CROSSFIRE: Debates in Electrodiagnostic Medicine

Faculty

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Dr. Johnson received his medical degree from Ohio State University in Columbus, Ohio, interned at Philadelphia General Hospital, and completed his residency in physical medicine and rehabilitation at Ohio State University under the sponsorship of the National Foundation of Infantile Paralysis. He has edited the textbook *Practical EMG*, and authored over 130 peer-reviewed articles. He established the Super EMG continuing medical education course in 1978, and is still involved in planning and teaching this course. Currently, Dr. Johnson is an emeritus professor at Ohio State University. He is a past-president of the AAEM, AAPM&R, AAP, former chair of the American Board of Electrodiagnostic Medicine, and has been editor of the *American Journal of Physical Medicine and Rehabilitation*.

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Dr. Robinson attended Baylor College of Medicine and completed his residency training in rehabilitation medicine at the Rehabilitation Institute of Chicago. He was on the faculty at the University of Pittsburgh, during which time his interests included, among other things, phrenic nerve lesions after cardiac surgery. He now serves as professor and chair of the Department of Rehabilitation Medicine at the University of Washington and is the director of the Harborview Medical Center Electrodiagnostic Laboratory. His current clinical interests include the statistical interpretation of electrophysiologic data, laryngeal electromyography, and the study of traumatic neuropathies. He recently received the Distinguished Academician Award from the Association of Academic Physiatrists.

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Dr. Kimura received his Bachelor of Technology in 1957 and MD in 1967 from Kyoto University in Japan. He came to the United States as a Fulbright scholar in 1962 for his neurology residency and electrophysiology fellowships at the University of Iowa. He has served as the AAEM’s secretary-treasurer, president of the AAEM, and editor of *Muscle & Nerve*. Dr. Kimura received the AAEM’s Distinguished Researcher Award in 1995 and Lifetime Achievement Award in 1999. He published the 3rd edition of *Electrodiagnosis in Diseases of Nerve and Muscle* in 2001. His current professional titles include Professor Emeritus at Kyoto University, Professor of Neurology at the University of Iowa, and president for the World Federation of Neurology.

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Dr. Fisher is a graduate of Harvard Medical School. He performed his residency in neurology at the Massachusetts General Hospital where he also served as a fellow in clinical neurophysiology. He is currently a professor of neurology at the Loyola University Stritch School of Medicine, Maywood, Illinois. Dr. Fisher is the director of the neuromuscular program at Loyola University Medical Center and the director of the Clinical Diagnostic Neurophysiology Laboratories at the Hines VA Hospital. He has long been active in research in clinical neurophysiology and has had a special interest in F waves. His recent interests have included the electrodiagnostic evaluation of patients with immunologically mediated neuropathies. Dr. Fisher has long been active in the AAEM and has served on the Board of Directors. He is also a past-president of the American Academy of Clinical Neurophysiology.

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The ideas and opinions expressed in this publication are solely those of the specific authors and do not necessarily represent those of the AAEM.
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Authors had nothing to disclose.

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# CROSSFIRE: Debates in Electrodiagnostic Medicine

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## Objectives

After attending this course, the participant will (1) understand appropriate approaches for diagnosing carpal tunnel syndrome (CTS), (2) understand the pros and cons of palmar stimulation in CTS, (3) learn the utility of F waves in entrapment neuropathies, (4) understand the benefit of incremental stimulation in focal entrapment neuropathies, and (5) become familiar with appropriate techniques in the evaluation of brachial plexopathies and thoracic outlet syndrome.

## Prerequisite

This course is designed as an educational opportunity for residents, fellows, and practicing clinical EDX consultants at an early point in their career, or for more senior EDX practitioners who are seeking a pragmatic review of basic clinical and EDX principles. It is open only to persons with an MD, DO, DVM, DDS, or foreign equivalent degree.

## Accreditation Statement

The AAEM is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education (CME) for physicians.

## CME Credit

The AAEM designates attendance at this course for a maximum of 3.5 hours in category 1 credit towards the AMA Physician's Recognition Award. This educational event is approved as an Accredited Group Learning Activity under Section 1 of the Framework of Continuing Professional Development (CPD) options for the Maintenance of Certification Program of the Royal College of Physicians and Surgeons of Canada. Each physician should claim only those hours of credit he/she actually spent in the activity. The American Medical Association has determined that non-US licensed physicians who participate in this CME activity are eligible for AMA PMR category 1 credit.
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**2003-2004 AAEM PRESIDENT**

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INTRODUCTION

Carpal tunnel syndrome (CTS) is a syndrome of pain, numbness, and weakness in the hand due to compromise of the median nerve within the carpal tunnel. Therefore, one must have the history and physical findings as well as the electrodiagnostic (EDX) evidence of compromise of the median nerve within the carpal tunnel prior to making a diagnosis.

The sensitivity and specificity of six signs of CTS have been studied by Kuhlman and Hennessey. They found that a square-shaped wrist (Johnson’s wrist ratio) was the most sensitive sign of CTS (69%) and that abductor pollicis brevis (APB) weakness was the second most sensitive sign (66%).

IS THERE A SIMPLE, YET RELIABLE SCREEN FOR CARPAL TUNNEL SYNDROME?

This author believes that comparing the median sensory nerve latency to the radial nerve latency to the thumb is the best and most reliable screen for CTS (Figure 1). A positive screen is indicated with a difference of greater than .3 ms assuming that the sensory nerve action potential (SNAP) is within normal limits—35 µV for the median nerve and 10-15 µV for the radial nerve. If latencies are within normal limits but amplitudes are small, then one must consider a brachial plexus compromise or a lesion of C6 nerve root distal to dorsal ganglion.

![Figure 1](image_url)
WHAT IS AN “ADEQUATE” ELECTRODIAGNOSTIC EXAMINATION?

Both the motor and sensory nerve fibers must be evaluated proximal and distal to the carpal tunnel. This concept is controversial because of the difficulties in stimulating and recording with this technique. This should include recording the SNAP over digit 3 with stimulation at the wrist (14 cm) and comparing it to the SNAP with stimulation at mid-palm (7 cm).

The physician should examine the amplitude, duration, and latency of the two segments. Usually the wrist to midpalm segment (14-7 cm) will have slightly more than half of the total latency. This is because of the smaller diameter of the distal portion of the nerve and because the finger is cooler than the palm. For the latter reason, SNAP amplitude at the palmar will normally be greater than 30% larger than the SNAP amplitude when stimulating at the wrist (Figure 2). If the hand is cool, all three parameters will be amplified—amplitude, latency, and duration of SNAP.

The median compound muscle action potential (CMAP) is evaluated by stimulating the median nerve fibers at the wrist (8 cm) as well as distal to the carpal ligament at the recurrent branch of the median nerve and recording over the APB.

This mid-palmar stimulation is technically difficult because the ulnar nerve could unintentionally be stimulated and the portion of the thenar muscles innervated by the ulnar nerve (usually deep head of flexor pollicis brevis) can contribute to the amplitude of the CMAP recorded (Figure 3).

To ensure that the ulnar nerve is NOT accidentally stimulated, note the shape and duration of the compound muscle action potential (CMAP). The duration of the negative component of the CMAP of a single thenar muscle will be less than 5 ms.

The shape must be the same at both wrist and palmar stimulation. If the ulnar nerve is unintentionally stimulated in the palm, the amplitude will be increased, but CMAP shape will differ and the negative component of the CMAP will have increased in duration.

While it is not especially difficult to stimulate only the median nerve at the palmar site with surface bipolar electrodes, the cathode must be tucked into the thenar muscle and the anode rotated minimally. The CMAP shape and duration should be noted.

It is easy to restrict the stimulus to the recurrent branch of the median nerve using a monopolar needle electrode for stimulation. The anode can be a large-surface electrode on the medial dorsal hand, or this author has used a ring electrode on base of digit 5.
If the CMAP is larger (greater than 10%) with distal stimulation, it is likely that there is a neurapraxic block of some of the median motor fibers within the carpal tunnel. There are persuasive studies in the literature showing this phenomenon.

Unfortunately, the palm to wrist (8 cm) transcarpal mixed nerve study which compares median and ulnar nerve latencies contains both motor and sensory fibers of both nerves. This technique therefore would not differentiate CTS which compromises only motor or only sensory axons of the nerve.

Kimura suggests that as many as 10% of CTS patients have only motor fibers involved.

This author and colleagues have reported “acute” CTS with largely or solely motor fibers compromised. These have usually been individuals who have repetitive, vigorous, or continuous grasping for 6-8 hours per day and several days in a row. Two examples are a high school hockey player who played 6-8 hours a day for 5 days consecutively, and several auto painters who squeezed a paint gun in a new job for 3-4 weeks in a row.

WHAT ABNORMALITIES CONFIRM CARPAL TUNNEL SYNDROME?

Absolute latencies are not reliably diagnostic and are never prognostic! To be more effective, one should compare latencies of an uninvolved nerve. One should also compare latencies from the wrist to the finger to latencies of the midpalm to the finger to reliably show slowing at the carpal tunnel. Evaluation of amplitudes are best for diagnosis and prognosis. This should be performed by comparing stimulation both proximal and distal to the carpal tunnel. Severity and prognosis is best determined by amplitude when stimulation is distal to the carpal tunnel.

CAN CARPAL TUNNEL SYNDROME BE DIAGNOSED IN THE PRESENCE OF DIABETIC POLYNEUROPATHY?

It is definitely possible to diagnose CTS in the presence of diabetic polyneuropathy. The examining physician should first compare two nerves—radial versus median to digit and ulnar versus median to digit 4 (Figure 4). If there is a disproportionate increase in latency or reduction of amplitude when comparing the two nerves, CTS is likely.

The examining physician should then compare the latency and amplitude of the SNAP of digit 3 with stimulation on both sides of the carpal ligament—noting the increase of latency and reduction in SNAP amplitude. If CTS is present, the distal latency (i.e., midpalm stimulation of 7 cm) will be greater than expected and the amplitude of SNAP will be less (Figure 5).
The last step to diagnosing an underlying diabetic polyneuropathy is to stimulate the sural nerve and note the latency and the amplitude. If diabetic polyneuropathy is present, the F wave of the ulnar or tibial nerve will be complex and the shortest latency prolonged.

SUMMARY

The SNAP of digit 1, both an analysis of the amplitude and the latency, is an excellent and reliable indication of CTS and is an accurate and reliable screen for CTS.

The best measure of the severity and the prognosis of CTS is recording the amplitude of the SNAP and CMAP when stimulating the median nerve both proximal and distal to the entrapment site which is the carpal ligament. Both sensory and motor axons must be evaluated separately. This author recommends digit 3 SNAPs for sensory fibers and the thenar CMAPs for motor axons, but the median nerve must be stimulated both proximal and distal to the carpal ligament.

Carpal tunnel syndrome can be diagnosed and assessed accurately in the presence of diabetic peripheral neuropathy. With CTS and an underlying diabetic polyneuropathy, the disproportionate increase in amplitude and latency of the comparison nerves is radial and median to digit 1 and ulnar and median to digit 4. Latency and amplitude of the sural SNAP and tibial or ulnar F wave should then be checked.

BIBLIOGRAPHY

CONTROVERSIES IN CARPAL TUNNEL SYNDROME

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INTRODUCTION

Although carpal tunnel syndrome (CTS) is the most frequently seen entrapment neuropathy in the electrodiagnostic (EDX) laboratory, there are multiple and varied approaches that have been described for diagnosing this condition. The lack of uniformity in approach suggests that there are likely many unanswered questions on how to best diagnose patients with this condition.

To determine the best approach, it is helpful to first list the criteria for the best testing strategy. The best EDX testing approach should be (in descending order of priority):

a. Specific (few false positives)
b. Sensitive (few false negatives)
c. Reliable (the same results on repeat testing)
d. Resistant to temperature effects
e. Efficient

As will be outlined, the best way to address these needs for diagnosing CTS is by using the combined sensory index (CSI), the modified CSI, median motor nerve conduction studies (NCSs) (don't stimulate in the palm!), and occasionally needle electromyography (EMG).

WHAT IS THE COMBINED SENSORY INDEX?

