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Motion Analysis of the Cervical Spine

By Patrick R. Luers, MD

A motion segment is defined in the Fifth Edition of the *Guides*, Section 15.1b, Description of Clinical Studies, as "two adjacent vertebrae, the intervertebral disk, the apophyseal or facet joints, and ligamentous structures between the vertebrae" (5th ed, 378). The range of motion from segment to segment varies.[1] Motion of individual spine segments cannot be determined by a physical examination but is evaluated with flexion and extension roentgenograms. Alteration of motion segment integrity, as defined in the Fifth Edition of the *Guides*, most often is due to decreased motion secondary to fusion. Alteration of motion segment integrity due to increased translation or angular motion as defined in the *Guides* is rare.

Section 15.1b, Description of Clinical Studies, defines loss of motion segment integrity as follows:

> Loss of motion segment integrity is defined as an anteroposterior motion of one vertebra over another that is greater than 3.5 mm in the cervical spine, greater than 2.5 mm in the thoracic spine, and greater than 4.5 mm in the lumbar spine (Figure 15-3a). Loss of motion segment integrity is also defined as a difference in the angular motion of two adjacent motion segments greater than 15˚ at L1-2, L2-3, and L3-4 and greater than 20˚ at L4 to L5. Loss of integrity of the lumbosacral joint is defined as angular motion between L5 and S1 that is greater than 25˚. In the cervical spine, loss of motion segment integrity is defined as motion at the level in question that is more than 11˚ greater than at either adjacent level. (5th ed, 379)

References cited for measurements are White and Panjabi's chapter in *Clinical Biomechanics of the Spine*, 2nd Edition,[2] and an article by Shaffer et al published in *Spine* in 1990.[3]

Normal flexion occurs in a steplike sequential fashion, initiating at C1-2, followed by C2-3, C3-4, C4-5, C5-6, and C6-7.[4] The greatest angulation and displacement at maximal flexion normally occurs at the C4 and C5 vertebral levels, most commonly at C4-5 and C5-6. C3-4 typically exhibits the next greatest angular motion and displacement, with the least motion noted at C2-3. Motion at one interspace occurs gradually and does not occur without similar motion occurring at adjacent levels.

Normal motion of the cervical spine is classically defined as follows:

> During flexion, there is a continual movement from the lower through the upper segments, characterized by each successfully higher segment translating (moving) progressively further forward while pivoting around its coronal axis. This change in the relationship of adjacent vertebrae may occur in one of two ways, each of which has a

distinctive radiographic appearance. In one instance, the forward translation of each successfully higher segment may be smoothly continuous so that connecting the posterior cortical margins of the centra results in an uninterrupted anteriorly concave imaginary line. In the other instance, flexion may occur as each successfully higher segment translates anteriorly a discrete distance so that flexion occurs through a series of seemingly separate, disconnected, segmental anterior increments. Regardless of the nature of the flexion, in extension, these vertebral movements are reversed, and each successfully higher segment may be posteriorly displaced with respect to the subjacent vertebrae as much as 3.5 mm physiologically. Finally, and with respect to the lateral radiograph of the cervical spine, in the neutral lateral position any lower cervical vertebrae may be normally anteriorly translated with respect to the adjacent segments by a distance that normally may be as much as 3.5 mm as measured from the posterior cortical margins of the adjacent vertebral bodies.[5]

Evaluation of cervical spine motion using fluoroscopy and cineradiographic techniques has been described in the literature, the bulk of which is from the 1950s and 1960s. A quantitative evaluation of cervical spine motion evaluated by fluoroscopy/cineradiography in the literature was presented by Hino et al in 1999.[6] In this study, cineradiographic frames were digitized to quantitatively measure initiation of motion at cervical vertebral levels of 10 healthy, asymptomatic volunteers and 12 symptomatic patients. The remaining studies in the literature describe qualitative evaluation of cervical spine motion using fluoroscopic or cineoradiographic techniques.

Intervertebral levels with pathological increased motion and/or instability demonstrate earlier than expected initiation of motion with flexion (often with a jumping or jerking type of motion), increased angulation (also referred to as *kinking*, *anterior subluxation*, or *hyperflexion subluxation*[7]), angular displacement (also referred to as *fanning*[8]) and/or increased translation (longitudinal displacement).

Increased Motion in the Cervical Spine

There are multiple etiologies of increased motion in the cervical spine, some of which are physiologic or compensatory and others of which are pathologic. The following four conditions may result in an increase in motion in cervical segments.

Increased Elasticity/Ligamentous Laxity in Immature Spines, Young Adults, and Double-Jointed Individuals

Increased mobility is identified at all cervical segments in adolescent spines compared to adult, mature spines. The findings are particularly evident in infants, neonates, and small children up to the age of 3 to 4 years, where rather marked displacement of vertebral bodies is identified with flexion (pseudosubluxation). Prior to complete maturation of the spine, the vertebral end plates are cartilaginous, which means they are more flexible than bone. Increased mobility is identified in children, adolescents, and young adults with immature spines.[7] Spinal maturity occurs with fusion of the cartilaginous ring apophyses, which typically occurs in the early to mid 20s.

Elastic fibers tend to degenerate with age, and collagen, which is the major component of ligaments, tends to lose elasticity with age. Cervical spine motion of young adults between the early 20s and early 30s is typically greater than that of older adults. Between the 2nd and 8th decades, spinal mobility and range of motion decrease by 21%.[9] Additionally, the degree of elasticity/ligamentous laxity present in adults varies. Double-jointed individuals possess a great deal of ligamentous laxity and elasticity in their ligaments, resulting in much greater than normal range of motion in their joints and spine.

