you see an experienced practitioner, this will change. But I would like to stress that the frequency can vary from case to case because of a variety of factors.

Can I do any harm?
If you identify the correct point and deactivate it with care and love, the answer is—probably not. There may well be some soreness for up to 48 hours after treatment. If the soreness lasts or gets worse, please discontinue treatment immediately and seek a medical opinion.

Will bruising occur?
Bruising should not occur if you follow the instructions, but may occur if you are on blood-thinning medication. With time and experience, bruising becomes increasingly rare. I have found that it is not the depth of treatment (force) that will cause a bruise but usually the result of pressure being applied too quickly (velocity). Try to feel the muscles and tender nodules beneath the skin. Arnica creams and tablets have been suggested to reduce the incidence and severity of bruising. Unfortunately some people bruise more easily than others.

Tip
Try to feel the muscles and tender nodules beneath the skin and build up the pressure slowly; do not come away too quickly.

Will I be sore afterwards or experience side effects?
It is not uncommon to feel sore or bruised for 24–36 hours after treatment, but it is unclear whether these conditions are treatment effects or side effects. Treatment reactions are common and most severe following cervical manipulation; they are, somewhat controversially, proportionally related to treatment efficacy. Reactions may include other associated symptoms, such as fatigue or "flu-like" feelings, increased peeing, lethargy, and increased sleepiness.

Stretching
It is advisable to stretch on the hour, every hour, on the day of the treatment and then three times per day thereafter for a few weeks to several months. Stretching diagrams for each muscle are presented where appropriate.

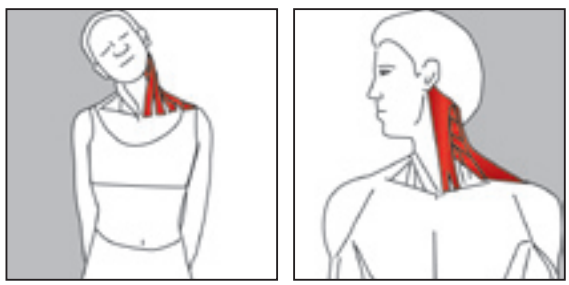

Figure 1.6: Stretching exercises for SCM.

Lifestyle and Diet
Studies have demonstrated that underlying health issues—such as folic acid, iron, vitamin, and/or mineral deficiency—may both contribute to and perpetuate trigger point activity. It is worth noting that tendons do not repair in the presence of nicotine! Furthermore, recent studies have indicated that the modern lifestyle tends to "underload" muscles and tendons, leading to internal fatty changes and increased vulnerability to damage. Other factors such as fatty foods and exposure to free radicals may also have a detrimental effect on our soft tissues. Supplements—for example omega-3, zinc, magnesium, iron, and vitamins K, B12, and C, as well as folic acid—may speed up your recovery.

Self-Help NAT Protocols
I have included my standard NAT protocols at the end of each colored muscle section. You will notice that these contain "super trigger points." While there is no "one size fits all" for all areas of the body, I have included protocols that have helped many thousands of patients over the years. For more information on super trigger points and NAT see Chapter 6.

What Is My Point?

At the beginning of each colored muscle section (Chapters 7–12) you will find a regional trigger point checklist. Have a good look through the muscle pages (see index on page 236 to locate the muscles indicated in the table below) and see if any of the pain maps seem familiar. The list of symptoms provided in Table 1.1 (below) should also help you to narrow down your search.

Signs and symptoms	Possible site(s) of trigger points (TPs)
Abdominal cramping/ colic	rectus abdominis—lateral border periumbilical
Abdominal fullness/ bloating/nausea	rectus abdominals, especially upper rectus abdominis paraxiphoid
Ankle weakness	tibialis anterior, fibularis
Anorexia	rectus abdominis
Bed wetting	active TPs in lower abdominal wall
Belching	abdominals (especially rectus abdominis), upper thoracic paraspinal
Bladder pain	upper adductor magnus
Bloating	transversus abdominis, rectus abdominis
Blocked ears/hearing loss/ hyperacusis/ hypoacusis	pterygoids, masseter
Blurred vision/visual disturbance	splenius capitis, eye muscles, sternal sternocleidomastoid, upper trapezius, orbicularis oculi, masseter (near vision)
Bruxism (grinding and/ or clenching of teeth)	temporalis
Buckling ankle	fibularis
Buckling hip	extension of both rectus femoris and upper vastus intermedius
Buckling knee	vastus medialis, vastus lateralis
Calf cramps	gastrocnemius
Cardiac arrhythmia	pectoralis major between 5th and 6th ribs, midway between nipple and sternum right side (inactivate sternal TPs first); pectoralis minor
Carsickness/ seasickness	sternocleidomastoid
Clumsy thumb (difficulty writing, buttoning)	adductor pollicis, opponens pollicis
Colic	transversus abdominis, rectus abdominis
Congestion/sinus pressure/sinus obstruction	masseter, pterygoids, internasal and sinus areas
Constipation	abdominal, possibly mesentery, obturator internus
Cough, dry hacking	convergence of sternal sternocleidomastoid and pectoralis
Diarrhea	lower abdominal area, right lower rectus abdominis, transversus abdominis
Difficulty climbing stairs	erector spinae, quadratus lumborum, tibialis anterior, soleus, long toe flexors
Difficulty swallowing	longus capitis, longus colli, medial pterygoid, digastricus
Diffuse abdominal/ gynecological pain	lower rectus abdominis, upper adductor magnus
Dimming of perceived light intensity	sternocleidomastoid
Disturbed weight perception of objects in hand	sternocleidomastoid
Dyspareunia (pain on sexual intercourse)	piriformis, upper adductor magnus
Elevated 1st rib	anterior scalene (can cause or contribute to costoclavicular syndrome)
Eye, explosive pressure in	splenius capitis
Eye, inability or slowness to raise upper lid	sternal sternocleidomastoid with spasm of orbicularis oculi
Eye, redness	frontalis, superior orbicularis oculi, sternal sternocleidomastoid
Eye irritation, redness	sternocleidomastoid, extrinsic eye muscles
Eye pain	sternocleidomastoid, occipitalis; longus capitis
Eye pain, behind the eye	temporalis, occipitalis, trapezius
Eye pain, deep	sternal sternocleidomastoid

Signs and symptoms	Possible site(s) of trigger points (TPs)
Eye tear production, excessive	front area temporalis, mid temporalis, sternal sternocleidomastoid, frontalis, superior orbicularis oculi
Female sexual dysfunction	piriformis and other short lateral rotators, pelvic floor
Flatulence	abdominals
Food intolerance	transversus abdominis
Foot drop; foot slap	tibialis anterior
Full sensation in rectum	obturator internus
Genital pain	upper adductor magnus, transversus abdominis
Grip strength, loss of	infraspinatus, scalenes, hand extensors, brachioradialis, abductor pollicis brevis
Heartburn	upper abdominal external oblique, upper rectus abdominis paraxiphoid, transversus abdominis
Hiccups	reflex contraction diaphragm, uvula
Hyperacusia (hypersensitive hearing)	temporalis, medial pterygoid
IBS (irritable bowel syndrome)	rectal TPs, abdominals (especially obliques), mid- and low-back multifidi, pelvic floor, upper adductor magnus
Impotence	piriformis and other short lateral rotators, pudendal nerve and blood vessel entrapment
Inability to stand up straight	psoas
Inability to sit still	gluteus maximus, obturator internus, gluteus maximus, upper adductor magnus
Incontinence, urinary and fecal	obturator internus (both)
Indigestion	rectus abdominis
Jaw opening, restriction of	masseter, many area TPs; the zygomaticus major alone may cause restriction of the opening by 10–20 mm
Knee weakness	rectus femoris, popliteus
Kneecap, locked	vastus medialis, vastus lateralis
Light sensitivity	frontalis, superior orbicularis oculi, sternal sternocleidomastoid, rectus capitis
Loss of attention or focus	rectus capitis anterior and lateral
Lumbago/low back pain	iliocostalis lumborum, longissimus thoracis, piriformis and other short lateral rotators, erector spinae, quadratus lumborum, gluteus medius, psoas major
Lump in throat	longus colli, longus capitis, digastricus
Nasal and sinus congestion	sternocleidomastoid, lateral pterygoid
Nausea	abdominals, upper thoracic paraspinals, transversus abdominis, temporalis
Nipple hypersensitivity/ intolerance to clothing	pectoralis major (check both sides)
Painful bowel movements	obturator internus
Palpable rigidity and deep tenderness of lower abdominal wall	T9 level of erector spinae
Petit mal seizure-like symptoms	rectus capitis major/minor
Phantom limb pain	after removal, TPs in the flesh surrounding the missing leg, arm, breast, or organ cause pain in the area of the removed tissue
Plantar fasciitis	superficial/deep intrinsic foot muscles
Postnasal drip	pterygoid, sternocleidomastoid
Projectile vomiting	"belch button" TP on either side, at or just below angle of 12th rib
Radial artery entrapment	pectoralis minor
Reflux	upper external abdominal oblique
Restless pain on prolonged sitting	gluteus maximus, piriformis, transverse perineal, inguinal ligaments, sacrotuberous ligament

Signs and symptoms	Possible site(s) of trigger points (TPs)
Retraction of the testicle	multifidi
Ringing in ears	pterygoids, masseters, medial pterygoid, splenius capitis, sternocleidomastoid, temporalis
Salivation, intense	mid temporalis
Sensitivity to sound and light	occipitalis
Shin-splint-type pain (anterior)	extensor digitorum longus, tibialis anterior
Shin-splint-type pain (posterior)	flexor digitorum longus, tibialis posterior
Shortness of breath	levator scapulae, scalenes
Shoulder impingement syndrome	serratus anterior
Stitch in side	serratus anterior and/or external oblique, diaphragm
Swallowing, sore and/or painful	pterygoids, digastricus, longus capitis, sternocleidomastoid
Swelling, foot and ankle	piriformis, soleus
Swelling, hands	scalene
Swelling, throat	digastric TPs (mimics swollen lymph nodes)
Swelling, leg	piriformis and other short lateral rotators, adductor longus/brevis
Swollen glands sensation	digastricus, sternocleidomastoid, pterygoids, anterior neck
Tachycardia, arrhythmia (including auricular fibrillation)	pectoralis major, intercostals, autonomic concomitants
Testicle, retraction	erector spinae
Thigh and leg weakness	rectus femoris
Thoracic-outlet-syndrome-type pain	scalenes, pectoralis major, latissimus dorsi, teres major and subscapularis, pectoralis minor, trapezius, levator scapulae, triceps brachii
Throat drainage	pterygoids, anterior neck muscles, digastricus
Thumb cramps	abductor pollicis longus
Tidal volume reduction	serratus anterior, intercostals
TMJS (temporomandibular joint syndrome)	lateral pterygoid, deep masseter
Toe cramps	long extensors of toes
Tooth pain and sensitivity (cold, heat, pressure)	clavicular sternocleidomastoid, trapezius, masseter, temporalis, upper trapezius, digastricus, longus capitis
Trigger finger	hand and finger flexors, finger flexor tendon sheath
Trigger thumb	flexor pollicis longus tendon sheath
Upper respiratory dysfunction	pectoralis major (bronchi), intercostals
Vertigo	sternocleidomastoid, upper trapezius, splenius capitis, semispinalis cervicis, temporalis
Vocal dysfunctions	pterygoids, anterior neck muscles, digastricus, laryngeal muscles
Vomiting	abdominals (especially rectus abdominis)
Vulvodynia	pelvic floor, psoas, rectus abdominis, and obturator internus
Writer's cramp	brachioradialis, forearm extensors

Table 1.1: Sites of TPs and associated symptoms (adapted from Starlanyl & Sharkey (2013)). These are listed in the following references: Bezerra Rocha et al. (2008), Doggweiler-Wiygul (2004), Funt & Kinnie (1984), Qerama et al. (2008), Sharkey (2008), Simons et al. (1998), Starlanyl & Copeland (2001), Teachey (2004), and Travell & Simons (1992).

Self-Help Trigger Point Release Techniques

For the purposes of this section, we will focus on two techniques—compression and deep stroking massage. These techniques are described in the work of Simons et al. (1998). More techniques are discussed in Chapter 4.

Inhibition Compression Technique

This technique involves locating the heart of the trigger/tender point. When this is compressed it may well trigger a specific referred pain map (preferably reproducing your symptoms). This technique involves applying direct, gentle and sustained pressure to the point:

PROCEDURE

1. Identify the tender/trigger point you wish to work on.
2. Place the host muscle in a comfortable position, where it is relaxed and can undergo full stretch.
3. Apply gentle, gradually increasing pressure to the tender point until you feel resistance. This should be experienced as discomfort and *not* as pain.
4. Apply sustained pressure until you feel the tender point yield and soften. This can take from a few seconds to several minutes.
5. Steps 3–4 can be repeated, gradually increasing the pressure on the tender/trigger point until it has fully yielded.
6. To achieve a better result, you can try to change the direction of pressure during these repetitions.

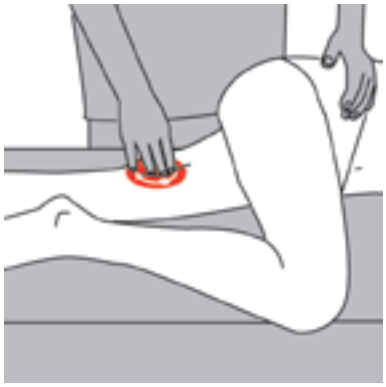

Figure 1.7: Inhibition compression technique.

Deep Stroking Massage Technique

This approach follows a technique advocated by Travell and Simons (Travell & Simons 1992; Simons et al. 1998), and involves a deep, slow stroking technique over a tender/trigger point rather than a compression as described in the previous technique. As well as deactivating the trigger point, this technique can have a stimulating or tonic effect on the host muscle.

PROCEDURE

1. Identify the trigger point and note the muscle fiber direction.
2. Place the patient in a comfortable position, where the affected/host muscle can undergo full stretch.
3. Lubricate the skin if required (I use simple blue Nivea Creme).
4. Identify and locate the tender/trigger point or taut band.
5. Working from the insertion of the muscle toward the muscle origin, perform slow stroking massage using your thumb/applicator just beneath the taut band, and reinforce with your other hand; it should feel a bit like squeezing toothpaste from a tube. This should be experienced as discomfort and *not* as pain.
6. Hold for 10–15 seconds and then complete the rest of the massage stroke toward the end of the muscle.

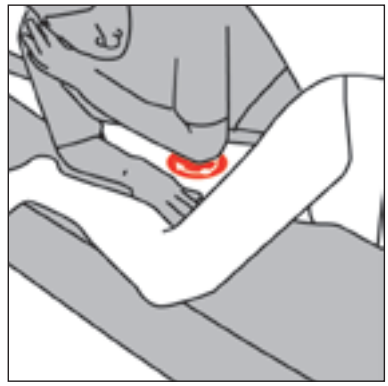

Figure 1.8: Deep stroking massage technique.

2 Skeletal Muscle, Muscle Mechanics, and Flexibility

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Skeletal Muscle

Skeletal muscles are where we find trigger points. The human body contains over 215 pairs of skeletal muscles, which make up approximately 40% of its weight. Skeletal muscles are so named because most attach to and move the skeleton, and so are responsible for movement of the body.

Skeletal muscles have an abundant supply of blood vessels and nerves, which is directly related to contraction, the primary function of skeletal muscle. Each skeletal muscle generally has one main artery to bring nutrients via the blood supply, and several veins to take away metabolic waste. The blood and nerve supply generally enters the muscle through its center, but occasionally toward one end, which eventually penetrates the endomysium around each muscle fiber.

Muscle Fibers

The three types of skeletal muscle fiber are: red slow-twitch, intermediate fast-twitch, and white fast-twitch. The color of each is reflected in the amount of myoglobin present, a store for oxygen. The myoglobin is able to increase the rate of oxygen diffusion, so red slow-twitch fibers are able to contract for longer periods, which is particularly useful for endurance events. The white fast-twitch fibers have a lower content of myoglobin; because they rely on glycogen (energy) reserves, they can contract quickly, but they also fatigue quickly, so are more prevalent in sprinters or in sports where short, rapid movements are required, such as weight lifting. World-class marathon runners have been reported to possess 93–99% slow-twitch fibers in their gastrocnemius (calf) muscle, while world-class sprinters only possess about 25% in the same muscle (Wilmore & Costill 1994).

Each skeletal muscle fiber is a single cylindrical muscle cell (Figure 2.1), which is surrounded by a plasma membrane called the *sarcolemma*. The sarcolemma features specific openings, which lead to tubes known as

transverse (or T) *tubules*. (The sarcolemma maintains a membrane potential, which allows impulses, specifically to the sarcoplasmic reticulum (SR), to either generate or inhibit contractions.)

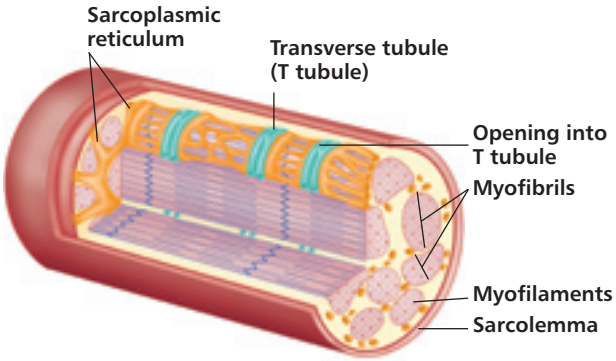


Figure 2.1: Each skeletal muscle fiber is a single cylindrical muscle cell.

Each muscle fiber is composed of small structures called *muscle fibrils* or *myofibrils* ("myo-" meaning "muscle" in Latin). These myofibrils lie in parallel and give the muscle cell its striated appearance, because they are composed of regularly aligned myofilaments. Myofilaments are chains of protein molecules, which under microscope appear as alternate light and dark bands. The light isotropic (I) bands are composed of the protein actin. The dark anisotropic (A) bands are composed of the protein myosin. (A third protein called *titin* has been identified, which accounts for about 11% of the combined muscle protein content.) When a muscle contracts, the actin filaments move between the myosin filaments, forming cross-bridges, which results in the myofibrils shortening and thickening. (See "The Physiology of Muscle Contraction.")

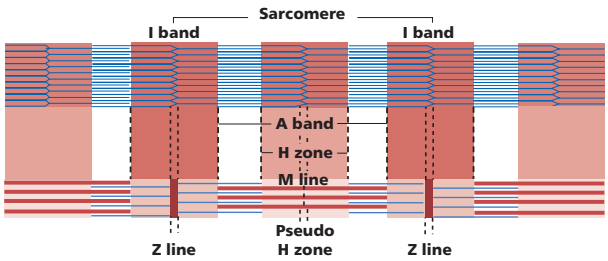


Figure 2.2: The myofilaments within a sarcomere. A sarcomere is bounded at both ends by the Z line; the M line is the center of the sarcomere. The I band is composed of actin, and the A band is composed of myosin.

Skeletal Muscle Composition

An individual skeletal muscle may be made up of hundreds, or even thousands, of muscle fibers bundled together and wrapped in a connective tissue sheath called the *epimysium*, which gives the muscle its shape, as well as providing a surface against which the surrounding muscles can move. Fascia, connective tissue outside the epimysium, surrounds and separates the muscles.

Portions of the epimysium project inward to divide the muscle into compartments. Each compartment contains a bundle of muscle fibers: each of these bundles is called a *fasciculus* (Latin = small bundle of twigs) and is surrounded by a layer of connective tissue called the *perimysium*. Each fasciculus consists of a number of muscle cells, and within the fasciculus, each individual muscle cell is surrounded by the endomysium, a fine sheath of delicate connective tissue.

Commonly, the epimysium, perimysium, and endomysium extend beyond the fleshy part of the muscle, the belly, to form a thick rope-like tendon or broad, flat, sheet-like tendinous tissue, known as an *aponeurosis*. The tendon and aponeurosis form indirect attachments from muscles to the periosteum of bones or to the connective tissue of other muscles. However, more complex muscles may have multiple attachments, such as the quadriceps (four attachments). Therefore a muscle typically spans a joint and is attached to bones by tendons at both ends. One of the bones remains relatively fixed or stable, while the other end moves as a result of muscle contraction.

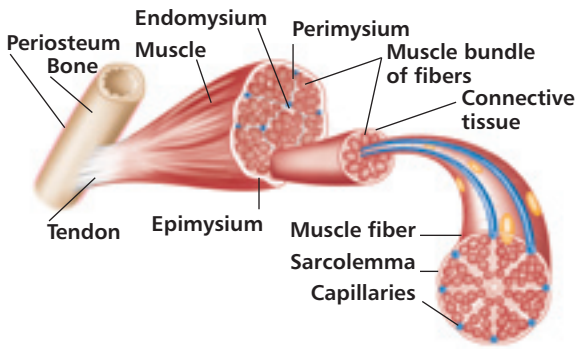


Figure 2.3: A cross-section of skeletal muscle tissue.

Types of Skeletal Muscle

Because of the arrangement of their fasciculi (English = fascicles), skeletal muscles come in a variety of shapes, depending on the function of the muscle in relation to its position and action. Parallel muscles have their fasciculi running parallel to the long axis of the muscle, e.g. sartorius. Pennate muscles have short fasciculi, which are attached obliquely to the tendon, and appear feather shaped, e.g. rectus femoris. Convergent (triangular) muscles have a broad origin with the fasciculi converging toward a single tendon, e.g. pectoralis major. Circular (sphincter) muscles have their fasciculi arranged in concentric rings around an opening, e.g. orbicularis oculi.

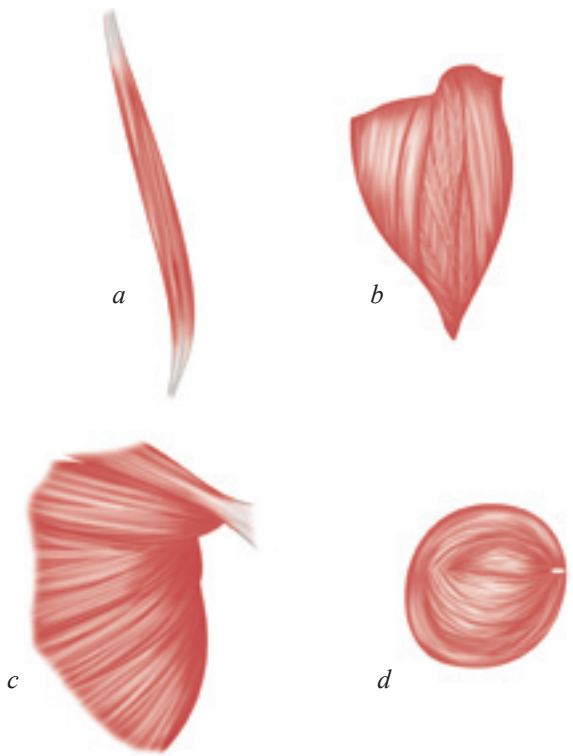


Figure 2.4: Muscle shapes: (a) parallel, (b) pennate, (c) convergent, and (d) circular.

Motor Units

Each muscle fiber is innervated by a single motor nerve fiber, ending near the middle of the muscle fiber. A single motor nerve fiber, together with all the muscle fibers it supplies, is known as a *motor unit*. The number of muscle fibers supplied by a single nerve fiber is dependent upon the movement required.

When an exact, controlled degree of movement is required, such as in eye or finger movement, only a few muscle fibers are supplied; when a grosser movement is required, as with large muscles like the gluteus maximus, several hundred fibers may be supplied.

Individual skeletal muscle fibers work on an "all or nothing" principle, where stimulation of the fiber results in complete contraction of that fiber, or no contraction at all—a fiber cannot be "slightly contracted." The overall contraction of any named muscle involves the contraction of a proportion of its fibers at any one time, with others remaining relaxed.

