

## Chapter 9

# Lumbar and thoracic spine

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Back pain is a persistent and costly problem for society today. It is estimated that approximately 70% of Americans will have back pain at some point in their life.<sup>1</sup> For many people these conditions will occur more than once. The direct and indirect medical costs of low-back pain (LBP) are staggering. In 1991 it was reported that these costs equaled somewhere between \$50 and \$100 billion per year in the United States, with more recent studies showing these costs increasing.<sup>2,3-5</sup> Attempts have been made to identify which treatments are most cost effective in dealing with this pervasive musculoskeletal disorder, but studies have yet to come away with a clear determination.<sup>6,7</sup> Other countries also report a high incidence of LBP indicating this disorder places a high economic burden on the health care system worldwide.<sup>8-12</sup>

The causes of LBP are poorly understood and treatment for the condition can be inadequate. For many years LBP was thought to result primarily from structural disorders, such as herniated discs.<sup>1</sup> While herniations do exist in some cases, many LBP complaints do not involve disc pathology. For numerous cases there is no easily identifiable structural or organic cause for the pain.

It is becoming increasingly clear that LBP problems are multi-dimensional, involving anatomy, biomechanics, and important psychosocial factors.<sup>1</sup> In fact, a back pain diagnosis is often dependent on the theoretical perspective of the health care practitioner treating the condition. In general, the type of health care specialist chosen mainly determines an individual's treatment options.<sup>13</sup> In LBP, these options run the gamut from surgery to soft-tissue therapy.

Despite the fact that a large percentage of the population develops back pain, the ability to effectively treat this problem has lagged. A large percentage of patients report dissatisfaction with the quality of care they receive for back pain.<sup>14,15</sup> This is one of the primary reasons that individuals seek help from various alternative therapies, such as massage. Non-specific back pain often has a muscular origin, resulting from hypertonicity or myofascial trigger points.<sup>16,17</sup> Massage is an ideal therapeutic intervention in many of these cases.<sup>18-23</sup>

## INJURY CONDITIONS

### NEUROMUSCULAR LOW BACK PAIN

#### Description

Muscular dysfunction is one of the most common sources of LBP and can lead to altered movement patterns as well as postural stress in both standing and sitting positions. The postural distortions that occur in the spine have led clinicians to focus on the bones of the spine as the root of the problem, with an associated emphasis on joint pathology.<sup>24</sup> In a large number of cases, however, the problem is muscular in nature, with postural changes a result not the cause.

Myofascial trigger points in muscles such as the quadratus lumborum, erector spinae, multifidi, and other short intrinsic muscles of the spine cause pain referral patterns in the back. A sudden and awkward loading movement or trauma often activates trigger points. Perpetual trigger points can become a chronic source of back pain if they are not properly neutralized.<sup>25-27</sup>

In some cases, a seemingly benign activity, like reaching down to pick up a pencil, can cause acute muscular back pain and the development of

subsequent trigger points. In these cases, lumbar musculature is close to a level of fatigue, and all it takes is a little bit of additional stress to cause overload and subsequent dysfunction. This kind of injury often results from the combined motions of lateral flexion and rotation, which put the lumbar spine in a mechanically disadvantaged position.<sup>28</sup> Recent studies in spinal biomechanics indicate that neuromuscular pain and acute back injury often start with dysfunctional coordination between muscle recruitment and fascial tension in the lumbar spine.<sup>29,30</sup>

Constant neuromuscular tension can produce a number of postural distortions. These postural distortions, conversely, can produce excess neuromuscular activity and create muscular imbalance. For example, the quadratus lumborum can be the cause of an apparent leg length difference because the muscle pulls superiorly on the pelvis. The difference in leg length can then cause neuromuscular dysfunction in the lumbar muscles, especially the quadratus lumborum. A cycle of pain and discomfort is the result.

Due to the nature of motor learning, movement and dysfunctional postural distortions follow a pattern that, once set, tend to be repeated.<sup>31</sup> Patterns of pain or dysfunction occur in the same location repeatedly, especially in the stabilizing postural muscles of the spine. Individuals with back pain regularly describe an area to which the pain always returns whenever their problem flares up.

Biomechanical ramifications of muscular dysfunction, while sometimes subtle, can have far-reaching effects. Movement of the spine is an integrated process of motion between each of the different functional segments. Restricted motion at one vertebral segment (from either joint pathology or soft-tissue dysfunction) can increase or decrease motion at another segment. The subsequent lack of proper coordination leads to mechanical overload and neuromuscular dysfunction of the numerous thoracic and lumbar muscles.<sup>32</sup>

#### Treatment

##### *Traditional approaches*

For many years bed rest was a treatment suggestion for muscular LBP. That idea has changed as evidence now suggests that bed rest is more detrimental than helpful.<sup>1,33</sup> Prolonged immobilization appears to cause further muscle splinting and limitations to

improved range of motion, despite the initial pain relief that may be felt during the rest. A complication of prolonged bed rest for back pain is the development of deep vein thrombosis in the lower extremity.<sup>34</sup>

Non-steroidal anti-inflammatory drugs (NSAIDs) are used with great frequency for neuromuscular back pain. In many cases there is no muscular inflammation, even though muscle is the primary pain-producing tissue in the condition. The rationale for use of anti-inflammatory medications is then questionable. The detrimental effects of prolonged NSAID are a factor in reconsidering this approach. Corticosteroid injections are also used for their anti-inflammatory and pain management properties.<sup>35,36</sup>

Physical medicine approaches, including exercise, stretching, or educational programs, have met with clinical success.<sup>37,38</sup> Physical intervention along with the active involvement of the patient are important components of this approach. Manipulation and joint mobilization also achieve favorable results in many cases.

### Soft-tissue manipulation

**General guidelines** While there is a limited research base for clinical massage, LBP is one area that is studied more than others. At this point the evidence is strong that massage is very helpful in neuromuscular LBP problems.<sup>18,19,21,39</sup> Myofascial techniques are a great way to start treatment of neuromuscular back pain. They are generally performed before lubricant is applied and produce an initial soothing sensation for the client. In addition, neurological responses in the fascia encourage reductions in muscular tension.<sup>40</sup> Myofascial techniques can be followed up with effleurage and sweeping cross fiber treatments to the back muscles. These techniques are effective at reducing superficial muscle tension, increasing tissue circulation, and enhancing pliability in the subcutaneous fascia. As treatment progresses pressure levels can increase to access the deeper muscles. Increasing pressure levels enhances the neurological and mechanical effects that help reduce tension throughout the spinal muscles.

Various deep and specific massage techniques are an excellent method for treating the muscular tension that is at the root of neuromuscular LBP. Static

compression, deep longitudinal stripping, pin and stretch, and active-assisted massage techniques are all effective methods for addressing neuromuscular back pain. Each of these techniques can be modified with different positions or methods to address specific muscles.

**Suggested techniques and methods** The suggested treatments section is expanded here to provide more attention to the different techniques that can be used to address the muscles in this area. Yet, this selection is only a small fraction of the many approaches and variations that are used to treat LBP.

**A. Myofascial approaches** These techniques can be performed in many areas of the lumbar or thoracic regions. The primary goal is to place a moderately light tangential (tensile) force on the subcutaneous fascia. Pulling the fascia in multiple directions enhances its pliability. Place the hands lightly on the client's back in the region where the myofascial stretch is to be applied. Pull the hands apart to take the slack out of the tissue and apply a light degree of tensile (pulling) force between the hands (Fig. 9.1). Once there is a slight degree of pull between the hands, hold this position until a subtle sensation of tissue release is felt between the hands.

**B. Effleurage and sweeping cross fiber** These techniques are some of the most effective methods for achieving relaxation of the superficial back muscles. They also produce extensibility in the superficial fascia. Effleurage is performed with long gliding strokes parallel to the muscle fiber



Figure 9.1 Myofascial techniques on lumbar area.



Figure 9.2 Sweeping cross fiber to lumbar muscles.

direction. The cross fiber techniques sweep diagonally across the primary fiber direction of the muscles. Perform both throughout the low back (Fig. 9.2). Increasing levels of pressure are used to access deeper muscles.

**C. Deep longitudinal stripping to spinal extensors** Deep stripping techniques are applied to the erector spinae and spinal extensor muscles. Use a broad contact surface of pressure at the outset, especially if there is a greater degree of tension in these muscles. After relaxation of superficial tissues and to work more specifically on particular regions of the muscle, apply the stripping techniques with a small contact surface pressure, such as the finger tips, thumb, or pressure tool (Fig. 9.3).

**D. Deep longitudinal stripping on quadratus lumborum** The client is in a prone or side-lying position. Use the thumb or fingertips to perform a longitudinal stripping technique on the quadratus lumborum (Fig. 9.4). Use stripping motions



Figure 9.3 Deep stripping on erector spinae.



Figure 9.4 Deep stripping on quadratus lumborum.

from the iliac crest to the transverse processes, iliac crest to twelfth rib, and transverse processes to twelfth rib as the quadratus lumborum has fibers running in all these directions. When working from lateral to medial on the fibers running from the iliac crest to the transverse processes, apply pressure deep enough to treat up under the lateral edge of the erector spinae muscle group. However, use caution not to apply too much pressure directly against the tips of the transverse processes.

**E. Static compression** To reduce muscular hypertonicity in a specific location, use static compression methods. With greater muscle tension, use a broad contact surface first such as the back side of the hand, palm, or fist. To treat more localized areas of tension or specific myofascial trigger points, use static compression with a small contact surface directly on those areas of increased tension (Fig. 9.5). Pressure maintained for 8–10 seconds is usually sufficient to achieve a reduction of muscle tension.

**F. Deep stripping in lamina groove** Numerous intrinsic muscles of the spine are difficult to treat unless deep specific work with a small contact surface is applied. Use the thumb, finger tip, or pressure tool to apply deep longitudinal stripping techniques to these muscles in the lamina groove. A hand position that uses two thumbs is the most effective way to perform this technique. This technique can be performed moving in a superior or inferior direction. The thumbs are positioned at right angles to each other. One thumb applies pressure against the spinous processes, while the other is applying pressure forward in the direction



Figure 9.5 Static compression (small contact surface) on lumbar muscles.

that the hands are moving (Fig. 9.6). Pause and repeat short stripping movements on any areas where increased muscle tension is palpated or the client reports greater tenderness.

**G. Pin and stretch for quadratus lumborum**

This technique uses the pin and stretch concept along with eccentric activity in the quadratus lumborum (QL) to reduce hypertonicity. The client is in a side-lying position. Bring the client's thigh into a position of abduction to shorten the QL muscle. Abducting the thigh shortens the QL because when the thigh is abducted the pelvis lifts a little higher on the same side. Ask the client to hold their leg in that position (as long as this does not produce further muscle pain in a client with muscle spasm). Apply static compression to the QL with the thumb while the client is holding the leg in the abducted position. Continue to



Figure 9.6 Deep stripping to the lamina groove.



Figure 9.7 Pin and stretch to quadratus lumborum. Photo shows position at the end of the stretch procedure.

apply the compression as the client slowly lowers the leg off the back-side of the table (Fig. 9.7).

This can feel intense for the client so gauge the pressure carefully as the technique is applied. A variation on this technique can be used by applying a stripping technique during the client's eccentric adduction of the thigh instead of static compression. This is even more effective in reducing tension in the QL.

**H. Active assisted stretching for quadratus** The client is in a side-lying position. Have the client reach up overhead and grasp the opposite end of the treatment table to both stabilize the torso and further stretch the lateral trunk muscles. Bring the client's thigh into a position of abduction to shorten the QL muscle just as in G above. Ask the client to hold the thigh in this position for 5–8 seconds, producing an isometric contraction in the QL. Have the client release the contraction and help them lower the abducted thigh off the back side of the table. As the thigh gets near the end range of motion press down on the thigh with one hand while the other hand pushes the client's pelvis in an inferior direction, creating a stretch (Fig. 9.8).

**Rehabilitation protocol considerations**

- Prior to constructing a treatment program for neuromuscular back pain, complete a thorough assessment to rule out more serious pathologies that need referral to another health professional.
- Facilitated patterns of postural distortion are an impediment to lasting change for



**Figure 9.8** Active assisted stretching for quadratus lumborum. Photo shows position at the end of the stretch procedure.

neuromuscular back pain. Even after an effective massage treatment that provides relief, the neuromuscular pain pattern can immediately recur when long-established dysfunctional movement or postural patterns are repeated. It is important to encourage changes in these movement or postural patterns early in the treatment process as muscle tension is addressed through massage treatment.

- Strengthening techniques are often suggested by physical therapists or exercise specialists as a means of addressing neuromuscular back pain. If the strengthening activities are engaged too early in the rehabilitative process, they can reinforce dysfunctional patterns of muscle tension or posture. Massage can help reinforce gains made with strengthening or conditioning activities as long as it is later in the rehabilitation stage. Stretching and flexibility enhancement are essential components of treating neuromuscular back pain. Stretching is most effective when it performed after the soft-tissue manipulation so the benefits of enhanced tissue pliability can be maximized. If strength training is used in the rehabilitation program, stretching should be an integral aspect of the treatment process. Stretching is beneficial after the exercises to reduce any lingering muscle tension.

**Cautions and contraindications** Pay close attention to the pain reported by the client when working in this area. The quality of pain the client

reports helps determine the primary tissues at fault. There can be other causes of LBP, such as systemic disorders, tumor, or infectious processes, and many of these conditions can mimic muscular pain problems. The general guideline of common sense should apply here. If there is doubt about the nature of the client's condition, refer that person to another more qualified practitioner for further evaluation.

Sometimes neuromuscular back pain appears to resolve while the client is on the treatment table, only to return when the client stands or performs some slightly awkward movement, such as getting dressed after the treatment. Decreasing pain-producing tension on the muscular soft tissues in the lumbar and thoracic region can dramatically alter muscular proprioception. When the individual moves around after treatment there are very different muscular recruitment patterns being used and these new patterns can overtax certain tissues that have not appropriately developed to accommodate for the new neuromuscular patterns. The body's reaction to the sudden overload is muscle spasm. The best way to avoid that occurring is to remind the client to move slowly and carefully when first getting up from the massage treatment and for a short time afterward.

#### Box 9.1 Clinical Tip

Despite the advances in high-tech diagnostic tools such as X-ray or MRI, these tools are not very helpful in identifying neuromuscular low back pain. One of the most valuable tools for identifying muscular back pain is the trained palpation of practitioners specializing in soft-tissue manipulation. Knowledge of anatomy combined with specialized palpatory skills enhances the practitioner's ability to accurately identify tissues responsible for neuromuscular pain complaints. The ability to identify the source of neuromuscular soft-tissue pain and then immediately treat it with manual therapy is a distinct advantage for the massage practitioner in addressing numerous cases of low back pain.

## HERNIATED NUCLEUS PULPOSUS

### Description

The herniated nucleus pulposus (HNP) is routinely considered a primary cause of LBP, especially if that pain involves neurological symptoms. This problem is also known as a herniated disc, or inappropriately in laymen's terms, as a *slipped disc*. The diagnosis of disc herniation as a cause of back pain has become so extensive in the medical field that it has been referred to as the *dynasty of the disc*.<sup>1</sup>

One of the first articles to appear in the scientific literature indicating the intervertebral disc as a cause of back pain was the paper published by Mixter and Barr in 1934.<sup>41</sup> After their original article, there were numerous other studies published in medical journals that set out to prove the intervertebral disc as being at fault in many, if not most, LBP cases. The Mixter and Barr article appeared at a time when there were significant developments occurring in surgical techniques that made back surgery more feasible. Because discs could be observed protruding near nerve roots, it was a logical conclusion to assume that they were the cause of much back pain. However, as it turns out, this concept contained a jump in logic that has not proven accurate now that more sophisticated evaluation techniques have been developed.

Herniation technically means a pushing through. The primary problem in this condition is that degeneration of the annulus fibrosis allows the nucleus to push through it (Fig. 9.9). As the nucleus continues to press into the annulus, it causes the annulus to change shape. Eventually, if not halted, the nucleus can push all the way through the annulus. Degeneration of the annulus can be the result of numerous factors, including poor disc nutrition, loss of viable cells, loss of water content, and others.<sup>42</sup> These problems typically originate from chronic excessive compressive loads on the spinal structures.

There are several names given to disc herniations, and these reflect the level of severity of the disc damage. These names are not always consistent in the literature, but they do give a greater degree of specificity as to the severity of the herniation.<sup>43</sup> Figure 9.10 illustrates the different types of disc herniation. In a disc protrusion (also called

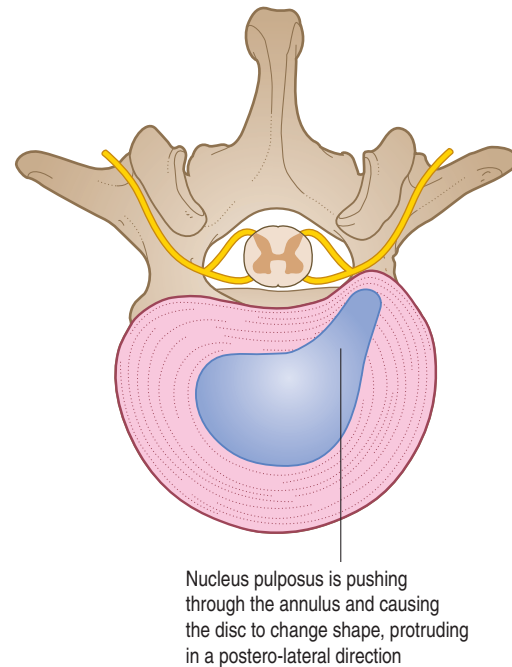


Figure 9.9 Herniated nucleus pulposus pressing toward a nerve root.

a bulge) the disc has changed shape, but the majority of the annulus fibrosus is still intact. Another type of disc protrusion is called a prolapse. In a prolapse the nucleus has not yet broken through the outer barriers of the annulus, but only the outermost fibers of the annulus are containing the nucleus. In an extrusion, the disc material has pushed through the outer border of the annulus, but is still connected to the nucleus in the center of the disc. The final stage of degeneration is the sequestration. In this stage, the disc material has actually separated from itself, and portions of the disc material can be floating freely in the spinal canal.

One of the apparent clinical challenges is trying to decipher when disc herniation is actually a cause of back pain. LBP can be a co-existing symptom along with disc herniation, but not necessarily caused by the disc herniation itself. A number of recent studies using magnetic resonance imaging (MRI) have examined low back structure in people without back pain. These studies show that herniated discs are often present in asymptomatic

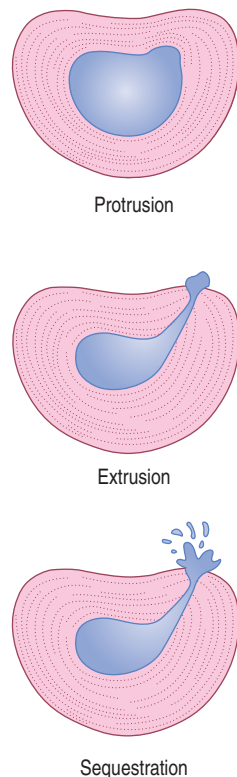


Figure 9.10 Different types of disc herniation.

individuals, which indicates that disc pathology is often not the source of LBP.<sup>44–48</sup>

When a lumbar disc protrudes against a nerve root, it is likely to cause symptoms in the distribution of that nerve root. Therefore, symptoms that are not neurological in nature and confined to the back are less likely to be coming from a disc pressing on a nerve root. However, if the symptoms are in the lower extremity and in characteristic dermatomes or myotomes, there is a greater likelihood of nerve root involvement.

## Treatment

### Traditional approaches

The HNP is usually treated conservatively with rehabilitative exercises. The exercise program developed by New Zealand physiotherapist Robin McKenzie has been quite effective for relieving symptoms thought to be originating from disc herniation.<sup>38,49,50</sup> In many cases, postural retraining along with rehabilitative exercise is sufficient for resolving the symptoms of disc herniation.<sup>51</sup>

Corticosteroid injections have been used with some success. There does appear to be some short-term pain relief with their use, although whether or not there is a long-lasting benefit is questionable.<sup>35</sup> Inflammation can be present, but is usually not a dominant aspect of this condition. The primary benefit of corticosteroid injections is pain relief more than reduction of inflammation.

