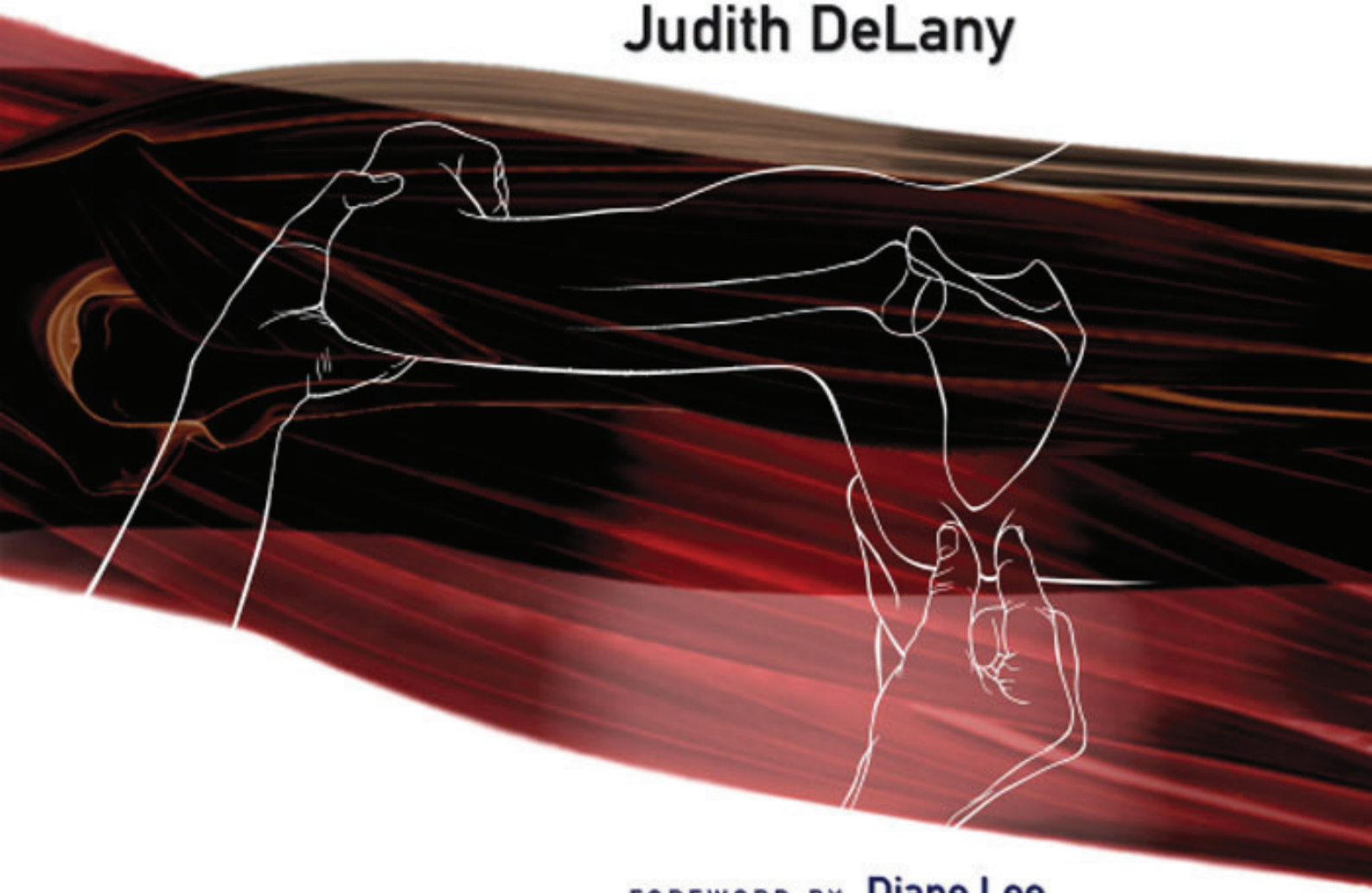
VOLUME 1:
The Upper Body

Clinical Application of NEUROMUSCULAR TECHNIQUES

SECOND EDITION

Leon Chaitow
Judith DeLany



FOREWORD BY Diane Lee

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Clinical Application of Neuromuscular Techniques

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Clinical Application of Neuromuscular Techniques

Volume 1 – The Upper Body

Second Edition

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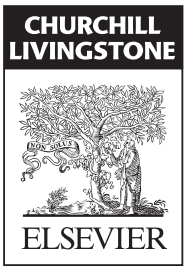

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Foreword

Headache, TMJ, neck/shoulder pain and tennis elbow are all common complaints of patients seeking help from various health practitioners. The source of the impairment and/or the pain is often found in the neuromyofascial system. As a novice, a clinician will approach the problem based on the paradigm taught in their formal training such as physiotherapy, osteopathy, massage therapy, Rolfing, acupuncture or chiropractic. Thus we see the advocacy of many different traditional treatments for myofascial pain such as:

- Physiotherapy – thermal agents followed by stretching exercises
- Osteopathy – strain/counterstrain, positional release, functional and muscle energy techniques
- Massage therapy – deep pressure on tender points, stroking, lymphatic massage techniques
- Rolfing – deep fascial release/stretching techniques
- Acupuncture – dry needling of 'Ah Shi' points
- Chiropractic – manipulation (high velocity, low amplitude thrust techniques) of the spinal segment which correlates to the segmental nerve supply of the affected muscle.

At this point, you may be thinking 'Wait a minute! I do more than that (or all of that, or some of that) for my patients with myofascial pain'. This is true enough, since over time most clinicians gain expertise and are exposed to the paradigms of other disciplines and thus their 'tool box' grows. This book is a wonderful representation of all the paradigms of the many disciplines that have ever considered how to relax/release a muscle or a trigger point in a muscle. Yet, this book is way more than this and even more than the title *Clinical Application of Neuromuscular Techniques* alludes to.

While this text relies heavily on the clinical expertise of both the authors and the historical leaders in both their professions and others, it also refers and draws on the current scientific evidence where it is available. Some may say that the techniques and suggested protocols in this text are not evidence-based and I think it is worthwhile defining exactly what evidence-based practice is. According to Sackett et al (2000),

> *Evidence-based practice is the integration of best research evidence, clinical expertise and patient values. External clinical evidence can inform, but can never replace individual clinical expertise, and it is this expertise that decides whether the external evidence applies to the patient at all, and if so, how it should be integrated into a clinical decision.*

What is expertise? Expertise has been defined as the ability to do the right thing at the right time (Ericsson & Smith 1991). Indeed, I believe that this monumental text is evidence-based since it includes the best available research evidence and integrates it with the multi-disciplinary clinical expertise that has accumulated over the last 100 years.

As mentioned earlier, this text is about more than neuromuscular techniques. It begins with an overview of the anatomy and function of connective tissue, fascia, muscles and the nervous systems (peripheral and central). The anatomical illustrations are clear, well-labeled and pertinent. Many of the current hypotheses regarding the causes of musculoskeletal dysfunction and the various patterns of presentation are outlined. There is an extensive discussion on the current theories and evidence pertaining to the cause, effect and clinical presentation of myofascial trigger points. While ultimately the text turns to the detailed treatment of every possible muscle you could think of in the upper half of the body, prior to this the authors discuss where, when and how the neuromuscular techniques fit into the entire treatment protocol. This ensures that the reader is not left with the impression that neuromuscular release is all that is needed for treating a patient. Once into treatment, consideration is given to the role of non-manual therapies such as thermal modalities, spray and stretch and exercise, and then the use of the manual techniques is explained in great detail. Following this, the upper half of the body is divided and each section begins with a review of

the regional anatomy and biomechanics and a listing of the muscles in which trigger points are commonly found. Each manual technique is illustrated and described in explicit detail. This is easy for the novice to follow and often contains 'pearls of clinical wisdom' for the expert clinician.

Leon Chaitow and Judith DeLany are to be congratulated for the second edition of *Clinical Application of Neuromuscular Techniques*, a text which is applicable to the novice and the expert of any discipline that deals with patients presenting with impairments of the neuromyofascial system.

White Rock, BC, Canada 2007 Diane Lee

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Ericsson KA, Smith J 1991 Towards a general theory of expertise: prospects and limits. Cambridge University Press, New York

Sackett DL, Strauss SE, Richardson WS, et al 2000 How to practice & teach evidence-based medicine. Elsevier Science, New York

Preface to the Second Edition

The clinical utilization of soft tissue manipulation has increased dramatically in recent years in all areas of manual health-care provision. A text that integrates the safe and proficient application of some of the most effective soft tissue techniques is both timely and necessary. The decision to write this book was therefore based on a growing awareness of the need for a text that describes, in some detail, the clinical applications of neuromuscular techniques in particular, and soft tissue manipulation in general, on each and every area of the musculoskeletal system.

There are numerous texts communicating the features of different manual therapy systems (osteopathy, chiropractic, physical therapy, manual medicine, massage therapy, etc.) and of modalities employed within these health-care delivery systems (high-velocity thrust techniques, muscle energy techniques, myofascial release and many, many more). There are also excellent texts that describe regional problems (say of the pelvic region, temporomandibular joint or the spine) with protocols for assessment and treatment, often presented from a particular perspective. Increasingly, edited texts incorporate a variety of perspectives when focusing on particular regions, offering the reader a broad view as well as detailed information on the topic. And then there are wonderfully crafted volumes, such as those produced by Travell and Simons, covering the spectrum of 'myofascial pain and dysfunction' and incorporating a deeply researched and evolving model of care.

We adopted Travell and Simons' view of the human body, which offers a valuable regional approach model on which to base our own perspectives. To this practical and intellectually satisfying model, we have added detailed anatomical and physiological descriptions, coupled with clinically practical 'bodywork' solutions to the problems located in each region. In this first volume of the text, the upper body is covered; in Volume 2, the region from the waist down is surveyed in the same way. As authors, we have attempted to place in context the relative importance and significance of local conditions, pain and/or dysfunction, which are quite logically the main focus for the patient. However, we believe it is vital that local problems should be commonly seen by the practitioner to form part of a larger picture of compensation, adaptation and/or decompensation and that the background causes (of local myofascial pain, for example) be sought and, where possible, removed or at least modified.

We also take the position that it is the practitioner's role to take account of biochemical (nutritional and hormonal influences, allergy, etc.), biomechanical (posture, breathing patterns, habits of use, etc.) and/or psychosocial (anxiety, depression, stress factors, etc.) influences that might be involved, as far as this is possible. If appropriate, suitable advice or treatment can then be offered. However, if the practitioner is not trained and licensed to do so, professional referral becomes the obvious choice. In this way, the focus of health care goes beyond treatment of local conditions and moves toward holism, to the benefit of the patient.

In this volume, the person applying the techniques is referred to as the 'practitioner' so as to include all therapists, physicians, nurses or others who apply manual techniques. To ease confusion, the practitioner is depicted as male and the recipient of the treatment modalities (the patient) is depicted as female so that gender references (he, his, she, hers) used within the text are not ambiguous. In Volume 2, the roles are reversed with the female practitioner treating the male patient.

The protocols described in this text fall largely within the biomechanical arena, with the main emphasis being the first comprehensive, detailed description of the clinical application of NMT (neuromuscular *therapy* in the USA, neuromuscular *technique* in Europe). The descriptions of NMT are mainly of the modern American version, as described by Judith DeLany, whose many years of involvement with NMT, both clinically and academically, make her a leading authority on the subject.

Additional therapeutic choices, including nutritional and hydrotherapeutic, as well as complementary bodywork methods, such as muscle energy, positional release and

variations of myofascial release techniques, and the European version of NMT, are largely the contribution of Leon Chaitow, as are, to a large extent, the opening chapters regarding the physiology of pain and dysfunction.

In addition to the practical application sections of the book, a number of chapters offer a wide-ranging overview of current thinking and research into the background of the dysfunctional states for which solutions and suggestions are provided in later chapters. The overview, 'big picture' chapters cover the latest research findings and information relevant to understanding fascia, muscles, neurological factors, patterns of dysfunction, pain and inflammation, myofascial trigger points, emotional and nutritional influences and much more. It is our assertion that the combination of the 'big picture', together with the detailed NMT protocols, offers a foundation on which to build the exceptional palpation and treatment skills necessary for finding effective, practical solutions to chronic pain conditions.

Some chapters, such as Chapters 6 and 7, have evolved substantially since the first edition, based on integration of our diverse viewpoints, with the occasional result being paradigm shifts that altered therapeutic platforms. We believe that this integration of new information and research, in tandem with our combined clinical experience, offers an expanded perspective. Readers can use these concepts to assist in safe application of the methods described, especially if they have had previous training in soft tissue palpation and treatment. The text of this book is therefore intended as a framework for the clinical application of NMT for those already qualified (and, where appropriate, licensed to practice), as well as being a learning tool for those in training. It is definitely not meant to be a substitute for hands-on training with skilled instructors.

To this volume is married the companion text for the lower body, the layout and style of which is very similar. Its foundational chapters cover posture, gait, balance, influences of the close environment surrounding the body, adaptations from sport and other repetitious use, and other contextual material that influences clinical thinking. Additionally, *Clinical Application of Neuromuscular Techniques – Practical Case Study Exercises* is now available to support the practitioner in developing a model by which to apply the protocols to clinical cases. The use of the study guide cases is enhanced with the addition of key words printed in red that may be found in the indices of the larger texts. We trust that these tools, together with practitioner's skills and training, will assure that NMT remains a powerful tool in the manual therapy fields.

London 2007 LC
Florida 2007 JD

Acknowledgments

In the first edition of this text and its companion volume for the lower body, a substantial number of people dedicated many hours of time to assure clarity and accuracy of the final text. Their contribution was not lost in the second edition. Instead, it served as a solid foundation to be built upon with the contributions of revised and added material.

The authors once again express sincere gratitude to the original team who help formulate this project many years ago and to the various authors and illustrators whose work was cited, quoted and borrowed. Additionally, contributions, support and inspiration for this revised edition were given by William Elliott, Donald Kelley, Ken Crenshaw, Ron Porterfield, Nathan Shaw, Mary-Beth Wagner, Andrew and Kaila DeLany, and Adam Cunliffe.

In the second edition of this book, a new team of talented staff members at Elsevier offered insightful ideas, patient support to achieve deadlines, and a variety of professional services in order for the work to evolve. Among those who made this second edition possible, the authors especially acknowledge and appreciate the efforts of Claire Wilson, Gail Wright, Claire Bonnett and the illustration team who gave visual life to the pages of text.

To Sarena Wolfaard, we express deep appreciation for her steady nature and for her ability to juggle the assorted deadlines and the many phases of the project so as to keep it close to its production schedule. She has proven herself as capable of filling the extraordinary shoes of Mary Law, who served as the editorial director of the first edition. As to Mary, her contributions will last forever and her presence is continually missed.

And, most endearingly, we offer our deepest gratitude to our families for their patience, support, and inspiration, all of which fills an ever-present and deep well from which we can draw to sustain and nurture ourselves. Their loving support is threaded through these pages in remarkable yet indiscernible ways.

ACKNOWLEDGEMENTS FROM THE FIRST EDITION

Books are written by the efforts of numerous people, although most of the support team is invisible to the reader. We humbly express our appreciation to our friends and colleagues who assisted in this project and who enrich our lives simply by being themselves.

From the long list of staff members and practitioners who dedicated time and effort to read and comment on this text, we are especially grateful to Jamie Alagna, Paula Bergs, Bruno Chikly, Renée Evers, Jose Fernandez, Gretchen Fiery, Barbara Ingram-Rice, Donald Kelley, Leslie Lynch, Aaron Mattes, Charna Rosenholtz, Cindy Scifres, Alex Spassoff, Bonnie Thompson and Paul Witt for reviewing pages of material, often at a moment's notice. And to those whose work has inspired segments of this text, such as John Hannon, Tom Myers, David Simons, Janet Travell and others, we offer our heartfelt appreciation for their many contributions to myofascial therapies.

John and Lois Ermatinger spent many hours as models for the photographs in the book, some of which eventually became line art, while Mary Beth Wagner dedicated her time coordinating each photo session. The enthusiastic attitudes and tremendous patience shown by each of them turned what could have been tedious tasks into pleasant events.

Many people offered personal support so that quality time to write was available, including Lois Allison, Jan Carter, Linda Condon, Andrew DeLany, Valerie Fox, Patricia Guillote, Alissa Miller, and Trish Solito. Special appreciation is given to Mary Beth Wagner and Andrea

Conley for juggling many, many ongoing tasks which serve to enhance and fortify this work.

Jane Shanks, Katrina Mather, and Valerie Dearing each put forth exceptional dedication to find clarity, organization and balance within this text, which was exceeded only by their patience. The illustration team as well as the many authors, artists and publishers who loaned artwork from other books have added visual impact to help the material come alive.

To Mary Law, we express our deepest appreciation for her vision and commitment to complementary medicine worldwide. Mary's ability to foster organization amidst chaos, to find solutions to enormous challenges and to simply provide a listening ear when one is needed has endeared her to our hearts.

And finally, to each of our families, we offer our deepest gratitude for their inspiration, patience, and ever present understanding. Their supporting love made this project possible.

Chapter 1

Connective tissue and the fascial system

CHAPTER CONTENTS

Connective tissue forms the single largest tissue component of the body. The material we know as fascia is one of the many forms of connective tissue.

In this chapter we will examine some of the key features and functions of fascia in particular, and connective tissue in general, with specific focus on the ways in which:

- these tissues influence myofascial pain and dysfunction
- their unique characteristics determine how they respond to therapeutic interventions, as well as to adaptive stresses imposed on them.

In order to understand myofascial dysfunction, it is important to have a clear picture of this single network that enfolds and embraces all other soft tissues and organs of the body, the fascial web. In the treatment focus in subsequent chapters, a great deal of reductionist thinking will be called for as we identify focal points of dysfunction, local trigger points, individual muscular stresses and attachment problems, with appropriate local and general treatment descriptions flowing from these identified areas and structures.

Box 1.1 Definitions

Stedman's Medical Dictionary (2004) says fascia is:

A sheet of fibrous tissue that envelops the body beneath the skin; it also encloses muscles and groups of muscles, and separates their several layers or groups

and that connective tissue is:

The supporting or framework tissue of the ... body, formed of fibrous and ground substance with more or less numerous cells of various kinds; it is derived from the mesenchyme, and this in turn from the mesoderm; the varieties of connective tissue are: areolar or loose; adipose; dense, regular or irregular, white fibrous; elastic; mucous; and lymphoid tissue; cartilage; and bone; the blood and lymph may be regarded as connective tissues, the ground substance of which is a liquid.

Fascia, therefore, is one form of connective tissue.

The truth, of course, is that no tissue exists in isolation but acts – is bound to and is interwoven – with other structures, to the extent that a fallen arch can directly be shown to influence TMJ dysfunction (Janda 1986). In contrast, loss of occlusal supporting zone can change weight distribution on the feet and alter overall body posture (Yoshino et al 2003a,b). When we work on a local area, we need to keep a constant awareness of the fact that we are influencing the whole body.

Remarkable research (see Box 1.5 in particular) is adding to our understanding of just how important connective tissues are in relation to musculoskeletal function, and to pain management (Chen & Ingber 1999, Langevin et al 2001, 2004, 2005, Schleip et al 2004). As a foundation of understanding of connective tissue is built within this chapter, this and other research evidence is presented that alters previous concepts of this extraordinary matrix.

THE FASCIAL NETWORK

Fascia comprises one integrated and totally connected network, from the attachments on the inner aspects of the skull to the fascia in the soles of the feet. If any part of this network becomes deformed or distorted, there will be compensating adaptive stresses imposed on other parts of the connective tissue web, as well as on the structures that it divides, envelopes, enmeshes, supports and with which it connects. There is ample evidence that Wolff's law (Wolff 1870) applies, in that fascia accommodates to chronic stress patterns and deforms itself (Cailliet 1996), something which often precedes deformity of osseous and cartilaginous structures in chronic diseases (see Box 1.3). As fascia, ligaments and tendons deform when accommodating to chronic stress (Dorman 1997, Lederman 1997), this might disrupt the homeostasis of the body (Keeffe 1999, Kochno 2001) and certainly interferes with normal function.

Visualize a complex, interrelated, symbiotically functioning assortment of tissues comprising skin, muscles, ligaments, tendons and bones, as well as the neural structures, blood and lymph channels and vessels which bisect and invest these tissues – all given shape, cohesion and functional ability by the fascia. Now imagine removing from this all that is not connective tissue. What remains would still demonstrate the total form of the body, from the shape of the eyeball to the hollow voids for organ placement.

FASCIA AND PROPRIOCEPTION

Research has shown that:

- muscle and fascia are anatomically inseparable
- fascia and other connective tissues form a mechanical continuum that extends throughout the body that includes even the innermost parts of each cell – the cytoskeleton (Chen & Ingber 1999, Oschman 2000)
- fascia moves in response to complex muscular activities acting on bone, joints, ligaments, tendons and fascia
- fascia, according to Bonica (1990), is critically involved in proprioception, which is, of course, essential for postural integrity (see Chapter 3)
- research by Staubesand (using electron microscope studies) shows that 'numerous myelinated sensory neural structures exist in fascia, relating to both proprioception and pain reception' (Staubesand 1996)
- after joint and muscle spindle input is taken into account, the majority of remaining proprioception occurs in fascial sheaths (Earl 1965, Wilson 1966)
- new research by Langevin et al (2001, 2004, 2005), described later in this chapter, suggests that a great deal of communication occurs by means of fascial cellular structures (integrins).

FASCIA: COLLAGENOUS CONTINUITY

Fascia is one form of connective tissue, formed from collagen, which is ubiquitous. The human framework depends upon fascia to provide form, cohesion, separation and support and to allow movement between neighboring structures without irritation. Since fascia comprises a single structure, from the soles of the feet (plantar fascia) to the inside of the cranium (dura and meninges), the implications for body-wide repercussions of distortions in that structure are clear. An example is found in the fascial divisions within the cranium, the tentorium cerebelli and falx cerebri, which are commonly warped during birthing difficulties (too long or too short a time in the birth canal, forceps delivery, etc.). They are noted in craniosacral therapy to affect total body mechanics via their influence on fascia (and therefore the musculature) throughout the body (Brookes 1984, Carreiro 2003, Von Piekartz & Bryden 2001).

Dr Leon Page (1952) discusses the cranial continuity of fascia:

> *The cervical fascia extends from the base of the skull to the mediastinum and forms compartments enclosing the esophagus, trachea and carotid vessels and provides support for the pharynx, larynx and thyroid gland. There is direct continuity of fascia from the apex of the diaphragm to the base of the skull. Extending through the fibrous pericardium upward through the deep cervical fascia the continuity extends not only to the outer surface of the sphenoid, occipital and temporal bones but proceeds further through the foramina in the base of the skull around the vessels and nerves to join the dura.*

FURTHER FASCIAL CONSIDERATIONS

Fascia is colloidal, as is most of the soft tissue of the body (a colloid is defined as comprising particles of solid material

Box 1.2 Biomechanical terms relating to fascia

Creep Continued deformation (increasing strain) of a viscoelastic material with time under constant load (traction, compression, twist)

Hysteresis Process of energy loss due to friction when tissues are loaded and unloaded

Load The degree of force (stress) applied to an area or an organism as a whole

Strain Change in shape as a result of stress (external force)

Stress Force (load) normalized over the area on which it acts (all tissues exhibit stress–strain responses)

Thixotropy A quality of colloids in which the more rapidly force is applied (load), the more rigid the tissue response and to become less viscous when shaken or subjected to shearing forces and to return to the original viscosity upon standing.