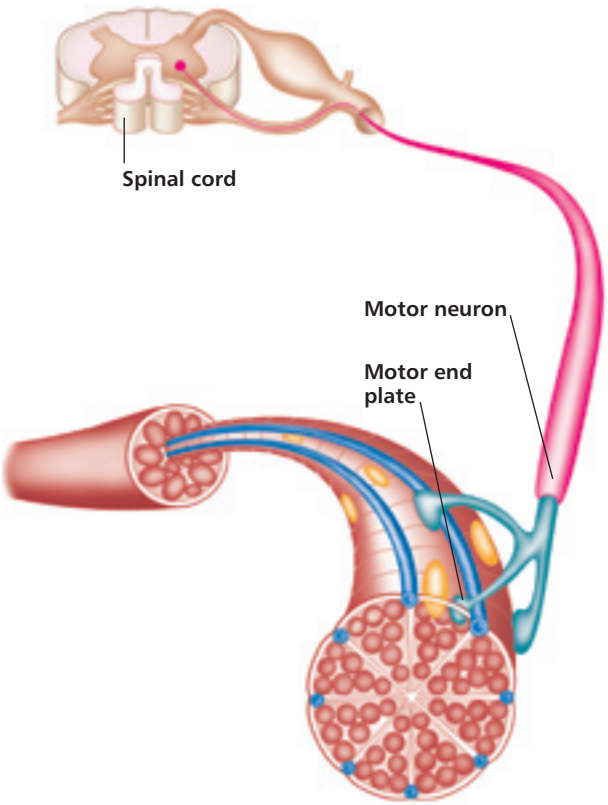


Figure 2.5: A motor unit of a skeletal muscle.

The Physiology of Muscle Contraction

Nerve impulses cause the skeletal muscle fibers at which they terminate to contract. The junction between a muscle fiber and the motor nerve is known as the *neuromuscular junction*, and this is where communication between the nerve and muscle takes place. A nerve impulse arrives at the nerve's endings, called *synaptic terminals*, close to the sarcolemma. These terminals contain thousands of vesicles filled with a neurotransmitter called *acetylcholine* (ACh). When a nerve impulse reaches the synaptic terminal, hundreds of these vesicles discharge their ACh. The ACh opens up channels, which allows sodium ions (Na+) to diffuse in. An inactive muscle fiber has a resting potential of about –95 mV. The influx of sodium ions reduces the charge, creating an endplate potential. If the endplate potential reaches the threshold voltage (approximately –50 mV), sodium ions flow in and an action potential is created within the fiber.

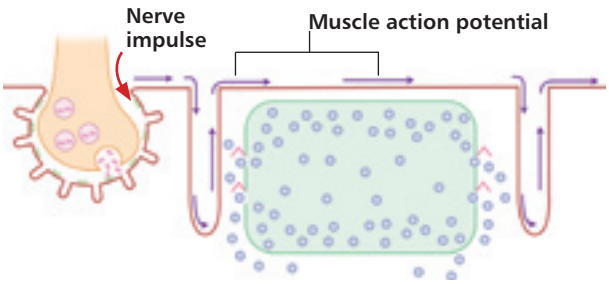


Figure 2.6: Nerve impulse triggering an action potential/muscle contraction.

No visible change occurs in the muscle fiber during (and immediately following) the action potential. This period, called the *latent period*, lasts from 3–10 msec. Before the latent period is over, the enzyme acetylcholinesterase breaks down the ACh in the neuromuscular junction, the sodium channels close, and the field is cleared for the arrival of another nerve impulse. The resting potential of the fiber is restored by an outflow of potassium ions. The brief period needed to restore the resting potential is called the *refractory period*.

Muscle Fiber Shortening

So how does a muscle fiber shorten? This has been explained best by the sliding filament theory (Huxley & Niedergerke 1954), which proposed that muscle fibers receive a nerve impulse (see above) that results in the release of calcium ions stored in the sarcoplasmic reticulum (SR). For muscles to work effectively, energy is required, and this is created by the breakdown of adenosine triphosphate (ATP). This energy allows the calcium ions to bind with the actin and myosin filaments to form a magnetic bond, which causes the fibers to shorten, resulting in the contraction. Muscle action continues until the calcium is depleted, at which point calcium is pumped back into the SR, where it is stored until another nerve impulse arrives.

Muscle Reflexes

Skeletal muscles contain specialized sensory units that are sensitive to muscle lengthening (stretching). These sensory units are called *muscle spindles* and *Golgi tendon organs* (GTOs), and they are important in detecting, responding to, and modulating changes in the length of muscle.

Muscle spindles are made up of spiral threads, called *intrafusal fibers*, and nerve endings, both encased within a connective tissue sheath; together, they monitor the speed at which a muscle is lengthening. If a muscle is lengthening at speed, signals from the intrafusal fibers will fire information via the spinal cord to the nervous system, so that a nerve impulse is sent back, causing the lengthening muscle to contract. The signals provide continuous information to and from the muscle about position and power (proprioception).

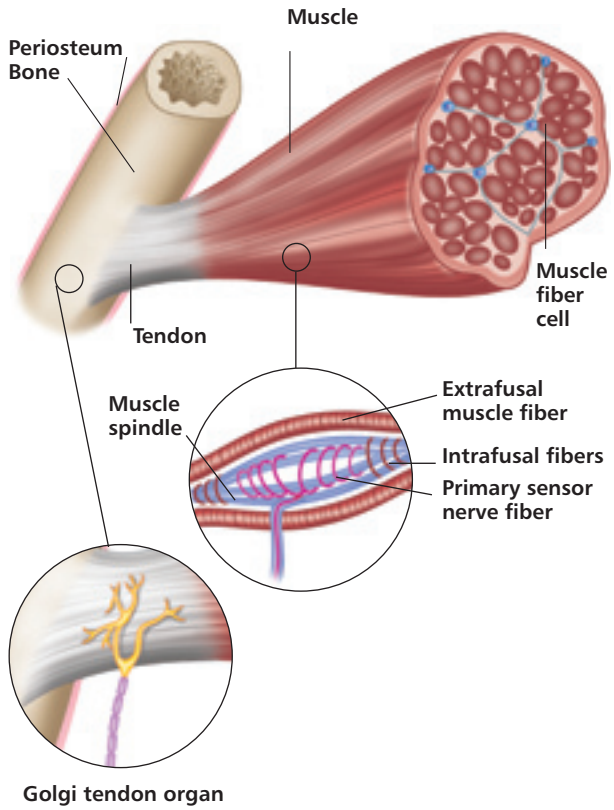


Figure 2.7: Anatomy of the muscle spindle and Golgi tendon organ (GTO).

Furthermore, when a muscle is lengthened and held, it will maintain a contractile response as long as the muscle remains stretched: this facility is known as the *stretch reflex arc*. Muscle spindles will remain stimulated as long as the stretch is held.

The classic clinical example of the stretch reflex is the knee jerk test, which involves activation of the stretch receptors in the tendon, which in turn causes a reflex contraction of the attached muscle, i.e. the quadriceps. Whereas the muscle spindles monitor the length of a muscle, the GTOs in the muscle tendon are so sensitive to tension in the muscle–tendon complex that they can respond to the contraction of a single muscle fiber. The GTOs are inhibitory in nature, performing a protective function by reducing the risk of injury. When stimulated, the GTOs inhibit the contracting (agonist) muscles and excite the antagonist muscles.

Musculoskeletal Mechanics

Most coordinated movement involves one attachment of a skeletal muscle remaining relatively stationary, while the attachment at the other end moves. The proximal, more fixed, attachment is known as the *origin*, while the attachment that lies more distally, and moves, is known as the *insertion*. (However, *attachment* is now the preferred term for origin and insertion, as it acknowledges that muscles often work so that either end can be fixed while the other end moves.)

Agonists, Antagonists and Synergists

Most movements require the application of muscle force, which often is accomplished by agonists, antagonists, and synergists. *Agonists* (or prime movers) are primarily responsible for movement and provide most of the force required for movement. *Antagonists* have to lengthen to allow for the movement produced by the prime movers and play a protective role.

Synergists assist the prime movers, and are sometimes involved in fine-tuning the direction of movement. They also prevent any unwanted movements that might occur as the prime mover contracts: in this context, such synergists are sometimes called *neutralisers*. This is especially important where a prime mover crosses two joints, because when it contracts, it will cause movement at both joints unless other muscles act to stabilize one of the joints. For example, the muscles that flex the fingers not only cross the finger joints, but also cross the wrist joint, potentially causing movement at both joints. However, it is because you have other muscles acting synergistically to stabilize the wrist joint that you are able to flex the fingers into a fist without also flexing the wrist at the same time.

A simple example of the role of agonists, antagonists, and synergists is the flexion of the elbow, which requires contraction (shortening) of the brachialis and biceps brachii (agonists) and relaxation of the triceps brachii (antagonist). However, the line of pull of the agonists (prime movers) will also supinate the forearm (twist the forearm, as in tightening a screw). If you want flexion to occur without supination, other muscles must contract to prevent this supination. The brachioradialis acts as the synergist by assisting the brachialis and biceps brachii.

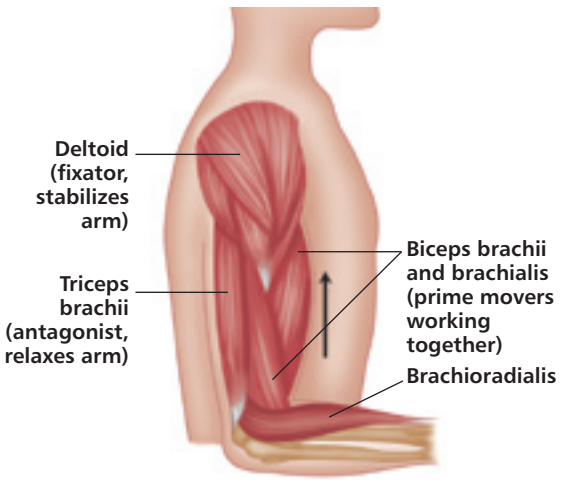


Figure 2.8: Flexion of the elbow, in which the brachialis and biceps brachii act as the agonists (prime movers), the triceps brachii as the antagonist, and the brachioradialis as the synergist.

Fixators

Synergists are more specifically referred to as fixators or stabilizers when they immobilize the bone of the prime mover's origin, thus providing a stable base for the action of the prime mover. The muscles that stabilize (fix) the scapula during movements of the upper limb are good examples. The sit-up exercise is another good example. The abdominal muscles attach to both the rib cage and the pelvis. When these muscles contract to enable you to perform a sit-up, the hip flexors will contract synergistically as fixators in order to prevent the abdominals tilting the pelvis, thus allowing the upper body to curl forward as the pelvis remains stationary.

Stabilizers and Mobilizers

Skeletal muscles can be broadly classified into two types:

1. *Stabilizers*, which essentially stabilize a joint. They are made up of slow-twitch fibers for endurance, and assist with postural holding. They can be further subdivided into *primary stabilizers*, which have very deep attachments, lying close to the axis of rotation of the joint; and *secondary stabilizers*, which are powerful muscles, with an ability to absorb large amounts of force. Stabilizers work against gravity, and tend to become weak and long over time (Norris 1998). Examples include the multifidus, transversus abdominis (primary), and gluteus maximus and adductor magnus (secondary).

2. *Mobilizers*, which are responsible for movement. They tend to be more superficial although less powerful than

stabilizers, but produce a wider range of motion. They tend to cross two joints, and are made up of fast-twitch fibers that produce power but lack endurance. Mobilizers assist with rapid or ballistic movements and produce high forces. With time and use, they tend to tighten and shorten. Examples include the hamstrings, piriformis, and rhomboids.

Importantly, all skeletal muscles are stabilizers and mobilizers—it depends on the movement and position of the body as to how the muscles are reacting at the time.

Types of Muscle Contraction

Muscle movement can be broken down into three types of contraction: concentric, eccentric, and static (or isometric). In many activities—such as running, Pilates, and yoga—all three types of contraction may occur to produce smooth, coordinated movement.

A muscle's principle action, shortening, where the muscle attachments move closer together, is referred to as a *concentric* contraction. Because joint movement is produced, concentric contractions are also considered *dynamic* contractions. An example is that of holding an object, where the biceps brachii contracts concentrically, the elbow joint flexes, and the hand moves up toward the shoulder.

A movement is considered to be an *eccentric* contraction where the muscle may exert a force while lengthening. As with a concentric contraction, because joint movement is produced, this is also referred to as a *dynamic* contraction. The actin filaments are pulled further from the center of the sarcomere, effectively stretching it.

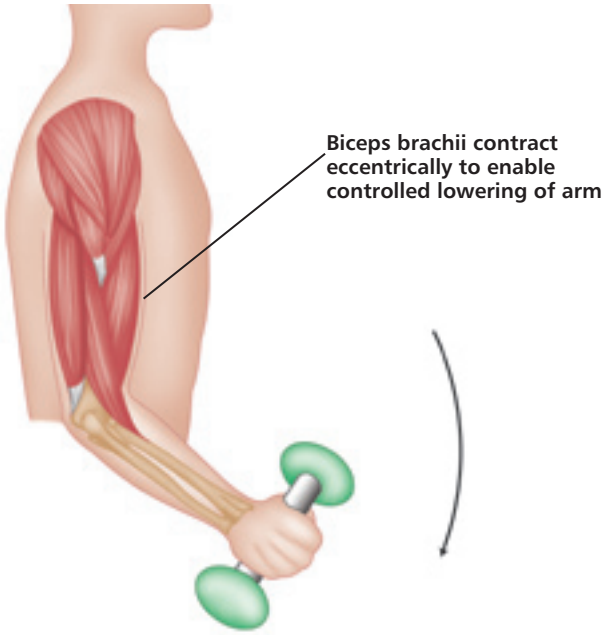


Figure 2.9: An example of an eccentric contraction is the action of the biceps brachii when the elbow is extended to lower a heavy weight. Here, the biceps brachii controls the movement by gradually lengthening in order to resist gravity.

When a muscle acts without moving, a force is generated but the length of the muscle remains unchanged. This is known as a *static* (isometric) contraction.

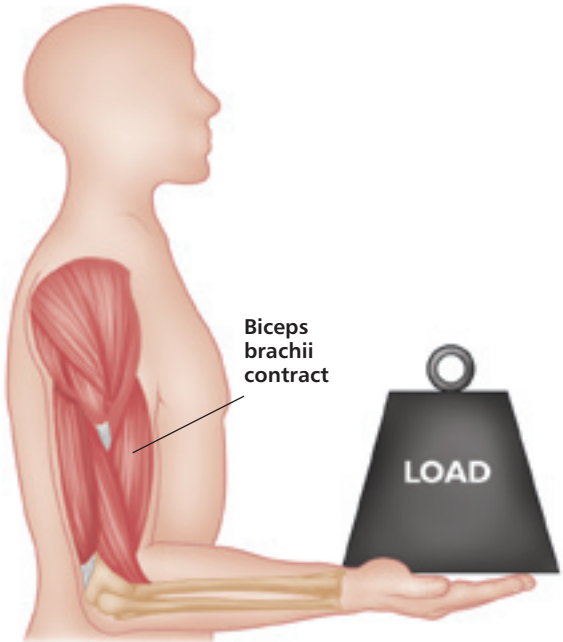
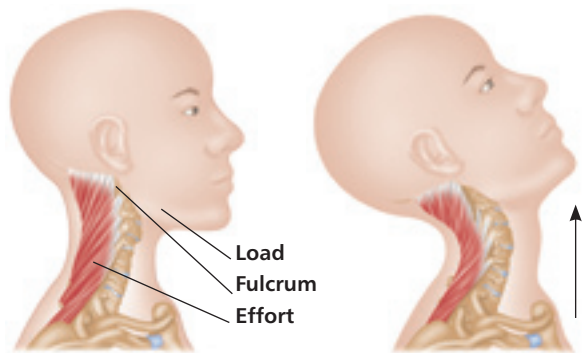


Figure 2.10: An example of a static (isometric) contraction, where a heavy weight is held, with the elbow stationary and bent at 90 degrees.

Transmission of Force

A lever is a device for transmitting (but not generating) a force and consists of a rigid bar moving about a fixed point (fulcrum). More specifically, a lever consists of an effort force, a resistance force, a rigid bar, and a fulcrum. The bones, joints, and muscles together form a system of levers in the body, where the joints act as fulcrums, the muscles apply the efforts, and the bones carry the weight of the body parts to be moved.

Levers are classified according to the positions of the fulcrum, resistance (load), and effort relative to each other. In a *first-class lever*, the effort and resistance are located on opposite sides of the fulcrum. In a *second-class lever*, the effort and the resistance are located on the same side of the fulcrum, and the resistance lies between the fulcrum and effort. Finally, in a *third-class* lever, the effort and resistance are located on the same side of the fulcrum, but the effort acts between the fulcrum and the resistance; this is the most common type of lever in the human body.



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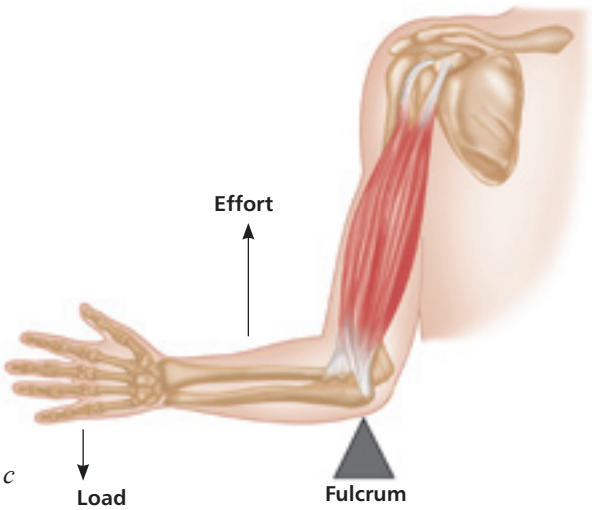

c

Figure 2.11: Examples of levers in the human body: (a) first-class lever, (b) second-class lever, and (c) third-class lever.

Generation of Force

The strength of skeletal muscle is reflected in its ability to generate force. If a weight lifter is able to lift 75 kg, the muscles are capable of producing enough force to lift 75 kg. Even when not trying to lift a weight, the muscles must still generate enough force to move the bones to which they are attached. A number of factors are involved in this ability to generate force, including the number and type of motor units activated, the size of the muscle, and the angle of the joint.

Reciprocal Inhibition

Most movement involves the combined effort of two or more muscles, with one muscle acting as the prime mover. Most prime movers usually have a synergistic muscle to assist them. Furthermore, most skeletal muscles have one or more antagonists that perform the opposite action. A good example might be hip abduction, in which the gluteus medius acts as the prime mover, with the tensor fasciae latae (TFL) acting synergistically and the hip adductors acting as antagonists, being reciprocally inhibited by the action of the agonists.

Reciprocal inhibition (RI) is the physiological phenomenon in which there is an automatic inhibition of a muscle when its antagonist contracts. Under special circumstances both the agonist and antagonist can contract together: this is known as a *co-contraction*.

Flexibility

When aiming to improve flexibility, the muscles and their fascia (sheath) should be the major focus of our flexibility training. While bones, joints, ligaments, tendons, and skin contribute to our overall flexibility, we have very little control over these factors.

Bones and joints are structured in such a way as to allow a specific range of motion. For example, the knee joint will not allow our leg to bend any further forward past a straight leg position, no matter how hard we try.

Ligaments connect bone to bone and act as stabilizers for joints. Stretching the ligaments should be avoided and can result in a permanent reduction of stability at the joint, which can lead to joint weakness and injury.

Tendons connect muscle to bone and consist of dense connective tissue. They are extremely strong yet very pliable. Tendons also play a role in joint stability and contribute less than 10% to a joint's overall flexibility; tendons should therefore not be a primary focus of stretching.

Stretching

Now that we have a general understanding of flexibility, muscles, and muscle mechanics, it will be useful to define what "stretching" is. *Stretching*, as it relates to physical health and fitness, is the process of placing particular parts of the body into a position that will lengthen the muscles and associated soft tissues (see also Chapter 5).

What happens when a muscle is stretched?

Upon undertaking a regular stretching program, you will notice that a number of changes begin to occur within the body and specifically within the muscles themselves. Other tissues that begin to adapt to the stretching process include the ligaments, tendons, fascia, skin, and scar tissue.

As discussed earlier in this chapter, the process of lengthening the muscles, and thereby increasing range of motion, begins within the muscles at the sarcomeres. When a particular body part is placed into a position that lengthens the muscle, the overlap between the thick and thin myofilaments begins to decrease. Once this has been achieved and all the sarcomeres are fully stretched, the muscle fiber is at its maximum resting length. At this point, further stretching will help to elongate the connective tissues and muscle fascia.

Embryological Development of Fascia

An overview of the embryological origin of connective tissues may provide some insights into the formation and location of trigger points. Trigger points tend to manifest within the epimysium according to myofascial strain patterns, which start to develop very early on in the developing embryo, and may also be related to fetal alignment in the womb. These strain patterns develop as we mature from childhood to adulthood and are influenced by, for example, posture, weight gain, and mechanical injury. Fascia supports organs, wraps around muscles, and condenses to form ligaments, aponeuroses, and even bone when infiltrated by calcium salts.

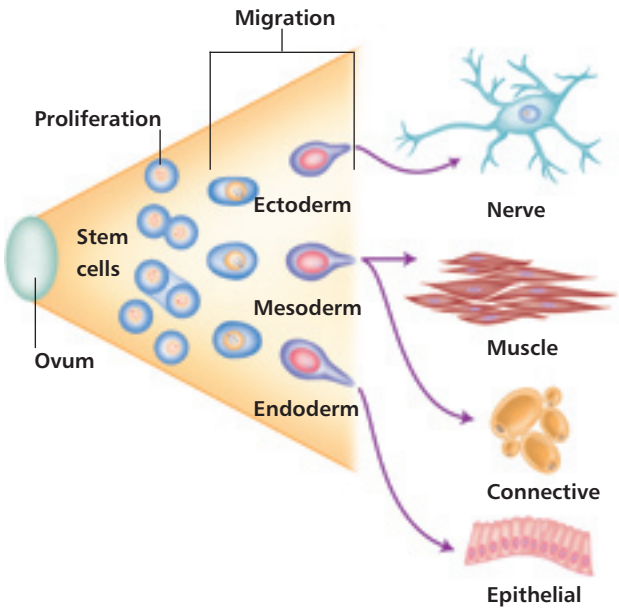

Figure 2.12: From the generalized ovum, cells proliferate, migrate, and differentiate into functionally specialized tissues.

By the end of the seventh week of development, the embryo has most of its organs, bones, muscles, and neurovascular structures in place. A group of "filler cells" begins to proliferate around these structures. This filler is derived from mesodermal tissue, a primitive fascia that is constructed from cells, fibers, and intercellular matrix. This matrix has the consistency of fiberglass insulation in a soft, jelly-like substrate. In most body areas, this primitive fascia remains supple until birth. In some areas, however, it condenses and becomes "directional" in response to internal and external pressures and tensions. Ligaments and tendons begin to form in these areas. Stress and strain lines develop in these tissues, and bone salts are laid down, causing primitive ossification. As the bones grow, they drag some of the connective tissue fibers into "differentiated" ligaments. An example of this

is the pre-vertebral cartilage, which grows and pushes into the mesodermal connective tissue beds. As it does so, it creates lines of stress that help to maintain integrity and provide a scaffold for further directional growth. As the bones start to grow, the complexity of strains and directional pulls results in the differentiated spinal ligaments (flavum, posterior longitudinal, etc.).

Furthermore, it has been reliably demonstrated that primitive organ growth relies on this mesodermal intracellular matrix. The "potential" pancreas, for example, will only differentiate into a mature organ in the specific presence of this "primitive" potential fascia. It has been suggested that the primitive or potential fascia creates a "specific energy field" in which the cells of the "potential" organ mature and differentiate (Schultz & Feitis 1996). This may make more sense when we consider that the bones, muscles, ligaments, and myofascial elements of connective tissue all share a characteristic pattern of growth.

