Table 2-2

Summary of Neurodynamic Tests for the Lower Limb and Trunk With Associated Maneuvers for Structural Differentiation[1,11,12,16,34,47,91-93]

TEST	STANDARD SEQUENCE*	TYPICAL STRUCTURAL DIFFERENTIATION MANEUVERS
PNF†	• Upper cervical flexion ("head on neck" flexion) • Lower cervical flexion ("neck on shoulder" flexion)	• None‡
SLR	• Pre-position with knee extended • Hip flexion	• Ankle DF • Hip ADD or ABD • Hip IR or ER • Neck flexion§
Slump	• Trunk flexion • Neck flexion • Ankle DF • Knee extension	• Release neck flexion
PKB	• Knee flexion	• None‖
Side-lying slump ("femoral slump")	• Pre-position in side lying with trunk and neck flexed; tested limb in 90-degree knee flexion • Hip extension • Knee flexion	• Release neck flexion

* Although standard sequences have been recommended to increase test reliability, clinicians are encouraged to adjust the order of movement to match an individual patient's presentation.

† May be performed in supine or sitting.[16,34]

‡ Focus on reproduction of symptoms in the lumbar region.[16,34]

§ Data suggest that neck flexion rarely changes SLR response (< 20%).[16,47]

‖ Focus on reproduction of symptoms consistent with nerve sensitivity.[93]

ABD = abduction; ADD = adduction; DF = dorsiflexion; ER = external rotation; EV = eversion; INV = inversion; IR = internal rotation; PKB = prone knee bend; PF = plantarflexion; PNF = passive neck flexion.

These biomechanical data suggest that neurodynamic tests are plausible tests for detecting nerve-related disorders. Strain, tension, and pressure from neurodynamic test movements will likely provoke mechanically sensitive neural tissues in patients who have nerve-related disorders.[94-99] Additionally, proximal or distal joint movements applied at the end of a neurodynamic test (structural differentiation) can likely help determine whether a test response is related to nerve mechanosensitivity because their biomechanical effects spread along the entire nerve.

Experimental pain studies also support the plausibility of neurodynamic tests. Experimental pain induced by injecting hypertonic saline into the thenar or calf muscles is not changed by applying structural differentiation maneuvers associated with the $ULNT1_{MEDIAN}$ or the SLR and slump tests, respectively.[100,101] Similarly, experimental pain induced by injecting hypertonic saline into the medial infrapatellar fat pad is not changed by neck movements performed at the end of the side-lying slump test (femoral slump test).[102] These data suggest that neurodynamic tests can potentially distinguish pain related to irritation of nonneural tissues from pain related to irritation of neural tissues.[31] Even though biomechanical and experimental pain data support using neurodynamic tests to detect nerve-related problems, plausibility is the lowest level of test validity.[28]

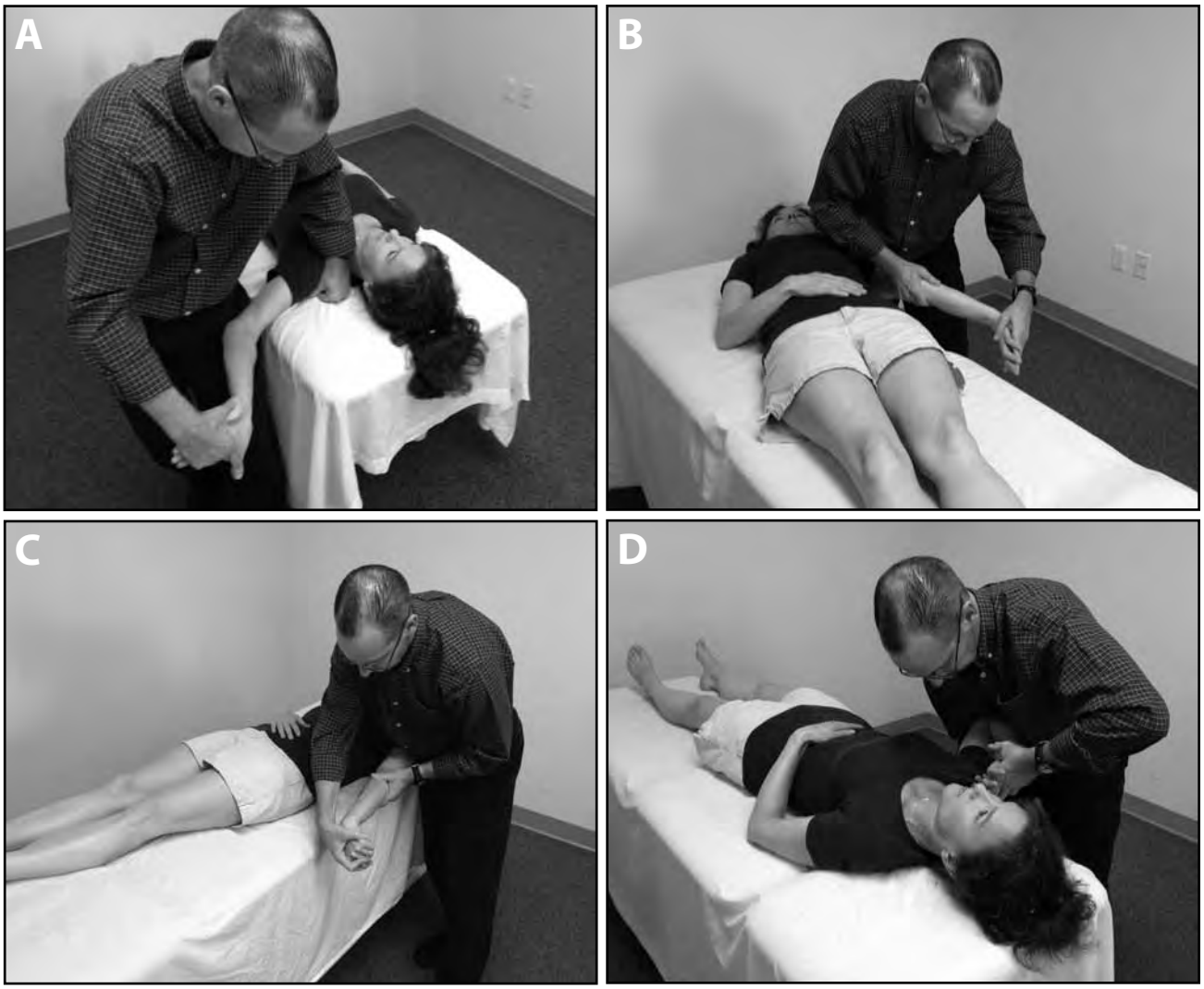


Figure 2-1. End position for each ULNT. (A) $ULNT1_{MEDIAN}$. (B) $ULNT2_{MEDIAN}$. (C) $ULNT_{RADIAL}$. (D) $ULNT_{ULNAR}$.

Criteria for a Positive Neurodynamic Test

Criteria for a positive neurodynamic test need to discriminate patients who have nerve-related disorders from asymptomatic individuals, and patients who have nerve-related disorders from patients who have competing diagnoses (concurrent validity).[30] Clinicians have been encouraged to assess sensory responses, resistance to movement, and range of motion (ROM) during a neurodynamic test to judge whether a patient has signs of increased nerve mechanosensitivity.[1,10] The ability of these components of a neurodynamic test response to discriminate patients who have nerve-related disorders from asymptomatic individuals forms the rationale behind proposed criteria for a positive neurodynamic test.[31] Concurrent validity is addressed later in this chapter.

Most asymptomatic individuals (greater than or equal to 80%) report sensory responses at the end of a neurodynamic test that change with structural differentiation.[19,31,91,103-108] Common descriptors include stretch, ache, pain, burning, and tingling.[19,31,91,103-109] Asymptomatic individuals, therefore, appear to have a certain level of nerve mechanosensitivity. The range of sensory responses reported by asymptomatic individuals makes it important to specify which sensory responses qualify as a positive neurodynamic test in symptomatic populations. A neurodynamic test is most likely identifying a patient with increased nerve mechanosensitivity when the test reproduces at least part of the patient's symptoms and the symptoms change with structural differentiation.[31]

Resistance to movement and ROM are not likely to discriminate patients who have nerve-related disorders from asymptomatic individuals. Different examiners cannot reliably identify the onset of resistance to elbow extension during $ULNT1_{MEDIAN}$.[110,111] The SDD between examiners at a 95% confidence level (SDD_{95})[112] for measuring the onset of resistance to elbow extension is 28 degrees.[31] Studies quantifying the onset of resistance during the SLR and slump tests have only reported intra-examiner reliability.[113,114] Large measurement error between examiners suggests that onset of resistance is not likely to be sensitive enough to discriminate

Figure 2-2. End position for each neurodynamic test for the lower limbs and trunk. (A) PNF. (B) Slump. (C) SLR. (D) PKB. (E) Side-lying slump ("femoral slump").

patients who have nerve-related disorders from asymptomatic individuals. Therefore, resistance to movement is unlikely to be useful for defining a positive neurodynamic test.[31]

Neurodynamic test ROM is most commonly measured at pain onset or pain tolerance during the last component of the test.[31] Examples include elbow extension during $ULNT1_{MEDIAN}$ or elbow flexion for $ULNT_{ULNAR}$, shoulder abduction during $ULNT_{RADIAL}$, hip flexion during SLR, and knee extension during the slump test. As with resistance to movement, there are relatively large errors between examiners for measuring neurodynamic test ROM at pain onset. SDDs (SDD_{95}) between examiners for measuring ROM at pain onset are approximately 15 to 20 degrees for the $ULNT1_{MEDIAN}$,[111,115,116] $ULNT_{ULNAR}$,[117] SLR,[118-120] and slump[120] tests (calculated from reported data).