The CSI is a combination of three sensory conduction comparisons.6 These include: median-radial sensory latency difference to the thumb at 10 cm (thumbdiff), median-ulnar sensory latency difference to the ring finger at 14 cm (ringdiff), and median- ulnar sensory latency difference across the palm at 8 cm (palmdiff). The CSI is calculated by simply adding the three latency differences (median minus ulnar, or median minus radial):

\[ \text{CSI} = \text{palmdiff} + \text{ringdiff} + \text{thumbdiff} \]

WHAT IS THE SPECIFICITY OF INDIVIDUAL NERVE CONDUCTION STUDIES AND THE COMBINED SENSORY INDEX IN THE DIAGNOSIS OF CARPAL TUNNEL SYNDROME?

Although sensitivity of various tests for CTS is often discussed,1,2 specificity is just as important or probably more important. It is at least as important to avoid operating on a patient without CTS, as it is to miss operating on someone with mild disease. In
order to measure and obtain high specificity, it is critical in any study to have a healthy control group either studied concurrently or studied in the same laboratory by the same investigators. Ideally, the control group should be similar in age and other demographic variables to the patient with the disease.

Usually, when collecting reference values from a control population, one should include approximately 97.5% of the healthy control subjects within the reference range. This is usually accomplished by measuring the mean value, standard deviation, and taking mean plus or minus two standard deviations as the reference values. This technique is appropriate if the latencies are distributed in a Gaussian fashion. Unfortunately, a Gaussian distribution often is not present and other techniques such as percentile estimation or transformation are required to establish reliable control values. Even after reference values are obtained, subsequent studies of the specificity of each technique should be measured using a control group—it cannot be assumed to be 95% simply because the mean ± 2 standard deviations from a reference group was used. For this reason, it is important to study a concurrent control group and measure the actual specificity, i.e., how many of the control subjects came out normal (true negatives) or abnormal (false positives).

The specificity and sensitivity of a technique is, of course, a function of where reference (normal) values are set. When these are set closer to the mean of the control group, sensitivity goes up and specificity goes down; the converse is true as the reference value moves further from the control group mean. Table 1 lists specificities for tests of the three NCSs listed above at defined reference values, and the CSI as a whole. Most authors would agree that a goal of 95-99% specificity is acceptable.

### WHAT IS THE SENSITIVITY OF INDIVIDUAL NERVE CONDUCTION STUDIES AND THE COMBINED SENSORY INDEX IN THE DIAGNOSIS OF CARPAL TUNNEL SYNDROME?

Although a number of values for specificity and sensitivity have been quoted in the literature, restriction of studies to those that meet certain criteria suggest that sensitivity of sensory studies is generally in the range of 50-80% while specificity is 95% or greater. This author has studied patients with relatively mild CTS, i.e., only those who have all sensory responses present. In this group, when specificity is 95-97%, sensitivity is 82% for the CSI and 70-76% for individual tests considered alone (Table 1). This higher sensitivity for the CSI is likely a result of the fact that some patients with mild CTS may have latencies in the high normal range for individual tests, but when this tendency is added together among three studies, the result will extend outside the reference range. It should be kept in mind that these figures for sensitivity are for those with mild disease; they will be greater when all-comers (including those with absent sensory responses) are considered.

### WHAT IS THE RELIABILITY OF INDIVIDUAL NERVE CONDUCTION STUDIES AND THE COMBINED SENSORY INDEX?

Ideally, tests for CTS or any other diagnosis should be repeatable when performed over two separate occasions. A variety of measures could be used to measure repeatability. For data that is not normally distributed, a useful statistic is the Spearman rho. As demonstrated in Figure 1, test-retest repeatability for performing a single test varies from a Spearman rho of 0.67-0.75 (1.0 is perfect repeatability). While test-retest differences are not large, they do in some cases differ sufficiently to cross the border between normality and abnormality.

It is theoretically expected that as one performs more tests and summarizes the results into a single variable, test-retest repeatability should be improved. As demonstrated in Figure 1, test-retest repeatability for the CSI (the summary of three variables) is excellent at 0.95.

It makes sense that a summary variable composed of multiple single tests is more reliable than a single test, as small random errors are neutralized by multiple measures. An analogy that can be used is multiple choice tests. What is a more reliable test—

<table>
<thead>
<tr>
<th>Test</th>
<th>Reference value</th>
<th>Sensitivity</th>
<th>Specificity</th>
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<tr>
<td>Med-Uln palmar</td>
<td>&lt; 0.3</td>
<td>70%</td>
<td>97%</td>
</tr>
<tr>
<td>Med-Uln ring finger</td>
<td>&lt; 0.4</td>
<td>74%</td>
<td>97%</td>
</tr>
<tr>
<td>Med-Rad thumb</td>
<td>&lt; 0.5</td>
<td>76%</td>
<td>97%</td>
</tr>
<tr>
<td>One of three tests abnormal</td>
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<td>85%</td>
<td>92%</td>
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<tr>
<td>Two of three tests abnormal</td>
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<td>74%</td>
<td>99%</td>
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<td>Three of three tests abnormal</td>
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<td>56%</td>
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<tr>
<td>Combined sensory index</td>
<td>&lt; 0.9</td>
<td>83%</td>
<td>95%</td>
</tr>
<tr>
<td>Combined sensory index</td>
<td>&lt; 1.1</td>
<td>82%</td>
<td>100%</td>
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Med = median; Uln = ulnar; Rad = radial
one with a good single question (that someone could make a simple error on) or one with many good questions?

HOW MANY TESTS SHOULD BE PERFORMED IN THE EVALUATION OF POSSIBLE CARPAL TUNNEL SYNDROME AND HOW DOES THIS AFFECT SENSITIVITY AND SPECIFICITY?

Given the number and variety of EDX tests available for diagnosing CTS, the question that inevitably arises is how many tests should be performed in each patient? At first, it is tempting to perform many tests so that one is more likely to detect any problem that might be subtle or might not show up on a smaller number of tests. Also, by performing more tests, the physician makes a more thorough assessment of different nerve fascicles than an assessment with a single test. There are, however, problems with performing multiple tests. The most significant problem is that of multiple comparisons. As more and more tests are performed, each of which has a 2.5% false-positive rate (under ideal circumstances), the chance of any one of the tests being abnormal goes up roughly additively. Thus, performing two tests yields a 4.9% chance of at least one test being abnormal in a control population. For three tests, the false-positive rate is 7.3%. Table 2 lists the false-positive rate as greater numbers of tests are performed.

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**Figure IA**  Test-retest reliability of a combined sensory index (CSI), Spearman rho = 0.95 = 0.75.

**Figure IB**  Test-retest reliability of ring-diff, rho = 0.67

**Figure IC**  Test-retest reliability of thumb-diff, rho = 0.75

**Figure ID**  Test-retest reliability of palm-diff, rho = 0.74

Palmdiff = medial-ulnar latency difference across the palm; Ringdiff = median-ulnar latency difference to the ring finger; Thumbdiff = median-radial latency difference to the thumb
The false-positive rate is lower if there is a requirement that multiple (two or more) tests be abnormal to make a diagnosis, however, this will lower sensitivity. Another practical problem with performing more testing is simply the time and cost required to complete the study. Obviously performing tests that do not add clinical or diagnostic information is not a good practice.

Thus, the question of how many tests to perform is not a trivial matter. The strategies for performing multiple tests should be decided upon before studying the patient, not handled in a casual manner during or after the test is performed.

To address the question of how to interpret multiple tests, this Robinson and colleagues\(^6\) have compared strategies of analyzing three different tests for CTS. In a comparison of 53 patients with CTS and 46 control subjects, three different EDX tests were performed in all subjects. This included latency difference with mid-palmar stimulation of median and ulnar nerves recording at the wrist (palmdiff), median and ulnar nerve stimulation with recording over the ring finger (ringdiff), and median and radial nerve stimulation with recording over the thumb (thumbdiff). Sensitivities of each of the tests varied from 70-76%, while specificities were 97% (Table 1). If a physician performed all three tests in each subject and simply took a single abnormality (one abnormality out of the three tests) as abnormal, the sensitivity rose to 85%. However, this is at the sacrifice of specificity, which fell from 97% to 92%. It was felt that this additional 5% false-positive rate is not acceptable. Alternative strategies of requiring two out three tests to be abnormal yielded a sensitivity rate of 74%, comparable to the single tests; specificity using this strategy was improved at 99%. Finally, the strategy of requiring all three tests to be abnormal yielded at a lower sensitivity of 56% but specificity was 100%. This change in specificity closely matches the theoretical predictions noted in Table 2.

The CSI calculated as the sum of the three latency differences

\[
(CSI = \text{palmdiff} + \text{ringdiff} + \text{thumbdiff})
\]

brings in the advantages of multiple tests (assessing multiple areas of nerve, enhancing reproducibility of findings, etc.), but does not create the problem of multiple comparisons. Thus, as long as the physician performs three tests but only makes a diagnosis based upon the summed result from all three tests, the problem of an additive false-positive rate is eliminated. Using this approach, sensitivity improved to 83% in the population described above with specificity still remaining high at 95%. These results assume a reference value for the CSI of less than or equal to 0.9 ms. Alternate use of a reference value of less than or equal to 1.1 ms yielded a sensitivity of 82% and specificity of 100% in the study sample.

Thus, it would appear that use of the CSI has advantages of improved sensitivity and high specificity compared to performing multiple individual tests or even a single individual test.

**WHAT IS THE INFLUENCE OF TEMPERATURE?**

Temperature is known to slow nerve conduction latencies and velocities. Thus, obtaining a median sensory latency will, to a large degree, be dependent upon the temperature. Much of this dependency, however, can be reduced by using comparisons of

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**Table 2** Probability of abnormal results by chance alone

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<th>Number of parameters studied</th>
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<tr>
<td>20</td>
<td>39.5</td>
<td>8.5</td>
<td>1.3</td>
<td>0.1</td>
<td>&lt; 0.1</td>
</tr>
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</table>

The probability of finding an abnormal result by chance alone according to the number of parameters studied. These calculations assume that 2.5% of an asymptomatic control population fall into the “abnormal” range for each parameter studied, and that each parameter is independent.
two nerves within the same limb. As noted in Table 3, temperature dependency of individual median nerve latencies is significant. Comparisons of the median nerve with another nerve in the same hand are not influenced by temperature. In addition, the CSI (made up of comparisons between median and other nerves in the same hand) is also not influenced significantly by temperature. Thus, to avoid influences of temperature, as well as age, height, and other influential variables, it is preferable to use comparisons of median nerve latency to other nerves within the same hand.

**IS THE ENTIRE COMBINED SENSORY INDEX NEEDED IN ALL PATIENTS?**

While the CSI represents an improvement over single tests, it has been noted that it might not be necessary to perform all three tests for the CSI when one or more are extreme values. Specifically, if the median latency is slow compared to the ulnar or radial nerve latency, additional testing might not help the diagnostic classification. Similarly, when results are “normal” additional testing might not be necessary. Robinson and colleagues have explored this to define endpoints for when more limited testing is possible. Figure 2 demonstrates a plot of latency difference between median and ulnar or median and radial nerves versus the probability of having an abnormal CSI. As noted on each graph, there is a range of normal individual test results that almost always predict a normal CSI. Similarly, there is a range of abnormal individual tests, which almost always predict an abnormal CSI. However, there is an uncertain range in the middle (this range varies for each test) that does not predict the CSI with a high degree of confidence. Thus, one could consider using the approach shown in Figure 3 whereby single tests could be performed when results are normal or abnormal, but the entire CSI

<table>
<thead>
<tr>
<th>Technique</th>
<th>Change in ms/°C</th>
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<tr>
<td>Median to ring</td>
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<tr>
<td>Ulnar to ring</td>
<td>-0.14 ms/°C</td>
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<tr>
<td>Median to thumb</td>
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<tr>
<td>Radial to thumb</td>
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<tr>
<td>Ring-diff</td>
<td>0.0003 ms/°C</td>
</tr>
<tr>
<td>Thumb-diff</td>
<td>-0.0002 ms/°C</td>
</tr>
<tr>
<td>Palm-diff</td>
<td>0.0005 ms/°C</td>
</tr>
<tr>
<td>Combined sensory index</td>
<td>0.0002 ms/°C</td>
</tr>
</tbody>
</table>

Palm-diff = medial-ulnar latency difference across the palm; ns = nonsignificant; Ring-diff = median-ulnar latency difference to the ring finger; Thumb-diff = median-radial latency difference to the thumb; CSI = combined sensory index; Palm-diff = medial-ulnar latency difference across the palm; Ring-diff = median-ulnar latency difference to the ring finger; Thumb-diff = median-radial latency difference to the thumb;
could be performed when results fall in the uncertain middle
ground. This approach is termed the modified CSI and it allows
for a more rapid diagnostic classification, though at the expense
of less test-retest reliability.

