# Evaluation of Nerve Injury and Nerve Compression in the Upper Quadrant

**Christine B. Novak, PT, MS**
*Division of Plastic and Reconstructive Surgery*
*Program in Occupational Therapy and Program in Physical Therapy*
*Washington University School of Medicine*
*St. Louis, Missouri*

**Susan E. Mackinnon, MD**
*Division of Plastic and Reconstructive Surgery*
*Washington University School of Medicine*
*St. Louis, Missouri*

**ABSTRACT**: Evaluation of the patient with nerve compression and/or nerve injury should include a complete motor and sensory evaluation to establish the level and degree of injury and/or compression. No one test has been accepted as the standard procedure for the evaluation of sensibility. The various sensory tests available for patient assessment will yield different information regarding the integrity of the quickly and slowly adapting sensory receptors. Tests such as provocative maneuvers and sensory thresholds (cutaneous and vibration) will be more sensitive in the evaluation of patients with nerve compression, and other discriminatory measures will yield better functional information in patients with nerve injury.

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Nerve injury and nerve compression may result in decreased sensory and/or motor function. Evaluation of the patient with nerve compression or nerve injury will provide information to identify the level of the lesion (injury or compression) and to document alterations in motor and/or sensory function.

## CHRONIC NERVE COMPRESSION

Patients with chronic nerve compression have wide variability in the presenting subjective symptoms and physical signs. In the motor system, these changes may progress from muscle ache and weakness to muscle atrophy. Sensory complaints will vary from intermittent paraesthesia to constant numbness. This spectrum of patient presentation likely relates to the range of neural histopathologic changes that occur with chronic nerve compression (Figure 1). Documentation of chronic histopathologic changes in humans is uncommon and therefore the animal model has assisted in the understanding of the changes that occur with chronic nerve compression.[1–4] The histopathology of chronic nerve compression is a continuum of neural changes that occur dependent upon the amount and duration of compression. The initial neural changes involve a breakdown of the perineurial blood–nerve barrier with subperineurial edema, followed by connective tissue thickening, segmental demyelination, diffuse demyelination, and finally axonal degeneration.[5] Initially, these neural changes do not occur equally across the nerve but may vary depending on the distribution of compression across the nerve. In general, the fascicles that are most superficial are affected sooner, and when this occurs varying patient symptoms within a single nerve distribution may result.[6]

## MULTIPLE AND DOUBLE CRUSH SYNDROMES

Upton and McComas[7] presented the concept of the double crush mechanism where a proximal level of nerve compression will cause the distal entrapment sites to be less tolerant to compressive forces. The authors presented a clinical patient review with a high prevalence of distal nerve compression with cervical root lesions.[7] Therefore, they concluded that the summation of neural compression may alter axoplasmic flow thus contributing to patient symptoms. Lundborg[6] introduced the concept of the reverse double crush where a distal compressive site will alter neural transmission, thus affecting the more proximal entrapment sites. The concept of the double or multiple crush should be considered in cases of nerve compression. Multiple sites of compression may cumulatively cause alteration in the neural transmission and produce patient symptoms, although each site in isolation may not be sufficient to cause patient symptoms (Figure 2).

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Correspondence and reprint requests to Christine B. Novak, PT, MS, Division of Plastic and Reconstructive Surgery, Washington University School of Medicine, 660 S. Euclid Avenue, St. Louis, MO 63110; e-mail: <novakc@wustl.edu>.

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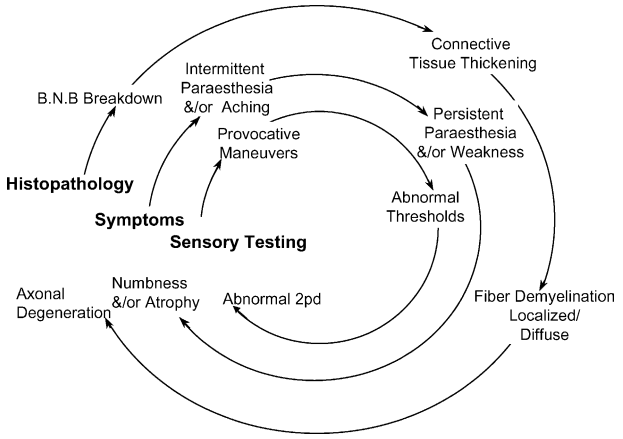


