

Rehabilitation of the Spine

A Practitioner's Manual

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McKenzie Spinal Rehabilitation Methods

Gary Jacob, Robin McKenzie, and Steve Heffner

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Learning Objectives

After reading this chapter you should be able to understand

- McKenzie Method descriptions of patterns of mechanical and symptomatic responses to movement and positioning
- McKenzie Method classification of mechanical and symptomatic response patterns into three syndromes: the postural, dysfunction, and derangement syndromes
- McKenzie Method pathoanatomical explanations of the syndrome patterns
- Management of the postural, dysfunction, and derangement syndromes

Introduction

The goal of rehabilitation is independence in self-care. To serve that purpose, spinal rehabilitation promotes self-efficacy. However, such efforts are often delayed when clinicians provide passive, palliative comfort care while waiting for things to "calm down" before the "good stuff" (rehabilitation) is introduced. The combined fears of patient and practitioner may be roadblocks to the exploration of patient self-generated movements for therapeutic purposes. The specter of dependency and deconditioning of physique and psyche is raised when patients are passive receptacles of care. Any delay in patient active participation is a delay in developing patient empowerment through self-management skills, the ultimate goal of rehabilitation.

This chapter introduces McKenzie Method management of common lower cervical and lower lumbar spinal symptoms, which uses patient-generated movements for acute and chronic symptoms. Whether acute or chronic, McKenzie Method concepts and skills promote independence in self-care from day one, without passive therapy detours on the rehabilitation road to recovery. The McKenzie Method educates patients regarding movement and positioning strategies that have the potential to rapidly ameliorate complaints if the practitioner and patient choose to make self-generated movement and positioning the centerpiece of care.

This chapter attempts to enrich the reader's appreciation of the conceptual foundations of the McKenzie Method to promote facility with its practical applications. Our consideration of McKenzie Method management of common lower cervical and lower lumbar symptoms is but a slice of the McKenzie Method "pie" and does not include appropriate McKenzie Method management of headaches, the extremities, adherent nerve root (epidural fibrosis), nerve root entrapment, and other conditions. Further study is encouraged by means of the texts authored by Robin McKenzie (5—7) and postgraduate study within the McKenzie Institute International (1). We close the chapter with only a brief consideration of the research literature and the reader is directed to the Literature Relevant to the McKenzie Method on the McKenzie Institute International web site (2) to peruse the expansive literature regarding the McKenzie Method.

The Three Syndrome Patterns and Explanations

The McKenzie Method recognizes three clinical patterns (syndromes) of mechanical and symptomatic responses to loading that are amenable to mechanical (movement and positioning)

therapies. The constructs of these three syndromes occur on two levels. The first level is the description of phenomenological patterns of mechanical and symptomatic responses to spinal loading. The second level is the pathoanatomical explanations of those phenomenological patterns. The syndromes are named after the pathoanatomical explanations, but this should not detract from the phenomenological observations on which those explanations are based.

We first consider the phenomenological patterns (the what) of the syndromes, after which we consider the pathoanatomical models proposed to make sense of what occurs (the why). Phenomenology gives equal importance to subjective and objective data and resists temptations to conjecture what the pathoanatomical underpinnings are. A phenomenological accounting for mechanical and symptomatic responses to loading includes a meticulous description of objective phenomena that can be observed and measured by clinicians (ranges of motion, antalgic posturing, etc.) and subjective phenomenon reported by the patient (symptom location, frequency, quality, duration, provocations/ palliations, etc.). Considering phenomenology before pathoanatomy permits a better appreciation of phenomena, permits the reader to posit his or her own pathoanatomical explanations to explain the why of what's going, and enables one to appreciate how McKenzie Method pathoanatomical explanations account for phenomenon.

The three syndrome patterns of mechanical and symptomatic responses to loading for which therapeutic movement and positioning strategies may be used are as follows.

The Postural Syndrome
The Dysfunction Syndrome
The Derangement Syndrome

Although the syndromes are named according to McKenzie Method pathoanatomical explanations, we will, for each syndrome, first consider how the patterns behave and then consider the explanation for those behaviors.

Postural Syndrome

Postural Syndrome: Phenomenological Pattern Examination of the postural syndrome patient reveals full and pain-free range of motion. Symptoms are only elicited with sustained end range loading, a "finding" typically obtained from history versus the examination.

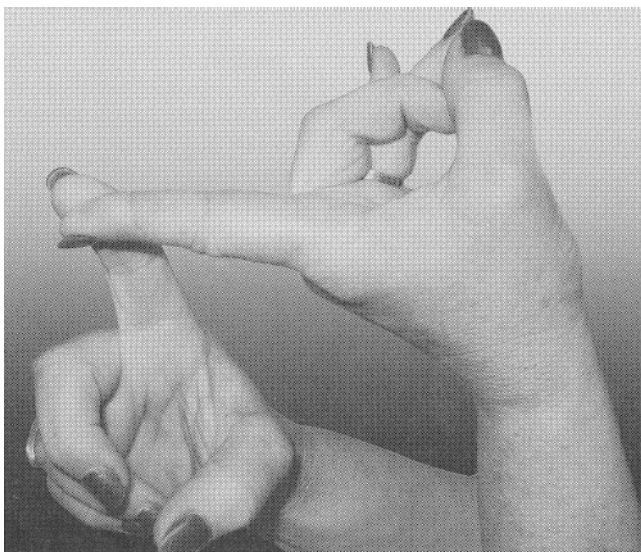


Figure 15.1 The bent finger.

Symptoms are intermittent because they only occur with sustained end range loading, a position typically only assumed intermittently. There are no responses at beginning or middle range. There is no practitioner-observed or patient-perceived range of motion loss or deviation from intended movement plane directions. There is no particular "curative" direction to load in.

It takes time for the end range loading to be provocative, i.e., the end range loading must be pro-longed and static. The symptoms at the mechanically unimpeded end range are in response to an abnormal amount or (more commonly) duration of load at that end range.

Symptoms cease once the end range loading ceases. The reaction occurs only at the end range being loaded. Loading in other movement plane directions has no effect on the reaction at the mechanically unimpeded end range, nor does loading at the mechanically unimpeded end range affect other movement plane directions.

The remedy is to avoid loading at the provocative mechanically unimpeded end range, which eventually results in resolution of its symptomatic effects.

Although the postural syndrome can occur in any movement plane direction, the movement plane direction most commonly culpable for lower cervical and lower lumbar postural syndrome symptoms is flexion.

Postural Syndrome: Pathoanatomical Explanation

Postural syndrome patterns are the result of an excessive amount or duration of end range loading

of normal articular containing or restraining elements. The solution is to avoid the excessive end range loading, i.e., to adopt new postural habits that do not challenge restraining or containing elements. Normal tissue can be symptomatic in response to abnormal forces without there having to be something wrong with the tissue. If the tissue is normal but the load is "wrong" (i.e., abnormal), symptoms may result.

The McKenzie Method uses the "bent finger" as a tool to educate about the postural syndrome (Fig. 15.1).

If a healthy finger is hyperextended far enough, an abnormal amount of force is brought to bear on normal structures, causing discomfort. If the finger were hyperextended to the point of pain and then backed off to the first point of no pain, sustaining that position over time would result in discomfort caused by the abnormal *duration* of force brought to bear on normal structures.

Postural Syndrome: Clinical Intervention

The most common postural syndrome provocateur for the lower cervical and lower lumbar spine is sustained flexion. For many, flexion is the most frequent posture assumed throughout the day as it is promoted with sitting slouched and other activities (Fig. 15.2).

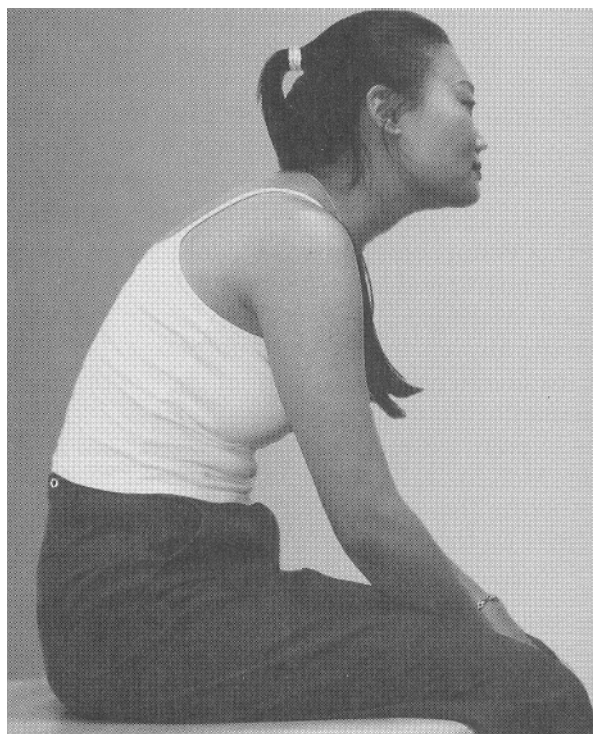


Figure 15.2 Slouched posture when sitting.

Practice-Base Problem

Certain patients report pain absent any range of motion loss or painful examination findings (e.g., mobility and orthopedic tests). What mechanical explanation would account for this if psychological and systemic factors have been excluded?

Sitting is frequently reported by patients to be causing, perpetuating, or aggravating lower cervical and lower lumbar symptoms. Reports of aggravation from sitting should raise suspicions that correction of sitting posture has clinical relevance. As a result of common relaxed slouched sitting, the upper cervical spine is at extension end range, whereas inferior spinal levels (i.e., the rest of the spine) are at as much flexion end range as slouched sitting position permits.

For the lower cervical and lower lumbar spine, aggravation from sustained flexion would cause one to consider the maintenance of lordosis (beginning range extension positioning) as a remedy. Considering the amount of time people spend sitting symptomatically slouched, McKenzie Method postural correction most often concerns correction of the slouched sitting posture. The McKenzie Method uses the slouch-overcorrect-relax strategy to help patients find appropriate lordotic sitting posture. The patient begins from the slouched, provocative sitting posture and then "overcorrects" by simultaneously hyper-extending the lumbar spine and hyper-retracting the head and neck. The patient then "lets go 10%" to find the neutral sitting posture (Fig. 15.3).