Table 2-3

Inter-Examiner Reliability for Identifying a Positive Neurodynamic Test*

TEST	KAPPA VALUE	95% CONFIDENCE INTERVAL
ULNTs[130]†	0.45	0.27, 0.63
SLR[120,131-133]	≥ 0.49	0.14, 1.00
Slump[120,134]	≥ 0.71	0.33, 0.97
Side-lying slump[92] (femoral slump)	0.71	0.33, 1.00

* Positive test defined as at least partial reproduction of the patient's symptoms and changing these symptoms with structural differentiation.

† Collective estimate for the median, radial, and ulnar nerve neurodynamic tests.

Even if reliability issues could be corrected, it is still unlikely that neurodynamic test ROM can discriminate patients who have nerve-related disorders from asymptomatic individuals. Neurodynamic test ROM is highly variable in asymptomatic and symptomatic populations.[31,104,107,109,114,117,118,120-125] There is also a lot of overlap in neurodynamic test ROM between asymptomatic and symptomatic individuals, and between the involved and uninvolved limbs of symptomatic individuals.[31,120,123-125] Because of this variability and overlap, it is unlikely that an absolute ROM cut-off would be useful for defining a positive neurodynamic test in an individual patient.

Neurodynamic test ROM can also be quantified as the deficit in ROM in the involved limb relative to the uninvolved limb (limb asymmetry). Asymptomatic individuals typically have 5 to 10 degree differences in ROM between limbs for $ULNT1_{MEDIAN}$,[126-129] $ULNT_{ULNAR}$,[117] and SLR[122] tests. It is still unclear if a certain amount of limb asymmetry in neurodynamic test ROM can discriminate patients who have nerve-related disorders from asymptomatic individuals. Therefore, similar to absolute ROM, it seems unlikely that limb asymmetry in ROM can help define a positive neurodynamic test.

Current evidence does not support using resistance to movement or ROM to define a positive neurodynamic test because of the previously described problems with measurement error and lack of discriminatory cut-off values. At this point, the recommended criteria for a positive neurodynamic test include at least partial reproduction of the patient's symptoms *and* a change in these symptoms with structural differentiation.[31] Reproducing the patient's symptoms helps distinguish the patient's response from sensory responses to neurodynamic tests that are typical for asymptomatic individuals. Changing the patient's symptoms with structural differentiation makes it more likely that these symptoms are at least partly related to increased nerve mechanosensitivity.

Reliability of a Positive Neurodynamic Test

Clinicians can make reliable decisions when using at least partial reproduction of the patient's symptoms and changing these symptoms with structural differentiation to define a positive neurodynamic test. Kappa values for the ULNTs,[130] SLR,[120,131-133] slump,[120,134] and side-lying slump[92] reflect adequate inter-examiner reliability for making this "yes" or "no" decision about a positive test (Table 2-3). Although kappa values between 0.41 and 0.60 suggest moderate reliability,[135] clinical tests with moderate reliability can still have enough concurrent validity to help make a diagnosis.[29,136]

Concurrent Validity of Neurodynamic Tests

Concurrent validity studies use results from a reference standard test to quantify the diagnostic performance of the clinical test of interest.[29,30] Radicular pain and carpal tunnel syndrome are the nerve-related disorders that have been addressed in concurrent validity studies on neurodynamic tests. Reference standard tests used to establish the presence of these nerve-related disorders include a grading system for diagnosing neuropathic pain,[137] electrodiagnosis (eg, nerve conduction studies, needle electromyography), and imaging (eg, magnetic resonance imaging [MRI], computed tomography scans). Strengths and limitations of these reference standards affect the conclusions drawn from these concurrent validity studies. It is also important to consider the criteria used to define a positive neurodynamic test.[138] This section focuses on studies where criteria for a positive neurodynamic test made it likely that symptoms were at least partly related to increased nerve mechanosensitivity.

Table 2-4

Diagnostic Accuracy of the Straight Leg Raise and Slump Tests for Lumbar Radicular Pain*

TEST	REFERENCE STANDARD	SENSITIVITY (95% CI)	SPECIFICITY (95% CI)	POSITIVE LR (95% CI)	NEGATIVE LR (95% CI)
SLR[140]†‡	Imaging or EDX	0.19 to 0.97	0.10 to 0.89	1.1 to 4.7	0.27 to 0.96
SLR[142]	Imaging§	0.59 (0.41, 0.75)	0.53 (0.41, 0.64)	1.26 (0.84, 1.87)	0.77 (0.47, 1.28)
SLR[142]	Imaging‖	0.93 (0.66, 1.00)	0.57 (0.45, 0.67)	2.1 (1.6, 2.8)	0.13 (0.02, 0.84)
SLR[142]	Imaging	0.32 (0.17, 0.52)	0.43 (0.33, 0.55)	0.56 (0.31, 1.03)	1.55 (1.08, 2.29)
SLR[143]†	EDX	0.63 (0.58, 0.69)	0.46 (0.39, 0.53)	1.2 (1.0, 1.5)	0.80 (0.64, 0.98)
Slump[139]	Imaging	0.84 (0.74, 0.90)	0.83 (0.73, 0.90)	4.9 (2.7, 9.0)	0.19 (0.11, 0.36)
Slump[142]	Imaging§	0.78 (0.59, 0.89)	0.36 (0.26, 0.48)	1.2 (0.93, 1.6)	0.61 (0.29, 1.3)
Slump[142]	Imaging‖	1.00 (0.77, 1.00)	0.38 (0.27, 0.49)	1.6 (1.4, 1.9)	0 (0.0, 0.0)
Slump[142]	Imaging¶	0.48 (0.29, 0.69)	0.26 (0.16, 0.37)	0.7 (0.42, 0.99)	2.0 (1.2, 3.5)
Slump[141]	Grading system#	0.91 (0.62, 0.98)	0.70 (0.40, 0.89)	3.0 (1.2, 8.0)	0.13 (0.02, 0.88)
Slump[141]**	Grading system#	0.55 (0.28, 0.79)	1.00 (0.72, 1.00)	11.9 (0.76, 188)	0.48 (0.26, 0.90)

* Unless noted otherwise, a positive test is defined as at least partial reproduction of the patient's symptoms and changing these symptoms with structural differentiation.

† Positive SLR was reproduction of patient's symptoms below the knee.

‡ Range of values reported from this systematic review.

§ MRI confirmed disk extrusion.

‖ MRI confirmed "high-grade" subarticular nerve root compression (obliteration of periradicular cerebrospinal fluid and fat).

¶ MRI confirmed high-grade foraminal nerve root compression.

Location and history of symptoms consistent with lumbar radicular pain *and* sensory signs present in areas consistent with lumbar radicular pain. Imaging findings could contribute to the diagnosis of lumbar radicular pain but were not required.[137,141]

** Positive slump test involved reproduction of patient's symptoms below the knee and changing these symptoms with structural differentiation.

CI = confidence interval; EDX = electrodiagnosis; LR = likelihood ratio.

Lumbar Radicular Pain

It appears that the slump test is more useful than SLR for diagnosing lumbar radicular pain, even when a positive SLR focuses on reproduction of symptoms below the knee (Table 2-4).[139-143] SLR results do not make clinically important changes in the odds of a patient having lumbar radicular pain because +LRs are consistently below 5.0 and -LRs are consistently above 0.2.[140,142-144] In contrast, both positive and negative slump test results are more commonly associated with clinically important changes in the odds of having this nerve-related disorder.[139,141,142,144] A positive slump test is particularly useful if it reproduces the patient's symptoms below the knee.[141] It appears, however, that diagnostic performance may vary according to the specific imaging finding used as the reference standard for radicular pain (eg, disk extrusion, subarticular nerve root compression, foraminal nerve root compression; see Table 2-4).[142]

One exception to the poor diagnostic performance of the SLR for detecting lumbar radicular pain is the crossed SLR. When SLR of the uninvolved limb reproduces symptoms in the involved limb (a positive crossed SLR), the patient is much more likely to have lumbar radicular pain related to lumbar disk herniation.[145] However, a positive crossed SLR is a relatively rare clinical finding.