***FIGURE 1.*** *The histopathology of chronic nerve compression spans a broad spectrum. Patient presentation and clinical findings will likely parallel the histopathologic changes occurring in the nerve. In the earliest stages of nerve compression, the patient may present with only intermittent symptoms and provocative maneuvers (positional and pressure) may be the only positive findings. In the later stages of nerve compression with Wallerian degeneration, patient complaints will include numbness and muscle atrophy. Sensory testing at this stage may reveal abnormal two-point discrimination. (Reproduced from Novak CB. Patient evaluation of nerve compression in the upper limb. In: Allieu Y, Mackinnon SE (ed). Nerve Compression Syndromes of the Upper Limb. London: Martin Dunitz; 2002, with permission.)*

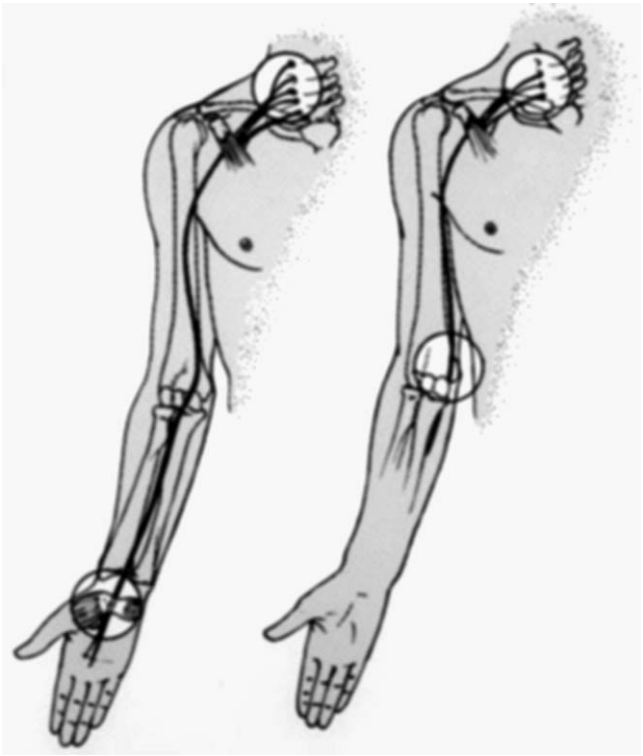

***FIGURE 2.*** *Several sites of nerve compression in the same extremity can cumulatively produce symptoms, such as illustrated here with compression at the cervical spine and distally at the cubital tunnel and carpal tunnel producing sensory disturbances in both the median and ulnar nerve sensory distributions. In the case of double crush, compression of several sites can cumulatively produce alteration of sensation, but each isolated site may be insufficient to produce patient symptoms. (Reproduced from Mackinnon SE. Double and multiple crush and entrapment syndromes and compression nerve syndromes of the upper extremity. Hand Clin North Am. 1992;8: 369–90, with permission.)*

The postures and positions that contribute to multiple levels of nerve compression may also contribute to muscle imbalance, which may further compress nerves (Figure 3).[8]

## NERVE INJURY

Disruption of nerve continuity and neural transmission will result in a number of alterations, not only at the site of the injury, but also at the proximal and distal nerve segment, the distal sensory/motor end organ, the cell body, and the central cortex.

In 1943, Seddon[9] introduced a classification system for nerve injury that included three levels: neurapraxia, axonotmesis, and neurotmesis. Sunderland[10] expanded the classification of nerve injury to five degrees of nerve injury (I–V). A first degree injury is comparable to a neurapraxia. It is a demyelination of the nerve resulting in a temporary conduction block. There will not be any axonal degeneration/regeneration. With remyelination of the nerve, the presenting motor and sensory dysfunction will be resolved. Complete recovery usually occurs by 12 weeks after injury. A second-degree injury is more severe and it is comparable to an axonotmesis as described by Seddon. Wallerian degeneration and proximal axonal degeneration will occur; however, the endoneurial tubes remain intact. Electrodiagnostic studies will be positive and muscle changes will be evident with electromyography. Neural regeneration will occur at the rate of 1 mm per day or 1 inch per month and may be assessed with an advancing Tinel's sign. Recovery will be complete, provided that reinnervation occurs in a timely fashion before muscle degeneration. A third-degree injury is more severe. The nerve will undergo Wallerian and proximal axonal degeneration, similar to a second-degree injury except that the endoneurial tubes are not intact. Therefore, when axonal regeneration occurs, the regenerating axons may not return to reinnervate their original end organs. Patients with third-degree injuries and mismatching of the regenerating axons will benefit most from motor and sensory reeducation to maximize functional outcome. A fourth-degree injury is a neuroma-in-continuity. There will be a proximal Tinel's sign, but it will not advance beyond the neuroma. These patients require surgical intervention to excise the neuroma and subsequent nerve coaptation. The injury will then regenerate as described with a third degree injury. A fifth-degree injury is complete nerve

