ISSUES AND CONTROVERSIES
IN THE INJURED WORKER

Laurence J. Kinsella, MD
Asa J. Wilbourn, MD

2005 AANEM COURSE C
AANEM 52ND Annual Scientific Meeting
Monterey, California
Issues and Controversies in the Injured Worker

Laurence J. Kinsella, MD
Asa J. Wilbourn, MD

2005 COURSE C
AANEM 52nd Annual Scientific Meeting
Monterey, California

Copyright © September 2005
American Association of Neuromuscular & Electrodiagnostic Medicine
421 First Avenue SW, Suite 300 East
Rochester, MN 55902

PRINTED BY JOHNSON PRINTING COMPANY, INC.
Issues and Controversies in the Injured Worker

Faculty

Nortin M. Hadler, MD*
Professor
Department of Medicine and Microbiology/Immunology
University of North Carolina
Durham, North Carolina

Dr. Hadler received his medical degree from the Harvard Medical School, and was trained at the Massachusetts General Hospital, the National Institutes of Health, and the Clinical Research Centre in London before joining the faculty of the University of North Carolina where he is Professor of Medicine and Microbiology/Immunology. He serves as Attending Rheumatologist at the University of North Carolina Hospitals. Dr. Hadler has authored over 200 papers and 12 books related to his special interest in the illness of work incapacity. He has lectured widely, garnered multiple awards, and served as a visiting professor in England, France, Israel, and Japan. He has been elected to membership in the American Society for Clinical Investigation and the National Academy of Social Insurance. The third edition of his monograph Occupational Musculoskeletal Disorders was published in 2005.

*Manuscript not included in the handout.

Laurence J. Kinsella, MD
Professor of Neurology
Department of Neurology
Saint Louis University
Saint Louis, Missouri

Dr. Kinsella earned his medical degree from St. Louis University, completed a neurology residency at Brown University/Rhode Island Hospital, and a neuromuscular fellowship at the Neurological Institute, Columbia-Presbyterian Hospital in New York. He currently is Chief in the Division of Neurology and Neurophysiology at Tenet-Forest Park Hospital in St. Louis, and is an associate professor of neurology at the St. Louis University School of Medicine. Dr. Kinsella's interests involve the clinical and electrophysiologic evaluation of patients with suspected peripheral neuropathies, especially those due to B12 deficiency and systemic medical illness.

Asa J. Wilbourn, MD
Director, EMG Laboratory
The Cleveland Clinic
Associate Professor
Department of Neurology
Case Western Reserve University
Cleveland, Ohio

Dr. Wilbourn received his neurology training at Yale University and his electrophysiology training at Mayo Clinic, Rochester, Minnesota. He is the director of the EMG Laboratory at the Cleveland Clinic, and is Associate Professor of Neurology at the Case Western Reserve University School of Medicine in Cleveland. Dr. Wilbourn has extensively published on all aspects of the electrodiagnostic evaluation of plexopathies, radiculopathies, entrapment neuropathies, and iatrogenic nerve injuries. He has served as a member of the AANEM Education and Training Program Committees, and has been the chair of both the Membership and Program Committees. Dr. Wilbourn has also served on the AANEM Board of Directors.

Course Chair: Alan R. Berger, MD

The ideas and opinions expressed in this publication are solely those of the specific authors and do not necessarily represent those of the AANEM.
Issues and Controversies in the Injured Worker

Contents

Faculty ii
Objectives iii
Course Committee vi

Neurotoxicology in the Workplace: How to Evaluate Patients with Suspected Neurotoxic Peripheral Nerve Disease 1
Laurence J. Kinsella, MD

The Role of EDX in Evaluating and Treating the Injured Worker 9
Asa J. Wilbourn, MD

CME Self-Assessment Test 17

Evaluation 19

Member Benefit Recommendations 21

Future Meeting Recommendations 23

Please be aware that some of the medical devices or pharmaceuticals discussed in this handout may not be cleared by the FDA or cleared by the FDA for the specific use described by the authors and are "off-label" (i.e., a use not described on the product's label). "Off-label" devices or pharmaceuticals may be used if, in the judgement of the treating physician, such use is medically indicated to treat a patient's condition. Information regarding the FDA clearance status of a particular device or pharmaceutical may be obtained by reading the product's package labeling, by contacting a sales representative or legal counsel of the manufacturer of the device or pharmaceutical, or by contacting the FDA at 1-800-638-2041.

OBJECTIVES Clinicians who specialize in neuromuscular disease are often asked to evaluate workers reportedly injured by their work activities. After attending this course, participants will: (1) understand the current workers' compensation and disability system and the role it plays in the pathogenesis of workers' symptoms, (2) appreciate the controversy surrounding the cumulative trauma theory to gain perspective when evaluating symptomatic workers, (3) understand the difference between low back pain and the injured back, and how confounding factors influence the pathogenesis, (4) understand the principles of neurotoxicology and develop a framework to evaluate whether a patient is suffering from neurotoxic peripheral nerve disease, and (5) understand the role and limitations of the electrodiagnostic evaluation of the symptomatic worker.

PREREQUISITE This course is designed as an educational opportunity for residents, fellows, and practicing clinical EDX physicians 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 AANEM is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education (CME) for physicians.

CME CREDIT The AANEM designates attendance at this course for a maximum of 3.25 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. CME for this course is available 9/05 - 9/08.
2004-2005 AANEM COURSE COMMITTEE

Kathleen D. Kennelly, MD, PhD
Jacksonville, Florida

Thomas Hyatt Brannagan, III, MD
New York, New York

Dale J. Lange, MD
New York, New York

Jeremy M. Shefner, MD, PhD
Syracuse, New York

Timothy J. Doherty, MD, PhD, FRCPC
London, Ontario, Canada

Subhadra Nori, MD
Bronx, New York

T. Darrell Thomas, MD
Knoxville, Tennessee

Kimberly S. Kenton, MD
Maywood, Illinois

Bryan Tsao, MD
Shaker Heights, Ohio

2004-2005 AANEM PRESIDENT

Gary Goldberg, MD
Pittsburgh, Pennsylvania
IDENTIFICATION OF TOXIC PERIPHERAL NEUROPATHIES

Despite widespread media attention to their rare epidemic occurrence, toxic polyneuropathies (TxPN) are relatively infrequent in North America. Most TxPN encountered in routine clinical practice are due to iatrogenic pharmaceutical intoxications; epidemic occupational exposure, as with large pharmaceutical companies, is unusual. The majority, and unfortunately the most difficult cases, of TxPN are individual intoxications due to small scale, often chance occupational exposures, or intentional and homicidal ingestion.