Increased range of motion in immature cervical spines, young adults, and double-jointed individuals will be observed throughout all levels of the cervical spine and is diffuse in nature, with maximal translation and angular motion occurring at the C4 and C5 vertebral levels. The increased motion

that is observed is symmetric, and initiation of flexion will be normal (beginning in a stepwise fashion at C1-2 to inferior levels). Objective measurements of translation, angular motion (fanning and kinking), and longitudinal displacement (translation) will be increased in all levels of the cervical spine in these conditions. Such patterns represent normal variations in development and should not be considered to be pathological or related to underlying ligamentous injury, which is most commonly more focal, involving a level.

Compensatory Increased Motion in Congenital Fusions, Surgical Fusions, Severe Degenerative Disk and Facet Disease, and Muscle Spasm/Guarding

If one or more vertebral levels in the cervical spine are immobile or fused, increased motion will occur at adjacent levels to compensate for the lost range of motion at the fused/immobile segments. Normal range of motion may not be accomplished for the entire cervical spine, but the levels adjacent to an immobile/fused level nearly always demonstrate increased compensatory motion. Relatively less increased compensatory motion is seen at levels more remote from the fused/immobile segments. Immobile segments include congenitally fused segments (such as those seen in Klippel-Feil syndrome), surgically fused levels, and levels in patients with severe degenerative disk and facet disease. In the latter condition, marked disk space narrowing with bridging osteophytes and large marginal facet spurs are commonly identified, resulting in autofusion. Disk spaces and facets may also become fused after an infection, such as diskitis or a septic joint.

Evaluating potential traumatic ligamentous injury in a patient with a fused cervical spine is extremely difficult without a pre-accident comparison study, as compensatory motion adjacent to the fused levels is expected to result in increased angular motion and translation. The standard objective measurements for angular motion (fanning and kinking) and longitudinal displacement (translation) do not apply in patients with fused/immobile cervical segments. If pre- and postaccident flexion and extension x-ray series are available for comparison, evaluation of potential superimposed ligamentous injury to the levels adjacent to a fusion is much more definitive.

Muscle spasm and guarding may result in either focally or diffusely decreased motion at affected levels in the cervical spine. The absence of any significant flexion angle (3˚ or greater) on an adequate flexion film (60˚ to 65˚ mandibular angle) is most commonly secondary to muscle spasm or guarding and is most typically associated with underlying ligamentous injury.[7,9] Head-tilting to the side of muscle spasm/guarding, elevation of the posterior first rib, scoliosis, rotation, or torticollis in the cervical spine are commonly associated with muscle spasm and guarding. Motion at less affected or unaffected levels is typically exaggerated in a compensatory fashion. Evaluation of ligamentous instability at levels affected by muscle spasm, as well as levels unaffected by muscle spasm, must be evaluated with great caution. Follow-up flexion and extension views are recommended to evaluate underlying instability in patients with no significant flexion angles (straightening of the cervical spine) on initial studies with an adequate flexion effort.[7]

Mild to Moderate Degenerative Disk and Facet Disease

Degenerative disk disease produces biochemical alterations in the nucleus pulposis and inner annulus fibrosis, resulting primarily in disk desiccation and disk space narrowing. Loss of disk height produces relative redundancy of the outer annulus and adjacent anterior and posterior longitudinal ligaments, resulting in diffuse disk bulging on cross-sectional imaging studies such as CT and MRI scans. The loss of disk height and relative redundancy of the outer annulus and adjacent ligaments produces increased motion at these segments and alters facet joint articulation. Facet joint arthritis typically is associated with degenerative disk disease and also contributes to increased motion at the degenerated segment level. Hino et al[6] found that early onset of abnormal motion and increased longitudinal displacement was identified at degenerated disk levels. Thus, degenerative disk disease commonly results in increased motion at the affected level, most typically seen in mild to moderate disk and facet disease, or decreased motion, which is typically seen in severe degenerative disk and facet disease. The abnormal motion identified at the degenerated levels always occurs focally at that level. Abnormal motion seen focally at a mildly to moderately degenerated level in the cervical spine is expected and does not imply associated ligamentous injury.

Musculoligamentous Injury

Significant trauma to the cervical spine commonly produces soft tissue injuries, predominantly to the muscles and ligaments, which can result in instability. Such injuries are graded from I to III depending on severity and are referred to as *sprain/strain* injuries. A grade I injury is defined as an intrasubstance tear of the ligament and/or muscle tendon that does not extend through the surface of the ligament or tendon. A grade II injury is defined as a tear that extends to one surface of the tendon or ligament but does not penetrate through both surfaces. A grade III injury is defined as complete disruption of the tendon or ligament. The extent of instability produced by ligament/musculotendinous injury depends on the grade of the resulting tear and the degree of involvement of adjacent ligaments and tendons in the cervical spine. Grade I injuries do not typically result in increased/abnormal motion in the cervical spine, but are commonly associated with overall decreased range of motion, muscle spasm, and guarding.

Ligamentous structures in the spine are typically classified into 3 groups/columns, consisting of anterior, middle, and posterior compartment ligaments. Greater motion and instability will be identified with grade III tears that involve more than 1 compartment or are associated with fractures through more than 1 compartment. Fluoroscopic analysis of flexion and extension typically reveals early motion with flexion and/or extension at the injured level, increased angular motion (fanning and

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Case Study: Carpal Tunnel Syndrome

The following case is that of a man who underwent bilateral carpal tunnel release. His rating was performed by two physicians in California. The AMA *Guides* are used to rate Longshore and Harbor Workers cases; however, at the time of these ratings, the *Guides* were not yet used in California for state workers' compensation cases. As of January 2005, the use of the Fifth Edition of the *Guides* will be required for all workers' compensation cases.