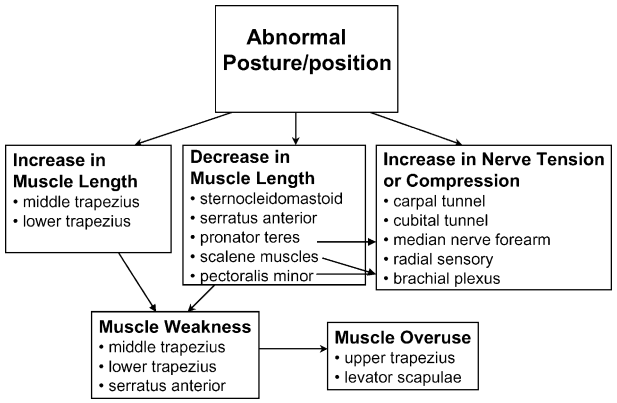


***FIGURE 3.*** *Certain postures and positions may cause increased pressure or tension on nerves contributing to nerve compression (wrist flexion/extension, elbow flexion, arm elevation). These positions will also place muscles in elongated or shortened positions, which will contribute to muscle imbalance, particularly in the cervicoscapular region. If the shortened muscles cross over a nerve, then more compression may be placed on that nerve; i.e., the pronator teres muscle compressing the median nerve in the forearm, the pectoralis minor or scalene muscles compressing the brachial plexus. (Reproduced from Novak CB, Mackinnon SE. Thoracic outlet syndrome. Orthop Clin North Am. 1996;27:747–62, with permission.)*

transection and will require surgery to establish nerve continuity. After surgery, nerve regeneration will occur at the rate of 1 mm per day and can be monitored with an advancing Tinel's sign. A sixth-degree nerve injury is a term used for a mixed nerve injury where varying degrees of nerve injury occur within the same nerve.[11]

In Sunderland degree II, III, IV, and V injuries, axonal degeneration will occur proximal and distal to the site of nerve injury.[11] The distance of proximal degeneration will vary dependent on the nature of the injury, but will extend to the proximal node of Ranvier. In very traumatic nerve injuries, the proximal degeneration may extend more proximally than the next node of Ranvier and may result in cell body death. At the site of nerve injury, axonal sprouting may occur within 24 hours after injury. Each single axon will produce multiple regenerating units, and with correct contact with a distal sensory and motor end organ, the unit will remain viable.

Good motor and sensory function after nerve injury depends on the reinnervation of the motor end plates and sensory receptors.[11–14] Recovery of motor function requires a critical number of motor axons to reinnervate the muscle fibers.[13] Because prolonged denervation of the neuromuscular junction may preclude reinnervation of the muscle, the motor axons must reach the target muscle within a critical period. The exact period for motor fiber reinnervation is unknown, but the critical period is shorter for transection nerve injuries (Sunderland degree V injury) compared with axonotmetic nerve injuries (Sunderland degree II and III injuries).[11] Sensory recovery, however, is possible for many years after nerve injury, but the quality of the recovery decreases with long delays in reinnervation.[11]

## EVALUATION

### Motor Evaluation

Muscle strength can be assessed qualitatively or quantitatively. Initial changes with chronic nerve compression will include muscle aching followed by weakness and finally muscle atrophy. However, alteration in muscle function may not be detected in patients with mild nerve compression. Muscle atrophy, if present, can be graded by visual assessment as mild, moderate, or severe. This will only occur in cases of traumatic injury to a motor nerve or with severe nerve compression.

There are several grading systems that have been described to classify muscle strength. First published in 1943, the British Medical Research Council (MRC) grading system[15] was presented to assess muscle strength on a scale from 0 to 5, where 0 = no muscle contraction, 1 = a flicker of contraction, 2 = movement with gravity eliminated, 3 = full range of motion against gravity, 4 = full range of motion with resistance, and 5 = normal muscle strength. Birch et al.[12] describe another MRC grading system proposed by Highet that includes grades from M0 to M5, where M0 = no contraction, M1 = visible contraction in proximal muscles, M2 = visible contraction in proximal and distal muscles, M3 = all important muscles both proximally and distally contract against resistance, M4 = return of function that all synergistic and independent movements are possible, and M5 = complete recovery. Kline and Hudson[16] called this system Grading of the Entire Nerve. Another grading system of the entire nerve (American System) that includes grade M0 to M6 was then described.[16] The use of multiple grading systems can result in confusion in the reporting of postoperative results. If the MRC grading system or another motor function grading system is used, it is important to identify the muscle grading system used and to follow the described system to ensure consistency in patient comparison.