Postural syndrome principles are consistent with stabilization philosophies of avoiding excessive end range loading and remaining safe within a neutral zone. The postural syndrome theme may therefore be characterized as end range loading.

Dysfunction Syndrome: Phenomenological Pattern

There is loss of range of motion with a new, premature, limited symptomatic end range being established. Loading at the premature, limited symptomatic end range results in a beneficial reaction at that end range only.

Repetitive loading at that end range results in no significant changes during the examination, other than a temporary increase in discomfort every time loading at the limited mechanically impeded end range occurs. It takes days, weeks, or months of repetitive mechanically impeded end range loading to achieve a beneficial effect. Benefit is not derived from avoiding any movement plane direction in particular.

The dysfunction syndrome pattern is one where-in loading at a mechanically impeded end range results in symptoms at that end range only, with symptoms ceasing once the end range loading ceases. The behavior (symptoms, range of motion) of the mechanically impeded end range does not substantially change in response to repetitive loading during the course of the examination. The reaction occurs at the same end range that is loaded. Movements in other movement plane directions have no effect on the reaction that occurs from loading at the mechanically impeded end range, nor does loading at the mechanically impeded end range affect the behavior of other movement plane directions.

Symptoms occur as soon as the mechanically impeded end range is reached. They are intermittent as they only occur at end range without responses to loading within the beginning or middle range in the same or other movement plane directions. Correction is achieved by loading at the mechanically impeded end range on a frequent basis.

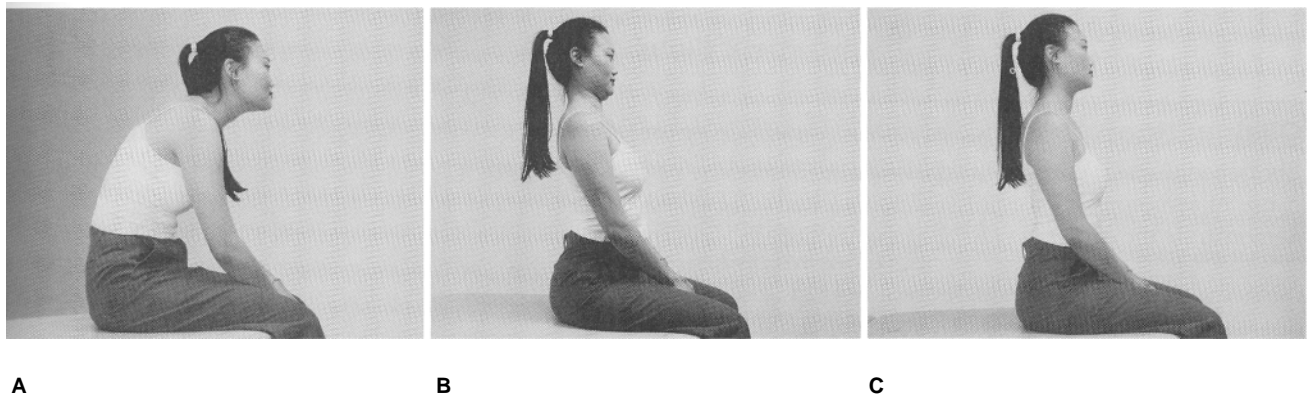


Figure 15.3 Slouched (A), overcorrect (B), let go 10% to neutral lordotic sitting (C).

Dysfunctions are named after the movement plane direction within which the mechanically impeded end range occurs.

Dysfunction Syndrome: Pathoanatomical Explanation

The model for the dysfunction syndrome is that of "short" tissue," i.e., tissue resistant to flexibility demands. It involves the adverse reaction of normal loads on abnormal tissue. The solution is to promote flexibility by means of frequent end range loading to remodel tissue. Improvement is increased flexibility, congruent with strategies to "stretch" or remodel short tissue.

Clinical Pearl

For the postural syndrome, the motto "if it hurts don't do it" applies; the remedy is to avoid loading at the symptomatic mechanically unimpeded end range. The principal is one of end range loathing. For the dysfunction syndrome, the motto "no pain no gain" applies; the remedy is to pursue loading at the symptomatic mechanically impeded end range. The principal is one of end range loading.

Dysfunction Syndrome: Clinical Intervention

Treatment of the dysfunction syndrome uses the remedy of "stretching."

For the McKenzie Method, an appreciation of how short tissue behaves is important to avoid treating short tissue that does not exist and to permit one to have greater success in identifying and treating short tissue when it does exist. Shortened muscular tissue is often the target of treatment when mechanical and symptomatic response patterns do not support the existence of the short tissue claimed. If the muscle is not "short," laboring toward making it long may not be prudent.

Clinical Pearl

Shortened tissue is often erroneously assumed to be the cause of symptoms. A careful evaluation often fails to demonstrate the expected painful loss of motion.

There are various terms used to describe muscle shortening, one of the most extreme being "spasm." Medically defined, spasm is the violent involuntary

sustained contraction of muscle that prohibits joint motion in the direction opposite the afflicted muscle's action. Therefore, if a particular muscle were claimed to be in spasm, that claim would predict a specific painful range of motion loss. Detection of the painful preclusion predicted by a specific spasm claimed would confirm that claim. When range of motion patterns fail to support the existence of the spasms claimed, or are the opposite of what is predicted, the clinical relevance of the claim can no longer be entertained.

Spinal antalgias are good examples of how spasm is inappropriately claimed. Consider the patient who presents with an acute lumbar kyphotic (Fig. 15.4) or the patient who presents with an acute lumbar scoliotic antalgia away from the side of pain (Fig. 15.5). It is not uncommon for these antalgias to be explained away as being caused by paravertebral muscle spasm despite the fact that the explanations predict antalgias opposite of the patient presentations.

Regarding kyphotic antalgia, paravertebral muscle spasm would result in fixed hyperextension of the spine, not the fixed flexion of kyphotic antalgia. Flexion positioning of the spine could not be attributed to spasms of muscles that extend the spine. For the acute left lumbar scoliotic antalgia away from a painful right side, right paravertebral muscle spasm is often blamed for the situation. Spasm of muscles to the right side of the spine would not permit an antalgia to the left but would result in an antalgia to the right. As these two cases demonstrate, discomfort localized to a muscle does not a spasm make.

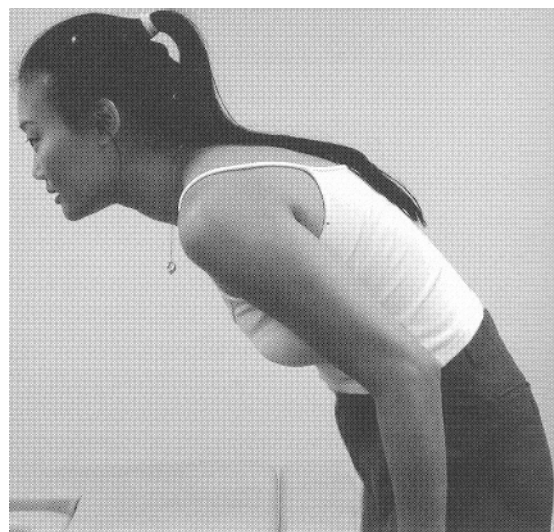


Figure 15.4 Lumbar kyphotic antalgia.

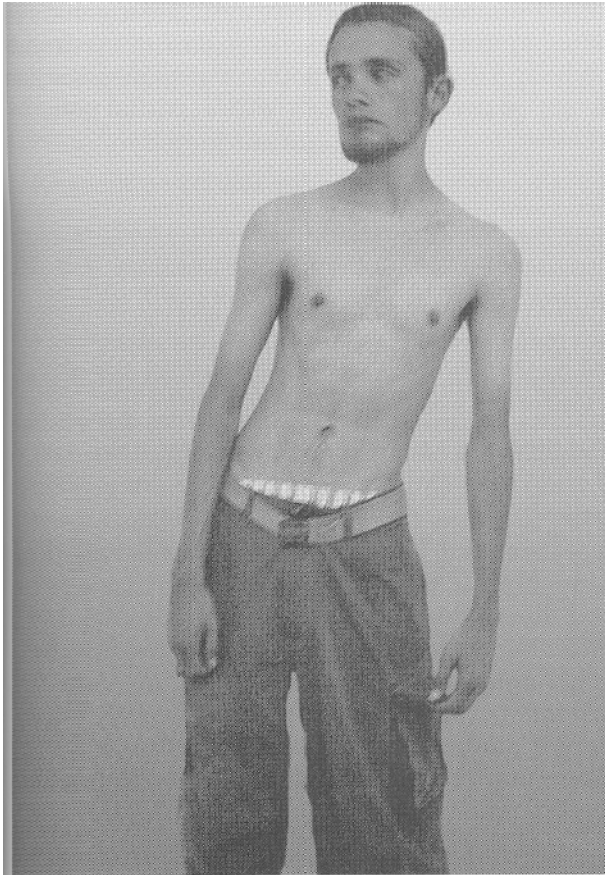


Figure 15.5 Left scoliotic antalgia.

Practice-Based Problem

Is the location of symptoms or palpation findings adequate to determine the presence of shortened muscular tissue? What other information might be important?

Other terms, such as hypertonicity, hyperactivity, contracture, scar, myofascitis, etc., are used to describe muscle shortening. These terms imply a lesser degree of shortening than spasm. With spasm, motion restriction is so great that the spine is "held" in the direction of the muscle action (pull) and neutral positioning cannot be achieved towards the movement plane direction opposite the muscles action (pull). With muscle shortening less severe than spasm, movement may be permitted beyond neutral, into the movement plane direction opposite the shortened muscle's pull, but a painful restriction is still predicted in that opposite direction. In summary, if a shortened muscle is culpable for symptoms, a specific painful range of motion restriction is predicted. Interventions designed to lengthen tissue have better outcomes when the short tissue targeted is really there.

Table 15.1 indicates the painful range of motion losses predicted if particular muscles were "short." One could not conclude that any of the listed muscles were short if the painful motion restrictions predicted did not exist or were the opposite of what is predicted.