Report 1: Dr Prior, November 26, 2002

I had the opportunity to reevaluate Mr Sample 11-26-02. It is my opinion that he is permanent and stationary as of that date. The following is a Permanent and Stationary Narrative Report.

History of Injury

Please see my initial dictation dated 09-04-00 for a complete history. Briefly, Mr Sample is a now 48-year-old, right-hand-dominant "Laborer" for Ship Enterprises, a company that does shipyard repairs. His work involves tank cleaning and use of air compression hoses, as well as hydraulic power tools.

The patient had the onset of numbness and tingling on or about 03-06-00. He was seen at the Industrial Clinic at St Mary Hospital and was advised that he probably had tendinitis. Initially, he was referred for evaluation by Dr Colleague who diagnosed carpal tunnel syndrome. The patient had a nerve conduction study and EMG on 07-14-00 that was positive for carpal tunnel syndrome in both hands. He has not been able to work since 03-20-00.

I diagnosed bilateral carpal tunnel syndrome and advised surgery. The patient, however, initially wished to try conservative management. He had difficulty with nonsurgical management and was unable to increase his activities. He, therefore, elected to go ahead with surgery, and this was done on 09-07-01, a right endoscopic carpal tunnel release. He felt that he experienced relief as a result of this treatment.

The patient underwent a left endoscopic carpal tunnel release on 07-15-02. Postoperatively, he had a fairly long course of occupational therapy and strengthening. At the time of my evaluation on 11-22-02, it is my opinion that he has maximized benefits from therapy and that most likely his condition has not changed in a reasonable period of time, and I felt he could be considered permanent and stationary for rating purposes.

Present Complaints

Mr Sample relates episodic pain in his bilateral hands and wrists. He feels that he has lost strength in his hands. He states that the numbness and tingling are gone, however, after his surgery.

Physical Examination

The patient has well-healed scars at his bilateral wrists at the flexor aspect from the endoscopic carpal tunnel releases. There is full range of motion of the elbows bilaterally. There is full range of motion of the wrists bilaterally. There is full range of motion of the thumbs bilaterally. There is full range of motion of the digits bilaterally. There is no thenar atrophy.

Grip strength was tested using the Jamar dynamometer set at the second notch with rapid exchange. Three measurements were obtained as follows: Right: 80, 90, and 80 lbs. Left: 80, 85, and 80 lbs. In my opinion, a full effort was provided by this right-hand-dominant, both-hand-injured patient. Estimated normal grip strength would be 120 lbs in the right dominant hand.

Diagnostic Impression

Right and left carpal tunnel syndrome, status post right and left endoscopic carpal tunnel release.

Permanent and Stationary Status

In my opinion, Mr Sample is permanent and stationary for rating purposes.

Subjective Factors of Disability

I would characterize his subjective factors of disability as occasional, slight pain at rest, increasing to intermittent, slight to moderate pain with repetitive gripping, grasping, pushing, and pulling with his bilateral hands.

Objective Factors of Disability

Objective factors of disability are decreased grip strength in the bilateral hands.

Permanent Disability

In my opinion, he has sustained permanent disability as a result of injury to his bilateral hands. I would characterize this disability as a loss of preinjury capacity for gripping and grasping with his bilateral hands estimated at 30%.

Reasonably, he would require preclusions from prolonged repetitive gripping bilaterally.

Report 2: Dr Later, April 21, 2003

This now 49-year-old, right-handed, 6'1", 170-lb laborer was reexamined in my Oakfield office for the second time on April 21, 2003. He had been previously seen last April. In the interim, he has undergone a left endoscopic carpal tunnel release. His grip strengths for me are improved but still diminished.

I do feel that he should be considered permanent and stationary at this time. His rating will be completed in accordance with the AMA *Guides to the Evaluation of Permanent Impairment, Fifth Edition*.

Review of Medical Records

There are handwritten PR-2 reports from Dr Colleague dated May 10, 2002, and May 31, 2002.
There is an operative report from the surgery center documenting the July 17, 2002, left carpal tunnel release using the Agee technique.

There are postoperative PR-2 reports dated August 2. There is a narrative report from Dr Prior dated November 26, 2002. He notes well-healed scars. His grip strength is measured as right (major) and left (minor): 80/80, 90/85, and 80/80. He does not perform an AMA rating but estimates that he has 30% of his grip and grip abilities.

Diagnosis

1. Bilateral carpal tunnel syndrome
2. Status post bilateral carpal tunnel releases

I would agree with Dr Prior that he should be considered permanent and stationary for the left arm as well as the right arm as of November 26, 2002, the date of Dr Prior's report.

His permanent impairment needs to be addressed via his grip strength loss. Looking at the AMA *Guides to the Evaluation of Permanent Impairment, Fifth Edition,* Tables 16-31 and 16-32 indicate a male average grip strength in manual laborers of 48.5 kg in the major hand and 44.6 kg in the minor hand. For someone his age, from Table 16-32, the numbers would be 49.0 kg in the major hand and 47.3 kg in the minor hand. However, please note that these are kilograms, and Dr and I have been using pounds. Translating this to pounds would yield 107.8 lbs on the right hand and 104 lbs on the left hand. By these calculations his grip strength would be somewhat more than 20% and significantly less than 30%.