The identification that a sporadic peripheral neuropathy results from toxin exposure in the occupational setting is often made difficult by an unclear exposure history. Toxic polyneuropathies are usually distal axonopathies and clinically and electrophysiologically resemble neuropathies from metabolic abnormalities, nutritional deficiencies, or systemic illness. Clinically relevant and reliable toxicologic tests are often unavailable or unhelpful, either because the necessary laboratory tests are not available, or the substance is undetectable because of the delay between exposure and examination. Consequently, when a naturally occurring medical cause is not readily apparent, there is an unfortunate tendency for many peripheral neuropathies to be misdiagnosed as toxic in nature. As a result, cases of naturally occurring polyneuropathy are more often misdiagnosed as being toxic in nature rather than the reverse.

The underlying pathology of many TxPN is the central-peripheral axonopathy. With continued exposure and worsening of the neuropathy, similar changes occur in the distal segments of the dorsal column, and the corticospinal and spinocerebellar tract axons. Initial clinical symptoms reflect dysfunction in peripheral axons. As the peripheral nerves recover, however, signs of central nervous system (CNS) impairment such as spasticity, mild ataxia, and persistent sensory loss may be evident. These latter deficits result from the lack of regeneration in central sensory and motor tracts.

Medicine’s limited knowledge of the biochemical and pathophysiologic mechanisms of most neurotoxins has led to a simplistic classification system according to compound class (e.g., solvents, metals). Such a classification is clinically unhelpful and potentially misleading. A compound cannot be presumed to be neurotoxic because of a superficial resemblance to a related known toxin of similar class; all compounds within the same class are not neurotoxic (e.g., acrylamide monomer is capable of producing a devastating peripheral neuropathy, while the polymer is innocuous).
Structure-toxicity relationships are clear for only a few classes of substances, such as organophosphates and hydrocarbons.

**CARDINAL TENETS OF NEUROTOXIC ILLNESS AFFECTING THE PERIPHERAL NERVOUS SYSTEM**

The identification of a neurotoxic illness should satisfy, or at least not be inconsistent with, the following basic principals of neurotoxic disease. The key to correctly recognizing the presence of a TxPN is not in remembering the characteristics of the many potential neurotoxins, but in understanding and applying these basic tenets.

**Strong Dose-Response Relationship**

Most neurotoxins produce a consistent pattern of disease, commensurate with the dose and duration of exposure. Neurotoxins rarely cause focal or asymmetric deficits. Since most neurotoxins cause diffuse myelin and/or neuronal dysfunction, their related symptoms and signs are usually widespread and symmetric. In the case of TxPN, this usually means a relatively symmetric distal axonopathy with initial symptoms in the feet and proximal progression, with continued exposure. Only rarely does an occasional toxin cause strikingly asymmetric or focal dysfunction (e.g., trichlorethylene and cranial neuropathies).

Some patients with pre-existing neuropathy may experience far greater toxicity than expected for the dose received, as seen in patients with unsuspected Charcot-Marie-Tooth disease who are exposed to chemotherapeutic agents.

**Consistency of Response**

All individuals with similar exposure to the same neurotoxin will invariably manifest similar signs and symptoms (if the chemical enters the circulation, and if the agent, its metabolite or intermediate, has similar access to the nervous system). Although the same toxin may produce strikingly different clinical syndromes if the exposure dose or duration is different, a similar and consistent illness should result in patients with similar exposures. There is usually no individual susceptibility or idiosyncratic reactions if dose and duration of exposure are similar. A neuropathy is unlikely to be neurotoxic if it occurs in only one member of a group with similar exposure history. Likewise, neurotoxicity should also be doubted when substantially different clinical manifestations occur in a group of individuals with identical chemical exposure.

**Proximity of Symptoms to Exposure**

Neurotoxic illness usually occurs concurrent with exposure or following a short latency. Neurologic symptoms do not begin months to years after exposure. The two most common exceptions are the 2-6 week delay following exposure to organophosphates and the occasional 2 month latency between cisplatin intoxication and neuropathic symptoms.

In addition, the extent and severity of neuropathy is usually commensurate with the degree of toxin exposure. It is unlikely that a single, brief, low-level exposure will result in a devastating peripheral neuropathy. Some lipid stored agents (e.g., chlorinated hydrocarbons) are detectable in fat biopsies years following exposure. Although this provides a valuable marker of previous exposure, there is no evidence that this state is associated with risk for future neurotoxicity, and attempts at removal or mobilizing the body burden are unnecessary.

**Improvement Usually Follows Cessation of Exposure**

Toxic polyneuropathies generally plateau and then gradually improve after removal of the neurotoxic agent. Some degree of recovery is the rule, except in the most severely affected cases. A neuropathy that shows no improvement or one that continues to deteriorate despite the cessation of exposure to a suspected neurotoxin is unlikely to be neurotoxic in nature. However, the clinical picture may become somewhat murky in certain toxic axonopathies in which cessation of exposure may be followed by worsening of symptoms (coasting) for several weeks before recovery commences.

**CONFUSING ASPECTS OF NEUROTOXIC ILLNESS**

**Multiple Clinical Syndromes May Result From Different Levels of Exposure to a Single Toxin**

Different exposure levels to the same substance may produce dramatically different syndromes. Most confusing is the bizarre constellation of symptoms that may arise from intoxication with intermediate levels of the neurotoxin. Examples include the different clinical syndromes produced by acute high-level and intermediate-level exposure, and prolonged low-level acrylamide intoxication. Exposures to high-level acrylamide causes early CNS dysfunction with drowsiness, disorientation, hallucinations, seizures, and severe truncal ataxia, followed by neuropathy of variable severity. Prolonged, lower-level exposure causes little CNS dysfunction but a marked peripheral neuropathy. Exposure to inter-
mediate levels of acrylamide causes hallucinations, mental confusion, and cognitive dysfunction, followed by sensory complaints affecting the distal limbs.

Another example is organophosphate poisoning in which there may be early, severe cholinergic symptoms resulting from excessive muscarinic receptor stimulation. Within 1-3 days there may occur generalized paralysis with respiratory distress owing to nicotinic receptor blockade. After a few weeks, a distal axonopathy may be evident.

In some instances, a single compound may produce similar clinical symptoms at both high- and low-level exposure, although different anatomic structures are affected. High-dose pyridoxine intoxication produces widespread sensory loss due to dorsal root ganglion dysfunction; low-level exposure produces similar symptoms but is due to a distal axonopathy.