Although SLR of the involved limb does not perform well as an isolated test, it may help detect lumbar radicular pain when combined with other clinical information. Two diagnostic decision tools suggest that provocation of a patient's typical leg symptoms during the SLR can help detect lumbar radicular pain when combined with other clinical information such as the distribution of the patient's symptoms and positive findings on a clinical neurological examination (ie, myotomes, reflexes, dermatomes).[146,147] However, the performance of these diagnostic decision tools needs to be confirmed in separate samples of patients before they can be recommended for use in clinical practice.[148]

Table 2-5

Diagnostic Accuracy of the Prone Knee Bend and Side-Lying Slump Tests for Lumbar Radicular Pain Affecting the L2 Through L4 Nerve Roots*

TEST	REFERENCE STANDARD	SENSITIVITY (95% CI)	SPECIFICITY (95% CI)	POSITIVE LR (95% CI)	NEGATIVE LR (95% CI)
PKB[93]†	Imaging	0.50 (0.31, 0.69)	1.00 (0.88, 1.00)	∞‡	0.50 (0.34, 0.75)
Side-lying slump[92]§ (femoral slump)	Imaging	1.00 (0.40, 1.00)	0.83 (0.52, 0.98)	6.0 (1.6, 19.4)	0.0 (0.0, 0.6)
Side-lying slump[142] (femoral slump)	Imaging‖	0.43 (0.16, 0.75)	0.64 (0.36, 0.85)	1.2 (0.37, 3.8)	0.90 (0.41, 2.0)
Side-lying slump[142] (femoral slump)	Imaging¶	1.0 (0.21, 1.00)	0.65 (0.41, 0.83)	2.8 (1.5, 5.4)	0.0 (0.0, 0.0)
Side-lying slump[142] (femoral slump)	Imaging#	0.17 (0.03, 0.56)	0.50 (0.25, 0.75)	0.33 (0.05, 2.2)	1.67 (0.85, 3.3)

* Unless noted otherwise, a positive test is defined as at least partial reproduction of the patient's symptoms and changing these symptoms with structural differentiation.

† Reproduction of patient's lower limb symptoms only because no structural differentiation for PKB.

‡ Not able to calculate +LR value because specificity was 100%.

§ Patients in sample with upper/mid lumbar nerve root involvement only had problems at L4; none had L2 or L3 nerve root involvement.

‖ MRI confirmed disk extrusion.

¶ MRI confirmed high-grade subarticular nerve root compression (obliteration of periradicular cerebrospinal fluid and fat).

MRI confirmed high-grade foraminal nerve root compression.

CI = confidence interval; LR = likelihood ratio.

The PKB and side-lying slump neurodynamic tests may help identify radicular pain affecting the L2 through L4 nerve roots (Table 2-5).[92,93,142] Reproducing low back-related leg pain during a PKB makes it more likely that upper/mid lumbar nerve root involvement is contributing to the patient's pain experience, but a negative PKB does not significantly reduce the odds of upper/mid lumbar nerve root involvement.[93] Both positive and negative findings from the side-lying slump test make clinically important changes in a patient's odds of having L2 through L4 nerve root involvement.[92,142] However, similar to the slump test, diagnostic performance of the side-lying slump test appears to vary depending on the specific imaging finding used as the reference standard for radicular pain at these lumbar nerve root levels (see Table 2-5).[142]

Additional research with larger samples is needed so that estimates of diagnostic performance of the PKB, slump, and side-lying slump tests can be more precise.[92,93,141,142] Future research on the diagnostic performance of the SLR should consistently incorporate structural differentiation into the definition of a positive test.[140]

Cervical Radicular Pain

The median and ulnar nerve neurodynamic tests can help diagnose cervical radicular pain (Table 2-6).[149,150] A positive ulnar nerve test is associated with a clinically important increase in the patient's odds of having cervical radicular pain. A negative median nerve test ($ULNT1_{MEDIAN}$) significantly decreases the patient's odds of having this condition. It is important to note that shoulder girdle depression and shoulder abduction of at least 100 degrees were the first 2 movements for both the median and ulnar nerve tests in this study.[149] These 2 movements apply tensile forces throughout the brachial plexus,[17,65] so it makes sense biomechanically that the median and ulnar nerve tests help diagnose cervical radicular pain when performed in this manner. Confidence in these findings will increase if they are replicated in a separate sample of patients.

Other published data suggest that $ULNT1_{MEDIAN}$ can help diagnose cervical radicular pain.[151] However, a positive test did not require both reproduction of symptoms and a change in symptoms with structural differentiation. It is

Table 2-6

Diagnostic Accuracy of Neurodynamic Tests for Cervical Radicular Pain[149]*†

TEST	SENSITIVITY (95% CI)	SPECIFICITY (95% CI)	POSITIVE LR‡ (95% CI)	NEGATIVE LR‡ (95% CI)
$ULNT1_{MEDIAN}$	0.88 (0.66, 0.93)	0.75 (0.48, 0.93)	3.3 (1.4, 7.8)	0.23 (0.1, 0.5)
$ULNT2_{MEDIAN}$	0.66 (0.48, 0.81)	0.75 (0.48, 0.93)	2.6 (1.1, 6.3)	0.46 (0.3, 0.8)
$ULNT_{RADIAL}$	0.43 (0.26, 0.61)	0.75 (0.48, 0.93)	1.7 (0.7, 4.3)	0.76 (0.5, 1.1)
$ULNT_{ULNAR}$	0.71 (0.54, 0.85)	0.87 (0.62, 0.98)	5.7 (1.5, 21.2)	0.33 (0.2, 0.6)

* Positive test is defined as at least partial reproduction of the patient's symptoms and changing these symptoms with structural differentiation.

† Reference standard for diagnosing cervical radicular pain involved patient history, clinical neurological examination, Spurling test, and MRI.

‡ Calculated by reconstructing 2 x 2 contingency tables from reported data.

CI = confidence interval.

Table 2-7

Diagnostic Accuracy of the Median Nerve Neurodynamic Test ($ULNT1_{MEDIAN}$) for Carpal Tunnel Syndrome*†

TEST	SENSITIVITY (95% CI)	SPECIFICITY (95% CI)	POSITIVE LR (95% CI)	NEGATIVE LR (95% CI)
Vanti et al[152]	0.29 (0.16, 0.45)	0.82 (0.69, 0.91)	1.6 (0.9, 2.8	0.87 (0.5, 1.5)
Bueno-Gracia et al[153]	0.58 (0.45, 0.71)	0.84 (0.72, 0.96)	3.7 (1.7, 7.9)	0.50 (0.4, 0.7)

* Positive test is defined as at least partial reproduction of the patient's symptoms and changing these symptoms with structural differentiation (contralateral side-bending of the neck).

† Reference standard for diagnosing carpal tunnel syndrome was electrodiagnosis with nerve conduction studies.

CI = confidence interval.

therefore unclear whether a positive neurodynamic test was at least partly related to increased nerve mechanosensitivity. The accompanying diagnostic clinical prediction rule that included $ULNT1_{MEDIAN}$ has not been validated, so it cannot yet be recommended for clinical practice.[148]

Carpal Tunnel Syndrome

The median nerve neurodynamic test ($ULNT1_{MEDIAN}$) might not help diagnose carpal tunnel syndrome (Table 2-7).[150,152,153] $ULNT1_{MEDIAN}$ results do not make clinically important changes in the odds of a patient having carpal tunnel syndrome because +LRs are consistently below 5.0 and -LRs are consistently above 0.2.[144,152,153] These findings support other data showing low correlation between $ULNT1_{MEDIAN}$ results and electrodiagnostically confirmed carpal tunnel syndrome.[154] Despite these data, neurodynamic testing may still be relevant for patients suspected to have carpal tunnel syndrome. The prevalence of cervical radicular pain in patients who have carpal tunnel syndrome is much higher than in the general population.[155] Including neurodynamic testing as part of a comprehensive examination may help determine whether a patient suspected to have carpal tunnel syndrome has coexisting cervical radicular pain.

Potential for Bias in Concurrent Validity Studies

In concurrent validity studies, it is important that the intent of the reference standard test matches the intent of the clinical test.[29] There is a mismatch between the intent of neurodynamic tests (identifying increased nerve mechanosensitivity) and the reference standard tests used in published concurrent validity studies. Electrodiagnostic tests have limitations as a reference standard because they focus on loss of function in large-diameter nerve fibers.[156,157] These tests cannot detect irritation of small-diameter afferents[94,95,97,157-159] or increased excitability in nociceptors innervating neural connective tissues[160-162] that can contribute to increased nerve mechanosensitivity. This explains why some patients

who have radicular pain[163] or carpal tunnel syndrome[164] have increased nerve mechanosensitivity even when electrodiagnostic tests are normal. Patients with a nerve-related disorder who have increased nerve mechanosensitivity but no loss of large-diameter nerve fiber function may therefore be misclassified by an electrodiagnostic reference standard as not having a nerve-related disorder. Misclassification of patients may also occur when imaging is the reference standard because there is often no strong correlation between imaging findings and nerve-related pain.[165] Misclassification of patients by these reference standards will bias estimates of the diagnostic accuracy of neurodynamic tests, or any other clinical test, for detecting nerve-related disorders.[166]

The difficulty in quantifying the concurrent validity of neurodynamic tests is that there is no agreed upon reference standard for establishing that an individual patient has increased nerve mechanosensitivity.[167] Until a reference standard for increased nerve mechanosensitivity can be agreed upon, using neurodynamic tests for diagnostic purposes will often be based on lower level evidence from previously described biomechanical and experimental pain studies.[168] Lateral elbow pain is a good example of this situation. A significant proportion of patients who have lateral elbow pain exhibit increased nerve mechanosensitivity with the radial nerve neurodynamic test ($ULNT_{RADIAL}$).[169-171] Lack of a reference standard for nerve mechanosensitivity means that the diagnostic validity of these $ULNT_{RADIAL}$ findings has not been quantified. Nevertheless, positive $ULNT_{RADIAL}$ findings should be monitored to make sure that nerve mechanosensitivity improves with interventions for lateral elbow pain. If nerve mechanosensitivity does not improve after evidence-based interventions such as eccentric exercise and mobilization with movement at the elbow,[172,173] neurodynamic treatment techniques may be indicated.