When Dysfunction Syndrome patterns are identified, procedures are instituted to improve flexibility, i.e., a stretching routine is instituted. There are numerous ways to "stretch" inflexible spinal joint complexes and most conservative spinal care specialists have expertise regarding stretching instruction. The McKenzie Method uses "stretches" for dysfunctions that are the same end range movements used to "compress" derangements.

Derangement Syndrome

Derangement Syndrome: Phenomenological Pattern

A mechanically unimpeded end range is an end range of motion that is not restricted by mechanical factors.

Table 15.1 Predicted Painful Motion Restrictions Based on Particular Muscles Being Short

Muscle Shortened	Motion Painfully Restricted
Paravertebral	Flexion
Suboccipital	Cervical flexion and lateral flexion to opposite side
Upper trapezius	Cervical flexion, rotation to opposite side
SCM	Cervical extension, rotation to same side
Levator scapulae	Cervical flexion, rotation to opposite side
Rhomboids	Raising of the arm on the same side
QL	Lumbar flexion, lateral flexion to opposite side
Psoas	Thoracolumbar and hip extension
Piriformis	Hip adduction and internal rotation
Gluteus maximus	Hip flexion and internal rotation

Any loss of motion toward that end range would be caused by symptoms or factors other than mechanical factors. A mechanically impeded end range is a premature, early end range, before normal end range, caused by mechanical factors versus symptoms, and may be perceived by the patient to be a mechanical limitation (stiffness or obstruction) with or without pain.

For the Derangement Syndrome, excessive loading toward, or at a mechanically unimpeded end range increases symptoms and promotes a mechanically impeded end range in another direction. The promotion of instability in one direction promotes rigidity in another. Conversely, loading in the direction of rigidity diminishes that rigidity and also diminishes the provocative power of the direction without restriction. The reduction of rigidity in one direction decreases the instability in another. These are some of the most important McKenzie Method observations.

The Derangement Syndrome patterns are complex co-reactions between movement plane directions precipitated by loading at beginning range, middle range, and/or end range (the latter being mechanically impeded or not). Treatment strategies involve avoiding a detrimental mechanically unimpeded end range direction (end range loathing) while pursuing a beneficial mechanically impeded end range (end range "loading"). Symptoms may be intermittent or constant. Changes may be slow and temporary or rapid and maintained, i.e., there is a high degree of reactivity to loading.

Unique to derangements are constant symptoms and adverse mechanical and symptomatic responses during motion occurring in the direction of a detrimental mechanically unimpeded end range. Not only do adverse responses occur at the mechanically unimpeded end range, as with the postural syndrome, but they also occur during motion in the same movement plane direction as the mechanically unimpeded end range. In addition, symptoms may centralize (retreat toward the center of the body) or peripheralize (away from the center towards the periphery, often into the extremities). Centralization is an optimistic prognosticator even when it is associated with increased central symptoms. Peripheralization is a dire prognosticator even when it is associated with a relative diminution of the level of symptom intensity.

There are three derangement syndrome subtypes, each with a unique pattern of potential mechanical and symptomatic responses to loading. The difference between the three subtypes concerns the movement plane directions within which the responses occur. What they have in common is that they all involve at least one potential "direction of detriment" and one "direction of correction," the former being a mechanically unimpeded, the latter a mechanically impeded, end range. The term potential signifies that there are

multiple possible mechanical and symptomatic responses to loading for each derangement syndrome subtype and that all of the potential responses may or may not be present. In other words, partial patterns may exist.

Practice-Based Problem

When a patient presents acute, how does one determine the appropriateness or inappropriateness of movement and positioning therapies, including which end ranges to load at and which end ranges to avoid?

The full palette of potential derangement phenomena is described, below, concerning what can occur when loading in the direction of detriment and in the direction of correction. Two qualifiers must be mentioned. The patterns, as described, represent cases that are amenable to mechanical therapy. Cases not amenable to mechanical therapy would evidence a different pattern. In addition, the patterns, as described, may require a few repetitions of movement to become established and clearly displayed.

Direction of Detriment

- A mechanically unimpeded end range (MUER) movement plane direction
- Adverse mechanical and/or symptomatic responses during motion towards and/or at the MUER
 - o Increased symptoms in the same movement plane direction
 - o Promotion of a mechanically impeded end range in another movement plane direction.

Direction of Correction

- A mechanically impeded end range (MIER) movement plane direction
- No mechanical and/or symptomatic responses during motion
- Beneficial mechanical and symptomatic responses at the MIER only
 - o Diminution of the MIER in the same movement plane direction
 - o Diminution of the provocative power of the MUER direction of detriment

The three derangement subtypes are named according to conclusions about the pathoanatomical mechanism, i.e., according to the direction of movement of intradiscal nuclear Derangement that

best explains the patient's mechanical and symptomatic responses to loading as follows.

1. Posterior derangements
2. Relevant posterolateral derangements
3. Anterior derangements

The examples considered for the three subtypes of derangements will be three spinal antalgias, which serve as excellent examples of derangements because all of the potential derangement features are present in these "extreme" cases.

An acute kyphotic antalgia (Fig. 15.4) would be an extreme example of a posterior derangement. An acute coronal antalgia (lumbar scoliosis or acute cervical torticollis) (Figs. 15.5 and 15.21) would be extreme example of a relevant posterolateral derangement. An acute lordotic antalgia (Fig. 15.24) would be an extreme example of an anterior derangement.

Derangement Syndrome: Pathoanatomical Explanation

Intradiscal nuclear derangement is the model used to explain the dramatic and long-lasting detrimental or beneficial responses to movement and positioning exhibited by lower cervical and lower lumbar derangement syndrome patterns.

The intradiscal nuclear derangement model considers compression rather than stretching forces to explain mechanical and symptomatic responses. Habitual loading in one movement plane direction compresses and displaces intradiscal nuclear material in another, often opposite, movement plane direction. Loading in directions that promote intradiscal derangement of nuclear material may cause adverse mechanical and symptomatic responses in the beginning, middle, and end range of that movement plane direction as the derangement progresses as movement progresses. The end range of the detrimental direction is mechanically unimpeded as intervertebral disc material has been displaced or "pushed out of the way," thus offering less resistance to compression of the intervertebral disc (approximation of vertebral end plates) in that direction.

The accumulation of displaced/deranged intradiscal nuclear material causes a painful obstruction to end range loading (mechanically impeded end range) in the movement plane direction it has deranged into. An example would be flexion causing derangement of intradiscal nuclear material posterior, which then obstructs (get in the way of) extension. The accumulated intradiscal nuclear material offers a

greater resistance to compressive forces (approximation of vertebral end plates). Mechanical and symptomatic responses in the movement plane direction within which the accumulated intradiscal nuclear material has deranged are not realized until the obstruction offered by that material is met, i.e., at the mechanically impeded end range. Mechanical and/or symptomatic responses do not occur *during motion* in the direction of the obstructed end range—the movement that caused the nuclear displacement is being avoided and the accumulated deranged nuclear material has yet to be encountered.

The remedy is to compress the accumulated de-ranged nuclear material (the obstruction to movement), to reduce the derangement, i.e., to send displaced nuclear material back from whence it came, i.e., to a more "central" intervertebral disc location.

As nuclear material migrates through posterior, lateral, or anterior annular tears, symptoms migrate in similar directions. If loading strategies cause nuclear material to migrate to a more central or peripheral location, the topography of symptoms follows suit. Changes in symptom location may be referred to as centralization and peripheralization, respectively. A response to loading involving an increase of central symptoms with diminution of peripheral symptoms (centralization) has a positive prognosis and is appreciated as reflecting the return of deranged intradiscal nuclear material to a more central location. As intradiscal nuclear material returns to a more central location, so do symptoms. As intradiscal nuclear material returns to a central, more confined, more highly pressurized environment, an increase of the intensity of central symptoms (at times, a pressure-type pain) may occur. A response to loading causing an increase of peripheral symptoms (peripheralization) has a negative prognosis, even if symptom intensity lessens, and is appreciated as reflecting intradiscal nuclear material deranging peripheral from its normal, central location.

The positing of the intervertebral disc nuclear derangement model fleshes out phenomenological observations, as follows.

Posterior Derangement Pathoanatomical

Explanation

Posterior Derangement Direction of Detriment: Flexion

- Flexion is the mechanically unimpeded movement plane direction. Loading in flexion displaces intradiscal nuclear material posterior resulting in less intradiscal resistance to flexion. If flexion is not possible, it is because of increased symptoms of, not mechanical resistance

from, deranged intradiscal nuclear material.

- Flexion loading has adverse mechanical and/or symptomatic responses at beginning, middle, and end range (including peripheralization) as nuclear material is progressively deranged posterior.
- Flexion loading promotes an obstruction to extension caused by the accumulation of deranged intradiscal nuclear material in that direction.

Posterior Derangement Direction of Correction: Extension

- Extension is mechanically impeded because of the accumulation of, and resistance to compression from, deranged intradiscal nuclear material.
- Extension loading has no responses during motion because it does not promote the derangement and has yet to meet the obstruction to movement from the derangement.
- Extension loading has beneficial mechanical and/or symptomatic responses at the mechanically impeded end range only (including centralization), the point at which the accumulated intradiscal nuclear material is compressed and returned to a more central location.
- Extension mechanically impeded end range loading results in flexion becoming less provocative. As nuclear derangement is reduced to a more central location, more flexion would be required to achieve the degree of posterior intradiscal nuclear derangement that existed before extension loading.

Clinical Pearl

In a posterior derangement, mechanically restricted extension is increased by flexion loading. Extension loading diminishes the provocative effect of flexion.

Relevant Posterolateral Derangement Pathoanatomical Explanation

A lateral component is "relevant" or not depending on whether loading outside the sagittal plane (i.e., loading laterally) is necessary to reduce the derangement. "Relevance" refers to the relevance of a lateral loading strategy. If there are symptoms that

are "lateral" but the derangement is reduced with loading in the sagittal plane, any lateral component to the intradiscal derangement is not considered relevant (to loading strategies).

Unilateral symptoms, including sciatica, are often adequately addressed with sagittal extension motions without having to resort to lateral techniques. A relevant lateral component is, therefore, not exhibited for these cases despite MRI that may demonstrate lateral intradiscal derangement. If symptoms are central and a lateral loading strategy is required for resolution, a relevant lateral component is considered to exist even though unilateral symptoms did not.