Viscoelastic The potential to deform elastically when load is applied and to return to the original non-deformed state when load is removed

Viscoplastic A permanent deformation resulting from the elastic potential having been exceeded or pressure forces sustained for too great a period of time

Box 1.3 Biomechanical laws

Mechanical principles influencing the body neurologically and anatomically are governed by basic laws.

- Wolff's law states that biological systems (including soft and hard tissues) deform in relation to the lines of force imposed on them.
- Hooke's law states that deformation (resulting from strain) imposed on an elastic body is in proportion to the stress (force/load) placed on it.
- Newton's third law states that when two bodies interact, the force exerted by the first on the second is equal in magnitude and opposite in direction to the force exerted by the second on the first.
- Ardnt–Schultz's law states that weak stimuli excite physiological activity, moderately strong ones favor it, strong ones retard it and very strong ones arrest it.
- Hilton's law states that the nerve supplying a joint also supplies the muscles that move the joint and the skin covering the articular insertion of those muscles.
- Head's law states that when a painful stimulus is applied to a body part of low sensitivity (such as an organ) that is in close central connection (the same segmental supply) with an area of higher sensitivity (such as a part of the soma), pain will be felt at the point of higher sensitivity rather than where the stimulus was applied.

suspended in fluid – for example, wallpaper paste or, indeed, much of the human body). Scariati (1991) points out that colloids are not rigid – they conform to the shape of their container and respond to pressure even though they are not compressible. The amount of resistance colloids offer increases proportionally to the velocity of force applied to them. A simple example that gives a sense of colloidal behavior is available when flour and water are stirred together with the resulting colloid being mixed into a paste, using a stick or spoon. A slowly moving stick or spoon will travel smoothly through the paste, whereas any attempt to move it rapidly will be met with a semirigid resistance (known as 'drag'). This makes a gentle touch a fundamental requirement if viscous drag and resistance are to be avoided when attempting to produce a change in, or release of, restricted fascial structures, which are all colloidal in their behavior.

ELASTICITY

Soft tissues, and other biological structures, have an innate, variable degree of elasticity, springiness, resilience or 'give', which allows them to withstand deformation when force or pressure is applied. This provides the potential for subsequent recovery of tissue to which force has been applied, so that it returns to its starting shape and size. This quality of elasticity derives from these tissues' (soft or osseous) ability to store some of the mechanical energy applied to them and to utilize this in their movement back to their original status. This is a process known as hysteresis (see below).

The stability and movement characteristics of each body part – whether this involves organs, vessels, nerves, muscles or bones – is defined by a fibrin matrix combined with other elements. For example, bone incorporates calcium phosphate to lend rigidity, while muscle contains neuroresponsive proteins that enable changes in shape. Each element in connective tissue contributes to its strength, resilience and compliance, with elastin allowing controlled, reversible deformation under strain, and fibrin, laid out along the lines of the local axis of motion, serving as a check on the extent of this deformation.

Although a certain amount of deformation is physiologically necessary, trauma may cause deformation beyond the elastic limit of the tissues, thereby causing permanent damage or possibly resulting in a semipermanent distortion of the connective tissue matrix if the damage is not too severe. Return to normal is then sometimes possible, but only with the reintroduction of sufficient energy to allow a reversal of the deformation process – for example, by means of manual therapy ('soft tissue manipulation'). Appropriately applied 'force' (i.e. slowly) can assist in resolving the deformation results of strain. In such processes energy is both absorbed and released. This energy transfer feature, known as hysteresis, is described further below (Becker 1997, Comeaux 2002).

PLASTIC AND ELASTIC FEATURES

Greenman (1989) describes how fascia responds to loads and stresses in both a plastic and an elastic manner, its response depending, among other factors, upon the type, duration and amount of the load. When stressful forces (undesirable or therapeutic) are gradually applied to fascia (or other biological material), there is at first an elastic reaction in which the degree of slack is reduced. If the force persists, this is

Box 1.4 Connective tissue

Connective tissue is composed of cells (including fibroblasts and chondrocytes) and an extracellular matrix of collagen and elastic fibers surrounded by a ground substance made primarily of acid glycosaminoglycans (AGAGs) and water (*Gray's Anatomy* 2005, Lederman 1997). Its patterns of deposition change from location to location, depending upon its role and the stresses applied to it.

The collagen component is composed of three polypeptide chains wound around each other to form triple helixes. These microfilaments are arranged in parallel manner and bound together by crosslinking hydrogen bonds, which 'glue' the elements together to provide strength and stability when mechanical stress is applied. Movement encourages the collagen fibers to align themselves along the lines of structural stress as well as improving the balance of glycosaminoglycans and water, therefore lubricating and hydrating the connective tissue (Lederman 1997).

While these bonding crossbridges do provide structural support, injury, chronic stress and immobility cause excessive bonding, leading to the formation of scars and adhesions which limit the movement of these usually resilient tissues (Juhan 1998). The loss of tissue lengthening potential would then not be due to the *volume* of collagen but to the random pattern in which it is laid down and the abnormal crossbridges which prevent normal movement. Following tissue injury, it is important that activity be introduced as soon as the healing process will allow in order to prevent maturation of the scar tissue and development of adhesive crosslinks (Lederman 1997).

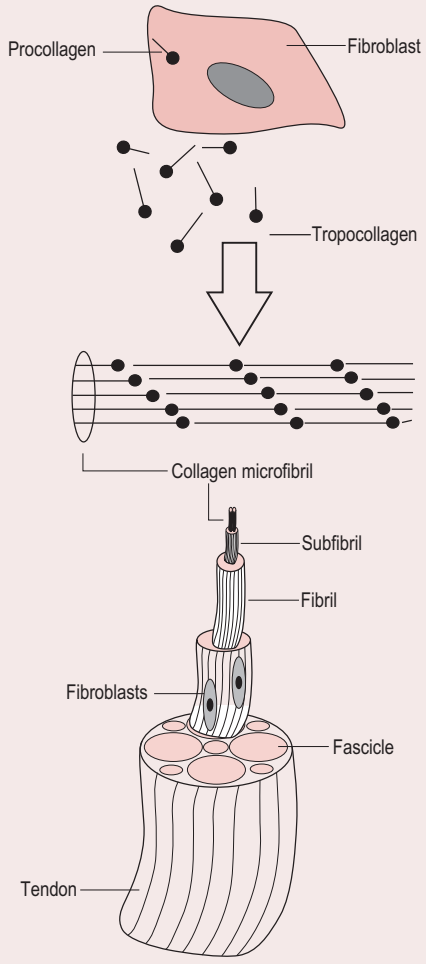


Figure 1.1 Collagen is produced locally for repair of damaged connective tissue. After Lederman 1997.

Lederman (1997) tells us:

The pattern of collagen deposition varies in different types of connective tissue. It is an adaptive process related to the direction of forces imposed on the tissue. In tendon, collagen fibers are organized in parallel arrangement; this gives the tendon stiffness and strength under unidirectional loads. In ligaments, the organization of the fibers is looser, groups of fibers lying in different directions. This reflects the multidirectional forces that ligaments are subjected to, for example during complex movements of a joint such as flexion combined with rotation and shearing ... Elastin has an arrangement similar to that of collagen in the extracellular matrix, and its deposition is also dependent on the mechanical stresses imposed on the tissue.

Elastin provides an elastic-like quality that allows the connective tissue to stretch to the limit of the collagen fiber's length, while absorbing tensile force. If this elastic quality is stretched over time, it may lose its ability to recoil (as seen in the stretch marks of pregnancy). When stress is applied, the tissue can be stretched to the limit of the collagen fiber length with flexibility being dependent upon elastic quality (and quantity) as well as the extent of crossbridging that has occurred between the collagen fibers. Additionally, if heavy pressure is suddenly applied, the connective tissue may respond as brittle and may tear more easily (Kurz 1986).

Surrounding the collagen and elastic fibers is a viscous, gel-like ground substance, composed of proteoglycans and hyaluronan (formerly called hyaluronic acid), which lubricates these fibers and allows them to slide over one another (Barnes 1990, Cailliet 1996, *Gray's Anatomy* 2005, Jackson et al 2001).

- Ground substance provides the immediate environment for every cell in the body.
- The protein component is hydrophilic (draws water into the tissue), producing a cushion effect as well as maintaining space between the collagen fibers (Jackson et al 2001).
- Ground substance provides the medium through which other elements are exchanged, such as gases, nutrients, hormones, cellular waste, antibodies and white blood cells (Juhan 1998).
- The condition of the ground substance can then affect the rate of diffusion and therefore the health of the cells it surrounds.

The consistency of the connective tissue varies from tissue to tissue. Where fewer fibers and more liquid is found, an ideal environment for metabolic activities abounds. With less fluid and more fibers, a soft, flexible lattice is achieved that can hold skin cells, nerve cells or organ tissue in place. With little fluid and many fibers, a tough, stringy material forms for use in muscle sacs, tendons and ligaments. When chondroblasts (cartilage-producing cells) and their hyaline secretions are added, a more solid substance occurs, and when mineral salts are added to achieve a rock-like hardness, bones are formed (Juhan 1998).

Unless irreversible fibrotic changes have occurred or other pathologies exist, connective tissue's state can be changed from a gelatinous-like substance to a more solute (watery) state by the introduction of energy through muscular activity (active or passive movement provided by activity or stretching), soft tissue manipulation (as provided by massage) or heat (as in hydrotherapies). This characteristic, called *thixotropy*, is a 'property of certain gels of becoming less viscous when shaken or subjected to shearing forces

box continues

Box 1.4 (continued)

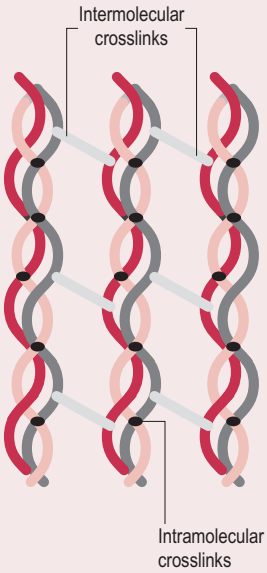


Figure 1.2 Collagen's triple helices are bound together by inter- and intramolecular crosslinking bonds. After Lederman (1997).

and returning to the original viscosity upon standing' (*Stedman's Medical Dictionary* 2004). Without thixotropic properties, movement would eventually cease due to solidification of synovium and connective tissue.

Oschman states (1997):

If stress, disuse and lack of movement cause the gel to dehydrate, contract and harden (an idea that is supported both by scientific evidence and by the experiences of many somatotherapists) the application of pressure seems to bring about a rapid solation and rehydration. Removal of the pressure allows the system to rapidly re-gel, but in the process the tissue is transformed, both in its water content and in its ability to conduct energy and movement. The ground substance becomes more porous, a better medium for the diffusion of nutrients, oxygen, waste products of metabolism and the enzymes and building blocks involved in the 'metabolic regeneration' process ...

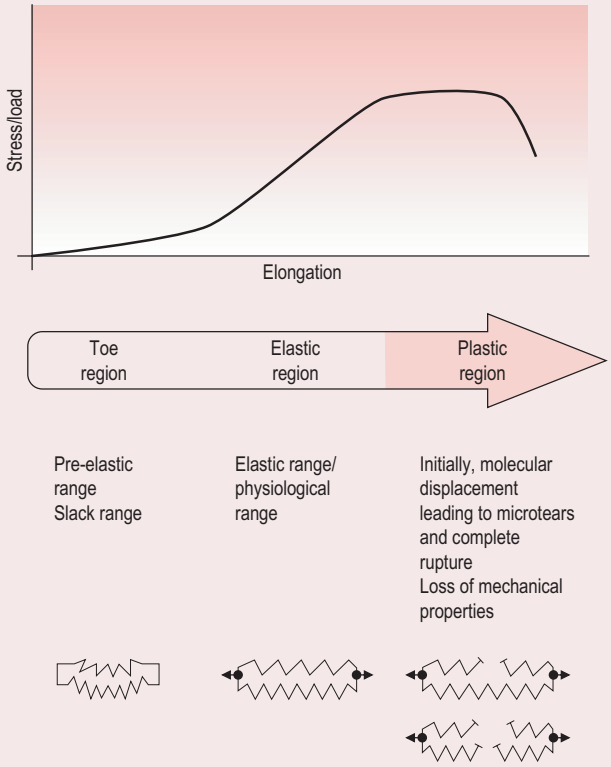


Figure 1.3 Schematic representation of the stress–strain curve. After Lederman (1997).

followed by what is colloquially referred to as *creep* – a variable degree of resistance (depending upon the state of the tissues). This gradual change in shape is due to the viscoelastic property of connective tissue.

Creep, then, is a term that accurately describes the slow, delayed, yet continuous deformation that occurs in response to a sustained, slowly applied load, as long as this is gentle enough not to provoke the resistance of colloidal 'drag'. During creep, tissues lengthen or distort ('deflect') until a point of balance is achieved. An example often used of creep is that which occurs in intervertebral discs as they gradually compress during periods of upright stance.

Stiffness of any tissue relates to its viscoelastic properties and, therefore, to the thixotropic colloidal nature of collagen/fascia. Thixotropy relates to the quality of colloids in which the more rapidly force is applied (load), the more rigid the tissue response will be – hence the likelihood of fracture when rapid force meets the resistance of bone. If force is applied gradually, 'energy' is absorbed by and stored in the tissues. The usefulness of this in tendon function is obvious and its implications in therapeutic terms profound (Binkley 1989).

Hysteresis is the term used to describe the process of energy loss due to friction and to minute structural damage that occurs when tissues are loaded and unloaded. Heat will be produced during such a sequence, which can be illustrated by the way intervertebral discs absorb force transmitted through them as a person jumps up and down. During treatment (tensing and relaxing of tissues, for example, or on-and-off pressure application), hysteresis induction reduces stiffness and improves the way the tissue responds to subsequent demands. The properties of hysteresis and creep provide much of the rationale for myofascial release techniques, as well as aspects of neuromuscular therapy, and

need to be taken into account during technique applications. Especially important are the facts that:

- rapidly applied force to collagen structures leads to defensive tightening
- slowly applied load is accepted by collagen structures and allows for lengthening or distortion processes to commence.

When tissues (cartilage, for example) that are behaving *viscoelastically* are loaded for any length of time, they first deform elastically. Subsequently, there is an actual volume change, as water is forced from the tissue as they become less *sol*-like and more *gel*-like. Ultimately, when the applied force ceases, there should be a return to the original non-deformed state. However, if the elastic potential has been exceeded, or pressure forces are sustained, a *viscoplastic* response develops and deformation can become permanent. When the applied force ceases, the time taken for tissues to return to normal, via elastic recoil, depends upon the uptake of water by the tissues. This relates directly to osmotic pressure, and to whether the viscoelastic potential of the tissues has been exceeded, which can result in a viscoplastic (permanent deformation) response.

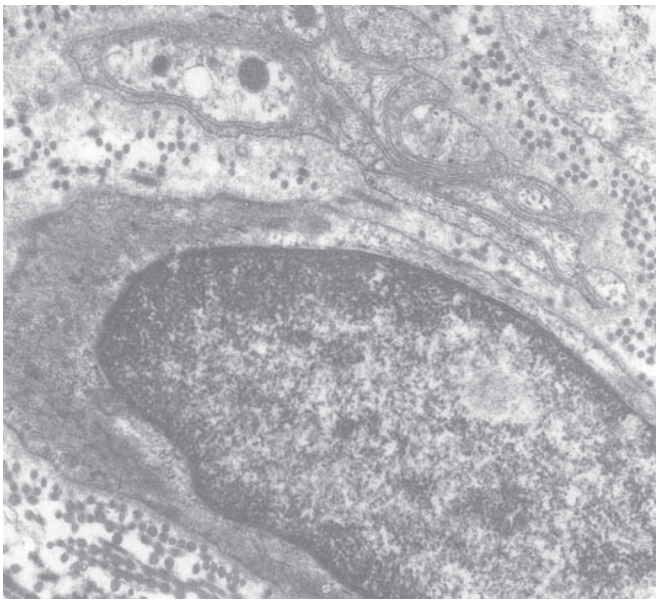


Figure 1.4 Electron photomicroscopy of a typical smooth muscle cell within the fascia cruris. Above it is the terminal portion of a type IV (unmyelated) sensory neuron. (Photo reproduced with the kind permission of Springer Verlag, first published in Staubesand 1996.) Reproduced with permission from *Journal of Bodywork and Movement Therapies* 2003; 7(2):104–116.

CONNECTIVE TISSUE AS A 'SPONGE'

Schleip et al (2004) have shown that when an isometric contraction takes place – as in sustained effort, or therapeutically with methods such as muscle energy technique (MET), proprioceptive neuromuscular facilitation (PNF) or other similar techniques (e.g. 'hold–relax') – fascia loses water and simultaneously loses some of its stability, making it easier to stretch.

It behaves like a sponge, and if the contraction is long and strong enough, and if no movement occurs after the contraction, the fascia reabsorbs water, becoming stiffer as it does so. Research into this phenomenon is in its early stages but at this time the researchers (Schleip et al 2004) have been able to report:

> *By carefully measuring the wet weight of our fascial strips, at different experimental stages, plus the final dry weight (after later drying the strips in an oven), we found the following pattern: During the isometric stretch period, water is extruded, which is then refilled in the following rest period. Interestingly if the stretch is strong enough, and the following rest period long enough, more water soaks into the ground substance than before. The water content then increases to a higher level than before the stretch. Fascia seems to adapt in very complex and dynamic ways to mechanical stimuli, to the degree that the matrix reacts in smooth-muscle-like contraction and relaxation responses of the whole tissue. It seems likely that much of what we do with our hands in Structural Integration and the tissue response we experience, may not be related to cellular or collagen arrangement changes, but to sponge-like squeezing and refilling effects in the semi-liquid ground substance, with its intricate scrub-like arrangement of water binding glycosaminoglycans and proteoglycans.*

Schleip et al (2004) have presented evidence that derives from the same German research, showing that the thoracolumbar fascia has the ability to contract, suggesting that the 'fascia may play an active role in joint dynamics and regulation'. Schleip et al also suggest that this research 'offers new insights into understanding low back instability, compartment syndrome, and myofascial release therapies'.

DEFORMATION CHARACTERISTICS

Cantu & Grodin (1992) describe what they see as the 'unique' feature of connective tissue as its 'deformation characteristics'. This refers to the combined viscous (permanent, plastic) deformation characteristic, as well as an elastic (temporary) deformation status discussed above. The fact that connective tissues respond to applied mechanical force by first changing in length, followed by some of the change being lost while some remains, has implications in the application of stretching techniques to such tissues. It also helps us to understand how and why soft tissues respond as they do to postural and other repetitive insults that exert load on them, often over long periods of time.

It is worth emphasizing that although viscoplastic changes are described as 'permanent', this is a relative term. Such

changes are not necessarily absolutely permanent since collagen (the raw material of fascia/connective tissue) has a limited (300–500 day) half-life and, just as bone adapts to stresses imposed upon it, so will fascia.

If negative stresses (e.g. poor posture, use, etc.) are modified for the better and/or positive (therapeutic) 'stresses' are imposed by means of appropriate manipulation and/or exercise, apparently 'permanent' changes can modify for the better. Dysfunctional connective tissue changes can usually be improved, if not quickly then certainly over time (Brown 2000, Carter & Soper 2000, Neuberger 1953). However, some connective tissue changes are more permanent.

Schleip et al (2004) have observed many examples of tissue contractions caused by connective tissue cells called myofibroblasts (see Box 1.5):

> *This happens naturally in wound healing, but also in several chronic fascial contractures. In the hand, it presents as palmar fibromatosis, also known as Dupuytren's contracture, or as a pad-like thickening of the knuckles. In the foot the same process is called plantar fibromatosis, while in club foot contraction of the myofibroblasts is focused on the medial side. In frozen shoulder, the contraction occurs in the shoulder capsule … considering the existence of pathological fascial contractures, it seems likely that there may be lesser degrees of fascial contractions, which may influence biomechanical behavior.*

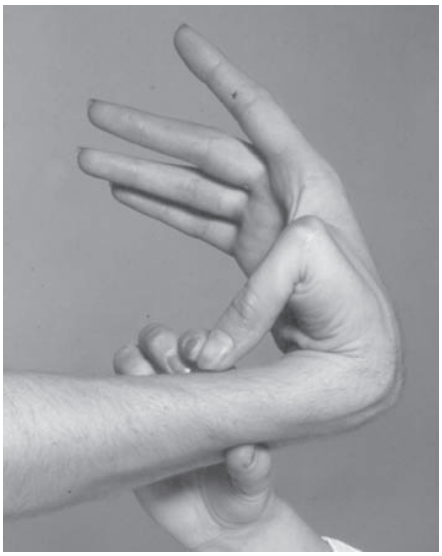
Important features of the response of tissue to load include:

- the degree of the load
- the amount of surface area to which force is applied
- the rate, uniformity and speed at which it is applied
- how long load is maintained
- the configuration of the collagen fibers (i.e. are they parallel to or differently oriented from the direction of force, offering greater or lesser degrees of resistance?)
- the permeability of the tissues (to water)
- the relative degree of hydration or dehydration of the individual and of the tissues involved
- the status and age of the individual, since elastic and plastic qualities diminish with age
- another factor (apart from the nature of the stress load) that influences the way fascia responds to application of a stress load, and what the individual feels regarding the process, relates to the number of collagen and elastic fibers contained in any given region.

HYPERMOBILITY AND CONNECTIVE TISSUE

- Ligamentous laxity and general increased mobility of the connective tissues creates a background of instability.
- Hypermobility is usually genetically acquired. Kerr & Grahame (2003) describe the sequence that leads to this as follows: 'Genetic aberrations affecting fibrous proteins give rise to biochemical variations, then in turn to

A



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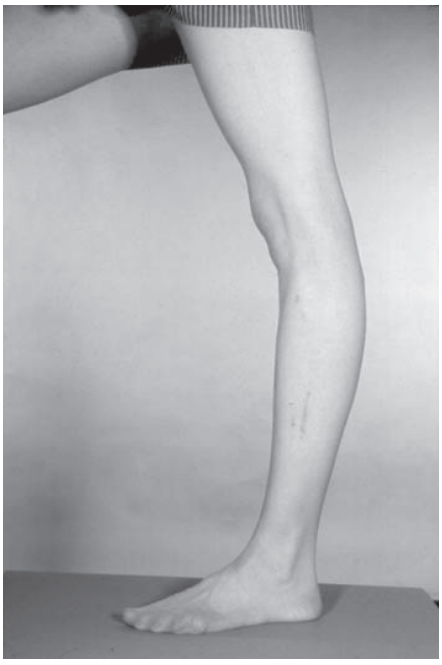


C

Figure 1.5 **A–C**: Examples of hypermobility. Reproduced with permission from Kerr & Grahame (2003).

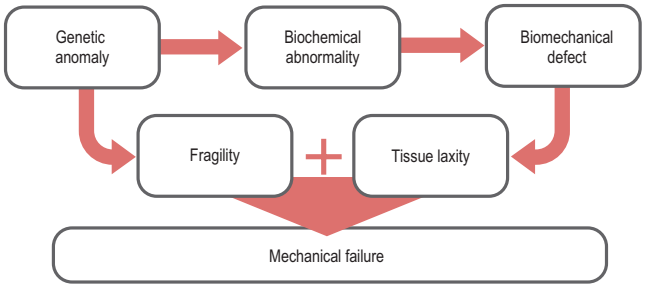


Figure 1.6 Pathophysiology of heritable connective tissue disorders. Reproduced with permission from Kerr & Grahame (2003).

impairments of tensile strength, resulting in enhanced mobility but at a cost of increased fragility, ultimately risking mechanical tissue failure.'