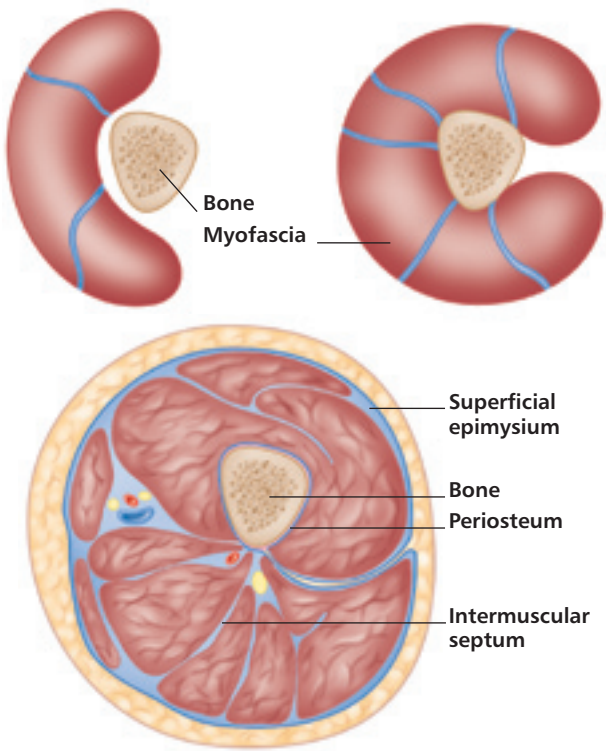

Figure 2.13: Fascial bag: the relationship between myofascia and bone.

The relationship between a developing muscle and its enveloping connective tissue, myofascia, is complex. The stress lines may provide a key to understanding this relationship. It has been suggested that during the second month of embryological development, connective tissue is laid down before muscle tissue, and that a clump of "potential muscle tissue, caught within this directional pull, differentiates into mature muscle oriented along the line of pull" (Schultz & Feitis 1996). These clumps of muscle tissue elongate through directional pressure. At this point they develop, differentiate, mature, and grow in size through mitotic cell reproduction to form the muscles as we know them.

In other words, it is the growth of fascia along lines of stress and strain that is the powerhouse of muscle orientation and development. This also explains why muscle action is not singular, but interconnected. For example, a contraction of the biceps brachii muscle will exert a force on the fascia of the whole arm, shoulder, and neck. Fascia has neither beginning nor end, and is described by anatomists according to location. On closer inspection the myofascial bags surrounding the muscles are actually part of a continuum. This may also go some way to explaining the referred pain patterns stimulated by pressing on a trigger point.

3 Trigger Points and Trigger Point Formation

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Trigger Point Definition

Drs. Janet Travell and David Simons (1992) described a trigger point as, "A highly irritable localized spot of exquisite tenderness in a nodule in a palpable taut band of (skeletal) muscle."

These hyperirritable localized spots can vary in size, and have been described as "tiny lumps," "little peas," and "large lumps"; they can be felt beneath the surface, embedded within the muscle fibers. If these spots are tender to pressure they may well be "trigger points." The size of a trigger point nodule varies according to the size, shape, and type of muscle in which it is generated. What is consistent is that they are tender to pressure. So tender in fact (hyperalgesia) that when they are pressed, the patient often winces from the pain; this has been called the "*jump sign*."

Myofascial trigger points may well be implicated in all types of musculoskeletal and mechanical muscular pain. Their presence has even been demonstrated in children and babies. Pain or symptoms may be directly due to active trigger points, or pain may "build up" over time from latent or inactive trigger points. Studies and investigations in selected patient populations have been carried out on various regions of the body. There is a growing amount of research evidence directly linking musculoskeletal pain to trigger points. A high prevalence of trigger points has been confirmed to be directly associated with myofascial pain, somatic dysfunction, psychological disturbance, and associated restricted daily functioning.

Etiology (Dommerholt et al. 2006)

Several possible trigger point mechanisms have been put forward, including:

- Low-level muscle contractions
- Uneven intramuscular pressure distribution
- Direct trauma

- Unaccustomed eccentric contractions
- Eccentric contractions in unconditioned muscle
- Maximal or submaximal concentric contractions

Embryogenesis

There is some evidence that myofascial trigger points may be present in babies and children (Davies 2004); they have also been demonstrated in muscle tissue after death.

Trigger points develop in the myofascia (hence the descriptor *myofascial trigger points* or MTPs), mainly in the center of the muscle belly where the motor endplate enters (primary or central). However, secondary or satellite trigger points often develop in a response to the primary trigger point. These satellite points often develop along fascial lines of stress, which may well be "built-in" at the time of embryogenesis. External factors—such as ageing, body morphology, posture, weight gain, or congenital malformation—also play a crucial role in trigger point manifestation and genesis. It has been suggested that myofascial trigger points are woven into the weft of the myofascial fabric as polymodal receptors; this may occur as far back as the splitting of the notochord and somites.

Evidence for Trigger Points

Studies over the past decade have imaged trigger points (Sikdar 2008, Shah 2005) and shown that their activation results in CNS activation through fMRI scanning (Niddam 2007), demonstrated electrophysiological activity at the trigger point (Hong 1998), and have shown biochemical changes in the trigger point zone (Shah 2005). Further studies have shown that manipulation of the trigger point modulates muscle function (Lucas 2010), and induces local and referred pain (Wang 2012).

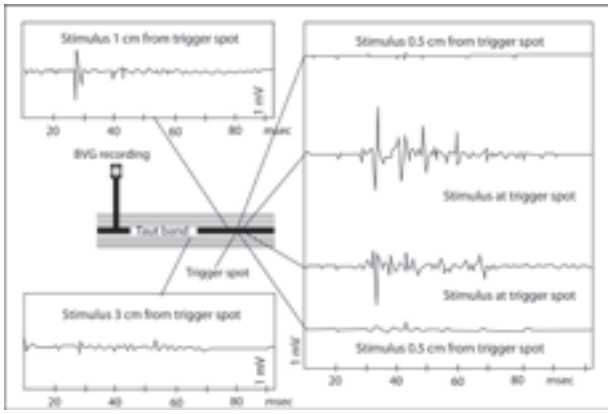


Figure 3.1: Local twitch response (LTR) in a rabbit tender spot. LTRs are elicited only when the needle is placed accurately within the trigger spot. (Adapted from: Hong 1996.)

In 1957, Dr. Janet Travell discovered that trigger points "generate and receive" minute electrical currents. She determined experimentally that trigger point activity could be accurately quantified by measuring these signals with an electromyogram (EMG). She went on to demonstrate that a trigger point could be accurately and reliably located by the same technique.

This is because electrical activity in a muscle in its resting state is "silent." When a small part of the muscle goes into contracture, as with a trigger point, a small, localized spike in electrical activity occurs. When needled with a monopolar Teflon-coated EMG needle, trigger points have been demonstrated to elicit a local twitch response (LTR). LTRs appear as high-amplitude polyphasic EMG discharges (Hong 1994, Wang & Audette 2000).

Trigger points have been reliably demonstrated by MRIs, and there is an abundance of studies showing their efficacy. In fact, only a few questions still remain unanswered:

- What is the action of a needle that causes a muscle to contract?
- Why is the twitch painful?
- Why does the pain go away quickly?

Further Evidence

Shah et al. (2003) performed a micro-dialysis experiment, in which two tiny microtubules were inserted (within a hollowed-out acupuncture needle) into the trigger point of the upper trapezius muscle. Saline solution was pumped through one tubule, while the other aspirated the local tissue fluid exudate; these microtubules were accurately positioned and maneuvered, under ultrasound guidance, from the outer zone of the trigger point toward the center.

Type of Trigger Point	Findings
Active	Lower pain thresholds, increased irritability, moderate hypoxia, lower pH, and highest levels of substance P, bradykinin, norepinephrine, and interleukin-1
Latent	Moderately increased levels of substance P, bradykinin, norepinephrine, and interleukin-1
Control group	Low levels of substance P, bradykinin, norepinephrine, and interleukin-1, normal pH

Table 3.1: Micro-dialysis findings.

Nine subjects were selected for the study; of these, three were said to manifest active trigger points, three latent trigger points, and three no trigger points (control group). To locate the trigger points, the subjects were first manually palpated, and then an algometer (pressure meter) was used to measure the amount of pressure required to elicit symptoms. In each of the nine subjects, the same zone of the upper trapezius was aspirated. The results have thrown some more light on the internal pathophysiology within trigger points, suggesting localized tissue hypoxia, increased acute inflammatory cascade, and lowered pH (acidosis) (see Table 3.1).

Referred Radicular Pain and Trigger Point Maps

Much like pain from a damaged nerve, trigger point stimulation causes referred pain. There are, however, several key differences, as highlighted in Table 3.2. It is advisable to perform neurological testing to eliminate any neural involvement.

Neural (Radicular) Referred Pain	Trigger Point Referred Pain
Specific dermatomal pattern	Map may extend across several dermatomes
Loss of sensitivity in dermatome	No loss of sensitivity
Loss of motor power to the point of paralysis	Weakness but no power loss on testing
Not induced by local muscle tissue pressure	Induced with local muscle tissue pressure
Loss of deep tendon reflex	No loss of deep tendon reflex

Table 3.2: Differences between neural and trigger point referred pain.

Trigger point referred pain is different to the referred shoulder pain of appendicitis or a jaw/arm pain with a heart attack. When you hold a trigger point for 5–6 seconds, part or all of the map should activate.

Acupuncture or Acupressure Points and Trigger Points

While there may be some overlap in trigger points and acupuncture points, they are not equivalent. Acupuncture points are said to be localized concentrations of "energy" that develop along electromagnetic lines (meridians). Trigger points, on the other hand, are discrete nodular tetherings in the myofascial tissues, which cause a specific and reproducible referred pain pattern when stimulated.

Acupuncture has long recognized painful points or "Ashi" points, often outside of a specific meridian. Some authorities believe these are trigger points. It has been suggested that the *general theory* of acupuncture points may have been put forward by ancient Chinese medicine as an "explanation" for the demonstrable and palpable presence of trigger points within myofascial tissues (Simons et al. 1998). Some authorities (Chaitow 1996) go much further, claiming that there is a 70% correlation between trigger points and acupuncture points. Furthermore, there is some evidence to demonstrate increased efficacy in pain relief when the trigger point is present at the site of an acupuncture point during treatment.

The "specific energy field" theory has been advanced by advocates of "Rolfing" (Hunt 1997; Myers 2001; Oschman 2003) as a bioenergetic field generated by the fascia itself. Some suggest that trigger points develop along lines of altered energetic activity or, at the very least, altered strain patterns.

Fibromyalgia

"Fibromyalgia is a complex syndrome characterized by pain amplification, musculo-skeletal discomfort, and systemic symptoms" (Starlanyl & Copeland 2001). Fibromyalgia means pain in the fibrous, connective, and tendinous tissues of the body, and fibromyalgia syndrome is characterized by widespread diffuse musculoskeletal pain and fatigue. The cause of the disorder is still unknown.

Like myofascial trigger points, fibromyalgic pain arises from the connective tissues, muscles, tendons, and ligaments; similarly, fibromyalgia does not involve the joints. Myofascial trigger points and fibromyalgia are often mistaken for each other; however, they are discrete conditions. Both conditions may be linked to psychological depression. Unlike trigger point manifestation, fibromyalgia is believed to have a systemic (CNS) origin. Starlanyl & Sharkey (2013) reviewed the current evidence and assert that, while fibromyalgia and MTPs are distinct entities, both are maintained by central and/or peripheral sensitization (see p.39).

Unlike with trigger points, which cause a specific and reproducible pattern of referral, patients with fibromyalgia describe that they "ache all over" (although some do describe localized tender spots). Patients with fibromyalgia also describe their muscles as feeling like they have been "pulled" or overworked. Sometimes the muscles twitch and at other times they burn. More women than men are affected by fibromyalgia, but there is no age profile. In contrast to fibromyalgic points, trigger points have been successfully photographed using electron microscopy. A listing of the basic differences is shown in Table 3.3.

	Pain Location	Type of Pain	Muscular Quality on Palpation
Trigger point	Specific and discrete Mediated locally in the region of the motor endplate from the peripheral nervous system (PNS)	Referred in a specific pattern	Tight and stiff, warm
Fibromyalgia	General Mediated centrally (CNS)	Vague, aching, burning, diffuse, widespread	Doughy and soft

Table 3.3: Some basic differences between fibromyalgia and trigger points (Juhan 1987).

Chapman's Reflex Points (Tender Points) vs. Trigger Points

These points were first described by Dr. Frank Chapman, D.O. in 1920. Chapman described his palpatory findings as "small pearls of tapioca that are firm, partially fixed, and located under the skin in the deep fascia." The points may be useful as a diagnostic aid for connecting internal pain to a specific pathology, and have been used diagnostically within osteopathic manipulative medicine (OMT).

Chapman's reflex points are distinct entities from trigger points. Unlike trigger points (which cause a specific referral map), Chapman's points are always small and discrete, with tissue local texture changes; moreover, they are located just deep to the skin. Practitioners claim they are an outward physical representation of internal dysfunction or pathology of an organ system. The current hypothesis is that they are caused by overstimulation of the sympathetic nervous system (SNS), resulting in a concentration of ionized fluid, although no histological basis has yet been established.

Nutritional and Biochemical Factors

Simons et al. (1999) suggested that changes in biochemical inputs might influence trigger point formation and/or perpetuation (see Table 3.4). Gerwin et al. (2004) expanded upon this, asserting that nutritional and biochemical factors may well both precipitate and maintain chronic myofascial pain and "must be" considered during treatment.

Factor	Influence
Allergic/ hypersensitivity	May have a potentizing effect (Brostoff 1992).
Hormonal	Estrogen and thyroid deficiency may impact the endoplasmic environment, leading to increased trigger point development and/or perpetuation (Lowe & Honeyman-Lowe 1998).
Chronic viral, yeast and/ or parasite infection	May increase the likelihood of trigger point formation (Ferguson & Gerwin 2004).
Vitamin C deficiency	May perpetuate trigger point longevity.
Iron deficiency (ferritin)	10–15% of people with chronic myofascial pain syndromes may be iron deficient (Simons et al. 1999). Serum levels of 15–20 ng/ml indicate depletion, but even levels below 50 ng/ml may be significant (Gerwin et al. 2004).
Vitamin B1, B6, B12 deficiency	May increase tiredness, fatigue, and chronic trigger point formation.
Magnesium and zinc deficiency	Levels in the lower realm of normal may be low for some people.
Vitamin D deficiency	Implicated in almost 90% of patients with chronic musculoskeletal pain (Plotnikoff 2003).
Cytochrome oxidase	Lowered levels are common in patients with myalgia. Associated with tiredness, coldness, extreme fatigue with exercise, and muscle pain.
Folic acid	May sufficiently change the internal endoplasmic environment to increase trigger point development and/or perpetuation.

Table 3.4: Biomechanical factors. (After Simons et al. (1999) and Gerwin (2004).)

Autonomic Nervous System (ANS) Involvement

Another important feature of trigger point activity is a change or modulation in the local ANS. As discussed above, various inflammatory chemicals have an effect on the ANS. Hubberd (1996) suggested the autonomic effects were due to dysfunctional changes in the muscle spindle. Gerwin & Dommerholt (2006) have suggested a possible mechanism involving alpha- and beta-adrenergic receptors at the motor endplate.

The altered chemical milieu around the active trigger point mentioned above (Shah et al. 2003) is also a recipe for sympathetic facilitation and mechanical sensitization. These chemicals are well noted for increased vasoconstriction, an increased sympathetic release of noradrenaline, and increased sensitivity to noradrenaline. Furthermore, the presence of interleukin IL-8 in the local chemical soup may also be implicated in ANS activity. IL-8 has been demonstrated to induce mechanical hyper-nociception, which is inhibited by beta-adrenergic receptor antagonists (Shah et al. 2005).

Known symptoms include:

- Hypersalivation—increased saliva production
- Epiphora—abnormal overflow of tears down the cheek
- Conjunctivitis—reddening of the eyes
- Ptosis—drooping of the eyelids
- Blurring of vision
- Increased nasal secretion
- Goose bumps

Differential Diagnosis

Many other conditions feature muscle pain and trigger points, including (Dommerholt & Issa 2003):

- Hypothyroidism
- Systemic lupus erythematosis (SLE)
- Lyme disease
- Ehrlichiosis
- Candida albicans infections
- Myoadenylate deaminase deficiency
- Hypoglycemia
- Parasitic (fascioliasis, giardia, amebiasis)

Trigger Points and Muscle Fiber Type

All muscles contain a blend of type 1 and type 2 fibers (Janda 2005; Lewit 1999). This has a direct correlation with how chronic symptoms might develop if left untreated.

1. Type 1 fibers are postural and tend to respond to stress or overuse by shortening and becoming hypertonic. A trigger point in a muscle with a high percentage of type 1 fibers may take longer to respond to treatment.

2. Type 2 fibers are built for explosive, short-term activity and tend to become weak, atrophic, and hypertonic under chronic or sustained endurance. A trigger point in a muscle with a high percentage of type 2 fibers may respond more rapidly to treatment.

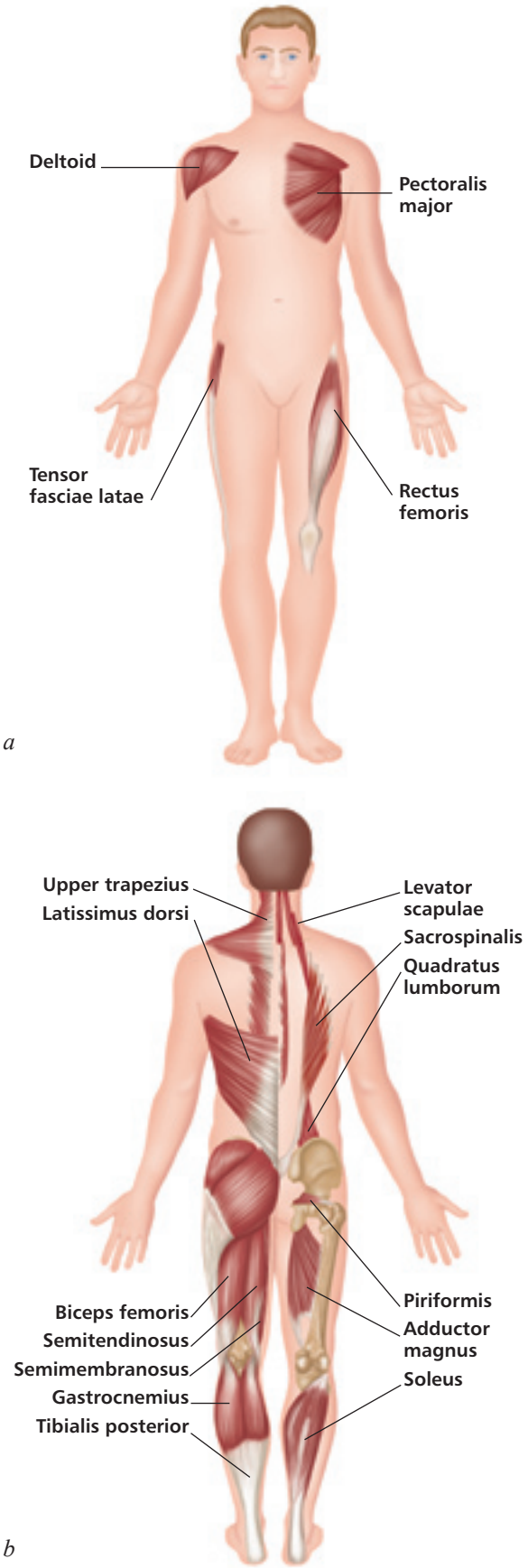


Figure 3.2: Major postural muscles of the body: (a) anterior view, (b) posterior view.

Trigger Point Formation and Posture

Poor posture is a powerful "activator and perpetuator" of myofascial trigger points (Simons et al. 1998) and is always worth considering in chronic trigger point syndromes. Postural muscles tend to have a greater percentage of type 1 fibers; this characteristic, as discussed, may lead to a more resistant type of trigger point. Human beings are four-limbed animals, and like our cousins, we are designed to move around and hunt for food. I am sure that if one put a gorilla in a chair all day, it would get a bad back!

It is a fact that in the developed world many occupations involve prolonged sitting, often at a computer screen. Ergonomics is a booming industry, focusing on the interactions of people and their working environments; however, not all workplaces can afford to implement proper ergonomic interventions. For many people, long and monotonous days spent in front of a computer screen often lead to chronic and maladapted postures. Where possible, it is essential to identify the postural abnormalities and how they impact the patient's symptoms, and offer to remedy the situation via ergonomic advice, treatment, and/or exercise.

The most common mechanical maladaptations are:

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

Trigger points are common in the following postural structures: upper trapezius, levator scapulae, sternocleidomastoideus (SCM), erector spinae, musculoligamentous apparatus of the lumbar spine, gluteus medius, and gastrocnemius/soleus complex.

Postural Trigger Points and "Cross Patterns"

Osteopathic, chiropractic, and other physical therapeutic modalities have all observed "cross-patterned" relationships within the body, from upper to lower and left to right. Janda (1996) recorded the two most common "crossover" postural strain patterns—*upper* and *lower*. Myers (2001) has further explored and developed these observations in his seminal book *Anatomy Trains*. These myofascial strain patterns have a profound effect on the pathogenesis and chronicity of trigger point development. Trigger points can be found throughout the muscles listed below.

Upper Crossed Pattern Syndrome

This can be observed in the "round-shouldered, chin-poking, slumped posture," which also compromises normal breathing. In such cases, pain is often reported in the neck, shoulder, chest, and thoracic spine (these areas are often restricted). An oblique cross can be drawn through the glenohumeral joint, indicating the functional "crossover" changes in muscular relationships. The main muscles in the upper cross pattern affected are shown in Figure 3.3.

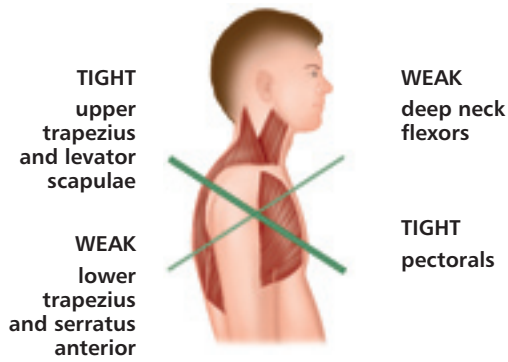


Figure 3.3: Upper crossed pattern syndrome.

Lower Crossed Pattern Syndrome

This can be observed in the "sway-back" posture, with weak abdominals and gluteals and overtight erector spinae, quadratus lumborum, TFL, piriformis, and psoas major (see Figure 3.4).

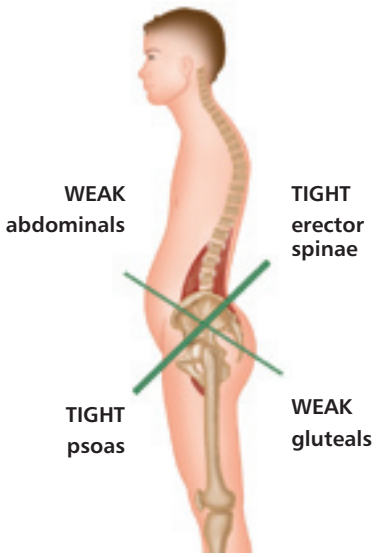


Figure 3.4: Lower crossed pattern syndrome.

Trigger Points Within Sarcomeres

Muscle contraction occurs at the level of the sarcomeres (see Chapter 2). Even the slightest of gross movements requires the coordinated contraction of hundreds of sarcomeres. The sliding process requires: (a) an initializing stimulation or impulse from a local motor nerve; (b) energy; and (c) calcium ions.

Physiology of Movement

When the brain wants to move a muscle, it fires a message through a motor nerve. The local motor nerve terminals translate this impulse chemically by producing ACh, which triggers an increase in sarcomere activity. The energy required for this process is released by the *mitochondria* (energy centers) in the cells. Calcium ions inhabit the sarcoplasmic reticulum, which is found in the sarcoplasm of skeletal muscle.

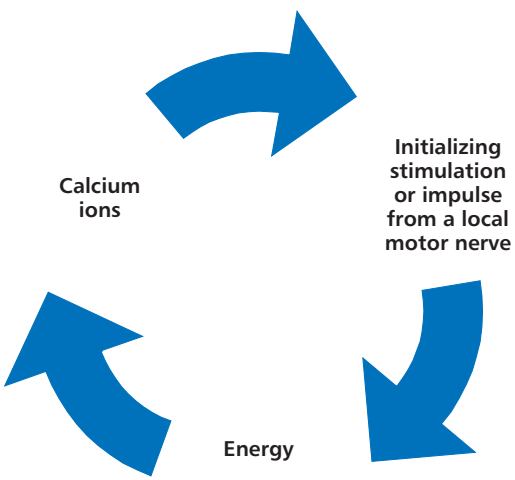


Figure 3.5: Flow chart of a nerve impulse which causes muscle contraction.