The mismatch between the intent of electrodiagnostic and neurodynamic tests also highlights the limitations of neurodynamic testing. The focus on nerve mechanosensitivity means that neurodynamic tests cannot capture changes in large-diameter (light touch, strength, reflexes) and small-diameter (pin prick, thermal) nerve fiber function that can be part of nerve-related problems.[137,157,174-177] A comprehensive examination, therefore, requires clinical neurological testing of nerve fiber function complemented by neurodynamic testing.

Test Application

Neurodynamic tests, like most orthopedic physical therapy examination techniques, are psychophysical tests because they require a patient to report the response to a physical stimulus. Cooperation from the patient is also required for proper execution of the test. The psychophysical and cooperative aspects of neurodynamic testing mean that patient-related factors may influence the test response and, therefore, diagnostic performance. Patients' pain cognitions, pain catastrophizing, and expectations of pain prior to testing influence neurodynamic test responses.[178-181] Clinicians should keep this in mind when explaining the purpose of neurodynamic testing to a patient. It may be best initially to describe a neurodynamic test as a general test of mobility or tolerance to movement and not a specific test of nerve sensitivity. If initially described as a test of nerve sensitivity, the patient's thoughts on whether or not the problem is nerve related may bias the response to the neurodynamic test and associated structural differentiation maneuvers.[182]

The spread of biomechanical effects along a nerve mean that a positive neurodynamic test by itself cannot identify the location of the problem.[1,15] It seems intuitive that nerve palpation could help identify the location of the problem. However, clinical observations suggest that tenderness to palpation can spread throughout the length of a sensitized nerve.[2] If present, tenderness to nerve palpation helps build a case for increased mechanosensitivity,[2,94,167,183] but it is not necessarily a good indication of the location of the problem.

Neurodynamic test sequencing has been proposed as a method to help identify the location of a nerve problem.[1] Sequencing is partly based on the belief that different orders of movement can apply different levels of strain to a particular nerve segment at the end of a neurodynamic test.[1] However, cadaveric data show that when joints are moved through similar ROM, different orders of movement do not change nerve strain at the end of a neurodynamic test.[46,55] Joints likely move through different ROM, however, when different neurodynamic test sequences are applied clinically. These potential differences between sequences in the amount of motion that occurs at each joint are more likely to affect nerve biomechanics at the end of a neurodynamic test than any specific effects from the order of movement.[46] It still needs to be determined whether different sequences can improve the diagnostic performance of a neurodynamic test.

Even if sequencing does not ultimately improve neurodynamic test performance, applying a test with different orders of movement may still be useful clinically.[168] A joint movement is not likely to reach full ROM when performed near the end of a neurodynamic test.[184] Clinicians can use this knowledge to modify a neurodynamic test when examining a patient with a sensitive or stiff body part. If a patient has a sensitive or stiff shoulder, neurodynamic testing of the median nerve may be best achieved with a sequence where shoulder abduction would be the last movement. Moving the shoulder last applies less mechanical load to the nonneural tissues in the shoulder but still applies adequate nerve strain, tension, and pressure to provoke sensitized neural tissues. Options for median nerve neurodynamic testing where shoulder abduction would be the last movement include performing $ULNT1_{MEDIAN}$ in a distal-to-proximal sequence or $ULNT2_{MEDIAN}$ in the standard sequence.

Different orders of movement can also help with structural differentiation. If a patient has plantar heel pain, performing ankle dorsiflexion and eversion prior to hip flexion during the SLR can help differentiate increased sensitivity originating from the tibial and plantar nerves from increased

sensitivity originating from the plantar fascia. Ankle dorsiflexion and eversion apply strain to the plantar fascia and tibial and plantar nerves simultaneously.[51] Subsequent hip flexion further increases strain on the tibial and plantar nerves without changing strain on the plantar fascia.[51] This modified SLR test sequence could help the clinician determine whether there is a nerve-related component to the patient's heel symptoms. Lastly, clinicians have always been encouraged to change the order of movement to match a patient's aggravating activities, especially in situations where results from standard neurodynamic tests are inconclusive.[1,10,11]

Educational Messages

Based on data available at this time, interpretation and explanation of a positive neurodynamic test should focus on increased mechanosensitivity, rather than restricted nerve movement or increased nerve stiffness. Even though nerve excursion is reduced in a significant proportion of patients who have carpal tunnel syndrome[185-187] or diabetes mellitus,[4] it does not appear to be reduced in patients who have neck-arm pain with signs of increased nerve mechanosensitivity.[188] For patients who have low back–related leg pain with signs of increased nerve mechanosensitivity, spinal cord movement may be reduced,[189] but there are no apparent restrictions in sciatic nerve movement in the posterior thigh.[60] Although sciatic nerve movement may not be restricted, preliminary data from a small sample suggest that patients who have low back–related pain greater than 6 months duration have increased sciatic nerve stiffness as measured by ultrasound shear-wave elastography.[190] Before concepts about altered nerve biomechanics can have a larger effect on the interpretation of a positive neurodynamic test, clinically feasible methods to determine whether an individual patient has altered nerve biomechanics need to be developed. Clinical trials also need to show that changes in nerve biomechanics are necessary for improvements in patients' nerve-related symptoms and activity levels.

The focus on nerve mechanosensitivity is consistent with the previous discussion on describing a neurodynamic test as a test of tolerance to movement. It is also consistent with helping patients who have a positive neurodynamic test understand why symptoms were changed by movement of a proximal or distal body part during structural differentiation. Explaining structural differentiation to patients provides an opportunity to describe the physical continuity of the nervous system and how different combinations of spine and limb movements apply more mechanical load to neural tissues than nearby nonneural tissues.[1,15] Connecting these concepts to aggravating activities can help patients better understand how increased nerve mechanosensitivity contributes to their symptoms.

A complete understanding and explanation of a patient's nerve-related disorder, including its location, can only come from synthesizing results from the entire examination (eg, distribution of symptoms, patient history, physical examination of nonneural tissues, neurological examination).[15,137,191,192] Discussing relevant impairments from the examination with a patient sets a foundation for implementing a variety of interventions to reduce nerve mechanosensitivity.

Neurodynamic Treatment

Neurodynamic treatment tries to reduce nerve mechanosensitivity by restoring homeostasis in and around the nervous system so that the patient can return to full activity without symptoms.[1,15] In musculoskeletal rehabilitation, there are 3 broad approaches that attempt to reduce nerve mechanosensitivity: (1) nerve mobilization exercises that move neural tissues relative to surrounding structures and can be described as sliding or tensioning techniques[13,193,194]; (2) contralateral cervical lateral glide (CCLG)[14,195] and side-lying lumbar foraminal opening[196] techniques that conversely mobilize structures around sensitized neural tissues[2]; and (3) interventions directed at other (nonneural) musculoskeletal impairments.[1,15] Regardless of the approach(es) used, education about the pain biology underlying the nerve-related disorder can improve outcomes[197,198] and may help the patient better understand the rationale for movement-based interventions.[1,15,199]

Nerve Mobilization Exercises

Nerve mobilization exercises are passive or active techniques that try to reduce nerve mechanosensitivity by moving neural tissues relative to surrounding structures.[13,193,194] Historically, nerve mobilization exercises were based on neurodynamic test movements where one or more joints were moved in a way that lengthened the anatomical course of the nerve (Figure 2-3).[168] These are now referred to as *tensioning techniques*[13,193,194] because while they do create excursion of the nerve relative to surrounding tissues, they also create significant increases in nerve strain.[13,59,60,193,194] However, different combinations of joint movements have markedly different effects on nerve biomechanics. When a joint movement that lengthens the anatomical course of the nerve is simultaneously offset by another movement that shortens the anatomical course of the nerve (see Figure 2-3), there are 2.5 to 5 times greater amounts of nerve excursion without significant increases in nerve strain.[13,59,193,194] The emphasis on nerve excursion over strain is why these movement combinations are referred to as *sliding techniques*.[13,193,194]

Despite different biomechanical effects, it is impossible to state that one type of nerve mobilization exercise is clinically superior to the other.[13] There are conflicting data on whether sliding or tensioning techniques have larger immediate hypoalgesic effects in asymptomatic individuals.[200-202] Additionally, there are no data to date comparing the effects of sliding and tensioning techniques in symptomatic populations over longer follow-up. Technique selection needs to