With relevant posterolateral derangement, extension loading is initially detrimental but after a course of coronal loading becomes beneficial. The initial phase of treatment, wherein lateral loading was required, represents a relevant lateral component. After lateral loading is successfully used, the relevant lateral component no longer exists.

A relevant posterolateral derangement may be thought of as a posterior derangement that has progressed to develop a relevant lateral derangement component as well. In the presence of a relevant lateral derangement, extension strategies fail to capture and return the lateral derangement to a more central location and, to the contrary, often promote the lateral component of the derangement. Treatment of the relevant posterolateral derangement is a two-step process. The first step reduces the lateral derangement with coronal (non-sagittal) loading strategies. The second step is to proceed with posterior derangement management, already considered above. The relevant lateral derangement must first be reduced to a more central location by means of lateral techniques, after which extension is transformed from being detrimental to being beneficial by reducing the posterior derangement that remains after the relevant lateral derangement is reduced (eliminated). We will now consider the case of an individual whose spinal symptoms are right-sided and whose mechanical and symptomatic responses are consistent with a relevant right posterolateral derangement.

Relevant Right Posterolateral Derangement Directions of Detriment: Flexion, Left Lateral, Extension

- Flexion and left lateral movements are the mechanically unimpeded movement plane directions. Loading in flexion and left lateral movements derange intradiscal nuclear material posterior and right lateral (i.e., right posterolateral) resulting in less intradiscal resistance to flexion and left lateral movements. If flexion or left lateral

movements are not possible, it is because of increased symptoms of, and not the mechanical resistance from, deranged intradiscal nuclear material.

- Flexion and left lateral loading have adverse mechanical and/or symptomatic responses at beginning, middle, and end range (including peripheralization) as nuclear material is progressively deranged right posterolateral.
- Flexion and left lateral loading promote an obstruction to extension and right lateral movements caused by the accumulation of deranged intradiscal nuclear material in those directions.
- Extension is a mechanically impeded movement plane direction that is initially detrimental to load at end range. Although extension is mechanically impeded because of accumulation of intradiscal nuclear material, intradiscal nuclear material has accumulated both posterior and right lateral. Extension end range loading fails to capture and return the relevant right lateral component to a more central location and promotes right lateral derangement of intradiscal nuclear material. It is the failure of extension to reduce the derangement that causes this type of derangement to be classified as relevant posterolateral.

**Relevant Right Posterolateral Derangement
Direction of Correction: Right Lateral
Loading**

- Right lateral loading is mechanically impeded because of the accumulation of, and resistance to compression from, deranged intradiscal nuclear material.
- Right lateral loading has no responses during motion because it does not promote the derangement and has yet to meet the obstruction to movement from the derangement.
- Right lateral loading has beneficial mechanical and/or symptomatic responses at end range only (including centralization); the point at which the accumulated deranged intradiscal nuclear material is compressed and returned to a more central location.
- Right lateral loading results in flexion, left lateral, and extension loading becoming less provocative as a result of a reduction of right lateral derangement of nuclear material. Because nuclear derangement is reduced to a more central location, a greater degree of

flexion, left lateral, and/or extension loading would be required to achieve the degree of lateral intradiscal nuclear derangement that existed before right lateral loading reduction of derangement.

- After right lateral loading is recovered, extension is no longer detrimental, but is transformed into something beneficial after the relevant lateral component is reduced, i.e., once the "lateral" component is taken out of the posterolateral derangement. Extension loading no longer promotes lateral derangement because there is no lateral derangement to promote. From this point on, the progression is as for posterior derangement, which essentially is what is left without the relevant lateral component. Extension results in further improvement as the remaining posterior component is reduced.

Anterior Derangement Pathoanatomical Explanation

**Anterior Derangement Direction
of Detriment: Extension**

- Extension is the mechanically unimpeded movement plane direction. Loading in extension deranges intradiscal nuclear material anterior resulting in less intradiscal resistance to extension. If extension is not possible it is because of increased symptoms of, and not the mechanical resistance from, deranged intradiscal nuclear material.
- Extension loading has adverse mechanical and/or symptomatic responses at beginning, middle, and end range (including peripheralization) as nuclear material is progressively deranged anterior.
- Extension loading promotes an obstruction to flexion because of the accumulation of deranged intradiscal nuclear material in that direction.

**Anterior Derangement Direction
of Correction: Flexion**

- Flexion is mechanically impeded because of the accumulation of, and resistance to, compression from deranged intradiscal nuclear material.
- Flexion loading has no responses during motion as it does not promote the derangement and has yet to meet the obstruction to movement from the derangement.

- Flexion loading has beneficial mechanical and/or symptomatic responses at the mechanically impeded end range only (including centralization), the point at which the accumulated intradiscal nuclear material is compressed and returned to a more central location.
- Flexion mechanically impeded end range loading results in extension becoming less provocative. As nuclear derangement is reduced to a more central location, more extension would be required to achieve the degree of anterior intradiscal nuclear derangement that existed before flexion loading.

Acute Spinal Antalgia Paradigms of McKenzie Method Derangement Management

With the McKenzie Method, antalgia is typically resolved within a few visits with self-generated movement initiated as the centerpiece of care beginning with the first visit. A prudent progression of forces is used to reverse the antalgia while being mindful of centralization and peripheralization phenomena to judge the appropriateness of the strategy.

Delay of movement therapy for spinal antalgia often results from the misconception that acute spinal antalgia represents the "wisdom" of the body avoiding a position that is deleterious. The situation, so conceived, precludes the exploration of movements to reverse the antalgia. Antalgia is rarely caused by neural or other pernicious pathological processes; standard history and examination procedures rule out these infrequent contributors.

Patients presenting with acute spinal deformities are unable to achieve neutral spinal positioning in the movement plane direction opposite the antalgia. It is as if the precluded movement plane direction has "col-lapsed" into the opposite movement plane direction within which the patient is "trapped." The McKenzie Method management strategy is to first achieve neutral spine positioning and then to "recover" the precluded movement plane direction, guided all the time by centralization and peripheralization phenomena.

The criteria for the preferred loading strategy are not only centralization phenomena but also the degree to which adverse mechanical responses resolve. Although the McKenzie Method is known for being mindful of symptomatic responses, mechanical responses are equally important and may, at times, be the only sign that a positive response to loading has occurred. For some patients, the presenting symptom may be perception of a mechanical restriction to motion, perceived as a stiffness

limitation versus significant pain.

For introductory educational purposes, the McKenzie Method management of spinal antalgias offers excellent examples of derangement management because the derangement subtype is easy to identify as opposed to the significant investigative efforts required when antalgia is absent. Appreciation of the presentation and management of the three acute spinal antalgias informs the process of learning how to detect, evaluate, and manage derangements when there is no antalgia, because most derangement presentations can be construed as partial patterns of the full antalgia patterns.

Kyphotic Antalgia Management—Extension Principle—Posterior Derangement

Lumbar Kyphotic Antalgia Management—Extension Principle—Posterior Derangement

The patient presenting with a lumbar kyphotic antalgia (Fig. 15.6) typically has symptoms that are central or symmetrical and do not radiate beyond the knee, consistent with a central, posterior derangement that does not affect more lateral articular or neurologic structures.

There are detrimental responses within the mechanically unimpeded flexion movement plane direction, both during motion and at end range. For extension there are responses at that mechanically impeded end range only. There are no responses "during motion" for extension because extension motion is not possible (there is no

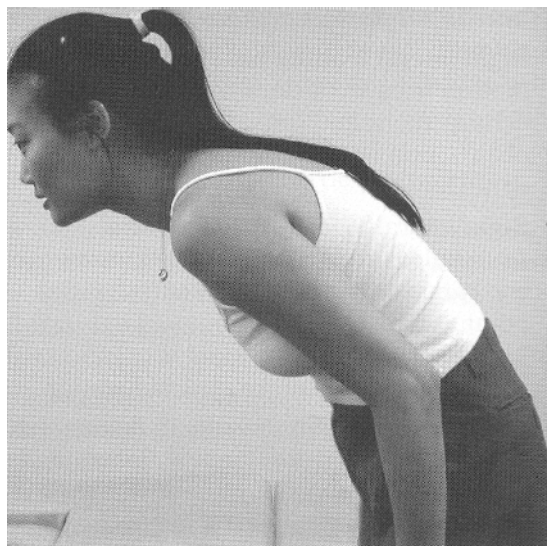


Figure 15.6 Lumbar kyphotic antalgia.

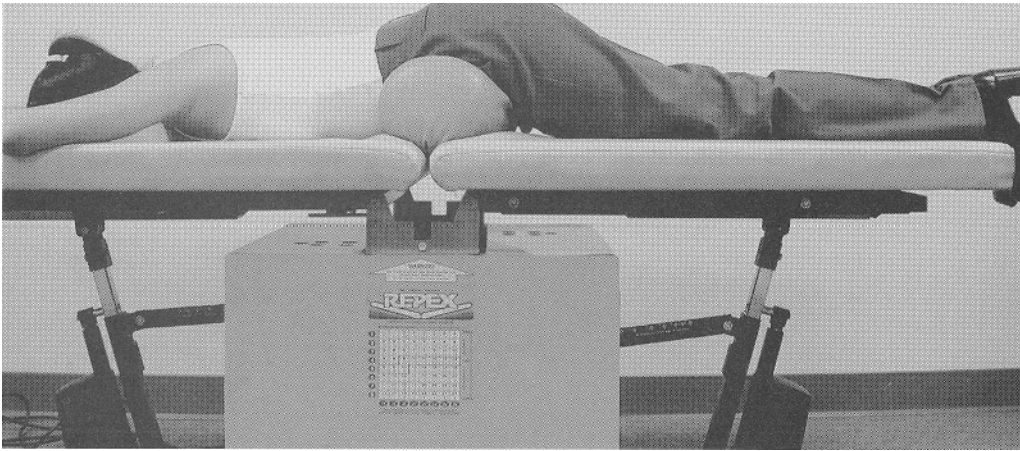


Figure 15.7 Prone patient on plinth with pillow.

extension); the mechanically impeded extension end range is met in the flexed position. As the patient improves, extension movements become possible but still evidence a mechanically impeded end range with responses continuing to occur at the mechanically impeded extension end range only.