**WHAT IS THE ROLE OF MEDIAN MOTOR RESPONSES?**

Median motor studies should be routinely obtained in the eval-
uation of CTS for two reasons. First, there is a small percentage
of patients with CTS who have selective motor slowing; this may
represent relatively more impact on the recurrent motor branch
than sensory fibers to the digits. Second, median motor studies
may help to further define the extent of motor axon loss.

However, please do not stimulate the median nerve in the palm
looking for neurapraxia. As has been shown by Park and col-
leagues, this is rather problematic. Due to stimulation of the
deep ulnar nerve (which on average is 1.2 cm from the recurrent
median), and/or crossing anomalous fibers from median to ulnar
nerves, there is a high frequency of larger responses with palm
stimulation than wrist stimulation. In fact, 53% of healthy
control subjects have a palm to wrist difference in amplitude
greater than reported control values, and 25% of control subjects
have an amplitude ratio (palm to wrist) outside the reference
range. Use of this technique for diagnosis produces an acceptably
high false positive rate.

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**Figure 3** Alternative diagnostic strategy for carpal tunnel syndrome

CSI = combined sensory index; Palmdiff = medial-ulnar latency difference across the palm; Ringdiff = median-ulnar latency difference to the ring finger; Thumbdiff = median-radial latency difference to the thumb
WHEN SHOULD NEEDLE ELECTROMYOGRAPHY OF THE THENAR MUSCLES BE PERFORMED?

There is considerable debate as to when needle EMG should be performed in the evaluation of possible CTS. This test is quite uncomfortable and it is also uncertain how the test results should influence treatment or assessment of prognosis.

It is this author's practice to perform thenar muscle needle EMG in those patients who have abnormal median motor responses, either in latency or amplitude, or those with a history of limb trauma. This approach is designed to include predominantly those patients who will have a higher likelihood of abnormalities, and spare those patients from the procedure who would have a low yield on the test. A cervical root screen is also performed on those presenting with a history and/or physical examination suggestive of possible cervical radiculopathy.

RECOMMENDED APPROACH

The approach to diagnosing CTS should be well thought out and strategized before the patient is referred to the EDX laboratory for testing. Specifically, the EDX consultant should know which tests are to be performed, how many will be performed, and how they will be interpreted to arrive at a diagnosis. Having a strategy worked out ahead of time allows the examiner to have a higher degree of confidence in the diagnosis.

This author recommends two alternative strategies. First, if the question is simply diagnostic classification (abnormal or normal), and repeat testing over time is not a likely consideration, then the strategy outlined in Figure 3 is a reasonable approach. Specifically, an EDX consultant could perform studies of the ring finger first and if results do not fall within the uncertain range, then the testing is completed. If, however, the results fall within the range of uncertainty, then the entire CSI should be performed.

When test-retest reliability is a consideration and the examiner wants to have a reliable result for the testing to be repeated, then the CSI is preferable to perform on all patients. This is the approach used in this author’s laboratory because of the possibility of repeat testing, should initial treatment prove unsuccessful or should the patient ever present with symptoms after treatment.

Median motor conduction studies should also be performed in all cases, but do not stimulate in the palm. Needle EMG should be performed in cases in which the median motor response is abnormal in latency or amplitude, or if there is a history of trauma.

REFERENCES

The Clinical Utility of Late Responses in Entrapment Neuropathies

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INTRODUCTION

With steady improvement and standardization of methods, nerve conduction studies (NCSs) have become a reliable means of testing peripheral nerve function. They supplement clinical observation by precisely localizing the lesion and characterizing conduction abnormalities. Delineating the extent and distribution of the neural lesion by this means also helps quantitate the degree of involvement. Optimal results can be expected only with the proper choice of techniques, which in turn depends on the type of lesions under consideration. Thus, the studies must be conducted as an extension of the clinical examination, rather than as a laboratory test.

This debate concerns the possible use of F waves in the evaluation of a radiculopathy, which represents a focal, in contrast to diffuse, involvement of the peripheral nerve. This manuscript will first describe the general principle of NCSs specifically dealing with the length of nerve segment under study. As a natural consequence of this argument, one must conclude that F-wave latencies cannot serve as a useful measure of radiculopathy simply because of its focal nature. Other negative aspects of F-wave study when used to diagnose radiculopathy, as well as a more general discussion on proper application of F-wave measurement for the study of diffuse neuropathic process will be discussed.

PRINCIPLES OF NERVE CONDUCTION STUDIES FOR FOCAL LESIONS

A question often posed but rarely tested relates to the length of the nerve segment under study to increase the yields of NCSs. Should one study a shorter or longer nerve segment for better results?

Ordinary NCSs suffice to approximate the site of involvement in a focal lesion. When more precise localization is required, inching the stimulus in short increments in the range of 1 to several centimeters along the course of the nerve can further isolate the site of involvement within the affected segment. In the evaluation of a focal lesion, studies of a longer segment lower the sensitivity of the test because the inclusion of the unaffected segments in the calculation dilutes the effect of slowing at the site of the lesion. In contrast, studying a shorter segment helps isolate a localized abnormality and provides better resolution of restricted lesions that may otherwise escape detection. Assume a nerve impulse conducting at a rate of 0.2 ms/cm (50 m/s) except for a 1-cm segment where demyelination has doubled the conduction time to 0.4 ms/cm. In a 10-cm segment, normally covered in 2 ms, a 0.2 ms increase would constitute a 10% change, or approximately 1 standard deviation, well within the normal range of variability. The same 0.2-ms increase, however, represents a 100% change in latency if measured over a 1-cm
neuropathy at the elbow,1 and peroneal nerve entrapment at the
knee,4 but also in characterizing the focal nature of some wide-
spread abnormalities such as multifocal motor neuropathies.3 It
does not help in evaluating proximal lesions as might be seen in
radiculopathy.

LIMITATION OF F-WAVE STUDIES IN RADICULOPATHIES

In radiculopathy, the site of involvement lies too proximal to
assess the lesion by short incremental stimulation as described
above. The latency of an F wave elicited after stimulation of the
nerve proximally close to the lesion can isolate a relatively short
central loop that contains the site of involvement. With proxim-
al stimulation, however, the F wave usually overlaps with the M
response, requiring a collision method to separate the two
components for latency determination.9 In addition, such a
central segment is not short enough to detect a focal radicular
lesion because a normally conducting unaffected segment dilutes
the abnormality.

F waves fall short of providing clinically useful information in
radiculopathy for a number of other reasons: (1) the minimum
F-wave latency will measure a surviving normal neuron in an in-
complete lesion, thus failing to test the affected neuron by sam-
pling error; (2) when recording from the intrinsic hand and foot
muscles, as customarily done, the study can target only C8 - T1
and S1 - S2 roots, precluding all the other levels from evaluation;
(3) F waves tested in nerves innervated by an unaffected root ob-
viously have no clinical relevance; and (4) F-wave abnormalities,
if detected in a patient with radiculopathy, have no localizing
value as the slowing may occur with lesions located distally or
proximally. Under these situations, the study, if abnormal, has
only limited clinical value in localizing the lesion and confirm-
ing the diagnosis of a radiculopathy.

SENSITIVITY OF F-WAVE STUDIES IN RADICULOPATHY

For all the reasons previously discussed, F-wave studies must, on
theoretical grounds, detect only small percentages of clinically
suspected cases of radiculopathy. In fact, although the F wave is
commonly used in the evaluation of suspected radiculopathies,
the yields have been disappointingly low. In one study,12 sensi-
tivity of the F wave in cervical radiculopathy ranged from 10-
20%. In this series, as in others, the needle examination was
abnormal more often than the F wave. F wave was abnormal in
10% of the 2093 patients who had clinical symptoms of cervi-
cal radiculopathy. This compares to 3% of the 1005 patients
who had normal clinical examinations. In some patients with
good clinical symptoms and abnormal needle study, 7% had ab-
normal F waves. Thus, the F wave was abnormal twice as often
in patients with clinical symptoms consistent with a radiculopa-
thy. In patients with an abnormal needle examination, indicat-
ing a C8 radiculopathy, the likelihood of finding an abnormal F
wave approached 20%. Patients with radiculopathies may show
statistically significant F-wave changes compared to control sub-
jects. A group difference does not suffice as a clinical diagnostic
measure that requires assessment of individual patients. F-wave
studies provide no additional information if a needle elec-
tromyography examination shows abnormalities consistent with
a radiculopathy. F-wave abnormalities found in patients with
normal needle studies also have limited clinical use because of
the lack of localizing value.

USEFULNESS OF F-WAVE LATENCIES FOR DIFFUSE
NEUROPATHIC CONDITIONS

In contrast to the usefulness of segmental studies for a focal
lesion, evaluation of a longer segment provides a better result in
assessing a more diffuse or multi-segmental process such as
demyelinating neuropathy. A longer path has an advantage in accumulat-
ing all the segmental abnormalities, which individually might
not show a clear deviation from the normal range. Assume a
nerve impulse conducting at a rate of 0.2 ms/cm (50 m/s). A
20% delay for a 10 cm segment is only 0.4 ms, whereas the same
change for a 100 cm segment amounts to 4 ms, an obvious in-
crease for easy detection. Thus, in general, the longer the
segment studied, the more evident the conduction delay is for a
diffuse process. Evaluating a longer segment also improves the
overall accuracy because the same absolute measurement error
constitutes a smaller percentage of change in latency and dis-
tance.

In a study testing reproducibility of conduction studies,10 the
measures showing the range of relative intertrial variation (RIV)
within ± 10% included F-wave latency and F-wave conduction
velocity of both median and tibial nerves and sensory conduction velocity of the median nerve. In general, amplitudes showed a greater RIV than latencies or nerve conduction velocities. Similarly, intraclass correlation coefficient (ICC) exceeded 0.9 for F-wave latency of the median and tibial nerves in both the healthy subjects and the patients. In some measures, a large among-subject variance of the amplitudes led to a high ICC despite a considerable intertrial variability. These included the amplitude of the median nerve sensory nerve potential and median and tibial nerve compound muscle action potentials.

CONCLUSION

This author's data indicate that the length of the nerve segment under study dictates the accuracy and sensitivity of measurement. Although studies of shorter or longer segments pose technical merits and demerits, the choice seems to depend entirely on the pattern of the conduction abnormalities. As evidenced by inching technique, a short segmental study uncovers focal lesions involving a restricted zone better than the evaluation of a longer distance, which tends to obscure the abnormality. In contrast, studies of a longer segment detect diffuse or multisegmental abnormalities better, increasing sensitivity and decreasing measurement errors, which, in percentage, diminish in proportion to the overall latency and surface distance. The increased accuracy of the techniques in turn improves the reproducibility of the results. Radiculopathies constitute a sharply focal lesion, but its proximal location precludes the application of segmental studies. Consequently, no currently available NCSs provide useful information in this condition. This and other characteristics make the use of F wave untenable as a measure of radiculopathies.

When using NCSs, short distances magnify focal conduction abnormalities despite increased measurement error; long distances, though insensitive to focal lesions, provide better yields and reliability for a diffuse or multisegmental process. These findings also underscore the importance of choosing nerve stimulation techniques appropriate for the lesion identified clinically. Thus, electrophysiologic studies are most useful when conducted as an extension of the history and physical examination, which provide the overall orientation for the subsequent physiologic evaluation.

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The Clinical Utility of Late Responses in Entrapment Neuropathies

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INTRODUCTION

There is little work supporting the use of F waves in entrapment neuropathies. It is the purpose of this manuscript to indicate that there is convincing evidence that F waves should be helpful in entrapment neuropathies.

F waves were originally described in the small muscles of the foot and hence their name. The interpretation of F waves requires an understanding of the electrogenesis of the F wave, those factors that modify the F wave, and techniques for eliciting the F wave.

THE PHYSIOLOGY OF THE F WAVE

It is now generally accepted that F waves are produced by antidromic activation of motoneurons (MNs). In healthy subjects the probability that any one motor unit (MU) will generate an F wave is small. Some stimuli in a train may not be followed by any F wave. Where F waves do follow the direct response, their shape and size changes from stimulus to stimulus (Figure 1) because motor unit action potentials (MUAPs) which generate the F wave change with each successive stimulus.