Pathophysiology of Trigger Points

The Integrated Trigger Point Hypothesis (ITPH)

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

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

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

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

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

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

Vicious Cycle of Energy Crisis

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

- Localized ischemia/hypoxia
- Increased metabolic needs
- Increased energy (required to sustain contraction)
- Failed reuptake of calcium ions into the sarcoplasmic reticulum
- Localized inflammation (to facilitate repair)
- Compression or watershed effect on local vessels
- Energy crisis
- Production of inflammatory agents (which sensitize local autonomic and nociceptive (pain) fibers)

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

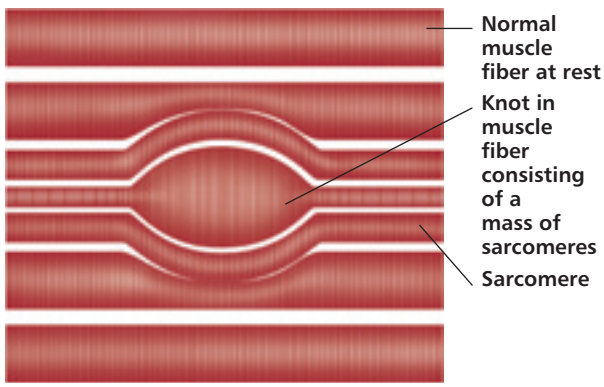


Figure 3.6: A trigger point showing 100 shortened sarcomeres without nerve stimulus and associated taut band.

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

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

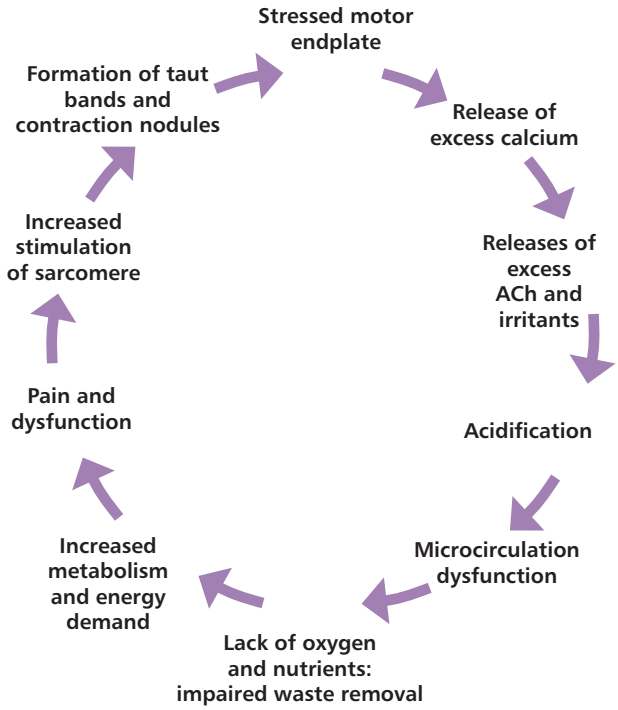


Figure 3.7: Cycle of misery: trigger point formation hypothesis – these individual links in the chain do not always occur in this order. (Sourced from: Starlanyl & Sharkey 2013.)

Other theories include *radiculopathic* theory and *polymodal* theory.

Radiculopathic Theory

Gunn (1997) and Quintner & Cohen (1994) have suggested an alternative mechanism for trigger point construction. This model suggests a causal relationship with intervertebral discopathy, nerve root impingement, and paraspinal muscle spasm. It is suggested that the irritation of these nerve roots (radicals) causes a compromise in neurovascular signals, distal muscle spasm, and trigger point pathogenesis.

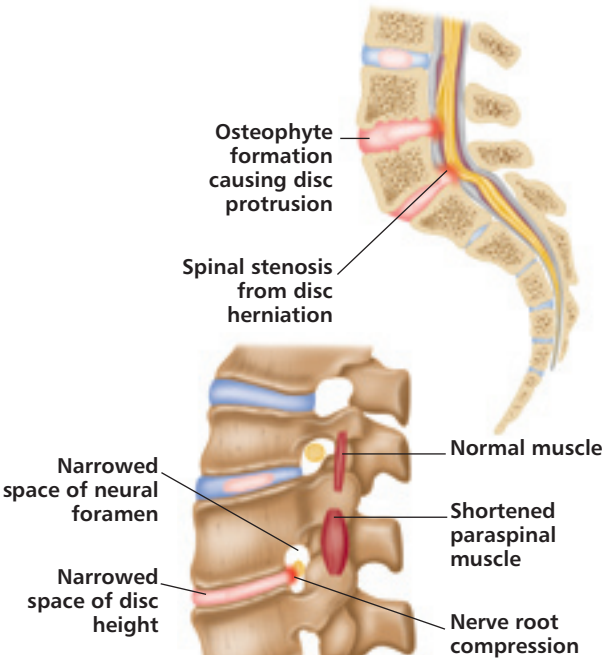


Figure 3.8: Nerve root irritation.

Polymodal Theory

Proposed by Kawakita et al. (2002), this alternative hypothesis describes trigger points themselves as "sensitized neural structures," called *polymodal receptors* (PMRs). It is suggested that these PMRs are a type of nociceptor, which responds to mechanical, thermal, and/or chemical stimuli. These PMR "sensory terminals" potentially exist in various tissues throughout the body as "free nerve endings." The theory is that the latent PMRs are "switched on" under certain physiological stimuli and become tender, morphing into the form we call *trigger points*. Although somewhat radical, this theory does explain a number of trigger point findings. Kawakita further suggests that PMRs may explain the link between acupuncture and trigger points.

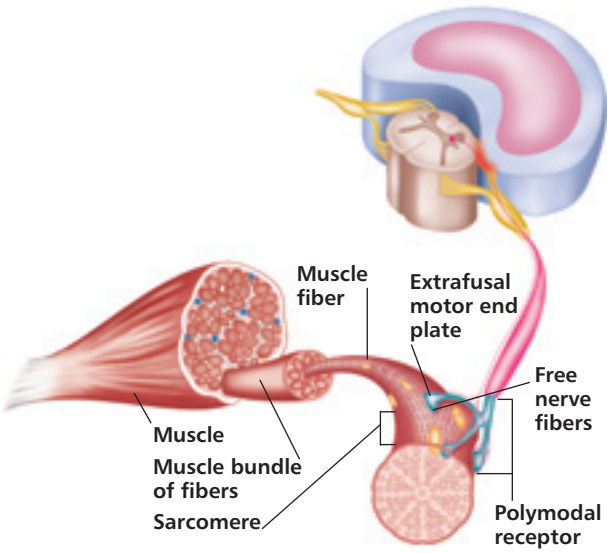


Figure 3.9: Nociceptive fiber accumulation (tree/dendrils).

Peripheral and Central Sensitization

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

Peripheral Sensitization

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

The suspected mechanism is:

- Substance P, released from nociceptor terminals, carries nociceptive signals for central processing, and alters local microcirculation and vessel permeability, leading to local edema, activating both mechanoreceptors and nociceptors, with subsequent increased tenderness and pain.
- Persistent activation with these algogenic substances leads to changes in nociceptor responsiveness both peripherally and centrally.

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

Central Sensitization (Spinal Hyperexcitability)

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

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

The suspected mechanism is:

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

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

Both peripheral and central sensitization can have serious unwanted effects: the advice therefore is to interfere with this process as soon as possible. The good news is that myofascial trigger point release and dry needling techniques have both been reliably demonstrated to reduce these effects.

Trigger Point Classification

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

Central (or Primary) Trigger Points

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

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

Satellite (or Secondary) Trigger Points

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

Attachment Trigger Points

As discussed in Chapter 1, myofascia is a continuum. It has been noted that the area where the tendon inserts into the bone (tendo-osseous junction) is often "exquisitely" tender (Simons et al. 1998; Davies 2004). This may well be the result of the existing forces travelling across these regions. It has also been suggested by the same authors that this may result from an associated chronic, active myofascial trigger point. This is because the tenderness has been demonstrated to reduce once the primary central trigger point has been treated; in such cases, the point is described as an *attachment* trigger point. Furthermore,

it has been suggested that if a chronic situation occurs where the primary and attachment trigger points remain untreated, "degenerative changes" within the joint may be precipitated and accelerated (Simons et al. 1998).

Ligamentous Trigger Points

There is some evidence that ligaments may develop trigger points but the relationships are not clear. The sacrotuberous and sacrospinous ligaments can refer pain down to the heel and the iliolumbar ligament can refer pain down to the groin and even into the testicles or vagina (Hacket 1991). Trigger points in the sacrotuberous ligaments can have profound effects on low back, lumbar erector spinae and pelvic pain (Starlanyl & Sharkey 2013) and may also be associated with backache, neck pain and even vocal dystonia (Lewit & Kolar, 1999). As well as stabilizing structures, ligaments have strong proprioceptive functions (Varga et al. 2008). Working on ligament trigger points therapeutically can be clinically useful as part of the neuroplastic model (explored later). Trigger points may manifest in the anterior longitudinal ligament (ALL) of the spine (e.g. after whiplash), which may result in neck instability (Stemper 2006). The fibular collateral ligament has a similar referred pain pattern to the vastus lateralis and trigger points in the ligamentum patellae are profoundly useful for treating knee pain syndromes.

Diffuse Trigger Points

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

Inactive (or Latent) Trigger Points

This applies to lumps and nodules that feel like trigger points. These can develop anywhere in the body and are often secondary. However, these trigger points are not painful, and do not elicit a referred pain pathway. The presence of inactive trigger points within muscles may lead to increased muscular *stiffness*. It has been suggested that these points are more common in those who live a sedentary lifestyle (Starlanyl & Copeland 2001). It is worth noting that these points may reactivate if the central or primary trigger point is (re)stimulated; reactivation may also occur following trauma and injury.

Active Trigger Points

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

Trigger Point Symptoms

Referred Pain Patterns

Pain is a complex symptom experienced differently and individually. However, *referred* pain is the defining symptom of a myofascial trigger point.

You may be used to the idea of referred pain of visceral origin: an example of this is heart pain. A myocardial infarct (heart attack) is often not experienced as crushing chest pain, but as pain in the left arm and hand, and in the left jaw. This type of pain is well documented, and known to originate from the embryological dermomyotome; in this case, the heart tissue, jaw tissue, and arm tissues all develop from the same dermomyotome.

Referred pain from a myofascial trigger point is somewhat different. It is a distinct and discrete pattern or map of pain. This map is consistent, and has no racial or gender differences, because stimulating an active trigger point generates the pain.

Patients describe referred pain in this map as having a *deep* and *aching* quality; movement may sometimes exacerbate symptoms, making the pain *sharper*. An example of this might be a headache. The patient often describes a pattern of pain, or ache, which can sometimes be aggravated and made sharper by moving the head and neck. The intensity of pain will vary according to the following factors (this list is not exhaustive):

- Location (attachment points are more sensitive)
- Degree of trigger point irritability
- Active or latent trigger points
- Primary or satellite trigger points
- Site of trigger point (some areas are more sensitive)
- Associated tissue damage
- Location/host tissue stiffness or flexibility
- Ageing
- Chronicity of trigger point

Physical Findings

The language for describing sensation is not highly sophisticated: unfortunately we have not yet evolved a suitable language to classify what we feel with our hands. With this in mind I will attempt to classify what trigger points feel like:

- Small nodules the size of a pinhead
- Pea-sized nodules
- Large lumps
- Several large lumps next to each other
- Tender spots embedded in taut bands of semi-hard muscle that feels like a cord
- Rope-like bands lying next to each other like partially cooked spaghetti
- Skin over a trigger point slightly warmer than the surrounding skin (due to increased metabolic/autonomic activity)

Examination

Examination may be performed standing, sitting, or lying down. The choice depends on both the area being examined and the type of muscle fiber suspected. You may want to examine a muscle *under load* if you suspect that this is an aggravating factor. For simplicity, *from this point forward* I will describe the examination of the pectoralis major and its trigger point(s).

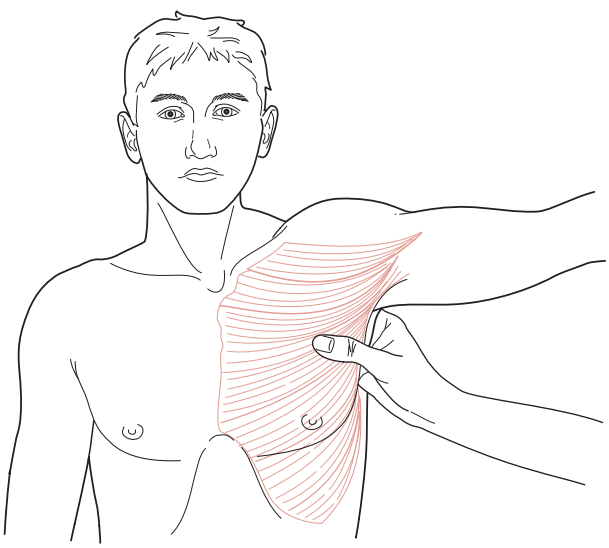


Figure 3.10: Pectoralis major examination.

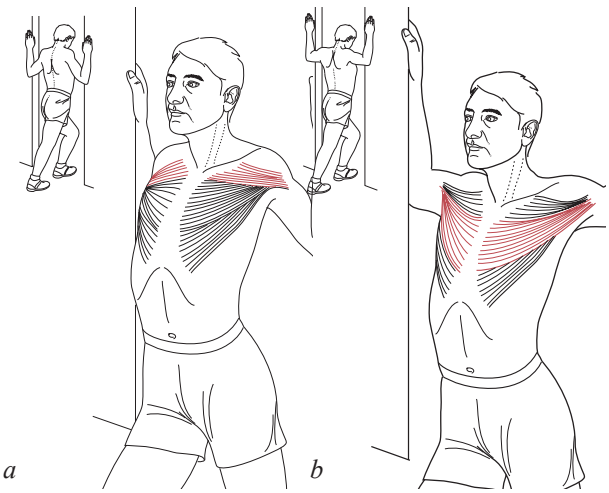
The main trigger points in the pectoralis major are to be found in the *clavicular* portion of the muscle. A *pincer-like* grip is the best way of examining for a trigger point in this region, while trigger points in the *parasternal* region of the muscle are best palpated with a *flat-handed* contact. The procedure is:

- Ask patient (sitting or standing) to abduct their arm 90 degrees to put the muscle into moderate tension.
- Palpate for a nodule or tight band.
- Feel for the *jump sign* or *twitch response*.
- Pressure should reproduce the symptoms experienced by the patient.
- Pressure should elicit a referred pain pattern.

Maintaining Factors

Several maintaining factors have been demonstrated for trigger points. The presence of one or several of the following factors may well present some difficulty in eliminating trigger points over the long term:

- Ageing
- Posture (including work)
- Obesity
- Anorexia
- Scar tissue (post-surgical)
- Sports, hobbies, habits
- Stress and strain patterns
- Metabolic disorders
- Disease or illness
- Sleep disturbance (including apnea)
- Iron deficiency
- Vitamin and mineral deficiency (folic acid, C, D, B1, B6, B12, iron, magnesium, and zinc)
- Congenital (bony) anomaly
- Type of muscle fiber
- Direction/orientation of muscle fiber
- Muscle shape/morphology (fusiform, etc.)
- Psychological factors—depression, anxiety, anger, and feelings of hopelessness
- Chronicity of trigger point



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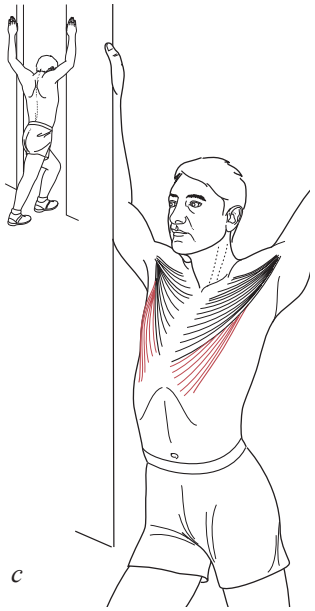

Figure 3.11: Stretching techniques for pectoralis major: (a) upper fibers, (b) middle fibers, and (c) lower fibers.

Advice to Patient

Once a therapeutic intervention has been performed, it is advisable to encourage the patient to get involved in managing their own symptoms. In this book, I have offered some specific advice under the heading "Advice to Patient." As a more general overview, you might want to include hints, tips, and advice using the following elements or components.

By way of an example, I will again use the pectoralis major muscle.

Strengthening

Muscles are more susceptible to damage, fatigue, and injury when they are weak. Weakness is often a contributory factor in the pathogenesis of myofascial trigger points: this is because the body overcompensates for the weakness and strains in the muscle, overloading and overstimulating the motor endplate.

One muscle should never be strengthened in isolation. If you decide to offer strengthening exercises, it is advisable to put them in context. An overall stretching program should be advised, perhaps utilizing a yoga-based regime.

Stretching

Stretching should be performed slowly, and *without* bouncing. Care must be taken to isolate the stretch to the specific muscle as far as possible. As a rule, stretches should be performed three times, slightly deepening the stretch with an out-breath each time. This sequence should be performed several times per day, for approximately 15–20 minutes. See Chapter 5.

Advice

Most of the advice you can offer is common sense. Explore and eliminate any chemical or nutritional deficit. Help patients to analyze different situations: for instance, "Look at your driving position" and "Look at your everyday work set-up." In the example of the pectoralis major muscle, you may ask the patient about their stress or anxiety levels (rib breathing mechanics). If the patient has large, heavy breasts, you may want to advise on a more appropriate bra or support. I have tried to offer some advice for each muscle in this book.

Posture

This may well have a crucial role in maintaining trigger point activity. Faulty sitting and/or standing postures are both a pathogenic and maintaining factor for trigger point activity. Advice and exercises for posture is often the key to unlocking both *central* and *satellite* points.

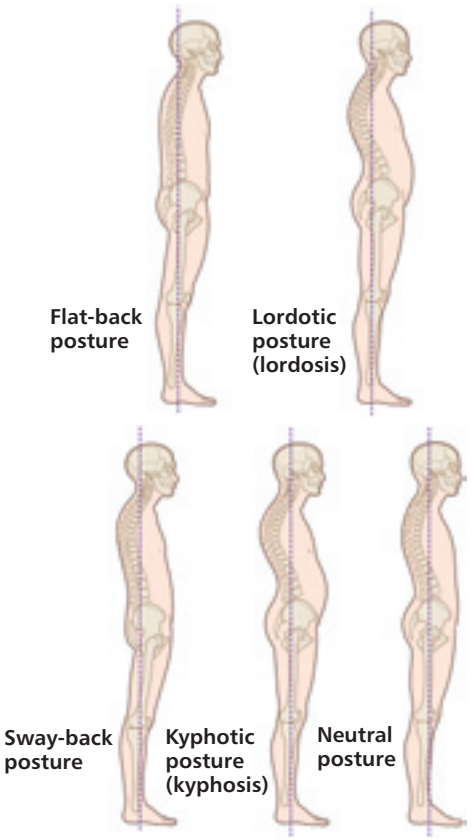

Figure 3.12: Posture.

Sleeping Posture

Patients frequently assume strange postures at night! This will sometimes be done to reduce the pain from either active or stiff latent trigger points. In such cases, patients often opt for a sleeping position that shortens the affected muscle: for example, they sleep with either the hands above the head (supraspinatus), or the arms folded over the chest (pectoralis major). In other cases, it may be that the sleeping position is a pathogenic or a maintaining factor.

Work Posture

Some patients may have manual or repetitive activities in the workplace; these may well have a role to play in trigger point pathogenesis or maintenance. Many patients spend their time at work sitting; Figure 3.11 illustrates an ideal sitting posture at work.

Figure 3.13: Ideal sitting posture at work.

Habitual Activity, Hobbies, and Sports

Similarly, it is important to ask the patient if they perform any repetitive or habitual activities apart from at work. Standing all day on one leg, for example, may well overload the TFL muscle. Sitting in a cross-legged position may affect a range of muscles, such as the hip flexors (iliopsoas), the buttock muscles (gluteals and piriformis), and the thigh muscles (quadriceps). Heavy smokers may develop trigger points in the shoulder (deltoid) and arm (biceps brachii) muscles.

Certain hobbies and sports may also lead to an increased incidence of trigger point pathogenesis. It is important to enquire carefully about such activities. What is their level of competence? Do they warm up, and cool down? How competitive are they? Is their level of activity realistic for their age? Posture? Body type? Physical health? You may want to explore these areas further. It is often useful to run through these activities and set the patient certain *activity goals* to achieve in between treatment sessions.

4 Therapeutic Technique Protocols

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Palpation

Palpation is part art and part science. Initially you should seek to relax the patient sufficiently to gain access to vulnerable and potentially painful treatment. Palpation comes through time and experience and a thorough knowledge of anatomy; it is the key to effective trigger point therapy. A thorough case history with thoughtful and directed questioning is essential, as is an engaging approach with the patient. It is important to talk to the patient: explaining procedures reduces the patient's anxiety levels, and allows participation in the treatment process. Involving the patient is a key step, as you rely on feedback to locate the exact center (heart) of the trigger point.

How do I know it is a trigger point?
You are looking for:

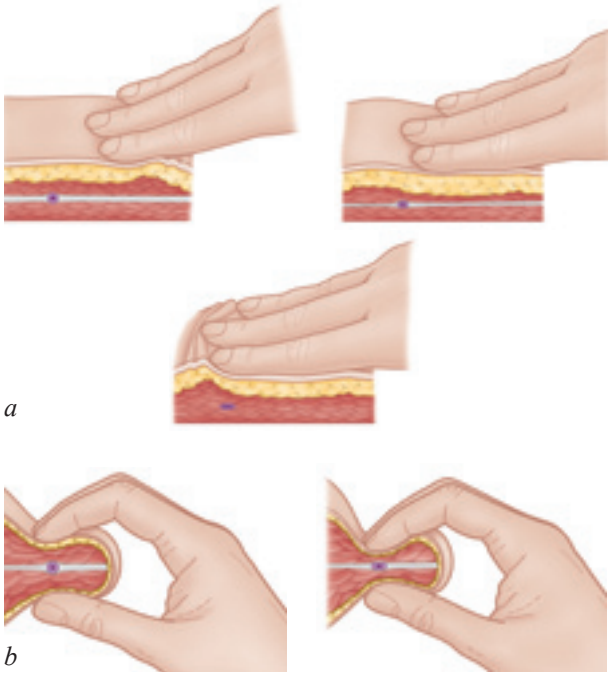
- Stiffness in the affected/host muscle
- Spot tenderness (exquisite pain)
- A palpable taut nodule or band
- Presence of referred pain
- Reproduction of the patient's symptoms (accurate)
- A warmer (or colder) region than the surrounding tissues
- A region more moist than the surrounding tissues
- An area that feels a little like sandpaper
- Possible loss of skin elasticity in the region of the trigger point

STAR/TART
Osteopathic medicine has long recognized the presence and clinical relevance of MTPs. In 1998 Dowling suggested the acronym STAR or TART for describing the somatic dysfunction associated with MTPs:

- Sensitivity
- Tissue texture change
- Asymmetry
- Range of motion reduced

What applicator should I use for palpation?

- Finger pads: remember to cut your fingernails (shorter is better).
- Flat finger: use the fingertips to slide around the patient's skin across muscle fibers.
- Pincer palpation: pinch the belly of the muscle between the thumb and the other fingers, rolling muscle fibers back and forth.
- Flat hand: useful in the abdominal region (viscera).
- Elbow: allows a stronger and shorter lever, which can be an advantage.



a

b

Figure 4.1: (a) Flat-finger palpation, (b) pincer palpation.

You may require instruments, such as a *dermometer* to accurately measure reduced skin resistance (needs calibration), and an *algometer* for measuring point tenderness and pain generated by pressure.

The "Jump and Twitch" Signs

This was first described in 1949 (Good 1950; Kraft et al. 1968). At first, you should discover that it is easier to locate a *central* trigger point. Firmly pressing it produces *exquisite pain* and often causes the patient to *jump* away. Using a quick *snapping* pincer palpation, or inserting a needle (stronger) into a trigger point, will often elicit a localized *twitch* response within the muscle (Simons et al. 1998). The twitch is due to the increased irritability of pain fibers (see Chapter 3). Pain from an active *central* trigger point commonly causes a specific referred pain map: this is a distinct map away from the point of pressure. In the therapeutic context, this pattern should, more often than not, reproduce the exact pain experienced by the patient.