When a patient presents with a lumbar kyphotic antalgia, the first step is to achieve neutral (0 degrees of flexion) positioning of the spine, which is difficult to accomplish in the erect standing posture. The patient is placed prone on the plinth with a bolster pillow under the abdomen (Fig. 15.7) to relax in a position accommodating the antalgia.

After some time, the pillow is removed and the patient is flat prone (Fig. 15.8) and may experience centralization discomfort as a result.

After achieving prone 0-degree flexion (neutral positioning), the next step is to recover extension. The patient is asked to rise up on elbows (Fig. 15.9) and to rest in that position for a few moments; again, an increase of centralization discomfort may be experienced.

Next, the patient is asked to perform a prone extension (Fig. 15.10).

From what may be described as a push-up position, the elbows are extended in an attempt to passively extend the trunk over the pelvis. Instruction is given to relax the buttocks because contraction of the gluteus maximus flexes the lumbar spine, a roadblock to extension. For patients having difficulty relaxing the buttocks, it is useful to assume a knocked-kneed, pigeon-toed positioning of the lower extremities to

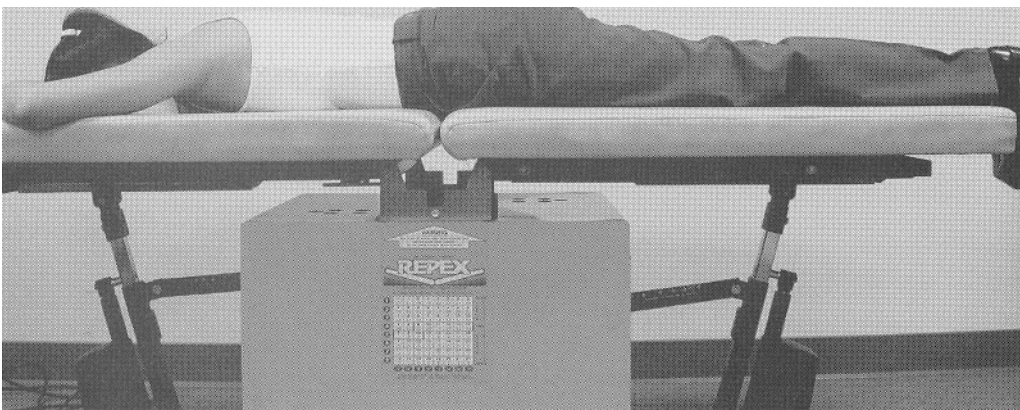


Figure 15.8 Prone patient on plinth.

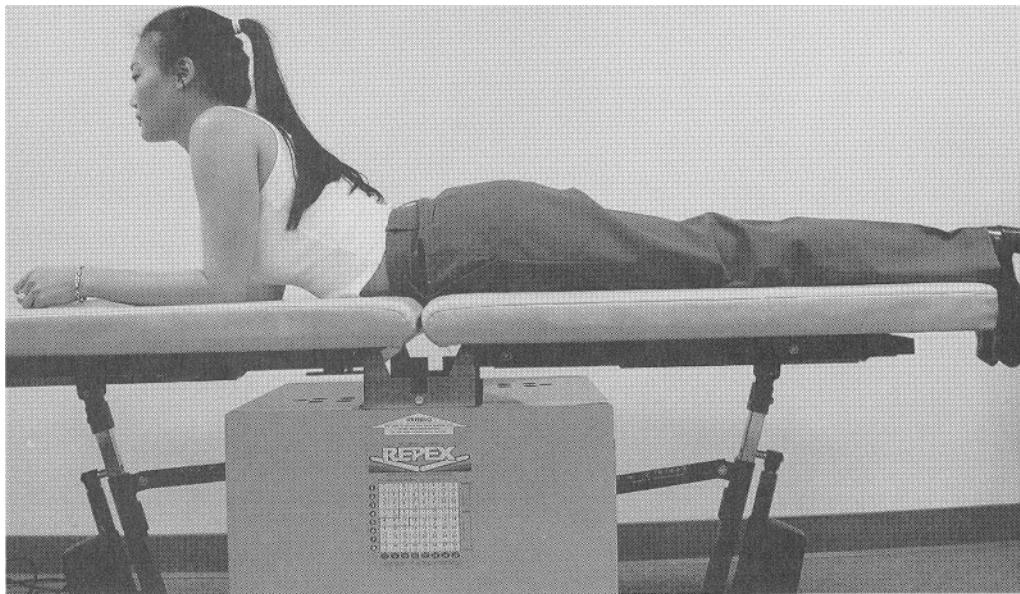


Figure 15.9 Prone on elbows.

stretch-relax the gluteus maximus. The patient is given the verbal cue to let the pelvis "sag" to the table. There is a momentary rest/pause at extension end range and then again at the starting position. The exercise is performed approximately 10 times.

When performing any end range loading exercise or mobilization, patients are asked to report when discomfort is perceived to change in any fashion. The clinician monitors whether these changes occur during

motion or at end range. Although the most important criteria is patient status subsequent to the performance of any exercise, during the exercise there is special interest as to what is occurring *at* the moment of end range loading and whether symptoms centralize or peripheralize *at* the moment of end range loading. Centralization and/or peripheralization reactions *at* the mechanically impeded extension end range herald whether benefit or detriment

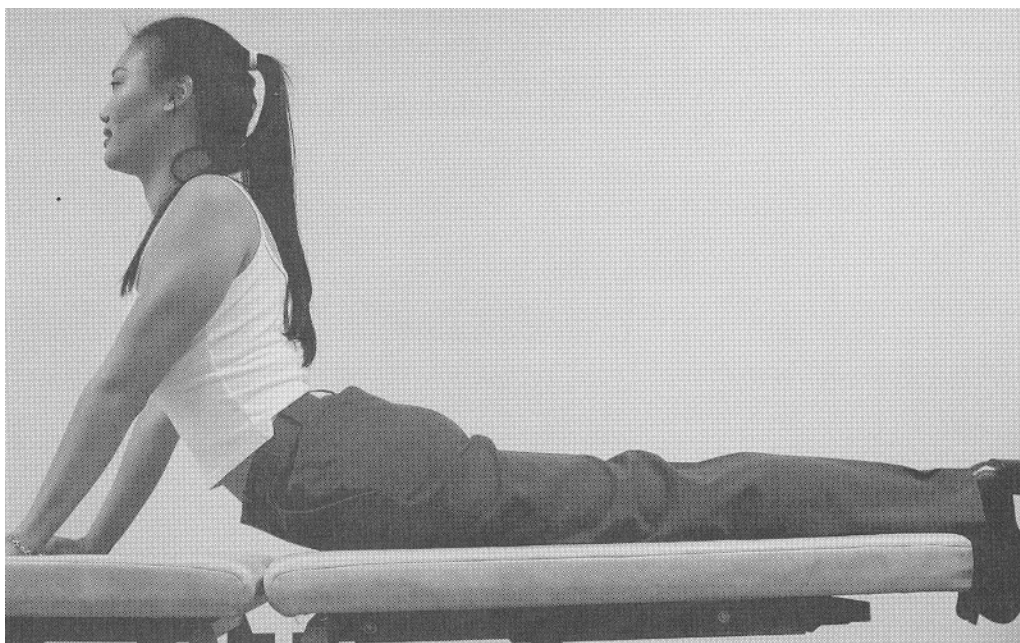


Figure 15.10 Prone extension.

will be experienced after end range loading ceases. It is an optimistic prognosticator if symptoms become more central or diminish at each extension end range loading. If radiation to the extremity occurs every time loading *at* end range is achieved, this would raise concerns that loading at that end range may not be the most prudent strategy.

After the patient is able to achieve extension from the prone position they should, within 1 or 2 days, be able to tolerate and benefit from extension in standing (Fig. 15.11) as an alternative self-treatment, in addition to prone extension.

Flexion postural syndrome principles of avoiding deleterious flexion end range and maintaining lumbar lordosis are used. Self-treatment for the posterior derangement centers on avoiding flexion, maintaining lumbar lordosis while sitting (and making transitions between postures) and periodically pursuing extension end range loading, either prone or standing (the former usually being more effec-



Figure 15.12 Cervical kyphotic antalgia.

tive). Education would be conducted concerning centralization and peripheralization phenomena.

One of the goals of care is the achievement of full pain-free extension, appreciated to represent the reduction of the posterior derangement. Subsequent to this, flexion would be revisited for two reasons. The first would be to confirm that flexion is no longer provokes derangement; the second is to explore whether a flexion dysfunction developed due to formation of scar tissue or avoidance of flexion during the course of care. Flexion would continue to be avoided if it was determined that it still had the power to promote posterior derangement. Flexion would be pursued if the pattern of reaction was consistent with flexion dysfunction. Flexion loading to remodel dysfunction would be followed by extension as a prophylactic measure to ensure that the recent reduction of the posterior derangement stayed that way.

Cervical Acute Kyphotic Antalgia Management—Extension Principle—Posterior Derangement

For cervical kyphotic antalgia (Fig. 15.12), the patient is unloaded in a supine position with additional unloading introduced by means of manual axial traction. Even though this requires "hands-on," patients are soon able to self-treat with techniques that resemble, and can replace, clinician manual methods used to get them "going."

The patient is initially made comfortable in the antalgic position. The supine patient's head rests on a pillow supporting the flexed antalgic position.

To achieve 0 degrees of flexion, manual axial traction is used (Fig. 15.13).