### Asymptomatic Disease

Prolonged, low-level exposure may occasionally produce widespread subclinical dysfunction. Clinical deficits may go unnoticed by the patient unless they perform an usually skilled job that requires fine-motor control or intact sensibility. Insidiously developing subclinical TxPN may occur in individuals who deny any disability.

### Enhancement by Bystander Chemicals

An agent without known neurotoxic activity may enhance the toxicity of a known neurotoxin that is present at a “no effect” level. This disquieting notion has raised the public’s fear that the combined effects of multiple chemicals in hazardous waste disposal sites may be more toxic than their separate effects. Such sites may contain low, presumably harmless levels of neurotoxic solvents, metals, or pesticides, whose neurotoxic potency conceivably might be enhanced by one of the other chemicals present. Neurotoxic potentiation is illustrated by the epidemic of peripheral neuropathy which occurred in German youths who abused paint thinner containing n-hexane. Initially there were no instances of neuropathy, but when the paint thinner was reformulated by lowering the concentration of n-hexane and adding methyl ethyl ketone (MEK), there resulted an epidemic of severe distal axonopathy. Experimental evidence subsequently showed that while MEK by itself was not neurotoxic, the compound dramatically potentiated the neurotoxic effects of n-hexane.

### Chemical Formula May Not Predict Toxicity

The neurotoxic potential of a compound cannot usually be predicted by its chemical formula. This is especially important to consider when evaluating cases of potential occupational exposure to chemicals that superficially resemble a known neurotoxin. An example is workers exposed to the innocuous acrylamide polymer who have been needlessly alarmed by physicians familiar only with the effects of acrylamide monomer, a potent neurotoxin. Unpredictability exists because the underlying biochemical mechanisms and active metabolites of most neurotoxins are unknown.

### IDENTIFICATION OF TOXIC PERIPHERAL NEUROPATHY

The presence of a TxPN is suggested by the following:

a. suspicion raised by history and reinforced by compatible findings on physical examination;

b. lack of naturally occurring alternative explanation;

c. consistency with basic principals of neurotoxic disease;

d. compatible electrodiagnosis;

e. confirmation by demonstration of elevated body burdens if appropriate, or resolution of condition with removal of potential exposure.

The initial step is a suspicion raised by a thorough occupational history. Unfortunately, because most TxPN are insidious in onset, many patients are unable to discern a relationship between their symptoms and chronic, low-level, toxin intoxication. Inquiry should focus on potential occupational, environmental, and iatrogenic exposures. The individual habits of a patient should be discussed. Does the patient wear protective devices? Do they change their clothes before coming home? Do they eat in the workplace and wash their hands prior to eating? What is the health of their peers? Do symptoms improve when they are away from the potential toxin, such as on weekends or holidays? Do workplace conditions (ventilation, drainage) predispose to an unacceptably high risk of toxin exposure? Answers may only be available after a visit to the home or workplace itself. In cases of suspected domestic poisoning or substance abuse, home visits may be needed to check hobby workshops, medicine cabinets, and food and water sources. Inquiry should be made about recent pesticide applications. A similar illness found in a neighbor may prove helpful in identifying a neurotoxin.

The nature of the suspected toxin should focus the physical examination to relevant deficits. Thus a suspicion of mercury poisoning should prompt a careful examination for tremor and mild cerebellar dysfunction. The function of the neurologic examination is to demonstrate that neurologic deficits are in a pattern and of a severity that is consistent with
neurotoxic illness. Since the clinical deficits resulting from a TxPN should be symmetric in distribution, the presence of multifocal deficits should suggest a diagnosis other than neurotoxic disease. In addition, since most TxPN affect mixed nerve function, finding a purely small fiber neuropathy makes neurotoxic disease unlikely.

Occasionally the sensory or motor predominance of the neuropathic deficit gives some clue to etiology (Table 1).

Because of the heterogeneity of toxins and their often-unknown mechanisms of action, Schaumburg suggests a clinical classification based on cardinal manifestations of disease. Those agents with an A rating have a strong association with a particular clinical presentation, and a B rating is less strong (Tables 2-5).

### Table 1 Clues to etiology

<table>
<thead>
<tr>
<th>Motor Greater than Sensory Toxic Neuropathies</th>
<th>Sensory Greater Than Motor Toxic Neuropathies</th>
</tr>
</thead>
<tbody>
<tr>
<td>dapsone</td>
<td>cisplatin</td>
</tr>
<tr>
<td>disulfiram</td>
<td>pyridoxine</td>
</tr>
<tr>
<td>nitrofurantoin</td>
<td>thalidomide</td>
</tr>
<tr>
<td>organophosphates</td>
<td>thallium</td>
</tr>
<tr>
<td>lead</td>
<td>arsenic</td>
</tr>
<tr>
<td>vincristine</td>
<td>polychlorinated biphenyls</td>
</tr>
</tbody>
</table>

(reprinted with permission, Schaumburg)

### Table 2 Agents associated with peripheral neuropathy (A rating)

<table>
<thead>
<tr>
<th>Acrylamide</th>
<th>Ethylene oxide</th>
<th>Organophosphates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allyl chloride</td>
<td>n-Hexane</td>
<td>Phenol</td>
</tr>
<tr>
<td>Apamin &amp; Hymenoptera</td>
<td>Hydralazine</td>
<td>Pyriminil</td>
</tr>
<tr>
<td>spp. venoms</td>
<td>Hydrazine</td>
<td>Thallium</td>
</tr>
<tr>
<td>Arsenic</td>
<td>Karwinskia</td>
<td>Vinyl chloride</td>
</tr>
<tr>
<td></td>
<td>humboldtiana</td>
<td></td>
</tr>
<tr>
<td>2-t-Butylalo-2-hydroxy-</td>
<td>Lead</td>
<td>Manihot esculenta</td>
</tr>
<tr>
<td>5-methylhexane</td>
<td>Arsenic</td>
<td>Methyl bromide</td>
</tr>
</tbody>
</table>

### Table 3 Agents associated with peripheral neuropathy (B rating)

<table>
<thead>
<tr>
<th>Carbamate pesticides</th>
<th>Germanium dioxide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chielanthes sieberi</td>
<td>Mercury, inorganic</td>
</tr>
<tr>
<td>Ciguatoxin</td>
<td>Methyl methacrylate</td>
</tr>
<tr>
<td>Dichloroacetic acid</td>
<td>Tetrachloroethane</td>
</tr>
<tr>
<td>Euphoribia spp.</td>
<td>“Spanish toxic oil”</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td></td>
</tr>
</tbody>
</table>