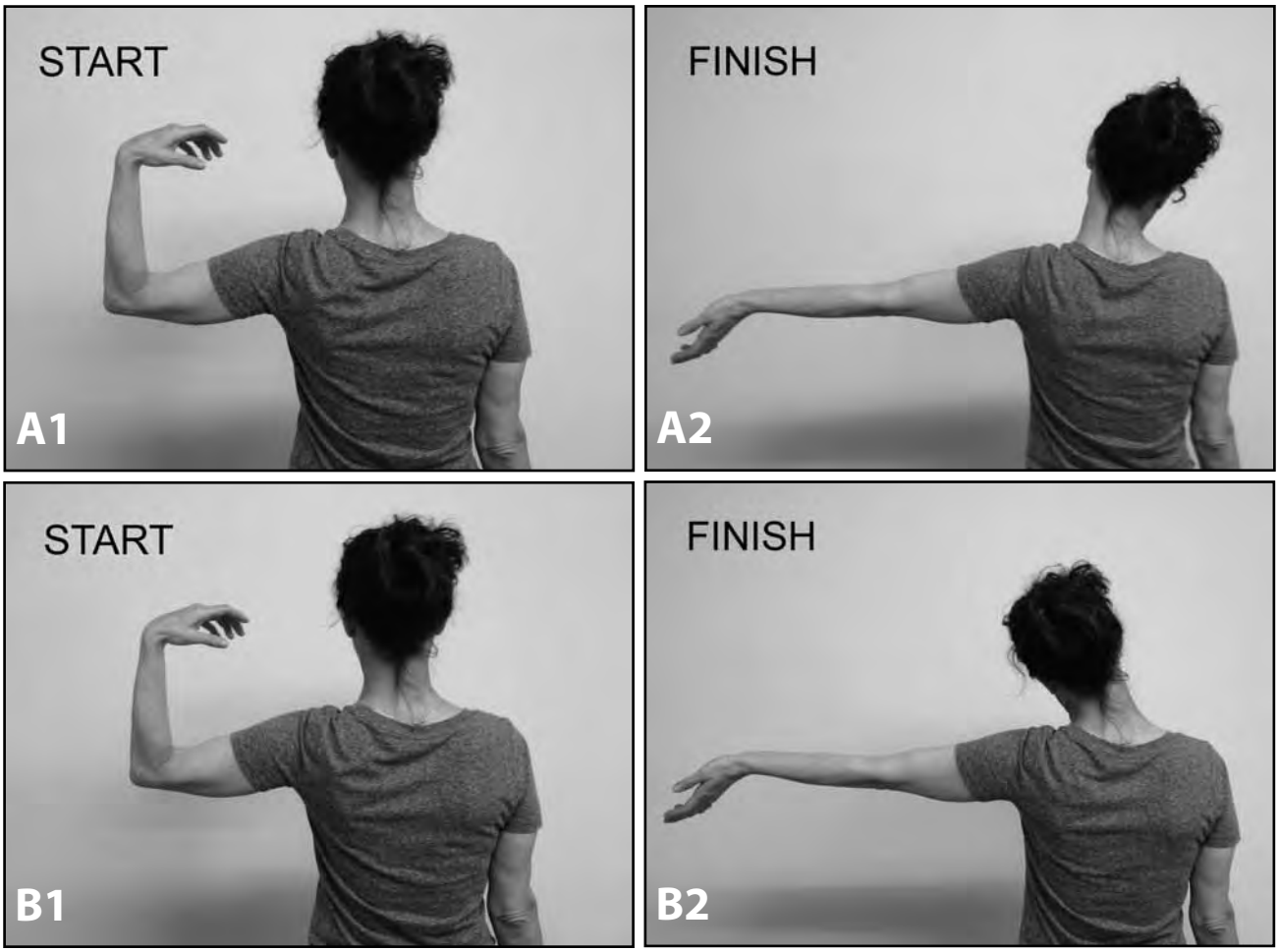


Figure 2-3. Examples of active nerve mobilization exercises for the cervical nerve roots and median nerve. (A) Tensioning technique where contralateral neck side-bending, elbow extension, and wrist extension lengthen the anatomical course of the nerve. (B) Sliding technique where elbow and wrist extension movements that lengthen the anatomical course of the nerve are offset by ipsilateral neck side-bending that shortens the anatomical course of the nerve.

be based on sound clinical reasoning that takes into account the movement requirements of the patient's activities. Sliding techniques are less vigorous biomechanically and, therefore, may be indicated when the patient's symptoms are more reactive or irritable, assuming that a movement-based intervention is deemed appropriate.[203-205] Tensioning techniques may be appropriate for less irritable conditions or when the patient's tolerance for movements that lengthen the anatomical course of the nerve has not been restored with other interventions.

Mobilizing Structures Around Sensitized Neural Tissues

Mobilizing structures around sensitized neural tissues is another approach that tries to reduce mechanosensitivity by creating movement between nerves and surrounding structures.[2,14] The CCLG technique, originally described by Elvey,[14] is the most commonly studied example of this type of neurodynamic treatment technique (Figure 2-4). It has shown immediate hypoalgesic effects in a variety of conditions such as nerve-related neck and arm pain,[206,207] lateral epicondylalgia,[208] and whiplash-associated disorder.[209] Furthermore, the CCLG technique in isolation[210] or as part of a neurodynamic treatment program[211,212] can improve the short-term natural history of nerve-related neck and arm pain. An analogous technique in the lumbar region is a side-lying foraminal opening technique where the affected lumbar spine motion segments are laterally flexed away from the symptomatic limb (Figure 2-5).[196] Observational[196] and clinical trial[213] data suggest that this technique may be helpful for patients who have low back–related leg pain with signs of increased nerve mechanosensitivity. When reassessment shows that the patient's rate of improvement has slowed or plateaued, a progression of these techniques would be to perform them with the limb positioned to pre-load the affected neural tissues (see Figures 2-4 and 2-5).[14]

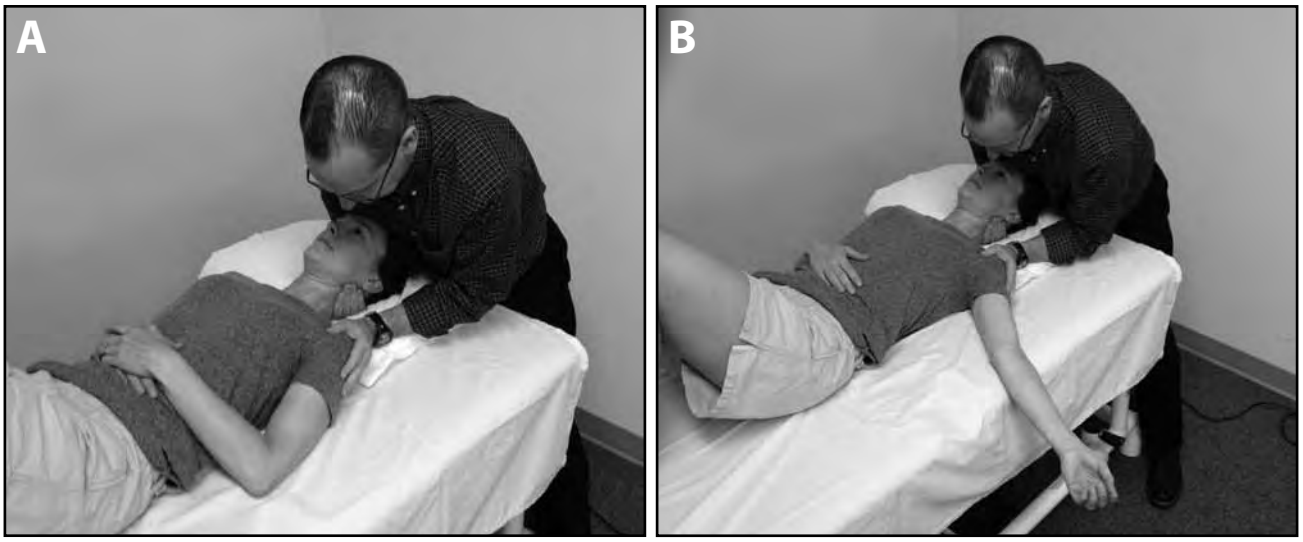


Figure 2-4. CCLG technique. (A) The head and neck are translated in the frontal plane away from the symptomatic arm so that there is minimal rotation or side-bending of the cervical spine. The hand on the shoulder girdle helps the clinician monitor resistance to movement. (B) The technique can be progressed by positioning the limb to pre-load the upper extremity neural tissues. The illustrated upper extremity position would pre-load the median nerve and associated neural tissues.