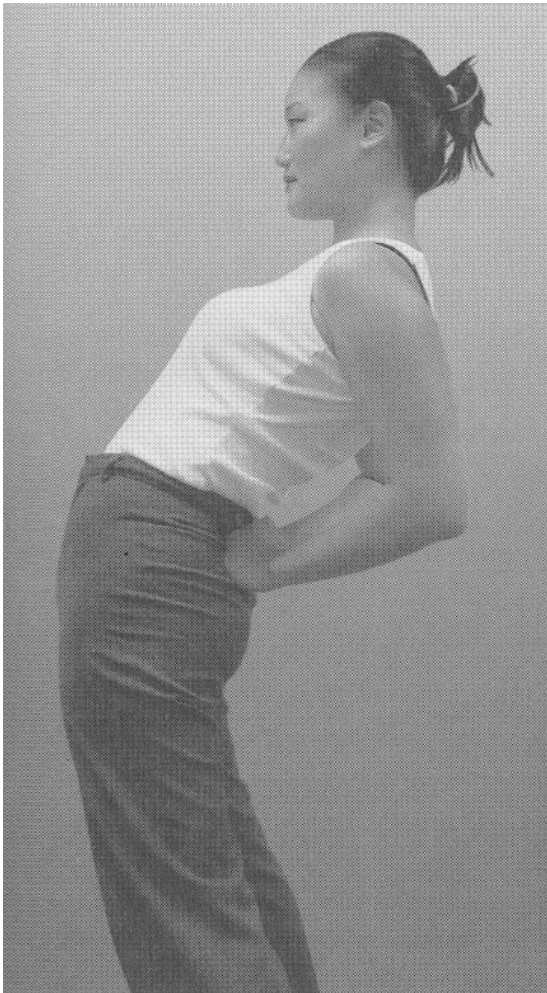


Figure 15.11 Extension in standing.

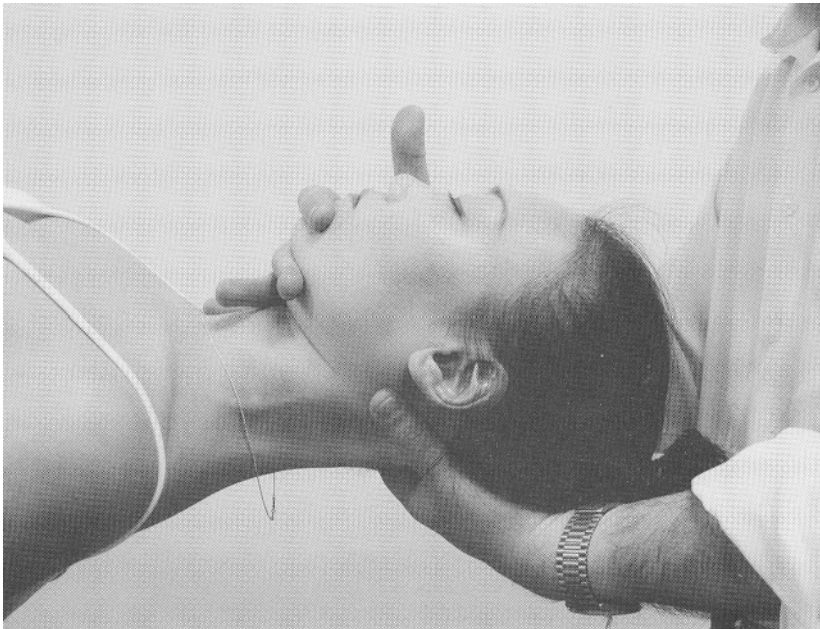


Figure 15.13 Manual supine cervical traction.

The therapist places the index and middle finger of one hand anterior and inferior to the chin, respectively. The thumb and index finger of the other hand abuts the inferior border of the occiput. The patient is asked to occlude (not clench) the teeth to avoid biting the tongue or disturbing the TMJ. Axial traction is then applied along the vector of the flexion antalgia. While maintaining the axial traction, cervical retractions are performed in a slow, gentle, repetitive manner to achieve beginning range lower cervical extension (lordosis) and neutral head and neck positioning (Fig. 15.14).

A momentary rest occurs at end range retraction and at the starting point for each repetition. Appropriateness is monitored by means of centralization and peripheralization.

Next, extension is introduced from the retracted position. As soon as extension is initiated, retraction forces are withdrawn, with axial traction forces maintained throughout (Fig. 15.15).

As always, centralization and peripheralization phenomena judge appropriateness. The head and neck are extended within tolerance. With each repetition further extension is attempted. At extension end range, gentle very small rotations of

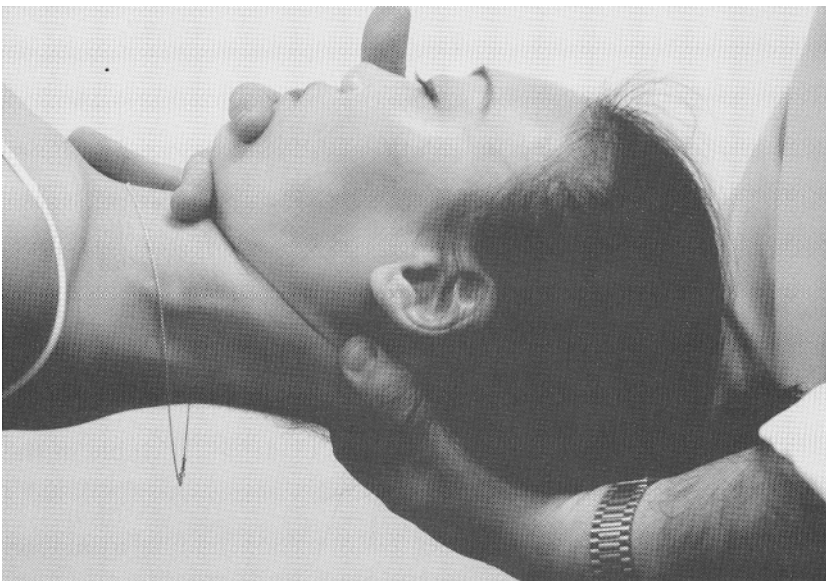


Figure 15.14 Manual supine cervical traction-retraction to introduce lordosis.



Figure 15.15 Manual supine cervical traction-retraction-extension.

the head are performed to facilitate further extension. As always, feedback from the patient is essential to evaluate what is occurring during motion and at end range. Responses at end range are of particular interest.

Patients are shown how to perform self-treatment exercises to the degree they are capable. Options include sitting retractions followed by sitting retraction-extension (Fig. 15.16).

For sitting cervical retractions, instruction to keep the head level to avoid nodding is helpful. Maintenance of lumbar lordosis is essential to achieve maximum cervical retraction or extension end range loading in the sitting position. Sitting extension is

performed from the retracted position to achieve maximum extension end range. Once extension is introduced, the retraction is not maintained (the retraction is "lost"). Gentle mini-rotations are performed at end range to permit further extension.

As with lumbar Kyphotic antalgia, self-treatment involves the flexion postural syndrome treatment principles of avoiding flexion and maintaining lumbar and cervical lordosis (the former required for the latter) while sitting and making transitions between postures. Periodically throughout the day, cervical retraction extensions are performed. Education regarding centralization and peripheralization would be conducted.

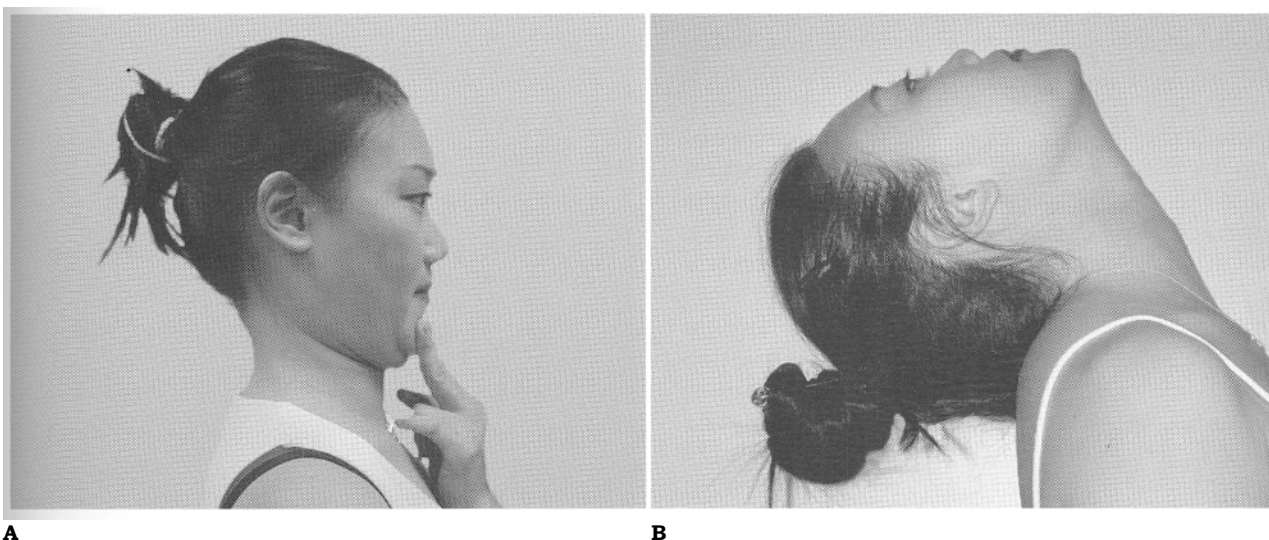


Figure 15.16 Sitting cervical retraction and sitting retraction-extension.

As with the lumbar spine, subsequent to the achievement of full pain-free extension, flexion would be revisited to confirm whether flexion is still provoked derangement or if a flexion dysfunction developed because of avoidance of flexion. Flexion would continue to be avoided if it was determined that it still promoted posterior derangement. Flexion would be pursued if the pattern of reaction was consistent with flexion dysfunction and would be followed by extension as a prophylactic measure to ensure the recent reduction of posterior derangement stayed that way.

Acute Coronal Antalgia Management: Lateral-Then-Extension Principle—Relevant Postero-Lateral Derangement

Unilateral and extremity symptoms are more common with coronal antalgia than with sagittal antalgias.

As with kyphotic lumbar and cervical antalgias, unloading tactics are used to initiate treatment for cervical coronal antalgia (acute torticollis) but may or may not be necessary for lumbar coronal antalgia (acute scoliosis). Acute lumbar scoliosis can often be corrected in the loaded standing position with strategies that may prove more effective than unloaded alternatives.

As with the kyphotic antalgias, the acute coronal antalgias can be visually identified. The coronal antalgias (lumbar scoliosis or cervical torticollis) may be associated with a kyphotic antalgia or not. Whether a kyphotic component is visualized or not, the treatment progression for coronal antalgia involves the two-step progression of recovering motion in the coronal plane opposite the antalgia (the relevant lateral component) followed by recovery of motion in the extension (sagittal) plane. The progression is the lateral-then-extension principle.