Another procedure that has been used with some degree of success is chemonucleolysis.<sup>52,53</sup> In this procedure, a derivative of the papaya enzyme is injected into the area of the protruding disc. The papaya enzyme breaks down the protruding disc material reducing the likelihood of its pressing on nearby nerve roots. This treatment is used less now due to problems with allergic reactions to the papaya derivative.<sup>54,55</sup>

One of the more recent treatment methods is percutaneous laser disc decompression (PLDD).<sup>56–58</sup> This is a procedure in which a reduction in disc pressure is achieved through a laser treatment. A needle is first inserted into the nucleus pulposus under local anesthesia. A small amount of the nucleus pulposus is vaporized with the laser energy. As a result there is a sharp decline in pressure within the disc and the herniation moves away from the nerve root. This procedure is performed on an outpatient basis, and requires no general anesthesia and greatly reduces rehabilitation time.<sup>59</sup>

Surgical approaches for treating disc herniation have been used extensively, although this trend is decreasing. Traditional procedures are laminectomy (removal of a portion of the lamina) or discectomy (removal of a portion of the protruding disc).<sup>60</sup> Because it appears that people can have herniated discs and no back pain, the need for surgery seems less urgent. In fact, some have suggested the need for disc surgery to be about 2% of the individuals with a diagnosis of herniated nucleus pulposus.<sup>61</sup>

There is increasing concern about the use of surgery for low back problems, particularly because there is no clearly defined pathological process in a large number of cases.<sup>62</sup> In the United States the rate of surgery for back pain is at least 40% higher than in any other country.<sup>63</sup> It appears that back surgery increases as the supply of orthopedic and neurosurgeons in the country increases. Whether there is excessive surgery or if those in other



countries are suffering from lack of surgery is yet to be determined.<sup>63</sup> What can be said is that in many cases conservative and less invasive treatments are more effective than surgical intervention for symptoms of disc herniation.

### *Soft-tissue manipulation*

**General guidelines** Soft-tissue treatment is not necessarily contraindicated for HNP, but should be used with caution. It is always a good idea to have the client evaluated by another health professional if a disc herniation is suspected. Determining the level of disc herniation will determine what level of soft-tissue treatment is acceptable.

Massage does not directly reduce disc herniation. However, disc herniation often results from chronic compressive loads over time and hypertonic lumbar muscles can be a major factor in producing these loads. Addressing the muscular components of the compressive loads can help reduce factors that aggravate the disc herniation.

**Suggested techniques and methods** The focus of massage treatment for disc herniations is to reduce the role that muscle tightness plays in the disc pathology. Attention is focused on low back muscles that contribute to the compressive forces on the spine. Any of the techniques discussed in the previous section on neuromuscular back pain could be used to address the muscular components that aggravate a disc herniation.

### **Rehabilitation protocol considerations**

- The primary focus of treatment is to reduce compression on neurological structures. Massage can be performed concurrently with other traditional conservative treatments such as manipulation or mobilization. Massage is a valuable adjunct for those treatments and is especially valuable if performed prior to those treatments. Reduction in muscle and soft-tissue tension allows these other techniques to be applied with greater ease and less resistance.
- Movement reeducation to reduce postural stress is an important aspect of treatment and can also be performed concurrently with massage treatment.
- If there is an acute disc herniation, significant muscle spasm can result. Protective muscle spasm

should be addressed first in order to reduce the perpetuation of dysfunctional muscular activity.

- Massage is also a valuable adjunct treatment in post-surgical rehabilitation. After surgery massage treatment should not be used anywhere near surgical incision sites, but is effective in reducing excess muscle tension and restoring proper neuromuscular activity in the affected muscles.
- If the disc herniation has become a chronic situation, slightly more aggressive massage treatment can be attempted, but treatment should never aggravate the client's symptoms.

**Cautions and contraindications** Because the symptoms that can occur with herniated discs involve neurological sensations such as paresthesia, numbness, or motor disturbance in the lower extremity, other causes of these problems should be investigated as well. If symptoms are bilateral, a cauda equina syndrome (pressure directly on the spinal cord) could exist and should be immediately referred to a physician for proper evaluation.

Massage should not aggravate symptoms of disc herniation, so if there is an increase in neurological sensations after massage treatment, the treatment should be adjusted accordingly. Use caution when performing techniques close to the spine. The location of disc herniation is not vulnerable to massage treatment because the transverse processes of the spine protect the region. However, any technique that puts anteriorly directed pressure on the spine could move vertebrae in a way that aggravates nerve-root compression.

## ZYGAPOPHYSIAL (FACET) JOINT IRRITATION

### Description

The zygapophysial, or facet joints, are responsible for guiding the degree and orientation of movement in different regions of the spine. In the lumbar region, the angle of the facet joints is mostly vertical (parallel with the sagittal plane), so there is more movement allowed in flexion and extension. Further up the spine in the thoracic region, the facet joints are more obliquely aligned, which allows for less flexion and extension, but

more rotation in the thoracic region. A functional unit of the spine is composed of two vertebrae and the intervertebral disc between them. There is a good indication that facet joint pain may be at least partially occurring from a lack of proper mobility in the functional units of the lumbar spine.<sup>64</sup>

In the spine, the vertebral body is the primary weight-bearing structure. The intervertebral disc sits directly on the body of the vertebra and acts as a cushion. There is some weight-bearing capacity of the posterior arch of the vertebrae. Thus, the contact points between adjacent vertebrae (the facet joints) are a partial weight-bearing joint. During extension, the center of gravity moves in a posterior direction. As a result, the posterior vertebral structures – the lamina, pedicle, pars interarticularis, posterior portion of the intervertebral disc, and facet joints – carry an increased percentage of weight. These structures are not designed for the weight increase and spinal pathology, such as facet joint dysfunction, disc herniation, spondylolysis, or spondylolisthesis, can result.

The amount of weight carried by the facet joints increases when the spine is in extension. When the spine is in extension, the center of gravity is more posterior, and this causes the facet joints to carry a greater load. There is greater weight carried by the facet joints in regions of the spine that have lordotic curvatures because the joints are already in extension. Due to the greater lordotic curvature, the low back region carries the greatest load. As a result, facet joint irritation is more common in this area than in other regions. Exaggerated lumbar lordosis increases the likelihood of facet joint irritation.

No specific tissue has been identified as the primary cause of pain in facet joint dysfunction, but there are several commonly suggested. The joint capsule is richly innervated, and certain postural strains on the facet joints can stretch or pinch capsular fibers causing significant pain. Chondromalacia of the joint surfaces, as well as capsular or synovial inflammation, has also been suggested as a cause of pain.<sup>31</sup>

Facet joint pain can be similar to pain that originates from other lumbar structures. For example, injection of a fluid irritant into the facet joints causes referred pain patterns similar to those of lumbar disc pathology.<sup>65</sup> There are certain signs

and symptoms that appear consistent with facet joint irritation. However, there is no gold standard for identifying facet joint pain and, therefore, it remains difficult to accurately identify and treat.<sup>66</sup>

## Treatment

### *Traditional approaches*

Oral anti-inflammatory medication is often prescribed for facet joint irritation. However, the purpose of anti-inflammatory medication is unclear, as the presence of inflammation is not always demonstrated in facet dysfunction. This could be the reason for variable effectiveness with anti-inflammatory medication.

Other conservative forms of treatment include instruction in body mechanics, stretching, and strength training. Instruction in body mechanics is helpful if the individual has a tendency toward an exaggerated lumbar lordosis. The client will benefit by reinforcing the postural corrections on a regular basis, otherwise treatment results generally do not last. Because excessive lordosis compresses the facet joints further, improvement of this postural distortion is one of the most important parts of a therapeutic approach.

Cryotherapy is sometimes used to address inflammatory processes in the facet joints. The effectiveness of this approach may be limited, because the presence of inflammation is not a consistent finding in facet joint dysfunction.<sup>67,68</sup> Facet joint injections may be used for diagnostic as well as treatment procedures. However, there is controversy about the effectiveness of this procedure, despite the fact that use of these injections is widespread. Corticosteroid injections into the facet joints have not been found to be of much value in treatment.<sup>69</sup> The lack of agreement in treatments for facet joint problems suggests that more research is needed to identify effective approaches.

### *Soft-tissue manipulation*

**General guidelines** A challenge for the soft-tissue practitioner treating facet joint dysfunction is how to restore proper joint biomechanics without increasing further trauma or aggravation in the area. Massage treatment has limited effectiveness in directly affecting joint biomechanics. However, a number of techniques can help to improve

proper joint function and, thus, reduce the aggravation of facet joint dysfunction.

Soft-tissue treatment begins with improved body mechanics that decrease irritation on the aggravated joint structures. Soft-tissue manipulation is enhanced if instruction in proper body mechanics is consistently followed during the treatment. As there is no clear-cut cause of a majority of facet joint dysfunction, the ideal treatment protocol remains unclear. However, reducing tension in the intrinsic spinal muscles is an important treatment goal as tightness in these muscles can contribute to facet joint dysfunction.

The muscular components of postural distortion that perpetuate excessive lumbar lordosis and increased facet joint compression must also be addressed. Massage treatment should emphasize the lumbar extensors, iliopsoas, and the rectus femoris, which all contribute to excessive lumbar lordosis. These muscles are effectively treated with techniques such as deep longitudinal stripping and active-assisted stretching procedures.

Stretching is helpful to reduce chronic muscular tension. At home the client should emphasize stretching in forward flexion and lateral flexion of the lumbar region. These are the areas, when hypertonic, that are most likely to aggravate facet joint compression.

### Suggested techniques and methods

**A. Deep longitudinal stripping to spinal extensors** Deep stripping techniques are applied to the erector spinae and spinal extensor muscles. Use a broad contact surface of pressure at the outset, especially if there is a greater degree of tension in these muscles. Once the muscles have begun to relax, more specific muscle treatment can be applied using stripping techniques with a small contact surface such as the finger tips, thumb, or pressure tool (Fig. 9.3).

**B. Deep longitudinal stripping on quadratus lumborum** The client is in a prone or side-lying position. Use the thumb or fingertips to perform a longitudinal stripping technique on the QL (Fig. 9.4). Use stripping motions from the iliac crest to the transverse processes, iliac crest to twelfth rib, and transverse processes to twelfth rib as the QL has fibers running in all these directions. When treating from lateral to medial on the fibers

running from the iliac crest to the transverse processes, apply pressure deep enough to work up under the lateral edge of the erector spinae muscle group. However, use caution not to apply too much pressure directly against the tips of the transverse processes.

**C. Deep stripping in lamina groove** Use the thumb, finger tip, or pressure tool to apply deep longitudinal stripping techniques to the muscles in the lamina groove. A hand position that uses two thumbs is the most effective way to perform this technique. This technique can be performed moving in a superior or inferior direction. The thumbs are positioned at almost right angles to each other. One thumb applies pressure against the spinous processes, while the other is applying pressure down and forward in the direction that the hands are moving (Fig. 9.6). Pause and repeat short stripping movements on areas where increased muscle tension is palpated or the client reports greater tenderness. Increased tension in the intrinsic spinal muscles is likely to be near the facet joint compression, so spend more time treating these areas. Some practitioners advocate working in an inferior to superior direction (opposite the direction of facet joint compression due to gravity) in order to decrease compression between adjacent vertebrae. However, there is no clear evidence that one is better than the other.

**D. MET for iliopsoas** The client is in a supine position with one thigh hanging off the side of the table. The client holds the opposite thigh in a fully flexed and bent knee position. The client attempts hip flexion of the hanging thigh, while the practitioner offers resistance. The client holds the contraction for about 5–8 seconds, and then releases the contraction. As the client releases the contraction, the practitioner pushes the thigh into extension to stretch the iliopsoas muscle (Fig. 9.11). If the client experiences discomfort, instruct the client to further flex the opposite hip, which will increase rotation of the pelvis, straighten the spine, and reduce facet joint compression.

### Rehabilitation protocol considerations

- In some cases facet joint dysfunction results from other conditions affecting spinal biomechanics, such as intervertebral disc degeneration. In these cases the dysfunctional spinal biomechanics or



Figure 9.11 MET in Thomas test position.

positions will need to be addressed sufficiently in order to resolve the facet joint dysfunction. Massage can be performed simultaneously with those other approaches.

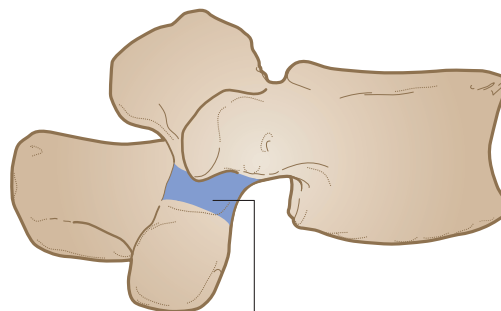
- Stretching is a valuable adjunct for improving postural strain that leads to facet joint irritation. Stretching procedures are more helpful if soft-tissue treatment, such as massage, is performed prior to the stretching.
- Strength training and conditioning can be helpful in establishing new movement patterns in the lumbar and trunk muscles. These approaches are most effective if there have been a number of massage treatments and reinforced postural changes prior to initiating the strengthening program. Otherwise, the benefits of soft-tissue treatment can be eliminated when the person walks out the door if they immediately adopt dysfunctional postural patterns.

**Cautions and contraindications** Symptoms of facet syndrome can be the same as many other back disorders, some of which are more serious. If symptoms get worse as a result of treatment, cease that approach and reinvestigate the problem. The client may need to be referred to another health care provider for further evaluation.

## SPONDYLOLYSIS AND SPONDYLOLISTHESIS

### Description

While these are two separate conditions, they are closely related to one another and are treated the



The region between the superior and inferior facets is the common site of stress fractures in spondylolysis and spondylolisthesis

Figure 9.12 Lateral view of a lumbar vertebra showing the pars interarticularis – site of stress fractures.

same, so they are described here together. By definition, spondylolysis is a breakdown of the vertebral body. The breakdown is a stress fracture to a region of the vertebra called the pars interarticularis (Fig. 9.12). The stress fracture results from repeated loads placed on the posterior aspect of the lumbar vertebrae. Most problematic are increased loads while the spine is in extension.<sup>70</sup>

The majority of weight is carried through the lumbar spine by the main body of each vertebra. However, during extension, the center of gravity moves in a posterior direction, and the posterior vertebral structures carry an increased percentage of that weight. Increased weight bearing, especially with repetitive or high-intensity loads, can lead to the development of stress fractures. Stress fractures produce pain themselves, but they also create problems as they progress into spondylolisthesis.

Spondylolisthesis is a forward slippage of one vertebra in relation to another and is often the result of bilateral spondylolysis. Once the stress fractures have occurred, on each side, forward slippage of the vertebra is more likely to occur. A common location for spondylolisthesis is at the L5–S1 junction (Fig. 9.13). Clients with spondylolisthesis report lumbar pain that is aggravated by strenuous activities, especially repetitive flexion and extension or hyperextension movements of the spine.

Spondylolisthesis is particularly common in the adolescent athletic population. The higher incidence of the condition in this group can be related

Bilateral stress fractures at the pars interarticularis have expanded allowing the body of the lumbar vertebra to migrate forward in relation to the adjacent vertebral or sacral segments

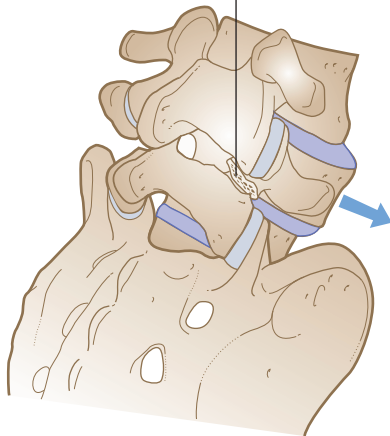


Figure 9.13 Spondylolisthesis at the L5–S1 junction.

to significant loads on the spine during skeletal immaturity. In adolescence bones are still developing some of their structural integrity and may not be ready to handle the load demand of vigorous athletic activities involving spinal extension.

In both conditions pain is diffuse in the lower lumbar and upper sacral regions. Pain is also common at the sacroiliac joint and there may be symptoms of radiating nerve pain with spondylolisthesis. Neurological sensations result from traction on the lower lumbar nerve roots and the cauda equina as a result of the anteriorly shifted vertebra.<sup>31,71</sup> Forward slippage of a vertebra also causes spinal stenosis (narrowing of the intervertebral foramen) at that vertebral level, which can increase the likelihood of nerve root impingement.<sup>72</sup>

One factor that makes the cause of pain confusing in spondylolysis and spondylolisthesis is that the severity of symptoms does not necessarily correlate with the degree of slippage.<sup>33</sup> An individual can have significant forward slippage with minimal pain where someone else may have only minor slippage, but experiences more pain.

Hamstring tightness is present in many individuals with spondylolisthesis. There is a strong proprioceptive function of the hamstrings as the body attempts to adjust to the forward slippage

of the lower lumbar vertebra. The hamstrings tighten in an effort to posteriorly rotate the pelvis. The posterior pelvic rotation decreases the potential for forward slippage of the lower lumbar vertebra and helps stabilize the lumbar region.<sup>73,74</sup>

## Treatment

### *Traditional approaches*

Treatment for spondylolysis is controversial. Because a stress fracture is involved, an important factor is reducing cumulative stress on the area so the fracture can heal appropriately. Activity modification is usually sufficient to reduce the cumulative trauma in the area. Some clinicians advocate rigid braces if bone scans reveal a greater severity of injury.<sup>70</sup> However, others question this protocol and propose rest from offending activities and stabilization exercises that emphasize flexion instead of extension (such as Williams flexion exercises) as a more valuable treatment.<sup>75</sup>

Treatment for spondylolisthesis follows the same protocol as that for spondylolysis. In most instances, conservative treatment is effective, and there is no need for surgery. If conservative treatment fails, surgery may be needed. Lumbar fusion is a procedure that is used in more severe cases.<sup>72</sup> Yet, some of the recent clinical evidence suggests that lumbar fusion may not be any more effective than the current methods of conservative treatment.<sup>60</sup> Additional research is needed to establish more definitive treatment guidelines for this problem.

### *Soft-tissue manipulation*

**General guidelines** Positioning on the treatment table is an important consideration in soft-tissue treatment for spondylolysis and spondylolisthesis. Back treatments are usually performed in a prone position, and include significant pressure applied to the lumbar area. A primary concern in these conditions is anterior translation of the lumbar vertebra. Caution is advised with techniques that put pressure on the lumbar area in an anterior direction as these could aggravate the problem. The client usually reports increased pain with pressure levels that aggravate the condition. Putting the client in a partially flexed position on the treatment table is helpful to reduce lumbar stress. Bolsters, pillows, or a number of commercially



Figure 9.14 Flexion protocol on cushioned support to keep the lumbar region in a partially flexed position.

available support cushions are useful for this positioning (Fig. 9.14).

The primary problem in these conditions involves structural deficiency in the bones, which soft-tissue manipulation can not change. The focus of soft-tissue treatment is to encourage restoration of biomechanical patterns that help reduce anterior vertebral translation.

#### Suggested techniques and methods

**A. Effleurage and sweeping cross fiber** Effleurage is performed with long gliding strokes parallel to the muscle fiber direction. The cross fiber techniques sweep diagonally across the primary fiber direction in different regions of the back (Fig. 9.2). Increasing levels of pressure are used to access deeper muscles. Use caution and relax pressure if the strokes increase the client's symptoms.

**B. Deep longitudinal stripping to spinal extensors** Deep stripping techniques are applied to the erector spinae and spinal extensor muscles. Use a broad contact surface of pressure at the outset, especially if there is a greater degree of tension in these muscles. Once the muscles have begun to relax, apply the stripping techniques with small contact surface pressure such as the finger tips, thumb, or pressure tool to work on specific regions of any particular muscle (Fig. 9.3).

**C. Deep longitudinal stripping to hamstrings** Reduction of hamstring tension can help aid the overall lumbo-pelvic balance. Start with a broad contact surface of pressure and then eventually perform the stripping techniques with a small contact surface (Fig. 9.15). Hamstring tightness is a result



Figure 9.15 Deep stripping to hamstrings.

of a compensation effort to posteriorly rotate the pelvis and decrease aggravating symptoms from anterior vertebral slippage. Some have suggested that massage should not be used on the hamstrings as it could reduce their ability to posteriorly rotate the pelvis and protect against anterior vertebral slippage. It is unlikely that reduction of tension in the hamstrings will decrease their effectiveness in making these biomechanical alterations. Once the individual stands and moves around, the body perceives a need for greater stability. At this time an appropriate protective contraction in the hamstrings is likely, even if they have been previously relaxed with massage treatment.

#### Rehabilitation protocol considerations

- Massage techniques are most helpful when they are performed in conjunction with exercise and activity modification. Massage can slightly alter soft-tissue proprioception. If massage is performed shortly before exercise, the client should slowly work into the exercise movements, allowing the nervous system time to integrate the new movement patterns.
- If the condition is severe, pressure on low-back muscles can aggravate pain too much and massage is not recommended. The bone and other soft-tissue damage will need time to heal prior to engaging soft-tissue therapy.