After a complete nerve injury, the patient will have immediate loss of function of those muscles innervated by the nerve. However, it may take several weeks for muscle atrophy to be visible, and this will vary with the type of muscle involved, with faster atrophy in type I muscles than in type II muscles. This may complicate patient examination immediately after a nerve injury when other uninjured muscles may provide a specific muscle action for the denervated muscle (i.e., shoulder abduction with supraspinatus for deltoid, elbow flexion with

brachioradialis for biceps). Resources such as the Medical Research Council handbook[15] can serve as an excellent reference for physical examination of individual muscle function.

A number of dynamometers have been described to quantify muscle strength.[17–21] Pinch and grip strength is commonly measured with closed hydraulic systems such as the B & L pinch gauge (B & L Engineering, Santa Fe, CA), Preston pinch gauge (Sammons Preston, Bolingbrook, IL) and Jamar pinch and grip dynamometer (Asimow Engineering Company, Los Angeles, CA). Reliability of measurement with these instruments has been demonstrated by taking the mean value of three measurements.[20] With five grip-handle positions on the Jamar, it is necessary to ensure that the same handle position is used for subsequent patient measures. Although grip strength is a common measure used in the evaluation of hand function, it will not yield information on a specific muscle or muscles. For example, grip strength weakness may result from weakness of the finger flexor or wrist extensor muscles. Also strength evaluation will not be very sensitive to the small changes that occur in the early stages of nerve compression. Weakness may not be evident until the nerve has undergone considerable degeneration from chronic nerve compression.

The Rapid Exchange Grip was described to identify patients giving submaximal effort with grip strength measurement.[22] However, with rapid alternating grip of the Jamar dynamometer, it has been reported that this test is not effective in detecting patients that are giving submaximal effort.[23,24] The simultaneous grip test uses one Jamar dynamometer in each hand and the patient grips both dynamometers simultaneously.[25] The authors reported good sensitivity and specificity with the simultaneous grip test, and noted that patients who did not give maximal effort also did not follow the test protocol.[25] To test simultaneous grip strength, the patient is instructed to hold one grip dynamometer in each hand and squeeze the dynamometers simultaneously, maximally, and quickly under the direction of the examiner for ten to 15 repetitions. Comparisons are made to the maximum static grip strength measured in both hands. In the patient who is giving submaximal effort, there will be increased grip strength measured in the affected hand or decreased grip strength noted in the unaffected hand. We have found measurement of simultaneous grip strength to be very useful in assessing maximal grip strength and degree of effort.

## Provocative Testing for Chronic Nerve Compression

In the early stages of nerve compression, provocation testing may be the only positive finding (Figure 1). Clinical evaluation for carpal tunnel syndrome using Tinel's sign, Phalen's test and other provocation tests have been well described and this concept of increasing nerve tension and compression to provoke symptoms may be extrapolated to other sites of nerve compression.[11,26–37] The importance of the double crush mechanism cannot be under estimated in evaluating patients with suspected nerve compression. Because compression at the proximal sites may affect the more distal sites and vice versa, all entrapment sites in the upper extremity should be evaluated for nerve compression. If all sites of nerve compression are not identified, treatment at only a single site in patients with multiple levels of nerve compression will be unsuccessful in eliminating all symptoms.

Tests of provocation using movement, pressure, and the Tinel's sign can be used to identify the sites of nerve compression in the upper extremity.[31–34,37–40] Evaluation of the more proximal entrapment sites may produce patient discomfort, and therefore testing should begin at the most distal entrapment sites and progress proximally. A Tinel's sign is performed at each entrapment site by applying four to six digital taps, and is considered positive with reproduction of patient symptoms in the appropriate neural distribution. At the carpal tunnel, the examiner applies digital taps just proximal to the carpal canal on the median nerve. To assess the median nerve in the forearm, the nerve is tapped in the region of the pronator teres. At the cubital tunnel, a Tinel's sign is assessed along the course of the ulnar nerve beginning proximal to the cubital tunnel and progressing distal through the cubital tunnel. At the brachial plexus, the Tinel's sign is applied supraclavicularly between the scalene muscles and is positive with radiation of symptoms into the upper extremity. Many patients may have local tenderness to the scalenes and this should be noted but not recorded as a positive Tinel's sign.

Positional and pressure provocative tests are held for a total of one minute and recorded as positive if there is alteration of sensation in the correct neural distribution. Pressure provocative maneuvers should be performed by placing digital pressure at each entrapment site including the carpal tunnel (median nerve proximal to the wrist crease), median nerve in the forearm (level of the pronator teres), cubital tunnel (ulnar nerve proximal to the cubital tunnel), and brachial plexus (supraclavicular between the scalene muscles).[31–34,37,38,40] Positional maneuvers should include carpal tunnel (wrist flexion or extension), cubital tunnel (elbow flexion), and brachial plexus (arm elevation).[31–34,37–40] Maximal provocation to the nerve can be performed by combining tension and digital pressure on the nerve (Table 1).