### Table 4 Agents associated with neuromuscular transmission syndromes (A rating)

<table>
<thead>
<tr>
<th>Black widow spider venom</th>
<th>Fasciculins</th>
<th>Pelamitoxin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Botulinum neurotoxin</td>
<td>Hydrophiidae toxins</td>
<td>Taipoxin</td>
</tr>
<tr>
<td>Carbamate pesticides</td>
<td>Lophotoxin</td>
<td>Trimethaphan</td>
</tr>
<tr>
<td>Ceruleotoxin</td>
<td>Mamba snake toxin</td>
<td>Erabutoxin</td>
</tr>
<tr>
<td>Cobra venom</td>
<td>Mandaratoxin</td>
<td>Organophosphorous</td>
</tr>
<tr>
<td>Crotoxin</td>
<td>Methyllycaconitine compounds</td>
<td></td>
</tr>
<tr>
<td>Delphinium spp.</td>
<td>Mojavetoxin</td>
<td>Dendroaspis angusticeps</td>
</tr>
<tr>
<td>Ixodes holocyclus</td>
<td>Dermocenter spp. Toxins</td>
<td></td>
</tr>
</tbody>
</table>

### Table 5 Agents associated with axon ion channel syndromes (A rating)

<table>
<thead>
<tr>
<th>Brevetoxins</th>
<th>Kalmia latifolia</th>
<th>Scorpion toxins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buhotus hottentota</td>
<td>Maitotoxin</td>
<td>Tetrodotoxin</td>
</tr>
<tr>
<td>Ciguatoxin</td>
<td>Pyrethroids</td>
<td>Tityustoxin</td>
</tr>
<tr>
<td>Dichlorophenylethanes</td>
<td>Saxitoxin</td>
<td>Veratridine</td>
</tr>
<tr>
<td>Germinene</td>
<td>Ptychodiscus brevis</td>
<td></td>
</tr>
</tbody>
</table>
DETERMINATION OF BODY BURDENS

In the usual scenario, screening levels for heavy metals are not fruitful. Often this is because the toxin exposure was too remote allowing time for the offending agent to be cleared from the serum. In some cases of prolonged exposures, like thallium or lead, finding elevated levels either in slow growing tissues such as hair, or in urine or serum after chelation is often diagnostic. In cases of prolonged exposure, the neurotoxin may be sequestered in various tissues and therefore unavailable for laboratory identification. With some chemicals, a safe level has not been established.

Serum lead levels greater than 30 mg/dL in adults and 10 mcg/dL in children are toxic, but symptoms are most likely with values greater than 80 mcg/dL. Free erythrocyte protoporphyrin is a useful screening test in children. The assessment of body burden of lead requires chelation with ethylene diamine tetra acetate followed by a 24-hour urine collection. Values greater than 600 mg/24 hours are considered toxic.

Caution must be taken when interpreting body burden results. An example is arsenic levels. These are frequently of little clinical value and may be raised by recent shellfood ingestion. Total arsenic levels may include both inorganic (toxic) and organic (nontoxic form from shellfish ingestion) components. Arsenic levels greater than 50 mcg/L are abnormal, but patients with signs of toxicity usually have levels greater than 200 mcg/L. Twenty-four-hour urine collection is helpful in those with elevated serum levels. Patients should abstain from shellfish for 3 days before measuring 24-hour urine or serum levels. The laboratory should separate results into organic and inorganic forms to eliminate false positives.

ELECTRODIAGNOSTIC ASSESSMENT

Electrophysiologic findings should be consistent with a distal axonopathy or mixed axonal, demyelinating neuropathy. Only a few rare neuropathies, such as n-hexane, perhexiline, amiodarone, and early arsenic poisoning, have predominant slowing of conduction velocities.

QUANTITATIVE SENSORY TESTING

Quantitative thresholds for thermal and vibration appreciation have proven extremely useful in documenting objective evidence of sensory impairment and monitoring the course of recovery or deterioration. These procedures are noninvasive and reproducible and can be performed by a trained technician. In an outbreak of arsenic poisoning, Kishi and colleagues documented recovery of vibration perception thresholds in survivors, whereas sural nerve responses showed no improvement. Berger demonstrated early abnormalities of thermal and vibratory thresholds in subjects with normal clinical examinations who had been exposed to excessive pyridoxine (1-3 g/day).

SYSTEMIC FEATURES SUGGESTIVE OF NEUROTOXIC DISEASE

The neuropathies resulting from most neurotoxins are remarkably similar in both their clinical and electrophysiologic characteristics (Table 6). Occasionally, there may be systemic complaints or signs that suggest the nature of the neurotoxic insult. Usually these symptoms/signs are apparent with either acute high-level, or chronic low-level intoxication. The clinical characteristics listed in the Table 6 may be the identifying feature that suggests a TxPN.

<table>
<thead>
<tr>
<th>Toxin</th>
<th>Systemic Signs and Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acrylamide</td>
<td>dermal contact associated with contact dermatitis, excessive sweating of hands and feet</td>
</tr>
<tr>
<td>Carbon disulfide</td>
<td>chronic low-level exposure associated with a variety of behavioral and psychiatric abnormalities along with peripheral neuropathy</td>
</tr>
<tr>
<td>Ethyl oxide</td>
<td>cognitive impairment and neuropathy with prolonged low-level exposure</td>
</tr>
<tr>
<td>Hexacarbons</td>
<td>acute, high-level exposure may mimic AIDP with prominent autonomic dysfunction</td>
</tr>
<tr>
<td>Lead</td>
<td>Mee's lines, blood abnormalities (basophilic stippling, anemia), GI abnormalities, and predominantly a motor neuropathy</td>
</tr>
<tr>
<td>Mercury</td>
<td>erythematous papulosis; tremor and ataxia with a predominantly sensory neuropathy</td>
</tr>
<tr>
<td>Methyl bromide</td>
<td>corticospinal and cerebellar dysfunction along with an axonal neuropathy</td>
</tr>
<tr>
<td>Organophosphate</td>
<td>early cholinergic symptoms, may have intermediate syndrome preceding neuropathy, late emergence of corticospinal tract dysfunction as the peripheral neuropathy resolves</td>
</tr>
<tr>
<td>Polychlorinated</td>
<td>symmetric sensory neuropathy associated with brown aceneiform skin eruption and brown pigmented nails</td>
</tr>
<tr>
<td>Biphenyls</td>
<td>prominent GI distress with high-level exposure, alopecia, Mee’s lines, hyperkeratosis with more prolonged exposure, sensory greater than motor neuropathy</td>
</tr>
</tbody>
</table>

AIDP = Acute idiopathic demyelinating polyneuropathy; GI = gastrointestinal.
Industrial workers and others may be exposed to a number of known neurotoxins. Table 3 lists the toxins, the occupations and clinical signs, aids in diagnosis and treatment, and prognosis. What little may be known about the pathophysiology is included.