**Cautions and contraindications** Because the symptoms of spondylolysis and spondylolisthesis can mimic other lumbar pathologies, it is important to accurately identify these problems before initiating treatment. Palliative care can be given

to the individual as long as the practitioner avoids any activity that aggravates the symptoms. Use great caution in applying pressure techniques to the lumbar area as these could further encourage anterior vertebral translation.

## POSTURAL DISTORTIONS

The next section includes a number of structural and postural disorders of the lumbar and thoracic spine. These disorders are not considered injury conditions, as are the prior conditions in this chapter. However, they can produce considerable stress on other tissues or structures and contribute to their dysfunction. Massage is not always used to correct these postural deviations, but it may be a helpful approach to restoring appropriate biomechanical balance in the region.

## EXAGGERATED LUMBAR LORDOSIS

### Description

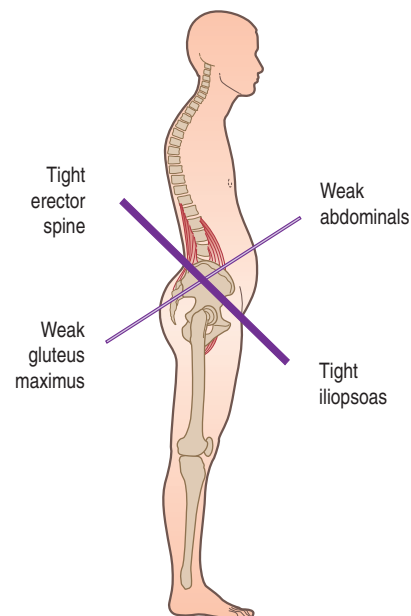
There is a natural lordotic curve in the lumbar region that is necessary for shock absorption. The lordotic curve is exaggerated during extension and is reduced during flexion. The primary muscles that produce spinal extension are the erector spinae group, quadratus lumborum, and several small intrinsic spinal muscles including the multifidi, rotatores, interspinales, and intertransversarii. LBP and myofascial trigger points can develop from hypertonicity in these muscles and exaggerate the lordosis.

In a normal lumbar lordosis the vertebral body is the primary weight-bearing structure. With an excessive lumbar lordosis, more of the body's weight is transferred to the posterior portion of the vertebrae. Increased weight is then borne by posterior vertebral arch structures such as the lamina, pedicle, pars interarticularis, posterior portion of the intervertebral disc, and facet joints. These structures are not designed for the weight increase and spinal pathology such as facet joint dysfunction, disc herniation, spondylolysis, or spondylolisthesis can result. Increased lumbar extension also narrows the intervertebral foramen and can lead to spinal nerve root compression.<sup>74</sup>

Dysfunctional patterns of muscular activation and poor posture can perpetuate an excessive lordosis. An

example is the lower crossed syndrome, originally described by Vladimir Janda.<sup>76</sup> He categorizes the body's muscles into two groups: postural or phasic muscles, which differ in their fiber type and activation patterns.<sup>77</sup> When overused and fatigued, postural muscles tend to become hypertonic, while phasic muscles tend to become weak and inhibited. The phasic muscles are antagonists to postural muscles. Because postural muscles tend toward hypertonicity, they create a functional weakness in the phasic muscles through the process of reciprocal inhibition.<sup>26</sup>

Postural muscles in the lumbar spine include the erector spinae, quadratus lumborum, and iliopsoas. Phasic muscles in this region include the abdominals, gluteus maximus and medius. An exaggerated lumbar lordosis also produces an anterior pelvic tilt and a postural distortion called the *lower crossed syndrome*. A graphical comparison of the functional and positional relationships of certain lumbopelvic muscles shows how they interact and where the term lower crossed syndrome originates from (Fig. 9.16). See the section in Chapter 8 on anterior pelvic tilts for additional descriptions of the lower crossed syndrome and reciprocal inhibition.



**Figure 9.16** Lower crossed syndrome (from Chaitow L, DeLany J. *Clinical Application of Neuromuscular Techniques*. Vol 1. Edinburgh: Churchill Livingstone; 2000).

## Treatment

### *Traditional approaches*

Traditional treatment approaches for exaggerated lumbar lordosis and the resulting lower crossed syndrome are the same as those described under the section on anterior pelvic tilt in Chapter 8.

### *Soft-tissue manipulation*

Soft-tissue treatment and rehabilitation protocol considerations for an excessive lumbar lordosis are the same as those described for anterior pelvic tilt in Chapter 8.

**Cautions and contraindications** Use caution with any iliopsoas treatment technique that uses direct pressure through the abdomen. There is a risk of adverse vascular responses with the external iliac artery if an aortic aneurysm exists. Some clients may report increased back pain or discomfort with MET stretching techniques for the iliopsoas performed from the supine position. To reduce client discomfort, have the client increase the level of flex in the opposite hip, which posteriorly rotates the pelvis, straightens the spine, and reduces facet joint compression.

### Box 9.2 Clinical Tip

One of the errors that health professionals make is looking at problems in certain regions as isolated mechanical or structural disorders. An excessive lumbar lordosis, for example, creates numerous patterns in other regions of the body. There is mechanical stress in the low back to be certain. However, the biomechanical ramifications of this postural distortion can produce numerous other pathologies. The excessive lumbar lordosis produces an anterior pelvic tilt. That anterior pelvic tilt causes increased length in the hamstring muscles. The increased hamstring length in an anterior pelvic tilt is associated with a higher incidence of hamstring strains. Therefore, preventive treatment of hamstring strains may need to focus on the low back region.

## KYPHOSIS

### Description

There is a natural slight kyphotic curvature in the thoracic region located between the lordotic curves of the cervical and lumbar spine. The primary function of this curve is shock absorption. Kyphosis occurs when the curvature is exaggerated and a subsequent postural distortion develops (Fig. 9.17). Kyphosis, also called hunchback, routinely occurs with advanced age and can also develop from chronically poor posture or degenerative changes such as osteoporosis.<sup>78</sup> The condition can adversely affect other physiological processes such as digestive function and breathing.<sup>79</sup>

When the thoracic kyphosis is increased, the head naturally tilts toward the floor. At the same time the body's righting reflex attempts to keep

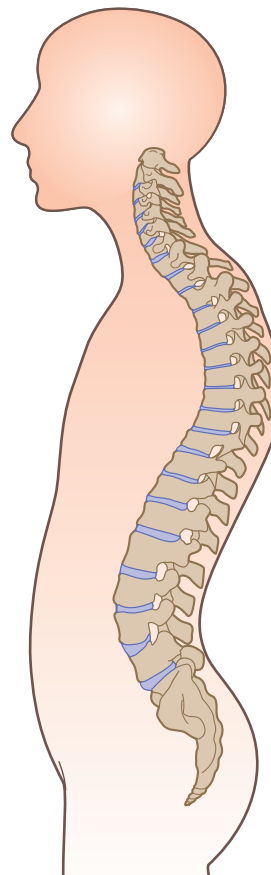


Figure 9.17 Kyphosis in the upper thoracic spine.



the eyes horizontal by contracting the cervical extensor muscles. The attempted postural compensation produces exaggerated compressive loads on the anterior aspect of the thoracic vertebrae and the posterior aspect of the cervical vertebrae. In some cases, the increased loads are enough to create vertebral stress fractures.<sup>80</sup>

An individual with kyphosis is likely to have an exaggerated cervical lordosis and forward head posture. Biomechanical patterns of muscular dysfunction, such as the upper crossed syndrome discussed in Chapter 10, are common in the client with kyphosis. In addition, there is increased tensile load on ligaments and muscle tissues in the posterior thoracic region.

Pathological kyphosis that causes clinical problems usually results from trauma, tumors, infection, tuberculosis, chronic postural stress, developmental disorders, rheumatoid arthritis, or other systemic conditions.<sup>81</sup> Bone weakness pathologies, such as osteoporosis or Scheuermann's disease, are also common causes of kyphosis. Osteoporosis involves a loss of bone density and is most prevalent in the elderly. Scheuermann's disease is a hereditary condition involving vertebral end-plate weakening and predominantly affects juveniles.<sup>82</sup>

Kyphosis can either produce or be caused by hypertonicity in the anterior chest muscles, such as the pectoralis major. Tightness in the anterior chest muscles pulls the arm into medial rotation and a corresponding scapular protraction develops. The altered scapular position limits function in the shoulder girdle, reducing range of motion and contributing to structural problems, such as shoulder impingement syndrome.<sup>83</sup>

Kyphosis does not necessarily produce pain or discomfort, but prolonged postural stress or more severe cases can produce a number of symptoms. Myofascial trigger points are typical in the upper thoracic or posterior cervical muscles and produce characteristic referral patterns. Pain in the upper thoracic region is typical and due to fatigue and overexertion in the upper thoracic spinal muscles.<sup>74</sup>

## Treatment

### *Traditional approaches*

Because kyphosis is a disorder of postural distortion, it is commonly treated using exercise and

postural re-education. The emphasis is on reducing the forward head posture, lifting the anterior chest region, and straightening the upper back. In more severe cases of structural kyphosis, such as those that result from Scheuermann's disease, surgical implantation of rods to straighten the spine may be used.<sup>84</sup>

### *Soft-tissue manipulation*

**General guidelines** This discussion of soft-tissue treatment for kyphosis assumes that the condition is primarily one of degenerative posture due to habitual neuromuscular patterns and not the result of problems with bony structure such as Scheuermann's disease or osteoporosis. In conditions of major bone structure deformity, massage can be used as palliative care or support for new neuromuscular patterns in the area, but it will not change the structural deformity.

Massage treatment for kyphosis focuses on the tissues that are short and hypertonic in the upper chest region, as well as those tissues that are fatigued from being held too long in an over-lengthened position in the upper back. On the anterior torso, attention is focused on the anterior deltoid, and pectoralis minor and major. In the upper back there are several layers of muscles that are emphasized in treatment including the rhomboids, middle and lower trapezius, serratus posterior superior, and intrinsic spinal muscles. These muscles are effectively addressed with a variety of techniques including static compression, deep longitudinal stripping, and active engagement methods.

### *Suggested techniques and methods*

**A. Sweeping cross fiber to anterior chest muscles** Treatments aimed at the anterior chest muscles reduce the medial shoulder rotation, scapular protraction, and thoracic kyphosis. This technique helps reduce tension in superficial fibers of pectoralis major and anterior deltoid. Stand facing the client's feet with the fingers anchored in the client's axilla. Use the thumb to perform sweeping cross fiber movements on the pectoralis major (Fig. 9.18). During the sweeping motion the pectoralis major is sifted between the fingers. Similar sweeping cross fiber techniques are applied to the anterior deltoid while facing in the same position (toward the client's feet).



Figure 9.18 Sweeping cross fiber on pectoralis major.



Figure 9.20 Deep stripping on pectoralis major.

**B. Static compression for anterior chest muscles**  
Apply static compression to areas of tension found in the pectoralis major when performing general warming and gliding techniques. Trigger points or areas of muscle hypertonicity are treated with both broad and small contact surface static compression methods (Fig. 9.19). Hold pressure on trigger points or areas of muscle tightness for about 5–8 seconds, or until a tissue release is felt.

**C. Deep stripping on pectoralis major** Use the thumbs, finger tips, or pressure tool to perform deep stripping techniques to the pectoralis major. Treatment can move from medial to lateral or lateral to medial (Fig. 9.20).

**D. Deep stripping on pectoralis minor** Perform a deep longitudinal stripping technique on the

pectoralis minor beginning at the coracoid process of the scapula and moving inferiorly to its attachments on ribs 3, 4, & 5 (Fig. 9.21). Performing this technique requires work directly through the pectoralis major, so use care in the amount of pressure applied as this region can be tender.

**E. Effleurage and sweeping cross fiber to upper back muscles** Use the palm, thumb, back of the hand, or other broad contact surface to apply effleurage and sweeping cross fiber strokes to the rhomboids and middle trapezius between the spine and the vertebral border of the scapula (Fig. 9.22). There are several layers of muscles being treated in this region, so it is advantageous to apply both the effleurage and sweeping cross fiber movements in multiple directions.



Figure 9.19 Static compression (small contact surface) on anterior chest muscles.



Figure 9.21 Deep stripping on pectoralis minor.



Figure 9.22 Sweeping cross fiber to rhomboids and mid trapezius.



Figure 9.23 Deep stripping to rhomboids, trapezius, serratus, etc.

#### F. Deep stripping to posterior back muscles

Perform deep longitudinal stripping techniques with a small contact surface to the rhomboids, mid trapezius and serratus posterior superior muscles (Fig. 9.23). There are likely to be myofascial trigger points in these areas and localized areas of tight fibers and increased tenderness. The tightness felt in these tissues is not from shortened muscles, but from taut muscles held for prolonged periods in a lengthened position. Static compression with a small contact surface can be applied to these tight focal regions to neutralize the muscle tissue dysfunction.

#### Rehabilitation protocol considerations

- In both the upper chest and upper back regions, there are several layers of muscles being treated.

It is important to relax the superficial muscles first prior to using greater pressure on the deeper muscles.

- Some people advocate that the shortened side (anterior chest muscles) should be treated prior to addressing the over-lengthened side (upper back). Generally this is a good guideline to follow. However, some individuals with kyphosis complain of much greater pain in the upper back region. Relieving pain in the back first appears to have beneficial effects on reducing tension in the anterior chest muscles. It is not clear that there is a firm guideline about which order to treat these areas, so experiment with both and communicate with the client.

**Cautions and contraindications** Some cases of kyphosis are caused by weakness and degeneration in bony structures as in osteoporosis. Many people develop their kyphotic posture with age as gravity gradually takes a toll on the upright vertical structure. In either case, especially that of osteoporosis, there can be fragility in the skeletal structures. Use caution when working in these areas, especially with techniques that use greater pressure, such as the deep stripping or static compression methods.

## SCOLIOSIS

### Description

Scoliosis is a lateral/rotary curvature in the spine and is relatively common, especially in children.<sup>81</sup> Most children grow out of scoliosis without need for further intervention. If the condition persists into adulthood, it can become seriously debilitating. The condition is caused by various diseases or muscular distortion.

There are two types of scoliosis: structural and functional. Structural scoliosis is caused by a fixed bony deformity, which can be inherited or acquired. A structural scoliosis is hard to correct and has detrimental long-term effects on spinal mechanics. The deformity could result from structural irregularities in the spine or a number of systemic disorders or neuromuscular

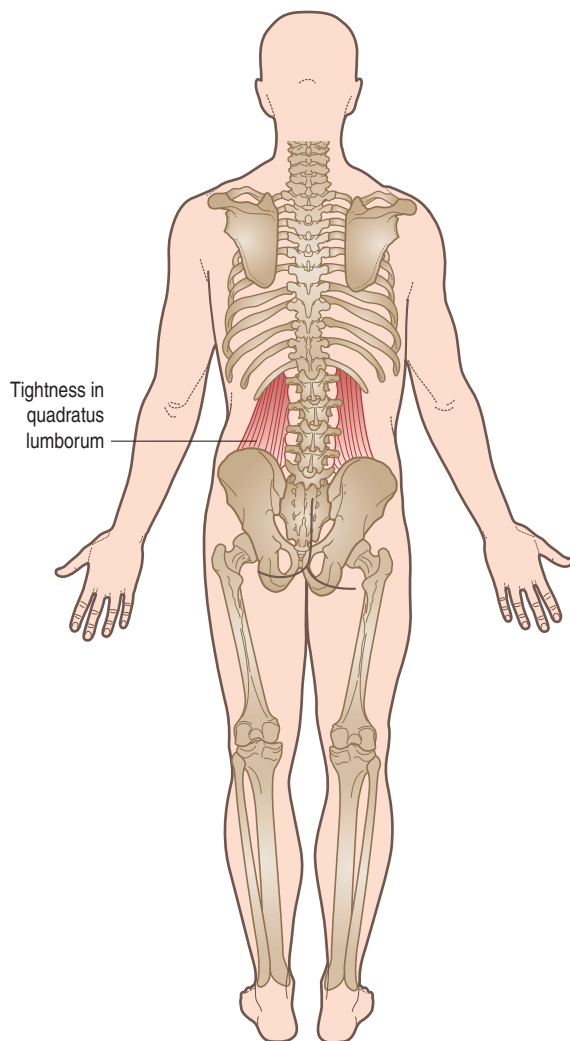


Figure 9.24 Functional scoliosis develops in the lumbar spine as a result of muscle tightness.

pathologies such as an upper or lower motor neuron lesion.<sup>43</sup>

Functional scoliosis develops from excessive muscle tension and not from deformity in the bones of the spine. For example, hypertonicity in the quadratus lumborum and iliocostalis lumborum produce a lateral pelvic tilt to the opposite side, creating a functional scoliosis in the lumbar region (Fig. 9.24).

In severe cases of scoliosis, especially structural forms, there is a characteristic rib hump visible in which one side of the back appears more pronounced in a posterior direction than the other.

The rib hump is often visible when the client is in a prone position and results from lateral curvature of the spine that also causes rotation of the individual vertebrae. The vertebrae rotate toward the side that is higher when the client is in a prone position.

If there is a single scoliotic curve, it is called a C curve. In other cases two curves are convex in opposite directions; this distortion is called an S curve. The scoliotic curve is named for the convex side of the curve. If, for example, the right quadratus lumborum is in spasm and the pelvis tilts to the left, a functional scoliosis in the lumbar region results that is convex on the left and concave on the right. When the convex side of the curve is to the left, the condition is called a levoscoliosis. If the convex side of the curve is to the right the condition is called a dextroscoliosis.<sup>74</sup>

While many cases of scoliosis begin as mild structural disorders, they can progress to more serious complaints because of the functional adaptation of muscles to the distorted postures. There is a focus on the role of paraspinal muscles and the quadratus lumborum in creating or perpetuating the lateral bending. The small intrinsic spinal muscles should not be overlooked, however, as critical components of scoliosis. Muscles such as the multifidi, rotatores, and transversospinalis govern spinal rotary movement and can play an important role in developing the condition.

## Treatment

### *Traditional approaches*

Structural scoliosis can often be prevented by detection early in life and use of corrective braces. If the scoliosis is severe and does not respond to braces or other conservative approaches, surgery might be suggested to straighten the spine. Surgical procedures used include spinal fusion and implantation of corrective devices such as Harrington rods. These are rods placed along each side of the spine and attached to the vertebral bodies to help keep the spine straight. The rods correct the lateral/rotary curvature and prevent the curvature from affecting other structures, such as spinal nerve roots or internal organs.

In certain cases a functional disorder involves skeletal structures, as in a structural leg-length discrepancy. The leg-length discrepancy can be corrected with orthotics and will help the scoliosis as well. Skeletal imbalances must be addressed first and then muscular compensations can be treated with massage.

### *Soft-tissue manipulation*

**General guidelines** Treatment of a functional scoliosis requires identification of biomechanical factors that led to the distortion. Massage is used to lengthen muscles that are shortened. In massage treatment of scoliosis emphasis is placed on those muscles on the concave side of the curve because they are shortened. For example, if the functional scoliosis results from a lateral pelvic tilt caused by muscular hypertonicity, attention should focus on reducing tightness or myofascial trigger points in the muscles on the concave side of the curve.

Massage treatments for scoliosis have not been adequately researched. Yet, there is reason to believe that treating hypertonic muscles on the concave side of the curvature is beneficial. Massage is also valuable for reducing general hypertonicity and pain associated with this postural disorder.

**Suggested techniques and methods** For the following techniques it is assumed that they will be applied in the lumbar region on the concave side of the scoliotic curvature. It may be beneficial to also apply the techniques to the convex side of the scoliotic curve, but emphasis in treatment should be on the concave side.

**A. Deep longitudinal stripping to spinal extensors** Deep stripping techniques are applied to the erector spinae and spinal extensor muscles. Use a broad contact surface of pressure at the outset, especially if there is a greater degree of tension in these muscles. Once the muscles have begun to relax, apply the stripping techniques with small contact surface pressure such as the finger tips, thumb, or pressure tool to work on specific areas of tightness in the muscles (Fig. 9.3).

**B. Deep longitudinal stripping on quadratus lumborum** The client is in a prone or side-lying position. Use the thumb or finger tips to perform

a longitudinal stripping technique on the QL (Fig. 9.4). Use stripping motions from the iliac crest to the transverse processes, iliac crest to twelfth rib, and transverse processes to twelfth rib as the QL has fibers running in all these directions. When working from lateral to medial on the fibers running from the iliac crest to the transverse processes, apply pressure deep enough to treat under the lateral edge of the erector spinae muscle group. However, use caution not to apply too much pressure directly against the tips of the transverse processes.