Using Table 16-34, also on page 509, this would become a 10% upper extremity restriction. However, this would be considered a bilateral and not a unilateral restriction. Using the combined values table from page 604, a 10% impairment combined with a second 10% impairment becomes a 19% upper extremity impairment.

Then using Table 16-3 on page 439, a 19% impairment of the upper extremity becomes an 11% impairment of the whole person.

Questions

1. What are the problems associated with these reports?
2. How should this case be rated?

See page 9 for the solution.

Cervical Spine (continued)

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kinking), and longitudinal displacement (translation). Relatively increased motion may be identified at immediately adjacent levels if muscle spasm/guarding is present at the injured level(s). The increased motion identified at traumatically injured levels always occurs focally at the level(s) injured. A single-level injury is most common. Two adjacent-level injuries are very uncommonly identified, while more than 2-level ligamentous injuries are exceedingly rare.[4]

Objective Measurements of Abnormal/Increased Motion and Instability in the Cervical Spine

The standard radiographic evaluation of instability and ligamentous injury in the cervical spine consists of lateral flexion and extension x-ray views.[7] No single pattern of injury is identified in whiplash injuries. It is probably more appropriate to describe a whiplash mechanism of injury as consisting of hyperextension followed by hyperflexion motion. The majority of cervical spine injuries identified after a whiplash mechanism of injury are related to the flexion component,[5] particularly in rear-end collision accidents involving a headrest. Anterior subluxation is the injury caused most frequently by the flexion component of whiplash injury. Complete disruption of the posterior column ligaments (supraspinous ligament, interspinous ligament, facet joint capsule, and posterior longitudinal ligament) is classified as a grade III ligament sprain and results in focal anterior subluxation. Incomplete disruption of the posterior column ligaments (grade II sprain) may also produce increased motion, although gross instability will be absent. Disruption of the posterior annulus fibrosis and posterior disk herniation (middle column) may be associated. Compression fracture of the vertebral end plates and fractures of the spinous processes, lamina, and/or facet may be identified in severe whiplash mechanism injuries.

Hyperextension injuries (disruption of the anterior column ligaments, traumatic spondylolisthesis, lamina fractures, extension teardrop fractures, etc) are not commonly identified in low-impact rear-end collision accidents and are more commonly seen in more violent, high-velocity motor vehicle accidents without headrests. A variety of injuries have been described in cases where the head forcibly strikes the windshield or other solid element in the car, resulting in axial compression and rotation forces, and in cases where the individual is ejected from the motor vehicle. This discussion focuses on relatively low-velocity motor vehicle rear-end collisions that result in a typical whiplash mechanism of injury without associated fractures (pure musculoligamentous sprain/strain injuries).

The standard radiographic evaluation of patients with a history of whiplash mechanism of injury consists of an initial cross-table lateral portable x-ray. This view is augmented with a cross-table lateral "swimmer's" view if lower cervical and upper thoracic elements are not adequately visualized. These views are evaluated for fracture, dislocation, malalignment, and anterior subluxation. If results are negative, erect anteroposterior, lateral, and odontoid views are typically performed. In patients with significant symptoms and a history of significant velocity motor vehicle accident without neurological symptoms and with negative initial plain film evaluation, flexion and extension views are commonly performed. In some facilities, flexion and extension views are performed at the time of the initial evaluation of trauma[7] although in low impact collisions, patients frequently are released in

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Assessing Common Issues in Electrophysiologic Studies

by Mitchell K. Ross, MD

Electrodiagnosis is a quantitative electrophysiologic test that may identify and localize the site of neurologic dysfunction and detect subclinical changes and compensatory processes. Electrodiagnostic (EDX) studies useful in evaluation of the peripheral nervous system and spinal cord include electromyography (EMG), nerve conduction studies (NCSs), and somatosensory evoked potentials (SEPs).[1-3] The most common applications in impairment assessment are to evaluate radiculopathy, entrapment neuropathy, or other peripheral nerve injury.[4] The report of an EDX study should present results completely, concisely, and in a standardized format understandable to the nonelectromyographer (because it will likely be reviewed by other physicians and possibly laypersons, including adjusters, attorneys, hearing officers, and judges who may have varying degrees of knowledge about neurophysiology and clinical neurology). Since medicolegal decisions are often based on EDX results, the findings must be valid and reliable. The physician examiner should be familiar with the concepts of EDX testing and the role it plays in impairment assessment.

Any EDX report should contain the fundamentals: test findings and a summary of the findings.[5] The former include data and other observations as well as "local" neurophysiological interpretation (results obtained in the given structure). The summary includes general neurophysiological interpretation, conclusions, and (when appropriate) clinical correlation. The analysis should use only relevant findings on which the electromyographer based the conclusions. The narrative summary offers quantitative findings that may be as absolute as any radiological report. The basis for this narrative summary may include raw data (traces, numbers, and parameters such as amplitude duration) and statistical descriptions in relation to references. These can be helpful to the reader, especially if there is internal consistency with conclusions supported by the data. The conclusion may give the main message first: eg, "moderate subacute axonal sensory and motor polyneuropathy, slight conduction block of the ulnar nerve in the cubital tunnel," "severe axonal lesion of the suprascapular nerve," or "a slight axonal and demyelinating median nerve lesion at the wrist."