Chinese herbal balls have been found to have potentially toxic levels of arsenic and mercury. They are a mixture of medicinal herbs and honey and are dissolved and drunk as a tea. Mercury content in the 32 balls tested varied between 7.8 and 621.3 mg per ball; arsenic content varied between 0.1 and 36.6 mg. Chronic poisoning has been reported in people ingesting as little as 10 mg per day of arsenic sulfide and among people ingesting approximately 260 mg per day of mercury sulfide.

SUMMARY

In summary, toxic neuropathies may occur from a variety of substances in the workplace and in the home, but remain rare. Often the physician is faced with the difficult task of ruling out toxic etiologies in patients who suspect they have been poisoned. A careful exposure history, and an examination consistent with known clinical patterns of injury are most important in reaching a diagnosis.

BIBLIOGRAPHY

2. Albers JW. Chronic low-level exposures and body burden: reason for concern or smoke screen? Presented at: Meeting of the American Academy of Neurology; April 9-16, 2005; Miami Beach, Fl.


The Role of the Electrodiagnostic Examination in the Evaluation and Treatment of the Injured Worker

Asa J. Wilbourn, MD
Director, EMG Laboratory
Cleveland Clinic
Cleveland, Ohio

INTRODUCTION

A long history is shared between the electrodiagnostic (EDX) examination and employees who have possibly sustained work-related peripheral nervous system (PNS) injuries. Electrodiagnosis generally is considered to have expanded from being solely a research tool to having clinical utility as well with the 1943 publication of Weddell, Feinstein, and Pattle's article called “The Clinical Application of Electromyography.”26 In 1950 and 1951, Woods and colleagues described the method currently used for identifying radiculopathies by the EDX examination.22,34 The fledgling diagnostic procedure took a large stride forward when motor nerve conduction studies (NCSs) were added to it in the mid-1950s, after techniques for assessing some motor fibers of all the major peripheral nerves were described by Hodes and colleagues in 1948.11 In 1956, Simpson reported using motor NCSs to detect entrapment neuropathies of the median nerve at the wrist, i.e., carpal tunnel syndrome (CTS), the ulnar nerve at the elbow, and the ulnar nerve in the hand.23 This was a major advancement, because focal nerve lesions manifesting only demyelination could now be identified with motor NCSs, thereby adding a new dimension to the EDX examination (which, heretofore, using needle EMG alone, had essentially been restricted to detecting axon loss lesions).

One of the first articles to discuss the use of the EDX examination in assessing patients with possible work-related PNS injuries was Marinacci’s “The Electromyogram in Industrial Medicine.”15 Published in 1954, it was surprisingly modern in its content, as judged by current standards. In regard to the EDX examination, it contained the following: criteria used for identifying nerve injuries; a method for distinguishing nerve injuries from psychogenic disorders; a procedure for recognizing a radiculopathy and nine reasons why this was beneficial with possible root lesions; and points regarding its usefulness in distinguishing changes, such as disuse atrophy of muscle or neurogenic findings caused by neuralgic amyotrophy, from abnormalities resulting from actual work-related PNS injuries.15 A notable omission was the lack of comments regarding CTS. However, it must be remembered that Marinacci’s article appeared in 1954, only 1 year after the clinical description of CTS was published by Kremer and colleagues,14 and 2 years before Simpson’s report recounted the use of motor NCSs for diagnosing CTS.23

In all industrialized countries, work-related injuries are responsible for an enormous amount of disability, expense, and lost production. The single most common symptom associated with such injuries is pain (less often, paresthesias), and the majority of these most likely are due to actual or
alleged PNS or musculoskeletal disorders. Carpal tunnel syndrome, as well as back, neck, and limb complaints, are responsible for a considerable number of claims. Noteworthy is that limb pains, particularly when they involve the lower extremity, are much more likely to be due to musculoskeletal causes as opposed to neurological problems (e.g., root compression).19

In any case, many persons with presumed work-related injuries are referred for EDX evaluations. Generally, the latter are obtained only under certain circumstances: (1) if the diagnosis is not clear after a detailed history has been obtained and a comprehensive physical examination has been performed; (2) if the results of the EDX examination would change the management of the patient in some fashion; or (3) if they are needed for medical-legal reasons.7 Many factors are involved in workplace injuries, not only occupational, but also medical, psycho-social, and legal. These cases often differ from similar ones that occur in the nonwork-related situations, in that patients frequently have incentives to fabricate or exaggerate symptoms. Moreover, they often “present with a complicated mixture of symptoms resulting from recent injury and exacerbation of pre-existing pathology.”16

Clinicians who examine patients with work-related injuries are not only responsible for diagnosing and appropriately treating them, but also for determining their cause, establishing a causal relationship between the worker’s complaint and the injury, and determining the degree of worker impairment.19 Moreover, the occupational physician is under considerable pressure to accurately diagnose and treat the injured worker promptly, because returning him/her to work is the primary goal, since there is compelling evidence that a major barrier to persons returning to work in these situations is the primary goal, since there is compelling evidence that a major barrier to persons returning to work in these situations is the amount of time they have already been away from work due to their injuries.325 Unfortunately, the treating physician can encounter a considerable number of impediments while trying to achieve these goals, beyond the problems posed by the PNS or musculoskeletal abnormalities that directly result from the injury itself. These may be financial, psychological, or administrative in nature.