You will find the maps of referred pain in Chapters 7–12.

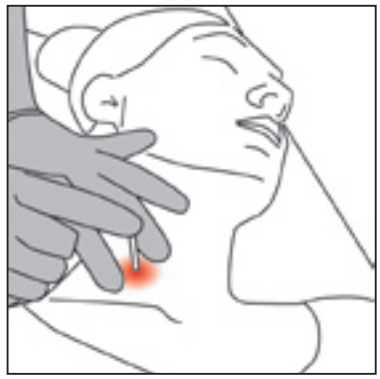


Figure 4.2: Therapeutic techniques.

Injections and Dry Needling

Dry needling is as effective as injection when it comes to relief of trigger point symptoms, but may result in a longer period of post-injection soreness. Table 4.1 offers some ideas as to when to consider injection or manual methods.

	Injection/ Needling	Manual Methods
Inexperienced therapist	**Not recommended**	**Not recommended**
Invasive	**Yes**	**No**
Quick response to treatment	**Yes**	**No**
Enables self-management	**No**	**Yes**
Point always accessible	**No**	**Yes**
Patient has low threshold	**Yes**	**No**
Patient is needle shy	**No**	**Yes**
Chronic setting	**Yes**	**Many require several sessions**

Table 4.1: When to use injection/needling versus manual methods.

There are three different approaches to needling (Simons et al. 1998):

1. Injection of a local anesthetic (alone)
2. Injection with botulinum toxin A
3. Dry needling with an acupuncture needle

A number of injections may be required although sometimes one is sufficient. Small amounts (<1 ml) of a non-myotoxic anesthetic are recommended. A local *twitch* response is a reliable indication of the correct position for needling, but EMG monitoring allows increased accuracy and specificity.

The following have been advocated for use when injecting:

- Procaine hydrochloride 1% solution
- Lidocaine hydrochloride (0.5%)
- Long-acting local anesthetics
- Isotonic saline
- Epinephrin
- Corticosteroid
- Botulinum toxin A

Dry Needling

Dry needling (Simons et al. 1998) is also known as *intramuscular stimulation* (IMS) and the "Gunn technique" after pioneering Canadian physician Dr. Chan Gunn. It borrows its needle technique from traditional Chinese acupuncture, but updates and enhances it with anatomy and neurophysiology.

In comparative studies, dry needling has been demonstrated to be as effective as injecting an anesthetic solution (procaine hydrochloride or lidocaine hydrochloride) in deactivating trigger points. Dry needling, however, causes localized soreness within 2–8 hours of injection; this soreness may be of a significantly greater intensity and/or duration than treatment with a wet injection.

For best practice, there are a number of protocols that should be followed before undertaking needling of trigger points. These include appropriate training, appropriate insurance cover, hygiene, knowledge of anatomical or mechanical risk for the points, and patient consent.

Complications of Dry Needling
(After Simons et al. 1998)

Pneumothorax
Pneumothorax is one of the most serious possible complications of dry needling. A pneumothorax occurs when air enters the pleural space between the visceral pleura and the parietal pleura. A pneumothorax is either partial or complete. The symptoms of a pneumothorax include sharp chest pain during breathing and coughing, shortness of breath, chest tightness, and sometimes coughing. The severity of symptoms can vary greatly between individuals. Other possible symptoms are fatigue, increased breathing rate (tachypnea), and tachycardia. Definitive diagnosis is made with a chest x-ray. Any suspicion of a pneumothorax needs an immediate referral to hospital or, at the very least, further investigation, especially if there are decreased breath sounds on auscultation.

Incidence of pneumothorax
While selection biased, one Japanese study found that 9% of 255 secondary pneumothoraces were the result of acupuncture (Nakamura et al. 1986). Another study reported two casualties (Gee 1984). While a unilateral pneumothorax may constitute a serious complication, bilateral pneumothoraces are life threatening. Therefore, it is advised against bilateral thoracic dry needling in one session.

Infections
Infections can result in other serious complications, such as erysipelas (a type of bacterial skin infection generally caused by group A streptococci) or viral infections. Extra caution is necessary in those patients with impaired immune systems (HIV/AIDS, advanced diabetes, or drug abuse). Entering the joint space or capsule with the needle should be avoided. Lymphedema should be avoided when needling, as there is a significantly increased risk of infection.

Injuries to Internal Organs, Nerves, Veins, and Arteries
Serious injuries as a result of dry needling are rare. However, superficial venous and arterial bleedings are fairly common: generally, they are harmless, but may result in hematoma. To prevent further bleeding you should apply direct compression, especially in those patients taking anticoagulants. Occasionally, you may hit a nerve, which will result in a sharp shooting pain down an extremity or into the groin. Hitting a nerve normally does not cause any damage. Areas that warrant extra caution are the suboccipital region between C0 and C2 (foramen magnum) and the sternum.

Autonomic Symptoms
Syncope, dizziness, vertigo, or sudden and excessive perspiration may occur occasionally but are usually transient. To reduce this risk, patients should be treated in supine, prone, or side-lying (lateral decubitus) positions.

Broken/Lost Needles
Rarely, the coiled metal handle of the needle may break off and the needle starts to get "sucked in" to the body cavity (it has happened to one of my colleagues). This may have potentially serious implications, so avoid putting the needle in all the way to the handle and always have a set of surgical forceps close by just in case. If multiple needles have been used in a particular session, you must account for all needles at the end of the session.

Cardiac Arrhythmia
Intramuscular electrotherapy stimulation (IMES) should not be used in a patient with a cardiac pacemaker or defibrillator.

Pregnancy
In traditional acupuncture, certain points are avoided during pregnancy. From an evidence-based perspective, there are no studies that support this.

Hygiene
Hygiene refers to the maintenance of *healthy* practices. With dry needling, relevant hygienic measures include cleanliness (such as keeping short and clean fingernails), hand washing (for at least 10 seconds with soap), disinfection, and protection (gloves to prevent infection). In some jurisdictions, local health codes may include specific guidelines for clinical hygiene.

Dry Needling Contraindications

General Contraindications
- Acute infections
- Blood thinning
- No consent from patient
- Fever
- Acute emergency
- Lymphedema
- Hematoma
- Osteosynthesis

Relative Contraindications
- Gravidity
- Children
- Psychiatric diseases
- Increased risk of infections, e.g. HIV or diabetes
- Contagious diseases

Complications
Once contraindications are taken into account and followed, there should not be any serious complications during professional application of trigger point needling. One frequent unpleasant reaction can be a more or less severe *hematoma*. Patients may also experience local muscle ache/soreness around the treated muscle area *up to four days* post-treatment. The patient should be informed about these possible reactions prior to treatment.

Consent
For best practice it is advisable to obtain informed consent whether verbal or written.

Equipment

Needles
Dry needling is performed with sterile acupuncture needles. There are various types of acupuncture needle available, particularly Chinese and Japanese ones. The Chinese needles have a conical shape, with the tip being thinner than the rest of the needle. Japanese needles have the same diameter. Whichever needle you choose, the recommendation is to use them only once, as they blunt fairly quickly; *never* share needles between patients. In the author's experience, most points can be reached using a 50 mm × 0.3 mm needle, but some deeper muscles may require a longer needle (up to 10 cm).

Disinfectants
Although the risk of infection is minimal with dry needling, it is recommended to disinfect the skin with antiseptic gels, either alcohol or isopropanol based, prior to needling. Remember to wait approximately 30 seconds before needling, to allow maximum antiseptic effect.

Needle Collector/Sharps Bin
It is recommended that used needles are discarded in a needle collector. Regulations may vary in different countries, states, and towns; you may well need to register with one or more local authorities.

Gloves
Wearing (latex) gloves is primarily for the clinician's protection, in case of an accidental needle stick: a glove provides a barrier to any body fluids and reduces the risk of infection. For best practice it is recommended you use a glove, at least for the palpating hand.

Swabs and Band-Aids
Occasionally there may be minor venous or arterial bleeding after removing the needle from the skin. If bleeding occurs, compression should be applied for up to 1 minute, followed by a Band-Aid as needed. Having alcohol swabs readily available also helps to stem the bleeding.

Something to Hug

Some authorities utilize a stuffed toy or a pillow for the patient to hug: they claim it reduces patient stress levels and increases compliance.

Dry Needling Technique

Dry needling is a relatively straightforward technique, although there are some variations. Needling can be performed with the patient sitting, prone, supine, or lateral decubitus. It is generally advised to needle with the patient in one of the lying positions: this reduces the side effect of syncope (fainting) and is generally more comfortable.

PROCEDURE

1. Locate the central trigger point; identify the muscle and fiber direction.
2. Be aware of any anomalies, nerves, nerve plexus, or blood vessels that may be in the area.
3. Choose holding grips depending on the muscle morphology and technique:
   a) Pincer grip: for trapezius, pectoralis major, etc.
   b) Flat-finger stretch: stretch the skin grip by sliding the thumb and forefinger of the palpating hand away from each other on either side of the needle (in the direction of the muscle fibers). This also helps you palpate and assess the twitch response.
4. Insert the needle perpendicular to the fiber until you see the twitch response (always check the anatomy).
   - Techniques then vary from immediately removing the needle to leaving the needle in the point for up to 26 minutes. There is some evidence that leaving the needle in situ for a few minutes diminishes small c-fiber activity and thus lessens the post-treatment soreness.
   - Manipulating the needle (once inserted) in different directions may appear to needle different areas of the same trigger point; however, studies have demonstrated that, once inside, the needle forms a channel or groove and will move in and out of this same groove.
5. Some authorities (Gerwin et al.) talk of "clusters" of points, and propose a technique of moving gently and rhythmically in and out of one point, removing the needle from the muscle, and then needling another point within the cluster.
6. Twiddling/twisting the needle is not recommended in general, as it has the effect of "winding up" the delicate muscle spindle and damaging it.

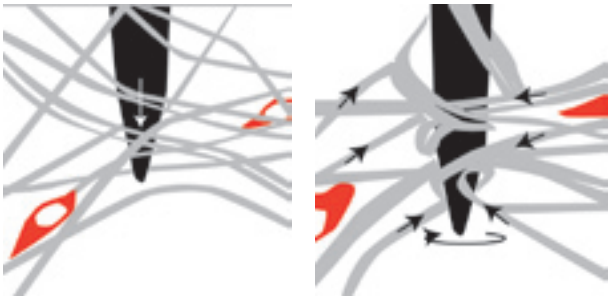
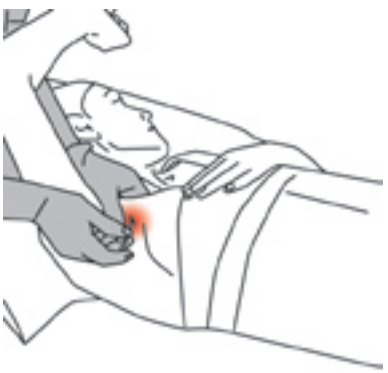


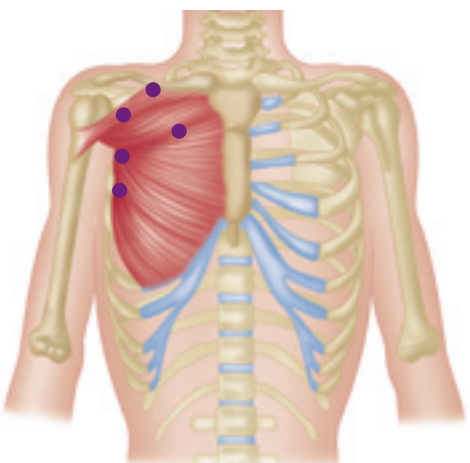
Figure 4.3: The "winding-up" effect of twiddling a needle.

PROCEDURE

1. Does not require the same precise localization of trigger points as for an injection
2. Two to three sweeps of spray are applied to the affected/host muscle whilst extending the muscle gently to its full stretch length;
3. The spray is aimed out of the inverted bottle nozzle at 30 degrees to the skin in a fine jet over a distance of about 30-50 cm (do not aim at a single spot).

Figure 4.4: Dry needling protocol.





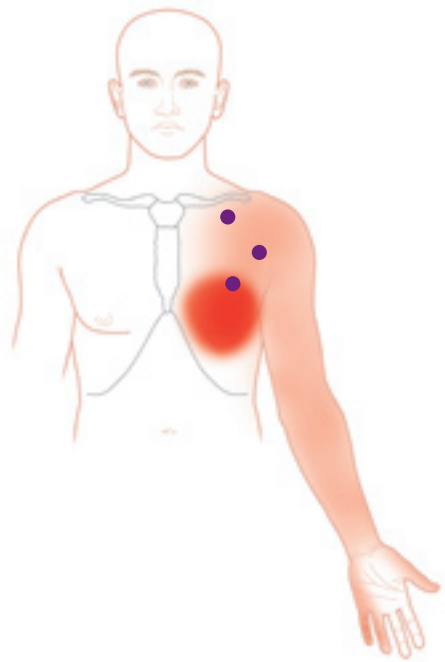


Figure 4.5: Pectoralis muscle needling.

Modifications for Dry Needling

The following modifications are useful in a chronic setting where there may be clusters of trigger points:

- Myofascial plane activation
- Periosteum pecking (for attachment trigger points)
- Two-needle techniques
- Nerve root activation needling technique
- Basal ganglia techniques

Myogenic Nerve Compression Syndromes

When a trigger point develops, the host muscle becomes shorter, thicker, and less efficient. Because muscles are made up of 75% water, and the fact that water does not compress well, they become functionally swollen, which often leads to a taut band that can have further pressure effects on local tissues. This is highly relevant in certain parts of the body, such as the thoracic outlet, where there may already be only a small space for the neurovascular bundle. The following conditions have been shown not only to have a trigger point component, but also to respond well to wet and dry needling interventions:

- Greater occipital neuralgia
  - Semispinalis capitis
  - Upper trapezius
  - Multifidus
- Thoracic outlet/inlet syndrome
  - Anterior and middle scalene
  - Supraspinatus
  - Infraspinatus
  - Levator scapulae
  - Latissimus dorsi
  - Pectoralis major
  - Subscapularis (wrist pain)
- Forearm—ulnar, radial, and median nerve entrapments by
  - Pronator teres
  - Supinator
  - Extensor carpi ulnaris
  - Flexor digitorum
  - Brachioradialis
- Meralgia paresthetica
  - Sartorius (mainly)
  - TFL (slightly)
- Sciatica (pseudo)
  - Piriformis

Prolotherapy

Originally proposed by US Dr. George S Hackett in the 1950s, prolotherapy is also known as "proliferation therapy," or "proliferative injection therapy". It involves injecting an irritant solution in the region of trigger points, tendons and/or ligaments and is claimed to be effective for 'strengthening weakened connective tissue' and alleviating chronic or recalcitrant musculoskeletal pain. Prolotherapy is usually performed by medical doctors; its primary therapeutic action is to stabilize joints and the soft tissues around them—the effects on trigger points that may have developed as a result of joint dysfunction can be profound.

The following compounds are most commonly used:

- hyperosmolar dextrose (a sugar) is the solution used;
- glycerine
- lidocaine (a commonly used local anaesthetic),
- phenol, and
- sodium morrhuate, a derivative of cod liver oil extract

The injection is administered at joints or at tendons where they connect to bone and/or into trigger points.

Prolotherapy treatment sessions are generally given every 2 to 6 weeks for several months in a series ranging from 3 to 6 or more treatments.

Spray and Stretch

Hans Kraus (1941), who was the first to describe the technique of spray and stretch using *ethyl chloride spray,* used the technique for treating aches and sprains in wrestlers. Since then, coolant techniques have been developed to treat almost all trigger points. They appear to have a myorelaxant effect within a few seconds of application.

These techniques are the "single most effective non-invasive methods" for deactivating trigger points. (Simons et al. 1998). Ethyl chloride spray is highly flammable and toxic, and is considerably colder than is necessary. It is volatile and has accidentally killed several patients and doctors. Vapocoolents, such as a Fluori-Methane spray, are a safer alternative, although being a fluorocarbon, it may affect the ozone layer. The recommended product is Gebauer's Spray and Stretch, as it is nontoxic and nonflammable. Machine-based products, such as Cryonics CRYO+, are also gaining popularity; they are far more predictable and controllable.

The physiological basis for these products is a type of "thermal shock." Research has indicated that these techniques work partly on the hypothalamus. The rapid skin cooling challenges the autonomic reflex pathways and forces a local homeostatic response, which may have a therapeutic action. A skin cooling of the skin by 2–5 degrees is enough to cause this reaction. The following beneficial effects have been suggested:

- Analgesic
- Anti-inflammatory
- Vasomotor
- Myorelaxant

Spray and Stretch Contraindications

- Allergy to cold
- Raynaud's syndrome
- Disorders of skin sensitivity
- Cryoglobulinaemia

Spray and Stretch Technique

The basic spray and stretch technique is quite straightforward, as it *does not require* the same precise localization of trigger points as for needling or injection; instead you need only locate and identify the affected/host muscle to release its fibers. However, it is advisable to locate the trigger point by palpation, since this reassures the patient as to the efficacy.

PROCEDURE

1. Does not require the same precise localization of trigger points as for an injection
2. Two to three sweeps of spray are applied to the affected/host muscle whilst extending the muscle gently to its full stretch length;
3. The spray is aimed out of the inverted bottle nozzle at 30 degrees to the skin in a fine jet over a distance of about 30-50 cm (do not aim at a single spot).

When to Use Spray and Stretch

- Young children
- Needle-shy patient
- Immediately after trigger point injection
- Post-hemiplegic—stroke rehabilitation
- Immediately following major trauma, e.g. fracture or dislocation
- After whiplash injury
- In a patient with myofascial trigger points and hyperuricemia (excess uric acid)
- Chronic or inhibition-resistant trigger points
- Attachment trigger points
- After sprains and burns

Tips

- Locating the central trigger point which causes a precise referred pain pattern is recommended, as it gives the patient a rationale for accepting treatment.
- Make sure the patient has recently eaten, as hypoglycemia aggravates trigger points.
- Provide a warmish surgery/room.
- Use a blanket to cover the body and areas not being cooled, as muscle warmth is more conducive to muscle relaxation.
- Remember to cover the eyes where appropriate.
- Do not aim at a single spot, as this can burn or cause urticaria.
- Do not force a stretch.
- If the patient is apprehensive, ask them to focus on their breathing.
- Test the range of motion before and after the spray and stretch technique.
- Make sure that the muscle to be treated is fully relaxed and support it where possible—treatment can be performed sitting, side lying, prone, or supine.
- To achieve a full stretch, you should anchor one side of the muscle, and move the other (passively).

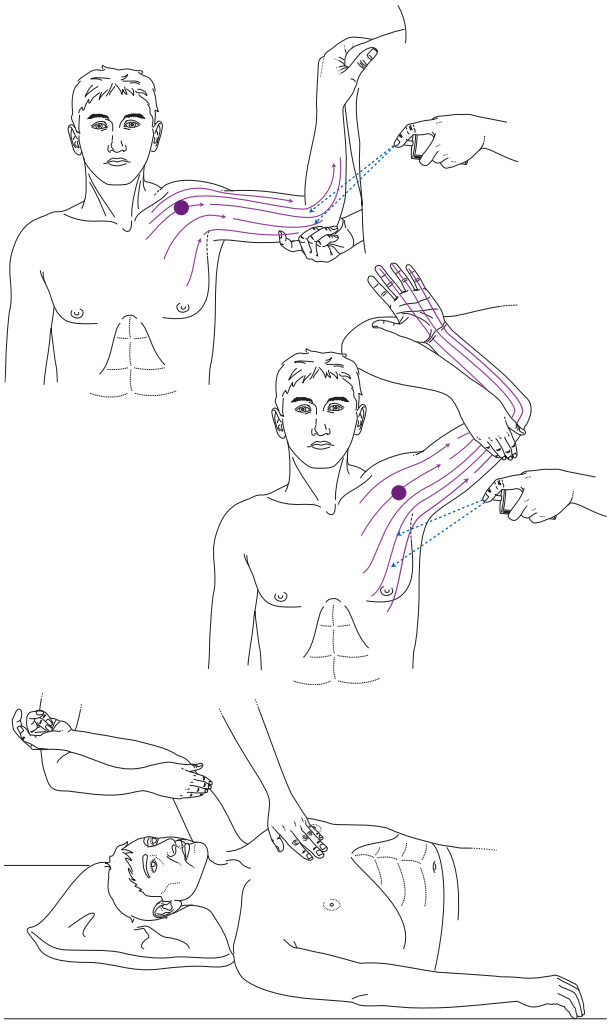


Figure 4.6: Spray and stretch technique for the pectoralis major.

Hands-On Therapy Trigger Point Release (TPR) Protocols

Niel-Asher Technique (NAT)

NAT is an advanced trigger point technique: it was developed in 1999 by this author—osteopath Simeon Niel-Asher. It was originally developed to treat complex shoulder problems, such as rotator cuff dysfunction and frozen shoulder syndrome/adhesive capsulitis.

NAT involves specific repeatable and deliberate neuromuscular and trigger point massage and compression algorithms. In NAT the idea is that the stimulation of a trigger point can be equally viewed as a neural input, promoting changes in feedback to and from the brain. With NAT these inputs are administered in a deliberate and repeatable sequence to trigger points within agonists and antagonists, and to super trigger points (see p.70) around the joints. The objective is to reduce the number of these neural inputs to a structured minimum. NAT deliberately utilizes automatic reflexes, including:

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

NAT is fast, effective, and evidence based, having successfully undergone randomized placebo controlled trials at Addenbrooke's Hospital, Cambridge, UK (Weis et al. 2003). The technique is now regularly used by thousands of therapists worldwide to treat a range of common musculoskeletal conditions (see Chapter 6). For more information please visit www.nielasher.com.

Neuromuscular Technique (NMT)

In Europe the neuromuscular technique was developed and adapted in the 1930s by pioneering osteopath Stanley Leif. In the US the technique was developed by chiropractor Dr. Raymond Nimmo in the 1920s, where it became known as the *receptor tonus method*. NMT offers a range of techniques that modulate or deactivate trigger points both locally and through reflex activity.

NMT addresses trigger point therapy as part of a multidimensional approach. Pain relief is addressed by treating the following six physiological factors (Chaitow & Delany 2000):

- Ischemia
- Trigger points
- Nerve compression/entrapment
- Postural distortion (biomechanics)
- Nutritional wellbeing
- Emotional wellbeing

Myotherapy (MT)

Myotherapy was developed in the US by exercise teacher Bonnie Pruddent. She created a system of manual therapy based on the work and research of Travell and Simons, Gunn, and Nimmo.

MT is defined as the comprehensive assessment, treatment, and management of neuromusculoskeletal disorders and conditions caused by improper biomechanical functioning.

Myotherapists take into account many aspects of health and wellness when treating patients—this includes the physical, psychological, and occupational aspects of the individual. MT can be used either as a single mode of treatment, or in conjunction with treatment provided by both medical and other allied-health practitioners.

Hands-On Techniques: The Details

(Inhibition) Compression Technique

This is the best technique to use on an active central trigger point. It involves first locating the trigger point that causes a specific referred pain pattern (preferably reproducing the patient's symptoms) and then applying a direct inhibitory pressure to the point. Although referred to as *ischemic*, it is now commonly accepted that you do not need to compress the trigger point to the point of ischemia, although you may wish to in certain situations. This technique is very effective, but is best used in conjunction with other stretch and release techniques. I have included a protocol that incorporates the current approach.

Personally, I find that it is easier to lean on the trigger point, and not to push or press on it. This literally means that once the point has been found, you lean your weight through the applicator rather than pushing or pressing it. This is much more comfortable for you and the patient.

PROCEDURE

1. Identify the trigger point.
2. Place the patient in a comfortable position, where the affected/host muscle can undergo full stretch.
3. Apply gentle and gradually increasing pressure to the trigger point, while lengthening the affected/host muscle until you hit a palpable barrier. This should be experienced by the patient as discomfort and not as pain.
4. Apply sustained pressure until you feel the trigger point soften. This can take from a few seconds to several minutes.
5. Repeat, increasing the pressure on the trigger point until you meet the next barrier, and so on.
6. To achieve a better result, you can try to change the direction of pressure during these repetitions.