The report should make it clear when facts other than neurophysiological data are included in the comments. Demographic data, reason for referral, tabulation of data, and summary should all contain meaningful verbiage and interpretations. Decimal places should imply truly obtainable precision and no more. More data is generally preferable to less, and the data should be sufficient to support the conclusions. For instance, it would be inappropriate to conclude there is a left ulnar neuropathy at the wrist without stimulation of the ulnar nerve distal to that point and without recording electrical activity of the abductor pollicis brevis or the first dorsal interosseous (to check the integrity of the deep branch).

The general neurophysiological interpretation should include localization, severity, pathophysiology, time course, and distribution. Recording sites for the NCS should be indicated. The interpretation of data obtained for muscles and nerve conductions may be quantitative but should include only relevant findings on which the conclusion is based. A contralateral Hoffman-reflex (H-reflex) latency should be determined if an absent H reflex is being used diagnostically. The absence of a sensory nerve action potential (SNAP) should be considered abnormal.

When interpreting distal motor latencies, it is preferable to record the proximal conduction velocity (CV) of the nerve as well, although sometimes it is difficult because of muscle bulk. If so, that problem should be noted in the report. Spontaneous activity on needle EMG should not be regarded as normal. The distribution of findings will vary, depending on the stage and severity of the disease. The report should reflect an understanding of muscular innervation. For instance, fibrillation potentials from only one muscle should not lead to a diagnosis of radiculopathy or polymyositis. The electromyographer should try to localize the abnormality within the expected abnormal and normal findings in the differential diagnosis of the disorder.

There should be consistency within a report; otherwise, suspected technique error may compromise credibility of the study. For instance, normal median sensory conduc-

Electrodiagnostic Abbreviations

CMAP	compound muscle action potential
CV	conduction velocity
EDX	electrodiagnostic
EMG	electromyography
F wave	F wave (responses evoked from muscle as the result of antidromic activation by a peripheral electrical stimulus)
H reflex	Hoffman reflex
M wave	M wave (measurement of a motor nerve's response)
MUAP	motor unit action potential
NCS	nerve conduction study
SEP	somatosensory evoked potential
SNAP	sensory nerve action potential

tion and latency, motor latency, and compound muscle action potential (CMAP) amplitude but slowing of motor CV is a dissociation that should be addressed before diagnosis. Median nerve abnormality should be defined by testing an adjacent nerve such as the ulnar. If this is abnormal, then additional sensory NCSs are required. Both sides of the body should be tested to maximize the probability of detecting asymmetries. When a peripheral neuropathy is considered, proximal nerves should be studied. Temperature should be recorded since latencies will be slightly prolonged (with relatively normal CMAP amplitudes) in cold extremities. The number of nerve roots involved should be in the impression.

Errors of fact and technique, incomplete or irrelevant data, and over-, mis-, and underinterpretation of data are seen all too frequently in EMG reports. Anatomic errors include incorrect cord levels as well as imprecise localization of explored muscles. Because any EDX study has potential medicolegal ramifications, the electromyographer must spend the time to be rigorous. The *Guides* Chapter 13, The Central and Peripheral Nervous System; Section 13.1b, Description of Clinical Studies, states that "nerve conduction and needle electromyography (EMG) studies help to determine which nerves are involved and their anatomic location. Also evident will be whether sensory, motor, or both fibers are predominantly involved and whether axonal degeneration, demyelination, or a combination of both is present. Skillful differentiation of peripheral neuropathy and neuromuscular disorders may also be possible. Expert neuromuscular knowledge and understanding of pathologic manifestations of disease processes are necessary for the appropriate application and performance of these tests, particularly the EMG" (5th ed, 307-308).

The needle EMG remains the most sensitive electrophysiologic test for determining a radiculopathy. Denervation should be demonstrated in multiple muscles innervated by a single root but by different peripheral nerves. Fibrillations in paraspinal muscles are good evidence of a radiculopathy. However, their absence does not exclude radiculopathy, primarily because these muscles are only a short distance from the site of root injury and therefore have greater opportunity for reinnervation. This same concept explains why fibrillations more commonly remain in distal rather than in proximal muscles after chronic root injury.

The specific systematic approach to a given clinical problem depends on the training and experience of the clinician. The newly revised guidelines[6] include practice parameters for carpal tunnel syndrome (CTS),[7] ulnar neuropathy at the elbow,[8] and cervical radiculopathy. These guidelines provide the EDX choices in these disorders as well as recommendations on the appropriate muscles and nerves to be included in a study.

EDX studies are useful in objectively defining peripheral nerve injuries, entrapment neuropathies, and

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Carpal Tunnel Syndrome Practice Parameters

In 2002, practice parameters for EDX studies in CTS were jointly presented by the American Association of Electrodiagnostic Medicine, the American Academy of Neurology, and the American Academy of Physical Medicine and Rehabilitation.[9] In patients with suspected CTS, the following EDX studies are recommended as a standard, guideline, or option:

1. Perform a median sensory NCS across the wrist with a conduction distance of 13 to 14 cm. If the result is abnormal, compare the result of the median sensory NCS to the result of a sensory NCS of one other adjacent sensory nerve in the symptomatic limb [standard].
2. If the initial median sensory NCS across the wrist has a conduction distance greater than 8 cm and the result is normal, one of the following additional studies is recommended:
   a. Comparison of median sensory or mixed nerve conduction across the wrist over a short (7 to 8 cm) conduction distance with ulnar sensory nerve conduction across the wrist over the same short (7 to 8 cm) conduction distance [standard].
   b. Comparison of median sensory conduction across the wrist with radial or ulnar sensory conduction across the wrist in the same limb [standard].
   c. Comparison of median sensory or mixed nerve conduction through the carpal tunnel to sensory or mixed NCSs of proximal (forearm) or distal (digit) segments of the median nerve in the same limb [standard].
3. Motor NCS of the median nerve recording from the thenar muscle and of one other nerve in the symptomatic limb to include measurement of distal latency [guideline].
4. Supplementary NCS: Comparison of the median motor nerve distal latency (second lumbrical) to the ulnar motor nerve distal latency (second interosseus), median motor terminal latency index, median motor nerve conduction between wrist and palm, median motor nerve CMAP wrist-to-palm amplitude ratio and median SNAP wrist-to-palm amplitude ratio (to detect conduction block), and short segment (1 cm) incremental median sensory nerve conduction across the carpal tunnel [option].
5. Needle EMG of a sample of muscles innervated by the C5 to T1 spinal roots, including a thenar muscle innervated by the median nerve of the symptomatic limb [option].

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a soft collar with a presumed mild sprain/strain (grade I to II) diagnosis if the initial trauma cervical spine x-ray series is negative and are evaluated with flexion and extension views on initial follow-up evaluation if symptoms persist or worsen.

Flexion and extension views of the cervical spine are physiologic "stress views." Adequate flexion is typically defined as 60˚ to 65˚ of mandibular angle in relationship to the floor on erect flexion and extension views. Grade I sprain/strain ligamentous injuries will demonstrate no evidence of increased abnormal motion but are commonly associated with muscle spasm and guarding. Grade II and III sprain/strain ligamentous injuries will result in increased motion unless masked by muscle spasm or guarding. The radiographic signs of anterior subluxation injuries consist of:

- A localized hyperkyphotic angulation (kinking) at the level of injury
- Widening of the interspinous and interlaminar space (fanning)
- Displacement of the inferior articular facets of the subluxed vertebrae with respect to their contiguous subjacent facets
- Widening of the space between the subluxed vertebrae and the subjacent superior articular process
- Posterior widening and anterior narrowing of the involved intervertebral disk space, with or without anterior translation of the subluxed vertebrae[10]

Bohrer et al reviewed 150 consecutive neck injury patients who underwent evaluation of the cervical spine after review of initial radiographs demonstrated no apparent unstable injury or fracture.[9] This study evaluated the adequacy of flexion and the presence of significant flexion angles (3˚ or more) and compared those results with clinical examination and follow-up clinical evaluations. Four different flexion patterns were identified: 21% demonstrated 3 or more significant flexion angles; 29%, 2 significant flexion angles; 25%, 1 significant flexion angle; and 25%, no significant flexion angles. The no-flexion-angle pattern was consistent with muscle spasm and/or guarding and was not considered an adequate stress film to exclude an underlying unstable ligamentous injury. Follow-up flexion and extension studies were recommended for this group. The *single-flexion-angle* pattern was most consistent with a grade II to III ligamentous injury if the single angle was *greater* than 11.5˚. If the single angle was less than 11˚, the pattern was felt to be consistent with a grade I or a mild grade II injury. Significantly greater pain and tenderness were present in this group compared to the 2- and 3-angle groups. Disk degeneration can also produce a single-angle pattern. The 2-angle pattern was most commonly visualized at adjacent vertebrae, typically around C4, C5, and C6, which are the levels that normally demonstrate maximum motion. Dr Bohrer noted that a 2-angle pattern is also identified around a single vertebra in asymptomatic normal individuals with an incomplete flexion effort. Normal clinical examinations and less tenderness were noted in the 2-angle group than in the 1- or no-angle groups. Bohrer suggested that the 2-angle group represents mild grade I sprain/strain injuries. Three or more flexion angles were considered a normal pattern and had the highest percentage of normal clinical examinations, good flexion attempts, and the least tenderness of any of the groups studied. If symptomatic, the 3-or-more flexion angle group appeared to have a minor grade I sprain/strain injury.

Griffiths et al reviewed 40 patients with clinically proven whiplash mechanism injuries and compared the flexion and extension radiographs to 105 normal control patients who were age-matched and had no history of neck injury. Kinking was evaluated only on flexion films by placing a line down the posterior margin of the vertebral body and measuring the angles produced between 2 adjacent vertebral levels. Fanning was measured by using a line drawn through the middle of the spinous processes and measuring the perpendicular distance between the tip of the spinous process above and the level below. Fanning was defined as the difference between this distance on flexion and extension views. Localized kinking greater than 10˚ (and at least 2˚ greater than the adjacent levels) and fanning greater than 12 mm (and at least 2 mm greater than the adjacent levels) predictably differentiated the patients with whiplash mechanism injuries from asymptomatic controls with 80% accuracy.

The AMA *Guides* defines the loss of motion segment integrity in the cervical spine as translation (anteroposterior motion or slipping of 1 vertebral body over another) greater than 3.5 mm, or a difference in angular motion of 2 adjacent motion segments greater than 11˚ in response to spine flexion and extension. The methodology of measuring abnormal translation and angular motion is based on the method described by Posner, White, et al.[4] Flexion and extension views must be obtained with standard radiographic technique, which implies a 72-inch source-to-image distance; 10% to 15% magnification is produced if the films are obtained with 40-inch source-to-image distance. The focal spot of the unit obtaining the films is not specified but will also affect magnification. It is assumed that the loss of motion segment integrity translation measurements will be focally abnormal and significantly greater than adjacent levels.