- A number of disorders derive from connective tissue pathophysiology, including Marfan syndrome, Ehlers–Danlos syndrome, osteogenesis imperfecta and joint hypermobility syndrome.
- The commonality of these different syndromes, all resulting from variations of connective tissue laxity, is a tendency toward hypermobility, arthralgia, tendency to dislocation (and possible fracture), osteoporosis, thin skin (and stretch marks), varicose veins, prolapse (rectal, uterine, mitral valve), hernia and diverticulae.
- Hypermobility has been shown to be a major risk factor in the evolution of back pain (Muller et al 2003).
- Hypermobile individuals often present with chronic pain syndromes and an increased tendency to anxiety and panic attacks (Bulbena et al 1993, Martin-Santos et al 1998).
- Hypermobility is more common in people of African, Asian and Arab origin where rates can exceed 30% (as compared with Caucasians ±6%), as well as being more frequently identified in the young compared with the elderly, and in females compared with males (Hakim & Grahame 2003).
- When joints are vulnerable because of hypermobility, passive stretches and end-range positions seem to be able to trigger musculoskeletal symptoms (Russek 2000).
- Patient care requires that patients modify their ergonomics and body mechanics (avoiding overuse and extreme positions) to avoid stretching their joints past end-range during activities of daily living (Russek 2000).
- Trigger point evolution in associated muscles is a common result of the relative laxity of joints (Kerr & Grahame 2003). The authors of this text *hypothesize* that these energy efficient (if painful) entities may offer an efficient means of achieving short-term stability in unstable areas (Chaitow 2000, Chaitow & DeLany 2002, DeLany 2000).
- The implications of this possibility are clear. If myofascial trigger points (MTrPs) are serving functional roles, such as in stabilization of hypermobile joints, deactivation of potentially stabilizing trigger points may ease pain but at the cost of stability (Simons 2002, Thompson 2001). Simons (2002) concurs:

> *In this case it is wise to correct the underlying cause of instability before releasing the MTrP tension. In fact, correcting the underlying instability often results in spontaneous resolution of the MTrP. It is important to identify and remove or modify as many etiological and perpetuating influences as can be found, however, without creating further distress or a requirement for excessive adaptation. It is also important to consider that, at times, apparent symptoms may represent a desirable physiological response (Thompson 2001).*

- A safer alternative is to encourage fitness training, along with the self-use of ice, hydrotherapy and gentle stretching and toning exercises (Goldman 1991). It might also be helpful to selectively deactivate the most painful MTrPs before movement therapies can begin; active movement and, therefore, toning can then be part of the immediate therapy session when the MTrPs are sufficiently reduced.

TRIGGER POINTS, FASCIA AND THE NERVOUS SYSTEM

Changes that occur in connective tissue, and which result in alterations such as thickening, shortening, calcification and erosion, may be a painful result of sudden or sustained tension or traction. Cathie (1974) points out that many trigger points (he calls them trigger 'spots') correspond to points where nerves pierce fascial investments. Hence, sustained tension or traction on the fascia may lead to varying degrees of fascial entrapment of neural structures and consequently a wide range of symptoms and dysfunctions. Neural receptors within the fascia report to the central nervous system as part of any adaptation process, with the pacinian corpuscles being particularly important (these inform the CNS about the rate of acceleration of movement taking place in the area) in terms of their involvement in reflex responses. Other neural input into the pool of activity and responses to biomechanical stress involve fascial structures, such as tendons and ligaments which contain highly specialized and sensitive mechanoreceptors, and proprioceptive reporting stations (see reporting stations, Chapter 3).

Additionally:

- German research has shown that fascia is 'regularly' penetrated (via 'perforations') by a triad of venous, arterial and neural structures (Heine 1995, Staubesand 1996)
- these seem to correspond with fascial perforations previously identified by Heine, which have been correlated (82% correlation) with known acupuncture points (Heine 1995). Further, Bauer & Heine (1998) showed that the triad of perforating neurovascular structures was regularly 'strangulated' by an excessive amount of collagen

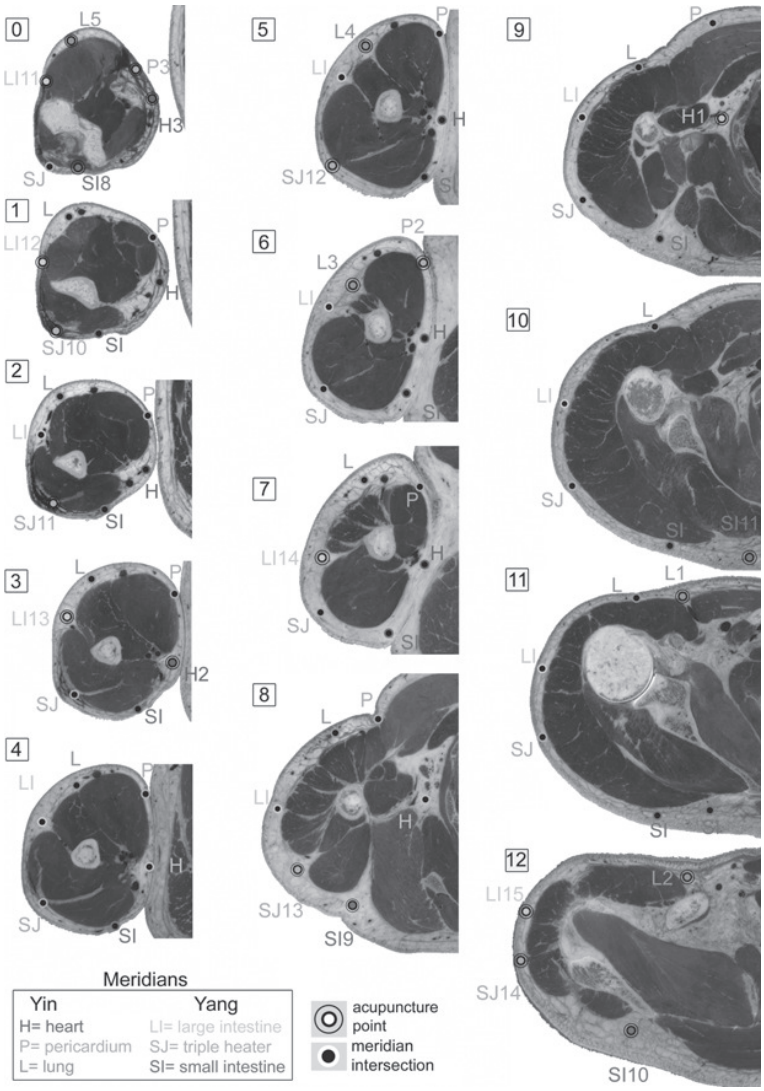


Figure 1.7 Location of acupuncture points and meridians in serial gross anatomical sections through a human arm. Reproduced from Langevin H M, Yandow J A Relationship of acupuncture points and meridians to connective tissue planes. Anatomical Record 269(6):257–265, 2002. Copyright 2002, Wiley-Liss, Inc. Reprinted with permission of Wiley-Liss, Inc., a subsidiary of John Wiley & Sons, Inc.

fibers around these openings in most of the acupoints of the painful region. When those strangulated areas were surgically opened a little, most of the patients experienced significant improvements (i.e. less pain)

- many of these fascial neural structures are sensory and capable of being involved in pain syndromes.

Staubesand states:

> *The receptors we found in the lower leg fascia in humans could be responsible for several types of myofascial pain sensations … Another and more specific aspect is the innervation and direct connection of fascia with the autonomic nervous system. It now appears that the fascial tonus might be influenced and regulated by the state of the autonomic nervous system … intervention in the fascial system might have an effect on the autonomic nervous system, in general, and upon the organs which are directly effected from it. (Schleip 1998)*

THE IMPORTANCE OF LANGEVIN'S RESEARCH

Ongoing research at the University of Vermont has produced remarkable new information regarding the function of fascia/connective tissue (Langevin et al 2001, 2004, 2005). In evaluating the importance of the research information (below) it is important to recall that approximately 80% of common trigger point sites have been claimed to lie precisely where traditional acupuncture points are situated on meridian maps (Wall & Melzack 1990). Indeed, many experts believe that trigger points and acupuncture points are the same phenomenon (Kawakita et al 2002, Melzack et al 1977, Plummer 1980).

Others, however, take a different view. For example, Birch (2003) and Hong (2000) have revisited the original work of Wall & Melzack (1990) and have both found this to be flawed, particularly when the acupuncture points referred to as correlating with trigger points are seen to be 'fixed' anatomically, as on myofascial meridian maps. Both

Birch and Hong agree, however, that so-called '*Ah shi*' acupuncture points may well represent the same phenomenon as trigger points. *Ah shi* points do not appear on the classical acupuncture meridian maps, but refer to 'spontaneously tender' points which, when pressed, create a response in the patient of, 'Oh yes' ('*Ah shi*'). In Chinese medicine *Ah shi* points are treated as 'honorary acupuncture points' and are needled or receive acupressure in the same way as regular acupuncture points, if/when they are tender/painful. This would seem to make them, in all but in name, identical to trigger points.

It is clearly important therefore, in attempting to understand trigger points more fully, to pay attention to current research into acupuncture points and connective tissue in general, as noted in the following research.

Langevin & Yandow (2002) have presented evidence that links the network of acupuncture points and meridians to a network formed by interstitial connective tissue. Using a unique dissection and charting method for location of connective tissue (fascial) planes, acupuncture points and acupuncture meridians of the arm, they note that: 'Overall, more than 80% of acupuncture points and 50% of meridian intersections of the arm appeared to coincide with intermuscular or intramuscular connective tissue planes.'

Langevin & Yandow's research further shows microscopic evidence that when an acupuncture needle is inserted and rotated (as is classically performed in acupuncture treatment), a 'whorl' of connective tissue forms around the needle, thereby creating a tight mechanical coupling between the tissue and the needle. The tension placed on the connective tissue as a result of further movements of the needle delivers a mechanical stimulus at the cellular level. They note that changes in the extracellular matrix '… may, in turn, influence the various cell populations sharing this connective tissue matrix (e.g. fibroblasts, sensory afferents, immune and vascular cells)'.

The key elements of Langevin's research can best be summarized as follows:

- Acupuncture points, and many of the effects of acupuncture, seem to relate to the fact that most of these localized 'points' lie directly over areas where there is fascial cleavage; where sheets of fascia diverge to separate, surround and support different muscle bundles (Langevin et al 2001).
- Connective tissue is a communication system of as yet unknown potential. The tiny projections emerging from each cell are called 'integrins'. Ingber demonstrated (Ingber 1993b, Ingber & Folkman 1989; see Box 1.6) integrins to be a cellular signaling system that modify their function depending on the relative normality of the shape of cells. The structural integrity (shape) of cells depends on the overall state of normality (deformed, stretched, etc.) of the fascia as a whole. As Langevin et al (2004) report:

  > *'Loose' connective tissue forms a network extending throughout the body including subcutaneous and interstitial connective tissues. The existence of a cellular network of fibroblasts within loose connective tissue may have considerable significance as it may support yet unknown body-wide cellular signaling systems … Our findings indicate that soft tissue fibroblasts form an extensively interconnected cellular network, suggesting they may have important, and so far unsuspected integrative functions at the level of the whole body.*

- Perhaps the most fascinating research in this remarkable series of discoveries is that cells change their shape and behavior following stretching (and crowding/deformation). The observation of these researchers is that: 'The

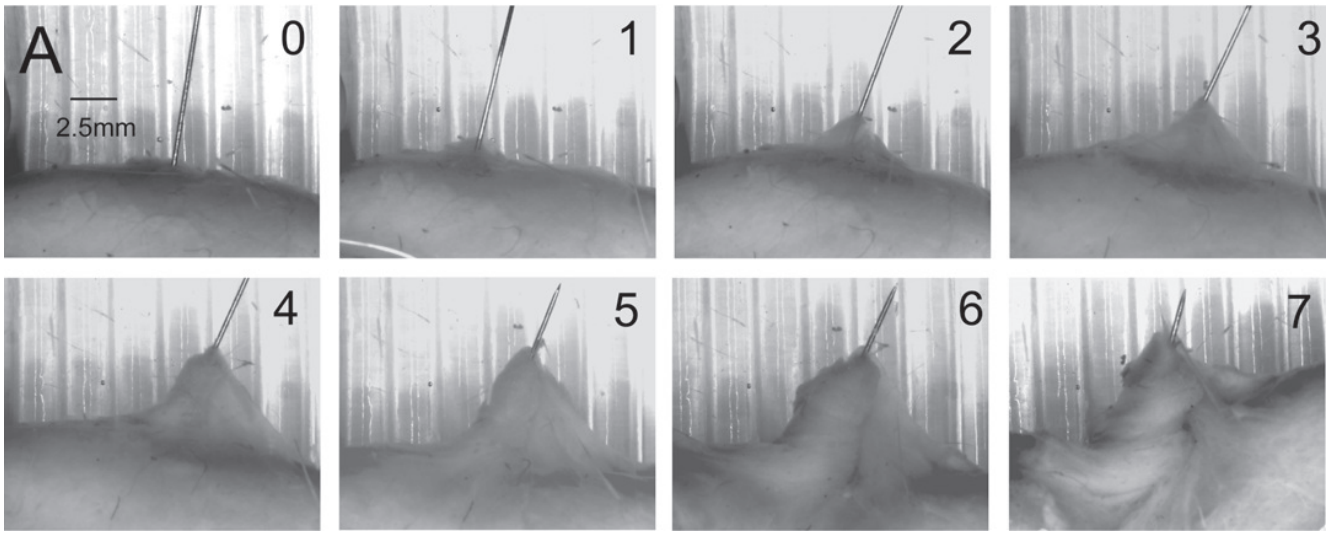


Figure 1.8 Formation of a connective tissue 'whorl' when an acupuncture needle was inserted through the tissue and progressively rotated. Reproduced from Langevin H M, Yandow J A Relationship of acupuncture points and meridians to connective tissue planes. Anatomical Record 269(6): 257–265, 2002. Copyright 2002, Wiley-Liss, Inc. Reprinted with permission of Wiley-Liss, Inc., a subsidiary of John Wiley & Sons, Inc.

dynamic, cytoskeleton-dependent responses of fibroblasts to changes in tissue length demonstrated in this study have important implications for our understanding of normal movement and posture, as well as therapies using mechanical stimulation of connective tissue, including physical therapy, massage and acupuncture' (Langevin et al 2005).

As will become clear, changes in the shape of cells also alter their ability to function normally, even in regard to how they handle nutrients. Ingber conducted research (Ingber 1993a,b, 2003, Ingber & Folkman 1989), much of it for NASA, into the reasons that astronauts lose bone density after a few months in space. He showed that cells deform when gravity is removed or reduced. The behavior of cells changes to the extent that, irrespective of how good the overall nutritional state is, or how much exercise (static cycling in space) is taking place, individual cells cannot process nutrients normally, and problems such as decalcification emerge.

The importance we give to this information should be tied to the awareness that, as we age, adaptive forces cause changes in the structures of the body, with the occurrence of shortening, crowding and distortion. With this, we are seeing in real terms, in our own bodies and those of our patients, the environment in which cells change shape. As they do so they change their potential for normal genetic

Box 1.5 Myers' fascial trains (Myers 1997, 2001)

Tom Myers, a distinguished teacher of structural integration, has described a number of clinically useful sets of myofascial chains. The connections between different structures ('long functional continuities') that these insights allow will be drawn on and referred to when treatment protocols are discussed in this text. They are of particular importance in helping draw attention to (for example) dysfunctional patterns in the lower limb which impact directly (via these chains) on structures in the upper body.

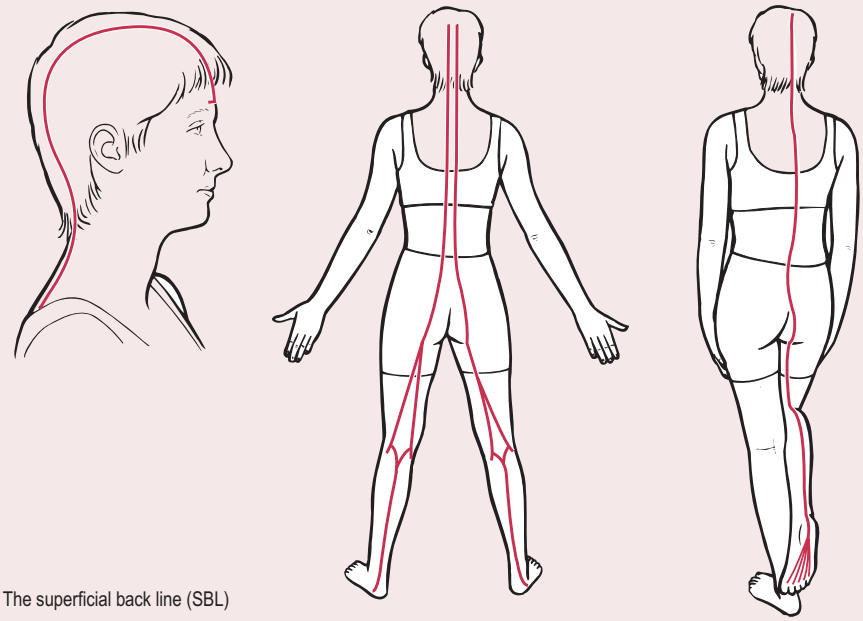
The five major fascial chains

The superficial back line (Fig. 1.9) involves a chain that starts with:

- the plantar fascia, linking the plantar surface of the toes to the calcaneus
- gastrocnemius, linking calcaneus to the femoral condyles
- hamstrings, linking the femoral condyles to the ischial tuberosities
- subcutaneous ligament, linking the ischial tuberosities to sacrum
- lumbosacral fascia, erector spinae and nuchal ligament, linking the sacrum to the occiput
- scalp fascia, linking the occiput to the brow ridge.

The superficial front line (Fig. 1.10) involves a chain that starts with:

- the anterior compartment and the periostium of the tibia, linking the dorsal surface of the toes to the tibial tuberosity
- rectus femoris, linking the tibial tuberosity to the anterior inferior iliac spine and pubic tubercle
- rectus abdominis as well as pectoralis and sternalis fascia, linking the pubic tubercle and the anterior inferior iliac spine with the manubrium
- sternocleidomastoid, linking the manubrium with the mastoid process of the temporal bone.



The superficial back line (SBL)

Figure 1.9 Myers' superficial fascial back line. Reproduced with permission from the *Journal of Bodywork and Movement Therapies* 1997; 1(2):95.

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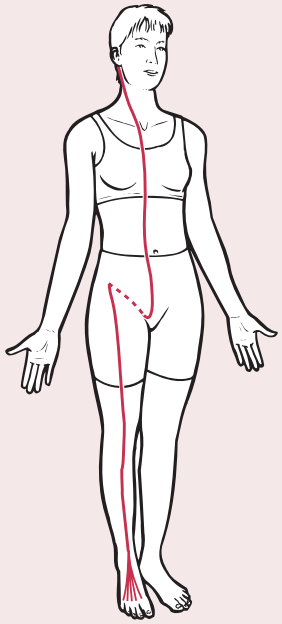
Box 1.5 (continued)



The superficial front line (SFL)

Figure 1.10 Myers' superficial fascial front line. Reproduced with permission from the *Journal of Bodywork and Movement Therapies* 1997; 1(2):97.

The lateral line involves a chain that starts with:

- peroneal muscles, linking the 1st and 5th metatarsal bases with the fibular head
- iliotibial tract, tensor fascia latae and gluteus maximus, linking the fibular head with the iliac crest
- external obliques, internal obliques and (deeper) quadratus lumborum, linking the iliac crest with the lower ribs
- external intercostals and internal intercostals, linking the lower ribs with the remaining ribs
- splenius cervicis, iliocostalis cervicis, sternocleidomastoid and (deeper) scalenes, linking the ribs with the mastoid process of the temporal bone.

The spiral line involves a chain that starts with:

- splenius capitis, which wraps across from one side to the other, linking the occipital ridge (say, on the right) with the spinous processes of the lower cervical and upper thoracic spine on the left
- continuing in this direction, the rhomboids (on the left) link via the medial border of the scapula with serratus anterior and the ribs (still on the left), wrapping around the trunk via the external obliques and the abdominal aponeurosis on the left, to connect with the internal obliques on the right and then to a strong anchor point on the anterior superior iliac spine (ASIS) (right side)
- from the ASIS, the tensor fascia latae and the iliotibial tract link to the lateral tibial condyle
- tibialis anterior links the lateral tibial condyle with the 1st metatarsal and cuneiform
- from this apparent endpoint of the chain (1st metatarsal and cuneiform), peroneus longus rises to link with the fibular head
- biceps femoris connects the fibular head to the ischial tuberosity
- the sacrotuberous ligament links the ischial tuberosity to the sacrum
- the sacral fascia and the erector spinae link the sacrum to the occipital ridge.

The deep front line describes several alternative chains involving the structures anterior to the spine (internally, for example):

- the anterior longitudinal ligament, diaphragm, pericardium, mediastinum, parietal pleura, fascia prevertebralis and the scalene fascia, which connect the lumbar spine (bodies and transverse processes) to the cervical transverse processes and via longus capitis to the basilar portion of the occiput
- other links in this chain might involve a connection between the posterior manubrium and the hyoid bone via the subhyoid muscles and
- the fascia pretrachealis between the hyoid and the cranium/mandible, involving suprahyoid muscles
- the muscles of the jaw linking the mandible to the face and cranium.

Myers includes in his chain description structures of the lower limbs that connect the tarsum of the foot to the lower lumbar spine, making the linkage complete. Additional smaller chains involving the arms are described as follows.

Back of the arm lines

- The broad sweep of trapezius links the occipital ridge and the cervical spinous processes to the spine of the scapula and the clavicle.
- The deltoid, together with the lateral intermuscular septum, connects the scapula and clavicle with the lateral epicondyle.
- The lateral epicondyle is joined to the hand and fingers by the common extensor tendon.
- Another track on the back of the arm can arise from the rhomboids, which link the thoracic transverse processes to the medial border of the scapula.
- The scapula in turn is linked to the olecranon of the ulna by infraspinatus and the triceps.
- The olecranon of the ulna connects to the small finger via the periostium of the ulna.
- A 'stabilization' feature in the back of the arm involves latissimus dorsi and the thoracolumbar fascia, which connects the arm with the spinous processes, the contralateral sacral fascia and gluteus maximus, which in turn attaches to the shaft of the femur.
- Vastus lateralis connects the femur shaft to the tibial tuberosity and (via this) to the periostium of the tibia.

Front of the arm lines

- Latissimus dorsi, teres major and pectoralis major attach to the humerus close to the medial intramuscular septum, connecting it to the back of the trunk.
- The medial intramuscular septum connects the humerus to the medial epicondyle which connects with the palmar hand and fingers by means of the common flexor tendon.
- An additional line on the front of the arm involves pectoralis minor, the costocoracoid ligament, the brachial neurovascular bundle and the fascia clavipectoralis, which attach to the coracoid process.
- The coracoid process also provides the attachment for biceps brachii (and coracobrachialis), linking this to the radius and the thumb via the flexor compartment of the forearm.
- A 'stabilization' line on the front of the arm involves pectoralis major attaching to the ribs, as do the external obliques, which

box continues

Box 1.5 (continued)

then run to the pubic tubercle, where a connection is made to the contralateral adductor longus, gracilis, pes anserinus and the tibial periostium.

In the following chapters' discussions of local dysfunctional patterns involving the cervical, thoracic, shoulder and arm regions, it will be useful to hold in mind the direct muscular and fascial connections that Myers highlights, so that the possibility of distant influences is never forgotten.

Dissection confirmation of fascial continuity (Fig. 1.11)
Barker & Briggs (1999) have shown the lumbodorsal fascia to extend from the pelvis to the cervical area and base of the cranium, in an unbroken sweep: 'Both superficial and deep laminae of the posterior layer are more extensive superiorly than previously thought.'