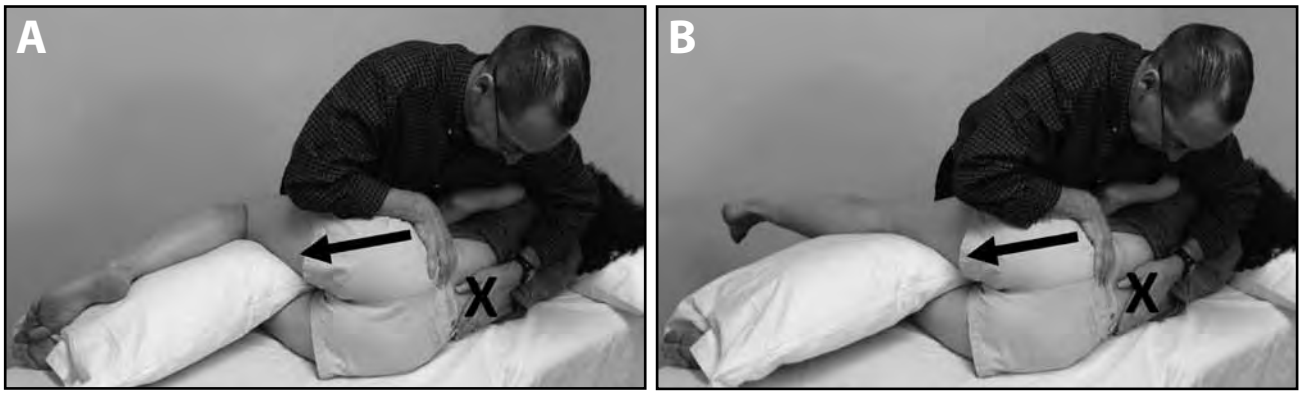
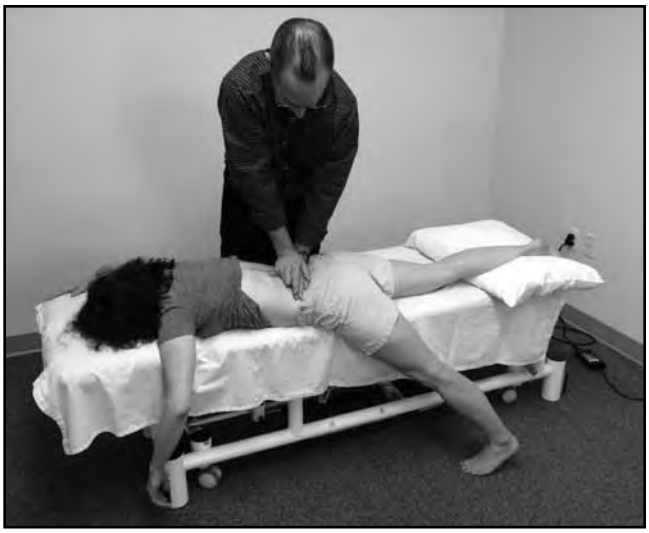


Figure 2-5. Lumbar foraminal opening technique. (A) Lumbar lateral flexion away from the symptomatic limb is created by moving the pelvis (arrow shows direction of force) while stabilizing the spinous process of the superior vertebra of the affected segment (X). (B) The technique can be progressed by positioning the limb to pre-load the lower extremity neural tissues.

Interventions Directed at Other (Nonneural) Musculoskeletal Impairments

Even though they do not try to create movement between neural tissues and surrounding structures, interventions targeting relevant nonneural musculoskeletal impairments can also be used to decrease nerve mechanosensitivity.[1,15] Examples include clinical trials for carpal tunnel syndrome[214,215] and a trial protocol for nerve-related neck and arm pain[216] where soft tissue techniques are applied at places along the nerve that can often be associated with increased mechanosensitivity (eg, scalenes, pectoralis minor, medial aspect of the upper arm, pronator teres, palmar aponeurosis). Case studies provide examples of a commonly observed clinical phenomenon where manual therapy applied to the spine (or extremities) is associated with immediate improvements in neurodynamic test findings.[217,218] The intensity of these manual therapy techniques can also be increased if necessary by using limb position to pre-load the affected neural tissues (Figure 2-6).[217,218] Therapeutic exercise targeting nonneural structures is another intervention that can reduce nerve mechanosensitivity. For example, in a patient who has clinical signs of glenohumeral instability,[219,220] associated problems with nerve mechanosensitivity may be reduced by therapeutic exercise targeting the rotator cuff and scapulothoracic musculature (Case Study One). The key clinical message is that patients who have symptoms related to increased nerve mechanosensitivity do not always have to receive nerve mobilization exercises as part of their intervention.

Figure 2-6. Example of progressing a manual therapy technique (lumbar central posterior-anterior mobilization) by placing the limb in a partial SLR position to pre-load the lumbosacral nerve roots and sciatic tract.



Potential Mechanisms of Neurodynamic Treatment

There are now preliminary data on aspects of pain biology involved in nerve-related disorders that may be influenced by neurodynamic treatment techniques.[13,168,199] These mechanistic studies have usually enrolled patients who have carpal tunnel syndrome (a common clinical model for nerve compression) or employed animal models of nerve-related pain. Although initial findings are promising, more research on the mechanisms of neurodynamic treatment is needed.[168]

Reduction of Edema and Pressure

Intraneural edema is part of the pathophysiology of nerve root and peripheral nerve disorders.[221-224] It reflects an inflammatory response to mechanical and chemical stimuli that compromise intraneural circulation.[222-224] Removing intraneural edema is difficult because nerve roots and peripheral nerves do not have a lymphatic system.[225,226] Persistent edema increases pressure inside nerve fascicles, creating a miniature compartment syndrome that perpetuates the problem.[221,225] Persistent intraneural edema also provides an environment for the development of fibrosis and can contribute to degradation of myelin and axon loss.[223]

Preliminary data suggest that neurodynamic treatment techniques can potentially reduce intraneural edema. A 1-week program of nerve mobilization exercises reduces MRI evidence of intraneural edema in patients who have carpal tunnel syndrome, something not observed in those who receive advice to remain active.[227] Cadaveric data show that tensioning techniques have immediate mechanical effects that produce dispersion of intraneural fluid in nerve roots and peripheral nerves.[80-82] Although limitations in applying cadaveric data to the clinical setting must be acknowledged, the authors hypothesized that these mechanical effects could help reduce intraneural edema.[80-82]

Edema around nerves and the associated increased pressure can contribute to some nerve-related disorders such as carpal tunnel syndrome[228] and radicular pain.[229,230] Although not specifically focused on nerve gliding, brief bouts of active wrist flexion and extension reduce carpal tunnel pressure in patients who have carpal tunnel syndrome.[231] It is plausible that nerve mobilization exercises for carpal tunnel syndrome could also have this effect because they can incorporate active wrist flexion and extension movements.[193,227,232] Reducing edema and pressure around nerves is important because it may help improve intraneural circulation and axonal transport. These 2 physiological processes have a significant effect on nerve function and mechanosensitivity.[95,226,233,234]

Reducing neural edema and pressure is not exclusive to neurodynamic treatment techniques. Wrist orthoses (splinting) can also reduce MRI evidence of intraneural edema in patients who have carpal tunnel syndrome.[227] The rationale for night splinting to prevent extremes of elbow flexion in patients who have cubital tunnel syndrome is to reduce strain and pressure applied to the ulnar nerve.[235] However, splinting may not be necessary when patients are educated on the pathomechanics of cubital tunnel syndrome and advised to avoid aggravating positions.[236]

Dispersal of Inflammatory Chemicals

Animal models of mild nerve injury have shown that the inflamed portion of the nerve can be extremely sensitive to stretch and pressure even when nerve conduction is largely unaffected.[94,95,98,99] The development of mechanosensitivity and associated behavioral signs indicative of nerve-related pain are partly due to the interruption of axonal transport.[95,97,99] Impaired axonal transport allows for accumulation of inflammatory and other chemical mediators that increase mechanosensitivity at the inflamed site of the nerve.[95,97,237]

Table 4-3

Cranial Nerve Examination

NERVE	FUNCTION	TEST PROCEDURE	SIGNIFICANT FINDINGS
I Olfactory	Smell	Odor recognition (eg, coffee, orange, vanilla)	Loss of smell
II Optic	Visual acuity	Snellen or Rosenbaum eye chart	Partial or complete vision loss
II Optic III Oculomotor	Pupillary light reflex	Place ulnar side of hand with fingers extended on bridge of patient's nose (block contralateral eye from light). Shine light to ipsilateral eye and (1) observe for pupil constriction, and (2) observe contralateral eye for consensual response; repeat for other eye.	Absent pupil constriction or consensual response to light
III Oculomotor IV Trochlear VI Abducens	Extraocular eye movements	(1) 12 to 18 inches from patient's eyes, perform "H" movement with finger.* Patient asked to follow with eyes only (head stationary). (2) 12 to 18 inches from patient's eyes, hold up 2 widely spaced targets* (eg, one finger from each hand). Ask patient to take eyes quickly from one finger to the other.	Abnormal gaze, nystagmus, or uncoordinated eye movement
V Trigeminal	Facial sensation	Assess sensation with cotton to facial areas V1, V2, and V3	Absent or asymmetrical
VII Facial	Facial expression symmetry	Ask patient to wrinkle (forehead), wink, whistle, and wince	Absent or asymmetrical
VIII Vestibulocochlear	Hearing, balance	Eyes closed, therapist rubs fingers near patient's ear	Absent or asymmetrical
IX Glossopharyngeal X Vagus	Gag reflex, swallowing, and uvula symmetry	Use a tongue depressor to stimulate the back of the throat Ask the patient to say "ah" and observe the uvula	Absent gag reflex Deviated uvula
XI Accessory	Head, neck motion	MMT trapezius and sternocleidomastoid	Weakness
XII Hypoglossal	Tongue movement	Tongue protrusion	Deviation to affected side

* Keep fingers in patient's midcervical spine ROM to prevent end-point (normal) nystagmus.