Evaluation of cervical spine motion using fluoroscopy or cineradiographic techniques has been described in the literature as being more sensitive in evaluating subtle abnormal motion in the cervical spine. Measurements of abnormal translation complying with the AMA *Guides* are not possible using current video fluoroscopic or cin-

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Case Study Solution: Carpal Tunnel Syndrome

Neither of these reports met standards defined in the *Guides*, and both ratings assumed Mr Sample was at maximum medical improvement (MMI) without support of this assumption. The evaluation by Dr Prior on November 26, 2002, was performed less than 5 months from the left carpal tunnel release (July 15, 2002). Mr Sample was probably not yet at MMI; therefore, it was inappropriate to rate permanent impairment. MMI may not be achieved until at least several months after carpal tunnel release, and grip strength may improve for up to 24 months postoperatively.[1-2] Hence, if symptoms and functional difficulties persist, it is appropriate to wait until the condition reaches a plateau before performing an impairment rating. This may be up to several months.[3]

Both histories were inadequate. Section 2.6, Preparing Reports (5th ed, 21), instructs an evaluator to "include a narrative history of the medical condition(s) with the onset and course of the condition, symptoms, findings on previous examinations(s), treatments, and responses to treatment, including adverse effects. Include information that may be relevant to onset, such as occupational exposure or injury. Historical information should refer to any relevant investigations. Include a detailed list of prior evaluations in the clinical data section.... Assess current clinical status, including current symptoms, review of symptoms."

Dr Prior's report did not list all pertinent physical examination findings. Noticeably absent are results of any sensory testing, which should include light touch, two-point discrimination, and perhaps monofilament testing. Motor examination was incomplete, consisting solely of grip strength testing (plus notation of thenar atrophy). Dr Later stated that he reexamined Mr Sample but listed no physical findings apart from "grip strengths for me are improved." The reader does not know if the grip strength testing was instrumented or what, if any, other testing was performed.

The clinical discussion was inadequate. The *Guides* advises examiners to "discuss diagnoses, impairment... Include a discussion of the anticipated clinical course and whether further medical treatment is required. Describe the residual function and the impact of the medical impairment(s) on the ability to perform activities of daily living and, if requested, complex activities such as work. List the types of affected activities (see Table 1-2). Identify any medical consequences for performing activities of daily living" (5th ed, 22). The reports did not appropriately explain how the rating was performed, eg, it should have included a thoughtful discussion of the rating process with reference to specific criteria: tables, figures, and page numbers in accordance with processes defined in the *Guides*. The Fifth Edition instructs one to "compare the medical findings with the impairment criteria listed within the *Guides* and calculate the appropriate impairment rating. Discuss how specific findings relate to and compare with the criteria described in the applicable *Guides* chapter. Refer to and explain the absence of any pertinent data and how the physician determined the impairment rating with limited data. Include an explanation of each impairment value with reference to the applicable criteria of the *Guides*" (5th ed, 22).

Although it was premature to rate permanent impairment, it is appropriate to review the process. Carpal tunnel syndrome impairment assessment is discussed in Section 16.5d, Entrapment/Compression Neuropathy (5th ed, 491-495), specifically on page 495. The *Guides* notes that "only individuals with an objectively verifiable diagnosis should qualify for a permanent impairment rating. The diagnosis is made not only on believable symptoms but, more importantly, on the presence of positive clinical findings and loss of function. The diagnosis should be documented by electromyography as well as sensory and motor nerve conduction studies" (5th ed, 493). "The sensory deficits or pain, and/or the motor deficits and loss of power, are evaluated according to the impairment determinations method described in Section 16.5b. In compression neuropathies, additional impairment values are not given for decreased grip strength"(5th ed, 494).

The Fifth Edition states on page 495:

> If, after an optimal recovery time following surgical decompression, an individual continues to complain of pain, paresthesias, and/or difficulties in performing certain activities, three possible scenarios can be present:
>
> 1. Positive clinical findings of median nerve dysfunction and electrical conduction delay(s): the impairment due to residual CTS is rated according to the sensory and/or motor deficits as described earlier.
> 2. Normal sensibility and opposition strength with abnormal sensory and/or motor latencies or abnormal EMG testing of the thenar muscles: a residual CTS is still present, and an impairment rating not to exceed 5% of the upper extremity may be justified.
> 3. Normal sensibility (two-point discrimination and Semmes-Weinstein monofilament testing), opposition strength, and nerve conduction studies: there is no objective basis for an impairment rating.)

According to Table 16-15, Maximum Upper Extremity Impairment Due to Unilateral Sensory or Motor Deficits or to Combined 100% Deficits of the Major Peripheral Nerves (5th ed, 492), the maximum upper extremity impairment for sensory and motor deficits of median nerve below midforearm are 39% and 10%, respectively. Sensory deficits are graded by Table 16-10, Determining Impairment of the Upper Extremity Due to Sensory Deficits or Pain Resulting from Peripheral Nerve Deficits (5th ed, 482). Dr Prior's November 26, 2002, report states that "the numbness and tingling are gone." Neither report in this case lists abnormal sensory findings. Hence, Mr Sample probably has no sensory

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impairment; however, this should be confirmed by an appropriate sensory examination.

Motor deficits are graded by Table 16-11, Determining Impairment of the Upper Extremity Due to Motor and Loss-of-Power Deficits Resulting from Peripheral Nerve Disorders Based on Individual Muscle Rating (5th ed, 484). Although Mr Sample reportedly had weakness of grip, the strength of muscles innervated by median nerve below midforearm was not documented.