**C. Deep stripping in lamina groove** Numerous intrinsic muscles of the spine are difficult to treat unless deep specific work using a small contact surface is applied. Use the thumb, finger tip, or pressure tool to apply deep longitudinal stripping techniques to the muscles in the lamina groove. A hand position that uses two thumbs is the most effective way to perform this technique. The thumbs are positioned at almost right angles to each other. One thumb applies pressure against the spinous processes, while the other applies pressure down and forward in the direction that the hands are moving (Fig. 9.6). Pause and repeat short stripping movements on areas where increased muscle tension is palpated or the client reports greater tenderness. This technique can be performed in a superior or inferior direction.

### **Rehabilitation protocol considerations**

- If the scoliosis is a more advanced structural scoliosis, consult with the client's physician or other health care provider about strategies being employed to address the structural disorder. It is important that massage approaches work in conjunction with these other treatments.
- If the scoliosis is predominantly a functional scoliosis resulting from postural disorders such as a lateral pelvic tilt, be sure to address all components of those disorders as well as treating the lateral curvature in the spine.

**Cautions and contraindications** Some people with a congenital scoliosis may already have rods or implanted stabilizing devices along the spine. Use caution when performing any specific massage treatments along the spine for anyone with surgically implanted instrumentation.

**Box 9.3** Case Study**Background**

Dennis is a 49-year-old construction contractor. This season has been a particularly hard winter which has meant a lot more snow shoveling. A week ago there were three straight days of heavy snow, and Dennis had to shovel his driveway each of those 3 days. He also helps the elderly couple next door by shoveling their driveway. He is in good physical condition but hurt his back with all this snow removal work over the last week.

His injury started when he had been working on snow removal for about 45 minutes. He had a moderately heavy pile of snow on his shovel and had turned in a bit of an awkward position to lift it up and away from his car in the driveway. When he turned and lifted his shovel he felt a sudden sharp pain in his back. He immediately fell to his knees and had a hard time getting up. The injury forced him to miss work for several days because he was in such pain. He put ice on it right after the injury and that helped a little with the pain. He has also been taking over the counter anti-inflammatory medication for the last week to deal with the pain and discomfort.

It has been hurting ever since, but decreasing a little in intensity with time.

**Questions to consider**

- What are several possible conditions that could be causing Dennis' back pain?
- Do you think he should see another health professional for evaluation prior to receiving massage? If so, who do you think he should see?
- What are several factors in his case that indicate a likely biomechanical overload on various structures in his lumbar spine?
- If his condition turns out to be primarily a muscular injury, such as muscle spasm, would massage treatment be helpful? If so, what techniques would be most appropriate?
- Based on the limited information you have about Dennis, what are several muscles that you think might be injured in this condition?
- Do you think his injury might be neurological, such as a herniated disc? If so, is this something you think should be treated with massage?

**References**

1. Waddell G. *The Back Pain Revolution*. Edinburgh: Churchill Livingstone; 1998.
2. Frymoyer JW, Cats-Baril WL. An overview of the incidences and costs of low back pain. *Orthop Clin North Am*. 1991;22(2):263–271.
3. Dagenais S, Caro J, Haldeman S. A systematic review of low back pain cost of illness studies in the United States and internationally. *Spine J*. 2008;8(1):8–20.
4. Nguyen TH, Randolph DC. Nonspecific low back pain and return to work. *Am Fam Physician*. 2007;76(10):1497–1502.
5. Pai S, Sundaram LJ. Low back pain: an economic assessment in the United States. *Orthop Clin North Am*. 2004;35(1):1–5.
6. van der Roer N, Goossens ME, Evers SM, van Tulder MW. What is the most cost-effective treatment for patients with low back pain? A systematic review. *Best Pract Res Clin Rheumatol*. 2005;19(4):671–684.
7. Maetzel A, Li L. The economic burden of low back pain: a review of studies published between 1996 and 2001. *Best Pract Res Clin Rheumatol*. 2002;16(1):23–30.
8. Lorusso A, Bruno S, L'Abbate N. A review of low back pain and musculoskeletal disorders among Italian nursing personnel. *Ind Health*. 2007;45(5):637–644.
9. Lorusso A, Bruno S, L'Abbate N. Musculoskeletal complaints among Italian X-ray technologists. *Ind Health*. 2007;45(5):705–708.
10. Feng CK, Chen ML, Mao IF. Prevalence of and risk factors for different measures of low back pain among female nursing aides in Taiwanese nursing homes. *BMC Musculoskelet Disord*. 2007;8:52.
11. Violante FS, Fiori M, Fiorentini C, et al. Associations of psychosocial and individual factors with three different categories of back disorder among nursing staff. *J Occup Health*. 2004;46(2):100–108.
12. Eriksen W. The prevalence of musculoskeletal pain in Norwegian nurses' aides. *Int Arch Occup Environ Health*. 2003;76(8):625–630.
13. Cherkin DC, Deyo RA, Wheeler K, Ciol MA. Physician variation in diagnostic testing for low back pain. Who you see is what you get. *Arthritis Rheum*. 1994;37(1):15–22.

14. Cherkin DC. Primary care research on low back pain. The state of the science. *Spine*. 1998;23(18):1997–2002.
15. McPhillips-Tangum CA, Cherkin DC, Rhodes LA, Markham C. Reasons for repeated medical visits among patients with chronic back pain. *J Gen Intern Med*. 1998;13(5):289–295.
16. Mense S, Simons DG. *Muscle Pain: Understanding Its Nature, Diagnosis, & Treatment*. Baltimore: Lippincott Williams & Wilkins; 2001.
17. Simons D. New aspects of myofascial trigger points: etiological and clinical. *Journal of Musculoskeletal Pain*. 2004;12(3/4).
18. Ernst E. Massage therapy for low back pain: A systematic review. *J Pain Symptom Manage*. 1999;17(1):65–69.
19. Cherkin DC, Eisenberg D, Sherman KJ, et al. Randomized trial comparing traditional Chinese medical acupuncture, therapeutic massage, and self-care education for chronic low back pain. *Arch Intern Med*. 2001;161(8):1081–1088.
20. Furlan AD, Brosseau L, Imamura M, Irvin E. Massage for low-back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine*. 2002;27(17):1896–1910.
21. Hernandez-Reif M, Field T, Krasnegor J, Theakston H. Lower back pain is reduced and range of motion increased after massage therapy. *Int J Neurosci*. 2001;106(3–4): 131–145.
22. Preyde M. Effectiveness of massage therapy for subacute low-back pain: a randomized controlled trial. *CMAJ*. 2000;162(13):1815–1820.
23. Tsao JC. Effectiveness of massage therapy for chronic, non-malignant pain: a review. *Evid Based Complement Alternat Med*. 2007;4(2):165–179.
24. Janda V. Rational therapeutic approach of chronic back pain syndromes. Paper presented at: Chronic back pain, rehabilitation, and self-help, 1985; Turku, Finland.
25. Chaitow L. *Modern Neuromuscular Techniques*. New York: Churchill Livingstone; 1996.
26. Chaitow L, DeLany J. *Clinical Application of Neuromuscular Techniques*. Vol 1. Edinburgh: Churchill Livingstone; 2000.
27. Simons D, Travell J, Simons L. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Vol 1. 2nd ed. Baltimore: Williams & Wilkins; 1999.
28. Panjabi M, White A. *Biomechanics in the Musculoskeletal System*. New York: Churchill Livingstone; 2001.
29. Gracovetsky S. Is the lumbodorsal fascia necessary. Paper presented at: Fascia Research Congress, 2007; Harvard Medical School, Boston, MA.
30. Trudeau M. The contribution of the thoracolumbar fascia to the spine's stiffness. Paper presented at: Fascia Research Congress, 2007; Harvard Medical School, Boston, MA.
31. Cailliet R. *Low Back Pain Syndrome*. Philadelphia: F.A. Davis; 1988.
32. Nordin M, Frankel V. *Basic Biomechanics of the Musculoskeletal System*. 2nd ed. Malvern: Lea & Febiger; 1989.
33. Liebenson CE. *Rehabilitation of the Spine*. Baltimore: Williams & Wilkins; 1996.
34. Slipman CW, Lipetz JS, Jackson HB, Vresilovic EJ. Deep venous thrombosis and pulmonary embolism as a complication of bed rest for low back pain. *Arch Phys Med Rehabil*. 2000;81(1):127–129.
35. Carette S, Leclaire R, Marcoux S, et al. Epidural corticosteroid injections for sciatica due to herniated nucleus pulposus. *N Engl J Med*. 1997;336(23): 1634–1640.
36. Fadale PD, Wiggins ME. Corticosteroid Injections: Their Use and Abuse. *J Am Acad Orthop Surg*. 1994;2(3): 133–140.
37. vanTulder M. Randomized trial comparing interferential therapy with motorized lumbar traction and massage in the management of low back pain in a primary care setting – Point of view. *Spine*. 1999;24(15):1584.
38. Busanich BM, Verscheure SD. Does McKenzie therapy improve outcomes for back pain? *J Athl Train*. 2006; 41(1):117–119.
39. Preyde M. Effectiveness of massage therapy for subacute low-back pain: a randomized controlled trial. *Can Med Assn J*. 2000;162(13):1815–1820.
40. Schleip R. Fascial plasticity – a new neurobiological explanation Part 2. *Journal of Bodywork and Movement Therapies*. 2003;7(2):104–116.
41. Mixer WJ, Barr JS. Rupture of the intervertebral disc with involvement of the spinal canal. *N Engl J Med*. 1934;211:210–215.
42. Buckwalter JA. Aging and degeneration of the human intervertebral disc. *Spine*. 1995;20(11):1307–1314.
43. Magee D. *Orthopedic Physical Assessment*. 3rd ed. Philadelphia: W.B. Saunders; 1997.
44. Haig AJ, Geisser ME, Tong HC, et al. Electromyographic and magnetic resonance imaging to predict lumbar stenosis, low-back pain, and no back symptoms. *J Bone Joint Surg Am*. 2007;89(2):358–366.
45. Haig AJ, Tong HC, Yamakawa KS, et al. Spinal stenosis, back pain, or no symptoms at all? A masked study comparing radiologic and electrodiagnostic diagnoses to the clinical impression. *Arch Phys Med Rehabil*. 2006; 87(7):897–903.
46. Jarvik JG, Hollingworth W, Heagerty PJ, Haynor DR, Boyko EJ, Deyo RA. Three-year incidence of low back pain in an initially asymptomatic cohort: clinical and imaging risk factors. *Spine*. 2005;30(13):1541–1548; discussion 1549.
47. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am*. 1990;72(3):403–408.
48. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med*. 1994;331(2):69–73.
49. Clare HA, Adams R, Maher CG. A systematic review of efficacy of McKenzie therapy for spinal pain. *Aust J Physiother*. 2004;50(4):209–216.
50. McKenzie R. Understanding centralisation. *J Orthop Sports Phys Ther*. 1999;29(8):487–489.

51. Bush K, Cowan N, Katz DE, Gishen P. The natural history of sciatica associated with disc pathology. A prospective study with clinical and independent radiologic follow-up. *Spine*. 1992;17(10):1205–1212.
52. Couto JM, Castilho EA, Menezes PR. Chemonucleolysis in lumbar disc herniation: a meta-analysis. *Clinics*. 2007; 62(2):175–180.
53. Guha AR, Debnath UK, D'Souza S. Chemonucleolysis revisited: a prospective outcome study in symptomatic lumbar disc prolapse. *J Spinal Disord Tech*. 2006; 19(3):167–170.
54. Chicheportiche V, Parlier-Cuau C, Champsaur P, Laredo JD. Lumbar Chymopapain Chemonucleolysis. *Semin Musculoskelet Radiol*. 1997;1(2):197–206.
55. Nordby EJ, Javid MJ. Continuing experience with chemonucleolysis. *Mt Sinai J Med*. 2000;67(4):311–313.
56. Maksymowicz W, Barczewska M, Sobieraj A. Percutaneous laser lumbar disc decompression – mechanism of action, indications and contraindications. *Ortop Traumatol Rehabil*. 2004;6(3):314–318.
57. McMillan MR, Patterson PA, Parker V. Percutaneous laser disc decompression for the treatment of discogenic lumbar pain and sciatica: a preliminary report with 3-month follow-up in a general pain clinic population. *Photomed Laser Surg*. 2004;22(5):434–438.
58. Sobieraj A, Maksymowicz W, Barczewska M, Konopielko M, Mazur D. Early results of percutaneous laser disc decompression (PLDD) as a treatment of discopathic lumbar pain. *Ortop Traumatol Rehabil*. 2004;6(3): 264–269.
59. Choy DS. Percutaneous laser disc decompression (PLDD): twelve years' experience with 752 procedures in 518 patients. *J Clin Laser Med Surg*. 1998;16(6):325–331.
60. Gibson JN, Grant IC, Waddell G. The Cochrane review of surgery for lumbar disc prolapse and degenerative lumbar spondylosis. *Spine*. 1999;24(17):1820–1832.
61. Deyo RA, Cherkin DC, Loeser JD, Bigos SJ, Ciol MA. Morbidity and mortality in association with operations on the lumbar spine. The influence of age, diagnosis, and procedure. *J Bone Joint Surg Am*. 1992;74(4):536–543.
62. Nachemson AL. Newest knowledge of low back pain. A critical look. *Clin Orthop*. 1992(279):8–20.
63. Cherkin DC, Deyo RA, Loeser JD, Bush T, Waddell G. An international comparison of back surgery rates. *Spine*. 1994;19(11):1201–1206.
64. Maitland GD, Banks K, English K, Hengeveld E. *Maitland's Vertebral Manipulation*. 6th ed. Edinburgh: Elsevier; 2001.
65. Mooney V, Robertson J. The facet syndrome. *Clin Orthop*. 1976(115):149–156.
66. Dreyer SJ, Dreyfuss PH. Low back pain and the zygapophysial (facet) joints. *Arch Phys Med Rehabil*. 1996;77(3):290–300.
67. Dreyfuss PH, Dreyer SJ, Herring SA. Lumbar zygapophysial (facet) joint injections. *Spine*. 1995; 20(18):2040–2047.
68. Schwarzer AC, Aprill CN, Derby R, Fortin J, Kine G, Bogduk N. Clinical features of patients with pain stemming from the lumbar zygapophysial joints. Is the lumbar facet syndrome a clinical entity? *Spine*. 1994;19(10): 1132–1137.
69. Carette S, Marcoux S, Truchon R, et al. A controlled trial of corticosteroid injections into facet joints for chronic low back pain. *N Engl J Med*. 1991;325(14):1002–1007.
70. Reeves RK, Laskowski ER, Smith J. Weight training injuries part 2: diagnosing and managing chronic conditions. *Physician Sportsmed*. 1998;26(3).
71. McCulloch J, Transfeldt E. *Macnab's Backache*. 3rd ed. Baltimore: Williams & Wilkins; 1997.
72. Bassewitz H, Herkowitz H. Lumbar stenosis with spondylolisthesis: current concepts of surgical treatment. *Clin Orthop*. 2001(384):54–60.
73. Amundson G, Edwards C, Garfin S. Spondylolisthesis. In: Herkowitz H, Garfin S, Balderston R, Eismont F, Bell G, Wiesel S, eds. *Rothman-Simeone: The Spine*. Vol 1. 4th ed. Philadelphia: W.B. Saunders; 1999:835–885.
74. Lowe W. *Orthopedic Assessment in Massage Therapy*. Sisters, OR: Daviau-Scott; 2006.
75. Torg J, Shephard R. *Current Therapy In Sports Medicine*. 3rd ed. St. Louis: Mosby; 1995.
76. Janda V. Muscles as a pathogenic factor in back pain. Paper presented at: IFOMT, 1980; New Zealand.
77. Janda V. Postural and Phasic Muscles in the Pathogenesis of Low Back Pain. Paper presented at: XIth Congress ISRD, 1968; Dublin.
78. *Dorland's Illustrated Medical Dictionary*. Philadelphia: Saunders; 2003.
79. Di Bari M, Chiarlone M, Matteuzzi D, et al. Thoracic kyphosis and ventilatory dysfunction in unselected older persons: an epidemiological study in Dicomano, Italy. *J Am Geriatr Soc*. 2004;52(6):909–915.
80. Neumann DA. *Kinesiology of the Musculoskeletal System*. St. Louis: Mosby; 2002.
81. White A, Panjabi M. *Clinical Biomechanics of the Spine*. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 1990.
82. Nowak J. Scheuermann Disease. September 1, 2004; www.emedicine.com. Accessed May 26, 2005.
83. Bullock MP, Foster NE, Wright CC. Shoulder impingement: the effect of sitting posture on shoulder pain and range of motion. *Man Ther*. 2005;10(1):28–37.
84. Lonner BS, Newton P, Betz R, et al. Operative management of Scheuermann's kyphosis in 78 patients: radiographic outcomes, complications, and technique. *Spine*. 2007;32(24):2644–2652.



## Chapter 10

# Cervical spine

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The cervical spine is susceptible to numerous soft-tissue disorders. The structural mechanics of this region are designed primarily to provide maximum mobility to the head. It is crucially important for survival that the head, where our primary sensory organs are located, is able to move around easily. Balance receptors that affect the whole body are also located in the head, so the role of various tissues in the neck is to maintain simultaneous stability and mobility.

The head of an adult can weigh close to 10 pounds. From a skeletal standpoint, this weight is balanced on a small contact surface (the surface area of the atlanto-occipital joint). To keep a weight this heavy balanced on such a small contact surface while it moves around in all different directions is challenging to say the least. The soft tissues of the cervical region are responsible for guiding this movement and providing stability.

Muscles, tendons, and ligaments have complex mechanical interactions that help produce the motion of the head and neck and maintain the head in an upright position. Due to biomechanical challenges in this region, muscle conditions involving chronic hypertonicity and myofascial trigger points are common. Nerve compression and tension syndromes are also common because of the brachial plexus and other sensitive neurological tissues, which are vulnerable to compression in the area. Pain and/or neurological sensations from these conditions can be felt in the neck or down the upper extremity.

Poor posture, faulty ergonomics, overuse, and stress can all cause various pathological conditions

of the neck, as can injuries such as impact trauma and whiplash. There are also several genetic conditions that can produce problems in this region. Massage is an excellent treatment choice for addressing these disorders because the soft tissues play a role in these pathologies.

## INJURY CONDITIONS

### NEUROMUSCULAR NECK PAIN

#### Description

The challenge of maintaining the head in an upright position places postural strain on muscles and other soft tissues of the cervical region. In neuromuscular neck pain there is hypertonicity either throughout the entire muscle or in localized areas, such as with myofascial trigger points. In the neck it takes very little muscular dysfunction to set off a cascading process of neuromuscular dysfunction, known as the *pain-spasm-pain cycle*.<sup>1</sup>

Postural distortions, such as a forward head posture, are a result of neuromuscular dysfunction and the constant effort to fight the pull of gravity on the head. If the head is maintained directly on top of the cervical spine, there is very little muscular effort needed to keep it in that position. Once the head moves forward of the center of gravity, even if it is just a few degrees, there is a significant increase in muscle activity to hold the head upright.<sup>2</sup> This constant muscle tension can lead to muscular hypertonicity and the development of myofascial trigger points.

Once these distortions develop, they cause a perpetuation of biomechanical dysfunction. As with other regions of the spine, primary health care providers often place emphasis on structural considerations, particularly joint pathology, as the source of most disorders. However, many of these problems are not structural but instead muscular in nature.<sup>3</sup>

Myofascial trigger points in muscles such as the posterior cervical muscles can become a constant source of pain if they are not properly neutralized. A sudden and awkward loading movement or trauma, as can happen in a motor vehicle accident, often activates these trigger points. The sudden

loading of these muscles stimulates excessive neurological activity in related muscles, and produces symptoms in other areas such as the temporomandibular joint.<sup>4</sup>

Because many cervical muscles maintain constant isometric contractions during the day just to keep the head erect, the patterns of dysfunction are facilitated by the very act of attempting to hold the head upright. These movement patterns and their dysfunctional fixations follow a pattern. Once they are set they tend to recur in the same region any time the individual is exposed to further stress. The stress does not have to be an excessive force to activate the pattern of neuromuscular pain or dysfunction; psychological or chemical stressors are just as likely to start a cascade of neuromuscular distress.