There is fibrous continuity throughout the lumbar, thoracic and cervical spine and with the tendons of the splenius muscles superiorly.

There is also growing interest in the possible effects that contractile smooth muscle cells (SMC) may have in the many fascial/connective tissue sites in which their presence has now been identified, including cartilage, ligaments, spinal discs and the lumbodorsal fascia (Ahluwalia et al 2001, Hastreiter et al 2001, Meiss 1993, Murray & Spector 1999).

For example, Yahia et al (1993) have observed that: 'Histologic studies indicate that the posterior layer of the (lumbodorsal) fascia is able to contract as if it were infiltrated with muscular tissue.'

Schleip and colleagues (2006) report that: 'Morphological considerations, as well as histological observations in our laboratory, suggest that the perimysium is characterized by a high density of myofibroblasts, a class of fibroblasts with smooth muscle-like contractile kinetics.'

Analysis of 39 tissue samples from the thoracolumbar fascia of 11 human donors (aged 19–76 years) by Schleip et al (2004) demonstrated the widespread presence of myofibroblasts in all samples, with an average density of 79 cells/mm^2 in the longitudinal sections.

Schleip et al (2006) suggest that: 'These findings confirm that fascial tissues can actively contract, and that their contractility appears to be driven by myofibroblasts. The question as to whether or not these active fascial contractions could be strong enough to exert any significant impact on musculoskeletal dynamics has previously been addressed in this journal (Schleip et al 2005) the following way: taking the greatest measured force of in vitro fascial contractions and extrapolating that to an average size of the superficial layer of the thoracolumbar fascia in humans the resulting contraction force can amount to 38 N, which may be a force strong enough to influence biomechanical behaviour, such as in a contribution to paraspinal compartment syndrome or in the prevention of spinal segmental instability.'

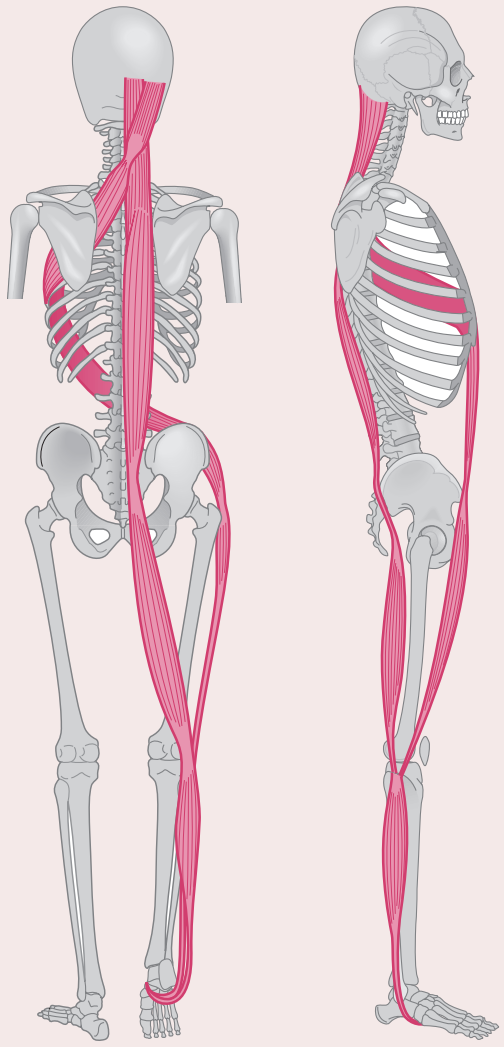


Figure 1.11 A&B: The continuity of vertical and spiral myofascial lines implies a mechanical connection from head to toe. Reproduced with permission from Myers (2001).

expression, as well as their abilities to communicate and to handle nutrients efficiently.

Reversing or slowing these undesirable processes is the potential of appropriate bodywork and movement approaches. It is yet to be precisely established to what degree cellular function can be modified by soft tissue techniques, such as those used in neuromuscular therapy. However, the normalizing of structural and functional features of connective tissue by means of addressing myofascial trigger points, chronic muscle shortening and fibrosis, as well as perpetuating factors such as habits of use, has clear implications. Well-designed research to assess cellular changes that follow the application of manual techniques that offer pain relief and improve function is sorely needed.

SUMMARY OF FASCIAL AND CONNECTIVE TISSUE FUNCTION

Fascia is involved in numerous complex biochemical activities.

- Connective tissue contains a subtle, bodywide signaling system with as yet unknown potentials.

- The fascial cleavage planes appear to be sites of unique sensitivity and of great importance in manual (and acupuncture) therapeutic focus.
- Connective tissue provides a supporting matrix for more highly organized structures and attaches extensively to and invests into muscles.
- Individual muscle fibers are enveloped by endomysium, which is connected to the stronger perimysium that surrounds the fasciculi.
- The perimysium's fibers attach to the even stronger epimysium that surrounds the muscle as a whole and attaches to fascial tissues nearby.
- Because it contains mesenchymal cells of an embryonic type, connective tissue provides a generalized tissue capable of giving rise, under certain circumstances, to more specialized elements.
- It provides (by its fascial planes) pathways for nerves, blood and lymphatic vessels and structures.
- Many of the neural structures in fascia are sensory in nature.
- Fascia supplies restraining mechanisms by the differentiation of retention bands, fibrous pulleys and check ligaments as well as assisting in the harmonious production and control of movement.
- Where connective tissue is loose in texture it allows movement between adjacent structures and, by the formation of bursal sacs, it reduces the effects of pressure and friction.
- Deep fascia ensheaths and preserves the characteristic contours of the limbs and promotes the circulation in the veins and lymphatic vessels.
- The superficial fascia, which forms the panniculus adiposis, allows for the storage of fat and also provides a surface covering that aids in the conservation of body heat.
- By virtue of its fibroblastic activity, connective tissue aids in the repair of injuries by the deposition of collagenous fibers (scar tissue).

Box 1.6 Tensegrity

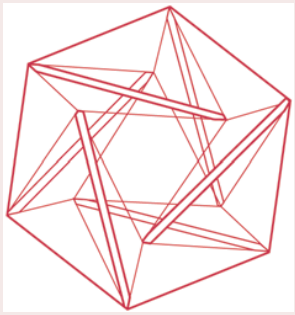
Tensegrity, a term coined by architect/engineer Buckminster Fuller, represents a system characterized by a discontinuous set of compressional elements (struts) which are held together, uprighted and/or moved by a continuous tensional network (Myers 1999, 2001, Oschman 1997, 2000). Fuller, one of the most original thinkers of the 20th century, developed a system of geometry based on tetrahedral (four-sided) shapes found in nature which maximize strength while occupying minimal space (maximum stability with a minimum of materials) (Juhan 1998). From these concepts he designed the geodesic dome, including the US Pavilion at Expo '67 in Montreal.

Tensegrity structures actually become stronger when they are stressed as the load applied is distributed not only to the area being directly loaded but also throughout the structure (Barnes 1990). They employ both compressional and tensional elements. When applying the principles of tensegrity to the human body, one can readily see the bones and intervertebral discs as the discontinuous compressional units and the myofascial tissues (muscles, tendons, ligament, fascia and to some degree the discs) as the tensional elements. When load is applied (as in lifting) both the osseous and myofascial tissues distribute the stress incurred.

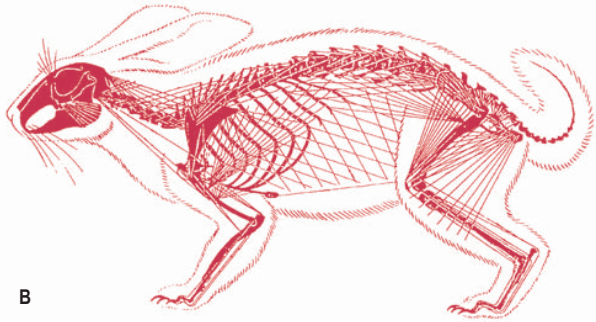
Ingber (1999) concurs with this concept and then adds to it:

In reality, our bodies are composed of 206 compression-resistant bones that are pulled up against the force of gravity and stabilized through interconnection with a continuous series of tensile muscles, tendons, and ligaments ... cells may sense mechanical stresses, including those due to gravity, through changes in the balance of forces that are transmitted across transmembrane adhesion receptors that link the cytoskeleton to the extracellular matrix and to the other cells (e.g. integrins, cadherins, selectins). The mechanism by which these mechanical signals are transduced and converted into a biochemical response appears to be based, in part, on the finding that living cells use a tension-dependent form of architecture, known as tensegrity, to organize and stabilize their cytoskeletons.

Oschman (2000) suggests that bones fit in both the strut and tensile categories, arguing that: 'Bones contain both compressive and tensile fibres, and are therefore tensegrity systems unto themselves.' Tensegrity allows mechanical energy to be transmitted away from the point of impact and to be absorbed throughout the structure. 'The more flexible and balanced the network (the better the tensional integrity), the more readily it absorbs shocks and converts them to *information* rather than *damage*.'



A



B

Figure 1.12 A&B: Tensegrity-based structures. Reproduced with permission from the *Journal of Bodywork and Movement Therapies* 1997; 1(5):300–302.

box continues

Box 1.6 (continued)

Regarding Ingber's work, Oschman (2000) points out that the living tensegrity network is not only a mechanical system, but also a vibratory continuum. When a part of a tensegrity structure is plucked, the vibration produced travels throughout the entire structure:

Restrictions in one part have both structural and energetic consequences for the entire organism. Structural integrity, vibratory integrity, and energetic or information integrity go hand in hand. One cannot influence the structural system without influencing the energetic/informational system, and vice versa. Ingber's work shows how these systems also interdigitate with biochemical pathways.

Of tensegrity, Juhan (1998) tells us:

Besides this hydrostatic pressure (which is exerted by every fascial compartment, not just the outer wrapping), the connective tissue framework – in conjunction with active muscles – provides another kind of tensional force that is crucial to the upright structure of the skeleton. We are not made up of stacks of building blocks resting securely upon one another, but rather of poles and guy-wires, whose stability relies not upon flat stacked surfaces, but upon proper angles of the poles and balanced tensions on the wires. ... There is not a single horizontal surface anywhere in the skeleton that provides a stable base for anything to be stacked upon it. Our design was not conceived by a stone-mason. Weight applied to any bone would cause it to slide right off its joints if it were not for the tensional balances that hold it in place and control its pivoting. Like the beams in a simple tensegrity structure, our bones act more as spacers than as compressional members; more weight is actually borne by the connective system of cables than by the bony beams.

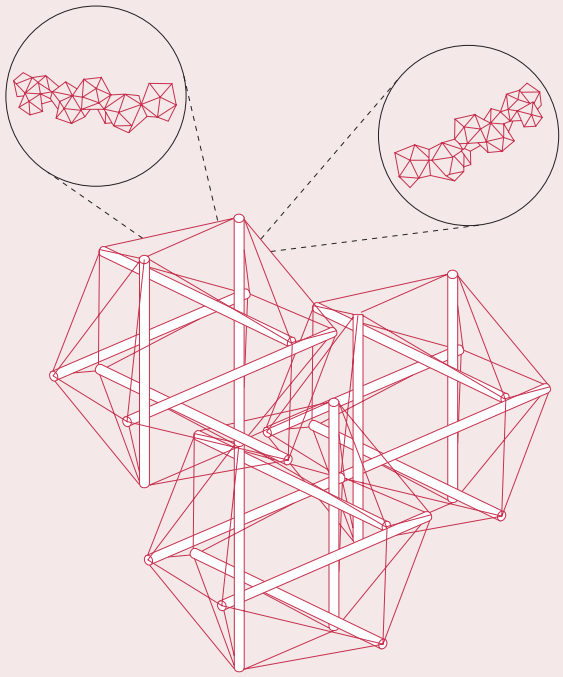


Figure 1.13 Tensegrity-based structures.

Oschman (1997) concurs, adding another element:

Robbie (1977) reaches the remarkable conclusion that the soft tissues around the spine, when under appropriate tension, can actually lift each vertebra off the one below it. He views the spine as a tensegrity mast. The various ligaments form 'slings' that are capable of supporting the weight of the body without applying compressive forces to the vertebrae and intervertebral discs. In other words, the vertebral column is not, as it is usually portrayed, a simple stack of blocks, each cushioned by an intervertebral disc.

These views are also suggested by Myers (2001) in his enlightening book, *Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists* (see also Box 1.4).

Later Oschman continues:

Cells and nuclei are tensegrity systems (Coffey 1985, Ingber & Folkman 1989, Ingber & Jamieson 1985). Elegant research has documented how the gravity system connects, via a family of molecules known as integrins, to the cytoskeletons of cells throughout the body. Integrins 'glue' every cell in the body to neighbouring cells and to the surrounding connective tissue matrix. An important study by Wang et al (1993) documents that integrin molecules carry tension from the extracellular matrix, across the cell surface, to the cytoskeleton, which behaves as a tensegrity matrix. Ingber (1993a,b) has shown how cell shape and function are regulated by an interacting tension and compression system within the cytoskeleton.

Levin (1997) informs us that once spherical shapes involving tensegrity structures occur (as in the cells of the body), a many-sided framework evolves which has 20 triangular faces. This is the hierarchically constructed tensegrity icosahedron (*icosa* is 20 in Greek) which are stacked together to form an infinite number of tissues.

Levin (1997) further explains architectural aspects of tensegrity as it relates to the human body. He discusses the work of White & Panjabi (1978) who have shown that any part of the body which is free to move in any direction has 12 degrees of freedom: the ability to rotate around three axes, in each direction (six degrees of freedom) as well as the ability to translate on three planes in either direction (a further six degrees of freedom). He then asks, how is this stabilized?

To fix in space a body that has 12 degrees of freedom it seems logical that there need to be 12 restraints. Fuller (1975) proves this ... This

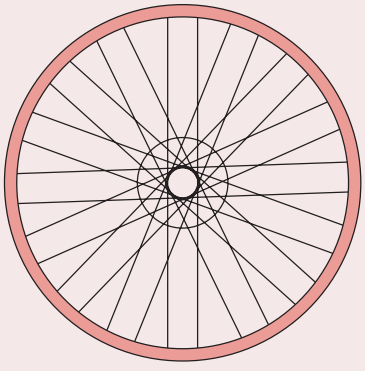


Figure 1.14 Cycle wheel structure allows compressive load to be distributed to rim through tension network.

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Box 1.6 (continued)

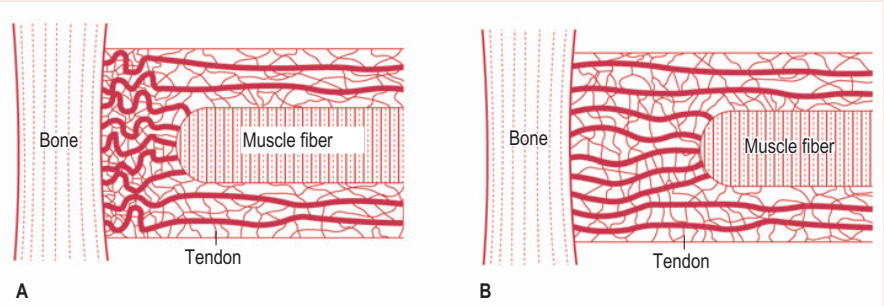

Figure 1.15 **A**: Dehydration of ground substance may cause kinking of collagen fibers. **B**: Sustained pressure may result in temporary solation of ground substance, allowing kinked collagen fibers to lengthen, thereby reducing muscular strain. Reproduced with permission from the *Journal of Bodywork and Movement Therapies* 1997; 1(5):309.

principle is demonstrated in a wire-spoked bicycle wheel. A minimum of 12 tension spokes rigidly fixes the hub in space (anything more than 12 is a fail safe mechanism).

Levin points out that the tension-loaded spokes transmit compressive loads from the frame to the ground while the hub remains suspended in its tensegrity network of spokes: 'the load distributes evenly around the rim and the bicycle frame and its load hangs from the hubs like a hammock between trees'.

Other examples of tensegrity in common use include a tent and a crane. In the body this architectural principle is seen in many tissues, most specifically in the way the sacrum is suspended between the ilia.

- The ensheathing layer of deep fascia, as well as intermuscular septa and interosseous membranes, provides vast surface areas used for muscular attachment.
- The meshes of loose connective tissue contain the 'tissue fluid' and provide an essential medium through which the cellular elements of other tissues are brought into functional relation with blood and lymph.
- This occurs partly by diffusion and partly by means of hydrokinetic transportation encouraged by alterations in pressure gradients – for example, between the thorax and the abdominal cavity during inhalation and exhalation.
- Connective tissue has a nutritive function and houses nearly a quarter of all body fluids.
- Fascia is a major arena of inflammatory processes (Cathie 1974) (see Chapter 7).
- Fluids and infectious processes often travel along fascial planes (Cathie 1974).
- Chemical (nutritional) factors influence fascial behavior directly. Pauling (1976) showed that 'Many of the results of deprivation of ascorbic acid [vitamin C] involve a deficiency in connective tissue which is largely responsible for the strength of bones, teeth, and skin of the body and which consists of the fibrous protein collagen'.
- The histiocytes of connective tissue comprise part of an important defense mechanism against bacterial invasion by their phagocytic activity.
- They also play a part as scavengers in removing cell debris and foreign material.
- Connective tissue represents an important 'neutralizer' or detoxicator to both endogenous toxins (those produced under physiological conditions) and exogenous toxins.
- The mechanical barrier presented by fascia has important defensive functions in cases of infection and toxemia.
- Fascia, then, is not just a background structure with little function apart from its obvious supporting role, but is an ubiquitous, tenacious, living tissue that is deeply involved in almost all of the fundamental processes of the body's structure, function and metabolism.
- In therapeutic terms, there can be little logic in trying to consider muscle as a separate structure from fascia since they are so intimately related.
- Remove connective tissue from the scene and any muscle left would be a jelly-like structure without form or functional ability.

FASCIAL DYSFUNCTION

Mark Barnes (1997) states:

> *Fascial restrictions can create abnormal strain patterns that can crowd, or pull the osseous structures out of proper alignment, resulting in compression of joints, producing pain and/or dysfunction. Neural and vascular structures can also become entrapped in these restrictions, causing neurological or ischemic conditions. Shortening of the myofascial fascicle can limit its functional length – reducing its strength, contractile potential and deceleration capacity. Facilitating positive change in this system [by therapeutic intervention] would be a clinically relevant event.*

Cantu & Grodin (1992) have stated that 'The response of normal connective tissue [fascia] to immobilization provides a basis for understanding traumatized conditions'.

A sequence of dysfunction has been demonstrated as follows (Akeson & Amiel 1977, Amiel & Akeson 1983, Evans 1960).

- The longer the immobilization, the greater the amount of infiltrate there will be.

- If immobilization continues beyond about 12 weeks, collagen loss is noted; however, in the early days of any restriction, a significant degree of ground substance loss occurs, particularly glycosaminoglycans and water. Loss of (47% of) muscle strength due to immobilization has been shown to occur in as little as 3 weeks (Hortobágyi et al 2000).
- Since one of the primary purposes of ground substance is the lubrication of the tissues it separates (collagen fibers), its loss leads inevitably to the distance between these fibers being reduced.
- Loss of interfiber distance impedes the ability of collagen to glide smoothly, encouraging adhesion development.
- This allows crosslinkage between collagen fibers and newly formed connective tissue, which reduces the degree of fascial extensibility as adjacent fibers become more and more closely bound.
- Because of immobility, these new fiber connections will not have a stress load to guide them into a directional format and they will be laid down randomly.
- Similar responses are observed in ligamentous as well as periarticular connective tissues.
- Mobilization of the restricted tissues can reverse the effects of immobilization as long as this has not been for an excessive period.
- If, due to injury, inflammatory processes occur as well as immobilization, a more serious evolution occurs, as inflammatory exudate triggers the process of contracture, resulting in shortening of connective tissue.
- This means that, following injury, two separate processes may be occurring simultaneously: there may be a process of scar tissue development in the traumatized tissues and also fibrosis in the surrounding tissues (as a result of the presence of inflammatory exudate).
- Cantu & Grodin (1992) give an example: 'A shoulder may be frozen due to macroscopic scar adhesion in the folds of the inferior capsule … a frozen shoulder may also be caused by capsulitis, where the entire capsule shrinks.'
- Capsulitis could therefore be the result of fibrosis involving the entire fabric of the capsule, rather than a localized scar formation at the site of injury.

Noted author Rene Cailliet (2004) points out that the viscoelastic properties of collagen are influence by temperature, 'which, when added to the equation of force and speed of stress, may cause irrecoverable damage'. Prolonged immobilization results in a number of alterations in tissue, including failure of collagen fibers to physiologically elongate and loss of collagen strength in as little as 4 weeks.

RESTORING GEL TO SOL

Mark Barnes (1997) insists that therapeutic methods that try to deal with this sort of fascial, connective tissue change (summarized above in relation to trauma or immobilization) would be to 'elongate and soften the connective tissue, creating permanent three-dimensional depth and width'.

To achieve this, he says:

> *Most important is the change in the ground substance from a gel to a sol. This occurs with a state phase realignment of crystals exposed to electromagnetic fields. This may occur as a piezoelectric event (changing a mechanical force to electric energy) which changes the electrical charge of collagen and proteoglycans within the extracellular matrix.*

In offering this opinion Barnes is basing his comments on the research evidence relating to connective tissue behavior which takes the properties of fascia into an area of study involving liquid crystal and piezoelectric events (Athenstaedt 1974, Pischinger 1991). Appropriately applied manual therapy can, Barnes suggests, often achieve such changes, whether this involves stretching, direct pressure, myofascial release or other approaches. As noted earlier, much that changes can be seen to possibly involve the 'sponge-like' behavior of connective tissues as they extrude and absorb water. All these elements form part of neuromuscular therapy interventions.

A DIFFERENT MODEL LINKING TRAUMA AND CONNECTIVE TISSUE

Discussion of trauma and connective tissue has focused thus far on the physical changes that evolve, and the adaptations and compensations that are often amenable to soft tissue therapeutic interventions.

Oschman (2006) offers a different perspective, which may be seen to build on the observations above on the work of Langevin, since both conceive connective tissue as (amongst other things) a communication network. Oschman summarizes this hypothesis as follows:

> *The hypothesis is that the connective tissue matrix and its extensions reaching into every cell and nucleus in the body is a whole-person physical system that senses and absorbs the physical and emotional impact in any traumatic experience. The matrix is also the physical material that is influenced by virtually all hands-on, energetic and movement therapies. It is suggested that the living [connective tissue] matrix is the physical substrate where traumatic memories are stored and resolved.*

Oschman continues:

> *The living matrix is a pervasive system, consisting of both the nerves and the connective tissues and cytoskeletons of every neural and non-neural cell in the body. On the basis of the known biophysical properties of this system, we can visualize this as a high-speed solid-state information processor with capabilities that far exceed the brightest minds and fastest computers. Intuition can therefore be described as an emergent property of a very sophisticated semiconducting liquid crystalline molecular matrix that is capable of storing, processing and communicating a vast amount of subliminal information that never reaches the nervous system*

Box 1.7 Postural (fascial) patterns (Zink & Lawson 1979)

Zink & Lawson have described patterns of postural patterning determined by fascial compensation and decompensation.

- Fascial compensation is seen as a useful, beneficial and, above all, functional adaptation (i.e. no obvious symptoms) on the part of the musculoskeletal system, for example, in response to anomalies such as a short leg, or to overuse.
- Decompensation describes the same phenomenon but only in relation to a situation in which adaptive changes are seen to be dysfunctional, to produce symptoms, evidencing a failure of homeostatic adaptation.