Cervical nerve root impingement can be clinically assessed with a Spurling's test.[41] The foraminal nerve encroachment test is performed by placing the

*TABLE 1.* **Provocative Tests for Nerve Compression in the Upper Extremity**

| *Nerve* | *Site of Entrapment* | *Provocative Test* |
|---|---|---|
| Brachial plexus | Supra/infraclavicular | Arm elevation<br>Pressure on the brachial plexus between the scalene muscles |
| Radial nerve | Distal forearm | Forearm pronation with wrist ulnar deviation<br>Pressure over tendinous junction of extensor carpi radialis and brachioradialis |
| Ulnar nerve | Cubital tunnel | Elbow flexion and pressure on ulnar nerve at cubital tunnel region |
| | Guyon's canal | Pressure at Guyon's canal |
| Median nerve | Proximal forearm | Forearm supination with pressure in region of the pronator teres |
| | Carpal tunnel | Wrist flexion and/or extension with pressure proximal to carpal tunnel |

patient's head in slight cervical extension and side flexion. Axial compression is then applied to the patient's head, and a positive response is noted when there is a ''spray'' of symptoms into the arm. The test should be repeated with cervical side flexion to the opposite side.

With a positive response to any provocation, the patient should be permitted time for the symptoms to cease before testing the next entrapment site. These provocation tests will allow identification of all entrapment sites, which are compressing the nerve(s) and potentially contributing to the patient's symptomatology. These tests, however, will not provide quantification of the neural changes or intensity of patient symptoms.

## Sensory Testing

Many instruments and assessment tools have been described for the evaluation of sensibility with little consensus on the standard procedure.[42–60] There is no one test that will be optimal in terms of sensitivity, specificity, and predictive values in all stages of nerve compression and nerve injury. Different sensory tests evaluate various parameters of nerve function. Therefore some assessment tools will be more useful in the varying stages of nerve compression or with nerve injury.

Sensory testing evaluates the different responses of the quickly and slowly adapting sensory receptors. Four sensory receptors have been described in the glabrous skin of the hand, and these receptors have been classified by receptive field and response qualities. Both quickly and slowly adapting receptors have been identified in human glabrous skin. The slowly adapting receptors (Merkel cell neurite complex and Ruffini end organ) respond to static touch. The Merkel cell neurite complexes are found in the basal layer of the epidermis. The Ruffini end organs have been identified electrophysiologically but not histologically in the glabrous skin. The quickly adapting receptors (Meissner and Pacinian corpuscles) respond to moving touch and the discharge impulses vary dependent on the stimulus frequency. The Meissner corpuscles are most sensitive to frequencies up to 30 Hertz and the Pacinian corpuscles respond to the higher frequencies.

Threshold refers to the minimum stimulus necessary to elicit a response. It can be assessed with vibration thresholds (quickly adapting receptors) and cutaneous pressure thresholds (slowly adapting receptors). Innervation density represents the number of innervated sensory receptors and can be assessed with two-point discrimination (2pd). Threshold testing of the sensory receptors will permit the earliest quantification of changes occurring with chronic nerve compression.[32,44,49,52,53,61–65] Alterations in the innervation density will not occur until the later stages of chronic nerve compression and measures of two-point discrimination will remain normal until the patient has more severe nerve compression.[11,32,66,67]

## Light Moving Touch

Evaluation of light moving touch can provide a quick screening of the large A-Beta fibers. This can be performed using the Ten Test.[60] The light moving touch test allows the patient to compare their sensation on the affected limb on a scale (from 0–10) to normal sensation on the contralateral limb. Strauch et al.[60] compared the Ten Test to Semmes-Weinstein monofilament testing and reported good validity and reliability. Patel and Bassini[68] compared the Ten Test to the Weinstein Enhanced Sensory Test and 2pd and reported the Ten Test to be the most sensitive test in patients with carpal tunnel syndrome. To perform the Ten Test, a moving light touch stimulus is applied with the examiner's finger to a normal area of sensation on the unaffected contralateral digit. This is to be ranked as normal sensation at 10/10. Then a similar stimulus is applied simultaneously to the digit to be tested and the patient is asked to assess the sensation on a scale from 0 to 10, with 0 = no sensation and 10 = perfect sensation.