Tip

Do not come away too quickly, as this can irritate the trigger point and make the symptoms worse. Feel, as well as think!

Deep Stroking Massage

Being more specific, as it is more directed than the spray and stretch technique, deep stroking massage is also considered by most authorities to be the safest and most effective hands-on method for treatment (Simons et al. 1998).

PROCEDURE

1. Place the patient in a comfortable position, where the affected/host muscle can undergo full stretch.
2. Lubricate the skin if necessary.
3. Identify and locate the trigger point or taut band.
4. Position your thumb/applicator just beyond the taut band, and reinforce with your other hand.
5. Apply sustained pressure until you feel the trigger point soften, and continue stroking in the same direction toward the attachment of the taut band. This should be experienced by the patient as discomfort and not as pain.
6. Repeat this stroking in the opposite direction.

Tip

Do not stroke too quickly or deeply, as this can irritate the trigger point and rupture the sarcomere, making the symptoms worse.

A modification to deep stroking massage is *strumming*, where the applicator is dragged perpendicularly across the taut band of muscle fibers. This is performed slowly and rhythmically using a light contact and pausing on the trigger point when it is palpated. It is especially useful for treating the medial pterygoid and masseter muscles.

Manual Lymphatic Drainage (MLD)

There is mounting anecdotal evidence that MLD techniques are very effective at releasing trigger points. This technique requires a more subtle approach and also a good knowledge of the morphology of the lymphatic system. Very light pressure is used to encourage lymph flow, as opposed to forcing blood through the system. MLD is especially useful for releasing trigger points in the scalenes, anterior cervical musculature, and clavipectoral fascia in the acute phase of whiplash injury.

Trigger point activity has been demonstrated to attenuate lymphatic flow in the following ways (Simons et al. 1998):

- Scalene trigger points (especially anterior) cause tension that interferes with drainage into the thoracic duct.

- This is compounded by restrictions in the 1st rib mechanics (often secondary to trigger points in the middle and posterior scalenes).
- The peristaltic movement of lymph is disrupted by trigger points in the scalenes.
- Lymph flow in the arms and breast may be disrupted by trigger points in the subscapularis, teres major, and latissimus dorsi.
- Lymph flow to the breast may be further disrupted by trigger points in the anterior axillary fold (especially in the pectoralis minor). This commonly results from a protracted, chronically round-shouldered posture (Zinc 1981).

It is suggested that MLD should be employed either before deeper work or after it, to help remove excessive toxins and/or waste products from the tissues (Chaitow & DeLany 2000).

PROCEDURE (Harris & Piller (2004))

1. Administer light, rhythmic, alternating pressure with each stroke.
2. Perform skin stretching and torque both longitudinally and diagonally.
3. Apply pressure and stretch in the direction of the desired fluid flow (not always in the direction of lymph flow).
4. Use light pressure over spongy, edematous areas and slightly firmer pressure over fibrotic tissue.
5. Do not exceed a pressure of 32 mmHg.

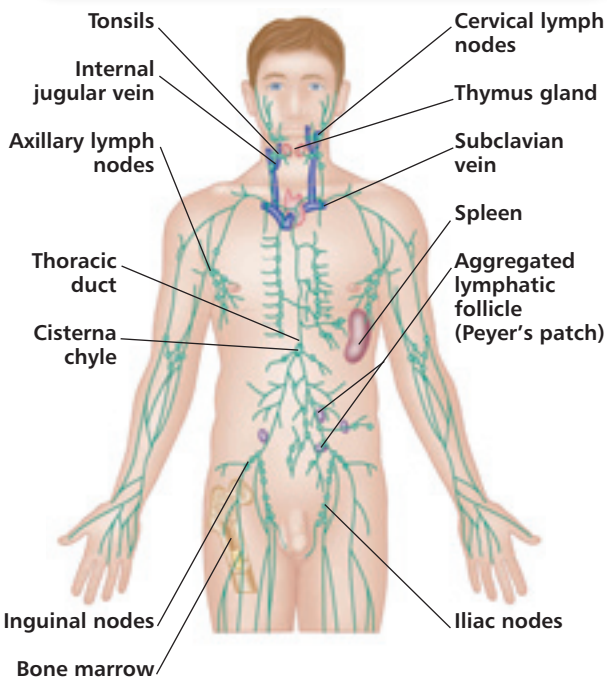


Figure 4.7: The lymphatic system.

Stretch and Release Techniques

These methods directly involve the patient, requiring them to actively contract the affected/host muscle and then to relax it. This sequence forms the basis for several extremely effective inhibitory techniques:

- Post-isometric relaxation (PIR)
- Reciprocal inhibition (RI)
- Contract and relax/hold and relax (CRHR)
- Contract relax/antagonist contract (CRAC)
- Muscle energy technique (MET)
- Positional release technique (PRT)
- Taping

These techniques are effective if you consider the concept of an overstimulated motor endplate, as already discussed in Chapter 2. Utilizing contraction and relaxation while fixing through the trigger point may well "normalize" the sarcomere length. This sets in place a cascade, releasing the affected actin and myosin, and reducing the energy crisis. In this case, taking up the slack while inhibiting the trigger point (as in a PRT) may be particularly useful.

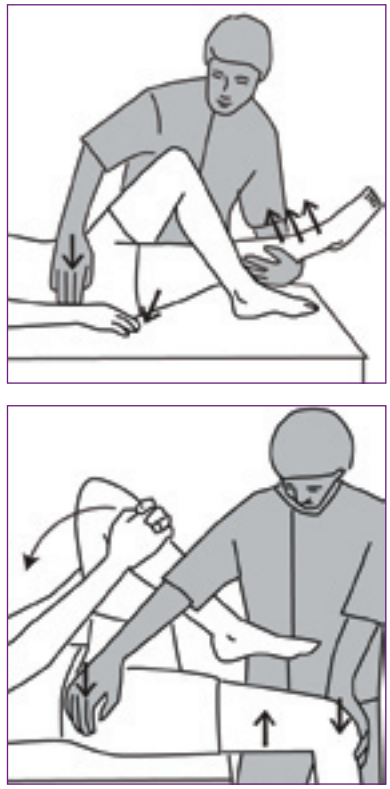


Figure 4.8: Stretch and release techniques.

Post-Isometric Relaxation (PIR) Technique

This technique was introduced by Karel Lewit (1981). The proposed complete technique incorporates the use of coordinated eye and respiratory movements (reflex augmentation).

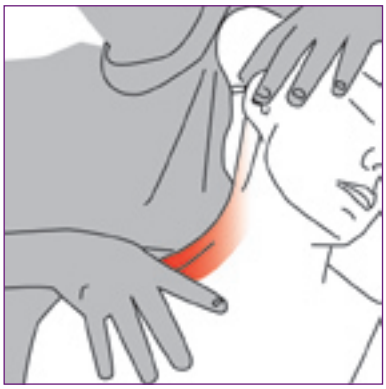


Figure 4.9: Post-isometric relaxation technique for upper trapezius.

PROCEDURE

1. Identify the trigger point.
2. Position the patient in a comfortable position, where the affected/host muscle can undergo full stretch.
3. Using 10–25% of their power, ask the patient to contract the affected/host muscle at its maximal pain-free length, while applying isometric resistance for 3–10 seconds; stabilize the body part to prevent muscle shortening.
4. Ask the patient to relax the muscle or "let it go."
5. During this relaxation phase, gently lengthen the muscle by taking up the slack to the point of resistance (passive)—note any changes in length.
6. Repeat several times (usually three).

Reciprocal Inhibition (RI) Technique

This is an indirect technique relying on the agonist–antagonist neurological reflex. It is often used to augment other techniques, adding the "finishing touch."

PROCEDURE

1. Identify the affected/host muscle and take it into relaxation.
2. Ask the patient to contract the antagonist muscle against 35–45% isometric resistance.
3. Manual therapy of the antagonist will have a reciprocal inhibition effect.

Contract and Relax/Hold and Relax (CRHR) Technique

Originally taught by osteopaths Knott and Voss (1968), the CRHR technique was devised to increase the passive range of motion of markedly stiff joints. The principles behind the technique have a direct relevance to myofascial trigger point therapy, because, as we have discussed, muscle tightness is often a sign of *active* or *latent* trigger points.

PROCEDURE

1. Identify the trigger point.
2. Place the patient in a comfortable position, where the affected/host muscle can undergo full stretch.
3. Take the stiff joint to a comfortable position near the endpoint, and ask the patient to actively contract the affected/host muscle.
4. Gently resist this voluntary contraction.
5. Allow relaxation.
6. During the relaxation phase, passively stretch the joint to a new (increased) endpoint.

Contract Relax/Antagonist Contract (CRAC) Technique

CRAC is a combination technique blending the neuromodulatory inputs of post-isometric relaxation and reciprocal inhibition. It is an excellent technique for moderate to chronic restricted joint conditions and recalcitrant trigger points. Readily used especially in painful, awkward regions.

PROCEDURE

1. Find the joint/soft tissue restriction or 'biting point'.
2. Contract agonist. Relax (agonist).
3. Contract antagonist. Stretch agonist.
4. Hold stretch for 15–30 seconds.
5. Repeat 3 times.

Muscle Energy Technique (MET)

METs are a broad classification of manual therapy methods directed at improving musculoskeletal function and reducing pain.

Historically, the concept emerged as a form of osteopathic manipulative diagnosis and treatment in which the patient's muscles are actively used on request, from a precisely controlled position, in a specific direction, and against a distinctly executed counterforce from the physician. It was first described in 1948 by Fred Mitchell, Sr., D.O. METs are used to treat somatic dysfunction, especially decreased range of motion, muscular hypertonicity, and pain.

These techniques are most appropriate for the following injury patterns:

- Decreased range of motion secondary to muscular spasticity
- Rigidity
- Hypertonicity or hypotonicity

Hypertonicity often follows overuse and can result in altered joint position, increased irritability, and decreased elasticity. This injury pattern is often accompanied by a nonspecific muscle ache in the area of injury.

In the case of an interneuronal injury, when dysfunction occurs at one joint or segment the related agonist muscles are also affected. If uncorrected, the antagonist muscles eventually become involved as well, leading to dysfunction of both muscle groups. This presents as decreased range of motion with pain and/or tenderness in the area.

The muscle energy technique is a *direct* and *active* method, meaning that it engages a restrictive barrier and requires the patient's participation for maximal effect. As the patient performs an isometric contraction, the following physiologic changes occur:

- Golgi tendon organ activation results in direct inhibition of agonist muscles.
- A reflexive reciprocal inhibition occurs in the antagonist muscles.
- As the patient relaxes, agonist and antagonist muscles remain inhibited, allowing the joint to be moved further into the restricted range of motion.

Despite the many claims made regarding the efficacy of these techniques, there are only two peer-reviewed studies that have shown that METs can significantly decrease disability and improve functionality in patients with disorders such as low back pain.

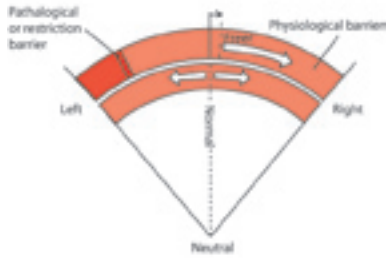


Figure 4.10: When you assess a tight muscle or a 'stuck' joint, you come to a barrier of restriction, which can be either physiological or protective. This diagram explores the end feel of the barriers.

METs can be applied to most areas of the body. Each technique requires eight essential steps:

1. Perform and obtain an accurate structural diagnosis.
2. Engage the restrictive barrier in as many planes as possible.
3. Physician and patient engage in an unyielding counterforce where the patient's force matches the physician's force.
4. The patient's isometric contraction has the correct amount of force, the correct direction of effort (away from the restrictive barrier), and the correct duration (3–5 seconds).
5. Complete relaxation occurs after the muscular effort.
6. The patient is repositioned into the new restrictive barrier in as many planes as possible.
7. Steps 3–6 are repeated approximately 3–5 times or until no further improvement in range of motion is observed.

8. The structural diagnosis is repeated to evaluate if the dysfunction has resolved or improved.

These techniques can be subdivided into three distinct approaches (Kuchera & Kuchera 1994).

Isometric Contraction Technique

PROCEDURE

1. Hold or fix through the trigger point of the affected/host muscle.
2. Ask the patient to actively contract the muscle with minimal resistance.
3. Hold until a softening is palpated in the trigger point.
4. Actively and passively stretch the muscle.

Isotonic Contraction Technique

PROCEDURE

1. Position the muscle in a mid-range comfortable position.
2. Ask the patient to actively contract the muscle for 7–15 seconds at about 35–45%, while you resist against it with less resistance, therefore allowing a concentric contraction.
3. Allow 5 seconds of rest before, on exhalation, taking the muscle to a new restriction barrier for 30 seconds.
4. Repeat 3 times.

Isolytic Contraction Technique

PROCEDURE

1. Position the muscle at the restriction barrier.
2. Ask the patient to actively contract the muscle for 2–4 seconds at about 10–25%, while you resist.
3. Overcome this resistance, actively pushing against the muscle into eccentric contraction towards the physiological barrier for 15–30 seconds.
4. Repeat 3–5 times.

Positional Release Technique (PRT)

PRTs were pioneered by osteopaths Harold Hoover, D.O., Charles H. Bowles, D.O., and William L. Johnston, D.O. PRTs are a range of methods that invite change, rather than force it: they attempt to find a way of offering an "opportunity for change." These techniques have a lot of crossover with other techniques.

Three of the main positional release approaches are:

- Strain–counterstrain technique
- Functional technique
- Facilitated positional release

Strain–Counterstrain (SCS) Technique

Created in the early 1960s by Lawrence Jones, D.O., the SCS technique uses very specific treatment positions held for 90 seconds (but which may be held for up to 3 minutes in neurological patients). During the procedure, the involved tissue is "slackened," causing a relaxation of the "spasm," which in turn allows local areas of inflammation trapped within the painful tissue to dissipate. Following this "release," there is an immediate reduction of pain and tension in the involved tissue.

One controlled study (Lewis et al. 2010), investigating the immediate and short-term effects of strain–counterstrain intervention on the pressure pain threshold at tender points in the low back region, suggested that strain–counterstrain treatment does elicit an immediate quantifiable reduction in tenderness at tender points. However, some of this reduction was found to be due to the manual-contact component of the treatment. The addition of the strain–counterstrain procedure to an exercise protocol was not more effective than exercise alone in reducing levels of low back pain and disability (Lewis et al. 2011).

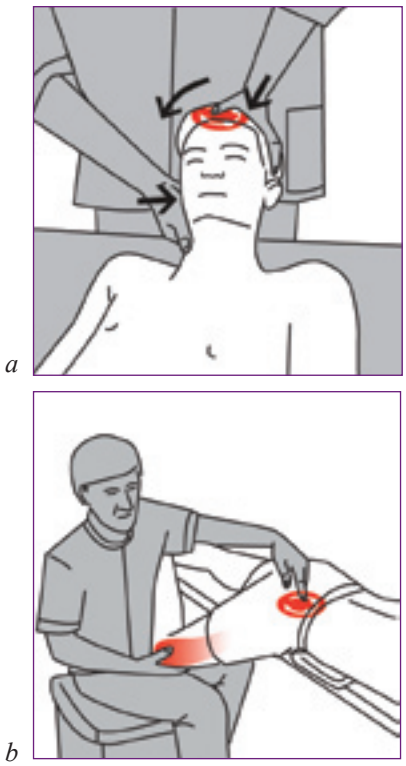


Figure 4.11: (a) Positional release technique, (b) facilitated positional release technique.

Functional and Facilitated Positional Release Techniques

PROCEDURE

1. With one hand monitoring (listening) and the other acting to introduce movement, take the tissues to a position of maximal ease in all available directions of motion—a point of dynamic neutral—in which one position of ease has been "stacked" on another.
2. Reassess for ease in different directions of movement, commencing at the point of ease revealed by the previous assessment. Repeat these steps if necessary.
3. Hold the position of dynamic neutral until a sense of warmth or pulsation or greater ease is noted (a minimum of 90 seconds is suggested)
4. Repeat the whole sequence at least once more (variations in the positions of ease will be evident as a consequence of changes resulting from the previous "treatment").

Taping

The use of taping has become increasingly prolific as an adjunct to manual therapy. Trigger point therapy offers a number of excellent opportunities to exploit taping. The *Journal of Sports Medicine* (Feb 2013) reviewed the evidence from 10 research papers about the effectiveness of kinesio tape in preventing sports injuries:

- No clinically important results were found to support the tape's use for pain relief.
- There were inconsistent range-of-motion results.
- Seven outcomes relating to strength were beneficial.
- The tape had some substantial effects on muscle activity, but it was not clear whether these changes were beneficial or harmful.
- The review concluded that there was little quality evidence to support the use of kinesio tape over other types of elastic taping to manage or prevent sports injuries.

Taping techniques are often performed after a manipulative or needling session, to "unload" the treated muscle or to improve lymphatic drainage. While kinesio tapes are becoming more widely available, any tape (zinc oxide etc.) can be used. There is some evidence to support the use of tape in aiding the reduction of pain (Thelen et al. 2008).

Unloading Taping Technique

PROCEDURE

1. Tape the affected/host muscle into a position of ease.
2. Leave the tape for a few hours to several days.
3. The taping may reduce nociceptor activity.
4. The taping may also improve oxygenation and reduce ischemia-related issues.

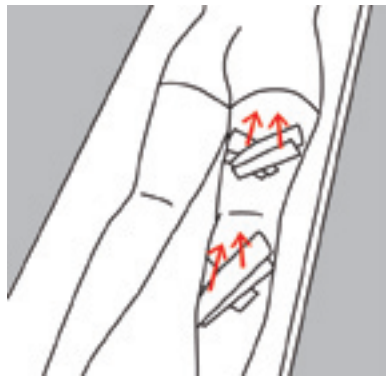


Figure 4.12: Unloading taping technique.

Star Technique

PROCEDURE

1. Precut six thin strips of tape.
2. Strips may be up to 15 cm in length, depending on the muscle.
3. Place the tapes in a star pattern, with the center located above the trigger point.
4. The tape should have minimal stretch at each end but 30% stretch in the middle.
5. Small slits may be cut in the tape to facilitate drainage, bruising, or hematoma.

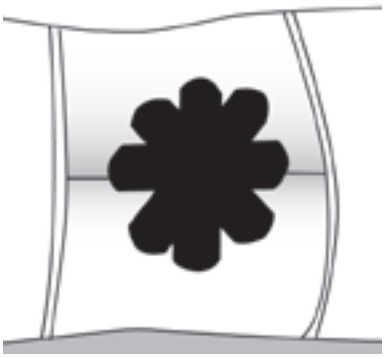


Figure 4.13: Star technique.

Multiple Trigger Point Taping

This can be a very useful post-treatment over a large area. Strips of tape are placed over the treatment area and/or the area of maximal trigger point activity. The tape can be left for up to three days. Several studies have shown that this increases the speed of bruising and soft tissue swelling, and may also help to deactivate large areas of trigger points.

Figure 4.14: Multiple trigger point taping.

Practitioners' Frequently Asked Questions (FAQs)

How much pressure do I use?

This is something that comes with experience, but as a rule of thumb the more painful the tissue, the slower and deeper the pressure. In all cases, the key words are "work slowly" and "thoroughly."

Another factor that determines the amount of pressure that you should apply is the muscle type (red/white fiber) and morphology of the patient. This will affect the depth of treatment. If the patient is stocky, you should expect to have to work quite vigorously, especially into the posterior capsule. If they are slight, you will not need to use as much force to cause a change in the tissues.

Which direction should the pressure/force be applied?

For those of you who have worked with trigger points before, this concept will be very familiar. For the rest of you, I am going to discuss the myofascial trigger point compression technique. Here we are going to apply steady, deep, direct pressure to the nodule or pea-like trigger point. I have tried to represent this by the idea of a "hot zone": the trigger point is located somewhere in this zone. You want to find the direction of pressure that, where possible, reproduces the exact pain that the patient is experiencing. It often amazes me that a slight change in the direction of the pressure can cause a totally different pain elsewhere. Again, you need to reproduce the patient's pain in these particular zones: ask the patient to tell you when you are "there."

Figure 4.15: Hot zones.

How do I know when I have done enough pressing?

Hold the trigger point until either (a) the patient's pain diminishes massively, or (b) the trigger point softens or evaporates beneath your pressure. With NMT it is recommended that if the trigger point does not yield within 6–10 seconds, you come away, do some massage, and repeat up to three times.

Follow all deep work with a gentle generalized effleurage massage. The area where you did the deep work may still be tender but do not avoid it. This will help to dispel pain-inducing toxins from the area and stimulate the repair of the fascia.

Are the trigger points and referred pain patterns the same for everyone?
Generally yes, although the following will have an effect on the pattern:

- Ageing
- Posture
- Obesity
- Anorexia
- Scar tissue
- Myofascial strain patterns
- Congenital anomaly
- Type of muscle fiber
- Direction/orientation of muscle fiber
- Type of muscle morphology (fusiform, etc.)
- Chronicity of trigger point

What effects do obesity, anorexia, and scar tissue have?
These factors will change the fat/muscle ratio and skew the position of the trigger points. They will also have an effect on the planes of the fascia, and hence the location of the trigger points. Similarly, scar tissue or keloid may cause a deviation in the myofascial strain pattern and hence the location of the trigger point.

What about the type and orientation of muscle fiber?
Depending on where they are in the body and the job they have to do, muscle fibers are arranged into various structures. This allows the muscles to generate either more force or a more specific force. Locating a central trigger point will vary therefore according to the arrangement of muscle fibers within any given muscle. In the multipennate fiber arrangement, for example, several trigger points may exist in the middle of each of the functional components.

Will bruising occur?
Bruising may occur if the patient is on blood-thinning medication. With time and experience, bruising becomes increasingly rare. In my experience, it is not the depth of treatment (force) that will cause the skin to bruise, but it is the treatment being done too quickly (velocity). In this respect it helps to visualize the tissues you are addressing as you treat them.

Tip
Try to feel the patient's muscles and tender nodules beneath the skin, and build up the pressure slowly; do not come away too quickly. Arnica creams and tablets may reduce the incidence and severity of bruising.

What creams or lotions can I use?
In general, it is better to avoid oils, as they may cause you to slide off from the pressure points once you have found them. A number of skin lubricants are available—I use plain blue Nivea Creme. Alternatively, arnica cream or plain aqueous cream mixed with some vitamin E oil may be sufficient. Petroleum gel, talcum powder, or massage oil may also be used, but beware in case the patient has a skin or lanolin allergy.

What is the frequency of treatment?
In my experience, for hands-on therapy you should perform three treatment sessions one week apart, another session four weeks later, and a final session twelve weeks after that. This is in line with the mechanical repair of fascia. You may want to review the patient again after this. Injections and dry needling have a much quicker response.

What is the best position for needling?
Needling can be performed with the patient sitting, prone, supine, or lateral decubitus. Unless you are very experienced, it is generally advised to needle with the patient in one of the lying positions: this reduces the side effect of syncope (fainting) and is generally more comfortable.

What happens if a needle gets stuck?
Leave it for up to a minute if the patient can tolerate it, otherwise you can often release it by inserting another needle next to it and then remove them in the order first in first out.

What happens if I hit an artery or a nerve?
Safety must come first! You should study the anatomy and surface anatomical landmarks thoroughly and gently palpate the area before needling. Remember, you are looking for a taut band. Arteries have thick round walls, so in most cases the needle just bounces off around the edge. If you do hit an artery dead-center, however, you should feel a strong pulse, which indicates you should not proceed. In the unlikely event of arterial bleeding, use pressure on the area for up to two minutes. Similarly, in the rare case of hitting a nerve, the sharp lacerating pain should remit when the needle is removed; there is very little chance of any permanent damage if using an acupuncture needle.

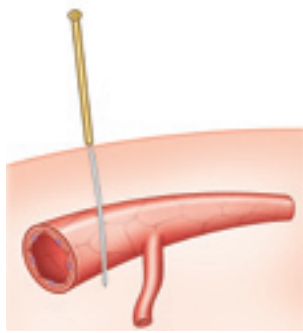


Fig 4.16: Structure of an artery, showing the thick round walls.