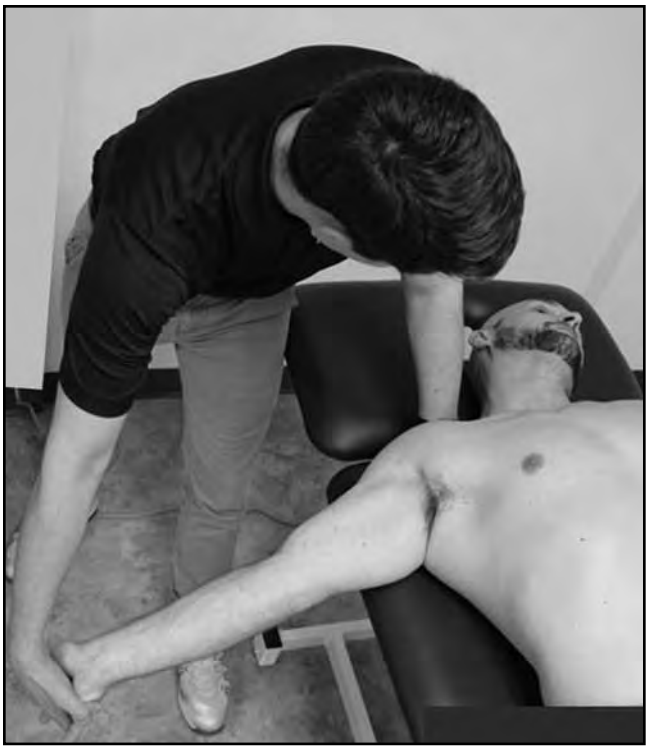


Figure 4-17. Upper limb tension testing median bias.

Figure 4-18. Upper limb tension testing radial bias.

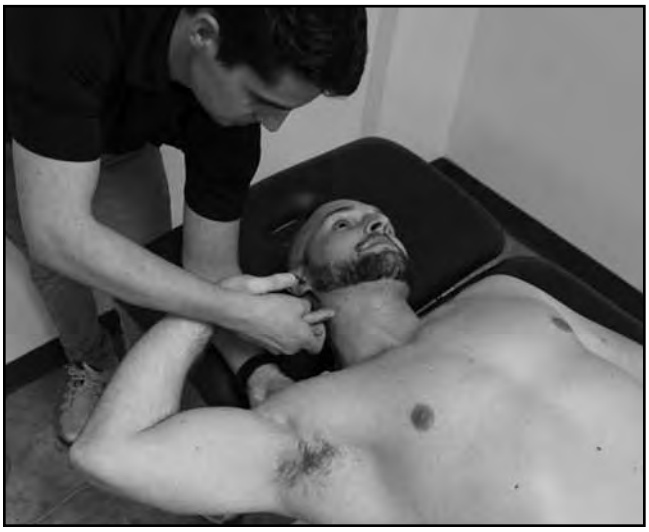


Figure 4-19. Upper limb tension testing ulnar bias.

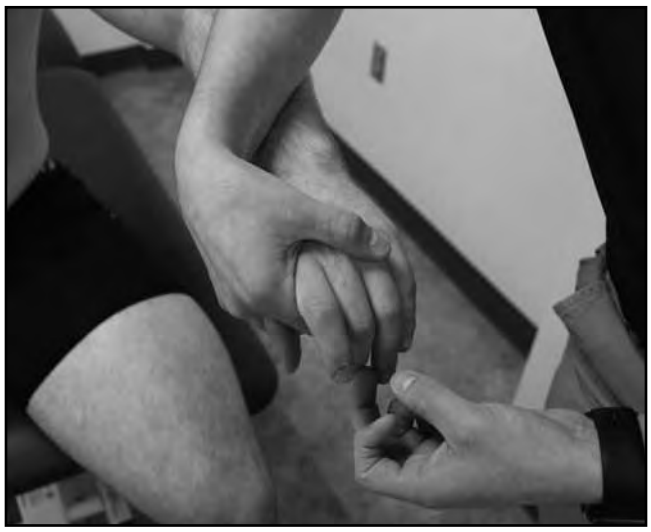


Figure 4-20. Hoffmann reflex.

measures help confirm or reject a hypothesis and/or support clinical decisions regarding diagnosis, prognosis, and the plan of care. The following is from a list of test and measure categories for the *Guide to Physical Therapist Practice*[2]:

- Aerobic Capacity/Endurance
- Balance
- Cranial and Peripheral Nerve Integrity
- Joint Integrity and Mobility
- Mental Functions
- Mobility (Including Locomotion)
- Motor Function
- Muscle Performance (Including Strength, Power, Endurance, and Length)
- Posture
- ROM
- Reflex Integrity
- Sensory Integrity
- Skeletal Integrity

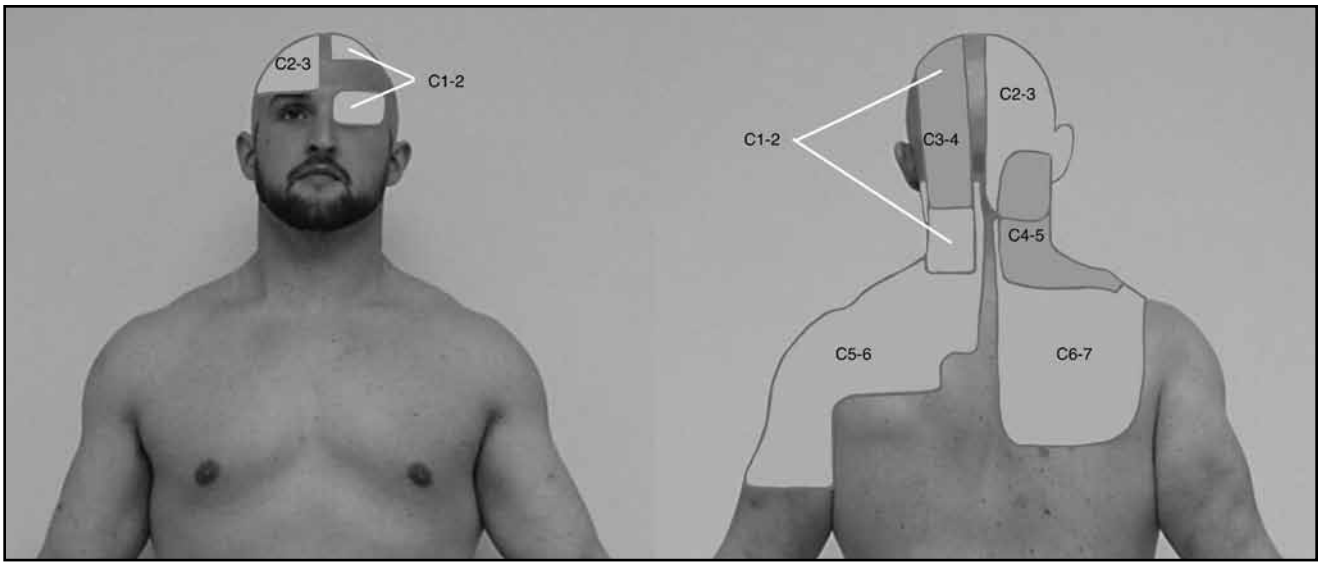


Figure 4-21. Facet joint referral patterns.

Palpation

Patient Position: Variable

Various texts recommend performing palpation early in the physical examination sequence. However, from clinical experience, early palpation can cause irritation, possibly altering the results of the remaining physical examination components. Regardless, layer palpation should be performed, which means the examiner works from superficial to deeper tissues. Any change in contour (ie, lump, bump, or lesion) should be brought to the patient's attention. Follow-up questions include determining whether their physician is aware of the lump and if there has been any change to it. Palpate in a systematic fashion the UQ soft tissues and bony structures. Knowledge of UQ lymph nodes and arteries is also important. In general, during palpation ask the patient if there is any discomfort or change in symptoms. The grading scale below can be used to quantify palpation.

- Grade 1 = Tenderness
- Grade 2 = Tenderness with flinch
- Grade 3 = Severe tenderness, withdrawal
- Grade 4 = Hyperalgesia

Upper Quarter Referral Patterns

For the purposes of this chapter and inconsistent use of definitions regarding referral patterns, it is important to begin by defining relevant terminology.[15,16] Referred pain can be either somatic or viscerogenic and is defined as pain perceived in an area of the body separate from the location of the actual source.[17] Common somatic pain generators include facet (zygapophyseal) joints, IVDs, and muscle tissue. Viscerogenic referred pain is believed to be a result of multisegmental innervation and direct pressure/shared pathways.[16] Radicular pain is due to a direct irritation of the nerve root and is not considered a type of referred pain.

Referred Pain—Facet Joints

Facet joints are innervated spinal synovial joints and a known structure to produce somatic referred pain (Figure 4-21). Facet joints as a pain generator is common with more than 50% of patients with cervical spine pain and more than 40% of patients with thoracic spine pain having at least one symptomatic facet joint.[18,19] Referral of pain beyond the upper arm, especially below the elbow, would likely rule out facet joint as a cause of the patient's pain.[18]

Published evidence to best distinguish facet joint pain from other causes from subjective examination findings is lacking.[20] A review of the literature concluded that the clinical presentation of cervical facet joint pain is similar to other axial neck pain etiologies with the main clinical feature being pain, which may radiate to the occiput, the shoulders, or midscapular region.[20] From clinical experience, patients are typically older (50 years or older), describe pain as worse in end-range extension, and abolished in flexion.