#### Treatment

##### *Traditional approaches*

The main approach to treating neuromuscular dysfunction in the cervical region is rest from offending activities. However, rest does not mean immobilization. In the past, a cervical collar was frequently advocated as a means of treating neuromuscular pain or muscle strain in the cervical region. Research has shown that long periods of immobilization are detrimental to the rehabilitation process unless the condition is severe and joint hypermobility is a serious concern. Rest from offending activities means going about normal daily functions, but being careful to avoid movements that aggravate the pain problem.

Various forms of physical therapy are used to address neuromuscular neck pain. These approaches include stretching, therapeutic exercise, or the various soft-tissue manipulation methods. Anti-inflammatory medication may be advocated for these problems as well. There is not an inflammatory process occurring in many cases, but these medications are effective in pain management and that is their primary benefit.

High-velocity manipulation/mobilization is used to treat neuromuscular dysfunction. However, muscle spindles can respond to the rapid rate of change in muscle length causing further contraction due to the myotatic (stretch) reflex. That is one reason why some manipulative therapy

practitioners are moving away from high-velocity manipulation adjustments in favor of various *low-force* techniques.<sup>5</sup>

### Soft-tissue manipulation

One of the most effective ways to address neuromuscular neck pain is with soft-tissue manipulation. The massage practitioner's ability to sense hypertonic regions and precisely locate myofascial trigger points or dysfunctional muscle tissue is a hallmark of effective massage. For the massage practitioner, numerous methods are effective in treating neuromuscular neck pain. Gentle effleurage and myofascial approaches are used initially if the condition is acute or involves excessive muscle spasm. Active-assisted stretching methods are also a way to begin treating muscles that are in pain or spasm. With muscle tension that is chronic, a slightly more aggressive treatment approach is appropriate. Effective techniques with chronic neuromuscular pain include static compression, deep stripping, and active engagement methods.

### Suggested techniques and methods

**A. Myofascial approaches** Myofascial techniques encourage fascial and muscular relaxation and enhance their pliability. The primary goal is to place moderately light pressure on the subcutaneous fascia while pulling the fascia in multiple directions. Place the hands lightly on the client's neck where the myofascial stretch is to be applied. Pull the hands apart to take the slack out of the tissue and apply a light degree of tensile (pulling) force between the hands (Fig. 10.1). Once there is a



Figure 10.1 Myofascial stretching on the neck.

slight degree of pull between the hands, hold this position until a subtle sensation of tissue release is felt. This technique can be applied to muscles of the posterior, anterior, and lateral cervical region. To retain the best contact between the client and the practitioner's hands, generally no creams or lotions are used.

**B. Effleurage and sweeping cross fiber** These techniques are effective for reducing tension in neck muscles. Effleurage is performed with long gliding strokes parallel to the muscle fiber direction. Cross fiber techniques sweep diagonally across the primary fiber direction of muscles; the direction of the movement changes depending on the muscles being addressed (Fig. 10.2). Use care with effleurage or sweeping cross fiber motions across tissues of the lateral and anterior neck region due to the superficial vascular structures in this area.

**C. Deep longitudinal stripping to cervical extensors** Deep stripping techniques are applied to the cervical erector spinae and extensor muscles. Due to the practitioner's hand size and the small region of the neck to be treated, it is not as easy to use a broad contact surface in this area. After reducing tension in superficial muscles with effleurage and cross fiber methods use small contact surface stripping techniques such as the finger, thumb, or pressure tool (Fig. 10.3). Stripping techniques can be performed in a caudal or cephalad direction and the client may be supine, prone, or in a side-lying position.

These same stripping techniques can be applied to muscles of the lateral neck region such as the



Figure 10.2 Sweeping cross fiber to neck muscles.



Figure 10.3 Deep stripping on posterior cervical muscles (small contact surface).



Figure 10.5 Deep stripping in lamina groove on posterior neck region.

upper fibers of the levator scapulae and posterior scalene. Use caution when applying stripping techniques near the anterior neck region due to the proximity of sensitive vascular structures and the brachial plexus.

**D. Static compression** To reduce muscular hypertonicity in a specific location, use static compression methods. Treat localized areas of tension or specific myofascial trigger points with small contact surface pressure (Fig. 10.4). Pressure maintained for 8–10 seconds is usually sufficient to achieve a reduction of muscle tension.

**E. Deep stripping in lamina groove** After working through tension in more superficial muscles, treat the deep intrinsic muscles of the cervical spine. Use the thumb or finger tip to apply deep



Figure 10.4 Static compression on cervical muscles.

longitudinal stripping techniques to the muscles in the lamina groove. One hand holds the head while the other hand performs the stripping technique in the lamina groove (Fig. 10.5). It is easiest to treat these muscles if the thumb is used. If it feels difficult to keep the thumb's interphalangeal joint straight, brace the thumb with the other fingers as pressure is applied in this stroke. Pause and repeat short stripping movements on any areas where increased muscle tension is palpated or the client reports greater tenderness.

**F. Massage with active engagement to cervical extensors** An effective way to treat chronic muscle tension in deeper neck muscles is using active engagement lengthening techniques. The client is in a supine position. Use one hand to hold the head and the other hand to perform the stripping technique on the cervical muscles, as in the deep stripping techniques in C and E. Instruct the client to push their head down with a moderate amount of effort into the practitioner's hand that is holding their head. Once a moderate isometric contraction is established, instruct the client to slowly let go of the contraction and gradually lift their head into full flexion. While they are releasing the contraction and the head is moving in flexion, perform a deep stripping technique on the cervical extensor muscles. It is easiest to perform this stroke moving down the neck in an inferior direction (Fig. 10.6).

**G. Massage with active engagement to lateral neck flexors** The same technique described in F



Figure 10.6 Stripping with active engagement on posterior cervical muscles.

above can be performed on the lateral neck flexors instead of the cervical extensors by altering the position slightly. Have the client attempt to laterally flex the neck to the side that is being treated while resistance is offered to that action. Instruct the client to slowly let go of the contraction. As the client slowly releases the contraction, gradually pull (or push) the head to the opposite side. As the client's head is slowly moving to the opposite side, perform a stripping technique on the lateral flexor muscles (Fig. 10.7). Use caution with pressure applied on the lateral side of the neck to avoid applying pressure to cervical vascular structures or the brachial plexus.

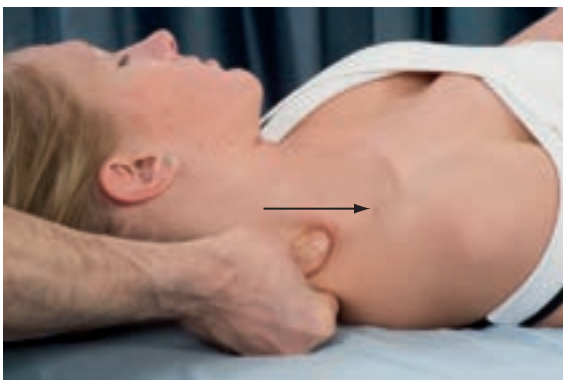


Figure 10.7 Stripping with active engagement on lateral flexor muscles.

#### Rehabilitation protocol considerations

- There can be a number of serious pathologies in the cervical region so perform a thorough assessment to rule out serious pathologies that need to be referred to another health professional.
- Neuromuscular pain and postural distortions are established by constant reinforcement. It is important to encourage changes in these movement or postural patterns early in the treatment process as muscle tension is addressed.
- Stretching and flexibility enhancement are essential components of treating neuromuscular neck pain. Stretching is most effective when performed after soft-tissue manipulation so the benefits of enhanced tissue pliability can be maximized.
- Certain techniques are more effective or appropriate at particular phases of the rehabilitation process. Techniques such as active engagement methods described in F and G are not suggested in acute or severe neuromuscular pain cases. These techniques can be intense for the client so they are used in the later stages of rehabilitation.

**Cautions and contraindications** Just as with the back, neuromuscular pain can resolve while the client is on the treatment table, only to return post massage. Decreasing pain-producing tension in the muscular soft tissues alters muscular proprioception. When the individual moves around after treatment there are different muscular recruitment patterns used, which can overtax tissues that have not adapted to the new patterns. The body's reaction to the sudden overload can be muscle spasm. The best way to avoid this situation is to move slowly when getting up from a massage treatment and shortly afterward so the body has time to integrate the new muscular proprioceptive patterns.

Some individuals are particularly sensitive to having their head and neck moved, which can have a profound effect on the treatment results. They may brace with protective muscle guarding as various movements or massage techniques are performed in this region. Be aware of these potential guarding patterns and make sure the quality of touch and the therapeutic environment created by the treatment encourages relaxation.

**Box 10.1 Clinical Tip**

In order to address a neck injury that has neuromuscular tension as a component it is crucial to help the client relax as much as possible. The neck muscles are accustomed to having to work all the time to maintain the head in an upright position. There can be significant tension in these muscles just holding the head in a static position, especially if the individual does not have good posture. Treating neuromuscular neck pain in a position with the client horizontal (preferably in a supine or side-lying position) allows the neck muscles to relax more completely so there is less muscle tension.

**HERNIATED NUCLEUS PULPOSUS****Description**

Intervertebral discs in the cervical region are exposed to compressive forces primarily from weight of the head. Other impacts can produce excess compression on the cervical discs, such as falling on the ground or having a load hit directly on top of the head. A common example where this occurs is someone diving into a shallow pool and hitting their head on the bottom of the pool.

The normal compressive load on the cervical spine is nowhere near that of the lumbar spine. Yet, there can still be enough compressive load to adversely affect the spinal structures. Because the cervical intervertebral discs are significantly smaller than their lumbar counterparts, it takes a smaller load to create damaging levels of compressive stress.

An accumulation of compressive forces on the cervical spine can cause degeneration of the intervertebral disc. Stenosis (narrowing) of the intervertebral foramen often accompanies these degenerative changes. Neurological symptoms can develop as nerve roots are compressed within the narrowing space around them. Muscle weakness and sensory symptoms including paralysis in the upper extremity occur in patients with spinal stenosis.<sup>6</sup>

Herniation technically means pushing through. The primary problem in this condition occurs

because degeneration of the annulus fibrosus allows the nucleus to push through it (see Fig. 9.9). As the nucleus continues to exert pressure on the annulus, it causes the annulus to change shape. Eventually, if not halted, the nucleus can push all the way through the annulus. Degeneration of the annulus can be the result of numerous factors, including poor disc nutrition, loss of viable cells, loss of water content, and others.<sup>7-9</sup> Most of these problems originate from excessive compressive loads on the spinal structures over time.

These factors cause the intervertebral disc to lose some of its thickness prior to the process of herniation. When a disc has lost some of its thickness it is common for an individual to be given a diagnosis of degenerative disc disease. Essentially, this means the disc has lost thickness and the vertebrae in the region are closer together. However, this does not necessarily mean that pathological symptoms follow. There are often no symptoms with degenerative disease of the spine and there may be no pathology related to the condition.<sup>10,11</sup>

There are several names given to the different degrees of disc herniation. While these names are not always consistent in the literature, they do offer a greater degree of specificity as to the severity of the disc herniation.<sup>12</sup> Figure 9.10 in the previous chapter illustrates the different degrees of disc herniation. In a disc protrusion (also called a bulge), the disc has changed shape, but the majority of the annulus fibrosus is intact. This disc is considered prolapsed if only the outer-most fibers of the annulus are still containing the nucleus. In an extrusion, the disc material has pushed through the outer border of the annulus, but it is still connected to itself. The final stage of degeneration is sequestration. In this stage the disc material has actually separated from itself, and portions of the disc material may be freely floating in the spinal canal.

Herniation of the nucleus pulposus in the cervical region is similar to that which occurs in the lumbar region. Yet, there are several reasons why cervical discs are not as vulnerable to disc herniations as those in the lumbar region. The ligament (Fig. 10.8) protects protrusions in the cervical region better than it does in the lumbar region, because it is proportionately wider in the cervical region. In the cervical region the posterior longitudinal ligament covers

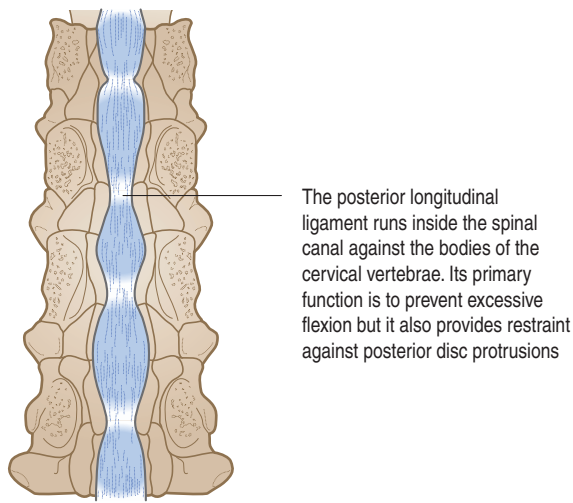


Figure 10.8 Posterior longitudinal ligament.

the majority of the posterior aspect of the disc. In addition, the nucleus is situated farther anteriorly in the cervical region, making the possibility of a posterior protrusion less likely.<sup>13</sup>

In the cervical region disc herniations are most likely to affect the nerve roots that make up the brachial plexus. Symptoms from compression of these nerve roots are felt down the length of the upper extremity. Pain can also come from the disc pressing on the posterior longitudinal ligament, dura mater, or spinal cord.<sup>13</sup> It is possible, though not common, for cervical pain associated with disc herniations to come from the disc itself without it pressing on nerve roots. Anatomical investigations have shown that the discs have nerve fibers and mechanoreceptors that can produce pain just from excessive pressure.<sup>14–17</sup>

## Treatment

### *Traditional approaches*

Conservative treatment is advocated for this condition prior to surgical intervention. Conservative treatment consists of rest, physical therapy modalities such as ice, heat, ultrasound, electrical stimulation, exercise, and traction. Manipulation and mobilization techniques are also used to reduce pressure on the disc. Cervical traction units come in various styles and are often effective for addressing disc pathology. Because a primary part of the problem is excessive compression on the disc,

reversing that compression through traction can provide effective relief. Anti-inflammatory medication may be used in pain management, although it is not always evident that inflammation is present.

If conservative treatment fails, surgery may be used to treat the problem. However, there is increasing controversy about when surgery should be used. Similar to problems in the lumbar spine, many asymptomatic individuals have been found with disc herniations in the cervical region.<sup>10</sup> Assuming the necessity of cervical disc surgery on the presence of disc herniation alone could lead to unnecessary surgery. New treatment approaches encourage a finding of adequate clinical signs showing disc pathology to warrant surgical intervention.

Laminectomy and vertebral fusion are some of the more common surgical approaches for treating cervical disc herniation. However, some of the procedures, such as cervical fusion, may place additional levels of strain on the other vertebral segments.<sup>18</sup> For that reason, newer and less invasive surgical procedures such as percutaneous laser disc decompression (PLDD) may be tried. A reduction in disc pressure is achieved through laser energy in this procedure.<sup>19</sup>

### *Soft-tissue manipulation*

**General guidelines** Soft-tissue treatments can be helpful in reducing symptoms in the area, but use caution not to aggravate the symptoms through pressure or movement applied to the area. Cervical disc pathologies may activate myofascial trigger points in the cervical muscles.<sup>20</sup> These activated trigger points can then be perpetuated by other factors. The resultant myofascial pain pattern can then linger long after the disc herniation symptoms have subsided. Massage is effective in addressing these patterns of dysfunctional muscular hypertonicity.

Disc protrusions onto cervical nerve roots can also produce upper-extremity neurological symptoms because of increased neural tension. The increased neural tension can make various regions of the upper extremity more susceptible to neurological pathology. For that reason, thorough soft-tissue treatment of the upper extremity is an important aspect of addressing neural pathology in the cervical region.

**Suggested techniques and methods** The focus of massage treatment for disc herniations is to reduce the role that muscle tightness plays in the disc pathology. Attention is focused on neck muscles that contribute to the compressive forces or postural distortion of the cervical spine. Any of the techniques in the previous section on neuromuscular neck pain could be used to address the muscular components that are aggravating a disc herniation.

Be careful of any technique that puts excessive pressure in the area or moves the neck in any way that aggravates symptoms. In some muscular pain conditions a degree of pain can be expected and even considered therapeutically helpful. Working deep tension out of muscles can be uncomfortable, but is often described as a sensation of pain that feels pleasant. Massage treatment of neurological conditions, such as a disc herniation, should not increase symptoms because that would indicate further pressure or aggravation of the nerve.

#### Rehabilitation protocol considerations

- The primary focus of treatment is to reduce compression on neurological structures. Massage can be performed at the same time as other traditional conservative treatments such as manipulation or mobilization. Massage is a valuable adjunct for manipulation or mobilization treatments and is especially valuable if performed prior to these treatments. Reduction in muscle and soft-tissue tension allows these other techniques to be applied with greater ease and less resistance.
- Movement re-education to reduce postural stress is an important aspect of treatment and can be performed along with massage treatment.
- If there is an acute disc herniation, significant muscle spasm is likely to result. Muscle spasm should be addressed first in order to reduce the perpetuation of dysfunctional muscular activity.
- Massage is a valuable adjunct treatment in post-surgical rehabilitation. After surgery, massage is effective in reducing excess muscle tension and restoring proper neuromuscular activity in the affected muscles. Treatment should not be used anywhere near surgical incision sites.
- If the disc herniation has become chronic, slightly more aggressive massage treatment can

be attempted, but always work within the client's symptom parameters so that neurological symptoms are not increased.

**Cautions and contraindications** Watch for any indication of symptom aggravation. If the procedure increases neurological symptoms, discontinue that treatment immediately. It is beneficial to have the client evaluated by another health care provider. Be particularly aware of any symptoms that are bilateral, as this may indicate a central protrusion onto the spinal cord.

Use caution when performing techniques close to spine. The location of disc herniation should not be vulnerable to massage treatment because the transverse processes of the spine protect the region. However, techniques that put anteriorly directed pressure on the spine could move spinal structures in a way that aggravates nerve-root compression by the disc.

Some clients demonstrate protective muscular guarding in their cervical region. Pain from a cervical disc herniation can produce anxiety and fears about treatment, which add to the muscle splinting and hypertonicity. To reduce the client's apprehension thoroughly describe the gentle nature of the soft-tissue treatment and make sure gentle, compassionate touch is conveyed through manual treatment. With these clients, it is best to begin with gentle techniques.

## THORACIC OUTLET SYNDROME

### Description

Thoracic outlet syndrome (TOS) is not a single pathology, but a term that encompasses several pathologies involving compression of arteries, veins, or nerves near the thoracic outlet (though the compression does not necessarily occur within the thoracic outlet). TOS is a complex condition and is often overlooked or misdiagnosed due to the difficulty in distinguishing between its variations.<sup>21,22</sup>

One area of confusion with this condition is the name thoracic outlet. In some anatomy texts, this region is referred to as the thoracic inlet. However, both names refer to the same general area. The thoracic outlet/inlet is the area where structures either exit (thoracic outlet) or enter



(thoracic inlet) the upper border of the thoracic rib cage. Therefore, whether the area is called the thoracic outlet or inlet depends upon the anatomical structure being discussed.

Thoracic outlet syndromes (TOS) were originally described in the medical literature as circulatory problems created by pressure on the arteries and veins in the upper-shoulder region. For that reason, many of the physical examination tests performed to evaluate this problem focus on circulatory responses. The pathology is sometimes defined as arterial (involving compression of the subclavian artery), venous (involving compression of the subclavian vein), or neurogenic (caused by compression of the brachial plexus). It is generally agreed upon today that the vast majority of symptoms from TOS, possibly as much as 90%, arise from neurological impairment.<sup>23,24</sup> For no known reason middle-aged women appear to be more susceptible to TOS than other groups.<sup>25</sup>

Four different pathologies can go by the name TOS. The first is a condition called true neurologic (or neurogenic) TOS. True neurologic TOS is caused by the presence of an unusual anatomical structure called a cervical rib, and is relatively rare (Fig. 10.9). The cervical rib is a pathological

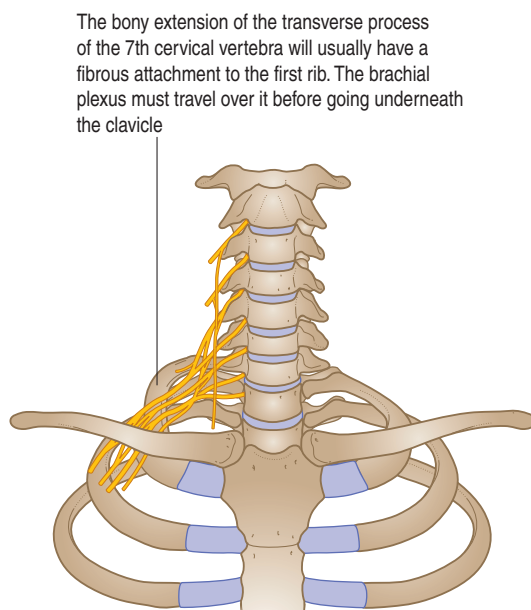


Figure 10.9 The cervical rib.

extension of the transverse process of the seventh cervical vertebra. It can be either a fibrous or an osseous structure and often connects the seventh cervical transverse process with the first rib. When the rib is present, the nerves of the brachial plexus must pass over it and, therefore, its presence can lead to neurological compression pathology.