## Vibration Thresholds

Vibration thresholds can be used to assess the quickly adapting receptor threshold and may be evaluated either qualitatively or quantitatively. Qualitatively, vibration thresholds may be evaluated with a tuning fork. A low-frequency tuning fork (30 cps) will be most useful in documenting return

of sensation in patients with nerve injuries. It is one of the first indications of reinnervation of the sensory receptors.[45] The tuning fork is applied to the area to be assessed and the patient indicates if the stimulus is felt. A positive response indicates reinnervation of the low-frequency quickly adapting receptors.

However, with chronic nerve compression, it is hypothesized that the high frequency quickly adapting receptors are first affected.[53] Therefore, assessment with a low frequency tuning fork will not be useful to detect nerve compression and assessment with a high-frequency tuning fork (i.e., 256 cps) will be more sensitive to neural function changes in the earlier stages of nerve compression. The tuning fork is applied to the digit pulp and a comparison is made to the contralateral area. The patient reports if the stimulus is more intense, less intense or the same. This test, however, requires subjective assessment by the patient regarding the intensity of the stimulus, and because the stimuli are not applied simultaneously, the patient must recall the previously applied vibration for comparison. Application of the tuning fork stimulus may vary with alteration of examiner technique. Therefore, accurate patient comparison requires that the examiner apply the same stimulus force each time and variations of application force have been reported.[69] This test may not be used in patients with bilateral hand symptoms.

Quantification of the vibration threshold may be assessed with a vibrometer and a number of vibrometers have been described.[45,46,48,49,52,53,63] The Vibratron II (Physitemp, Clifton, NJ) is a fixed-frequency (120 Hz) variable-amplitude device used to evaluate the minimal vibration stimulus necessary to elicit a response.[48,57] This fixed-frequency vibrometer has a non–force-sensitive transducer on which the patient places his or her digit and indicates when the vibration is felt. Through a method of limits and force choice methodology, good reliability has been reported with this vibrometer.[57] The greatest limitation with the Vibratron II is that the vibration threshold is assessed only at a single frequency. If the higher frequencies are abnormal, then a single-frequency vibration threshold at a lower frequency will not detect the abnormality. In the early stages of nerve compression, the patient may be asymptomatic at rest and measurement at a single frequency without any provocation may not detect abnormalities in vibration thresholds. When using a single-frequency vibrometer and baseline measures are within normal limits, patient testing of vibration thresholds should be combined with provocation of symptoms. In a group of thoracic outlet syndrome patients, vibration thresholds in the small finger were significantly elevated after provocation (arms elevated), whereas baseline measures remained normal.[32] However, in another study evaluating vibration thresholds in TOS patients, baseline thresholds were not significantly different than normal control subjects.[70]

The Bruel and Kjaer vibrometer (Type 9627, Naerum, Denmark) allows the measurement of vibration thresholds at seven frequencies ranging from 8 to 500 Hz.[52,53] The patient places the digit on a 5-mm$^2$ probe and the intensity of the vibration is controlled by a switch in the nontest hand. The vibration threshold is determined by a method of limits. It is hypothesized that the higher vibration frequencies are usually first affected in the early stages of chronic nerve compression and with increasing age.[53] Therefore, evaluation of these higher frequencies may permit earlier identification of these discrepancies.[53]

## Cutaneous Pressure Thresholds

Cutaneous pressure thresholds evaluate the threshold of the slowly adapting sensory receptors (Merkel cell neurite complexes). Initially, Von Frey described using hair of varying diameters to evaluate pressure thresholds and Semmes-Weinstein monofilaments (Sammons Preston, Bolingbrook, IL) are now commonly used to measure pressure thresholds.[42,71] These nylon monofilaments vary in diameter thereby requiring different application forces thus producing different pressure thresholds (Figure 4). Between each monofilament within the set there is an incremental increase on a logarithmic scale (log 10 force of 0.1 mg). The monofilaments are applied to the test area in a consistent fashion and the smallest filament that is perceived by the patient is recorded as the threshold. Alteration in the monofilament diameter or in the application technique will alter the stimulus and thus the recorded pressure threshold.[51,72] In a recent evaluation of normal subjects, variability in Semmes-Weinstein monofilament measures was found with repeated testing.[73] Criticism of the Semmes-Weinstein monofilaments includes the logarithmic scale between the monofilaments and the variability in the size and shape of the nylon filament.[74]

More recently, a computerized one-point discrimination system has been described. This system permits measurement of cutaneous pressure thresholds on a continuous scale.[75] This system has a two rounded, blunt, 0.9 mm probes that allow the assessment of one-point or two-point discrimination.[75] For one-point discrimination, the probe is applied to the patient's digit and the patient indicates when the stimulus is felt by pressing a button in the contralateral hand. After five trials, the highest and lowest values are discarded and the mean value of the remaining three values is recorded as the cutaneous pressure threshold.[74]