With coronal antalgia, if extension end range loading is performed before recovery of the coronal movement in the direction opposite the antalgia (i.e., reduction of the relevant lateral component), the patient may worsen. However, after recovery of motion in the coronal movement plane direction opposite the antalgia, extension end range loading is transformed from detrimental to beneficial. In fact, the tolerance of, and/or benefit from, extension is a sign of progress.

For our examples of lumbar and cervical coronal antalgias, we will consider a patient with right-sided symptoms and a coronal antalgia to the left, interpreted as a right posterolateral derangement. The goal is to first recover movement in the right coronal movement plane direction (i.e., to reduce the right lateral component of the derangement) and then to recover extension (to reduce the posterior derangement that remains).

Lumbar Acute Scoliosis Antalgia Management

When considering acute lumbar scoliosis, two terms are useful, those being lateral shift and side gliding. The term "lateral shift" is equivalent to antalgia and is referenced as right or left depending on the direction of the coronal deviation of the trunk over the pelvis. Someone with a left antalgia has a left lateral shift (Fig. 15.17).

If lateral shift refers to a position in the coronal plane, side gliding is the *movement* that gets you to, or away, from that position. Side gliding is movement of the trunk relative to the pelvis in the coronal plane with the shoulders kept level.

For our patient with a left lateral shift, the first intervention to explore is side gliding against the wall, which permits self-correction in the loaded standing posture without need to visit the plinth (Fig. 15.18).

Our left lateral shift patient is positioned with the left side of the body toward the wall. The medial epicondyle of the left elbow remains in contact with the left rib cage on the axillary line. The patient leans the lateral aspect of the left arm against the wall. The feet are placed together a few feet away from the wall. The patient places the right hand on the superior aspect of

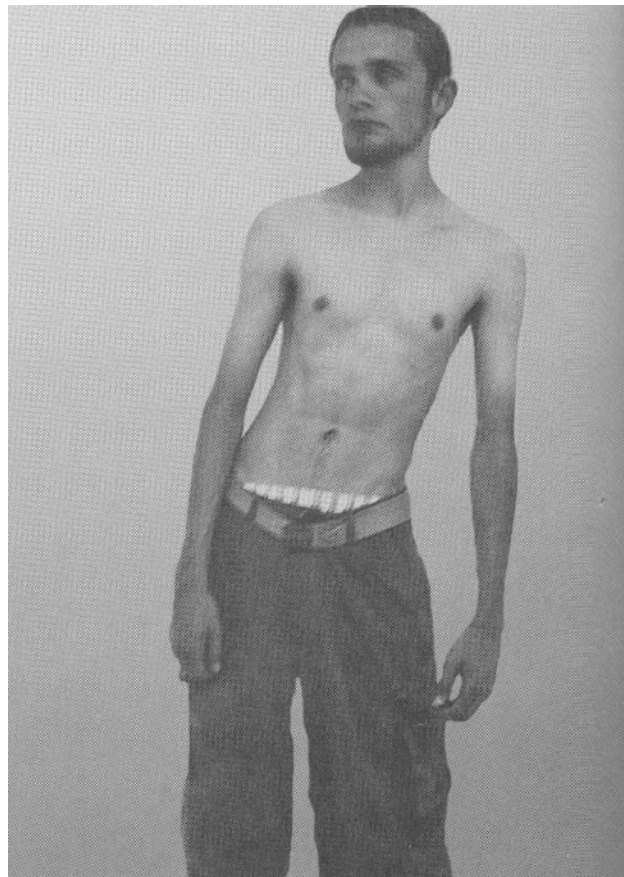


Figure 15.17 Left lumbar lateral shift.

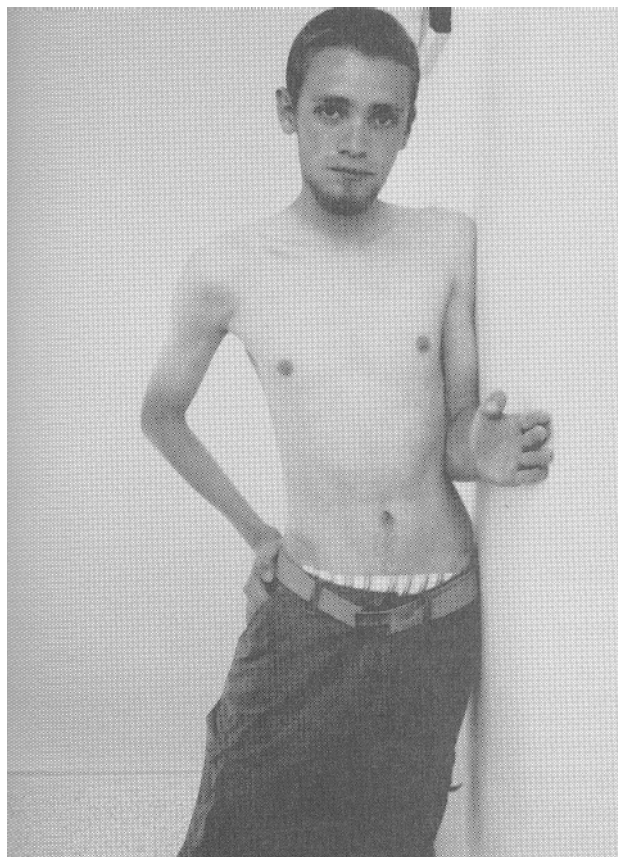


Figure 15.18 Right side-gliding against the wall to correct a left lateral shift.

the lateral right ilium and pushes the pelvis toward the wall until the painful obstruction is met; this end range loading is maintained for a moment. The pelvis is then backed off to the first point of tolerable discomfort; there is a moment of rest and the procedure is repeated. With each repetition, further progression to the wall should be achieved. If the feet are placed a proper distance from the wall, contact between the pelvis/hip and the wall should not occur, even as side gliding improves. The appropriateness of the intervention, as always, is judged by centralization and peripheralization phenomena.

If considerable improvement is noted, extension may be performed at the end range of the coronal movement opposite the antalgia; however some patients do not benefit from extension until some days have passed. Should side gliding not be well-tolerated, the introduction of a slight degree (e.g., 10 degrees) of flexion may transform the maneuver into something of benefit. As the patient progresses, the need to flex should resolve and tolerance and benefit from extension should evolve.

If the patient cannot adequately achieve coronal end range movements with side gliding against the

wall, therapist overpressure may be required. If this does not turn out well, the therapist may have to offer even more assistance by manually inducing side gliding maneuvers absent benefit of the wall. In essence, the therapist becomes a wall with arms (Fig. 15.19).

The patient stands with feet shoulder width apart with the left arm positioned as it would be to lean against the wall. The therapist is on the patient's left-side, oriented in the patient's coronal plane and adopting a three-point stance with the forward foot behind the patient. The angle of the therapist's neck/shoulder girdle contacts the patient's left arm just above the elbow. The therapist reaches around the patient, inter-lacing fingers just below the crest of the right ilium. Therapist mobilizations are then applied by simultaneously pulling the pelvis (with the interlaced hands) and pushing the trunk (with the angle of neck/shoulder girdle against the patient's arm) in the coronal movement plane. Use of a mirror helps ensure that the patient's shoulders remain level so that side gliding correction is used as opposed to lateral flexion. As with the wall side gliding, adverse reactions often indicate the need for a slight degree of flexion. As with wall side gliding, if significant benefit is experienced, extension can be added at the point of coronal end range in the direction opposite the antalgia. To do this, our patient's right hand would be placed on the therapist's right wrist (behind the patient) and used as a fulcrum to lean back on.

Should standing side gliding strategies prove futile, prone extensions from a lateral shift position may be explored. For our patient with a left lateral shift, this

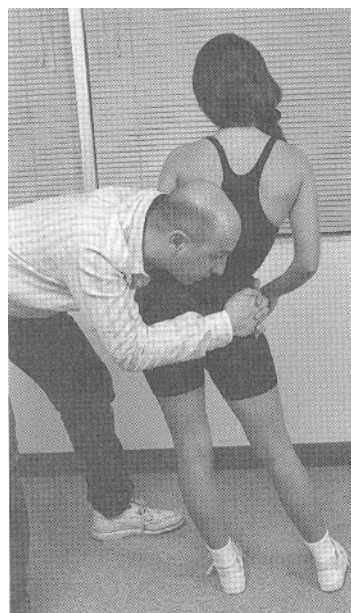


Figure 15.19 Therapist-assisted right side-gliding to correct a left lateral shift.

would be prone extensions from a right lateral shift position (Fig. 15.20).

In the prone position, the pelvis is positioned at coronal end range in the direction opposite the presenting coronal antalgia. Our patient places the pelvis to the left (essentially performing a right lateral shift as the trunk is now to the right of the pelvis) and prone extensions are performed from the right lateral shift position. Benefit is monitored by centralization as well as the ability of the exercise to diminish the antalgia, once performed.

Self-care would include use of flexion postural syndrome principles (avoiding flexion and maintaining lumbar lordosis while sitting and making transitions between postures) with the periodic performance of the preferred coronal end range loading strategy. Although extension end range loading initially fails to benefit, or is of detriment, maintenance of a minimal lordosis (beginning range extension positioning) is usually tolerated and avoids the deleterious effects of flexion. Education regarding centralization and peripheralization would be conducted.

After recovery of movement in the coronal movement plane direction opposite the presenting coronal antalgia, self-treatment continues by using the extension principle for the posterior derangement that remains once the relevant lateral component of the postero-lateral



Figure 15.21 Torticollis: left coronal antalgia demonstrated.

derangement is reduced/eliminated.

Cervical Acute Torticollis Antalgia Management

As with lumbar scoliosis, cervical antalgia in the coronal plane (i.e., torticollis) (Fig. 15.21) may or may not be associated with kyphotic antalgia. As with lumbar scoliosis, whether a kyphotic component is visible or not, after the coronal movement plane direction opposite the antalgia is recovered, the extension principle is explored. As with cervical acute kyphotic antalgia, manual axial traction is required to get things going. Soon thereafter, the responsibility of treatment is transferred to the patient using techniques resembling what the clinician used.