Because most weakness falls in grade 4, some examiners choose to subdivide it into 4-, 4, and 4+. The next step is to select a percentage motor deficit, sometimes nicknamed a multiplier, from the right column of Table 16-11. For grade 4 weakness the range of motor deficits is 1% to 25%. The *Guides* states that interpolation is used when "deciding where to place an individual's impairment rating within a range" (5th ed, 20). More specifically, "impairment values for*...measurements falling between those shown in a...chart may be adjusted or interpolated proportionally* in the corresponding interval" (5th ed, 461). Example 16-58 (5th ed, 487-488) implies percentage motor and sensory deficits should be "selected on clinical judgment." A more specific and defensible means to select from within a range of percentage motor deficits becomes available if the examiner is able to further categorize the weakness within a grade. Grade 4-, 4, and 4+ would correspond to the low end, midportion, and high end of the range of 1% to 25%, respectively. The evaluator also determines the impact of the impairment on the ability to perform activities of daily living (ADL) and uses that information to estimate where the individual stands within that class (5th ed, 4-5).

If Mr Sample had grade 4 weakness in median innervated muscles—which affected some ADL such as tactile feeling and grasping (5th ed, 4)—it would be appropriate to select the midportion of the range: 13%. This motor deficit would be multiplied by the maximum upper extremity impairment for motor deficit of median nerve below midforearm: 10% x 13% = 1.3%, rounded to 1% upper extremity impairment.

However, it is inappropriate to rate Mr Sample based on weakness of grip strength. The *Guides* discusses strength evaluation and its limited role in impairment evaluation in Section 16.8, Strength Evaluation (5th ed, 507):

> Because strength measurements are functional tests influenced by subjective factors that are difficult to control, and the *Guides* for the most part is based on anatomic impairment, the *Guides* does not assign a large role to such measurements. Those who have contributed to the *Guides* believe further research is needed before loss of grip and pinch strength is given a larger role in impairment evaluation.

Furthermore, Section 16.8b, Grip and Pinch Strength, emphasizes the importance of obtaining reliable measurements with less than 20% variation. Measurements are "usually repeated three times with each hand at different times during the examination." This section describes 2 techniques to determine if maximum effort was used, plotting measurements obtained at each the 5 handle settings of a dynamometer to determine if a bell-shaped curve was obtained, and comparing readings to the rapid exchange grip technique. Dr Prior reportedly performed 3 measurements "with rapid exchange." Unclear is what Dr Later did. Regardless, Section 16.5d, Entrapment/Compression Neuropathy, states that "in compression neuropathies, additional impairment values are not given for decreased grip strength" (5th ed, 494). Hence, grip strength cannot be used directly to rate impairment due to carpal tunnel syndrome. It has been used by some examiners to assist in assigning a grade of muscle function; however, grip strength is determined by the neuromusculoskeletal capacity of digits, hands, wrist, forearm, and elbow, as well as psychological factors. Therefore, grip strength reflects multiple factors and is not a direct reflection of median nerve innervated muscle function.

Assuming no ratable sensory deficits and grade 4 weakness of median nerve innervated muscles, Mr Sample's rating would be 1% upper extremity impairment, which converts to 1% whole person permanent impairment per Table 16-3 (5th ed, 438). If the weakness was bilaterally symmetrical, the combined total would be 2% whole person permanent impairment.

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Cervical Spine (continued)

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eradiographic techniques. Relative areas of increased motion can be identified with these techniques. The increased motion identified must then be evaluated to determine whether it represents normal physiologic increased motion, normal compensatory motion, increased motion related to underlying degenerative disk and/or facet disease, or increased motion related to ligamentous injury.

Summary

Alteration of motion segment integrity due to loss of motion segment integrity (increased translation or angular motion) as defined in the AMA *Guides* is rare. The most common cause of alteration is due to decreased motion resulting from surgical fusion. Flexion and extension x-rays are indicated only if the physician suspects motion segment alteration from history or findings on routine x-rays. Physicians reviewing these x-rays need to ensure that the studies are performed and interpreted as instructed in the *Guides*.

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Date		Activity	Location	Organization
Oct	22	How to Write Winning Reports	Chicago, IL	ABIME
	22–23	Understanding the AMA Guides: A Critical Approach	Los Angeles, CA	CAAA/ AADEP
	23–24	ABIME Certification Review and AMA Guides Fifth Edition Training Course	Chicago, IL	ABIME
	25	ABIME Advanced Topics	Chicago, IL	ABIME
Nov	3	AMA Guides-Fifth Edition: The Basics	Oakland, CA	COA/AAOS/ AADEP
	4	AMA Guides-Fifth Edition: The Basics	Irvine, CA	COA
	4–5	IME Summit	Orlando, FL	SEAK
	4–5	AMA Guides in Workers' Compensation	Anaheim, CA	AMA
	8–9	AMA Guides in Workers' Compensation	South San Francisco, CA	AMA
	10	Advanced Skills Development	Miami, FL	AADEP
	19–21	Survival Guides to Impairment Evaluations Schedule	San Francisco, CA	AAEME
	11–13	AADEP Annual Scientific Session and Business Meeting	Miami, FL	AADEP
Dec	4–5	A New Approach to the AMA Guides Training Course, Fifth Edition	Cleveland, OH	AADEP

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Clinical Update (continued)

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radiculopathies. Therefore, it is imperative that these studies are performed by physicians with adequate training and are interpreted correctly. The findings must be clearly presented with supportable conclusions.

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