## Two-point Discrimination

Tactile discrimination measured with 2pd more accurately reflects the quantity of innervated sensory

***FIGURE 4.*** *Semmes-Weinstein monofilaments are applied to the digit pulp and the smallest filament that the patient can detect is the pressure threshold.*

***FIGURE 5.*** *The Disk-Criminator can be used to measure moving and static two-point discrimination (2pd) by applying the probes to the digit pulp. The smallest distance by which the patient can differentiate two probes from one probe is recorded as the 2pd.*

receptors.[45] Initially, Moberg[55,56,76] described using a paper clip to measure 2pd. However, because of inconsistency in the distance between the ends and unreliable blunt ends, instruments such as the Disk-Criminator (Neuroregen, Baltimore, Maryland) and the Two-point Aesthesiometer (Smith + Nephew, Germantown, WI) were introduced.

To evaluate static 2pd, either one or two probes are applied to the digit pulp with only enough force to produce minimal skin deformation and the probes are held in one place for 5 seconds. The patient is asked to identify if one or two probes were applied. The smallest distance that the patient can correctly differentiate one from two probes is recorded as the static 2pd. To evaluate moving 2pd, testing is performed by placing the probes perpendicular to the digit and moving them longitudinally along the digit pulp (proximal to distal). The smallest distance that the patient can correctly identify two probes from one is documented as the moving 2pd. The Disk-Criminator with dull rounded probes permits 2pd measures between 2 mm to 15 mm in 1-mm increments with good interrater reliability (Figure 5).[57,77]

The description of static 2pd measurement does not differentiate whether the probes are to be placed perpendicular or parallel to the digit pulp, and many reports indicate that the probes were placed parallel to the digit.[56,76,78] In Onne's[79] review of patients with nerve injury, some of the reported 2pd were very large and could only have been obtained by placing the probes parallel to the digit pulp. However, the measurement of moving 2pd was described by placing the probes perpendicular to the digit pulp, and therefore assessment with the probes placed parallel to the digit pulp is using incorrect measurement technique.[43] In some cases, patients with nerve injury or with severe nerve compression may not be able to identify two probes before the distance between the probes exceeds the diameter of the patient's finger when placed perpendicular to the digit. In our experience, a 2pd that exceeds 10 or 11 mm is the ''functional'' equivalent of no 2pd. Therefore, in our measures, we have standardized the measurement of moving and static 2pd to apply the probes perpendicular to the digit pulp. If the distance between the probes exceeds the digit diameter, then this is recorded as no 2pd. Standardization of measurement technique is necessary to ensure reliable measurement of both moving and static 2pd. Some authors have advocated five out of seven trials; however, patient fatigue can influence the assessment, and we use two out of three trials for the correct response. Criticism of 2pd assessment includes variability in the application force and not knowing the application force.[43,69,80] However, testing with the Disk-Criminator has been shown to have good intertester reliability when used in a consistent fashion.[57,77]

## Sensory Recovery Grading System

The sensory grading system is less defined than the numerous motor grading systems. Highet's[12] scheme included grades from S0 (no sensation) to S4 (complete recovery). This scheme was modified by Zachary and Holmes[45] to gradations of recovery within the grading scale and then the system was modified to include 2pd. The modification of the sensory classification system includes the following: S0 = no sensation, S1 = recovery of deep pain sensibility, S1+ = recovery of superficial pain sensibility, S2 = recovery of pain and some touch sensibility, S2+ = recovery of pain and some touch sensibility with some over-response, S3 = recovery of pain and some touch sensibility with no over-response with 2pd greater than 15 mm, S3+ = sensory localization and 2pd recovery between 7 and 15 mm, and S4 = complete recovery with 2pd between 2 and 6 mm.[45]

Not all sensory evaluation tools will be equally effective in the assessment of nerve injury and nerve compression. Tests that are most useful in detecting abnormalities in nerve injury may not be the most sensitive measures in the different stages of nerve compression. In the early stages of nerve compression, all sensory tests may be normal because symptoms are intermittent and the histopathologic changes in the nerve are minimal. Therefore initially tests of provocation to identify the site of compression may be the only positive test. However, with increased compression and progression to more chronic nerve compression, the threshold measures will become abnormal and finally with severe nerve compression, 2pd will become abnormal. Because 2pd becomes abnormal in the severe stages of nerve compression, it will not be a very sensitive measurement in many patients with chronic nerve compression.[31,32]