By testing the tissue 'preferences' in different areas it is possible to classify patterns in clinically useful ways:

- *ideal* (minimal adaptive load transferred to other regions)
- *compensated* patterns which alternate in direction from area to area (e.g. atlantooccipital, cervicothoracic, thoracolumbar, lumbosacral) and which are commonly adaptive in nature
- *uncompensated* patterns which do not alternate and which are commonly the result of trauma.

Functional evaluation of fascial postural patterns

Zink & Lawson (1979) have described methods for testing tissue preference.

- There are four crossover sites where fascial tensions can be noted: occipitoatlantal (OA), cervicothoracic (CT), thoracolumbar (TL) and lumbosacral (LS).
- These sites are tested for their rotation and side-bending preferences.
- Zink & Lawson's research showed that most people display alternating patterns of rotatory preference with about 80% of people showing a common pattern of left-right-left-right (termed the common compensatory pattern or CCP) 'reading' from the occipitoatlantal region downwards.
- Zink & Lawson observed that the 20% of people whose compensatory pattern did not alternate had poor health histories.
- Treatment of either CCP or uncompensated fascial patterns has the objective of trying, as far as is possible, to create a symmetrical degree of rotatory motion at the key crossover sites.
- The treatment methods used to achieve this range from direct muscle energy approaches to indirect positional release techniques.

Assessment of tissue preference

Occipitoatlantal area (Fig. 1.16)

- Patient is supine.
- Practitioner sits at head, and cradles upper cervical region.
- The neck is fully flexed.
- The occiput is rotated on the atlas to evaluate tissue preference as the head is slowly rotated left and then right.

Cervicothoracic area (Fig. 1.17)

- Patient is seated in relaxed posture with practitioner behind, with hands placed to cover medial aspects of upper trapezius so that fingers rest over the clavicles.

Figure 1.16 Alternative hand positions for assessment of upper cervical region tissue direction preference.

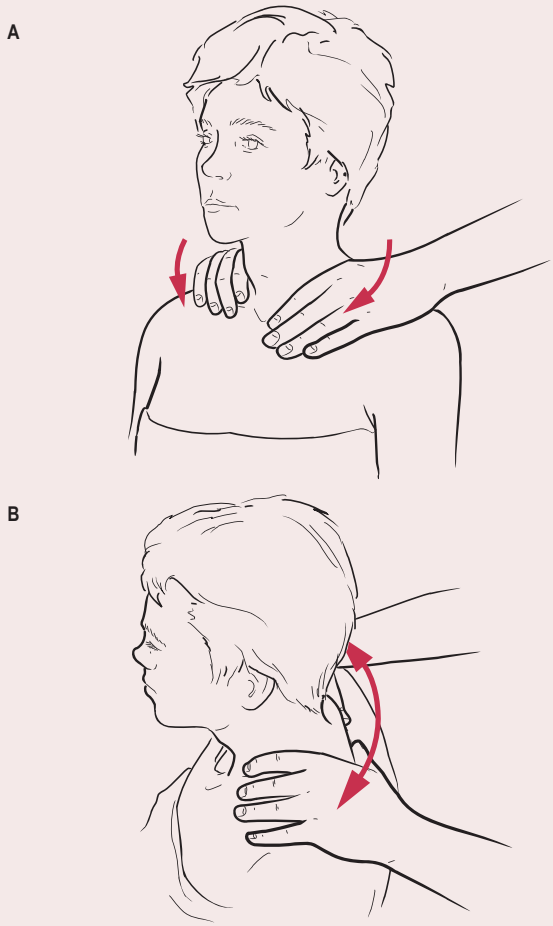

Figure 1.17 A&B: Hand positions for assessment of upper cervicothoracic region tissue direction preference.

box continues

Box 1.7 (continued)

- The hands assess the area being palpated for its 'tightness/looseness' preferences as a slight degree of rotation left and then right is introduced at the level of the cervicothoracic junction.

Thoracolumbar area

- Patient is supine, practitioner stands at waist level facing cephalad and places hands over lower thoracic structures, fingers along lower rib shafts laterally.
- Treating the structure being palpated as a cylinder, the hands test the preference the lower thorax has to rotate around its central axis, one way and then the other.

Lumbosacral area

- Patient is supine, practitioner stands below waist level facing cephalad and places hands on anterior pelvic structures, using the contact as a 'steering wheel' to evaluate tissue preference as the pelvis is rotated around its central axis while seeking information as to its 'tightness/looseness' preferences.

NOTE: By holding tissues in their 'loose' or ease positions, by holding tissues in their 'tight' or bind positions and introducing an isometric contraction or just by holding tissues at their barrier, waiting for a release, changes can be encouraged. The latter approach would be inducing the myofascial release in response to light, sustained load.

Questions following assessment exercise:

1. Was there an 'alternating' pattern to the tissue preferences?
2. Or was there a tendency for the tissue preference to be the same in all or most of the four areas assessed?
3. If the latter was the case, was this in an individual whose health is more compromised than average – in line with Zink & Lawson's suggestion?
4. By means of any of the methods suggested in the 'Note' above, are you able to produce a more balanced degree of tissue preference?

and consciousness directly. A computer, with its software programs and memory and information storage capacities pales to insignificance in comparison with the evolutionarily ancient solid-state system that is expressed within every cell and sinew of the body.

Since the primary channels of this informational system are the acupuncture meridians, it is not surprising that there are energy psychology methods that involve tapping on key points on the meridian system. Such tapping will introduce electrical fields into the meridian system because of the piezoelectric or pressure-electricity effect (e.g. Lapinski 1977, MacGinitie 1995). Such currents, then, will be transduced into signals that will be propagated through the meridian/living matrix system for a certain distance, since the meridians are low resistance pathways to the flow of electricity (e.g. Reichmanis et al 1975).

THERAPEUTIC SEQUENCING

Cantu & Grodin (1992) conclude that therapeutic approaches which sequence their treatment protocols to involve the superficial tissues (involving autonomic responses) as well as deeper tissues (influencing the mechanical components of the musculoskeletal system) and that also address the factor of mobility (movement) meet with the requirements of the body when dysfunctional problems are being treated. NMT, as presented in this text, adopts this comprehensive approach and achieves at least some of its beneficial effects because of its influence on fascia.

In the upcoming chapters we will see how influences from the nervous system, inflammatory processes and patterns of use affect (and are affected by) the fascial network. In the second volume of this text, the principles of tensegrity, thixotropy and postural balance will be seen to form an intricate part of the foundations of whole-body structural integrity. As will become clear in the next chapter, Ingber (2003) now tends to use the term 'structural continuum' as an advance on the tensegrity model, wherein the entire body and all its myriad structures are seen to be interdependently enmeshed. The authors of this text believe that an understanding of these different ways of appreciating the structures of the body is a foundation for the use of therapeutic bodywork methods.

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Chapter 2

Muscles

CHAPTER CONTENTS

In this chapter our focus of attention is placed on the prime movers and stabilizers of the body, the muscles. It is necessary to understand those aspects of muscle structure, function and dysfunction that can help to make selection and application of therapeutic interventions as suitable and effective as possible. Unless otherwise noted, the general muscle discussions in this chapter refer to skeletal muscles.

The skeleton provides the body with an appropriately semirigid framework that has facility for movement at its junctions and joints. However, it is the muscular system, given cohesion by the fascia (see Chapter 1), that both supports and propels this framework, providing us with the ability to express ourselves through movement, in activities ranging from chopping wood to brain surgery, climbing mountains to giving a massage. Almost everything, from facial expression to the beating of the heart, is dependent on muscular function.

Synchronized and coordinated movement depends on structural integration, in which the form of the body parts, and how they interrelate spatially, from the smallest to the largest, determines the efficiency of function. It is in this complex setting that muscle function (and dysfunction) should be seen.

DYNAMIC FORCES – THE 'STRUCTURAL CONTINUUM'

It may be useful to qualify the description above, in which a division is suggested between the semirigid skeleton and the attaching elastic soft tissues that propel and move it. In fact, the integrated systems of the body are better described as representing a series of interrelated tensegrity structures.

It was Fuller (1975) who used the term tensegrity to describe structures whose stability, or *tensional integrity*, required a dynamic balance between discontinuous compression elements (such as bones) connected (and moved) by continuous tension cables (such as the soft tissues of the body, e.g. ligaments, tendons, muscle and fascia). There

Figure 2.1 The miraculous possibilities of human balance. Reproduced with permission from *Gray's Anatomy* (1995).

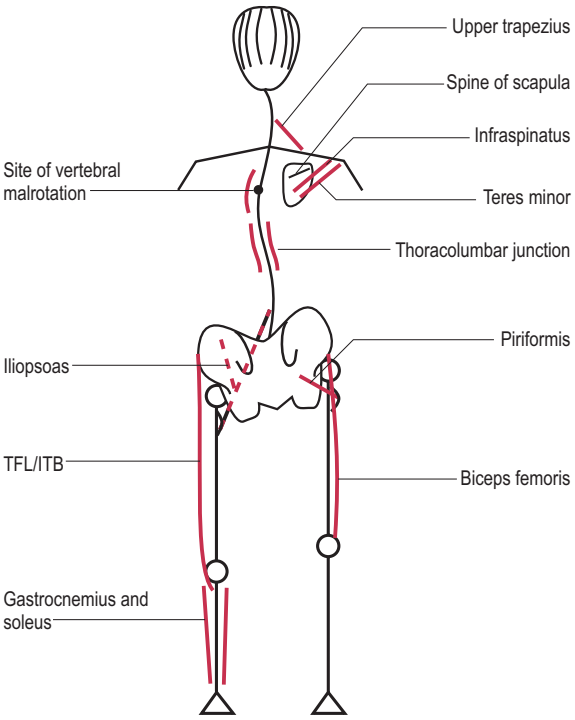

Figure 2.2 Typical sites of increased muscle/tendon tension and tenderness resulting from malalignment. The drawing also indicates the typical lateralization; if the structure is involved bilaterally, the one indicated here is usually affected more severely. TFL/ITB, tensor fascia lata/iliotibial band. Redrawn with permission from Schamberger (2002).

was, in this construct, the implied balance created between *tension* and *compression*, involving all tissues, from an intra- and extracellular level, to the gross skeletal and muscular structures of the physical body (Ingber 1993, 2003).

Ingber (2003) has, in fact, moved beyond the tensegrity model in his descriptions, having more recently discussed what he terms a 'structural continuum', in which everything from the macro (skeleton, muscles, organs, etc.) to the micro (intra- and extracellular structures) are interdependently enmeshed. Ingber summarizes this when he states: 'Mechanical deformation of whole tissues [the outcome of the interaction between tensional, shear and compression forces] results in coordinated structural re-arrangements on many different size scales.'

He uses the word *mechanotransduction* to summarize the effects of shear and other forces on cells, which change their shape and function, including gene expression. These processes occur in tissues that have been, or are being, over- or underused, or abused. This implies that functional misuse (poor posture, for example) leads to structural modifications, and that once such structural rearrangements have occurred, normal (or at least optimal) function may become impossible.

The interlocking elements of structure, function and dysfunction are the territory of the manual therapist, as we evaluate in our patients these processes of 'coordinated structural rearrangement' that are capable of affecting *all* tissues, including neural, fascial and muscular. The end results of such 'rearrangement' will be noted when a muscle is found to be shortened, fibrotic or to contain trigger points. These symptom-producing changes (reduced range of motion, tense, tight and/or indurated muscles that may be housing trigger points) are the manifestation of rearrangement of the structural continuum. An example of a 'rearranged' structure is given by Schamberger (2002) who describes an example of what he terms a 'malalignment syndrome' (Fig. 2.2). In this example rotational and other malalignments are seen to cause increased muscular tensions and corresponding adaptations.

Fortunately, 'coordinated structural rearrangement' in a positive direction is also possible, when appropriate therapeutic measures are initiated to help restore the 'structural continuum', offering the chance for function to improve, or

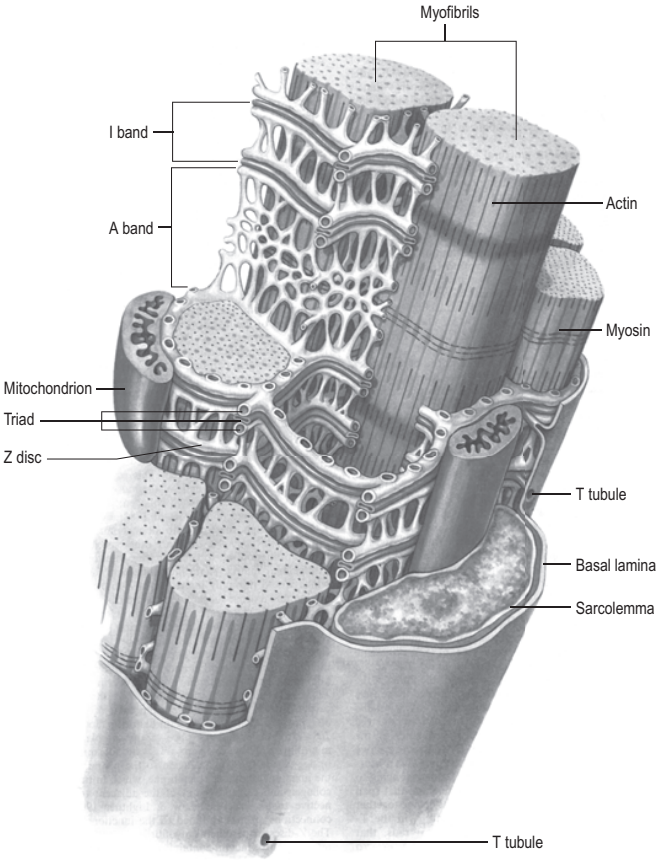

Figure 2.3 Details of the intricate organization of skeletal muscle. Reproduced with permission from *Gray's Anatomy* (2005).

normalize. It is within this context that you should consider our survey of fascia (Chapter 1) and muscles (this chapter) and the dysfunctions that are described and the treatments proposed throughout the book.

SIGNALS

Healthy, well-coordinated muscles receive and respond to a multitude of signals from the nervous system, providing the opportunity for coherent movement. When, through overuse, misuse, abuse, disuse, disease or trauma, the smooth interaction between the nervous, circulatory and the musculoskeletal systems is disturbed, movement becomes difficult, restricted, commonly painful and, sometimes, impossible. Dysfunctional patterns affecting the musculoskeletal system (see Chapter 5) which emerge from such a background lead to compensatory adaptations and a need for therapeutic, rehabilitation and/or educational interventions. This chapter will highlight some of the unique qualities of the muscular system. On this foundation it will be possible to commence exploration of the many dysfunctional patterns that can interfere with the quality of life and create painful modifications of the framework, thus leading to degenerative changes.

Because the anatomy and physiology of muscles are adequately covered elsewhere, the information in this chapter will be presented largely in summary form. Some specific topics (muscle type, for example) receive a fuller discussion due to the significance they have in regard to neuromuscular therapy.

ESSENTIAL INFORMATION ABOUT MUSCLES (Fritz 1998, Jacob & Falls 1997, Lederman 1997, Liebenson 1996, MacIntosh et al 2006, Schafer 1987)

- Skeletal muscles are derived embryologically from mesenchyme and possess a particular ability to contract when neurologically stimulated.
- Skeletal muscle fibers comprise a single cell with hundreds of nuclei.
- The fibers are arranged into bundles (fasciculi) containing approximately 100 fibers, with connective tissue filling the spaces between the fibers (the endomysium) as well as surrounding the fasciculi (the perimysium).
- Entire muscles are surrounded by denser connective tissue (fascia, see Chapter 1) where it is known as the epimysium.
- The epimysium is continuous with the connective tissue of surrounding structures.
- Individual muscle fibers, which are bundles of 1000–2000 myofibrils, can vary in length from a few millimeters to about 12 cm. When a muscle appears to be longer than this, it has fibers arranged in series, separated into compartments by inscriptions. The sartorius, for instance, has three such inscriptions (four compartments), with each compartment having its own nerve supply (MacIntosh et al 2006).
- Individual muscle fibers can vary in diameter from 10 to 60 μm, with most adult fibers being around 50 μm.
- Individual myofibrils are composed of a series of sarcomeres, the basic contractile units of a skeletal muscle, connected end to end. Actin and myosin filaments overlap within the sarcomere and slide in relation to one another to produce shortening of the muscle (see Box 2.1).

TYPES OF MUSCLE

Muscle fibers can be broadly grouped into those that are:

- *longitudinal* (or *strap* or *parallel* or *fusiform*), which have lengthy fascicles, largely oriented with the longitudinal axis of the body or its parts. These fascicles favor speedy action and are usually involved in range of movement (sartorius, for example, or biceps brachii)

Box 2.1 Muscle contractile mechanics and the sliding filament theory

Striated (skeletal) muscles are composed of fasciculi, the number of which is dependent upon the size of the muscle. Each fascicle is made up of bundles of (approximately) 100 fibers with each fiber containing up to around 2000 myofibrils (MacIntosh et al 2006, Simons et al 1999). Each myofibril is composed of a series of sarcomeres laid end to end; these contain two primary types of protein filament, actin and myosin, as well as a stabilizing filament (titin) and other proteins, such as troponin, tropomyosin and nebulin. In most anatomy books the reader can easily find illustrations and discussions regarding the distinct bands and shadings, such as the Z-line, H-zone and M-region, which are created by the myofibril components.

The sliding filament theory, first proposed by biophysicist Jean Hanson and physiologist Hugh Esmor Huxley in 1954, offers an explanation of how muscles shorten during contraction. Although scientists have failed to fully explain the biomechanics of movement, the sliding filament theory remains today as the foundational platform. The following illustrates the basis of this theory.

Figure 2.4 illustrates the relationship of actin, myosin and other components of the muscle cell during contraction. As ATP binds to the myosin heads (which form the crossbridges between the two filaments), it partially hydrolyzes them to produce an energized (pre-cocked) myosin head. This preloaded thick filament has a high affinity for the thinner actin component. When a muscle is at rest, binding of the two filaments must be blocked or else continual contraction will result, such as seen in rigor mortis. The tropomyosin filament overlies the myosin binding sites on the actin molecule, thereby preventing coupling of the two filaments.

As an action potential spreads across the muscle fiber, signaling contraction, it travels down the transverse tubules, which lie close to the terminal cisternae (lateral sacs), the storage site for Ca^{2+}. As the action potential progresses, it causes a depolarization of the membrane, an opening of the calcium channels and the release of Ca^{2+} from the sarcoplasmic reticulum.

The release of Ca^{2+} catalyzes troponin to change its shape, thereby moving tropomyosin aside. This process exposes the binding sites on the actin molecule and allows myosin to attach itself to the actin filaments. This occurs to many filaments simultaneously, not just the one described here. The myosin heads (and possibly shafts) flex, causing numerous myosin and actin filaments to slide past each other, resulting in muscle contraction.

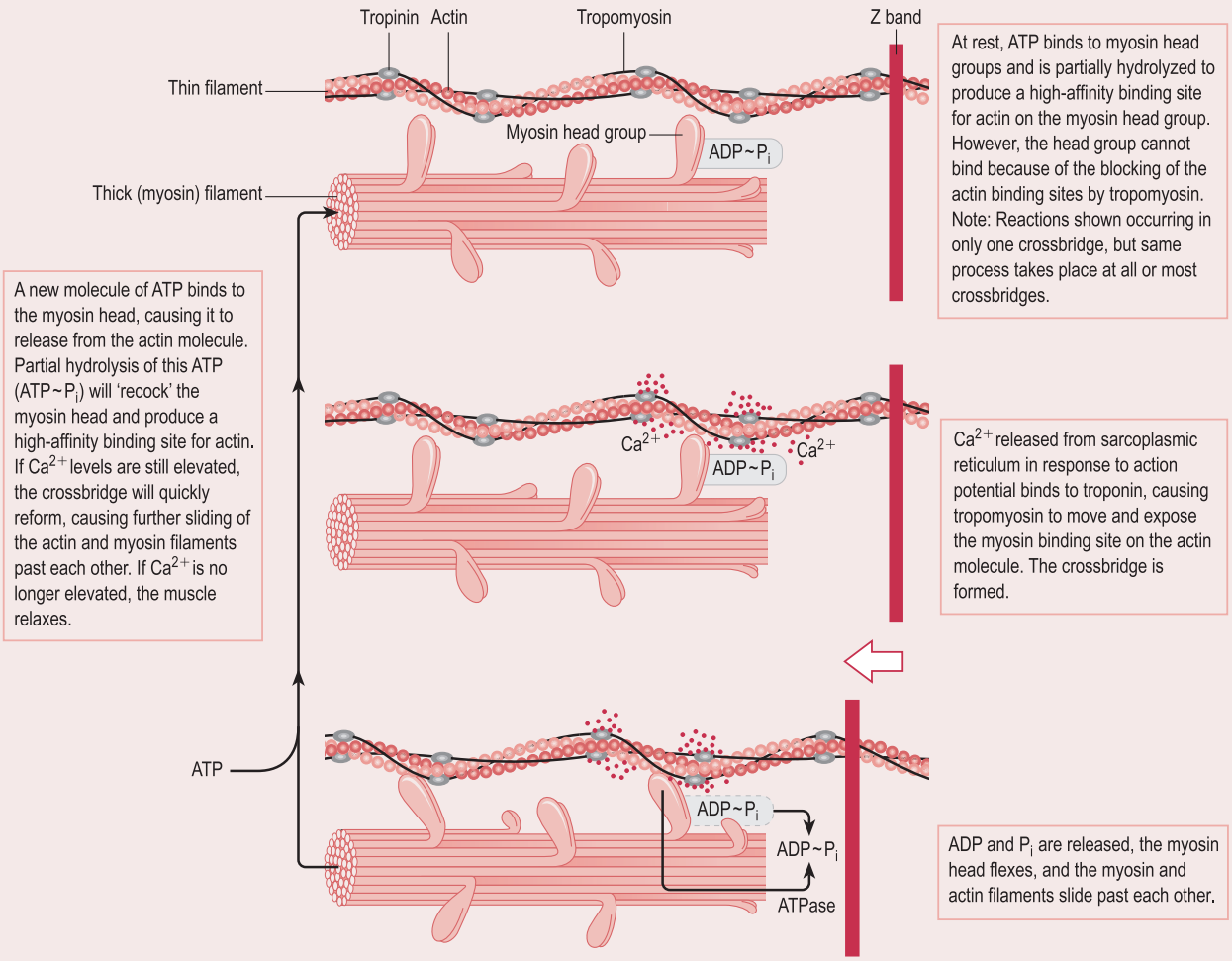

Figure 2.4 The contraction of the myofilaments results from the interaction of actin and myosin. Redrawn after Hansen & Koeppen (2002).

box continues

Box 2.1 (continued)

Once this occurs, the myosin loses its energy and remains bonded to the actin until it is re-energized with ATP. In other words, the ATP *unlocks* the myosin head and preloads it for the next cycle. However, the absence of adequate ATP and the presence of Ca^{2+} can cause the filaments to remain in a shortened position for an indefinite period of time.

After the contraction is completed, if adequate ATP is available, the myosin can be detached, the Ca^{2+} can be actively transported back into the terminal cisternae of the sarcoplasmic reticulum, thereby allowing the tropomyosin to slide back into place and cover the actin-reactive sites. Muscle fiber relaxation occurs.

For best results (maximal force output and functional shortening) the filaments should begin at normal resting length, neither overapproximated nor overstretched. This will allow the maximal number of myosin heads to be used. Adequate ATP is needed for myosin energy and Ca^{2+} must be available as a catalyst to troponin. A functional calcium pump will allow for removal of the molecule. ATP is also needed for this step since the calcium requires active transportation, which requires energy.