At least two financial disincentives can be encountered. First, the injured worker may be receiving a portion of their salary while not working. The amount received varies, but can be substantial—up to 70% of normal earnings. While the logic for this arrangement is obvious (i.e., to prevent financial hardship for the worker and his family), the practice often acts as a major deterrent to the worker returning to work, particularly if there are associated psychological factors. Second, sometimes the potential exists for the employee to obtain significant monetary gain via litigation. This is especially likely to occur in situations where the worker has sustained a past injury and received a favorable settlement for it. In this situation, the worker has strong motivation for not returning to work because his/her absence strengthens the argument that substantial personal injury has occurred (and, therefore, a higher monetary settlement is in order).8

Psychological factors are often prominent. Many of these are directly work-related, such as job satisfaction related to non-challenging, or repetitive work, being a new employee, and strained relationships between the employee and employer.8 Thus, Bigos and colleagues noted that a recent (preceding 6 months) poor employee job performance evaluation was a major psychologic factor that impeded the return to work of employees with low back injuries.4 The psychological factors also can impact the injured employee’s nonwork environment. Stress and turmoil within the family can develop if injured workers relinquish social responsibilities that they have in their homes, even though they could perform them despite their impairments. Moreover, some workers use their injuries as control factors to manipulate family members. In addition, some of them have underlying psychiatric disorders such as depression, which manifest under the circumstances and impede their return to work.8

There are psychological issues pertaining to diagnostic testing that may have a negative impact on return to work. Excessive diagnostic testing may increase the risk of false-positive studies, thereby creating the impression that patients have more profound illnesses. It may also result in incorrect diagnoses. Thus, obtaining an excessive number of spinal imaging procedures on patients with low back pain may lead to misdiagnosis, since a co-existing but unrelated imaging abnormality may be considered the cause for the patient’s current symptoms. This can create psychological stress for patients and their families and encourage those litigious workers to file lawsuits. In both instances, return to work is delayed.8

There are administrative factors that can thwart the occupational physician’s goal of promptly returning the worker to his employment. These may be both system-wide or related to a specific employer. Many workers are covered by the state’s Worker’s Compensation system. Although these vary considerably from state to state, they all have one thing in common—they are complicated.19 Often delays are encountered before proper authorization can be obtained for procedures to be performed that the physician considers necessary (including EDX examinations). Under the Worker’s Compensation system, physicians usually are authorized to treat only the work-related injury.8 In regard to EDX examinations, that means only the symptomatic limb can be assessed (i.e., no studies can be performed on the contralateral, normal limb, for comparison purposes). In many instances this is a reasonable approach (e.g., a young, otherwise healthy man with major nerve injuries in one upper limb). However, in other instances it is a major obstacle to diagnosis (e.g.,
patients with brachial plexopathies or elderly diabetic patients with unilateral distal lower limb injuries). In some situations, it is impossible to determine the significance of a particular EDX finding obtained on the symptomatic limb without having results obtained on the contralateral normal limb to which to compare it. Administrative factors may also be limited to the work site. The injured worker may be unable to obtain any workplace accommodations, such as modified or light duty, because the employer is unwilling or unable to provide these accommodations.\(^8\)\(^19\)

Obviously injured workers may have a number of reasons for resisting a return to work. Moreover, many of these factors are operative at the time of the initial evaluation and they often seriously compromise the physical examination, particularly in regard to suspected PNS injuries. Thus, the procedures employed during the physical examination to assess the muscle strength integrity of the PNS rely on the patient performing maximal effort. Similarly, when testing sensory functions or determining symptoms produced with provocative maneuvers, the patient’s honesty in reporting is necessary. In situations in which patient efforts or veracity are questioned, EDX examinations often prove helpful, since they can provide objective results compared to those obtained during the physical examination.\(^7\)

Many physicians who order EDX examinations for injured workers do not perform the procedure themselves, and know little about its characteristics, particularly in regard to its limitations. In this author’s experience, they often are doubly unenlightened. First, they are oblivious to the circumstances in which the EDX examination should be obtained, because it will provide information useful in patient management. Second, they frequently display unrealistic expectations of the EDX examination’s capability to provide diagnostic help in situations in which it is extremely unlikely to do so.

**LIMITATIONS OF THE ELECTRODIAGNOSTIC EXAMINATION**

The major limitations of the EDX examination should be explained to all physicians who request such studies on their patients.

Virtually all components of the standard EDX examination assess only large myelinated fibers, specifically, the motor fibers that transmit impulses to the extrafusal muscle fibers and the sensory fibers that convey impulses dealing with position, vibration, and light touch. Pain fibers, being unmyelinated, are not evaluated by any component of the EDX examination. Consequently, if pain is the sole symptom, the EDX examination is not likely to be revealing unless the pain is due to a PNS lesion which has concomitantly damaged large myelinated fibers and evidence of the latter is still detectable on either the NCSs or the needle EMG (or both).\(^33\)

The ability of the EDX examination to detect a specific PNS disorder varies enormously, based on the particular injury in question and its duration. This is important point because many physicians are aware of the high sensitivity the EDX examination has for identifying CTS and they erroneously assume that it is equally sensitive for detecting all other PNS disorders. For example, most patients who have intermittent paresthesias in the median nerve-innervated fingers because of CTS have positive EDX examinations; however, many patients who have similar sensory symptoms in the ulnar nerve-innervated fingers because of an ulnar neuropathy at the elbow have completely normal EDX examinations. Physicians who do not know that the EDX examination’s capability to identify a lesion varies substantially may mistakenly assume that a particular PNS disorder has been excluded by a normal EDX examination when it has not. This can lead to their attributing their patients’ symptoms to some other PNS disorder or even to psychogenic factors.

The EDX examination can detect the presence of a lesion and provide useful information regarding its pathophysiology, severity, and extent, but generally cannot establish its etiology. It is difficult to assess patients with possible work-related injuries in this regard, because the injury’s cause is often questionable. Typically, the EDX findings concerning etiology are noninformative, so the referring physician must rely heavily on the history—often provided only by the patient and which may be self-serving. However, there are a few exceptions. The EDX findings of acute compression of the median nerve in the carpal tunnel are distinct from those typically seen with chronic compression of the same nerve at the same location (i.e., CTS). The former findings are often incorrectly referred to as “acute CTS,” which is a contradiction in terms because CTS by definition is due to chronic nerve entrapment. Acute CTS results from abrupt injury of the median nerve within the carpal tunnel and its pathophysiology is either demyelinating conduction block or conduction failure caused by axon loss. In contrast, CTS is caused by chronic or subacute compression of the median nerve within the carpal tunnel, and its characteristic pathophysiology is demyelinating conduction-slowing until it is far advanced. Because of these different pathophysiologies, the clinical and EDX features of these two types of median nerve lesions are dissimilar. Patients with acute CTS clinically manifest marked weakness of the median nerve-innervated thenar muscles and fixed sensory deficits in the median nerve distribution in the hand and fingers. During the EDX examination, the compound muscle action potentials (CMAPs) recorded from the lateral thenar (median nerve-innervated) muscles and the sensory nerve action potentials (SNAPs) recorded from the median nerve-innervated fingers usually
are unelicitable, although occasionally they are simply low in amplitude. If they are the latter, then invariably the distal and peak latencies for the responses are normal or only slightly prolonged (consistent with the severe degree of demyelinating conduction block or axon loss present). When performing needle EMG of the lateral thenar muscles, fibrillation potentials are seen if the lesions are of 3 or more weeks duration, and either no motor unit action potentials (MUAPs) can be activated, or there is markedly reduced MUAP recruitment. These clinical and EDX changes are unlike those seen with typical CTS: the most prominent symptom usually is intermittent paresthesias in the median nerve-innervated fingers and, on EDX examination, the median NCSs are prolonged in latency (usually with normal amplitudes) and needle EMG of the lateral thenar muscles typically is normal.31