Patients with brachial plexus nerve compression often have pain associated with the positions that compress the brachial plexus, and therefore patients will alter their positions to minimize discomfort. Because the progression of nerve compression is dependent upon the duration of compression, brachial plexus nerve compression is less likely to progress to the more severe stages. Therefore, clinical findings and testing will reflect these changes with positive provocative maneuvers and abnormal sensory thresholds, whereas 2pd will remain normal.[31,32]

After nerve injury, 2pd may be a better indicator of functional recovery than threshold measures because threshold measures may not return to normal values. Object identification is often used as a functional outcome to evaluate patient recovery after nerve reconstruction or decompression. A strong correlational relationship has been reported between object identification and 2pd[57,81,82]; therefore, with respect to object identification, 2pd is a good predictor of function.

## Pain Evaluation

Pain can be a significant component of patient complaints with nerve injury and/or nerve compression. Particularly in patients with multiple level nerve compression and soft-tissue disorders, the patient's response to pain can impact on successful management. Therefore, assessment of pain and the impact of this pain on the patient's life is an important component of the total evaluation. Our pain evaluation,[83] which has been modified from the McGill pain questionnaire,[84] Hendler's back pain questionnaire,[85] and a previous modification of the pain questionnaire,[11,33] consists of a series of questions (e.g., regarding work, home, medications), pain descriptors, a body diagram, and visual analogue scales for pain, stress, and coping. Each component is scored and considered positive with use of more than three descriptors, a body diagram that does not follow a known anatomical pattern, or a questionnaire score exceeding 20. If more than two of these components are positive, the patient should be sent for further psychological evaluation to determine the impact of pain on their life. Successful management will only be achieved if the psychological component of this problem is also addressed. The first page of this pain evaluation questionnaire, which includes the body diagram, pain adjectives and the 1- cm visual analogue scales for pain can be used at subsequent patient visits to monitor progress.

## Evaluation of the Cervicoscapular Region

Patients with brachial plexus nerve compression will often present with pain and discomfort resulting from muscle imbalance in the cervicoscapular region. A thorough evaluation of the shoulder, scapular and cervical movements should be included in patients who report proximal symptoms. Relaxed standing posture is evaluated with comparison to the ideal posture[86]; patients typically have a head-forward posture with loss of cervical lordosis, increased thoracic flexion, scapulae abduction, and shoulder internal rotation. Prolonged positioning in altered postures will result in adaptation of the muscle length and resultant muscle imbalance.[87] Cervical range of motion should be evaluated for the degree of movement in addition to associated pain and movement abnormalities. Individual muscles in the cervicoscapular region should also be evaluated for evidence of tightness, weakness and tenderness. Shoulder range of motion should be evaluated in addition to the associated scapular motion. With active shoulder flexion and abduction, weakness in the serratus anterior or trapezius muscles, if present,

can be seen with abnormal scapular winging and/or motion.

Neural tension and length must also be considered in the evaluation of patients with upper quadrant symptoms, particularly in those with complaints of paraesthesia and numbness.[88–90] Because the nerve is composed of connective tissue, prolonged duration in shortened positions may produce relative neural shortening. There will also be increased fibrosis at the entrapment sites and perhaps adhesions, which may tether the nerve at these sites. Neural tissue is relatively intolerant of being stretched without causing symptoms, and thus excessive stretching will cause increased symptoms both proximally and distally extending to the nerve's sensory distribution. Therefore, overstretching of the neural line will potentially increase symptoms throughout the upper quadrant. The irritability of the patient's condition must guide the aggressiveness of the evaluation and the time at which neural tension testing will provide the most useful information. Butler's proposed neural stress tests of the upper extremity are important in determining decreased mobility of the neural tissues.[89] However, these tests can also produce pain patterns in asymptomatic individuals, and the therapist can best attain the most useful information by becoming familiar with the response in asymptomatic individuals before applying these tests to the patient with nerve-related symptoms. These tests are best used in the later stages of management, when the irritability of the condition has decreased and segmental mobility of the cervical spine and scapula has been achieved.

## CONCLUSION

The evaluation of patients with nerve injury and/or nerve compression requires an accurate history and subjective report to determine the tests that will be most useful in providing the essential information. Motor and sensory evaluation are necessary in global mixed nerve injuries, but in cases of nerve compression, tests of provocation will give more accurate information in detecting the site of nerve compression. Multiple levels of nerve compression can increase sensory sensitivity and can confuse the interpretation of findings. However, because there is no standard test in the evaluation of patients with nerve injury and/or compression, a battery of valid and reliable sensory and motor tests will provide the most complete information to formulate a treatment plan.

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