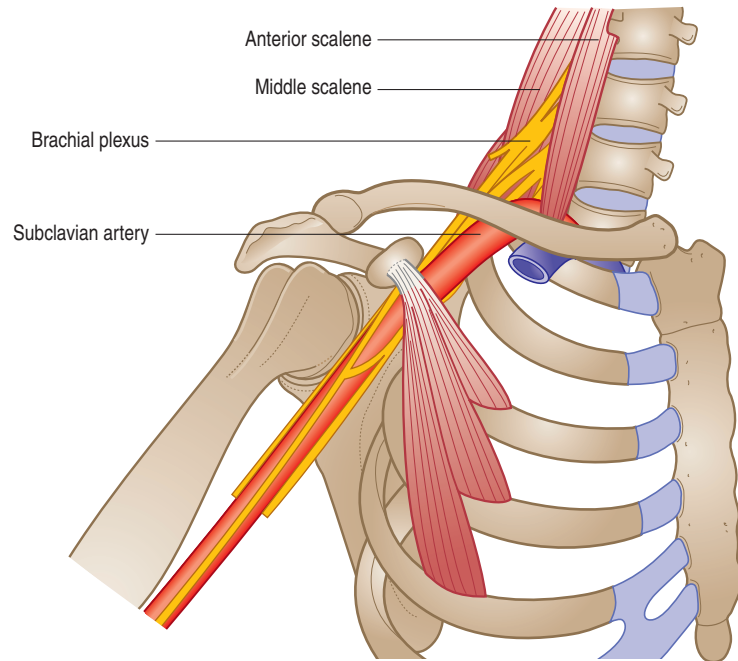
The other three conditions that go by the name TOS are all neurovascular compression pathologies in regions near the thoracic outlet. They include anterior scalene syndrome, costoclavicular syndrome, and pectoralis minor syndrome. In anterior scalene syndrome, the neurovascular structures are compressed between the anterior and middle scalene muscles (Fig. 10.10). In costoclavicular syndrome they are compressed between the clavicle and first rib (Fig. 10.11). In pectoralis minor syndrome the neurovascular structures are compressed between the pectoralis minor muscle and the upper rib cage (Fig. 10.12).

The medial, lateral, and posterior cords of the brachial plexus are its three primary divisions in the thoracic outlet region. Of those three, the medial cord is the most inferior, which makes it most susceptible to compression against the bones that are underneath it. As a result, symptoms are common in the nerves derived from fibers in the medial cord. Most of these fibers make up the ulnar nerve; therefore, it is usual for symptoms from the various forms of TOS to be felt first and foremost in the cutaneous distribution of the ulnar nerve. The cutaneous distribution of the ulnar nerve is pictured in Figure 10.13.

Several factors can lead to the development of TOS. Its onset can be acute or chronic. However, acute onset TOS is rare, and is usually the result of serious trauma, such as a direct blow to the clavicular region. A clavicular dislocation may change the structural arrangement enough to cause compression of the neurovascular structures in the area. An adequate history should provide insight into the onset of the condition.

More common causes of TOS are chronic postural distortions with resultant muscular dysfunction. Tightness or myofascial trigger points in the anterior and middle scalene muscles can be enough to make them press against the brachial plexus. Tightness in the coracobrachialis and

Figure 10.10 Anterior scalene syndrome. Compression of neurovascular structures between the anterior and middle scalene muscles.



biceps brachii can pull the coracoid process in an anterior/inferior direction. If this occurs, the pectoralis minor is in a shortened position. It may become hypertonic in this shortened position and compress the brachial plexus against the upper rib cage (pectoralis minor syndrome).

Either of these postural distortions can lead to costoclavicular syndrome. Postural or movement patterns that put affected muscles under prolonged contraction also play a part in these pathologies. Examples include maintaining long periods of abduction in the shoulders, such as

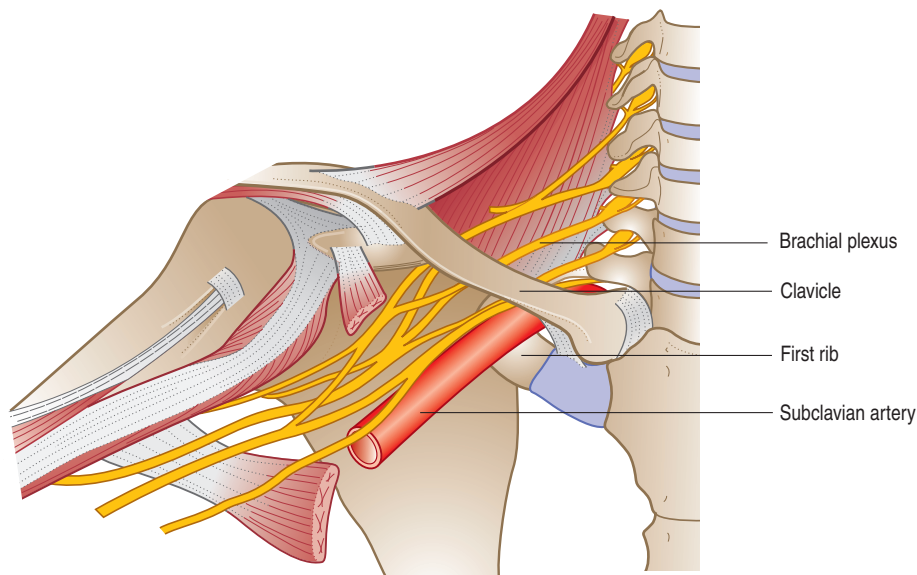


Figure 10.11 Costoclavicular syndrome. Neurovascular structures are compressed between the clavicle and first rib.

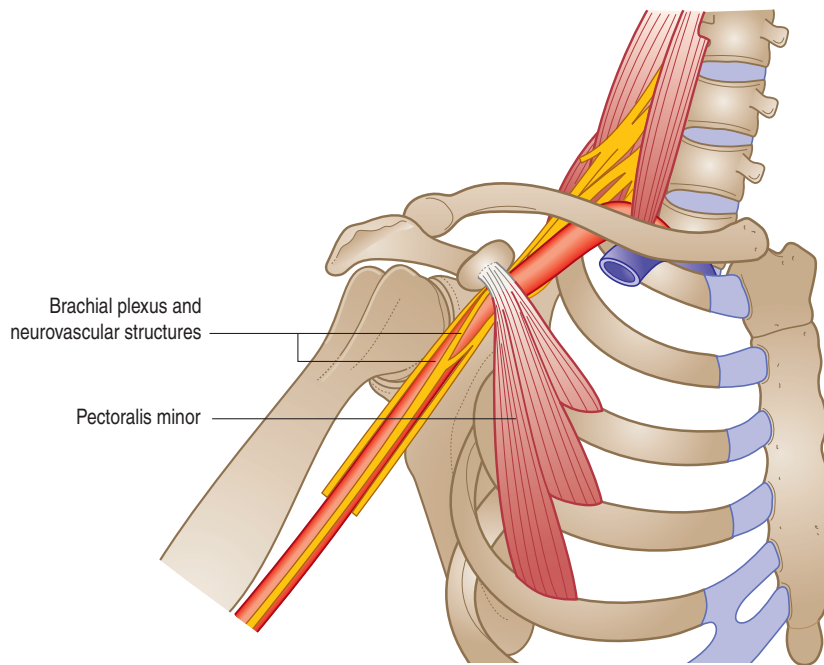


Figure 10.12 Pectoralis minor syndrome. Neurovascular compression under the pectoralis minor muscle.

in hair styling, or the position a violinist must keep while playing their instrument. Wearing a heavy backpack or carrying heavy objects in the affected upper limb could aggravate symptoms as well.

Symptoms that clients experience include pain or paresthesia down the arm into the hand, feelings of heaviness in the upper limb, coldness or discoloration of the upper limb, or muscular atrophy of the thenar muscles of the hand. The pain, aching, or paresthesia that is felt in the arm and hand is usually in the distribution of the ulnar

nerve or the medial antebrachial cutaneous nerve along the ulnar side of the forearm.

Problems in the thoracic outlet region are commonly involved in double crush injuries (see Chapter 2 for additional information on the double crush phenomenon). There are several locations in the upper extremity where compression

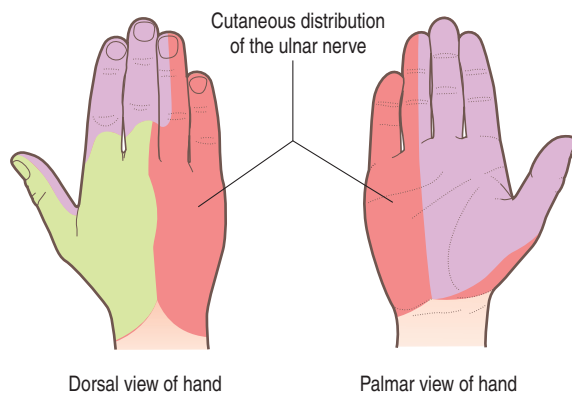


Figure 10.13 Cutaneous distribution of ulnar nerve.

**Box 10.2 Clinical Tip**

The ulnar nerve is the one most commonly involved in the different variations of thoracic outlet syndrome. However, it is important not to overlook the other major nerves of the upper extremity, which can also be involved. A more comprehensive physical examination with functional testing will be required to determine which nerves are primarily responsible for the adverse symptoms. Regardless of which nerve(s) appear to be at fault, it is advantageous to treat the entire neck and upper extremity as if the thoracic outlet nerve entrapment were affecting any or all nerves in the area. The enhanced mobility of all nerves in the upper extremity is valuable in reducing the potential for double crush injuries.

pathologies routinely occur so the likelihood of involvement of at least one, if not more, of these areas is increased. Common postural distortions can adversely affect more than one nerve compression site near the thoracic outlet, so double crush symptoms are common from thoracic outlet pathologies.<sup>26</sup>

## Treatment

### *Traditional approaches*

Conservative treatment is usually successful with TOS variations. In the majority of cases, some form of postural re-education is of crucial importance because postural change may be sufficient to remove the dysfunctional compression on the neurovascular structures. Postural re-education can be enhanced through stretching and various strength training methods as well.

If conservative treatment is unsuccessful, surgery may be the next option. Surgical approaches are most effective when dealing with compression caused by a cervical rib. Excision (removal) of the cervical rib often brings immediate relief of symptoms. However, there are those that question the necessity of surgery for this problem, even if a cervical rib exists.<sup>27</sup> If an individual with a cervical rib has made it through their life without symptoms, it is questionable whether the surgery is absolutely necessary. More research is needed to determine which conservative treatments are most effective in treating a neurogenic TOS caused by a cervical rib.

Surgery can be used to treat some of the other soft-tissue variations of TOS. The anterior scalene syndrome is sometimes treated surgically by removing some of the scalene muscles or a portion of the first rib.<sup>28</sup> However, removal of the muscles or the first rib is likely to have other detrimental effects on muscular function in the region.<sup>29</sup>

### *Soft-tissue manipulation*

**General guidelines** Particular attention is paid to treating the soft tissues involved in the specific region of compression. It is helpful to treat all of these areas regardless of where the primary site of compression is occurring, because more than one site may be involved. It is beneficial to treat these other areas, even if they are not directly involved in the compression pathology.

If neurological or vascular symptoms are increased during treatment, move to another region, as it is likely that nerve compression is being increased. Several muscles, including the pectoralis major, latissimus dorsi, teres major, and subscapularis, can have myofascial trigger points that mimic some TOS symptoms. It is advantageous to treat these muscles in the process as well. If they are holding dysfunctional trigger points, their treatment helps restore proper muscular balance.

Because these neurovascular compression disorders are often caused by postural distortion, it is important to address postural dysfunctions. A critical component of postural correction for these disorders is to repeat the corrections frequently. The more a postural or movement pattern is repeated, the more likely it is to become ingrained in the neurological system and become a natural aspect of posture or movement.

Stretching muscles of the cervical region and shoulder girdle is an important part of treating all the variations of TOS. When stretching muscles take the muscle to the point of mild pain or discomfort and hold it there until the pain or discomfort subsides. In doing this, the connective tissue component of the muscle is elongated, and the neuromuscular component changes the rate of stimulation in the muscle to reduce tightness. In the process of stretching, the client may report an exacerbation of neurological symptoms in certain positions. This is ordinarily due to stretching of nerve tissue in these positions. If there is adverse neural tension throughout any of the nerves in the upper extremity due to one of these problems, some degree of neural stretching may be helpful to improve mobility of the nerves.

The same positions that give an increase in symptoms can be used for neural mobility enhancement. However, be very careful in the way neural stretching procedures are applied, because it is different from stretching myofascial tissues. With nerve tissue it is more beneficial to encourage mobility between the nerve and adjacent structures by repeatedly bringing the nerve to the end of its extensibility, and then removing tension from it (shortening it) without holding it in the fully stretched position.<sup>30,31</sup> It is the repetition that encourages greater neural mobility and not the tensile load on the nerve.

Holding the nerve in its fully stretched position is likely to increase the symptoms, and does not achieve a significant increase in mobility. The primary function of neural stretching procedures is not to increase the length of nerve tissue as it is with an elastic tissue like muscle. The primary goal is to increase the mobility between the nerve and adjacent structures. Neural mobility procedures are more effective when the soft tissues along the entire path of the nerve are as relaxed and pliable as possible.

**Suggested techniques and methods** The following techniques may be effective for addressing one variation of TOS, but not for another variation. They are categorized in different groups for ease of presentation. It is still valuable to treat all the potentially involved tissues regardless of the variation that exists.

**True neurogenic thoracic outlet syndrome** No soft-tissue treatment technique can remove the obstruction of the cervical rib. Massage treatment for a true neurogenic TOS focuses on reducing the likelihood of additional neurovascular compression in the area and enhancing neural mobility. The techniques described below in the other categories are valuable for this approach.

#### **Anterior scalene syndrome and costoclavicular syndrome**

**A. Myofascial approaches** Myofascial techniques encourage fascial and muscular relaxation. Gentle pressure is placed on the fascia in multiple directions. Relaxing the fascia has benefits in reducing muscular tension and decreasing restrictions on neural mobility. The primary goal is to place a moderately light pressure on the subcutaneous fascia. Pulling the fascia in multiple directions appears to enhance its pliability the most. Place the hands lightly on the client's neck where the myofascial stretch is to be applied. Pull the hands apart to take the slack out of the tissue and apply a light degree of pulling force between the hands (Fig. 10.1). Once there is a slight degree of pull between the hands, hold this position until a subtle sensation of tissue release is felt. These approaches are effective when applied to muscles of the posterior, anterior, and lateral cervical region.

**B. Effleurage and sweeping cross fiber** These techniques are effective for reducing tension in neck muscles that may be contributing to neurovascular



Figure 10.14 Static compression on anterior scalenes.

compression. Effleurage is performed with long gliding strokes parallel to the muscle fiber direction. Cross fiber techniques sweep diagonally across the primary fiber direction of muscles in different regions of the neck (Fig. 10.2). Use care with effleurage or sweeping cross fiber motions across tissues of the lateral and anterior neck region due to the superficial vascular structures in this area.

**C. Static compression** Use small contact surface static compression to reduce muscular hypertonicity or treat myofascial trigger points in specific muscles, such as the scalenes, that can be compressing neurovascular structures (Fig. 10.14). Pressure maintained for 8–10 seconds is usually sufficient to achieve a reduction of muscle tension.

**D. Deep stripping on scalenes** Due to the location of the scalene muscles deep to the sternocleidomastoid and other neck muscles, they are not easy to access along their whole length. Do not exert a great deal of pressure in the anterior neck region as the brachial plexus and vascular structures are superficial in this area. The client is in a supine position with the head turned slightly away from the side being treated. Use the finger tips or thumb to perform a longitudinal stripping technique on the anterior and middle scalene muscles, just lateral to the lateral edge of the clavicular head of the sternocleidomastoid (SCM) (Fig. 10.15). The entire length of the muscle is not accessible with this stripping technique so just treat the accessible fibers. If the client reports an increase in neurological or vascular symptoms, immediately cease the technique as there may be additional pressure on the primary region of pathology.



Figure 10.15 Deep stripping on scalenes.

**E. Stripping with active engagement** It can be difficult to apply adequate and effective pressure on the scalene muscles with stripping techniques because of the sensitivity of this region and the delicate structures nearby. Adding active engagement increases the effectiveness of the stripping technique. The client is in a supine position with the head partially rotated to the opposite side and off the end of the table. If the client does not feel comfortable with the head off the end of the table, use a bolster or pillow under the upper back so there is room to move the head in hyperextension.

Hold the client's head with one hand while the other hand is prepared to apply the stripping technique. Ask the client to lift the head slightly and hold it there for a few seconds (the practitioner's hand keeps light contact with the client's head, but the client is now holding their own head up). After holding that contraction for 5–8 seconds, instruct the client to slowly let go of the contraction. As the contraction is slowly released, gently guide their head into hyperextension with one hand while the other hand performs a stripping technique on the anterior and middle scalenes as in D above (Fig. 10.16). Repeat this technique several times for maximum effectiveness. Do not perform this technique if there is a concern about vertebrobasilar insufficiency (see the cautions and contraindications below).

**F. Muscle energy technique for anterior/lateral neck muscles and fascia** In some cases additional pressure in the region of the brachial plexus is uncomfortable for the client because it aggravates neurological or vascular symptoms. In those cases



Figure 10.16 Stripping with active engagement (lengthening) on scalenes.

this MET procedure is an effective way to enhance lengthening in the scalene muscles without putting direct pressure on them. This technique is best performed without lubricant on the client's skin. The client is in a supine position and in a position like E above where the head can be relaxed off the end of the table or over a bolster. One hand holds the client's head and the other hand is positioned over the scalene muscles or their distal fascial connections. Ask the client to lift their head out of the hand and hold it there for a few seconds (the practitioner's hand keeps contact with the client's head, but the client is now holding the weight of their own head). After holding that contraction for 5–8 seconds, instruct the client to slowly let go of the contraction. As they slowly let go of the contraction let their head drop back into extension while applying an inferiorly directed traction force to the scalenes and the fascial tissues connected with them (Fig. 10.17). The force of the fingertips pulls on the skin and superficial fascia more than pressing down into the scalenes. Repeat this technique several times and apply at different points along the length of the scalene muscles or their distal fascia. Do not perform this technique if there is a concern about vertebrobasilar insufficiency (see the cautions and contraindications below).

#### *Pectoralis minor syndrome*

**A. Deep stripping on pectoralis minor** The client is in a supine position. Perform sufficient warming techniques, such as effleurage and sweeping cross fiber, to reduce tension in the pectoralis



Figure 10.17 MET for scalenes and their fascial connections.

major muscle so the pectoralis minor is more easily accessible. Perform a deep longitudinal stripping technique on the pectoralis minor beginning at the coracoid process of the scapula and moving inferiorly to its attachments on ribs 3, 4, and 5 (Fig. 10.18). Pressure is applied directly through the pectoralis major muscle and this region can be somewhat tender, so use care in the amount of pressure that is applied. Immediately move off the area if the technique reproduces or exacerbates neurological symptoms.

**B. Pin and stretch for pectoralis minor** The client is in a supine position. The practitioner is at the level of the client's head facing their feet. Place a thumb or finger tip in contact with the pectoralis minor muscle but not on any area that exaggerates or reproduces neurological symptoms when pressure is applied. Instruct the client to

reach as far toward their toes as possible. This motion depresses the scapula and puts the pectoralis minor in a shortened position. With the muscle in this position apply moderate to significant pressure (within the client's comfort tolerance) to the pectoralis minor muscle. Then instruct the client to hike the shoulder up as if bringing it as close to the ear as possible. While they are elevating the shoulder girdle, apply static compression or a short stripping technique to the pectoralis minor muscle (Fig. 10.19). Repeat this technique several times to encourage lengthening of the pectoralis minor.