Physiological Factors Influencing the Probability of an F Wave

Electrical stimulation of peripheral nerves produces antidromic activation of motor nerve fibers as well as orthodromic impulses in sensory fibers. Both might influence the excitability of MNs and thereby the chance of an F wave. Ultimately, an F wave is dependent on an orthodromic response following the antidromic activation. This requires the orthodromic response to traverse an MN initial segment that has previously been depolarized by the initial antidromic volley. As such, any factors tending to speed up the recovery of the initial segment or which delay or increase the magnitude of any antidromically induced depolarization of the somadendritic membrane might well increase the chance of an F wave. F waves may therefore be influenced by differences in "central" excitability. For this reason, the frequency of F-wave discharge following a series of stimuli (persistence) is characteristically about 80-90% in antigravity muscles such as the abductor pollicis brevis, calf, and abductor hallucis, but only about 30-40% in the antigravity antagonists such as the forearm extensors, tibialis anterior, and extensor digitorum muscles.

The question of a selective bias in the selection of MUs in F waves is important and has been controversial. All studies questioning a selective activation of larger MUs in F waves have been performed at submaximal stimulation in comparison to the supramaximal stimulation used in the clinical situation. The diameters of the largest nerve fibers in the ventral roots are roughly twice those of the smallest ventral root fibers. Ranges of conductions at least twice the 15-20% that are normally observed in F waves have been noted in human motor fibers in individual subjects. Changes in F-wave latency are similar to changes in maximal evoked response latencies when the sites of stimulation are changed. These observations would be consistent with a
selective bias of larger MUs in F waves. This is supported by findings based on studies of single MUs in F waves as well as analysis of F-wave conduction velocities. Fortunately, whatever the situation, the observed variability in F-wave latencies is not so random or great as to preclude their clinical use.

TECHNIQUES FOR STUDYING THE F WAVE

F waves are usually recorded using surface electrodes arranged in a belly-tendon fashion with the active electrode positioned over the innervation zone of the target muscle. F waves recorded from distal muscles in the limbs are usually clearly separated from the direct motor response (M wave). The more proximal the site of stimulation, however, the greater the chance that F waves may be obscured by the preceding direct M wave. This feature of F waves provides a practical limitation of recording F waves from proximal muscles.

Supramaximal stimulation should be used for recording F waves. The size and persistence of F waves increase as the stimulus intensity increases, and supramaximal stimulation provides a physiologically consistent environment for analyzing other F-wave parameters.

F waves may be affected by a previous conditioning stimulus. As such, F waves should be recorded at rates no faster than 0.5 Hz.

METHODS FOR ANALYZING F WAVES

Individual F waves are generated by the recurrent discharges of anywhere from one to at the most a few MUs whose associated MUAPs and latencies differ. This accounts for the small size of the F wave relative to the size of the direct M potential. The moment-to-moment changes in which MUs generate F waves accounts for the inherently variable size, latency, and shape of the F wave from stimulus to stimulus (Figure 1). This inherent variability also means that the analysis of F waves requires a series of F waves and the evaluation of a number of parameters.

The most common way of assessing F waves has been to measure the shortest latency F wave following a series of 10 or more stimuli. Measuring the shortest F-wave latency, however, may be difficult. In addition, individual F waves may overlap and be confused with axon reflexes or A waves. A more reliable method of comparing latencies in F waves is to calculate the mean latency. The latter does not depend on correctly identifying and measuring the latency of the shortest latency F wave, reflects the range of F latencies, and is more reproducible than minimal latencies. For these reasons, mean latencies have been recommended by a number of studies. Recent computer-assisted analysis of F waves has used median latency values. F-wave latencies will vary with limb length and, to a lesser degree, age. Predicted F-wave latencies need to be adjusted for height or limb length. This is done best by using regression equations that include height or limb length and age. Chronodispersion refers to the difference between the minimal and maximal latencies and thereby reflects the range of latencies in a series of F waves. Persistence indicates the percentage of discernible responses, (generally exceeding 20 mV), following a series of stimuli. Identical responses in a series of F waves are called repeater waves. Because of the variability of F waves, the amplitudes of F wave are best measured as mean values and related to the amplitude of the maximum M potential, i.e., the mean F/M ratio. Normal values may be found in recent references.
Because of the inherent variability of F waves, sufficient F waves must be collected to provide representative data. Ten stimuli yielding anywhere from 7-10 F waves may suffice for most studies of persistence and latencies. However, 20 or more stimuli providing anywhere from 16-20 F waves may be needed for accurate measurements.\textsuperscript{11,13,26,30} This can be important when comparing relatively small latency differences between sides, as may be necessary in radiculopathies. A series of 20 F waves is also adequate for the measuring mean F/M amplitude ratios and the percentage of repeater waves. As few as two F waves may establish an abnormal chronodispersion if the separation in latency between these two responses is greater than normal. Accurate measurement of chronodispersion requires more than 20 stimuli, and may possibly require as many as 50-60 stimuli.\textsuperscript{11,23,25} At least 100 stimuli are required to determine the number of individual repeater waves. These data are based on recordings from antigravity muscles. Additional stimuli will be needed if recording from antigravity antagonists with their associated lower persistences.

**F WAVES AND ENTRAPMENT NEUROPATHIES**

There is evidence supporting the value of F waves in entrapment neuropathies. F-wave latencies may be prolonged in neuropathies and may be abnormal even when peripheral motor conduction studies are normal. F waves may also be more sensitive than conventional motor conduction studies in axonal neuropathies.\textsuperscript{12} Prolonged F-wave latencies exceeding 150% of the upper limit of normal have been considered suggestive of demyelination as has the absence of F-waves in the presence of relatively preserved maximum M potentials.\textsuperscript{12} F-wave latencies have been reported to be the most stable and reliable measurement for sequential nerve conduction studies in the same subjects.\textsuperscript{17}

F-wave parameters other than latency are important for defining nerve dysfunction. Abnormal F waves have a high sensitivity in acquired demyelinating neuropathies.\textsuperscript{12,16} This high sensitivity is consistent with the effects of focal proximal demyelination. Increased chronodispersion and decreased persistence may occur in up to 50% of the nerves in patients with acquired demyelinating polynuropathies and may be the only abnormality in those nerves.\textsuperscript{16} Repeater waves and mean F/M ratios are increased in patients with axonal injury. The latter is consistent with a decreased MN pool available for activation, the latter with increased motor unit size but decreased M-wave amplitudes.

**Thoracic Outlet Syndrome**

The potential value of F waves in entrapment neuropathies can be surmised from one of the earliest reports dealing with this issue.\textsuperscript{38} This study included methodological features required for meaningful F-wave studies; namely, an adequate number of stimuli and the evaluation of parameters other than latency. F waves were recorded from hypothenar muscles in five patients with true neurogenic thoracic outlet syndromes following 60 stimuli. F waves in the affected arms were prolonged by at least 2 ms in comparison to the unaffected side. The F waves in the affected arms were also prolonged in comparison to data from control subjects with the data related to arm length. F-wave persistence was also decreased in the involved arms in comparison to both the uninvolved arms as well as in control subjects. In one subject, the F-wave latency prolongation resolved following removal of the causative cervical band.

**Carpal Tunnel Syndrome**

Macleod\textsuperscript{19} compared F waves recorded from the abductor pollicis brevis (APB) in 52 healthy nerves and 147 nerves from patients with symptomatic carpal tunnel syndrome (CTS). All of the patients had prolonged median sensory conductions across the wrists. Analyses were based on responses following 100 supramaximal stimuli. The F-wave persistence was significantly decreased (p<0.001) in the symptomatic nerves. The mean persistence in the symptomatic nerves was 60% compared to about 83% in the normal nerves (Figure 2). Due to the wide range of normal values, however, persistence was considered an insensitive measure of CTS. Percent repeater waves (%RF) was also calculated where %RF was the number of recurring identical F waves divided by the total number of discernible F waves. In the control group, the mean %RF value was about 15% while in those with CTS, the mean %RF was about 60%. In 84.4% of the studies in nerves with CTS, %RF was above the reference 90% upper limit of normal. In comparison, only 35% of median distal motor latencies in the patients were prolonged. The author concluded that in injured nerves there is a reduction in the size of the alpha MN pool capable of generating F waves and an enhanced backfiring capacity in that MN pool. Since Macleod\textsuperscript{19} used 100 supramaximal stimuli, the clinical utility of this method would be limited. Nevertheless, the study indicates that F waves may be abnormal in CTS and could be clinically helpful if one were interested in a sensitive measure of the incidence of injury to motor fibers in patients with CTS.

Median F-wave latencies are usually 1-2 ms shorter than ulnar F-wave latencies when recorded from hand muscles. “Inversion” of this latency is abnormal and is consistent with CTS.\textsuperscript{22} This inversion may be due to lesions proximal to the wrist but, in the appropriate context, may readily provide evidence for injury to motor fibers in CTS not otherwise available.

Carpal tunnel syndrome has provided a laboratory for defining the potential effect of focal nerve injury on F waves. F-wave persistence may be decreased where there is evidence of axonal injury defined either by decreased M-wave amplitudes\textsuperscript{24} (60 supramaximal stimuli) or abnormal spontaneous activity\textsuperscript{10} (20 supramaximal stimuli). Chronodispersion may be significantly
increased (p<0.025) in those patients with prominently prolonged median distal motor latencies (DML) (i.e., >5.7 ms in comparison to an upper limit of normal of 4.2 ms) in comparison to those with lower DMLs. Repeater waves may be significantly higher (p<0.001) in those with APB abnormal spontaneous activity. F-wave amplitudes (mF/M ratios) are almost always larger (p<0.001) in the hands with the more prolonged DMLs in those with bilateral CTS.10

The use of F waves to help define proximal injury in CTS was also the basis of one of the earliest reports of F waves in entrapment neuropathies.6 Regression lines with 99% confidence limits were determined in normal subjects for M latency versus minimal F-wave latency stimulating at the elbow and recording from the APB. In 3 of 30 patients with CTS, the F latencies were outside the 99% confidence limits consistent with an associated proximal lesion.

Leffler and colleagues18 evaluated a large series of hands (203) in patients with possible median nerve injury at the wrist. Based on a multivariate logistic regression model using clinical, DML, and F-wave latency information, the median F-wave latency has been found to be an independent predictor of CTS study. The “gold standard” for a diagnosis of CTS was the physician’s diagnosis after formal clinical and electrodiagnostic studies. These data—both DML and F waves—were acquired using a standardized computer-based stimulation and recording system. These types of systems and analyses—automated recordings and multivariate regressions—may soon become routine and F waves will be part of these evaluations.

**Hemifacial Spasm**

Hemifacial spasm (HFS) can be considered an entrapment neuropathy due to vascular compression of the facial nerve in the brainstem. F waves have been recorded in patients with HFS (32 supramaximal stimuli) recording from the mentalis muscle stimulating the marginal mandibular branch of the facial nerve.14 In this study, the low cutoff filter was set at 100 Hz in order to minimize interference from the associated M wave. In 14 patients, F waves were suppressed during inhalation of anesthesia on the normal side, but in only 4 of the 14 nerves with HFS. F-wave suppression occurred following surgical decompression at the same time as there was disappearance of the aberrant motor responses characteristic of HFS. In addition, F-wave studies of the involved facial nerves revealed decreased persistences, F/M amplitude ratios, and response durations as well as increased latencies after surgery in comparison to studies prior to surgery. These studies, therefore, not only support increased facial nucleus excitability in HFS and indicate a clinical use of F waves, they also reinforce the discernible changes in F-wave parameters that may occur with focal nerve entrapment.
Radiculopathies

Nerve roots are susceptible to focal injury due to limited collagen supporting tissue and relatively fixed anatomical constraints limiting the ability of nerve roots to move away from a site of compression. As such, symptomatic root injury can be considered to fall under the rubric of entrapment neuropathies.