A clinical decision guide (CDG)[21] has been developed, which can help rule in or out cervical facet joint as the cause of symptoms for patients with a chief complain of neck pain. The CDG includes the following physical examination findings:

- PST: Reproduction of familiar pain/tenderness with palpation of segmental musculature (patient in prone)
- Manual spinal extension: Reproduction of familiar pain with a posterior to anterior force (patient in prone)
- Extension-rotation test: Reproduction of familiar pain with active end-range extension, followed by rotation (patient seated)

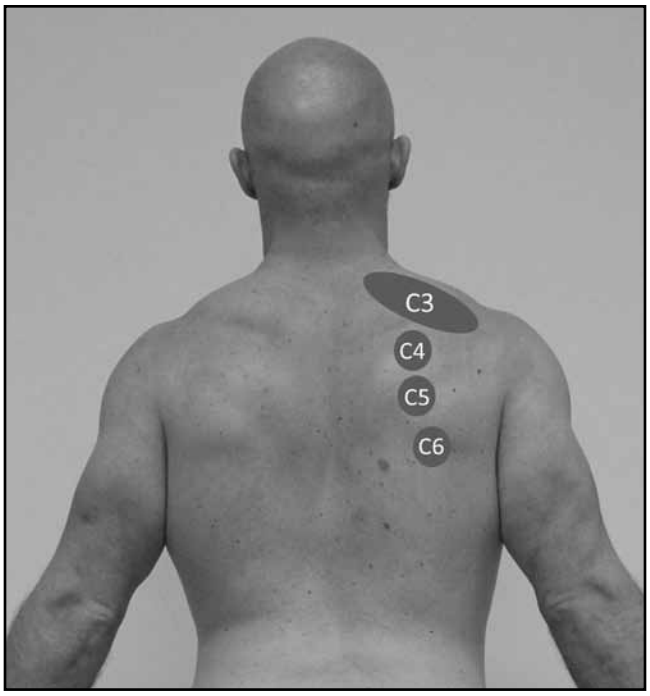


Figure 4-22. IVD referral patterns.

If each test is positive, the CDG increases the posttest probability from 42% to 78%. Conversely, the negative result for PST is good for ruling out the facet joint as the cause of the patient's pain due to its low negative likelihood ratio (0.08 [95% confidence interval (CI), 0.03 to 0.24]). That is, a negative result for the PST reduces the posttest probability of a diagnosis of facet joint pain from 42% to 5%. Although this CDG was developed for patients with neck pain, it may be plausible to adapt this guide for patients with thoracic spine pain; however, it is important to note that this CDG has yet to be validated in any population.[21]

Referred Pain—Intervertebral Disk

Referral of pain from IVDs (also known as the *Cloward sign*) is well documented. Mapping of cervical IVD referral patterns is established (Figure 4-22), and although referral patterns of thoracic IVD have not been mapped, it is believed to be a pain generator.[22-24] It is important to note this is somatic referred pain from the IVD itself, not impingement upon the nerve root (ie, radiculopathy).[15]

Somatic structures, such as facet joints and IVD, which are innervated by the same spinal segment, result in neural convergence of afferent nociception, making differentiation between structures difficult.[17] Therefore, because the goal of the examination is to make a physical therapist diagnosis and begin treatment, if it is determined that the source of the patient's symptoms is appropriate for physical therapist intervention (ie, musculoskeletal), then differentiating the anatomical source of pain is not required.

While it may be useful from a prognosis standpoint to identify the pathoanatomical structure due to differences in tissue healing time, it is well known that cervical spine pathological changes observed on imaging are common in asymptomatic individuals, making pathoanatomical findings suspect and less likely to help guide treatment in symptomatic individuals.[25] As musculoskeletal experts, physical therapists can best guide examination and treatment by response to movement and/or loading, which is congruent with treatment-based classifications that use physical therapist examination findings to establish and guide the physical therapist treatment plan.[26,27]

Referred Pain—Muscular

Common terminology to describe muscular pain includes *myofascial pain*, *myofascial trigger points*, or simply *trigger point*. A trigger point is a palpable tender spot in a taut band of skeletal muscle that produces pain in a predictable referral pattern and a local twitch response.[28,29] Unfortunately, the definition of a trigger point is inconsistent, therefore making comparisons in the literature regarding diagnostic criteria difficult.[29] In addition, locating trigger points via palpation, the main tool advocated for identifying these structures, has been found to have marginal to poor reliability, even after training.[30,31]

Although the authors of this chapter are speculating, trigger points may be a sign of nervous system irritability, similar to neural tension testing, as opposed to a local tissue issue. It appears palpation of the irritable tissue may refer pain consistent with its innervation. For example, the purported infraspinatus muscle trigger point referral pattern is consistent with the C5-C6 dermatome distribution.[28] The infraspinatus is innervated by the suprascapular nerve, which arises from the superior trunk of the brachial plexus, consisting of C5 and C6.[10]

Referred Pain—Viscerogenic

Although the abdomen is often associated with the lower quarter examination, abdominal structures must be included as a potential source of UQ pain due to referral patterns. Viscerogenic referred pain is believed to be a result of 3 mechanisms. First, embryologic development is believed to play a role. For example, during human embryonic development, the pericardium is formed in the gut, which may help explain why a myocardial infarction can refer pain to the abdomen.[16] Second, there is multisegmental innervation of viscera, resulting in overlap with somatic structures that share the same spinal afferent pathway, a concept known as *visceral-organ cross-sensitization*.[16] Last, viscerogenic referred pain can be caused by direct pressure of an inflamed visceral structure on the respiratory diaphragm.[16] The respiratory diaphragm is innervated by the phrenic nerve (C3-C5), which shares common innervation with the shoulder. Refer to Figure 4-23 for common viscerogenic referral sites.

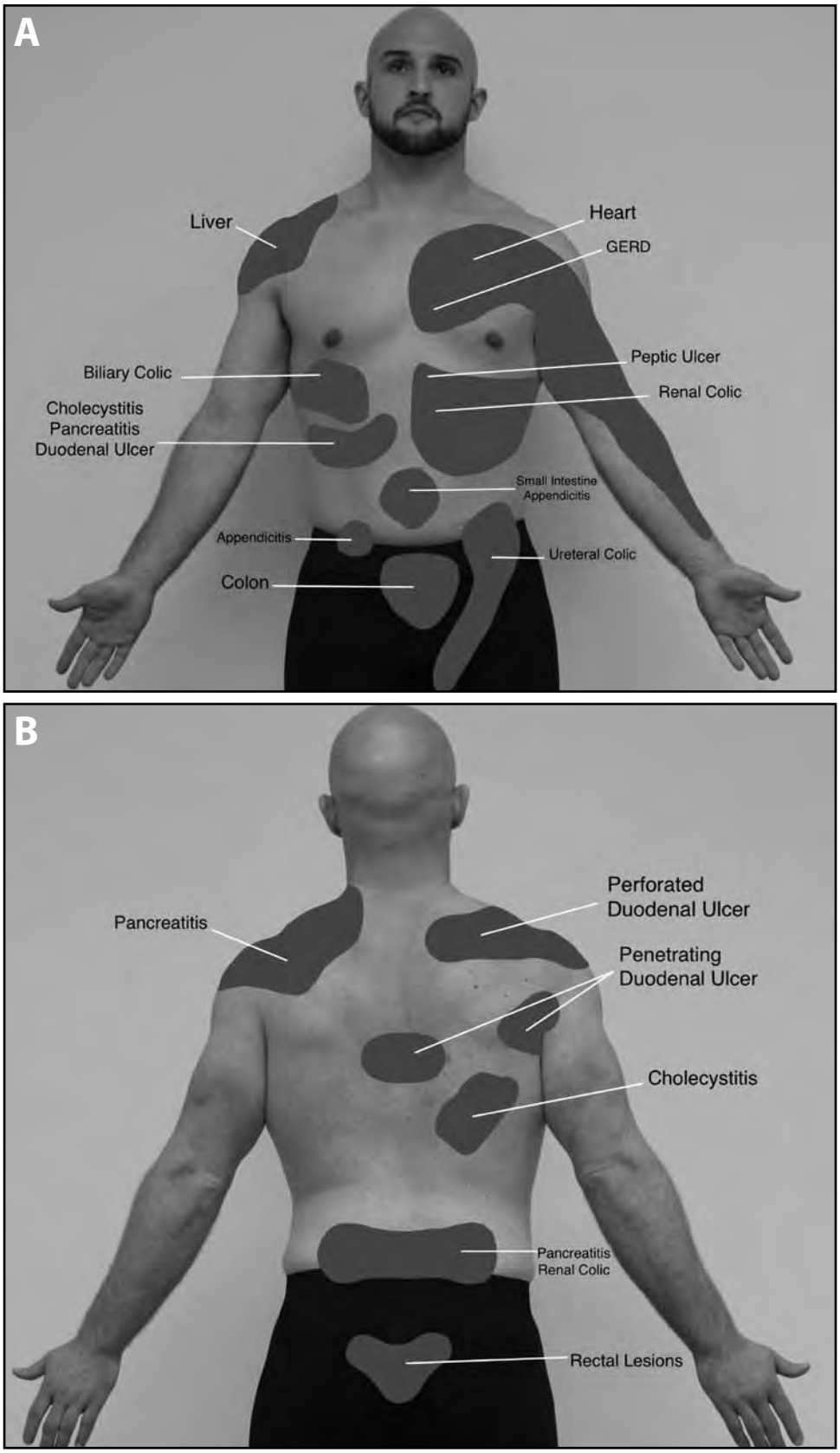


Figure 4-23. Viscerogenic.