The EDX examination has little ability to “date” (i.e., to determine the duration of) any PNS lesions it detects. A major exception is axon loss injuries that are severe enough to affect the amplitudes of the NCSs, and the latter are performed during the first 10 days after lesion onset. During this “hyperacute” period, the NCS findings are predictable. Following a severe, focal axon loss lesion, the CMAP responses on stimulating and recording distal to the lesion site are normal for the first 2-3 days. Then they begin to progressively decrease in amplitude as more fibers are unable to conduct impulses to the recorded muscle. The CMAP amplitudes reach 15% of their pre-injury height at 5 days post-injury, and their nadir by 7 days post-injury. The SNAP amplitudes also progressively decrease on stimulating and recording distal to an axon loss lesion (at least one at or distal to the dorsal root ganglion), but they follow a different time course than the CMAPs. The SNAPs are unaltered for the first 5 days after lesion-onset, and then drop progressively over the next 4-5 days, reaching their nadir at day 10-11 after onset. While the motor and sensory NCS responses are decreasing in amplitude, but before they become unelicitable, the rates of conduction along the surviving fibers remain within or near the normal range. In any case, the NCSs with axon loss lesions are as severely affected as they will be by 10-11 days after lesion onset. With substantial axon loss lesions, the needle EMG will show reduced MUAP recruitment immediately after lesion onset and will persist, whereas fibrillation potentials develop anywhere between 10-35 days after onset, depending upon such factors as the distance between the lesion site and the muscle being sampled. To complicate matters, some patients are “late fibbers” and do not develop fibrillation potentials until several weeks (4-6) after lesion onset. With abrupt onset focal NCS lesions, detecting a demyelinating conduction block usually indicates the lesion is of less than 6 weeks duration, because most neurapractic injuries of such nature resolve within that time period. On needle EMG, fibrillation potentials tend to disappear from denervated muscles as time passes, either because of progressive reinnervation or because of muscle degeneration. However, the time course is variable, depending on such factors as the density of the fibrillation potentials initially present, and the distance between the injury site along the PNS fibers and the muscle being sampled. In general, relatively few fibrillation potentials are detectable in muscles which have been denervated more than 2 years. Complex repetitive discharges (CRDs) can help establish the duration of the lesion. Even though they are nonspecific (i.e., seen in both neuropathic and myopathic disorders), they generally do not appear with the former until the lesions are of at least 6 months duration. Whenever motor axon loss has been substantial enough to result in an appreciable amount of collateral nerve fiber sprouting, chronic neurogenic MUAPs are seen. These can help in “dating” a PNS lesion, since they generally require at least 4-6 months to develop. However, once present, they can persist indefinitely.31,33

After EDX abnormalities resulting from a focal PNS lesion appear, they can be quite variable in their duration. In some static focal disorders (e.g., CTS), they can remain relatively unchanged indefinitely. Conversely, with other disorders (e.g., radiculopathies) they have a notorious tendency to normalize with time. How stable the EDX findings are depends principally on the pathology at the lesion site. The changes seen with demyelinating conduction slowing lesions often are far more persistent than those seen with axon loss, which begin to resolve (or attempt to do so) almost as soon as they appear. For this reason, there is a window of time during which an EDX examination is likely to reveal abnormalities following an axon loss lesion. The EDX examination, specifically the needle EMG, should be performed no sooner than 3 weeks after lesion onset (to allow fibrillation potentials to develop), but ideally within the first 6 weeks (i.e., before fibrillation potentials may begin to disappear from muscles because of collateral sprouting).33

Generally, EDX examinations are not as helpful as detailed histories and clinical examinations in situations with multiple confounding factors. Thus, if the findings on the clinical examination are so complex and confusing that they are unintelligible, then the results of the EDX examinations are likely to be as equally complex and confusing.

Electrodiagnostic examinations are essentially worthless in chronic pain syndromes. This is partly because pain fibers are not assessed by the EDX examination, and partly because many of the responsible lesions are affecting sensory fibers proximal to the dorsal root ganglia. Also, many pain syndromes are due to factors other than PNS disorders. Regardless, in these patients the EDX studies are almost always normal, or show only “anticipated” abnormalities (e.g., absent H response or chronic neurogenic MUAPs in a distal S1 distribution in a patient who underwent prior lumbar
laminectomy for an S1 radiculopathy). A critical point is that these findings in this situation have no exclusionary value—specifically, they do not exclude ongoing compression of preganglionic sensory root fibers.31,32

The workplace is diverse, which means the type and severity of injuries that employees can sustain are variable. Even when restricted to PNS and various musculoskeletal injuries, the diversity is striking. For example, almost every peripheral nerve can be injured at virtually any point along their course.

In many instances, these lesions are straightforward and their EDX presentations are identical to those seen in lesions of a similar type that are not work-related.

Two PNS disorders responsible for a disproportionate number of work-related referrals to the EDX laboratory merit special comment: lumbosacral radiculopathies and CTS.