**C. Neural mobilization technique** Neural mobilization is helpful to reduce binding or restriction on nerves of the upper extremity that could be affected by regions of entrapment near the thoracic outlet. Most thoracic outlet variations affect the ulnar nerve so this neural mobility technique emphasizes the ulnar nerve. The client is in a supine position with the arm at the side. Hyperextend the client's wrist and fingers and bring the forearm into position of elbow flexion. From that position abduct the shoulder so it appears the client's hand is coming close to covering the ear (Fig. 10.20). At the far end of this movement instruct the client to laterally flex the head to the opposite side as the hand is brought up near the side of the head. Do not hold the final stretch position. Bring the shoulder and elbow back to neutral positions. Immediately repeat this series of movements several times. The idea is to repeatedly pull on the nerves of the brachial plexus and reduce binding or restriction between the nerves and adjacent tissues.



Figure 10.18 Deep stripping on pectoralis minor.



Figure 10.19 Pin and stretch on pectoralis minor.



Figure 10.20 Neural mobilization for ulnar nerve.

### Rehabilitation protocol considerations

- Strength training for shoulder or neck muscles is sometimes advocated to address postural distortions that contribute to neurovascular compression. Postural retraining should be initiated and routinely reinforced prior to significant strength training activities so dysfunctional patterns are not strengthened or reinforced.
- In more severe cases, some of the suggested treatment techniques are not recommended because the neurological symptoms are severe and any additional pressure on the region aggravates the symptoms. In those cases, simply reduce the pressure applied in the techniques and focus on treatment methods such as the MET technique described above that doesn't put additional pressure on the neurovascular structures.
- Be sure to treat the entire upper extremity as part of the TOS treatment strategy because there can be multiple regions of nerve entrapment that could be aggravating symptoms.
- The neural mobility technique described in C under Pectoralis minor syndrome treatment above should be performed after the entire upper extremity has been treated. Neural mobility procedures are more effective when the soft tissues along the entire path of the nerve are as relaxed and pliable as possible.

**Cautions and contraindications** The practitioner should be cautious about applying pressure in any of the affected regions. Because TOS involves nerve compression, additional compression can aggravate the problem. The practitioner should stay in close

communication with the client about symptoms so treatment can be immediately modified.

Some of the neural stretching procedures can create an immediate increase of symptoms. Exacerbation of symptoms can be kept to a minimal level if treatment is ceased when symptoms increase. Do not overstretch neural structures when performing neural mobility techniques. If symptoms recur prior to the ending position of the described movements, do not pursue additional movements past the point where symptoms recur.

The position with the client supine and head moving into hyperextension described in E and F under anterior scalene syndrome and costoclavicular syndrome above can produce an adverse effect in some people. This position with the head turned to the side while it is being hyperextended can compress the vertebral artery and decrease blood flow to the brain causing dizziness, vertigo, blurred vision, or fainting. This is a condition called *vertebrobasilar insufficiency*. Prior to performing this technique it is helpful to perform the vertebral artery test to see if the client is potentially susceptible to this compression. With the client in a seated position ask them to look up and over their shoulder so the head is in hyperextension and rotation. Hold that position for about 30 seconds. If the client begins to feel sensations of dizziness, vertigo, nausea, vision problems, or other neurological symptoms, this is a positive result and indicates they are susceptible to vertebral artery compression. If that is the case, those treatment techniques should be avoided.

## SPASMODIC TORTICOLLIS

### Description

Spasmodic torticollis is a condition of continual muscle spasm that affects the extensor and rotator muscles of the neck. It usually makes the individual's head turn to the side in lateral flexion and/or rotation with some hyperextension as if they were attempting to look over their shoulder. It is most common on one side only. Another form of torticollis, congenital torticollis, arises from difficulty in traveling through the birth canal. Spasmodic torticollis is a different process; it appears to be a central nervous system dysfunction.



This condition is also called wry neck or cervical dystonia. Dystonia is a neurological movement disorder characterized by involuntary muscle contractions that force the body into abnormal and sometimes painful movements or postures. The cause for spasmodic torticollis is unknown, although there does seem to be some central nervous system dysfunction. In spasmodic torticollis, muscles develop a degree of fibrotic change and contracture within the tissue. Pain in this condition may not be limited to the muscles alone, but may involve dysfunctional central nervous system responses.<sup>32</sup>

Some individuals may develop spasm in cervical muscles. These spasms can happen as the result of sleeping in an awkward position for long periods or even from having a cool draft on the cervical region during the night.<sup>20</sup> Spasmodic torticollis is different as the degree of spasm in the muscles is much greater with torticollis, and torticollis is often harder to resolve than a muscle spasm that results from long periods of awkward positioning.

## Treatment

### *Traditional approaches*

Stretching and various physical therapy modalities are commonly used for treatment of spasmodic torticollis. A primary focus is on the level of muscle dysfunction, as well as attempting to normalize any contributing factors from the central nervous system. Medications are sometimes used to address the muscle spasms. Biofeedback and hypnosis have also been used with some clinical success.

When conservative measures are not effective, another treatment that is used is injection therapy. In this procedure, a small amount of botulinum toxin A (known by its commercial name, BOTOX®) is injected into the affected muscles. BOTOX® is a neurotoxic substance that essentially prevents the release of acetylcholine, which interrupts muscle function.<sup>33</sup> The interruption of muscle function is an effective means of reducing the spastic contractions in the neck muscles. The amount of botulinum toxin used in this procedure is well below the level that is poisonous to a human.

In addition to botulinum toxin injections, surgical procedures may also be used. Surgery will focus on denervating the involved muscles so they

do not perpetuate their dysfunctional spasm. Surgery alone is generally not as effective as surgery combined with the injection therapy.<sup>34</sup>

### *Soft-tissue manipulation*

**General guidelines** The primary focus of massage treatment for torticollis is to reduce the chronic muscle spasms that occur in this condition. It is not clear if massage can help with the aspects of this condition that are mediated by the central nervous system. However, due to the systemic effects of massage in lowering stress levels and overall neuromuscular tension, massage might be beneficial for the central nervous system components of the disorder as well.

Always strive to work gently within the client's comfort zone. Any increased tonus level that is stimulated through excessive pressure may cause an increased central nervous system response. This will be detrimental to the primary goal of treatment. The foremost aim of treatment is to reduce muscle spasms and excess central nervous system activity that is contributing to the disorder. A treatment environment that is conducive to overall relaxation is desirable for achieving those outcomes. The cumulative effect of environmental factors on reducing central nervous system stress should not be ignored; for example, a long treatment session (more than 45 minutes), a dimly lit room, and relaxing music. The factors that contribute to nervous system relaxation should be maximized as much as possible.

### *Suggested techniques and methods*

**A. Myofascial approaches** Myofascial techniques encourage fascial and muscular relaxation. Gentle pressure is placed on the fascia in multiple directions. Relaxing the fascia has benefits in reducing muscular tension and decreasing any restrictions on neural mobility. The primary goal is to place a moderately light pressure on the subcutaneous fascia. Pulling the fascia in multiple directions appears to enhance its pliability the most. Place the hands lightly on the client's neck where the myofascial stretch is to be applied. Pull the hands apart to take the slack out of the tissue and apply a light degree of pulling force between the hands (Fig. 10.1). Once there is a slight degree of pull between the hands, hold this position until there

is a subtle sensation of tissue release. Apply these fascial techniques specifically to the muscles in the neck that are engaged in spasm due to torticollis.

**B. Effleurage and sweeping cross fiber** These techniques are effective methods for reducing tension in superficial neck muscles. Effleurage is performed with long gliding strokes parallel to the muscle fiber direction. The cross fiber techniques sweep diagonally across the primary fiber direction of muscles in different regions of the neck (Fig. 10.2). Use care with the pressure level in effleurage or sweeping cross fiber motions as there are sensitive vascular structures that are superficial in the anterior neck region. Emphasize long gliding strokes with the muscles that are in spasm as these long strokes help reduce excess neurological activity.

**C. Pin and stretch on sternocleidomastoid** The SCM is one of the primary muscles that maintain the dysfunctional torticollis position when in spasm. The SCM can be difficult to treat. Due to the layers of soft tissues and sensitive structures underneath the SCM, significant pressure should not be used. Depending on the severity of the torticollis, the head may not be able to move much. Perform what is within the client's comfort tolerance and gradually work towards improvement. The description below assumes a greater range of motion is possible.

This pin and stretch technique allows direct pressure and stretching on the SCM without pushing down into anterior neck structures. This technique can be performed actively with the client moving their own head or passively with the practitioner moving the client's head. Place one hand underneath the client's head in a position where their head can adequately be moved in a number of different directions. Move their head into a position of flexion and contralateral rotation (e.g. turning left if treating the right SCM). This position puts the SCM in a fully shortened position. Grasp the SCM muscle between the thumb and fingers. Either actively or passively rotate the client's head into extension and ipsilateral rotation, as if they are looking up over their shoulder (Fig. 10.21). Do not perform this technique if there is a concern about vertebrasilar insufficiency (see the cautions and contraindications below).



Figure 10.21 Pin and stretch on sternocleidomastoid (SCM).

**D. MET using reciprocal inhibition for neck rotators** In many cases muscle spasm does not respond well to direct manipulation, but active-assisted stretching using reciprocal inhibition can achieve beneficial results by using appropriate neurological principles. This technique assumes attention is focused on the SCM, a contralateral neck rotator muscle. The technique can be modified to address the contributions of other muscles, such as the flexors or lateral flexors of the neck that may be contributing to the disorder. The description assumes the right side is being treated. Reverse the instructions to address the left side SCM. The client is in a supine position. Place one hand gently on the right side of the client's forehead. Instruct the client to attempt to rotate the head to the right while offering resistance for about 5–8 seconds. This resisted action engages the left SCM (and the right side ipsilateral rotators). When they contract there is decreased neurological activity in our target muscle, the right SCM. Instruct the client to release the contraction. As they let go of the contraction gently attempt to turn the head in same direction that they were attempting (to the right). This will stretch the SCM on the right side (Fig. 10.22).

#### Rehabilitation protocol considerations

- Depending on the severity of the spasm in torticollis, different massage techniques are used. In a severe case, there may be very little that direct soft-tissue manipulation can do due to client pain. In these cases, begin with less invasive



Figure 10.22 MET with reciprocal inhibition for cervical contralateral rotators.

techniques such as the MET with reciprocal inhibition described in D above.

- Myofascial techniques have clear effects on reducing neuromuscular activity through mild tensile loads applied to the tissues. Due to their gentle intervention, these techniques are advantageous in more severe cases when there is a significant amount of muscle spasm.

**Cautions and contraindications** There may be significant pain and discomfort associated with spasmodic torticollis. Be aware of the client's reported level of discomfort and adjust treatments accordingly. It is more beneficial to work slowly on improving central nervous system effects over a number of treatments, rather than trying to reduce all the symptoms in one or two sessions.

Special precautions should be taken with massage treatment for a client who is currently receiving botulinum injection therapy. No studies have been published on the effects of massage in conjunction with botulinum toxin injections. Because of the potential that massage treatments have to enhance circulation, there may be some adverse interference with the local administration of the medication. It is best to get clearance from a physician about massage treatment in this area for a client who is currently receiving injection therapy.

The position with the client supine and head moving into hyperextension described in C can produce an adverse effect in some people. This position with the head turned to the side while it is being hyperextended can compress the vertebral

artery and decrease blood flow to the brain causing dizziness, vertigo, blurred vision, or fainting. Prior to performing this technique, perform the vertebral artery test to see if the client is potentially susceptible to this compression. With the client in a seated position ask them to look up and over their shoulder so the head is in hyperextension and rotation. The client holds the position for about 30 seconds. If the client begins to feel sensations of dizziness, vertigo, nausea, vision problems, or other neurological symptoms, they are susceptible to vertebral artery compression and those treatment techniques should be avoided. Depending on the severity of the torticollis the client may not be able to make these movements in a position that would be enough to compress the vertebral artery. However, as greater motion is achieved it could be an issue.

## WHIPLASH

### Description

Whiplash is a frequently misunderstood condition, as it is not a single condition. Whiplash is an injury that occurs as the result of a sudden acceleration or deceleration of the head and neck in relation to the torso. Numerous problems can result from whiplash. Consequently, it is not appropriate to speak of whiplash as an isolated condition. It is more appropriate to describe it as a broad spectrum of possible disorders. It is now commonly referred to as whiplash associated disorder (WAD), indicating that whiplash is the mechanism of injury, but numerous tissues could be injured as a result.

One of the most important classifications of WAD published to date came from the Quebec Task Force on Whiplash-Associated Disorders.<sup>35</sup> They have classified WAD into four categories of severity:

- Category 1: Neck complaint without musculoskeletal signs such as loss of mobility.
- Category 2: Neck complaint with musculoskeletal signs such as loss of mobility.
- Category 3: Neck complaint with neurological signs.
- Category 4: Neck complaint with cervical fracture or dislocation.

It is apparent from looking at this classification that various pathologies fall under the description of whiplash – from simple musculoskeletal

irritation, all the way to cervical fracture and severe neurological impairment. Assessment of the injury level is crucial for any practitioner attempting to treat whiplash disorders.

The approach to classifying whiplash and designing appropriate treatment protocols has been challenging because of the different symptoms that occur some time after the initial incident. Although the Task Force has set up a helpful system for classifying whiplash problems, their conclusions are not without controversy. There is still a call for a better understanding of WAD.<sup>36,37</sup>

Whiplash injuries can cause damage to muscles, neural structures, ligaments, tendons, fascia, facet joints, bones, intervertebral discs, or vascular structures. Pain may be local in the neck, or it may radiate into the head, shoulder, or upper extremity. Some people suffering from whiplash experience impairment of memory, concentration, or sleep, as well as fatigue, depression, and various forms of psychological distress.<sup>38,39</sup> The practitioner should be on the lookout for any of these symptoms if their client has suffered a whiplash trauma.

A challenging aspect of WAD is matching the onset of symptoms to the actual structures that are damaged. It is common for whiplash symptoms to emerge days, weeks, or even months after the initial trauma. Explanations for this delay of symptoms include delayed inflammatory effects or altered mechanics that take a while to produce symptoms. There is also evidence of increased central nervous system sensitivity that could be playing a role in delayed onset of tissue sensitivity.<sup>40,41</sup>

If not severe, whiplash conditions are generally limited to soft-tissue injury. Cervical muscles can be strained or under protective spasm. Ligaments of the cervical region can be sprained from excess joint motion. In many instances, myofascial trigger points develop in the cervical muscles following the injury. Trigger points lead to patterns of facilitated muscular dysfunction that often linger for months or years after the initial trauma.<sup>20</sup>

## Treatment

### *Traditional approaches*

As with other conditions, rest from offending activity is essential, especially in the early phases

after the injury. It is common practice to begin rehabilitation of whiplash injury as soon as the inflammatory phase has subsided. This may be difficult to identify in many cases, however, because visible inflammation is mostly absent. A general guideline of 48–72 hours after the initial incident is a common period for the inflammatory stage. When there is relief of symptoms from various positions other than static rest, some degree of managed treatment can begin.<sup>5</sup>

For years, the cervical collar was a mainstay of treatment for whiplash, and the practice persists. However, health care practitioners now realize that unless there is a severe level of damage, early, protected mobilization is more beneficial than immobilization in a collar.<sup>42–45</sup> When cervical collars are used, whether soft or hard collars, they can cause further complications from the initial trauma. It is common for temporomandibular dysfunction, joint adhesions, muscle atrophy, and myofascial trigger points to develop from long periods of immobilization in a cervical collar.<sup>46</sup>

Various drugs, such as anti-inflammatory medications or muscle relaxants, may be used to treat whiplash symptoms. Cervical traction units may also provide relief for symptoms. This will be especially true in instances where there is compression of the vertebral bodies or facet joints.<sup>47</sup> Remember that numerous tissues can be the source of whiplash pain so evaluation of these potential problems should be thorough.

### *Soft-tissue manipulation*

**General guidelines** If a specific tissue injury can be identified as the source of pain, such as a strain to the splenius capitis, massage treatment focuses directly on that tissue injury. Use comprehensive orthopedic assessment to clarify the nature of the tissue injury as accurately as possible. Without identification of a specific tissue that is damaged, soft-tissue treatments are more general in nature. For example, it is difficult to treat some ligaments of the cervical spine that are sprained because they are deep along the cervical spine or near the atlanto-occipital joint and too deep to access. However, the resultant muscular dysfunction that occurs from excessive neurological activity and movement restriction can become a serious part

of the whiplash-associated disorder, and this muscular dysfunction is treatable with massage.

The appropriate form of massage depends greatly upon what tissues are injured. Techniques such as effleurage, sweeping cross fiber, active engagement, as well as active-assisted massage are useful at various stages of whiplash rehabilitation. Gentle stretching in the cervical region is also an integral part of the treatment process.

**Suggested techniques and methods** Depending on the exact nature of the injury, which is determined by proper assessment, any of the techniques A–G described in the section Neuromuscular neck pain can be valuable in treating WAD. In some cases the muscles or other soft tissues of the cervical region are too sensitive for direct soft-tissue manipulation. The additional techniques included below can be used in the early stages of rehabilitation when it is too early for other soft-tissue work.

Because there are a number of tissues that can be injured in a whiplash injury, it may be difficult to identify the severity or nature of the injury. In cases that appear to be more than a mild muscular whiplash disorder referral to a physician is strongly recommended to screen for more serious pathologies.

**A. MET with reciprocal inhibition for cervical extensors** This technique emphasizes muscular dysfunction in the cervical extensors following the whiplash injury. The client is in a supine position to begin. Gently cradle one hand behind the client's head and place the other hand on the client's forehead. Instruct the client to slowly lift the head off the treatment table against resistance. Hold the contraction for about 5–8 seconds. Depending on the severity of the whiplash injury, engaging the cervical muscles in a contraction may be uncomfortable or cause further pain. Only engage this muscle contraction within the client's pain or comfort tolerance. Instruct the client to release the contraction. As they release the contraction gently push the head farther into flexion to stretch the posterior cervical muscles (Fig. 10.23).

**B. MET with reciprocal inhibition for lateral flexors** The client is in a supine position. Instructions for this technique assume that the lateral rotators on the right side of the neck are being



Figure 10.23 MET with reciprocal inhibition for cervical extensors.

treated. Place one hand against the left side of the client's head and the other against the client's right shoulder. It might be more helpful to cross the arms and have the right hand underneath the client's head with fingers on the side of their head and the left hand on the client's shoulder. Instruct the client to hold this position as the practitioner attempts to laterally flex the client's head to the right (the client engages the left lateral flexors to resist this motion). The client holds that contraction for about 5–8 seconds. Instruct the client to release the contraction and as the contraction is released, move the head into left lateral flexion to stretch the lateral flexor muscles on the right side (Fig. 10.24).

**C. MET with reciprocal inhibition for anterior neck flexors** Deep flexors of the neck, such as the



Figure 10.24 MET with reciprocal inhibition for lateral neck flexors.

longus colli and longus capitis, are commonly injured in rear-impact whiplash incidents. These muscles are deep in the neck and lie very close to the cervical spine. This MET procedure avoids pressure on the baroreceptors in the carotid sinus (see this important contraindication below), but still helps treat the deep neck flexor muscles.

The client is in a supine position with the head off the end of the table or the upper back supported by a bolster so the head can move back in extension. Both of the practitioner's hands are holding the head. Holding the head with both hands is important to give the client maximum confidence that no sudden or forceful neck movement will result. Starting in a neutral position instruct the client to gently push their head into the hands for about 5–8 seconds as resistance is offered. After holding that contraction, instruct the client to let go of the contraction and gently let the head drop back into further extension stretching the deep neck flexors on the anterior side of the neck (Fig. 10.25). Only perform this stretch within the client's comfort tolerance. Do not perform this technique if there is a concern about vertebrobasilar insufficiency (see the cautions and contraindications below).

#### Rehabilitation protocol considerations

- WAD runs the gamut from mild muscular irritation to severe and life-threatening cervical injury. Accurate assessment of the injury is crucial to determine the severity of tissue damage and what types of massage treatment are appropriate or potentially contraindicated.



Figure 10.25 MET with reciprocal inhibition for deep anterior neck flexors.

- In the early stages of the injury use gentle techniques that emphasize neurological responses without putting excess pressure on muscle and other soft tissues. Myofascial approaches and active-assisted stretching procedures, such as the MET with and without reciprocal inhibition, are particularly well suited to the early stages of a whiplash injury
- Positional release is also a good treatment choice in the early stages of whiplash injury because it does not require invasive pressure and uses gentle neurological responses to achieve treatment objectives.
- Due to excessive neuromuscular tension following WAD injuries, thermal modalities are useful adjuncts to soft-tissue treatment. Ice and cold applications are preferable in the immediate aftermath of the injury to reduce inflammatory effects. However, ice is also valuable in the later stages with muscular spasm because of its ability to slow nerve conduction velocity and interrupt muscle spasm. If the WAD has moved into the sub-acute or chronic phase, individuals respond well to heat applications because they reduce neuromuscular tension and settle down excessive sympathetic nervous system activity.
- Early mobilization and gentle exercise is a mainstay of WAD treatment. More vigorous movement retraining, strengthening, and conditioning are also a part of WAD treatment to restore proper strength and resilience to the tissues in this area. However, due to the heightened neurological stress in the area following this type of injury and the increased central nervous system sensitivity, it is best to wait later in the rehabilitation process for significant strength-training activities. Strength training or conditioning that is begun too soon can cause reactive muscular spasms as the body is readjusting to proprioceptive feedback from the injured muscles.