The role of F waves in radiculopathies has been controversial. Theoretical arguments against the use of F waves have included the long course of F waves and subsequent “dilution” of any change in latency, as well as the multiple root innervation of most muscles. The latter argument is not supported by observations in patients with other types of focal proximal nerve injury. The former argument is particularly irrelevant since F-wave parameters such as chronodispersion are arguably of unique value in circumstances where some axons may be injured and others not. Studies that have questioned the use of F waves in radiculopathies could be criticized today for their methodology. For example, in one commonly cited study, F waves were recorded following only 10 stimuli from a muscle with a characteristically low persistence (the extensor digitorum brevis) such that analyses were made on only 3-4 F waves.

Recent studies examining F-wave parameters in addition to latency have supported the value of F waves in the electrophysiological evaluation of radiculopathies. In a study of 96 patients with L5/S1 radiculopathies, 2 over 40% of these patients had absent or prolonged F-wave latencies and 76% had abnormal chronodispersion. In a similar series of patients with L5/S1 injury and using similar F-wave parameters, needle electromyography (EMG) studies were abnormal in 70% while F-wave abnormalities were present in 69%. F-wave abnormalities were found in 13 of the 23 patients where the only evidence for ongoing root injury was needle EMG abnormalities in the parasartnal muscles thereby providing unique evidence of injury to anterior primary rami. In another series of 95 patients with L5 or S1 root lesions, F waves were abnormal in 70% of the patients and needle EMG was abnormal in 77%. Abnormal F-wave parameters for this study included chronodispersion. Using similar criteria for normal versus abnormal F waves, improvement in F-wave parameters has been correlated at a statistically significant level with recovery in strength following surgery. In 20 patients with surgically verified L4, L5, or S1 radiculopathies, needle EMG was abnormal in 12. F-wave abnormalities, including persistence, were present in eight patients. In three of these eight patients, the needle examinations were unrevealing. Using increased minimal latencies or chronodispersion, 69% of the tibial or peroneal nerves studied in 10 patients with spinal stenosis had abnormal F waves while this was true in only 24% of the nerves in 11 patients with L5/S1 root compression syndromes. In all of these patients, 3 minutes of standing produced increased F-wave abnormalities, most noticeably up to an 8 ms increase in chronodispersion. This study supports the idea that the frequency of F-wave abnormalities may be higher when multiple roots are involved such as in spinal stenosis. This study also supports the idea of “provocative” F-wave studies, namely definable changes in F waves associated with maneuvers that reproduce patients’ symptoms. A composite nerve conduction measurement using multivariate logistic regression analysis including tibial and peroneal F-wave latencies (40 stimuli) recorded from foot muscles is reported to have diagnostic specificity of 84.3% and a sensitivity of 83.3% for L5, S1 root injury. The electrophysiological data was recorded using a computer-based automatic nerve conduction testing instrument. Current reports therefore indicate that F waves recorded and analyzed appropriately may be a sensitive and valuable means of studying L5/S1 radiculopathies. These radiculopathies are of course common.

The value of F waves in cervical radiculopathies is more problematic. Most cervical radiculopathies involve the C5, C6, or C7 roots—roots that do not readily lend themselves to F-wave studies due to their relatively proximal innervation. A recent report, however, describes radial F waves recorded from the extensor indicis. These F-wave latencies were reported prolonged in patients with C7 radiculopathies.

CONCLUSION

F waves are a valuable tool for the evaluation of peripheral nerves. This should be true for entrapment neuropathies, but will require a knowledgeable use of F waves and analysis of F-wave parameters other than latency. The potential availability of automated analysis of F waves should enhance this process.

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The Clinical Utility of Late Responses in Entrapment Neuropathies

Controversies in Evaluating Brachial Plexopathies and Thoracic Outlet Syndrome

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INTRODUCTION

The term thoracic outlet syndrome (TOS) is applied to a number of conditions attributed to compromise of peripheral nerve fibers (components of the brachial plexus), blood vessels (subclavian/axillary artery or vein), or both, at one or more points between the base of the neck and the axilla.\(^\text{17}\)

Thoracic outlet syndrome is a confusing topic for several reasons, the most basic being that typically the term is used in the singular sense, when it is actually an umbrella title for a number of very distinct disorders that vary in their validity, and which have little in common beyond their known or presumed site of occurrence.\(^\text{17}\) (It should be stressed at this point that, of the various types of TOS, only one is controversial and it alone is the principal subject of this manuscript.)

There have been at least two classifications of TOS: The original one, proposed by Peet and colleagues in 1956, and one advanced by Wilbourn some 3 decades later, in 1984; the latter has undergone various modifications.\(^\text{2,5,14,17}\) Peet and colleagues’ classification consisted of the following: (1) cervical rib syndrome; (2) scalenus anticus syndrome; (3) subcoracoid-pectoralis minor syndrome (i.e., the hyperabduction syndrome); (4) costoclavicular syndrome; and (5) first thoracic rib syndrome.\(^\text{2}\) This original TOS classification was based principally on what was assumed at that time to be the source of symptoms and not on the structures affected. It has little current relevance, because most of the entities included under it have been either abandoned or significantly modified with the passage of time.

In contrast to the original classification, the current one subdivides TOS by the particular neurovascular structure(s) compromised: plexus, artery, or vein. This current classification consists of the following: (1) vascular TOS, subdivided into arterial TOS and venous TOS; (2) neurologic TOS, subdivided into true and disputed TOS; and (3) vascular plus neurologic TOS, subdivided into traumatic TOS and sometimes disputed TOS.\(^\text{2}\) This classification appears to be a reasonable approach to the topic since, with the exception of traumatic TOS, seldom is more than one of these structures affected simultaneously.\(^\text{14}\)

All of the TOS subgroups, with the glaring exception of disputed neurologic TOS (N-TOS), share several features in common. All of them are rare entities, are unilateral disorders, have an uncontested cause, have a characteristic clinical presentation, manifest objective clinical findings, produce generally acknowledged abnormalities on various objective confirmatory tests, and are uni-
versally acknowledged disorders. Disputed N-TOS is highly controversial, in large part because it lacks all of the previously noted characteristics.\textsuperscript{13,15,17}

**VASCULAR THORACIC OUTLET SYNDROME**

Both the arterial and the venous types of vascular TOS are rare lesions. The arterial type, also called the arterial cervical rib syndrome, is due to a congenital bony anomaly, generally a full-formed cervical rib or an abnormal first thoracic rib. This abnormal structure compresses the subclavian artery as the latter passes from the thorax to the upper limb. Distal to the compression site, post-stenotic dilatation develops because of turbulent blood flow. Thrombi form on the wall of the dilated segment and then embolize peripherally. Depending upon the site, severity, and persistence of arterial occlusion, symptoms range from intermittent bouts of strictly unilateral Raynaud’s syndrome to sudden, severe, tissue threatening ischemia of the more distal portion of the limb. This disorder is diagnosed by dynamic venography (because nearly always a bony anomaly is present at the base of the neck), and by various vascular procedures, particularly transarterial arteriography. Prompt surgical treatment is mandatory. In fact, this entity sometimes is considered a surgical emergency by many surgeons.\textsuperscript{4,17}

The venous type of vascular TOS also is known as effort thrombosis, Paget-von Schroetter disease, and idiopathic, spontaneous occlusion of the subclavian-axillary vein. The cause of this disorder is somewhat uncertain because typically there is no bony anomaly present in the thoracic outlet, as there is with the arterial type. Most investigators believe that it is caused by congenital narrowing of the space that the subclavian vein passes through to reach the innominate vein. The symptoms of venous vascular TOS often develop rather abruptly, with the entire limb becoming swollen, cyanotic, and somewhat painful. Dilated veins, serving as collateral channels, appear over the lateral chest. Dynamic venography is reported to be the most reliable diagnostic test. Although intuitively one would assume that the appropriate treatment is surgical thrombectomy, this is seldom successful alone because the thrombosis often reforms. The most efficacious treatment appears to be thrombolytic therapy, often followed by elective removal of the first thoracic rib (the latter is performed on the assumption that the vein is being compressed in a congenitally tight costoclavicular space). Sometimes balloon dilation of a stenotic segment of vein, or vein patch angioplasty, is required.\textsuperscript{7,17}

Neither the arterial nor the venous types of vascular TOS primarily affect the peripheral nervous system. Although neurological symptoms such as pain, paresthesias, fatigue, and weakness can occur with vascular TOS (particularly the arterial type), these are secondary in nature, caused by the initial compromise of the vascular system, and are not the result of simultaneous brachial plexus damage. The electrodiagnostic (EDX) examinations with both types of vascular TOS are normal.

**TRAUMATIC NEUROVASCULAR THORACIC OUTLET SYNDROME**

Traumatic neurovascular TOS is an uncommon disorder that most often results from mid-clavicular fractures. It almost exclusively affects adults. (The clavicle is the most commonly fractured bone in the human body.) Either at the time of the injury, or up to several months later as a result of nonunion or the formation of a large callus, one or more of the neurovascular structures situated between the clavicle and the first thoracic rib are compromised. The peripheral nervous system structures injured are typically the proximal portions of one or more cords of the brachial plexus. These may be damaged either directly (e.g., by a blunt trauma, by bony fragments, or by a compressive callus) or indirectly (e.g., by hematoma formation or pseudoaneurysm resulting from primary vascular injury). Invariably, plain radiographs of the clavicle reveal abnormalities. With neurological damage, the EDX examination shows abnormalities whereas with vascular damage, arteriograms, venograms, and other diagnostic vascular procedures reveal abnormalities. Many of these patients require operative treatment, often with several surgeons—neurologic, orthopedic, and vascular—all involved.\textsuperscript{2}

**TRUE NEUROLOGIC THORACIC OUTLET SYNDROME**

True N-TOS also called neurogenic cervical rib syndrome and cervical rib and band syndrome, was first described by Thomas and Cushing in 1903. Refinements in its clinical presentation (i.e., that the muscles composing the lateral thanar eminence are the most severely affected) were provided by Thorburn in 1905. Unfortunately, almost immediately after it was described, true N-TOS was confused with the then unknown carpal tunnel syndrome (CTS). For this and other reasons, it was soon plunged into a sea of medical confusion, from which it was not extracted until 1970, when Gilliatt and co-workers described its characteristic clinical, radiographic, and electrophysiological presentation.\textsuperscript{3,13,14,17} Patients diagnosed with true N-TOS generally are young to middle-aged, and far more often women than men. It is caused by two linked congenital anomalies: (1) a rudimentary cervical rib, or an elongated C7 transverse process, from the tip of which, (2) a radiolucent taut band extends to the first thoracic rib. The proximal lower trunk of the brachial plexus, or more often, the distal T1 (and to a lesser extent, the distal C8) anterior primary ramus that will form the lower trunk is stretched and angulated around this band. Characteristically, motor changes overshadow sensory changes, to the point that Gilliatt himself referred to this disorder as a “motor syndrome.”\textsuperscript{17} Although many patients report experiencing intermittent aching along the medial forearm and hand, (sometimes for many years)
few seek medical attention for these sensory complaints. Instead, patients first ask for medical help upon noticing that one of their hands is weak and wasted. Preferential involvement of the lateral thenar muscles (i.e., the median nerve-innervated) is characteristic of this disorder. In approximately one-fourth of patients, only these muscles are wasted, although all of the intrinsic hand muscles are weak. In the remaining three-fourths, all of the intrinsic hand muscles are wasted and weak, although invariably the lateral thenar muscles are the most severely involved. In most patients, a patchy sensory loss can be demonstrated along the medial forearm and hand. Helpful ancillary studies include plain neck radiographs and the EDX examination. The neck radiographs demonstrate the rudimentary cervical rib or elongated C7 transverse process (often with a larger cervical rib on the contralateral, asymptomatic side). The EDX studies reveal a chronic, axon loss, lower trunk brachial plexopathy whose features, because of the T1 anterior primary ramus emphasis and the marked chronicity of the lesion, are almost pathognomonic. Patients with true N-TOS require surgical treatment, specifically, sectioning of the cervical band via a supraclavicular approach. Following operation, sensory symptoms generally promptly resolve. The patients often report some subjective increase in hand strength, although this is difficult to prove. In any case, hand wasting is arrested but, because of the nerve fibers affected and the chronicity of the lesion, atrophy of the intrinsic hand muscles does not change significantly.