Low back pain, with or without lower limb radiation, is probably the most common cause for workers being referred to physicians for evaluation and treatment. Most low back injuries occur in workers who are 20-40 years of age (similar to the age range for persons whose back injuries are not work-related). The occupations with the highest incidence of back injuries that result in workman’s compensation are nurses, truck drivers, and machine operators.19

Most patients with low back pain do not have lumbosacral radiculopathies and, hence, if EDX examinations are performed they will be normal. There are certain facts regarding the EDX assessment of lumbosacral radiculopathies, including those that are work-related, that must be appreciated. First, the timing of an EDX examination is critical if it is to reveal evidence of a radiculopathy. The study must be performed during the period when any fibrillation potentials present have had time to develop fully, but not yet had time to disappear because the muscle fibers generating them have been reinnervated via collateral sprouting. Too often in this author’s experience, for one reason or another, worker’s compensation cases referred for EDX examination have had static symptoms for many months to years. Such late referrals almost guarantee that, even if a radiculopathy were present, it would be undetectable on EDX examination. Second, a normal EDX examination never excludes a radiculopathy, particularly a chronic, static radiculopathy, because false-negative studies are inherent to the procedure. Any physician who uses such negative results under these circumstances to reassure the patient that he/she does not have a radiculopathy is providing misleading information.30,32 Third, EDX examinations and magnetic resonance imaging (MRI) studies are competitive procedures, not complementary, as is often assumed.17 Thus, usually they are helpful or unhelpful in the same groups of patients. Because of this, if one study has not provided useful information in a given patient, typically it is pointless to perform the other. The majority of clinicians—with the exception of some EDX physicians—obtain MRI studies on their patients with suspected radiculopathies, including the possible work-related ones, in lieu of EDX examinations. Consequently, EDX examinations are not performed because they usually are not indicated. There are a few seldom encountered exceptions in which the EDX examinations can be of benefit when MRI studies cannot. The first is when marked weakness is present with a recent onset root lesion. If the parietic/paralyzed muscle is one that can be used as a recording site during motor NCSs (e.g., tibialis anterior with peroneal motor NCSs), and the symptoms are of at least 6 days duration, then the pathophysiology of the lesion responsible for the weakness can be determined—demyelinating conduction block or axon loss/conduction failure.31 Also, radiculopathies caused by something other than compression (e.g., diabetes, mellitus, HIV, and Lyme disease) can be detected, and proximal PNS disorders other than root lesions (e.g., plexopathies) can be identified. A special situation occurs when pregnant employees develop possible work-related radiculopathies. In these situations, the EDX examination often becomes the diagnostic procedure of choice.20

Carpal tunnel syndrome is the most common human entrapment neuropathy. When it is suspected of being a manifestation of a work-related injury, it also becomes the most contentious of any focal nerve lesion.5,6,18,21 More publications have focused on occupational CTS than any other single work-related topic (a recent Medline search yielded over 100 articles which have appeared in less than a decade). Unfortunately, the number of articles is matched only by the number of controversies they have generated. The following statement understates the major controversy: “Some debate exists within the medical, political, and business arenas regarding the strength of the associations between repetitive work-related activities and musculoskeletal disorders (including CTS).”7 This altercation is aptly illustrated by excerpts from two publications that appeared in 2002 which are impossible to reconcile. The first is “at least one-half of all cases of carpal tunnel syndrome in North America appear attributable to occupational hand activities.”21 The second states “except in the case of work that involves cold temperatures (possibly in conjunction with load and repetition)…work is less likely than demographic and disease-related variables to cause CTS.”6 Virtually every aspect of work-related CTS is in dispute, including how to diagnose it (and the value of NCSs) and, if it is found in an employee, what its relationship is to the work he/she performs. Although NCSs of various types are considered by many to be as close to the gold standard as can be achieved at present for diagnosing CTS,10 they are a major part of the controversy. Establishing “normal” and “abnormal” has proven contentious.5,21 The high sensitivity reported for median NCSs in relationship to CTS, encountered in the clinical setting
with symptomatic patients, is not found in cross-sectional workplace screening. Moreover, the specificity is lower than it was earlier thought to be.29

A recent report by Cosgrove and colleagues illustrates the problem well.5 They selected 900 railroad workers from more than 2500 who reputedly had work-related CTS. All had initiated, or were in the process of initiating, legal proceedings against their employer. Of the 900, 248 had already undergone CTS surgery. Although all of the claimants reportedly had some type of NCS positive for CTS, the authors found (at least for the operated cases) that the EDX data “were difficult to interpret for reasons such as the absence of ulnar or radial distal latencies, failure to account for skin temperature, poor description of techniques, or no numerical data supplied.” All participants “were subject to a comprehensive history, physical examination, medical record review,” and an EDX assessment. The EDX study consisted of a “median minus ulnar distal latency differential,” always utilizing both motor and sensory responses, and selectively using mid-palmar responses. They found a statistically significant relationship between CTS and body mass index, age, and wrist index. Almost all the participants were men, and most were overweight and relatively old. They found no association, however, between CTS and job classification. Using their diagnostic criteria, more than half the studied population did not have CTS, and among the surgical cases, based on perioperative data, they could not determine the justification for operation in one-third of them because the NCS results appeared normal both pre- and post-surgery.5

Of all the work-related PNS disorders assessed in the EDX laboratory, CTS is the only one often screened for (in mostly asymptomatic persons), either during the pre-employment assessment, or in the post-offer preplacement (POPP) period.9 Whether such screening is beneficial also is debated.9,27

A review of the literature reveals that there is some (but definitely not universal) consensus regarding patient occupation and CTS:

1) The prevalence of CTS among any patient group decreases significantly if the diagnosis rests on the presence of both clinical and EDX findings, rather than solely on the former.2,10,12,21,24 Thus, in one study of dental hygienists, 42% could be classified as having CTS based on symptoms alone, but this figure dropped to 8.4% when abnormal median NCSs were added to the diagnostic requirements.2

2) Workers in certain occupations have a substantially higher risk than normal for developing CTS. Examples include those workers in the meat, fish, poultry processing industry, as well as those who use vibrating tools.13,21 Conversely, workers in certain other occupations, despite the fact that they have substantial upper limb symptoms, either have no appreciable increased risk of having CTS (computer and keyboard users),1,12 or only a moderately increased risk (dental professionals).8,20,28 One problem in determining the prevalence of CTS in various occupational groups is that often little attempt is made to distinguish one neuromuscular disorder from another. Thus, neuromuscular limb complaints are common among dental hygienists, but these are more often related to epicondylitis, particularly lateral epicondylitis, than to CTS.20,28

3) Abnormal median NCSs found in asymptomatic workers probably have little predictive value in regard to whether those workers will subsequently develop CTS.12,21

In contrast to the above, there is no consensus, in regard to workers with CTS, concerning the relative contribution of such factors as obesity, cigarette smoking, age, gender, and wrist index, compared to their work activities, in the genesis of their PNS disorder.5,6,18,21

CONCLUSION

The EDX examination can be beneficial in assessing workers with reputed or definite job-related PNS injuries. However, it has many limitations which the referring physician must be cognizant.

REFERENCES