When ischemia reduces the availability of elements used by the local mitochondria to produce ATP, a local energy crisis develops. When this is taken into account with the above description, one can readily understand how persistent muscle fiber shortening (contractures) might form. Due to the unavailability of ATP to drive the calcium pump, the continual presence of Ca^{2+} in the immediate vicinity of the filaments would add to the continuity of muscle shortening. It is also easily apparent that these would be chemically induced by local factors rather than neuronally driven.

In Chapter 6 we will explore what occurs when some of these steps are altered from their normal process (by trauma, overuse, strain, etc.) and how these filaments produce some of the most vicious, unrelenting, pain-producing elements – myofascial trigger points.

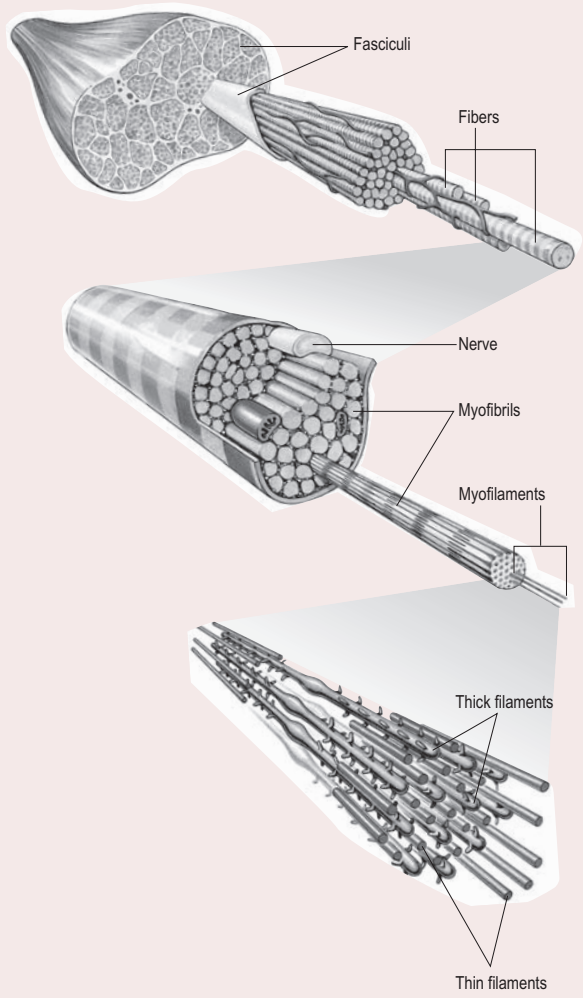

Figure 2.5 From whole muscle to the sarcomere's actin and myosin elements. Reproduced with permission from *Gray's Anatomy* (2005).

- *pennate*, which have fascicles running at an angle to the muscle's central tendon (its longitudinal axis). These fascicles favor strong movement and are divided into *unipennate* (flexor pollicis longus), *bipennate*, which has a feather-like appearance (rectus femoris, peroneus longus) and *multipennate* (deltoid) forms, depending on the configuration of their fibers in relation to their tendinous attachments
- *circular*, as in the sphincters
- *triangular* or *convergent*, where a broad origin ends with a narrow attachment, as in pectoralis major
- *spiral* or *twisted*, as in latissimus dorsi or levator scapulae.

ENERGY PRODUCTION IN NORMAL TISSUES

- Muscles are the body's force generators. In order to achieve this function, they require a source of power, which they derive from their ability to produce mechanical energy from chemically bound energy (in the form of adenosine triphosphate – ATP).
- This process of energy production depends on an adequate supply of oxygen, something that will be normal in aerobically fit tissues, but not in the tissues of the deconditioned individual (see below).
- Some of the energy so produced is stored in contractile tissues for subsequent use when activity occurs. The force that skeletal muscles generate is used to produce or prevent movement, to induce motion or to ensure stability.
- Muscular contractions can be described in relation to what has been termed a *strength continuum*, varying from a small degree of force, capable of lengthy maintenance, to a full-strength contraction, which can be sustained for very short periods.
- When a contraction involves more than 70% of available strength, blood flow is reduced and oxygen availability diminishes.

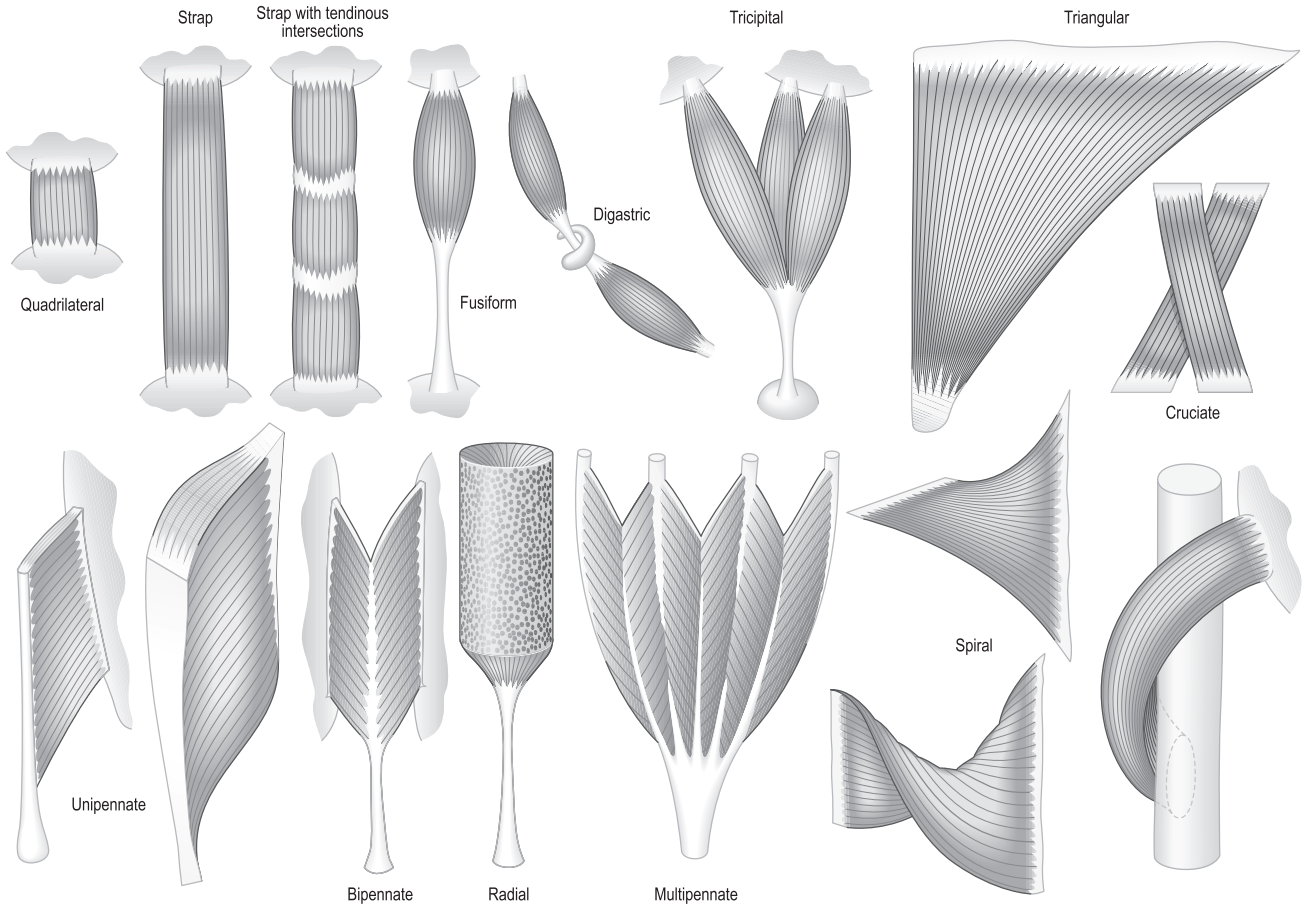

Figure 2.6 Types of muscle fiber arrangement. Reproduced with permission from *Gray's Anatomy* (2005).

ENERGY PRODUCTION IN THE DECONDITIONED INDIVIDUAL

- When anaerobic energy (ATP) pathways are activated in the tissues of deconditioned individuals, the result is accumulation of incompletely oxidized metabolic products, such as lactic acid and pyruvic acid (Fried 1987, Nixon & Andrews 1996).
- The effects of this are described by Nixon & Andrews (1996) as leading to: 'Muscular aching at low levels of effort; restlessness and heightened sympathetic activity; increased neuronal sensitivity; constriction of smooth-muscle tubes [e.g. vascular, respiratory and gastrointestinal], accompanying the basic symptom of inability to make and sustain normal levels of effort.'
- Aerobic activity, if at all possible, is the solution to such problems.
- As outlined later in this chapter, another feature that can result in anaerobic glycolysis is a disturbed breathing pattern, where excessive levels of CO_2 are exhaled (as in hyperventilation).

MUSCLES AND BLOOD SUPPLY

Gray's Anatomy (2005, p. 118) explains the intricacy of blood supply to skeletal muscle as follows:

> *In most muscles the major source artery enters on the deep surface, frequently in close association with the principal vein and nerve, which together form a neurovascular hilum. The vessels course and branch within the connective tissue framework of the muscle. The smaller arteries and arterioles ramify in the perimysial septa and give off capillaries which run in the endomysium. Although the smaller vessels lie mainly parallel to the muscle fibres, they also branch and anastomose around the fibres, forming an elongated mesh.*

Gray's also tells us that the capillary bed of predominantly red muscle (type I postural, see below) is far denser than that of white (type II phasic) muscle.

Research has shown that there are two distinct circulations in skeletal muscle (Grant & Payling Wright 1968).

> ***Nutritive circulation*** *derives from arteriolar branches of arteries entering by way of the neurovascular hilus. These*

penetrate to the endomysium where all the blood passes through to the capillary bed before collection into venules and veins to leave again through the hilus. Alternatively, some of the blood passes into the arterioles of the epi- and perimysium in which few capillaries are present. Arteriovenous anastomosis [a coupling of blood vessels] are abundant here, and most of the blood returns to the veins without passing through the capillaries; this circuit therefore constitutes a ***non-nutritive*** *[collateral] pathway through which blood may pass when the flow in the endomysial capillary bed is impeded, e.g. during contraction.*

In this way blood would keep moving but would not be nourishing the tissues it was destined for, if access to the capillary bed was blocked for any reason. This includes when ischemia is present in the tissues due to overuse, prolonged shortening due to postural positioning, and tight clothing, such as an elastic waistband in pants applying pressure to the lower back tissues.

This is also particularly relevant to deep pressure techniques, designed to create 'ischemic compression' – for example, when treating myofascial trigger points. When ischemic compression is applied, the blood destined for the tissues being obstructed by this pressure (the trigger point site) will diffuse elsewhere until pressure is released, at which time a 'flushing' of the previously ischemic tissues will occur. A blanching/flushing combination repeated several times can act as a local 'irrigation pump' to significantly increase blood flow to localized ischemia.

As explained below, when a situation of increased alkalinity (respiratory alkalosis) leads to the smooth muscles around blood vessels constricting, blood supply will be diminished. In addition, oxygen release to the tissues will also be reduced in such a setting due to the Bohr effect (Pryor & Prasad 2002).

Some areas of the body have relatively inefficient anastomoses and are termed *hypovascular*. These are particularly prone to injury and dysfunction. Examples include the supraspinatus tendon, which corresponds with 'the most common site of rotator cuff tendinitis, calcification and spontaneous rupture' (Cailliet 1991, Tulos & Bennett 1984). Other hypovascular sites include the insertion of the infraspinatus tendon and the intrascapular aspect of the biceps tendon (Brewer 1979).

The lymphatic drainage of muscles occurs via lymphatic capillaries that lie in the epi- and perimysial sheaths. They converge into larger lymphatic vessels that travel close to the veins as they leave the muscle.

Box 2.2 The lymphatic system

Coming in contact with lymph is to connect with the liquid dimension of the organism. (Chikly 1996)

The lymphatic system serves as a collecting and filtering system for the body's interstitial fluids, while removing the body's cellular debris. It is able to process the waste materials from cellular metabolism and provide a strong line of defense against foreign invaders while recapturing the protein elements and water content for recycling by the body. Through 'immunological memory', lymphocyte cells, which reside in the lymph and blood and are part of the general immune system, recognize invaders (antigens) and rapidly act to neutralize these. This system of defending during invasion and then cleaning up the battleground makes the lymphatic system essential to the health of the organism.

Organization of the lymph system

The lymphatic system comprises an extensive network of lymphatic capillaries, a series of collecting vessels and lymph nodes. It is associated with the lymphoid system (lymph nodes, spleen, thymus, tonsils, appendix, mucosal-associated lymphoid tissue such as Peyer's patches and bone marrow), which is primarily responsible for the immune response (Braem 1994, Chikly 1996, 2001). The lymphatic system is:

- an essential defensive component of the immune system
- a carrier of (especially heavy and large) debris on behalf of the circulatory system
- a transporter of fat-soluble nutrients (and fat itself) from the digestive tract to the bloodstream.

Chikly (2001) notes:

The lymphatic system is therefore a second pathway back to the heart, parallel to the blood system. The interstitial fluid is a very important fluid. It is the real 'interior milieu' (Claude Bernard, 1813–1878) in which the cells are immersed, receive their nutritive substances and reject damaging by-products. Lymph is a fluid which originates in the connective tissue spaces of the body. Once it has entered the first lymph capillaries ... this fluid is called lymph.

Collection begins in the interstitial spaces as a portion of the circulating blood is picked up by the lymphatic system. This fluid is comprised primarily of large waste particles, debris and other material from which protein might need to be recovered or that may need to be disposed. Foreign particulate matter and pathogenic bacteria are screened out by the lymph nodes, which are interposed along the course of the vessels. Nodes also produce lymphocytes, which makes their location at various points along the transportation pathway convenient should infectious material be encountered.

Lymph nodes (Chikly 2001):

- filter and purify
- capture and destroy toxins
- reabsorb about 40% of the lymphatic liquids, so concentrating the lymph while recycling the removed water
- produce mature lymphocytes – white blood cells that destroy bacteria, virus-infected cells, foreign matter and waste materials.

Production of lymphocytes increases (in nodes) when lymphatic flow is increased (e.g. with lymphatic drainage techniques).

A lymphatic capillary network made of vessels slightly larger than blood capillaries drains tissue fluid from nearly all tissues and organs that have a blood vascularization. The blood circulatory system is a closed system, whereas the lymphatic system is an open-end system, beginning blind in the interstitial spaces. The moment the fluid enters a lymph capillary, a flap valve prevents it from returning into the interstitial spaces. The fluids, now called 'lymph', continue

box continues

Box 2.2 (continued)

coursing through these 'precollector' vessels which empty into lymph collectors.

The collectors have valves every 6–20 mm that occur directly between two to three layers of spiral muscles, the unit being called a *lymphangion* (Fig. 2.7). The alternation of valves and muscles gives a characteristic 'moniliform' shape to these vessels, like pearls on a string. The lymphangions contract in a peristaltic manner that assists in pressing the fluids through the valved system. When stimulated, the muscles can substantially increase (up to 20–30 times) the capacity of the whole lymphatic system (Chikly 2001).

The largest of the lymphatic vessels is the thoracic duct, which begins at the cisterna chyli, a large sac-like structure within the abdominal cavity located at approximately the level of the 2nd lumbar vertebra. The thoracic duct, containing lymph fluids from both of the lower extremities and all abdominal viscera except part of the liver, runs posterior to the stomach and intestines. Lymph fluids from the left upper extremity, left thorax and the left side of cranium and neck may join it just before it empties into the left subclavian vein or may empty nearby into the internal jugular vein, brachiocephalic junction or directly into the subclavian vein. The right lymphatic duct drains the right upper extremity, right side of the head and neck and right side of the thorax and empties in a similar manner to that of the left side.

Stimulation of lymphangions (and therefore lymph movement) occurs as a result of automotoricity of the lymphangions (electrical potentials from the autonomic nervous system) (Kurz 1986). As the spiral muscles of the vessels contract, they force the lymph through the flap valve, which prevents its return. Additionally, stretching of the muscle fibers of the next lymphangion (by increased fluid volume of the segment) leads to reflex muscle contraction (internally stimulated), thereby producing peristaltic waves along the lymphatic vessel. There are also external stretch receptors that may be activated by manual methods of lymph drainage which create a similar peristalsis.

Lymph movement is also augmented by respiration as the altering intrathoracic pressure produces a suction on the thoracic duct and cisterna chyli and thereby increases lymph movement in the duct and presses it toward the venous arch (Kurz 1986, 1987). Skeletal muscle contractions, movement of limbs, peristalsis of smooth muscles, the speed of blood movement in the veins into which the ducts empty and the pulsing of nearby arteries all contribute to lymph movement (Wittlinger & Wittlinger 1982). Exposure to cold, tight clothing, lack of exercise and excess protein consumption can hinder lymphatic flow (Kurz 1986, Wittlinger & Wittlinger 1982).

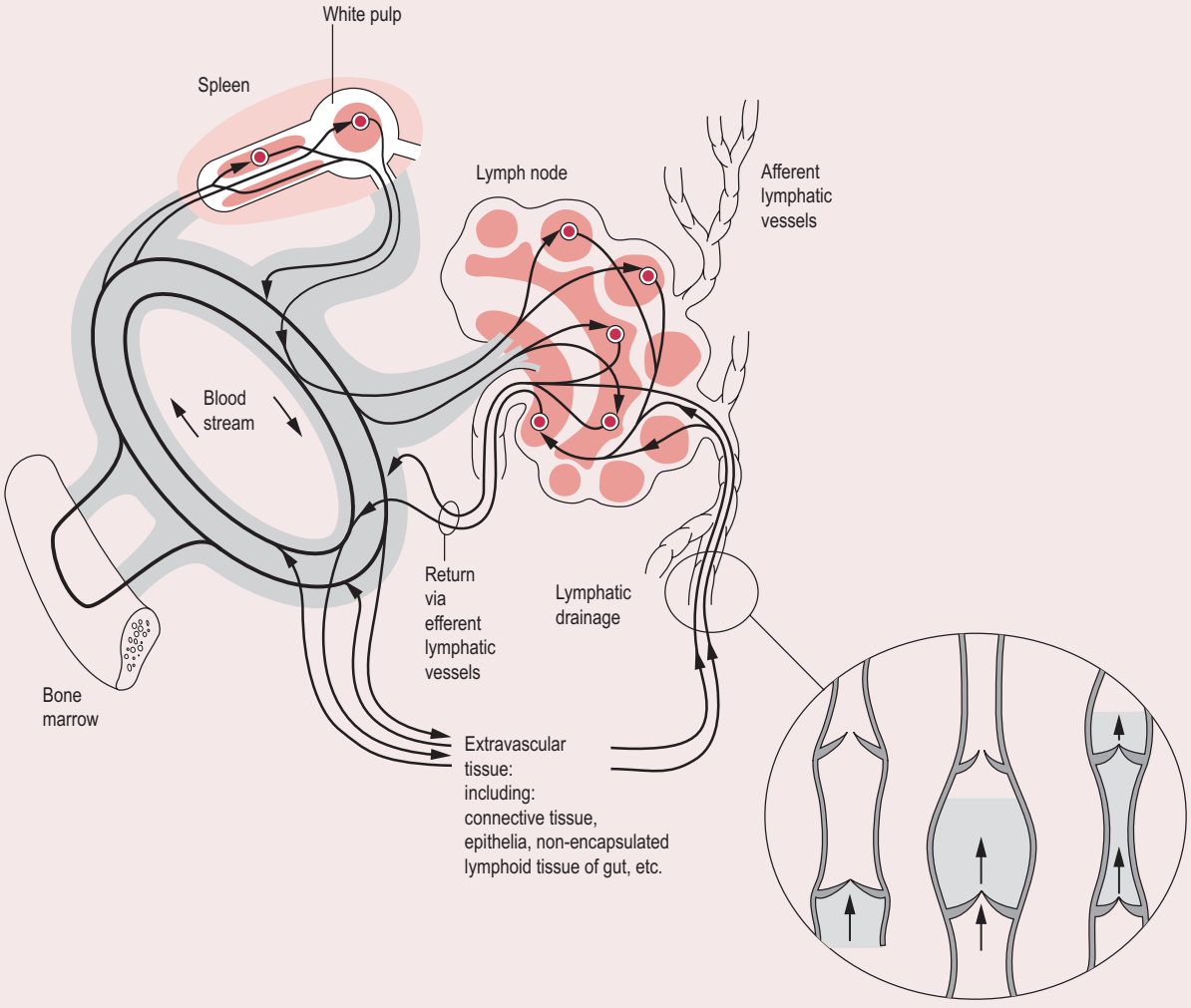

Figure 2.7 Lymph pathway (a lymphangion is shown in insert).

box continues

Box 2.2 (continued)

Contraction of neighboring muscles compresses lymph vessels, moving lymph in the directions determined by their valves; extremely little lymph flows in an immobilized limb, whereas flow is increased by either active or passive movements. This fact has been used clinically to diminish dissemination of toxins from infected tissues by immobilization of the relevant regions. Conversely, massage aids the flow of lymph from oedematous regions. (Gray's Anatomy 1995)

By recovering up to 20% of the interstitial fluids, the lymphatic system relieves the venous system (and therefore the heart) of the responsibility of transporting the large molecules of protein and debris back to the general circulation. Additionally, the lymphocytes remove particulate matter by means of *phagocytosis*, that is, the process of ingestion and digestion by cells of solid substances (other cells, bacteria, bits of necrosed tissue, foreign particles). By the time the fluid has been returned to the veins, it is ultrafiltered, condensed and highly concentrated.

In effect, if the lymphatic system did not regain the 2–20% of the protein-rich liquid that escaped in the interstitium (a large part of which the venous system cannot recover), the body would probably develop major edemas and autointoxication and die within 24–48 hours. (Chikly 2001, Guyton 1986)

Conversely, when applying lymph drainage techniques, care must be taken to avoid excessive increases in the volume of lymph flow in people who have heart conditions as the venous system must accommodate the load once the fluid has been delivered to the subclavian veins. Significantly increasing the load could place excessive strain on the heart.

Lymphatic circulation is separated into two layers. The superficial circulation, which constitutes approximately 70% of all lymph flow (Chikly 2001), is located just under the dermoepidermic junction. The deep muscular and visceral circulation, below the fascia, is activated by muscular contraction; however, the superficial circulation is not directly stimulated by exercise. Additionally, lymph capillaries (lacteals) in the jejunum and ileum of the digestive tract absorb fat and fat-soluble nutrients that ultimately reach the liver through the blood circulation (Braem 1994).

Manual or mechanical lymphatic drainage techniques are effective ways to increase lymph removal from stagnant or edemic tissue. The manual techniques use extremely light pressure, which significantly increases lymph movement by crosswise and lengthwise stretching of the anchoring filaments that open the lymph capillaries, thus allowing the interstitial fluid to enter the lymphatic system. However, shearing forces (like those created by deep pressure gliding techniques) can lead to temporary inhibition of lymph flow by inducing spasms of lymphatic muscles (Kurz 1986). Unless the vessels are damaged, lymphatic movement can then be reactivated by use of manual techniques that stimulate the lymphangions.

While each case has to be considered individually, numerous conditions, ranging from postoperative edema to premenstrual fluid retention, may benefit from lymphatic drainage. There are, however, conditions for which lymphatic drainage would be contraindicated or precautions exercised. Some of the more serious of these conditions include:

- acute infections and acute inflammation (generalized and local)
- thrombosis
- circulatory problems
- cardiac conditions
- hemorrhage
- malignant cancers
- thyroid problems
- acute phlebitis.