**DISPUTED NEUROLOGIC THORACIC OUTLET SYNDROME**

Disputed N-TOS has accumulated a great number of names since it was first designated disputed N-TOS in 1984; these include aspecific N-TOS, assumed N-TOS, nonspecific N-TOS, and symptomatic N-TOS. This reputed lesion, similar to true N-TOS, has a long history. It first appeared in 1903 when Bramwell described a patient who probably had true N-TOS. Unfortunately, that was the same year that Thomas and Cushing were to describe true N-TOS, and Bramwell did not obtain cervical radiographs on his patient. Consequently, he attributed his patient’s symptoms to a lower trunk brachial plexopathy with the affected axons being compressed by a normal first thoracic rib. A version of this disorder, the scalenus anticus syndrome, frequently was diagnosed in the United States, beginning in 1935. That was the year that Ochner, Gage, and DeBakey claimed that the scalenus anticus muscle could compromise the lower trunk of the brachial plexus and the subclavian artery in certain predisposed individuals, by establishing a “vicious circle” of compression, even without any type of bony anomaly being present. Thus, this disorder was described as being cervical rib syndrome sans cervical rib. It was a popular diagnosis for approximately 20 years, and anterior “scalenotomies were carried out with abandon” to treat it. However, its popularity plummeted after real causes for upper limb complaints were delineated, specifically cervical radiculopathy in 1943 and CTS in 1953. Only in retrospect did many surgeons in the 1960s concede that their surgical failure rates with scalenus anticus syndrome had been inordinately high (up to 60%). Disputed N-TOS experienced its third, and current, rise in popularity in the 1960s. In 1962, Claggett, a thoracic surgeon, stated that the optimal surgical treatment for all reported types of TOS (as described by Peet and colleagues) was removal of the normal first thoracic rib, because in all patients it acted as one arm of vice (the other arm varied, depending upon the exact site of the lesion), compressing the neurovascular structures. Claggett also contended that thoracic surgeons should be performing this type of surgery (even though they may be viewed as “claim jumpers” by their neurosurgical colleagues), because they possessed the requisite surgical expertise to remove the first thoracic rib. Claggett’s presentation generated much interest but few TOS operations, because the surgical procedure he was advocating to remove the first thoracic rib (the posterior thoracotomy approach) was formidable, and left an extensive scar. In 1966, Roos, a general surgeon, described a surgical technique for removing the first thoracic rib through the axilla—the transaxillary first rib resection. Because this surgery left an inconspicuous scar—and therefore women who underwent the procedure could subsequently wear swimsuits and evening gowns—it was considered acceptable and was offered to patients who “had only subjective complaints.” Within a few years, transaxillary first rib resections became as popular as anterior scalenotomies had been a few decades earlier, and they were being performed on essentially the same type of patient—a young to middle-aged woman who had vague forequarter sensory symptoms.

In contrast to all the other TOS subgroups, disputed N-TOS has extremely broad and poorly defined boundaries. Almost every aspect of it is controversial, including its incidence, etiology, underlying pathophysiology, lesion location within the thoracic outlet, clinical profile, ancillary diagnostic procedures of value, optimal treatment, and if the latter is surgical, the operation(s) of choice (Figure 1). Surprisingly, many of the disputes on these points are among the proponents of this entity, who often seem to agree upon little about it except that it is a common disorder, it affects women more than men, and that it frequently requires surgical treatment. Initially, disputed N-TOS was attributed to simultaneous compromise of both nerve fibers and blood vessels. However, due principally to the insistence of Roos, most proponents now contend that it affects only portions of the brachial plexus (i.e., it is solely a neurological disorder). Over the years proponents have linked it increasingly to trauma and repetitive limb use. Because of this, unlike the other TOS subgroups, it is in the legal/quasi-legal sphere. Proponents also claim that, unlike all of the other types of TOS, it is a common disorder, affecting up to 8% of the population. Nonetheless, it has no characteristic clinical presentation. Some proponents believe it causes only sensory symptoms, without objective findings, whereas others contend it produces some detectable (although usually “subtle”) physical examination changes as well.
was reported to cause symptoms solely in a C8 distribution, the so-called “lower plexus presentation.” However, an upper plexus presentation was described in the early 1980s, in which symptoms reputedly are experienced principally around the upper arm, shoulder girdle, and posterior neck and head. Typically, the diagnosis is made whenever the patient has a “characteristic clinical presentation,” whatever that may be, and some ancillary diagnostic procedure is positive. However, there is absolutely no agreement among the various proponents concerning which of the many available ancillary procedures are valuable in this regard. At least 20 have been proposed, including physical examination, electrophysiologic examination, neuroimaging, and miscellaneous procedures. None is universally accepted, although the most widely employed is the elevated arm stress test (EAST procedure), first described by Roos in 1966. Yet, this is a non-specific test, being positive in great numbers of normal persons and particularly in those with CTS. Moreover, it is a claudication test (i.e., its end point is arterial compromise), a fact that is at variance with the concept that disputed N-TOS involves the brachial plexus and not the blood vessels.

Concerning the utility of the EDX examination with this disorder, its proponents hold three different views: (1) an upper limb motor nerve conduction study can definitely diagnose the entity. Urschel, a thoracic surgeon, is the most vocal advocate of this approach; unfortunately, he appears to have a poor understanding of both motor nerve conduction studies and peripheral nervous system anatomy; (2) although an EDX examination cannot diagnose this entity, it is nonetheless useful, because it can detect other peripheral nervous system lesions, such as CTS and cervical radiculopathies, with which disputed N-TOS can be confused; and (3) it is of no value at all in the assessment of patients.
with this suspected disorder. Roos, considered by many to be the father of disputed N-TOS, has stated this opinion repeatedly.2,6,9,12,17

Disputed N-TOS is treated both conservatively (with physical therapy, postural exercises, etc.) and surgically. Operations are performed on a considerable number of patients who are said to have intractable pain. There is considerable disagreement among the various proponents regarding which operation is optimal. Initially, almost all TOS surgery after 1966 consisted of transaxillary first rib resections to treat the lower plexus presentation. However, a few surgeons had never abandoned operation on the scalene muscles, although the procedure had been expanded from an anterior scalenectomy to anterior and middle scalenectomies. Roos contends that the latter operation should be reserved for patients with the upper plexus presentation. Nonetheless, some surgeons perform either operation, regardless of presentation (i.e., upper plexus or lower plexus type), whereas others perform one and, if symptoms are not relieved, they perform the other, regardless of presentation. Still others perform both procedures as two portions of the same operation. Moreover, other approaches for first rib removal, including the supraclavicular and posterior thoracotomy approach, are sometimes employed (the latter particularly for reoperation). In addition, some surgeons perform epineurectomies of various portions of the brachial plexus.6,10,11,15

The results of TOS surgery are vigorously debated. For many years high operative success rates, typically approaching 90%, were claimed, but these almost always were reported by the surgeons who had performed the operations after short follow-up periods. Over the past 2 decades, far more pessimistic results have been published, with much lower operative success rates (35-65%). In one report, based on a 5-year survey of the operative results on Workman’s Compensation patients in the state of Washington, the surgical success rate was described as “dismal.”13,15,17

The many skeptics of disputed N-TOS have several problems with the diagnosis. First, it is often being made by physicians who have had little if any training or experience in diagnosing neurologic disease, particularly brachial plexopathies, or in formulating the differential diagnosis for them. Also, many proponents appear remarkably naïve regarding functional symptoms and secondary gain factors in patients who have personal injury litigation or Workman’s Compensation claims pending. In addition, some proponents now claim that this type of TOS can precipitate a great variety of additional peripheral nervous system and musculotendinous disorders, including cervical radiculopathies, various entrapment neuropathies, etc., many of which may require additional operative treatment. Finally, most skeptics have serious concerns regarding the number of what they consider to be completely unnecessary surgical procedures being performed on patients who are labeled with this diagnosis, because of the potential for these operative procedures to cause grave harm to the patients, such as infection, severe permanent brachial plexus injuries, and even death by exsanguination.12-17

An important point is that the skeptics of disputed N-TOS do not hold identical opinions regarding it. Many consider it to be a nonexistent disorder, one formulated primarily because an operative technique had been devised to remove the normal first thoracic rib while leaving an inconspicuous scar. Others believe that it probably exists, although its incidence undoubtedly is far less than its proponents proclaim, but contend that its diagnosis is markedly difficult and relies principally on the “gut feeling” of the clinician making the diagnosis. Unfortunately, many such clinicians appear to experience such an “abdominal experience” whenever they encounter any patient with vague four-quarter symptoms.

CONCLUSIONS

The term TOS encompasses several different disorders, only some of which involve peripheral nervous system structures and only one of which is highly controversial. However, the controversial one is also the only one that occurs with any frequency.

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INTRODUCTION

The subject of thoracic outlet syndrome (TOS) remains one of the most contentious in all of medicine. Virtually every aspect of this disorder, including its existence, name, clinical features, methods of diagnosis, pathophysiology, and treatment all evoke varying degrees of controversy and debate. Indeed, mention of the diagnosis of TOS, particularly among neurologists and physiatrists, will often elicit obvious expressions of disbelief, snickers, and scornful looks. Why, then, would a neurologist agree to debate in favor of this concept, particularly against one of its most vigorous and knowledgeable opponents? The main reason is that is author happens to believe that this is a useful diagnosis, one that can be made by careful clinical assessment and can lead to an effective treatment program in the majority of patients so diagnosed.

This manuscript will focus on the so-called “nonspecific neurogenic” TOS (NSN-TOS) or “symptomatic type,” more preferable terms than the more pejorative (although accurate) “disputed” TOS favored by others. The main reason is that is author happens to believe that this is a useful diagnosis, one that can be made by careful clinical assessment and can lead to an effective treatment program in the majority of patients so diagnosed.

This manuscript will focus on the so-called “nonspecific neurogenic” TOS (NSN-TOS) or “symptomatic type,” more preferable terms than the more pejorative (although accurate) “disputed” TOS favored by others. There is not much to say in favor of the usefulness of electrodiagnostic (EDX) techniques to investigate this disorder. A variety of such procedures have been proposed and they can be helpful in excluding alternative, or co-existing, conditions. Similarly, this manuscript will not be promoting the surgical treatment of NSN-TOS, although it can be successfully utilized for patients who remain symptomatic despite aggressive and skillfully applied non-surgical techniques.

This author's experience with TOS is primarily among instrumental musicians, a group that seems to be particularly prone to develop symptoms. This diagnosis as been made by this author in 75 instrumentalists out of the total of 1480 evaluated over the past 25 years. No patient in this series, or in any other involving musicians, to this author's knowledge, has been diagnosed with the true neurogenic form. Lascelles and colleagues identified 31 patients with different forms of TOS, including 5 with what he called the “pain/paresthesia” type. One young woman in this group played the flute. Charness identified TOS in 59 limbs in 40 of 117 instrumental musicians (34%) with nerve entrapment syndromes. In a series from the Massachusetts General Hospital Musical Medicine Clinic, TOS was identified in 7% of the 1000 instrumentalists seen (included in the 32% with a neurologic diagnosis). Newmark discussed three musicians with symptoms suggesting NSN-TOS in his 1996 report. In a series from the United Kingdom, Wynn Parry included 14 patients diagnosed with TOS among 257 musicians with a “clear diagnosis” of upper limb problems out of the 617 seen at that clinic.
MECHANISMS OF SYMPTOM PRODUCTION

Most authors have assumed that compression of the “neurovascular” structures in the thoracic outlet is largely responsible for the production of symptoms. Three potential sites of compression have been identified, including the interscalene triangle, the costoclavicular space, and the retro- or subpectoralis minor space. While it is generally agreed that anatomical factors may predispose to the development of TOS, including the presence of cervical ribs or possibly fibrous bands, some other process also seems necessary. Specifically, some form of trauma, either an acute event or, as in the musician population, repetitive stress and postural factors, is hypothesized. Sanders and Hammond have suggested that scarring of the scalene muscles following trauma might lead to compression of the brachial plexus and offer as evidence a single histologic study in support of this concept. Pascarelli and Hsu, reviewing work-related upper extremity symptoms in 485 patients (including 134 musicians), conclude that postural misalignment is a major factor in producing the symptoms of NSN-TOS in the 339 persons so diagnosed in their series. Lindgren and colleagues have identified a “functional” mechanism for production of symptoms of TOS, based on the demonstration of subluxation of the first rib at the costotransverse joint associated with work-related upper extremity stress, leading to tension in the scalene muscles and subsequent brachial plexus compression.