5 Stretching and Exercise

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Fitness and Flexibility

An individual's physical fitness depends on a vast number of components, and flexibility is only one of these. Although flexibility is a vital part of physical fitness, it is important to see it as only one "spoke" in the "fitness wheel." Other components include strength, power, speed, endurance, balance, co-ordination, agility, and skill.

Although particular sports require different levels of each fitness component, it is essential to plan a regular exercise or training program that covers all the components of physical fitness. Rugby and American football (gridiron), for example, rely heavily on strength and power; however, the exclusion of skill drills and flexibility training could lead to serious injury and poor performance. Strength and flexibility are of prime concern to a gymnast, but a sound training program would also improve power, speed, and endurance.

The same is true for each individual: while some people seem to be naturally strong or flexible, it would be foolish for such persons to completely ignore the other components of physical fitness. And just because an individual exhibits good flexibility at one joint or muscle group, it does not mean that the entire individual will be flexible. Therefore, flexibility must be viewed as specific to a particular joint or muscle group.

The Dangers and Limitations of Poor Flexibility

Muscles that are tight and stiff limit our normal range of motion. In some cases, lack of flexibility can be a major contributing factor to muscle and joint pain. In the extreme, lack of flexibility can mean it is difficult, for example, to even bend down or look over the shoulder.

Tight, stiff muscles also interfere with proper muscle action. If the muscles cannot contract and relax efficiently, the result will be a decrease in performance and a lack of muscle movement control. Moreover, short, tight muscles cause a dramatic loss of strength, power, and efficiency during physical activity.

In a very small percentage of cases, muscles that are tight and stiff can even restrict blood circulation. Good blood circulation is vitally important in helping the muscles receive adequate amounts of oxygen and nutrients. Poor circulation can result in increased muscle fatigue and, ultimately, will impede the muscles' repair process and the ability to recover from strenuous exercise.

Any one of these factors can greatly increase the chances of becoming injured. Together they present a package that includes muscular discomfort, loss of performance, an increased risk of injury, and a greater likelihood of repeated injury.

Causes of Restricted Flexibility

The muscular system needs to be flexible in order to achieve peak performance, and stretching is the most effective way of developing and retaining flexible muscles and tendons. However, a number of other factors also contribute to a decrease in flexibility.

Flexibility, or range of motion, can be restricted by both internal and external factors. Internal factors such as bones, ligaments, muscle bulk, muscle length, tendons, and skin all restrict the amount of movement at any particular joint. As an example, the human leg cannot bend forward beyond a straight position, because of the structure of the bones and ligaments that make up the knee joint.

External factors such as age, gender, temperature, restrictive clothing, and of course any injury or disability will also have an impact on one's flexibility.

Flexibility and the Ageing Process

It is no secret that with each passing year, the muscles and joints seem to become stiffer and tighter. This is part of the ageing process and is caused by a combination of physical degeneration and inactivity. Although we cannot help getting older, this should not mean that we give up trying to improve our flexibility.

Age should not be a barrier to a fit and active lifestyle, but certain precautions should be taken as we get older. Participants just need to work at it for longer, be a little more patient, and take a lot more care.

Stretching and Strengthening Exercises

Exercise should be regarded in the same way as a doctor's prescription: it should have the appropriate dosage and, for maximum effectiveness, be targeted where needed (amount and type of exercise). While some specific exercises are suggested in Chapters 7–12, to achieve the optimum results the best person to prescribe these would be your appropriate therapist.

Stretching

Stretching has a host of benefits, including:

- Improved range of motion
- Increased power
- Diminished post-treatment soreness
- Reduced fatigue

Stretching the muscles with trigger points, or the muscles that you are trying to strengthen, is important for breaking old holding patterns, restoring range of motion, and preventing injury. Gently stretching after a trigger point treatment session or after strengthening exercises can help reduce muscle soreness and keep your muscles long and flexible.

Types of Stretching

There are many different ways to stretch, each with its advantages and disadvantages. The two most recommended techniques are: (1) *passive/static stretching*, best used at home or after treatment; and (2) *proprioceptive neuromuscular facilitation* (PNF), best used with a partner. There is no such thing as a "good" or "bad" stretch, and the effects of different techniques vary from person to person. It is advised to warm up for 10 minutes before stretching, whether with some cardiovascular exercises or a warm/hot shower.

Passive/Static Stretching Technique

This technique is safe and effective for the novice.

PROCEDURE

1. Place the body in a position where the muscle you want to stretch can be put under tension.
2. Slowly and cautiously approach the stretch.
3. Do not stretch to the point of pain—discomfort is expected, but be cautious not to force the stretch.
4. Hold for a minimum of 20 seconds (45–60 is best) and allow the muscle to lengthen.
5. Breathe and relax.
6. Gently come away and rest for 45–60 seconds.
7. Repeat the stretch 2–3 times.
8. Repeat 2–3 times daily.
9. To make this more effective, stretch the antagonist (the opposite muscle) straight afterwards.

Figure 5.1: A passive/static stretching exercise: lateral side stretch.

Foam Roller Stretching

Foam rollers have been used since the 1950s to stretch ease and 'rebalance' muscular tension. Dr. Moshe Feldenkrais is credited with having been the first person to use them for therapeutic purposes. Foam rollers come in various shapes, sizes and densities; they are cheap to buy and easy to use. Selecting the best roller is down to personal choice. Often this depends on your height, weight and the area you are looking to stretch.

Rollers can be very effective at deactivating trigger points both on their own, after hands-on techniques, and after dry needling. Using a foam roller is simple; used properly they can be very effective for improving:

- Balance
- Flexibility
- Coordination
- Relaxation
- Range of motion

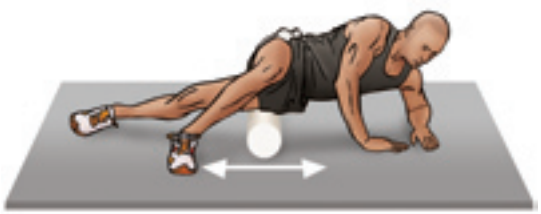

Figure 5.2: Foam rolling the outside of the thigh.

Self-massage

With self-help foam rolling, you are able to determine the amount of pressure you apply to a particular area and the duration of time you hold the point. Whilst they are relatively safe and easy to use, it is advised to seek an opinion from a doctor or informed therapist about the best way to use them. Here are some considerations for trigger point massage:

- A firm circular roller is advised
- Always study the anatomy of the muscle before rolling
- When applying pressure, try to rest on the muscles/soft tissues and not the bones or joints
- Start close to the body and roll away from it. Roll up and down the taut band until you hit the trigger point/sweet spot
- Pause on the pain spot/trigger point for up to 5 minutes, or until you feel it melt away. Repeat as necessary
- Be aware of the posture of the rest of your body when rolling
- Move up and down from the trigger point slowly and carefully
- The foam roller can be used up to 6 times a day

Warning

Remember – pain is an alarm bell, so if you feel exaggerated pain, stop! Be gentle and respectful to the points.

Proprioceptive Neuromuscular Facilitation (PNF)

This is a more advanced technique and may be used for obtaining more permanent results; it also improves muscular strength. There are several forms of PNF stretches, including "hold relax stretch" or "contract relax stretch." Another variation is post-isometric relaxation (PIR) (see p.55).

PROCEDURE

1. Position the muscle group so that it is under tension, and hold.
2. Contract the stretched muscle for 5–6 seconds while a partner resists you moving the joints.
3. Stretch the muscle again for approximately 30 seconds.
4. Rest/recover for 30 seconds.
5. Repeat the procedure 2–4 times (up to 10 minutes).
6. Repeat 2–3 times daily.

Protocol for Stretching

As a rule any stretching program should be continued for four to six weeks, unless otherwise specified by your practitioner, doctor, or physical therapist. After your recovery, these exercises can be continued as a maintenance program for lifelong protection and health. Performing the exercises two to three days a week will maintain strength and range of motion. A goal should be to make a regular time at home every day for stretching the affected muscles toward obtaining full range of motion. It is also advised to keep a diary of any stretches that aggravate your trigger point symptoms.

Remember to warm up before doing stretches: perform 5 to 10 minutes of low-impact activity, such as walking or riding a stationary bicycle.

NB: *Do not* ignore pain**.** It is important to be aware that overzealous stretching can reactivate latent trigger points. The advice is to progress gradually from one stretch to another and listen to your body; different stretches work different types of fiber and afford the brain a better sense of self. You should not feel severe pain during or after a stretch: in general, if a stretch activates your trigger point pain, it should be stopped.

Pain on rest can indicate that the trigger points are very active. The advice here is either to rhythmically move the effected area in warm water or to apply moist heat and the gentlest of massage.

Talk to your practitioner, doctor, or physical therapist if you have any pain while stretching.

Strengthening

Strengthening the muscles improves their tolerance and stamina to exercise. Keeping muscles strong can relieve pain, improve the function of muscles with trigger points, and prevent further injury. As a rule, strengthening a muscle occurs when you hold maximal muscle contraction for 5–10 seconds.

Types of Strengthening

Here we will talk about two types of strengthening exercise—*isometric* and *isotonic*—although others exist.

Isometric Strengthening

Isometric exercises keep the joints in the same position and are non-traumatic. They are relatively simple to perform, require very little equipment, and do not require any previous fitness experience. They are a great first place to start on your strengthening program. Isometric strengthening occurs when you exert variable force to a fixed position: yoga and Pilates, for example, rely a lot on isometric loading. A simple example of an isometric exercise is the "plank":

- Lie face down on the floor or a yoga mat, with your hands directly beneath your shoulders.
- Press down with your hands to lift your torso off the floor.
- Flex your abdominal muscles to keep your back straight.
- Your body should create a long, straight line.

Figure 5.3: An isometric strengthening exercise: the "plank."

Tips

- Isometric exercises are specific to the joint angle: the greater the angle, the longer the lever and the more force required to maintain the position.
- To increase difficulty you can repeat isometric positions every 15–20 degrees throughout the range of motion.
- Hold most contractions for about 6–30 seconds, and repeat if you want to increase the effects.
- Do not forget to breathe.
- If you feel dizzy or light-headed, you should stop.

Isotonic Strengthening

This occurs when you resist against a uniform force. This may involve:

- Weights—barbells, dumbbells, or resistance machines
- Body resistance, e.g. press-ups
- Resistance bands
- Kettle bells

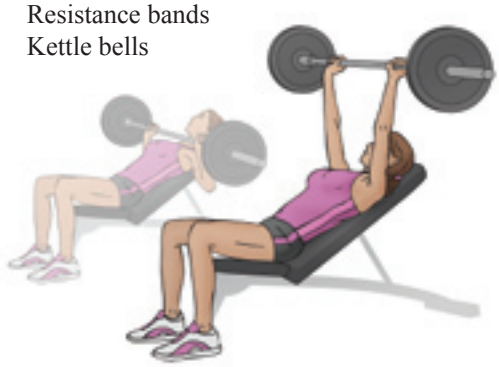

Figure 5.4: An isotonic strenthening exercise: incline barbell press.

Tips

- Exercise each muscle group at least twice a week.
- It is important that you get adequate rest between exercise sessions—at least 48 hours. Isotonic exercise strengthens muscles by creating small tears in them, which then repair. As you rest after your workout, your muscles heal and grow stronger.
- Always warm up before working out and cool down afterwards.
- Stretch at the end of every exercise session.

Protocol for Strengthening

Before doing strengthening exercises, warm up with 5 to 10 minutes of low-impact activity, such as walking or rowing.

NB: *Do not* ignore pain. You should not feel severe pain during or after exercise: in general, if an exercise activates your trigger point pain, it should be stopped.

Talk to your practitioner, doctor, or physical therapist if you have any pain while exercising.

6 Beyond the Trigger Point

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"The fascia is the place to look for the cause of disease and the place to consult and begin the action of remedies in all diseases."

"It is my hope and wish that every osteopath will go on and on in search of scientific facts as they relate to the human mechanism and health, and to an ever extending, unfolding of nature's truths and laws."

Dr. Andrew Taylor Still—Founder of osteopathic medicine, Kirksville, Missouri

Putting It All Together

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

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

Protection

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

then arranged hierarchically into functional units of agonist, antagonist, fixator, and synergist.

In myofascial dysfunction, "switch-off" mechanisms work to avoid the noxious stimuli. We are forced to recruit synergists, fixators, and agonists, often in a less efficient manner, to perform our daily tasks. This is fine in the short term, but over time it can lead to neuroplastic changes in the spinal cord and brain (sensitization). These mechanisms often include reflexes maintained locally in the spinal cord, and centrally in the brain.

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

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

Pain Is a Big Stimulus

With regard to myofascial trigger points, the stressor is acute or chronic pain, either in a joint or in the myofascial matrix. In both cases the body "switches off" around the stimulus; this switching is maintained both locally and centrally. The phenomenon is observable in muscles around a fracture site, a slipped disc, or, for example, a frozen shoulder. Painful stimuli are often mediated by inflammation and its noxious exudates, which is part of a well-demonstrated cascade. When our feedback mechanisms are altered, the brain is forced to adapt and compensate. Pain is a highly motivating symptom for the nervous system: it is our alarm bell that something is wrong.

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

Trigger points on demand—TODs

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

Reciprocal Inhibition

Reciprocal inhibition is an important reflex within our nervous system and has a major role in the control of voluntary movement. It describes the "automatic" process that occurs when muscles on one side of a joint relax to accommodate contraction on the other side of that joint.

As discussed in Chapter 2, joints are controlled primarily by opposing sets of muscles, extensors, and flexors, which must work in synchrony for smooth movement. When a muscle spindle is stretched and the stretch reflex is activated, the opposing muscle group must be inhibited to prevent it from working against the resulting contraction of the homonymous muscle. This inhibition is accomplished by the actions of an inhibitory interneuron in the spinal cord.

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

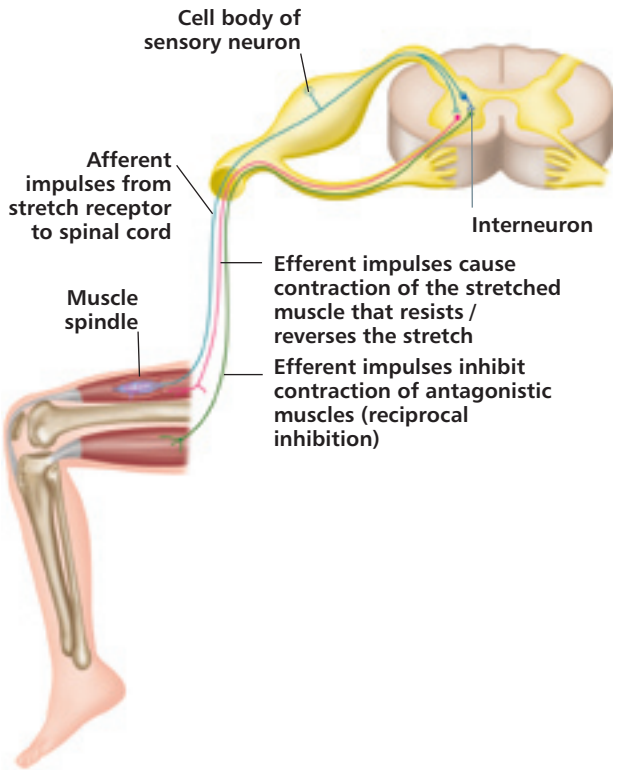

Figure 6.1: The stretch reflex arc and reciprocal inhibition (RI).

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

Holding Patterns

A few years ago, I was stuck in an airplane for almost an hour, circling around Heathrow Airport, waiting for a "landing window." The captain informed us that we were in a holding pattern and should be landing shortly. I have thought a lot about this phrase ever since. For me it neatly encapsulates the way I see a patient when they present in the therapeutic setting. Patients may come with acute or chronic symptoms, but, whatever the origin, the body's myofascial framework adapts and changes in a protective "holding pattern." Over time the "normal" muscle functioning fails, often resulting in multiple trigger point formation. The longer a problem persists, the more rigid these patterns may become. Chains of sarcomeres fail and chronic recalcitrant trigger points form. Peripheral and central sensitization play a role in maintaining this holding pattern, but so does the adapted myofascial infrastructure.

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

Figure 6.2: Shoulder holding patterns.

Complexity

"Complexity theory" may also have a part to play in the trigger point story. Chaos theory and its offshoot complexity theory represent a new paradigm in scientific thought. Complexity theory is pan-disciplinary, relating equally to economics, medicine, anthropology, history, politics, computer science, etc. It offers a framework for addressing many old and fundamental philosophical questions arising from complex systems.

Complexity theory examines the connectivity that is more than the "sum of the parts," and in doing so attempts to answer some fundamental questions. It might also help us conceptualize a model for the "why" of trigger points. Complexity is *deterministic*: in other words, it is grounded in real and measurable math, calculations, theorems, and proofs.

Some of the relevant aspects of complexity theory for this subject are:

1. The idea of different *attractor types* in complex systems
2. The concept of *positive feedback* in complex systems
3. The concept of *emergence*
4. The idea of order existing at the "edge of chaos," the *creation zone*, and self-similarity (fractals)

Chaos theory emerged from the earlier work of scientists such as Henri Poincaré in the 1880s while he was studying the problem of three bodies in nonperiodic orbits. It took hold in the 1980s, when it was used to explain and model nonlinear systems such as the weather. Its implications have been profound, showing how very simple dynamic rules can give rise to extraordinarily intricate and complex behavior: witness the endlessly detailed beauty of fractals, or the foaming turbulence of a river. Chaos is not the same as randomness. For example, when you look down at a busy city from a 30-story building, the cars, buses, and people seem to be moving around randomly. The

truth is that everyone is going somewhere; each person has a vector. What looks like randomness is actually a highly mathematically predictable phenomenon.

The advent of ever-increasing computer power has allowed models to explore chaos theory further in real-world situations. As a result, many researchers from different disciplines have been exploring this scientific frontier. All modalities have independently stumbled upon an eerie but important principle: order, complexity, and structure exist in a narrow band at the edge of chaos. The resulting themes of this research have resulted in complexity theory. This theory explores the simple rules that build complex systems, such as the stock market, a social network, and the musculoskeletal system.

Strange Attractors

Life on our planet exists on a "knife's edge" that some have called the *Goldilock's zone*. Had Earth's orbit been closer to the sun, the water vapor would have boiled away and life could not have started. Had Earth been further away, like Mars, it would have been frozen and stagnant. Had the valency of hydrogen not allowed it to form a stable bond with oxygen, we would not be here. Again and again in almost every system, this pattern emerges. On one side of the boundary there is chaos, this nonlinear dimension of constant turmoil, upheaval, and change; on the other side there is rigidity, structure, and order. Using computer models, such as cellular automata, this principle has been explored further.

Physics and computer whiz kid Stephen Wolfram (at the age of 12, he wrote a dictionary of physics) performed a breakthrough experiment in 1984. He was studying a simulated population of cells called *cellular automata* on his computer. He noticed that by tampering with the variables (such as food and sunlight) certain patterns on his computer screen emerged again and again. To his surprise this behavior looked very lifelike.

This behavior pointed to an underlying type of organization. Wolfram's genius was to recognize that there were certain underlying principles at work. He observed certain types of "attractor" which appeared, disappeared, and sometimes stayed in place:

- **Class 1:** Point attractor
- **Class 2:** Periodic attractor
- **Class 3:** Strange attractor

In his automata, Class 1 attractors led to stagnation and stasis, like rolling a marble in a bowl where the cells started whizzing about but then coalesced at the bottom in a clump. Class 2 attractors seemed to have two poles between which the automata would coalesce and occasionally flit off to the other (like a binary star). Only Class 3 attractors produced "lifelike" results. The rules of complex systems only work because of the emergence of these attractors, especially the *strange attractors* (see *Complexity* by Mitchell Waldrop (1992)).

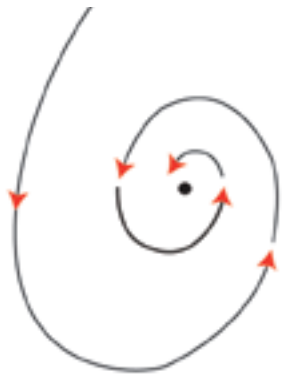

Figure 6.3: Point attractor.

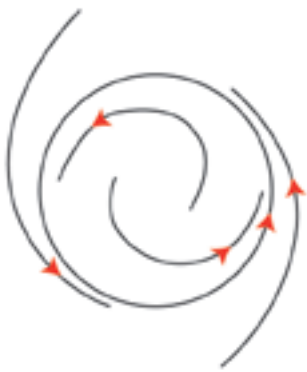

Figure 6.4: Periodic attractor.

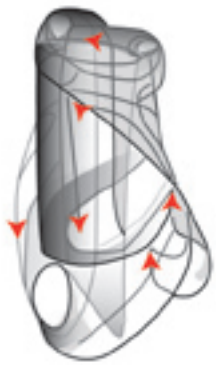

Figure 6.5: Strange attractor.

It would appear that strange attractors act like "organizing catalysts" that seem to spontaneously emerge in all complex dynamic systems. Wolfram's experiments with cellular automata have been extrapolated universally.

Again and again, in all types of complex dynamic system, attractors seem to emerge. They appear to arise spontaneously as an absolute necessity of the system itself. They are like organizing- and structure-giving still points, similar to the still point at the center of a whirlwind.

Attractors Within Complex Human Systems

The human body as a complex dynamic system can be seen to exhibit a range of attractors operating at various levels, and operating in the parts and in the whole. The following systems seem to demonstrate this principle in some way (this list is indicative only):

- The heart, with point, periodic, and strange attractors operating (Mills 2005).
- Homeostatic (dynamic) functions, such as body temperature or the menstrual cycle.
- Spindle formation in embryology.
- The liver—macro and micro functions.
- The reticulo-lymphatic system.
- Osteogenesis and fascial dissemination.

The musculoskeletal system is complex and therefore must exhibit point, periodic, and strange attractors emerging within it. These attractors could well be the "polymodal receptors" which may become myofascial trigger points under certain circumstances.

Trigger Points Are Strange Attractors

Kawakita et al. (2002) proposed the hypothesis that trigger points may come from "sensitized neural structures" called *polymodal receptors* (PMRs). It was suggested that PMRs are a type of nociceptor, which responds to mechanical, thermal, and/or chemical stimuli. PMR "sensory terminals" potentially exist in various tissues throughout the body as "free nerve endings." The theory is that the latent PMRs are "switched on" under certain physiological stimuli and become tender, morphing into the form we call *trigger points*. This theory is also supported by some of the findings in peripheral and central sensitization.

I would like to go further and assert that certain PMRs are *class 3 attractors*—or *strange attractors*—within the complex myofascial web; they emerge and are activated on demand. They are there because they have to be there; they emerge from the complexity and, under certain physiological circumstances and environments, "switch on" as trigger points. They are organizational and also part of the nervous system's negative feedback response to noxious stimuli.

Certain trigger points seem to be constantly "switched on": I suggest that these trigger points are the strange attractors and have named them *super trigger points* (STPs).

Super Trigger Points (STPs)

STPs seem to be active all the time in everyone: it is like they "have to be there." They are the myofascial strange attractors. Releasing trigger points in these muscles appears to have greater systemic effects than expected, often including profound physiological effects (such as autonomic changes). These effects are well beyond the "normal" trigger point reactions, hence the name "super" trigger points.