**Cautions and contraindications** One of the concerns in working with clients with a WAD injury is identifying what the problem really is. Because symptoms can be delayed, do not perform any treatment technique that is going to make the condition worse. It is not unusual for clients to go through

therapy, massage and other types, for whiplash treatment and come out feeling worse. In these cases, the practitioner may have been too aggressive with the treatment, or simply used an approach that was improper for the nature or stage of the injury.

Acquire thorough and complete information from the client related to the symptoms they have experienced and those they are currently experiencing in order to make responsible clinical decisions. Watch for symptoms that indicate the need for intervention of another health professional for further and more complete evaluation. If there is uncertainty of the severity or nature of the whiplash injury, it is best to refer that person out for a more comprehensive evaluation prior to treatment. Underlying dysfunctions such as severe joint instability may not be immediately apparent due to protective muscle spasm. Overly enthusiastic stretching or movement techniques could then cause joint subluxation or dislocation.

Some treatment techniques for the deep neck flexors, such as the longus colli and longus capitis, advocate direct treatment of the muscles. This direct deep treatment usually involves displacing the trachea to the side so muscles in the anterior neck can be directly accessed. Unfortunately these muscles are also very close to sensitive tissues such as the thyroid gland and vascular structures such as the carotid artery and carotid sinus. The carotid sinus contains sensitive neurological receptors called baroreceptors that are important in regulating blood pressure. Attempts at putting pressure on the deep neck flexors could inadvertently put pressure on the baroreceptors and cause an unintentional alteration of blood pressure with adverse effects. Indirect treatment techniques such as active-assisted stretching (see the MET procedures described in C above) avoid direct deep pressure in the anterior neck region, but still provide effective treatment of these important muscles.

The position with the client supine and head moving into hyperextension described in MET with reciprocal inhibition for anterior neck flexors can produce an adverse effect in some people. This position can compress the vertebral artery and decrease blood flow to the brain causing dizziness, vertigo, blurred vision, or fainting (a condition called vertebrobasilar insufficiency). Prior to performing this technique it is helpful to perform

the vertebral artery test. With the client in a seated position ask them to look up and over their shoulder so the head is in hyperextension and rotation. The client holds the position for about 30 seconds. If the client begins to feel sensations of dizziness, vertigo, nausea, vision problems, or other neurological symptoms, this is a positive result and indicates they are susceptible to vertebral artery compression. If that is the case, this treatment technique should be avoided.

## POSTURAL DISORDERS

This section includes a discussion of forward head posture, the most common postural distortion in the cervical region. It is not considered an injury condition, as are the prior conditions in this chapter. However, it can produce considerable stress on other tissues or structures and contribute to their dysfunction. Massage is a valuable tool in addressing this postural condition, due to the combination of muscle dysfunctions that create it.

### FORWARD HEAD POSTURE

Because humans stand upright in a vertical gravity plane and the head is a heavy weight, we are susceptible to the distortion of a forward head posture. Chronic dysfunctional posture is the likely cause. This condition usually involves muscular distortion in the thoracic and cervical regions. In forward head posture, there is extension in the upper cervical vertebrae and flexion in the lower cervical vertebrae. When the head is positioned forward of the line of gravity, a tensile load is placed on the posterior cervical extensor muscles. These muscles keep the eyes looking straight ahead and prevent the head from falling forward. Even a slight degree of forward head posture increases stress on the posterior neck muscles.

In addition there is an increased compressive load on the posterior vertebral arch structures. For every inch the head moves forward from its normal posture, the compressive load on the lower neck is approximately equal to that number times the weight of the head (e.g. 2 inches forward head posture equals two times the weight of the head).<sup>5</sup>

In a forward head posture the posterior cervical muscles must work harder to keep the head upright. As a result fatigue, tension, and myofascial trigger points develop. Muscle tension and myofascial trigger point referrals can produce pain in the head, neck, upper back, and temporomandibular joint. Muscular irritation from forward head posture is at the root of numerous head and neck pain conditions. For example, myofascial trigger points that develop in the sub-occipital muscles from forward head posture frequently produce headache pain. The increased compressive loads can eventually lead to vertebral stress fractures or cervical facet (zygapophysial) joint irritation.

The previous chapter discussed the lower crossed syndrome, a condition that involves the relationship between postural and phasic muscles in the lumbar region. A similar relationship exists in the cervical region and is referred to as the upper crossed syndrome.<sup>46</sup> The upper crossed syndrome often exists in conjunction with the lower crossed syndrome. Postural and phasic muscles differ in their fiber type and activation patterns. When overused and fatigued, postural muscles tend to become hypertonic, while phasic muscles tend to become weak and inhibited. The phasic muscles are antagonists to postural muscles. Because postural muscles tend toward hypertonicity, they create a functional weakness in the phasic muscles through the process of reciprocal inhibition. A graphical comparison of their position and functional relationships with each other shows how they interact in the upper crossed syndrome (Fig. 10.26).

The postural muscles in the neck prone to hypertonicity include the upper trapezius, levator scapulae, and cervical extensors on the posterior aspect, as well as the pectoralis major and minor in the front.<sup>5</sup> The postural muscles are known to house active myofascial trigger points and refer characteristic sensations when palpated. Phasic muscles that are prone to weakness include the deep neck flexors, such as the longus colli and longus capitis, as well as the lower trapezius and serratus anterior.<sup>5</sup>

Forward head posture results from poor postural habits, such as leaning forward toward a computer screen for long periods. This condition also occurs alongside upper thoracic kyphosis or

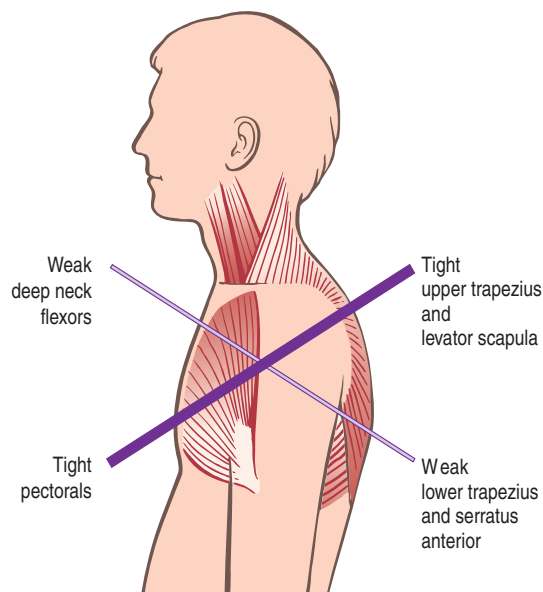


Figure 10.26 Forward head posture and upper crossed syndrome (from Chaitow L, DeLany J. *Clinical Application of Neuromuscular Techniques*. Vol 1. Edinburgh: Churchill Livingstone; 2000).

excessive lordosis and can be a result of osteoporosis. Due to the postural compensations that naturally occur with age, forward head posture is more prevalent in older adults.

## Treatment

### *Traditional approaches*

Traditional treatment focuses on strength training and postural re-education. Ergonomic changes at work play an important role and any method of postural retraining that reinforces correct cervical alignment is beneficial. Certain postural retraining techniques, such as the Alexander technique, are frequently successful in the correction and retraining of forward head posture. Faulty joint alignment of the forward head posture is often treated with manipulation or mobilization.

### *Soft-tissue manipulation*

**General guidelines** The primary focus of massage treatment in a forward head posture is to reduce tension in the shortened muscles so proper alignment can be regained. Superficial posterior cervical



muscles, such as the trapezius, splenius capitis, and semispinalis capitis, are shortened in the condition and should be a focus of treatment. In addition, it is important to treat the four small sub-occipital muscles on each side of the spine. These muscles develop myofascial trigger points from their chronic shortened position and are a frequent cause of muscle tension headaches.

Forward head posture originates not only in the cervical muscles, but also in the corresponding pattern of kyphosis that develops in the upper thoracic region. Treatment emphasizes the shortened muscles of the upper anterior torso and posterior cervical region. These hypertonic muscles often develop myofascial trigger points so they can be treated with static compression, deep longitudinal stripping, and eventually with active engagement methods as well. Certain muscles, such as the rhomboids and mid-trapezius that are held in a lengthened position due to this posture, also become painful from fatigue. Similar techniques can be applied to these muscles.

**Suggested techniques and methods** Soft-tissue treatment and rehabilitation protocol considerations for a forward head posture are the same as those described for kyphosis in Chapter 9. In addition, the techniques listed under neuromuscular neck pain at the beginning of this chapter are valuable for treating forward head posture. Two other techniques that are helpful in treating the suboccipital muscles are also included.

**A. Static compression on suboccipital muscles** The client is in a supine position. To deactivate myofascial trigger points in the posterior cervical and suboccipital muscles, apply static compression with the fingertips or thumb to the posterior cervical and suboccipital region one side at a time (Fig. 10.27). Hold the static compression on these regions until the client reports a decrease in the irritability of the muscle tension (about 5–8 seconds).

**B. Static compression with head rotation** This technique is a variation of the one described above. It allows for an alternating amount of pressure to the suboccipital muscles. The practitioner is to the side of the client's head. Gently rotate the client's head to the opposite side with one hand. With the other hand place the thumb on the



Figure 10.27 Static compression on suboccipital muscles.

suboccipital muscles so that it is pressing up into the muscle tissue just below the occiput. With the hand that is holding the head, gently rotate the head onto the thumb of the other hand, which gradually applies more pressure to the suboccipital muscles (Fig. 10.28). Hold the static compression until there is some sensation of tissue release in the suboccipital muscles or the client reports a reduction in pain sensations.

**Cautions and contraindications** Forward head posture and kyphosis are related. Some cases of kyphosis are caused by weakness and degeneration in bony structures as in osteoporosis. People often develop their kyphotic posture with age as gravity gradually takes a toll on the upright vertical structure. In either case, especially that of osteoporosis, there can be fragility in the skeletal structures. Use caution when working in these



Figure 10.28 Static compression on suboccipital muscles with head turned into thumb.

areas, especially with techniques that use greater pressure, such as the deep stripping or static compression methods.

When treating the suboccipital muscles be cautious of treatment techniques that apply pressure into the suboccipital region bilaterally at the same time. These techniques can potentially produce compression of the vertebral arteries and reduce cranial blood flow (vertebrobasilar insufficiency). A way to avoid the potential of arterial insufficiency is to only treat the suboccipital region on one side of the head at a time. That way there is still an opportunity for normal blood flow on one side.

If there is concern that the client might have adverse arterial compression with treatment, evaluate their susceptibility with the vertebral artery test. With the client in a seated position ask them to look up and over their shoulder so the head is in hyperextension and rotation. Hold that position for about 30 seconds. If the client begins to feel any sensations of dizziness, vertigo, nausea, vision problems, or any other neurological symptoms, they are susceptible to vertebral artery compression, and extra care should be exercised when treating the suboccipital region.

### Box 10.3 Case Study

#### Background

Miriam is a 52-year-old who is in good physical condition. She spends a good deal of her day at the computer doing freelance writing projects for the local magazines and newspaper. Three weeks ago she was in an automobile accident where someone ran a traffic light and hit her on the passenger side of her car. She was not hurt badly in the accident and was able to walk away. However, she did go see a doctor the next day because she was having neck and arm pain. The doctor said there were no fractures or serious joint trauma and most likely her pain was related to soft-tissue injury.

The neck and arm pain has persisted since her injury. She has also been experiencing regular headaches that come on about halfway through her day. She is not able to spend anywhere near as much time at her computer now because the headaches as well as neck and arm discomfort prevent long periods of her writing. She reports that ice applications have been helpful initially with managing the pain.

#### Questions to consider

- Due to the mechanics of the injury in the automobile accident, what are several different tissues that could have been injured?
- She reported that ice has been helpful in pain management. What are the primary physiological effects of ice applications that are likely to be helping reduce her pain?
- She was hit broadside from the passenger side of her car. Which side of her neck received the initial tensile stress from the immediate force of the collision?
- What are several factors that may help you decide if it is appropriate to treat Miriam with massage?
- What are several questions you would want to ask Miriam in your initial evaluation with her?
- If it turns out that she has a sudden tensile stress injury to the brachial plexus from the collision, is this something that can be treated with massage?

### References

1. Mense S, Simons DG. *Muscle Pain: Understanding Its Nature, Diagnosis, & Treatment*. Baltimore: Lippincott Williams & Wilkins; 2001.
2. Neumann DA. *Kinesiology of the Musculoskeletal System*. St. Louis: Mosby; 2002.
3. Janda V. Rational therapeutic approach of chronic back pain syndromes. Paper presented at: Chronic back pain, rehabilitation, and self-help, 1985; Turku, Finland.
4. Friedman MH, Weisberg J. The craniocervical connection: a retrospective analysis of 300 whiplash patients with

- cervical and temporomandibular disorders. *Cranio*. 2000;18(3):163–167.
5. Liebenson Ce. *Rehabilitation of the Spine*. Baltimore: Williams & Wilkins; 1996.
  6. Pavlov H, Torg JS, Robie B, Jahre C. Cervical spinal stenosis: determination with vertebral body ratio method. *Radiology*. 1987;164(3):771–775.
  7. Buckwalter JA. Aging and degeneration of the human intervertebral disc. *Spine*. 1995;20(11):1307–1314.
  8. Le Maitre CL, Freemont AJ, Hoyland JA. Accelerated cellular senescence in degenerate intervertebral discs: a possible role in the pathogenesis of intervertebral disc degeneration. *Arthritis Res Ther*. 2007;9(3):R45.
  9. Podichetty VK. The aging spine: the role of inflammatory mediators in intervertebral disc degeneration. *Cell Mol Biol (Noisy-le-grand)*. 2007;53(5):4–18.
  10. Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am*. 1990;72(8):1178–1184.
  11. Matsumoto M, Fujimura Y, Suzuki N, et al. MRI of cervical intervertebral discs in asymptomatic subjects. *J Bone Joint Surg Br*. 1998;80(1):19–24.
  12. Magee D. *Orthopedic Physical Assessment*. 3rd ed. Philadelphia: W.B. Saunders; 1997.
  13. Cailliet R. *Neck and Arm Pain*. Philadelphia: F.A. Davis; 1991.
  14. Mendel T, Wink CS, Zimny ML. Neural elements in human cervical intervertebral discs. *Spine*. 1992;17(2):132–135.
  15. Roberts S, Eisenstein SM, Menage J, Evans EH, Ashton IK. Mechanoreceptors in intervertebral discs. Morphology, distribution, and neuropeptides. *Spine*. 1995;20(24):2645–2651.
  16. Bogduk N, Windsor M, Inglis A. The innervation of the cervical intervertebral discs. *Spine*. 1988;13(1):2–8.
  17. Bogduk N, Tynan W, Wilson AS. The nerve supply to the human lumbar intervertebral discs. *J Anat*. 1981;132(Pt 1):39–56.
  18. Matsunaga S, Kabayama S, Yamamoto T, Yone K, Sakou T, Nakanishi K. Strain on intervertebral discs after anterior cervical decompression and fusion. *Spine*. 1999;24(7):670–675.
  19. Choy DS. Percutaneous laser disc decompression (PLDD): twelve years' experience with 752 procedures in 518 patients. *J Clin Laser Med Surg*. 1998;16(6):325–331.
  20. Simons D, Travell J, Simons L. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Vol 1. 2nd ed. Baltimore: Williams & Wilkins; 1999.
  21. Sheth RN, Belzberg AJ. Diagnosis and treatment of thoracic outlet syndrome. *Neurosurg Clin N Am*. 2001;12(2):295–309.
  22. Huang JH, Zager EL. Thoracic outlet syndrome. *Neurosurgery*. 2004;55(4):897–902; discussion 902–893.
  23. Sanders RJ, Hammond SL, Rao NM. Diagnosis of thoracic outlet syndrome. *J Vasc Surg*. 2007;46(3):601–604.
  24. Dawson D, Hallett M, Wilbourn A. *Entrapment Neuropathies*. 3rd ed. Philadelphia: Lippincott-Raven; 1999.
  25. Sucher BM. Thoracic outlet syndrome – a myofascial variant: Part 1. Pathology and diagnosis. *J Am Osteopath Assoc*. 1990;90(8):686–696, 703–684.
  26. Smith TM, Sawyer SF, Sizer PS, Brismee JM. The double crush syndrome: a common occurrence in cyclists with ulnar nerve neuropathy – a case-control study. *Clin J Sport Med*. 2008;18(1):55–61.
  27. Sucher BM. Thoracic outlet syndrome – a myofascial variant: Part 2. Treatment. *J Am Osteopath Assoc*. 1990;90(9):810–812, 817–823.
  28. Baltopoulos P, Tsintzos C, Prionas G, Tsironi M. Exercise-induced scalenus syndrome. *Am J Sports Med*. 2008;36(2):369–374.
  29. Altobelli GG, Kudo T, Haas BT, Chandra FA, Moy JL, Ahn SS. Thoracic outlet syndrome: pattern of clinical success after operative decompression. *J Vasc Surg*. 2005;42(1):122–128.
  30. Butler D. *Mobilisation of the Nervous System*. London: Churchill Livingstone; 1991.
  31. Shacklock M. *Clinical Neurodynamics*. Edinburgh:Elsevier; 2005.
  32. Kutvonen O, Dastidar P, Nurmikko T. Pain in spasmodic torticollis. *Pain*. 1997;69(3):279–286.
  33. Comella CL, Jankovic J, Brin MF. Use of botulinum toxin type A in the treatment of cervical dystonia. *Neurology*. 2000;55(12 Suppl 5):S15–21.
  34. Smith DL, DeMario MC. Spasmodic torticollis: a case report and review of therapies. *J Am Board Fam Pract*. 1996;9(6):435–441.
  35. Spitzer WO, Skovron ML, Salmi LR, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining “whiplash” and its management. *Spine*. 1995;20(8 Suppl):1S–73S.
  36. Alexander D. Quebec task force on whiplash associated disorders challenged. *Journal of Soft Tissue Manipulation*. 1998;6(2):2–3.
  37. Freeman MD, Croft AC, Rossignol AM. “Whiplash associated disorders: redefining whiplash and its management” by the Quebec Task Force. A critical evaluation. *Spine*. 1998;23(9):1043–1049.
  38. Wallis BJ, Lord SM, Barnsley L, Bogduk N. The psychological profiles of patients with whiplash-associated headache. *Cephalalgia*. 1998;18(2):101–105; discussion 172–103.
  39. Young WF. The enigma of whiplash injury; current management strategies and controversies. *Postgrad Med*. 2001;109(3):179–186.
  40. Petersen-Felix S, Arendt-Nielsen L, Curatolo M. Chronic pain after whiplash injury – evidence for altered central sensory processing. *Journal of Whiplash & Related Disorders*. 2003;2(1):5–16.
  41. Curatolo M, Arendt-Nielsen L, Petersen-Felix S. Evidence, mechanisms, and clinical implications of central hypersensitivity in chronic pain after whiplash injury. *Clin J Pain*. 2004;20(6):469–476.

42. Mealy K, Brennan H, Fenelon GC. Early mobilization of acute whiplash injuries. *Br Med J (Clin Res Ed)*. 1986; 292(6521):656–657.
43. Gross AR, Goldsmith C, Hoving JL, et al. Conservative management of mechanical neck disorders: a systematic review. *J Rheumatol*. 2007;34(5):1083–1102.
44. Conlin A, Bhogal S, Sequeira K, Teasell R. Treatment of whiplash-associated disorders – part I: Non-invasive interventions. *Pain Res Manag*. 2005;10(1):21–32.
45. Schnabel M, Ferrari R, Vassiliou T, Kaluza G. Randomised, controlled outcome study of active mobilisation compared with collar therapy for whiplash injury. *Emerg Med J*. 2004;21(3):306–310.
46. Chaitow L, DeLany J. *Clinical Application of Neuromuscular Techniques*. Vol 1. Edinburgh: Churchill Livingstone; 2000.
47. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash. A placebo-controlled prevalence study. *Spine*. 1996;21(15): 1737–1744; discussion 1744–1735.