The patient is placed supine with the head comfortably placed on a pillow in a manner that does not challenge the antalgia. The therapist's manual contacts are the same as were used with the cervical kyphotic antalgia. Axial traction is applied, at first in the direction of the antalgia. While maintaining axial traction, a lateral flexion mobilization is conducted in the direction opposite the antalgia until the painful obstruction is met at which point there is a momentary pause (Fig. 15.22). The therapist then backs off to the first point of tolerable discomfort, pauses a moment (traction maintained throughout), and repeats the procedure, gaining lateral flexion in the direction opposite the antalgia with each repetition. If lateral flexion fails, the coupled motion of rotation may be attempted in its place, using the same protocols.

As with the lumbar spine, premature attempts to recover extension may be detrimental. Unlike the lumbar spine, combined lateral and extension movements are not used. As is occasionally the case with the lumbar spine, cervical coronal antalgia more often requires a degree of flexion be maintained when recovering lateral movements. As with the lumbar spine, subsequent to the



Figure 15.20 Prone extension from a right lateral shift position to correct a left lateral shift.



Figure 15.22 Correction of left torticollis.



Figure 15.23 Sitting cervical retraction/lateral flexion to recover coronal motion.

recovery of coronal movement in the direction opposite the antalgia, extension end range loading is transformed from detrimental to beneficial. As always, the appropriateness of any loading strategy is audited by centralization and peripheralization.

Patient self-care includes the employment of flexion postural syndrome principles as well as self-generated lateral flexion mobilizations in the direction opposite the coronal antalgia. Education regarding centralization and peripheralization is provided. At first, lateral flexions may only be possible supine with the head on a pillow. As the patient progresses, the ability to perform and benefit from lateral flexion mobilizations in a seated, retracted head and neck position (which promotes lower cervical lordotic extension) is of benefit (Fig. 15.23).

Subsequent to the achievement of end range in the coronal movement plane direction opposite the coronal antalgia, treatment progress is to the extension principle whether there is a visible acute kyphotic antalgic component or not.

Acute Lordotic Antalgic Management— Flexion Principle—Anterior Derangement

Clinical Pearl

Considering the lower cervical and lower lumbar flexion stressors in everyday life (e.g., prolonged sitting, bending), one would predict that flexion as a treatment of lower cervical and lower lumbar symptoms would be the exception rather than the rule. It has been our experience that conditions requiring flexion are less common than those requiring extension.

Regarding the kyphotic and coronal antalgias, a similar mechanical "deformity" occurs in both the lumbar and cervical areas. Lordotic antalgia differs inasmuch as it occurs for the lumbar spine but not for the cervical spine. Nonetheless, patients presenting with cervical symptoms amenable to flexion end range loading strategies have many of the same mechanical and symptomatic responses to loading as those presenting with an acute lumbar lordotic antalgia except, of course, for the lack of an antalgia that can be visualized.

In addition, the lumbar lordotic antalgia has a unique feature. Whereas most low backs that respond to the extension principle do not present with an acute lumbar kyphosis, most low backs that respond to the flexion principle present with an acute lordotic antalgia (Fig. 15.24).

Manual therapists are usually more adept at promoting flexion end range loading than they are at promoting extension end range loading strategies. Typically these skills have been acquired and used according to the notion that short posterior muscular structures are culpable for symptoms and need to be stretched. The McKenzie Method more often uses flexion loading strategies to compress deranged intradiscal nuclear material that has accumulated within the anterior intervertebral disc space to return that material to a more central location as opposed to promoting the flexibility of posterior extra-articular structures. Figs. 15.25 and 15.26 demonstrate lumbar and cervical flexion strategies.

Self-treatment involves education regarding centralization and peripheralization but there would be

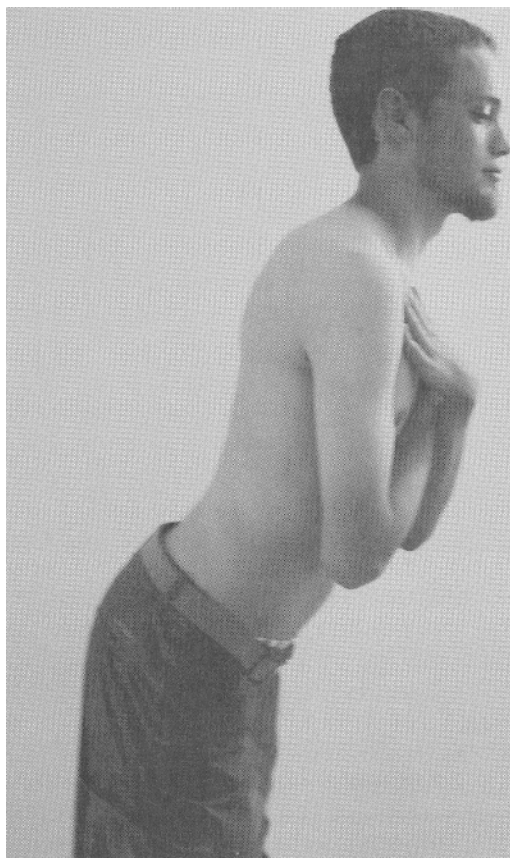


Figure 15.24 Lumbar acute lordosis antalgia: attempting flexion.

no education regarding flexion postural syndrome treatment principles, because flexion is not of detriment. The patient would be dissuaded from any extension end range loading postural habits or any

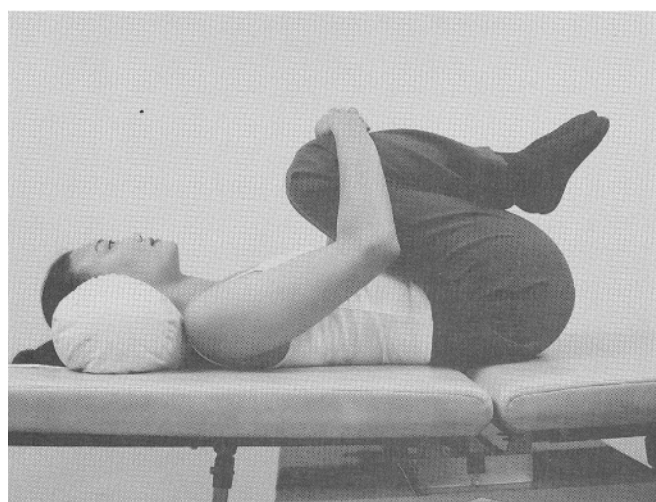
extension end range loading for that matter, including "McKenzie prone extensions!"

■ CONCLUSION

McKenzie Method clinical reasoning would predict that a majority of individuals with spinal symptoms would benefit from minimizing flexion and periodically pursuing extension, considering the amount of time we spend flexed in everyday life. The McKenzie Method predicts that loading in one movement plane direction may be more beneficial than loading in other movement plane directions whether symptoms are acute or chronic. These predictions have been verified within the recent peer-reviewed evidence-based literature.

Snook (8) demonstrated how controlling lumbar flexion in the early morning serves as a form of self-care for reducing pain and costs associated with chronic, non-specific low back pain. The McKenzie Method predicts that avoiding flexion would minimize low back pain for most patients. Early morning flexion is perceived to be particularly provocative because of imbibition of fluid by intradiscal nuclear material over a night of unloading. Theoretically, if the patient has posterior derangement of nuclear material, the imbibition of fluid makes intradiscal nuclear pressures and the risk of debilitating derangements even greater.

Larsen, Weidick and Leboeuf-Yde (3) demonstrated it may be possible to reduce the prevalence of back problems and use of health care services during military service, at a low cost, using lumbar prone extensions with a back/ergonomic school including McKenzie Method disc theories. Military recruits



A



B

Figure 15.25 Promotion of lumbar flexion.

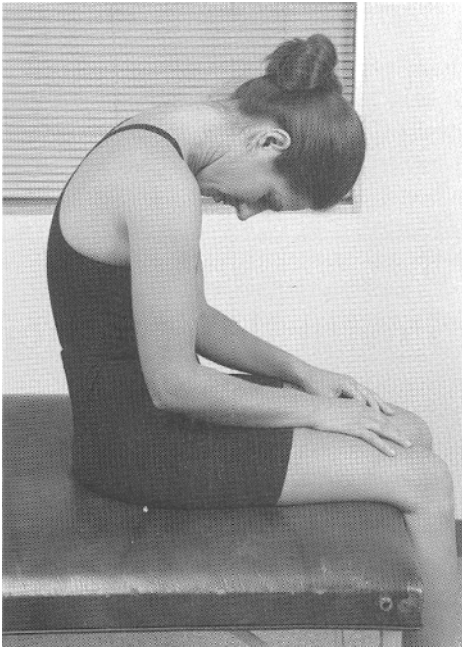


Figure 15.26 Promotion of cervical flexion.

were taught McKenzie Method extension principles (including lumbar lordotic body mechanics and prone extensions were performed periodically throughout the day) resulting in the favorable outcomes noted.

Long, Donelson, and Fung (4) showed that a McKenzie assessment could identify a large subgroup of acute, subacute, and chronic low back patients with a direction of preference ("an immediate, lasting improvement in pain from performing either repeated lumbar flexion, extension or side glides/rotation tests"). Regardless of the direction of preference, "the response to contrasting exercise prescriptions was significantly different." Exercises matching the patient's direction of preference significantly and rapidly decreased pain and medication use and improved disability, degree of recovery, depression, and work interference outcomes. Of the original 312 subjects who underwent assessment, 53.5% demonstrated a directional preference for pure sagittal extension, the remainder required prone extensions from a lateral shift position or movements in other planes. The majority of subjects, therefore, required an extension component to their preferred loading strategy.

Audit Process

Self-Check of the Chapter's Learning Objectives

- What are the three syndromes as defined by the McKenzie Method?
- What are the responses during motion and at end range for each syndrome?
- What is the role of pursuing or avoiding end range loading for each syndrome?
- According to the McKenzie Method, what are the possible reasons a patient might experience increased discomfort with sitting versus standing and vice versa? Consider each syndrome and subtypes to account for the phenomena.

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