Brantigan and Roos have emphasized the role of anatomic abnormalities, especially fibrous bands, in the etiology of symptoms in neurogenic TOS. They have suggested that the major difference between NSN-TOS and true neurogenic TOS is time and advocate treatment before the clinical (and electrical) indicators of nerve and muscle injury have appeared. Wilbourn and Porter have argued strongly, and effectively, against this idea. I have identified five patients in my series with NSN-TOS who have had symptoms for 8-12 years. If there were an evolution from NSN-TOS to the true neurogenic form based primarily on time, it would seem likely that these patients would have at least some evidence of impairment clinically or electrically. They, in fact, did not.

Stretching of the brachial plexus has also been suggested as a mechanism for symptom production. Klein first suggested the term “droopy shoulder syndrome” for painful shoulders associated with presumed stretching of the brachial plexus. Swift and Nichols reported 10 such patients, with pain or numbness in one or both upper extremities associated with this configuration, and suggested that brachial plexus stretch accounted for most of the cases of NSN-TOS in their experience. Approximately half of this author’s patients with TOS have the long neck, sloping shoulders, and horizontal clavicles characteristic of this disorder.

Ide and colleagues studied 150 patients with TOS, injecting contrast material into the supraclavicular plexus under fluoroscopic guidance with and without provocative maneuvers. They found that 18% had only compression of the plexus, 8% showed evidence of stretching only, and 74% demonstrated a combination of the two mechanisms. Eleven of the 12 patients showing stretch only had the droopy shoulder configuration.

CLINICAL FEATURES OF NONSPECIFIC NEUROGENIC THORACIC OUTLET SYNDROME

One of the most disturbing aspects of NSN-TOS disorder is the lack of consistent and validated clinical diagnostic criteria. In a series published by this author, the following five features were utilized. At least four features were required to be satisfied for the diagnosis to be made.

1. The patients have arm pain, most commonly distal and usually maximal along the ulnar aspect of the forearm and hand. Less commonly, there may be pain in the upper arm and ipsilateral upper trunk.

2. Sensory symptoms, including numbness, tingling, burning, or a swollen feeling are described, again most commonly along the ulnar forearm and hand.

3. The symptoms are associated with or aggravated by specific positions, such as holding the arm at or above shoulder level.

4. Symptoms can typically be provoked by specific maneuvers, including hyperabduction of the arm, abduction and extension of the arm, and downward traction on the arm, often with internal rotation at the shoulder.

5. Despite these symptoms, the neurologic examination is virtually always normal, with no demonstrable weakness, sensory loss, or reflex change. Extreme care must be taken to exclude apparent weakness related to pain or poor effort and subjective, often non-anatomic sensory impairment.

Some patients will have tenderness in the supraclavicular fossa overlying the scalene muscles or over the pectoralis minor insertion and others may have a Tinel sign with percussion in these areas. However, these signs are not sufficiently helpful to use as additional diagnostic criteria.

A variety of provocative maneuvers have been advocated over the years for helping to identify patients who have NSN-TOS. Unfortunately all of these, including the extended arm stress test.
(EAST) as described by Roos, can be criticized as being non-specific or irrelevant.

Using the criteria outlined above, this author identified NSN-TOS in 75 instrumental musicians, including 56 women and 19 men. These ranged in age from 15-47 years at the time of diagnosis (mean = 25 years). The disorder was found in all instrument groups, including 32 bowed string players (25 violinists or violists), 19 keyboard instrumentalists, and 13 woodwind players (9 flutists). The only group showing a significant lateralization were the violinists/violists, with 19 having left arm symptoms and 8 right arm symptoms (2 bilateral).

DIFFERENTIATION OF THORACIC OUTLET SYNDROME FROM OTHER DISORDERS

A number of identifiable syndromes can be considered in the differential diagnosis. These would include cervical radiculopathy, neuralgic amyotrophy (before the appearance of obvious weakness and atrophy), carpal tunnel syndrome, ulnar neuropathy, and other nerve entrapments. The clinical features of these entities, with characteristic muscle weakness, variable sensory loss, and reflex changes are well known and need not be elaborated further here. The major problem confronting the clinician is to distinguish patients with NSN-TOS from the large and heterogeneous group presenting with regional cervicobrachial pain syndromes, especially those resulting from trauma. This can be accomplished in many cases by relying heavily on adherence to the criteria outlined earlier. The large majority of patients with TOS have unilateral or clearly asymmetrical symptoms which, at least early in the course, are position-dependent. The ability to reproduce the symptoms specifically and repeatedly with provocative maneuvers is also a dependable test, particularly in the group with droopy shoulder configuration, where depression of the shoulder with internal rotation for 1 minute or less produces symptoms on the affected side, with immediate relief on releasing the downward traction on the arm.

Howard and colleagues attempted to increase the sensitivity of clinical testing with provocative maneuvers by utilizing quantitative touch threshold and grip-strength devices at rest and in provocative positions in patients with TOS. They reported a sensitivity of 82% and a specificity of 100% for “clinically severe” brachial plexus compression (as judged by the EAST and Tinel signs). They further indicated that, in 16 of 17 patients tested before and after surgery, the response pattern reverted to normal.

A variety of ancillary procedures have been advocated for aiding in the diagnosis of NSN-TOS. Cervical spine radiographs can identify the elongated C7 transverse process or cervical rib but, in the absence of the clinical and electrical findings of true neurogenic TOS, these abnormalities are not helpful in diagnosis. Nannapaneni and Marks reported 51 patients with “neurogenic TOS” identified and surgically treated in a 13-year period. All had radiographic abnormalities, including 5 with complete and 34 with incomplete cervical ribs and 20 with enlarged transverse processes. It is not possible to determine whether any (or all) of these had true neurogenic TOS, since they found sensory abnormalities in 71%, motor weakness in 59%, and atrophy in 25%. They also found EDX testing to be negative, except when there was muscle wasting and then characterized it as “nonspecific.” Gillard and colleagues utilized ultrasound and helical CT in 48 patients, presumably with NSN-TOS. These were effective in identifying vascular compression, but they questioned whether these studies are useful in establishing a diagnosis of TOS.

Electrodiagnosis of NSN-TOS has almost as long and equally controversial history as does the clinical diagnosis. Most experienced EDX physicians would agree that electrophysiologic confirmation of this disorder is not possible. They would also agree that an EDX study is particularly useful in excluding a number of alternative diagnoses. Recording of the ulnar motor conduction velocity across the “thoracic outlet,” as promoted by Urschel and colleagues, has been effectively shown to be of no value. A number of alternative EDX studies, including F-wave analysis, cervical root stimulation, and somatosensory evoked potentials, which at least theoretically should be helpful in studying this area, have proved disappointing. Magnetic stimulation may overcome some of the shortcomings of proximal electrical stimulation in this situation. Misawa and colleagues compared symptomatic and asymptomatic limbs in patients with TOS as well as a healthy control group and found a significant prolongation of compound muscle action potential latency, recording both abductor pollicis brevis and abductor digiti minimi, in the symptomatic limbs.

TREATMENT OF NONSPECIFIC NEUROGENIC THORACIC OUTLET SYNDROME

The debate regarding treatment of NSN-TOS has proven every bit as vigorous and acrimonious as the various aspects discussed above. While even the most aggressive among the surgical specialists treating these patients would agree that nonsurgical options should be tried first, the implication is that all such methods have been exhausted by the time surgery is carried out.
A variety of surgical approaches have been advocated, often depending on the distribution of symptoms and the purported site of compression or stretching, and a wide range of results have also been described. Much of this variability can be attributed to failure to differentiate the true from the non-specific neurogenic types in some series as well as differences in duration of follow-up and rigor in clinical assessment. The potential dangers in surgical management have also been widely quoted, although some authors would obviously dispute the likelihood of injury to the brachial plexus.

The vast majority of patients with NSN-TOS can and should be managed non-surgically, with a combination of postural modification, exercise, and pain control. A number of therapeutic regimens have been suggested over the years, beginning with the exercise program described by Peet and colleagues. Most such regimens attempt to normalize the neck and upper trunk relationships, thereby minimizing the potential compression or stretch at the interscalene, costoclavicular, or sub-pectoralis minor sites. Lindgren and colleagues have proposed a series of exercises to reduce the reported subluxation of the first rib in patients with this demonstrated abnormality. The addition of a comprehensive pain management program, with all the tools currently available, may be necessary in those patients who progress to a chronic regional pain syndrome. The combination of medication, psychotherapy, nerve blocks, and even electrical stimulation or infusion techniques may be required to provide symptomatic relief, particularly in those patients who decline or are not offered surgical treatment.

Of the 75 instrumental musicians diagnosed with TOS in the authors study, only 2 have undergone surgical decompression, both successfully. Approximately 75% have responded favorably to conservative treatment, including 60% who have either no symptoms or mild and nondisabling complaints, and another 15% who are limited in their ability to play but continue in their careers or avocation. Most of the remaining 25% have been lost to follow-up and their current status cannot be documented. A few who did not improve have been offered surgical treatment, but have declined.

CONCLUSION

Nonspecific neurogenic TOS represents an identifiable entity that commonly produces pain and sensory symptoms in the upper limb, particularly among certain occupational groups, as well as those suffering acute trauma. The combination of upper limb pain and positional paresthesias suggests that intermittent compression, or stretching, of the brachial plexus is responsible for the production of symptoms. Nonspecific neurogenic TOS remains a clinical diagnosis since reliable diagnostic studies that can confirm it are not yet available. The majority of such patients can be helped by nonoperative treatment; only a small minority require a surgical approach. Lacking definitive clinical, electrophysiologic, radiographic, or other criteria, NSN-TOS is likely to remain a source of controversy, potentially to the detriment of the afflicted patient.

REFERENCES

1. The **best** screening test for carpal tunnel syndrome (CTS) is:
   A. Median motor latency to lumbrical.
   B. Median sensory latency to digit 2.
   C. Median/ulnar motor latency to interosseus.
   D. Sensory nerve action potential (SNAP) amplitude to digit 1.
   E. Median/radial sensory latency to digit 1.

2. Which is the **most** specific and sensitive physical finding in CTS?
   A. Phalen sign.
   B. Wrist ratio.
   C. Reduced 2-point discrimination.
   D. Tinel's sign over median nerve at wrist.
   E. Tethering sign.

3. In order to estimate the normal compound muscle action potential (CMAP) amplitude of bilateral CTS, which is the **best** method?
   A. Kimura's values in his text (3rd edition).
   B. 10 mV +/- 2.
   C. Use CMAP of abductor digit 5.
   D. Add 30% to CMAP of non-dominant hand.
   E. Stimulate distal to carpal ligament and measure.

4. In order to minimize the phase cancellation phenomenon in sensory conduction what should the "inter-electrode" separation be when recording?
   A. 2 cm.
   B. 10 mm.
   C. 6 cm.
   D. 4 cm.
   E. It really doesn't matter.

5. What happens if both e-1 and e-2 are both placed over the thenar muscle?
   A. Latency shortens.
   B. Latency lengthens.
   C. CMAP gets larger.
   D. CMAP gets smaller.
   E. Nothing happens.

6. What happens if one unintentionally stimulates both median and radial nerves simultaneously while recording the SNAP on digit 1?
   A. Diagnosis of CTS will be missed.
   B. False positive diagnosis of CTS.
   C. If CTS is present the Bactrian sign will occur.
   D. Only median SNAP will appear.
   E. Only radial SNAP will occur.

7. Specificity of a test for CTS should be:
   A. 100%.
   B. 95-99%.
   C. 90-95%.
   D. 80-90%.
   E. 70-80%.

8. When reference values are set to give similar high specificity, which test is most sensitive?
   A. Mid-palmar studies.
   B. Ring finger comparisons.
   C. Thumb comparisons.
   D. Median motor latency.
   E. Combined sensory index.

9. Which nerve conduction result has highest test-retest reliability?
   A. Mid-palmar studies.
   B. Ring finger comparisons.
   C. Thumb comparisons.
   D. Median motor latency.
   E. Combined sensory index.

10. Which nerve conduction measurement will be **least** affected by cold?
    A. Median sensory latency.
    B. Median sensory amplitude.
    C. Median motor latency.
    D. Median motor conduction velocity.
    E. Median-ulnar sensory latency differences.
11. With median nerve stimulation at the wrist, a 7 mV response is recorded from abductor pollicis brevis (APB). Palm stimulation produces a 10 mV response from APB. This most likely indicates:
   A. Carpal tunnel syndrome.
   B. Martin-Gruber anastomosis.
   C. Neurapraxia.
   D. Axonotmesis.
   E. Nothing.