I have observed that incorporating these "super" trigger points into a treatment protocol acts as a type of shortcut, rapidly releasing deep-seated and chronic pain syndromes. At the end of each muscle chapter, I have suggested some basic NAT protocols which incorporate STPs. Examples of these physiological or "super" trigger points can be found in:

- Sternocleidomastoideus: headaches
- Scalenes: hand and wrist pain and neurovascular problems such as CRPS I
- Infraspinatus near medial scapula; subscapularis and long head biceps brachii: shoulder pain
- Gluteus medius: low back pain
- Ligamentum patellae (patellar ligament) patella insertion: knee pain
- Popliteus: knee pain
- Extensor digitorum longus (at junction of talocrural joint): ankle balance (post-fracture rehabilitation) and ankle pain

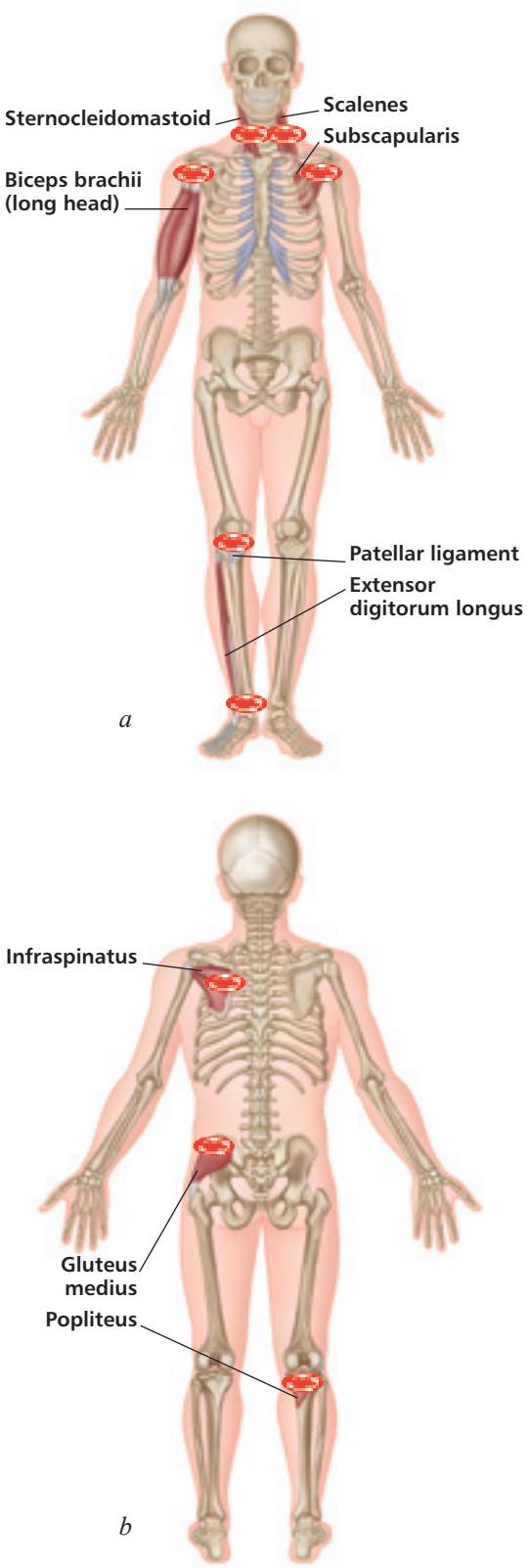

Figure 6.6: Super trigger points: (a) anterior view, (b) posterior view.

Myofascial Meridians

Trigger Points Tend to Develop Along "Myofascial Meridians"

Clinically, trigger points (and super trigger points) tend to emerge in the warp and weft of the myofascia along certain predetermined lines of force, or meridians. The reasons for this have been suggested by Thomas Myers (2001) and are based on the earlier work of Ida Rolf. The concept of "myofascial channels," or chains, helps to explain the way the body dissipates and distributes forces from right to left, up to down, and deep to superficial. It is useful therefore to understand and visualize these myofascial meridian train lines.

Muscles do not operate in isolation, but might be regarded as the contractile elements within a myofascial continuum, which runs throughout the body. These meridian maps may help to explain how and why the development of primary, central trigger points in one area of the body may lead to secondary or satellite trigger points distally. The maps may also explain the "crossover" patterns discussed in Chapter 3. The term *meridian* derives from acupuncture and Traditional Chinese Medicine (TCM): it describes bioenergetic lines or channels that are said to flow throughout the body.

Myokinetic Chains and Sublinks

The brain/body employs a range of neuromuscular strategies to coordinate muscular contraction and thus facilitate stability and spatial orientation. All of our body systems and structures work together in interdependent and connected ways. Myers (2001) presented several ideas for the myofascial component of these connections in his seminal work *Anatomy Trains*, labeling them "myofascial meridians." Sharkey (2008) developed this concept further: he presented these meridians as a series of "functional kinetic chains." Sharkey suggested that the body dissipates kinetic forces (energy) through the "spiral/oblique chain, lateral chain, posterior sagittal chain, and anterior sagittal chain." Several other secondary chains and/or connections also coexist, being both deep and superficial.

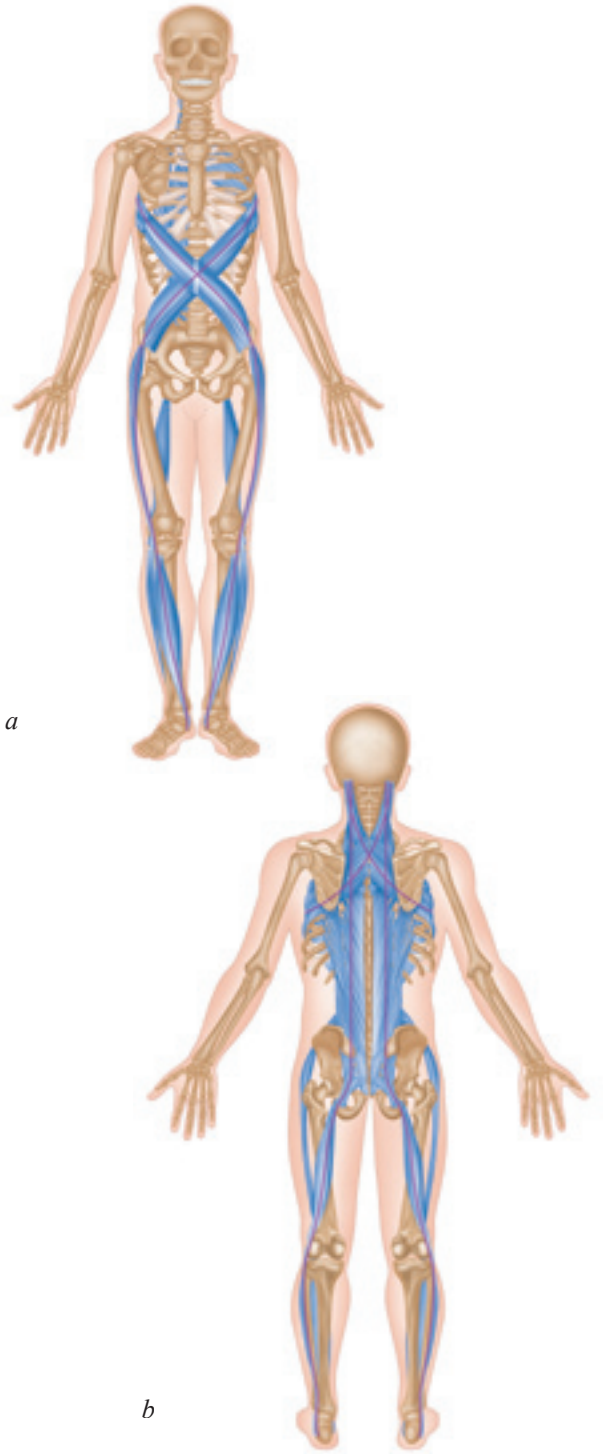

Figure 6.7: The spiral (oblique) chain (S/OC): (a) anterior view, (b) posterior view.

The Spiral (Oblique) Chain (S/OC)

The spiral (oblique) chain includes the external oblique, internal oblique (contralateral), adductors, iliotibial (IT) band, tibialis anterior, and peroneus longus/brevis. This chain may also include the following links: serratus anterior, ipsilateral rhomboids, and contralateral splenius capitis.

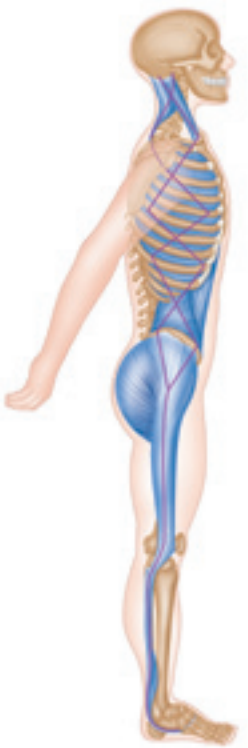

Figure 6.8: The lateral chain (LC).

The Lateral Chain (LC)

The lateral chain includes the peroneals, IT band, TFL, gluteals, external and internal obliques, ipsilateral adductors, and quadratus lumborum (contralateral). This chain may also include the following links: intercostals, sternocleidomastoideus, splenius capitis/cervicis, and scalenes.

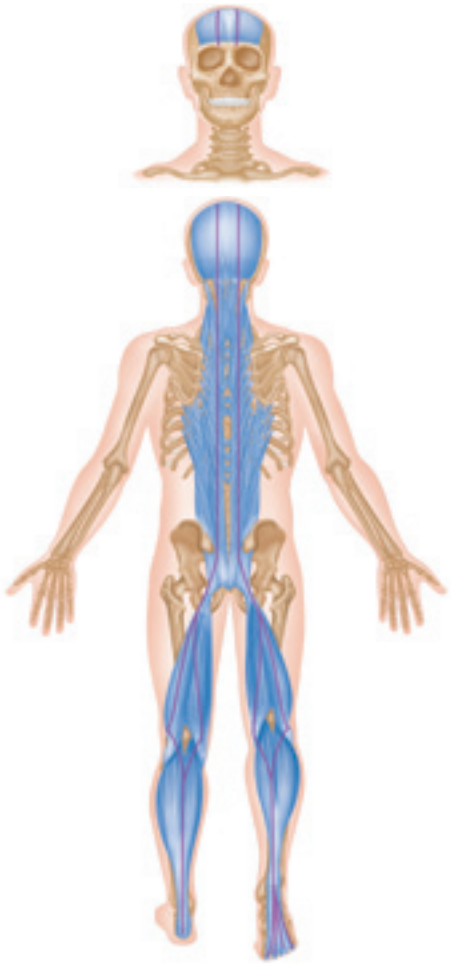

Figure 6.9: The posterior sagittal chain (PSC).

Posterior Sagittal Chain (PSC)

The posterior sagittal chain includes the thoracolumbar fascia and muscular links both above and below, offering movement and support to the joints of the periphery as well as to the spinal joints. At the midsection, sublinks include the transversus abdominis and posterior fibers of the internal obliques. The pelvic floor muscles include the pyramidalis, multifidi, and lumbar portions of the longissimus, iliocostalis, and diaphragm, more commonly known collectively as the *core muscles*. Of course, this joint support system is also present at the glenohumeral and lumbopelvic-hip complexes.

A deep posterior or sagittal chain involves local, deep, segmentally related muscles providing localized support for motion in segments or joints (tonic or type II fibers).

A superficial oblique posterior chain involves prime movers or more global muscles that are, as the name implies, predominantly superficial. These muscles are primarily phasic and are heavily populated with type I fibers with a high resistance to fatigue.

The posterior sagittal chain includes the occipitofrontalis, erector spinae, thoracolumbar fascia, multifidus, sacrotuberous ligament, and biceps femoris (short head). This link can be continued to include the gastrocnemius and plantar fascia. The posterior oblique link (POL) includes the latissimus dorsi, contralateral gluteus maximus, and thoracolumbar fascia. This chain can be continued to include the following links: IT band, tibialis anterior, and peroneals.

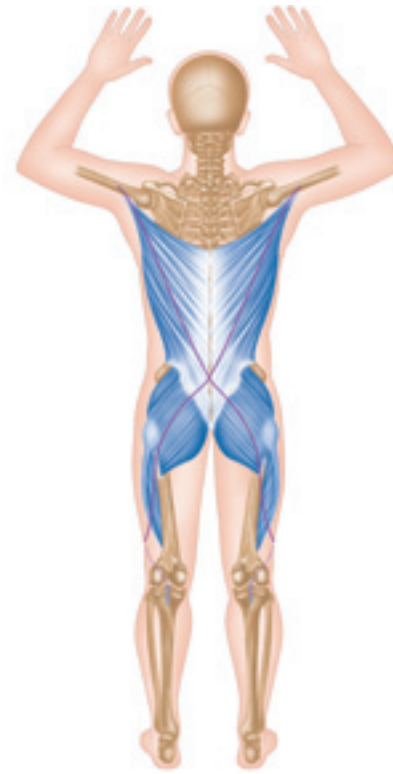

Figure 6.10: The posterior oblique link (POL).

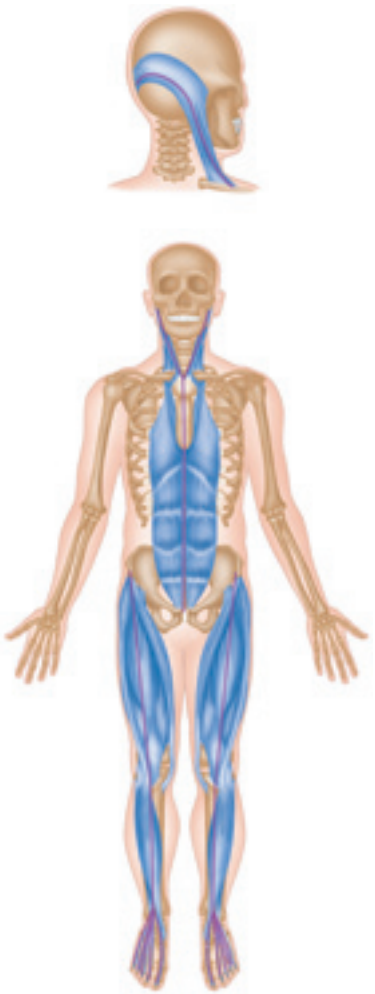

Figure 6.11: The anterior sagittal chain (ASC).

The Anterior Sagittal Chain (ASC)

The anterior sagittal chain includes the dorsal surface of the foot, tibial periosteum, rectus femoris (including articularis genu), anterior inferior iliac spine (AIIS), pubic tubercle, rectus abdominis, sternal periosteum, sternocleidomastoideus, and periosteum of the mastoid process.

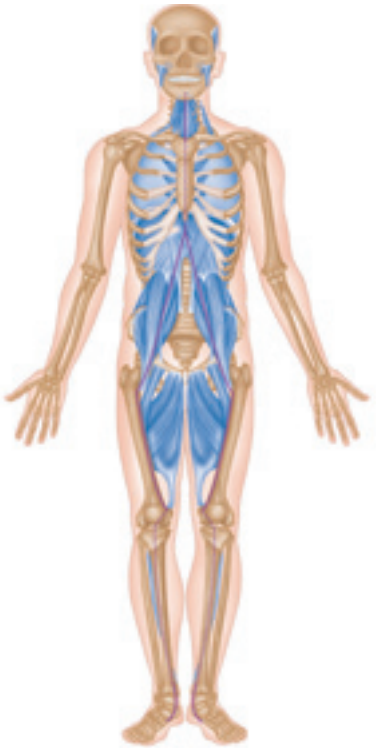

Figure 6.12: The deep anterior chain (DAC).

The Deep Anterior Chain (DAC)

The deep anterior chain includes the inner arch of the plantar surface (first cuneiform), tibialis posterior, medial tibial periosteum, adductors, linea aspera, ramus of the ischium and pubis, lesser trochanter, iliacus, anterior longitudinal ligament, psoas major, central tendon of the diaphragm, mediastinum and pericardium, pleural fascia, prevertebralis fascia, fascia scalenes, longus capitis, hyoid and associated fascia, mandible, occiput, and galea aponeurotica.

The Niel-Asher Technique (NAT)

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

During most hands-on treatments, a haphazard stream of various mechanoreceptors are stimulated. In NAT, the number of inputs is reduced to an absolute and purposeful minimum. Inputs are made via the trigger points in repeatable sequences, which always include the manipulation of STPs as well as trigger points in agonists and antagonists (see the next section on 3-D release). Part of the NAT input sequence is performed three times: repeating something three times (either verbally or somatically) seems to help the nervous system "get the point." Stroking massage is performed in "one direction" only, and compression techniques are performed to the point of *pain* (and are sometimes held for up to 10 minutes). NAT is best performed using the elbow.

The concept of there being one way of treating somatic dysfunction is anathema to some therapists; we have all been taught to treat individuals in context and that each case, and therefore treatment protocol, is different. All I can say is, try NAT for yourself and see—it works every time.

I originally developed NAT in 1999 to treat frozen shoulder syndrome (adhesive capsulitis). The shoulder is one of the most complex regions in the body, involving four joints and eighteen muscles. A frozen shoulder is considered by many to manifest the worst components of all other shoulder problems put together. It is in many ways an enigma, and like other enigmas, solving the puzzle helps us gain many truths and insights of the inner workings of the nervous system. I observed that specific tender/trigger points seemed to be present in the same distribution in *every single patient*. To my surprise, by manipulating these points in a sequence, I discovered that my patients' long-standing frozen shoulders seemed to melt away in as few as one or two treatments. This rapid "defrosting" could only be explained by a neurological process which led to the current theoretical model.

Many thousands of frozen shoulders have now been successfully treated with NAT. It is evidence based and is now used to treat a wide range of musculoskeletal conditions.

More information at www.nielasher.com.

NAT Theory

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

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

The nervous system responds to these input sequences by releasing the "holding pattern," normalizing motor unit output, and improving co-coordination. Clinically, after each NAT session, patients describe a sense of joints being "oiled inside" or feeling that "normal" muscular control has been regained. With regard to somatic dysfunction, NAT is readily used to reinvigorate and release protective joint postures (such as with spondylolisthesis) and/or treat protective spasm around joint problems (such as an arthritic hip).

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

Three-Dimensional (3-D) Release

The brain has 3-D sensory and motor maps soft-wired into the cortex. Our brain (motor cortex) responds to our movement demands by coordinating complex sequences of motor units. These motor units can contract singly or collectively, and when more power is demanded, groups of units combine (recruitment). One of the key ways the motor system achieves smooth coordinated movement is by utilizing the type of triangulation known as *antagonism*. The triangle is formed by agonists, antagonists, and fixators (the *fixators* hold the joints still so that the agonist and antagonist can operate efficiently—see Chapter 2). When a trigger point develops in one of these three groups, the others are forced to compensate. That is why it is important to treat trigger points in the antagonist as well as the agonist, in other words a 3-D release. A number of factors come into play, which magnify these effects over time. These factors are: reciprocal inhibition (where an antagonist is partially or fully switched off), pure facilitation (where an antagonist is made stronger), and co-facilitation (where increased power is routed to teams of secondary muscles).

Much of the experimental data demonstrating antagonism have been generated on healthy volunteers. I would like to suggest that in the pathological situation (such as a frozen shoulder), the brain is often forced to compromise this antagonism and, to this end, it exhibits a degree of neuroplasticity. In other words, the agonist/antagonist relationship may not operate in the classically described manner.

The frozen shoulder, for example, might be considered to be one of the built-in protective responses of our nervous system to avoid noxious stimuli (such as reactive tendinopathy and pain). Every time you try to push the frozen shoulder in one direction, it pushes against you in the opposite one. The brain is constantly working to protect against what it perceives as a threat. This manifests as a stiff and painful (frozen) shoulder, with many trigger points in many muscles. (To a certain extent, we can see this protective pattern in all painful shoulder complaints.) It is as if the brain needs to take the painful shoulder (neurologically) into a "sling-like" position and hold it there for months, or even years, after the pain stimulus has remitted. This is possibly due in part to both peripheral and central sensitization.

3-D NAT Sequences

I have included some of the 3-D NAT release techniques as examples at the end of each of the colored muscle sections (Chapters 7–12). For more details of NAT please visit www.nielasher.com.

Here is an example of a basic NAT treatment for mild to moderate osteoarthritic hip (O/A hip):

1. Assess ROM.
2. Examine the muscles and note trigger points.
3. Treat trigger points in the *adductors* (with patient in a lateral decubitus position on the affected side), using deep stroking massage in one direction only, from the knee to the groin.
4. Use inhibition compression technique, pausing on the trigger point at the adductor insertion.
5. Turn patient onto the opposite side and treat trigger points in the TFL (deep stroking massage in a hip-to-ankle direction only) and pause on the *gluteus minimus/medius* trigger point using inhibition compression.
6. Articulate the hip in passive circumduction.
7. Repeat steps 4 and 5 three times.
8. Turn the patient supine and treat trigger points in the *pectineus* and small *internal rotator hip muscles*.
9. Recheck ROM.

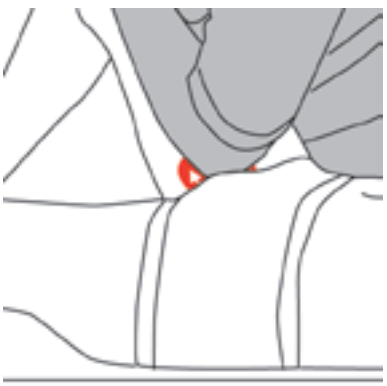

Figure 6.13: Supine inhibition of the pectineus using the elbow.

Altered Antagonism

Having treated well over a thousand frozen shoulders, I have observed that in someone with acute shoulder pain the biceps brachii and triceps brachii pairing stops operating properly. Instead, the biceps brachii and the infraspinatus pair off; similarly, the triceps brachii and the pectoralis minor seem to change their functional relationship. NAT takes these functional antagonistic changes into account during treatment sequences.

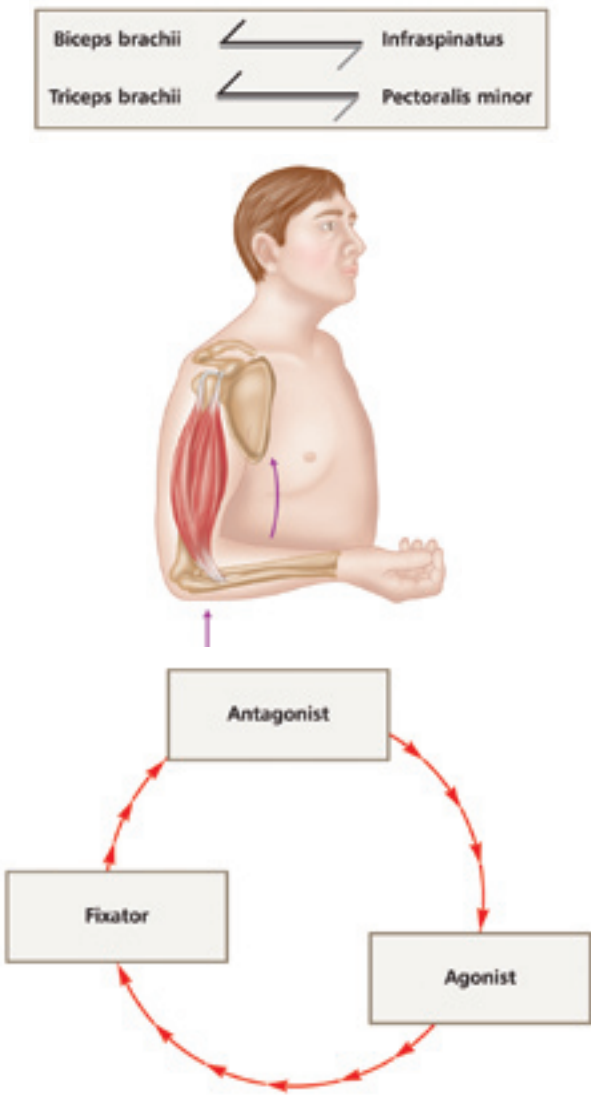

Figure 6.14: Observational functional antagonist reflex patterns for the "capsular pattern" of the frozen shoulder.

You can observe this phenomenon for yourself. If you stimulate the trigger point in the infraspinatus somewhere near the lateral scapular border in a patient with a frozen shoulder when they are supine, they will almost always tell you that they can feel referred pain in the anterior shoulder region of the deltoid and (long head) of the biceps brachii. In other words, treating a trigger point in the functional antagonist may reflect pain and reproduce the trigger point symptoms in the agonist.

Treat Trigger Points in Reverse

The types of functional relationship described above become apparent in particular in muscles with chronic trigger points. In such cases, it pays to establish the primary tissues that are causing symptoms and then look at the antagonistic "holding pattern." I have found that treating the secondary satellite or latent trigger points first, and only then the central myofascial trigger points, makes treatment more effective and longer lasting. Stimulating a sequence of three points three times (one of these points should be an STP) allows the brain to triangulate the sensory input. The motor cortex responds by automatically releasing the holding patterns, which have become established in the 3-D map. There is an old osteopathic adage: "Treat the secondary (holding) pattern and the primary problem will sort itself out."