Conditions that might benefit from lymphatic drainage but for which precautions are indicated include:

- certain edemas, depending upon their cause, such as cardiac insufficiency
- carotid stenosis
- bronchial asthma
- burns, scars, bruises, moles
- abdominal surgery, radiation or undetermined bleeding or pain
- removed spleen
- major kidney problems or insufficiency
- menstruation (drain prior to menses)
- gynecological infections, fibromas or cysts
- some pregnancies (especially in the first 3 months)
- chronic infections or inflammation
- low blood pressure.

MOTOR CONTROL AND RESPIRATORY ALKALOSIS

Motor control is a key component in injury prevention. Loss of motor control involves failure to control joints, commonly because of incoordination of agonist–antagonist muscle coactivation. According to Panjabi (1992), three subsystems work together to maintain joint and spinal stability:

1. The central nervous subsystem (control)
2. The muscle subsystem (active)
3. The osteoligamentous subsystem (passive).

Anything that interferes with any aspect of these features of normal motor control may contribute to dysfunction and pain. This includes a condition in which the bloodstream increases in alkalinity because of overbreathing (for example hyperventilation, the extreme of overbreathing, see below), which interferes with the first two of those three elements – the CNS as well as muscle function.

People who 'overbreathe', or who have marked upper chest breathing patterns ('breathing pattern disorders' or BPD), automatically exhale more carbon dioxide (CO_2) than is appropriate for their current metabolic needs. Exhaled CO_2 derives from carbonic acid in the bloodstream, and an excessive reduction of this leads to a situation known as respiratory alkalosis, where the pH of the blood becomes more alkaline than its normal of ±7.4 (Lum 1987, Pryor & Prasad 2002).

There are a number of major consequences of increased alkalinity, one of which is a contraction of smooth muscle cells (SMC). This reduces the diameter of all structures surrounded by smooth muscles, such as the blood vessels and intestinal structures. Reduced diameter of blood vessels limits blood supply to the tissues and the brain, thereby

resulting in a variety of symptoms (see below), one of which is increased fatigability. It is postulated that SMC contraction may also influence fascial tone (Schleip et al 2004). (See Chapter 1 for information regarding smooth muscle cells and their location and behavior in connective tissues.)

TWO KEY DEFINITIONS

- *Hypocapnia*: Deficiency of CO_2 in the blood, possibly resulting from hyperventilation, leading to respiratory alkalosis.
- *Hypoxia*: Reduction of O_2 supply to tissue, below physiological levels despite adequate perfusion of the tissue by blood.

Lum (1987) reports that research indicates that not less than 10% of patients attending general internal medicine practice in the US have such breathing pattern disorders as their primary diagnosis. Newton (2001) agrees with this assessment.

The authors of this text suggest that there exists a large patient population with BPDs who do not meet the criteria for hyperventilation, but whose breathing patterns may contribute markedly to their symptom picture, and whose motor control is likely to be negatively affected as a result (Chaitow 2004).

- Breathing pattern disorders are female dominated, ranging from a ratio of 2:1 to 7:1 (Lum 1994).
- Women are more at risk, possibly because progesterone is a respiratory accelerator (Damas-Mora et al 1980).
- Progesterone is known to cause hyperventilation and hypercapnia in the luteal phase of a normal menstrual cycle (Brown 1998, Rajesh et al 2000, Stahl et al 1985).
- During post ovulation phase, CO_2 levels drop ±25% (Lum 1994).
- Additional stress then, 'increases ventilation when CO_2 levels are already low' (Lum 1994).

THE BOHR EFFECT (Fried 1987, Pryor & Prasad 2002)

The Bohr effect states that a rise in alkalinity (due to a decrease in CO_2) increases the affinity of hemoglobin (Hb) for oxygen (O_2). This means that when tissues, and the bloodstream, increase in alkalinity the Hb molecule binds more firmly to the oxygen it is carrying, releasing it less efficiently, which leads to hypoxia. Increased O_2–Hb affinity also leads to changes in serum calcium and red cell phosphate levels which both reduce.

Additionally, there is a loss of intracellular Mg^{2+} as part of the renal compensation mechanism for correcting alkalosis. The function of motor and sensory axons will be significantly affected by lower levels of calcium ions and these sensitive neural structures will tend toward hyperirritability, negatively affecting motor control (Seyal et al 1998).

Lum (1994) explains: 'Loss of CO_2 ions from neurons stimulates neuronal activity, causing increased sensory and motor discharges, muscular tension and spasm, speeding of spinal reflexes, heightened perception (pain, photophobia, hyperacusis) and other sensory disturbances.' Muscles affected in this way inevitably become prone to fatigue, altered function, cramp and trigger point evolution (George et al 1964, Levitzky 1995, Macefield & Burke 1991).

CORE STABILITY, TRANSVERSUS ABDOMINIS, THE DIAPHRAGM AND BPD

It is well established that the tone of both the diaphragm and transversus abdominis hold the key to maintenance of core stability (Panjabi 1992).

McGill et al (1995) have observed a reduction in spinal support if there is both a load challenge to the low back, combined with a demand for increased breathing (imagine shoveling snow!). 'Modulation of muscle activity needed to facilitate breathing may compromise the margin of safety of tissues that depend on constant muscle activity for support.'

Hodges & Gandevia (2000) reported that after approximately 60 seconds of overbreathing, the postural (tonic) and phasic functions of both the diaphragm and transversus abdominis are reduced or absent.

SUMMARY

- BPDs alter blood pH, thereby creating respiratory alkalosis.
- This induces increased sympathetic arousal, which affects neuronal function (including motor control).
- There will be an increased sense of apprehension and anxiety. As a result, the person's balance may be compromised (Winters & Crago 2000).
- Depletion of Ca and Mg ions enhances neural sensitization, encouraging spasm and reducing pain thresholds.
- As pH rises, smooth muscle cells constrict, leading to vasoconstriction that reduces blood supply to the brain and tissues (particularly the muscles) and possibly alters fascial tone.
- Reduced oxygen release to cells, tissues and brain (Bohr effect) leads to ischemia, fatigue and pain, and the evolution of myofascial trigger points.
- If the individual is deconditioned, not involved in aerobic activity, this sequence will trigger release of acid wastes when tissues attempt to produce ATP in a relatively anaerobic environment (as discussed earlier in this chapter).
- Biomechanical overuse stresses emerge along with compromised core stability and postural decay.

What this (overbreathing) scenario illustrates is that when pain and dysfunction involving neuromuscular imbalance are evident in a patient, any therapeutic intervention that fails to pay attention to breathing patterns is less likely to be successful than if this receives appropriate clinical evaluation and rehabilitation, if necessary (see Chapter 4).

MAJOR TYPES OF VOLUNTARY CONTRACTION

Muscle contractions can be:

- *isometric* (with no movement resulting)
- *isotonic concentric* (where shortening of the muscle produces approximation of its attachments and the structures to which the muscle attaches) or
- *isotonic eccentric* (in which the muscle lengthens during its contraction, therefore the attachments separate during contraction of the muscle).

TERMINOLOGY

- The terms *origin* and *insertion* are somewhat inaccurate, with *attachments* being more appropriate. Attachments can be further classified as proximal or distal (in the extremities) or by location, such as sternal, clavicular, costal or humeral attachments of pectoralis major.
- In many instances, muscular attachments can adaptively reverse their roles, depending on what action is involved and therefore which attachment is fixed. As an example, psoas can flex the hip when its lumbar attachment is 'the origin' (fixed point) or it can flex the spine when the femoral attachment becomes 'the origin', i.e. the point toward which motion is taking place.

MUSCLE TONE AND CONTRACTION

Muscles display *excitability* – the ability to respond to stimuli and, by means of a stimulus, to be able to *actively contract*, *extend* (lengthen) or to *elastically recoil* from a distended position, as well as to be able to *passively relax* when stimulus ceases.

Lederman (1997) suggests that *muscle tone* in a resting muscle relates to biomechanical elements – a mix of fascial and connective tissue tension together with intramuscular fluid pressure, with no neurological input (therefore, not measurable by EMG). If a muscle has altered morphologically, due to chronic shortening, for example, or to compartment syndrome, then muscle tone, even at rest, will be altered and palpable.

He differentiates this from *motor tone*, which is measurable by means of EMG and which is present in a resting muscle only under abnormal circumstances – for example, when psychological stress or protective activity is involved.

Motor tone is either *phasic* or *tonic*, depending upon the nature of the activity being demanded of the muscle – to move something (phasic) or to stabilize it (tonic). In normal muscles, both activities vanish when gravitational and activity demands are absent.

Contraction occurs in response to a motor nerve impulse acting on muscle fibers.

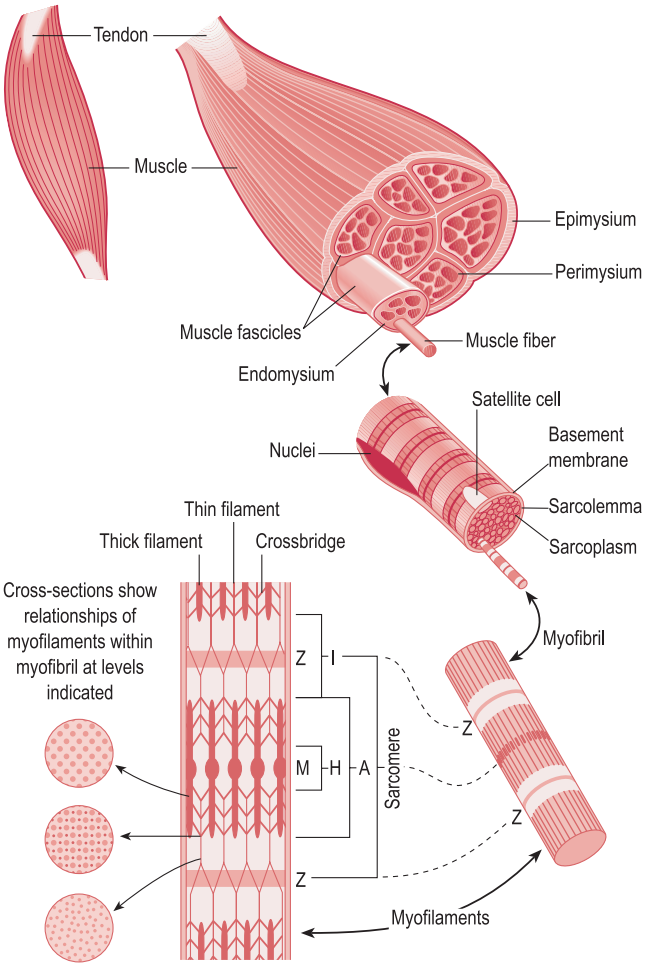

Figure 2.8 Organization of skeletal muscle. Redrawn after Hansen & Koeppen (2002).

A motor nerve fiber will always activate more than one muscle fiber and the collection of fibers it innervates is called a *motor unit*. The greater the degree of fine control a muscle is required to produce, the fewer muscle fibers a nerve fiber will innervate in that muscle. This can range from 10 muscle fibers being innervated by a single motor neuron in the extrinsic eye muscles to one motor neuron innervating several hundred fibers in major limb muscles (*Gray's Anatomy* 2005, p. 121).

Because there is a diffuse spread of influence from a single motor neuron throughout a muscle (i.e. neural influence does not necessarily correspond to fascicular divisions) only a few need to be active to influence the entire muscle.

The functional contractile unit of a muscle fiber is its *sarcomere*, which contains filaments of actin and myosin. These myofilaments (actin and myosin) interact in order to shorten the muscle fiber. *Gray's Anatomy* (2005) describes the process as follows:

> *At higher power, sarcomeres are seen to consist of two types of filament, thick and thin, organized into regular arrays.*

The thick filaments, which are c. 15 nm in diameter, are composed mainly of myosin. The thin filaments, which are 8 nm in diameter, are composed mainly of actin. The arrays of thick and thin filaments form a partially overlapping structure … The A-band consists of the thick filaments, together with links of thin filaments that interdigitate with, and thus overlap, the thick filaments at either end … The I-band consists of the adjacent portions of two neighbouring sarcomeres in which the thin filaments are not overlapped by thick filaments. It is bisected by the Z-disc, into which the thin filaments of the adjacent sarcomeres are anchored. In addition to the thick and thin filaments, there is a third type of filament composed of the elastic protein, titin … The banded appearance of the individual myofibrils is thus attributable to the regular alteration of the thick and thin filaments arrays.

VULNERABLE AREAS

- In order to transfer force to its attachment site, contractile units merge with the collagen fibers of the tendon which attaches the muscle to bone.
- At the transition area, between muscle and tendon, these structures virtually 'fold' together, increasing strength while reducing the elastic quality.
- This increased ability to handle shear forces is achieved at the expense of the tissue's capacity to handle tensile forces.
- The chance of injury increases at those locations where elastic muscle tissue transitions to less elastic tendon and finally to non-elastic bone – the attachment sites of the body.

MUSCLE TYPES

Muscle fibers exist in various motor unit types – basically type I slow red tonic and type II fast white phasic (see below). Type I are fatigue resistant while type II are more easily fatigued.

All muscles have a mixture of fiber types (both I and II), although in most there is a predominance of one or the other, depending on the primary tasks of the muscle (postural stabilizer or phasic mover).

Those which contract slowly (slow-twitch fibers) are classified as *type I* (Engel 1986, Woo 1987). These have very low stores of energy-supplying glycogen, but carry high concentrations of myoglobulin and mitochondria. These fibers fatigue slowly and are mainly involved in postural and stabilizing tasks. The effect of overuse, misuse, abuse or disuse on postural muscles (see Chapters 4 and 5) is that, over time, they will shorten. This tendency to shorten is a clinically important distinction between the response to 'stress' of type I and type II muscle fibers (see below).

There are also several phasic (*type II*) fiber forms, notably:

- type IIa (fast-twitch fibers) which contract more speedily than type I and are moderately resistant to fatigue with relatively high concentrations of mitochondria and myoglobulin
- type IIb (fast-twitch glycolytic fibers) which are less fatigue resistant and depend more on glycolytic sources of energy, with low levels of mitochondria and myoglobulin
- type IIm (superfast fibers) which depend upon a unique myosin structure that, along with a high glycogen content, differentiates them from the other type II fibers (Rowlerson 1981). These are found mainly in the jaw muscles.

As mentioned above, long-term stress involving type I muscle fibers leads to them shortening, whereas type II fibers, undergoing similar stress, will weaken without shortening over their whole length (they may, however, develop localized areas of sarcomere contracture, for example where trigger points evolve without shortening the muscle overall).

Shortness/tightness of a postural muscle does not necessarily imply strength. Such muscles may test as strong or weak. However, a weak phasic muscle will not shorten overall and will always test as weak.

Fiber type is not totally fixed, in that evidence exists as to the potential for adaptability of muscles, so that committed muscle fibers can be transformed from slow twitch to fast twitch, and vice versa (Lin 1994).

An example of this potential, which has profound clinical significance, involves the scalene muscles. Lewit (1985) confirms that they can be classified as either a postural or a phasic muscle. The scalenes, which are largely phasic (type II) and dedicated to movement, can have postural functions thrust upon them, as with forward head postures, or when chronically contracted to maintain a virtually permanently elevated status of the upper chest, as in asthma. If these postural demands are prolonged, more postural (type I) fibers may develop to meet the situation. If overuse continues (as in upper chest breathing involving the upper ribs being regularly elevated during inhalation), these now postural muscles will shorten, as would any type I muscle when chronically stressed (Janda 1982, Liebenson 2006).

The following findings, relating to the scalene muscles, were reported in a study that evaluated the link between these and inappropriate breathing patterns, in this instance, mainly asthma.

The incidence of scalene muscle pathology was assessed in 46 consecutively hospitalized patients with bronchial asthma and irritable cough diagnoses. Three tests described by Travell & Simons were used in patient evaluation, including palpation for scalene trigger points and the use of Adson's test. Breathing patterns were also evaluated in all patients for the presence of paradoxical breathing patterns. Scalene muscle pathology [dysfunction] was identified in 20 of the 38 bronchial asthma patients (52%), and in 5 of the

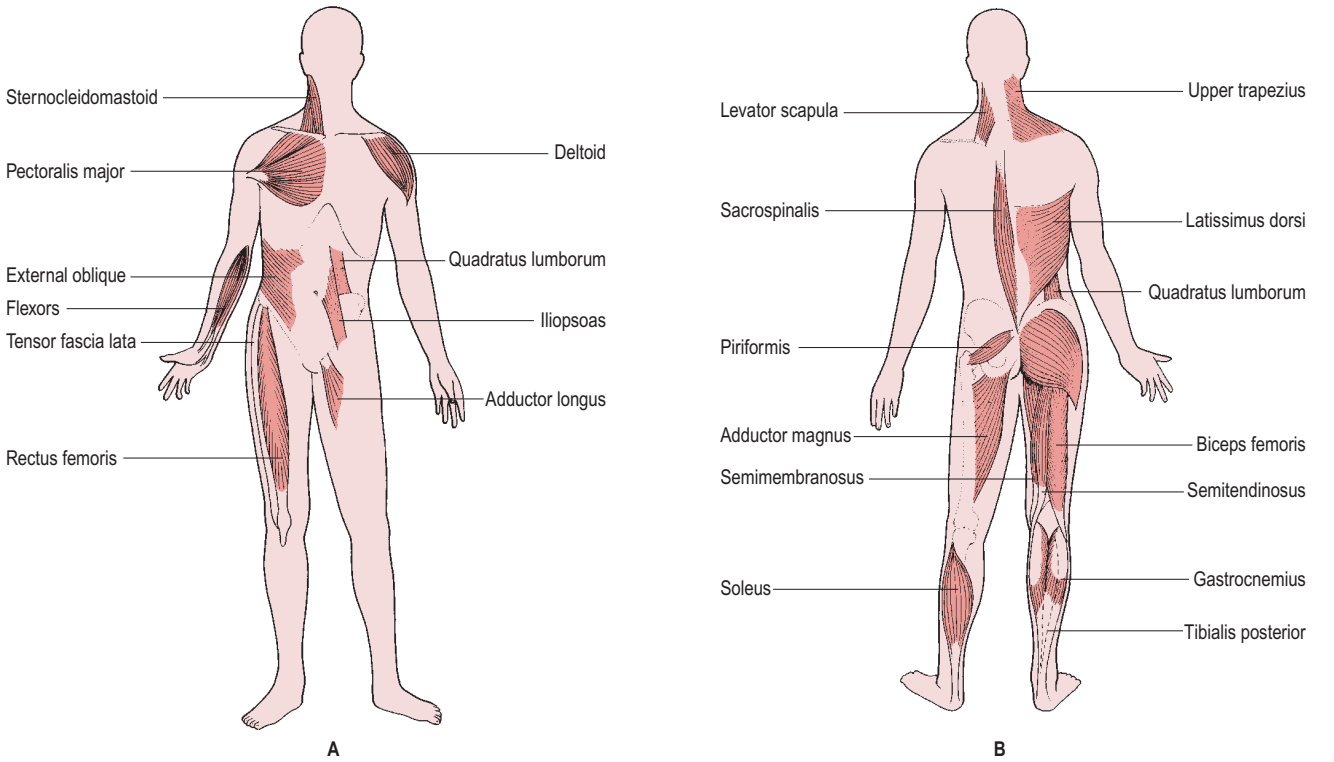

Figure 2.9 Major postural muscles. **A**: Anterior. **B**: Posterior. Reproduced with permission from Chaitow (1996).

8 irritable cough syndrome patients (62%). Postisometric relaxation technique [muscle energy] was used in those with scalene dysfunction. Self-administered stretching techniques for home use were also taught. One patient with paradoxical breathing pattern was taught an alternative breathing pattern. The authors are of the opinion that bronchial asthma and irritable cough syndrome patients should be examined and evaluated by Rehabilitation Medicine Department staff for functional pathology of the scalene muscles. They are also of the opinion that examination, treatment and self-administered stretching techniques should be a part of routine management of bronchial asthma patients. (Pleidelova et al 2002)

Among the more important postural muscles that become hypertonic in response to dysfunction are:

- trapezius (upper), sternocleidomastoid, levator scapula and upper aspects of pectoralis major in the upper trunk and the flexors of the arms
- quadratus lumborum, erector spinae, oblique abdominals and iliopsoas in the lower trunk
- tensor fascia latae, rectus femoris, biceps femoris, adductors (longus, brevis and magnus), piriformis, semimembranosus and semitendinosus in the pelvic and lower extremity region.

Phasic muscles, which weaken in response to dysfunction (i.e. are inhibited), include the paravertebral muscles (not erector spinae), scalenii and deep neck flexors, deltoid, the abdominal (or lower) aspects of pectoralis major, middle and lower aspects of trapezius, the rhomboids, serratus anterior, rectus abdominis, gluteals, the peroneal muscles, vasti and the extensors of the arms.

Some muscle groups, such as the scalenii, are equivocal. Although commonly listed as phasic muscles, this is how they start life but they can end up as postural ones if sufficient demands are made on them (see above).

COOPERATIVE MUSCLE ACTIVITY

Few, if any, muscles work in isolation, with most movements involving the combined effort of two or more, with one or more acting as the 'prime mover' or *agonist*.

Almost every skeletal muscle has an *antagonist* that performs the opposite action, with one of the most obvious examples being the elbow flexors (biceps brachii) and extensors (triceps brachii).

Prime movers usually have synergistic muscles that assist them and which contract at almost the same time. An example of these roles would be hip abduction, in which gluteus medius is the prime mover, with tensor fascia latae and gluteus minimus acting synergistically and the hip adductors acting as antagonists, being *reciprocally inhibited* (RI) by the action of the agonists if movement is to occur. RI is the physiological phenomenon in which there is an automatic inhibition of a muscle when its antagonist contracts, also known as Sherrington's law II.

Box 2.3 Alternative categorization of muscles

It is generally accepted that muscles respond to overuse, misuse or disuse by either shortening or weakening (and possibly lengthening). As Kolar has explained (in Liebenson 2006, p. 533): 'There is clinical and experimental evidence that some muscles are inclined to inhibition (hypotonus, weakness, inactivity), while other muscle groups are likely to be hyperactive with a tendency to become short.' It was Janda (1969, 1983a) who first showed that these changes followed certain rules, and who named them as phasic (those tending to inhibition) and postural (those tending to shortening). A plethora of different descriptors have been used to label these two muscle groups, including stabilizer, mobilizer; global, local; superficial, deep, etc. (Norris 1995a,b), adding a sense of potential disagreement and confusion to the understanding of what is in essence relatively simple: some muscles follow one pathway toward dysfunction, while others follow a different pathway – whatever names they are ascribed. In the interest of simplicity, the authors of this text have continued to designate these different muscle types as postural and phasic.

Liebenson (2006, p. 411) discusses Janda's classification of tense and tight muscles and further separates muscle dysfunction into a variety of different treatment-specific categories that are either neuromuscular or connective tissue related.

These classifications are as follows:

- Neuromuscular:
  1. *Reflex spasm*: As a response to nociception, this often acts as a splinting mechanism. Treatment would aim toward removal of the cause of pain, such an inflamed appendix.
  2. *Interneuron*: This delicate part of the reflex arc can become involved when afferent information is sent from spinal or peripheral joints. Treatment would aim to normalize the involved joints.
  3. *Trigger point*: This is thought to be associated with localized congestion within the muscle stemming from short muscle fibers. A variety of treatments are offered in this book to normalize myofascial tissue.
  4. *Limbic*: This is associated with psychological stress. It can be treated with counseling, stress management and a variety of relaxation methods including yoga and meditation.
- Connective tissue:
  1. *Overuse muscle tightness*: This stems from muscle imbalances, overuse, faulty movement patterns and other stresses that result in postural adaptations. Treatment would aim to normalize the tissues and lengthen the fibers.

Many of these categories interface – for instance, overused tight muscles tend to create joint pressure leading to interneuron responses. Psychological stress might result in muscle tightening and trigger point formation. Although the body has a number of response choices that it can make to cope with the load to which it is adapting (biochemical, biomechanical and psychosocial), the practitioner also has a wide range of choices in the way of interventions. Chapters 9 and 10 carry a full discussion of some of those options.