12. F-wave persistence characteristically can vary considerably in different muscles.
   A. True.
   B. False.

13. Which of the following F-wave parameters can be abnormal in CTS?
   1. Repeater waves.
   2. Persistence.
   3. Chronodispersion.
   A. 1 and 2 are correct.
   B. 1 and 3 are correct.
   C. 2 and 3 are correct.
   D. All are correct.
   E. None are correct.

14. Which of the following are true about F waves?
   1. They are inherently variable in latency, amplitude, and configuration.
   2. Chronodispersion reflects a range of conductions in motor axons.
   3. Latency abnormalities cannot be detected in radiculopathies.
   A. 1 and 2 are correct.
   B. 1 and 3 are correct.
   C. 2 and 3 are correct.
   D. All are correct.
   E. None are correct.

15. Which of the following are true about minimal F-wave latencies?
   1. Accurate values are always obtained after 10 stimuli.
   2. They are the most reliable measure of F-wave latencies.
   3. They are not prolonged with focal nerve injury.
   A. 1 and 2 are correct.
   B. 1 and 3 are correct.
   C. 2 and 3 are correct.
   D. All are correct.
   E. None are correct.

16. F waves have been reported abnormal with focal injury to peripheral nerves, plexi, and roots.
   A. True.
   B. False.

17. According to the proponents of disputed (symptomatic) neurologic thoracic outlet syndrome (TOS), electrodiagnostic studies:
   A. Are of no benefit in assessment.
   B. Can help in diagnosis by identifying other disorders.
   C. Permit objective confirmation.
   D. All of the above.
   E. None of the above.

18. In practical terms, automobile accidents could be responsible for only two types of TOS:
   A. Traumatic and disputed neurologic TOS.
   B. True neurologic and disputed neurologic TOS.
   C. Arterial vascular and traumatic TOS.
   D. Venous vascular and true neurologic TOS.
   E. Disputed neurologic and venous vascular TOS.

19. The most common symptoms attributed to disputed (symptomatic) neurologic TOS are:
   A. Paresthesias and weakness.
   B. Weakness and pain.
   C. Limb coolness and discoloration.
   D. Paresthesias and pain.
   E. Weakness and paresthesias.

20. Very low incidence, typical clinical presentation, and objective laboratory findings are some of the features common to all types of TOS EXCEPT:
   A. True neurologic TOS.
   B. Disputed neurologic TOS.
   C. Traumatic TOS.
   D. Venous vascular TOS.
   E. Arterial vascular TOS.

21. Congenital anomalies of various types characteristically are present with:
   A. True neurological and arterial vascular TOS.
   B. Arterial vascular and venous vascular TOS.
   C. Disputed neurologic and arterial vascular TOS.
   D. Traumatic and true neurologic TOS.
   E. Disputed neurologic and traumatic TOS.
22. The diagnosis of non-specific (symptomatic) thoracic outlet syndrome (NS-TOS) is best reserved for those with:
   A. Unilateral intrinsic hand muscle atrophy.
   B. Position-dependent arm pain and paresthesias.
   C. Radial pulse obliteration produced by arm hyperabduction.
   D. Sensory loss along the ulnar forearm and hand.
   E. Reduced amplitude of the ulnar compound muscle action potential.

23. The “droopy shoulder syndrome” is characterized by:
   A. A long, thin neck and sloping shoulders.
   B. Unilateral atrophy of the trapezius muscle.
   C. Hypotrophy of the clavicles bilaterally.
   D. Bilateral winging of the scapulae.
   E. Unilateral laxity of the acromiohumeral joint.

24. Electrodiagnostic testing in NS-TOS:
   A. Serves no useful role.
   B. Often demonstrates slowing of ulnar motor conduction velocity.
   C. Will usually demonstrate prolonged ulnar F-wave latency.
   D. Can help to exclude alternative diagnoses.
   E. Usually reveals reduced ulnar sensory response amplitude.

25. Successful treatment of NS-TOS:
   A. Can hardly ever be accomplished.
   B. Can usually be achieved with postural correction and therapeutic exercise.
   C. Usually requires psychotherapy.
   D. Usually requires first rib resection.
   E. Usually requires scalenotomy plus pectoralis minor tenotomy.

26. NS-TOS among violinists and violists most commonly affects:
   A. Middle-aged men.
   B. The right (bowing) arm.
   C. Young women in their third decade.
   D. Beginning students.
   E. Those who are left-handed.
### EVALUATION

Select ANY of the answers that indicate your opinions.

Your input is needed to critique our courses and to ensure that we use the best faculty instructors and provide the best course options in future years. Please use the computer form to answer the following questions. For the purpose of tabulating evaluations, please enter the last 4 digits of your telephone number in the ID NUMBER box beginning with the left column and fill in the appropriate ovals below each number. Make additional comments or list suggested topics or faculty for future courses on the comment form provided at the end.

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
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| 27. How would you rate the quality of instruction received during Dr. Johnson's presentation? | A. Best possible.  
B. Good.  
C. Average.  
D. Fair.  
E. Worst possible. |
| 28. Select any item(s), that, if changed, would have appreciably improved Dr. Johnson's presentation: | A. Quality of slides.  
B. Quality of handout.  
C. Amount of clinically relevant information in the presentation.  
D. Amount of scientific content in the presentation.  
E. Other: please explain on the comment form at the back of this handout. |
| 29. How would you rate the quality of instruction received during Dr. Robinson's presentation? | A. Best possible.  
B. Good.  
C. Average.  
D. Fair.  
E. Worst possible. |
| 30. Select any item(s), that, if changed, would have appreciably improved Dr. Robinson's presentation: | A. Quality of slides.  
B. Quality of handout.  
C. Amount of clinically relevant information in the presentation.  
D. Amount of scientific content in the presentation.  
E. Other: please explain on the comment form at the back of this handout. |
| 31. How would you rate the quality of instruction received during Dr. Kimura's presentation? | A. Best possible.  
B. Good.  
C. Average.  
D. Fair.  
E. Worst possible. |
| 32. Select any item(s), that, if changed, would have appreciably improved Dr. Kimura's presentation: | A. Quality of slides.  
B. Quality of handout.  
C. Amount of clinically relevant information in the presentation.  
D. Amount of scientific content in the presentation.  
E. Other: please explain on the comment form at the back of this handout. |
| 33. How would you rate the quality of instruction received during Dr. Fisher's presentation? | A. Best possible.  
B. Good.  
C. Average.  
D. Fair.  
E. Worst possible. |
| 34. Select any item(s), that, if changed, would have appreciably improved Dr. Fisher's presentation: | A. Quality of slides.  
B. Quality of handout.  
C. Amount of clinically relevant information in the presentation.  
D. Amount of scientific content in the presentation.  
E. Other: please explain on the comment form at the back of this handout. |
35. How would you rate the quality of instruction received during Dr. Wilbourn’s presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Fair.
   E. Worst possible.

36. Select any item(s), that, if changed, would have appreciably improved Dr. Wilbourn’s presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment form at the back of this handout.

37. How would you rate the quality of instruction received during Dr. Lederman’s presentation?
   A. Best possible.
   B. Good.
   C. Average.
   D. Fair.
   E. Worst possible.

38. Select any item(s), that, if changed, would have appreciably improved Dr. Lederman’s presentation:
   A. Quality of slides.
   B. Quality of handout.
   C. Amount of clinically relevant information in the presentation.
   D. Amount of scientific content in the presentation.
   E. Other: please explain on the comment form at the back of this handout.

39. As a result of your attendance at this course, did you learn anything that will improve the care of your patients?
   A. Yes, substantially.
   B. Yes, somewhat.
   C. Not sure.
   D. Probably not.
   E. This course was not applicable to my patients.

40. Select ALL items where improvement was needed.
   A. The accuracy of advance descriptions of this course.
   B. The specific topics selected for presentation.
   C. The number of speakers in this course.
   D. The amount of time allotted for discussion in this course.
   E. Other: please add other areas and outline specific recommendations for areas needing improvement on the comment form at the back of this handout.

41. Should this topic be presented in the future by a different method of presentation.
   A. No, the topic of presentation should remain as a course.
   B. Yes, the topic should be presented as a dinner seminar.
   C. Yes, the topic should be incorporated into the plenary session.
   D. Yes, the topic should be discussed during a breakfast session.
   E. Yes, the topic should be organized as a special interest group.
FUTURE MEETING RECOMMENDATIONS

Select ANY of the answers that indicate your opinions.

The following questions are included with all dinner seminar, course, and plenary session evaluations. It is only necessary to answer these questions once during the course of the entire meeting.

42. Please indicate below your specialty:
   A. Neurologist.
   B. Physiatrist.
   C. PhD.
   D. Other.

43. How often do you attend AAEM meetings?
   A. Annually.
   B. Every 2-3 years.
   C. Every 4 or more years.
   D. This is the first AAEM meeting I have attended.

44. With regard to this year’s meeting, which of the small group sessions did you attend? (mark all that apply)
   A. Experts’ roundtables.
   B. Workshops.
   C. Dinner seminars.
   D. None of the above.

45. If you answered none of the above to the previous question, please answer the following. The reason I did not attend the small group sessions was due to:
   A. The timing of the event.
   B. The cost of the event.
   C. My lack of interest in the topics offered.
   D. The session was full.

46. Did this meeting provide information that will enhance care of your patients?
   A. Extremely.
   B. Somewhat.
   C. Very little.
   D. Not at all.

47. With regard to the social event:
   A. I am signed up to attend the social event.
   B. I did not sign up because of the cost of the event.
   C. I did not sign up because of the day the event was offered.
   D. I did not sign up because I am not interested in attending this type of function.

48. How would you rate this meeting?
   A. Poor.
   B. Fair.
   C. Good.
   D. Very good.
   E. Excellent.

49. Did this meeting meet your expectations?
   A. Not at all.
   B. Somewhat.
   C. As expected.
   D. Exceeded expectations.
   E. Best ever.

50. Was the printed program clear and easy to follow?
   A. Yes.
   B. No.

51. With regard to the meeting hotels:
   A. I stayed at one of the meeting hotels.
   B. I did not stay at one of the meeting hotels.

52. If you answered B to the question above, please explain why you did not stay at one of the meeting hotels (please make your comments under Comments, page 43).

53. How did you first learn about the meeting? (choose the method where you first learned about the meeting)
   A. Preliminary brochure mailing.
   B. Registration brochure mailing.
   C. The internet.
   D. Email message.
   E. From a friend.

54. Did you perceive any commercial bias in any of the educational sessions offered by the AAEM at this meeting?
   A. Yes.
   B. No.
55. Did you attend any of the Industry Forums provided this year?
   A. Yes.
   B. No.

56. If you answered yes to question 55 and you attended the Pfizer Industry Forum, how would you rate the quality of the session?
   A. Best possible.
   B. Good.
   C. Average.
   D. Fair.
   E. Worst possible.
   On page 43 under comments, please provide any other comments you have about your attendance at the Pfizer Industry Forum.

57. If you answered yes to question 55 and you attended the Allergan Industry Forum, how would you rate the quality of the session?
   A. Best possible.
   B. Good.
   C. Average.
   D. Fair.
   E. Worst possible.
   On page 43 under comments, please provide any other comments you have about your attendance at the Allergan Industry Forum.

58. How do you prefer to learn new information?
   A. Lecture only.
   B. Lecture in conjunction with questions and answers.
   C. Small group hands-on.
   D. Small group discussion.

59. I plan to attend the 2005 AAEM meeting in Monterey, California, September 21-24.
   A. Yes, definitely.
   B. No, definitely.
   C. Will wait to see the program content.
   D. Will wait to see if budget allows my attendance.

60. I would be more likely to attend the 2005 AAEM meeting if (please make your comments under Comments, page 43):
COMMENTS

Given time and budget constraints, is there something we could do in terms of altering the format of the meeting that would significantly increase the likelihood of your attendance at future AAEM meetings? Explain:

Write out any additional comments about specific courses or the plenary session (please indicate which), and list suggestions for topics and speakers for future meetings. Leave at the AAEM Registration and Information Center or mail to the AAEM Executive Office at 421 First Avenue SW, Suite 300 East, Rochester, MN 55902.