In summary, whatever the causes, there are two main responses by muscles when chronically stressed:

1. They are inhibited and show evidence of hypotonus and weakness (phasic), or
2. They develop hypertonus, and possibly spasm and rigidity (postural).

These changes appear to involve mainly the contractile elements of muscles. However, in some instances, connective tissue may also be involved, resulting in contracture (Janda 1991).

There is quite naturally not only a functional but also a structural aspect to these differences, and these have been identified by physiologists. As Kolar explains (Liebenson 2006, p. 533):

Differences are found in the nervous structure in control of these [different] muscles, for it is the type of neurons that determines the type of muscle fibre. It is therefore better to speak of tonic and phasic motor units. Tonic motorneurons, i.e. small alpha motor cells, innervate red muscle fibres, whereas phasic motorneurons (large alpha cells) innervate white muscle fibres. In humans, both types of motor units are present in every muscle, in different proportions.

Examples of patterns of imbalance which emerge as some muscles weaken and lengthen and their synergists become overworked, while their antagonists shorten, can be summarized as follows.

Lengthened or underactive stabilizer	Overactive synergist	Shortened antagonist
1. Gluteus medius	TFL, QL, piriformis	Thigh adductors
2. Gluteus maximus	Iliocostalis lumborum & hamstrings	Iliopsoas, rectus femoris
3. Transversus abdominis	Rectus abdominis	Iliocostalis lumborum
4. Lower trapezius	Levator scapulae/Upper trapezius	Pectoralis major
5. Deep neck flexors	SCM	Suboccipitals
6. Serratus anterior	Pectoralis major/minor	Rhomboids
7. Diaphragm	Scalenes, pectoralis major/minor	

Observation

Observation can often provide evidence of an imbalance involving cross patterns of weakness/lengthening and shortness. A number of tests can be used to assess muscle imbalance: postural inspection, muscle length tests, movement patterns and inner holding endurance times. Posture is valuable because it provides a quick screen.

box continues

Box 2.3 (continued)

Muscle inhibition/weakness/lengthening	Observable sign
Transversus abdominis	Protruding umbilicus
Serratus anterior	Winged scapula
Lower trapezius	Elevated shoulder girdle ('gothic' shoulders)
Deep neck flexors	Chin 'poking'
Gluteus medius	Unlevel pelvis on one-legged standing
Gluteus maximus	Sagging buttock

Inner range endurance tests

'Inner holding isometric endurance' tests can be performed for muscles that have a tendency to lengthen, in order to assess their ability to maintain joint alignment in a neutral zone. Usually a lengthened muscle will demonstrate a loss of endurance, when tested in a shortened position. This can be tested by the practitioner passively prepositioning the muscle in a shortened position and assessing the duration of time that the patient can hold the muscle in this position. There are various methods used, including 10 repetitions of the holding position for 10 seconds at a time. Alternatively, a single 30-second hold can be requested. If the patient cannot hold the position actively from the moment of passive prepositioning, this is a sign of inappropriate antagonist muscle shortening.

Norris (1999) describes an example of inner range holding tests.

- *Iliopsoas*: Patient is seated. Practitioner lifts one leg into greater hip flexion so that foot is well clear of floor and the patient is asked to hold this position.
- *Gluteus maximus*: Patient is prone. Practitioner lifts one leg into extension at the hip (knee flexed to 90°) and the patient is asked to hold this position.
- *Posterior fibers of gluteus medius*: Patient is sidelying with lower leg straight and uppermost leg flexed at hip and knee so that the medial aspect of both the knee and foot are resting on the floor/surface. Practitioner places the flexed leg into a position of maximal unforced external rotation at the hip, so that sole of foot is in contact with the floor surface, and the patient is asked to maintain this position.

Norris states:

Optimal endurance is indicated when the full inner range position can be held for 10 to 20 seconds. Muscle lengthening is present if the limb falls away from the inner range position immediately.

Movement can only take place normally if there is coordination of all the interacting muscular elements. With many habitual complex movements, such as how to rise from a sitting position, a great number of involuntary, largely unconscious reflex activities are involved. In many cases, patterns of dysfunction, including muscle substitution and changes in firing sequence, develop and often add undesirable consequences. Altering such patterns has to involve a relearning or repatterning process (see Chapters 4 and 5).

The most important action of an antagonist occurs at the outset of a movement, where its function is to facilitate a smooth, controlled initiation of movement by the agonist and its *synergists*, those muscles that share in and support the movement. When agonist and antagonist muscles contract simultaneously they act in a stabilizing *fixator* role, which results in virtually no movement.

Sometimes a muscle has the ability to have one part acting as an antagonist to other parts of the same muscle, a phenomenon seen in the deltoid, where its anterior fibers are antagonistic to its posterior fibers during internal and external rotation of the humerus. Interestingly, these same fibers become synergists in the movement of lateral abduction of the humerus. Hence the role that various fibers play, even within the same muscle, changes dependent upon the desired effect.

The ways in which skeletal muscles produce or deny movement in the body, or in part of it, can be classified as:

- *postural*, where stability is induced. If this relates to standing still, it is worth noting that the maintenance of the body's center of gravity over its base of support requires constant fine tuning of a multitude of muscles, with continuous tiny shifts back and forth and from side to side
- *ballistic*, in which the momentum of an action carries on beyond the activation produced by muscular activity (the act of throwing, for example)
- *tension movement*, where fine control requires constant muscular activity (playing a musical instrument, such as the violin, for example, or giving a massage).

MUSCLE SPASM, TENSION, ATROPHY (Liebenson 1996, Walsh 1992)

Muscles are often said to be short, tight, tense or in spasm; however, these terms are often used very loosely.

Muscles experience either neuromuscular, viscoelastic or connective tissue alterations or combinations of these. A tight muscle could have either increased neuromuscular tension or connective tissue modification (for example, fibrosis) that results in it palpating as tight.

It is worthwhile differentiating between three commonly used terms: contraction, spasm and contracture. With regards to skeletal muscles, each of these produces a shortening or increase in tension of a muscle. However, they are unique in many ways.

CONTRACTION (TENSION WITH EMG ELEVATION, VOLUNTARY)

- Muscle tension, usually with shortening, that denotes the normal function of a muscle.
- Electromyographic (EMG) activity is increased in these cases.
- Contraction is voluntary, not obligatory, i.e. one can voluntarily relax a contraction if desired.
- While contraction usually produces movement of the joint(s) on which the muscle acts, it can also contract to produce stability in a moving joint, as a result of anxiety or for postural purposes.

SPASM (TENSION WITH EMG ELEVATION, INVOLUNTARY)

- Muscle spasm is a neuromuscular phenomenon relating either to an upper motor neuron disease or an acute reaction to pain or tissue injury.
- Electromyographic (EMG) activity is increased in these cases.
- Spasm is involuntary, i.e. one cannot voluntarily relax a spasm.
- Examples include spinal cord injury, reflex spasm (such as in a case of appendicitis) or acute lumbar antalgia with loss of flexion relaxation response (Triano & Schultz 1987).
- Long-lasting noxious (pain) stimulation has been shown to activate the flexion withdrawal reflex (Dahl et al 1992).
- Using electromyographic evidence Simons (1994) has shown that myofascial trigger points can 'cause reflex spasm and reflex inhibition in other muscles, and can cause motor incoordination in the muscle with the trigger point'.

CONTRACTURE (TENSION OF MUSCLES WITHOUT EMG ELEVATION, INVOLUNTARY)

- Increased muscle tension can occur without a consistently elevated EMG.
- Contracture is involuntary, i.e. one cannot voluntarily relax a contracture.
- An example is trigger points, in which muscle fibers fail to relax properly.
- Muscle fibers housing trigger points have been shown to have different levels of EMG activity within the same functional muscle unit.
- Hyperexcitability, as shown by EMG readings, has been demonstrated in the nidus of the trigger point, which is situated in a taut band (that shows no increased EMG activity) and has a characteristic pattern of reproducible referred pain (Hubbard & Berkoff 1993, Simons et al 1999).
- When pressure is applied to an active trigger point, EMG activity is found to increase in the muscles to which sensations are being referred ('target area') (Simons 1994).
- A contracture differs from a contraction in that it is involuntary and that activation of the myofibrils is prolonged in the *absence* of action potential activity (MacIntosh et al 2006, Simons et al 1999).
- These types of 'physiologic' contractures are differentiated from the 'pathologic' contractures associated with permanent shortening of muscles produced by excessive growth of fibrous tissue, such as seen in Duchenne muscular dystrophy (MacIntosh et al 2006).

INCREASED STRETCH SENSITIVITY

- Increased sensitivity to stretch can lead to increased muscle tension.
- This can occur under conditions of local ischemia, which have also been demonstrated in the nidus of trigger points, as part of the 'energy crisis' which, it is hypothesized, produces them (Mense 1993, Mense et al 2001, Simons 1994) (see Chapter 6).
- Many free nerve endings in group III (smallest myelinated) and IV (non-myelinated) afferent fibers are sensitive to pressure or stretch (MacIntosh et al 2006) and would likely be affected by the degree of ischemia within the muscle.
- These same afferents also become sensitized in response to a build-up of metabolites (MacIntosh et al 2006) when sustained mild contractions occur, such as occurs in prolonged slumped sitting (Johansson 1991).
- Mense (1993) and Mense et al (2001) suggest that a range of dysfunctional events emerge from the production of local ischemia that can occur as a result of venous congestion, local contracture and tonic activation of muscles by descending motor pathways.
- Sensitization (which, in all but name, is the same phenomenon as facilitation, as discussed more fully in Chapter 6) involves a change in the stimulus–response profile of neurons (Mense et al 2001), leading to a decreased threshold as well as increased spontaneous activity of types III and IV primary afferents.
- Schiable & Grubb (1993) have implicated reflex discharges from (dysfunctional) joints in the production of such neuromuscular tension. Liebenson (2006) notes that 'joint inflammation or pathology initiates a complex neuromuscular response in the dorsal horn of the spinal cord, resulting in flexor facilitation and extensor inhibition'.

- According to Janda (1991), and agreed to by Liebenson (2006), neuromuscular tension can also be increased by central influences due to limbic dysfunction.

VISCOELASTIC INFLUENCE

- Muscle stiffness is a viscoelastic phenomenon that has to do with fluid mechanics and viscosity (so-called sol or gel) of tissue (Liebenson 2006, Walsh 1992), which is explained more fully in Chapter 1.
- Fibrosis occurs gradually in muscle or fascia and is typically related to post trauma adhesion formation (see notes on fibrotic change in Chapter 1, p. 16).
- Fibroblasts proliferate in injured tissue during the inflammatory phase (Lehto et al 1986).
- If the inflammatory phase is prolonged then a connective tissue scar will form as the fibrosis is not absorbed.

ATROPHY AND CHRONIC BACK PAIN

- In chronic back pain patients, generalized atrophy has been observed and to a greater extent on the symptomatic side (Stokes et al 1992).

Box 2.4 Muscle strength testing

For efficient muscle strength testing it is necessary to ensure that:

- the patient builds force slowly after engaging the barrier of resistance offered by the practitioner
- the patient uses maximum controlled effort to move in the prescribed direction
- the practitioner ensures that the point of muscle origin is efficiently stabilized
- care is taken to avoid use by the patient of 'tricks' in which synergists are recruited.
- Muscle strength is most usually graded as follows.
- Grade 5 is normal, demonstrating a complete (100%) range of movement against gravity, with firm resistance offered by the practitioner.
- Grade 4 is 75% efficiency in achieving range of motion against gravity with slight resistance.
- Grade 3 is 50% efficiency in achieving range of motion against gravity without resistance.
- Grade 2 is 25% efficiency in achieving range of motion with gravity eliminated.
- Grade 1 shows slight contractility without joint motion.
- Grade 0 shows no evidence of contractility.

Box 2.5 Two-joint muscle testing

As a rule when testing a two-joint muscle good fixation is essential. The same applies to all muscles in children and in adults whose cooperation is poor and whose movements are uncoordinated and weak. The better the extremity is steadied, the less the stabilizers are activated and the better and more accurate are the results of the muscle function test. (Janda 1983b)

- Type I (postural or aerobic) fibers *hypertrophy* on the symptomatic side and type II (phasic or anaerobic) fibers *atrophy* bilaterally in chronic back pain patients (Fitzmaurice et al 1992).

WHAT IS WEAKNESS?

True muscle weakness is a result of lower motor neuron disease (e.g. nerve root compression or myofascial entrapment) or disuse atrophy. In chronic back pain patients, generalized atrophy has been demonstrated. This atrophy is selective in the type II (phasic) muscle fibers bilaterally.

Muscle weakness is another term that is used loosely. A muscle may simply be inhibited, meaning that it has not suffered disuse atrophy but is weak due to a reflex phenomenon. Inhibited muscles are capable of spontaneous strengthening when the inhibitory reflex is identified and remedied (commonly achieved through soft tissue or joint manipulation). A typical example is reflex inhibition from an antagonist muscle due to Sherrington's law of reciprocal inhibition, which declares that a muscle will be inhibited when its antagonist contracts.

- Reflex inhibition of the vastus medialis oblique (VMO) muscle after knee inflammation/injury has been repeatedly demonstrated (DeAndrade et al 1965, Spencer et al 1984).
- Hides et al (1994) found unilateral, segmental wasting of the multifidus in acute back pain patients. This occurred rapidly and thus was not considered to be disuse atrophy.
- In 1994, Hallgren et al found that some individuals with chronic neck pain exhibited fatty degeneration and atrophy of the rectus capitis posterior major and minor muscles as visualized by MRI. Atrophy of these small suboccipital muscles obliterates their important proprioceptive output, which may destabilize postural balance (McPartland et al 1997) (see Chapter 3 for more detail on these muscles).

Various pathological situations have been listed that can affect either the flexibility or the strength of muscles. The result is muscular imbalance involving increased tension or tightness in postural muscles, coincidental with inhibition or weakness of phasic muscles.

TRICK PATTERNS

Altered muscular movement patterns were first recognized clinically by Janda (1982) when it was noticed that classic muscle-testing methods did not differentiate between normal recruitment of muscles and 'trick' patterns of substitution during an action. So-called trick movements (see below) are uneconomical and place unusual strain on joints. They involve muscles that function in uncoordinated ways and are related to both altered motor control and poor endurance.

In a traditional test of prone hip extension it is difficult to identify overactivity of the lumbar erector spinae or hamstrings as substitutes for an inhibited gluteus maximus. Tests developed by Janda are far more sensitive and allow us to identify muscle imbalances, faulty (trick) movement patterns and joint overstrain by observing or palpating abnormal substitution during muscle-testing protocols. For example, in a prone position, hip extension should be initiated by gluteus maximus. If the hamstrings undertake the role of prime mover and gluteus maximus is inhibited, this is easily noted by palpating activity within each of them as movement is initiated.

Similar imbalances can be palpated and observed in the shoulder region where the upper fixators dominate the lower fixators by inhibiting them, which results in major neck and shoulder stress. These patterns have major repercussions, as will become clear when crossed syndromes, and Janda's functional assessment methods, are outlined in Chapter 5 (Janda 1978).

As Sterling et al (2001) explain:

> *Musculoskeletal pain potentially produces many changes in motor activity. Some of these changes can be explained by peripheral mechanisms in the muscles themselves and by mechanisms within the central nervous system. Certainly, pain has a potent effect on motor activity and control.*
>
> *The dysfunction that occurs in the neuromuscular system in the presence of pain is extremely complex. In addition to the more obvious changes, such as increased muscle activity in some muscle groups, and inhibition of others, more subtle anomalous patterns of neuromuscular activation seem to occur … Loss of selective activation and inhibition of certain muscles that perform key synergistic functions, leading to altered patterns of neuromuscular activation, and the ensuing loss of joint stability and control, are initiated with acute pain and tissue injury. However, these phenomena persist into the period of chronicity and could be one reason for ongoing symptoms.*

Examples

- Pain may lead to inhibition or delayed activation of specific muscles or muscle groups involved in key synergistic functions. This seems to most commonly occur in the deep local muscles that perform a synergistic function to control joint stability (Cholewicki et al 1997).
- EMG has been used to detect selective fatigue of lumbar multifidus, as opposed to other erector spinae muscles (Roy et al 1989).
- Ultrasonography was used by Hides et al (1994) to identify a marked atrophy of lumbar multifidus ipsilateral to the patients' symptoms. These changes remained even after the patients had ceased to report pain (Hides et al 1996).
- A delay of contraction of transversus abdominis was noted in subjects with low back pain when they performed limb movements (Hodges & Richardson 1999). This was in contrast to subjects without low back pain who showed that contraction of transversus abdominis precedes contraction of the muscles involved in limb movement (Hodges & Richardson 1996).
- The upper and deep cervical flexor muscles (type II, phasic) have been shown to lose their endurance capacity in subjects with neck pain and headache (Watson & Trott 1993).
- When testing for activity in these deep flexor muscles, it has been found that patients with neck pain tend to substitute with the superficial flexor muscles (sternocleidomastoid and scalenes) to achieve the desired position of the neck (Jull 2000).
- The posterior suboccipital muscles, which control the position of the head, have been shown to atrophy in patients with chronic neck pain (McPartland et al 1997). The synergistic function of these muscles may be lost so that other muscles, such as upper trapezius and levator scapulae, substitute for the suboccipital muscles during functional movements. This is confirmed by studies that have reported increased activity in these muscles in people with neck pain (Bansevicius & Sjaastad 1996).

These examples offer insights into the adaptive capacity of the musculoskeletal system when faced with problems of pain, overuse and disuse. There is clear evidence that some muscles respond by becoming inhibited and/or by losing stamina, while others shorten.

JOINT IMPLICATIONS

When a movement pattern is altered, the activation sequence, or firing order of different muscles involved in a specific movement, is disturbed. The prime mover may be slow to activate while synergists or stabilizers substitute and become overactive. When this is the case, new joint stresses will be encountered. Sometimes the timing sequence is normal yet the overall range may be limited due to joint stiffness or antagonist muscle shortening. Pain may well be a feature of such dysfunctional patterns.

WHEN SHOULD PAIN AND DYSFUNCTION BE LEFT ALONE?

Splinting (spasm) can occur as a defensive, protective, involuntary phenomenon associated with trauma (fracture) or pathology (osteoporosis, secondary bone tumors, neurogenic influences, etc.). Splinting-type spasm commonly differs from more common forms of contraction and hypertonicity because it often releases when the tissues that it is protecting, or immobilizing, are placed at rest.

When splinting remains long term, secondary problems may arise in associated joints (e.g. contractures) and bone (e.g. osteoporosis). Travell & Simons (1983) note that, 'Muscle-splinting pain is usually part of a complex process. Hemiplegic and brain-injured patients do identify pain that

depends on muscle spasm'. They also note 'a degree of masseteric spasm which may develop to relieve strain in trigger points in its parallel muscle, the temporalis'.

Travell & Simons (1983) note a similar phenomenon in the lower back:

> *In patients with low back pain and with tenderness to palpation of the paraspinal muscles, the superficial layer tended to show less than a normal amount of EMG activity until the test movement became painful. Then these muscles showed increased motor unit activity or 'splinting' … This observation fits the concept of normal muscles 'taking over' (protective spasm) to unload and protect a parallel muscle that is the site of significant trigger point activity.*

Recognition of this degree of spasm in soft tissues is a matter of training and intuition. Whether attempts should be made to release, or relieve, what appears to be protective spasm depends on understanding the reasons for its existence. If splinting is the result of a cooperative attempt to unload a painful but not pathologically compromised structure, in an injured knee or shoulder for example, then treatment is obviously appropriate to ease the cause of the original need to protect and support. If, on the other hand, spasm or splinting is indeed protecting the structure it surrounds (or supports) from movement and further (possibly) serious damage, as in a case of advanced osteoporosis for example, then it should clearly be left alone.

BENEFICIALLY OVERACTIVE MUSCLES

Van Wingerden et al (1997) report that both intrinsic and extrinsic support for the sacroiliac joint (SIJ) derives, in part, from hamstring (biceps femoris) status. Intrinsically, the influence is via the close anatomic and physiological relationship between biceps femoris and the sacrotuberous ligament (they frequently attach via a strong tendinous link). They state: 'Force from the biceps femoris muscle can lead to increased tension of the sacrotuberous ligament in various ways. Since increased tension of the sacrotuberous ligament diminishes the range of sacroiliac joint motion, the biceps femoris can play a role in stabilization of the SIJ' (Van Wingerden et al 1997; see also Vleeming 1989).

Van Wingerden et al (1997) also note that in low back pain patients forward flexion is often painful as the load on the spine increases. This happens whether flexion occurs in the spine or via the hip joints (tilting of the pelvis). If the hamstrings are tight and short, they effectively prevent pelvic tilting. 'In this respect, an increase in hamstring tension might well be part of a defensive arthrokinematic reflex mechanism of the body to diminish spinal load.'

If such a state of affairs is long standing, the hamstrings (biceps femoris) will shorten (see discussion of the effects of stress on postural muscles, p. 25), possibly influencing sacroiliac and lumbar spine dysfunction. The decision to treat a tight hamstring should therefore take account of why it is tight and consider that, in some circumstances, it is offering beneficial support to the SIJ or that it is reducing low back stress (Simons 2002, Thompson 2001). It is possible to conceive similar supportive responses in a variety of settings, including the shoulder joint when lower scapular fixators have weakened, thus throwing the load onto other muscles (see discussion of upper crossed syndrome in Chapter 5).

SOMATIZATION – MIND AND MUSCLES

It is entirely possible for musculoskeletal symptoms to represent an unconscious attempt by the patient to entomb their emotional distress. As most cogently expressed by Philip Latey (1996), pain and dysfunction may have psychological distress as their root cause. The patient may be somatizing the distress and presenting with apparently somatic problems (see Chapter 4).

BUT HOW IS ONE TO KNOW?

Karel Lewit (1992) suggests that, 'In doubtful cases, the physical and psychological components will be distinguished during the treatment, when repeated comparison of (changing) physical signs and the patient's own assessment of them will provide objective criteria'. In the main, he suggests, if the patient is able to give a fairly precise description and localization of his pain, we should be reluctant to regard it as 'merely psychological'.

In masked depression, Lewit suggests, the reported symptoms may well be of vertebral pain, particularly involving the cervical region, with associated muscle tension and 'cramped' posture. As well as being alerted by abnormal responses during the course of treatment to the fact that there may be something other than biomechanical causes of the problem, the history should provide clues. If the masked depression is treated appropriately, the vertebrogenic pain will clear up rapidly, he states.

In particular, Lewit notes, 'The most important symptom is disturbed sleep. Characteristically, the patient falls asleep normally but wakes within a few hours and cannot get back to sleep'. Pain and dysfunction can be masking major psychological distress and awareness of it, how and when to cross-refer should be part of the responsible practitioner's skills base.

Muscles cannot be separated, in reality or intellectually, from the fascia that envelops and supports them. Whenever it appears we have done so in this book, it is meant to highlight and reinforce particular characteristics of each. When it comes to clinical applications, these structures have to be considered as integrated units. As muscular dysfunction is being modified and corrected it is almost impossible to conceive that fascial structures are not also being remodeled. Some of the quite amazingly varied functions of fascia are

detailed in Chapter 1. In this chapter we have reviewed some of the important features of muscles themselves, their structure, function and at least some of the influences that cause them to become dysfunctional, in unique ways, depending in part on their fiber type.

In the next chapter, as we review the myriad reporting stations embedded in the soft tissues in general and the muscles in particular, it becomes clear that muscles are as much an organ of sense as they are agents of movement and stability.

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