AMA Guides™ Newsletter

Expert advice, practical information, and current trends on impairment evaluation

July/August 2012

In this issue

Upcoming issues

The *AMA Guides™ Newsletter* provides updates, authoritative guidance, and AMA interpretations and rationales for the use of the *AMA Guides to the Evaluation of Permanent Impairment.*

Evaluating Causation for the Opposite Upper Limb

J. Mark Melhorn, MD, James B. Talmage, MD, Charles N. Brooks, MD, and Christopher R. Brigham, MD

Workers' compensation and personal injury claims often become embroiled in debates over the cause of the clinical presentation. The deliberation stems in large part from an administrative need to determine if an employer, insurer, or some other potentially responsible party is financially liable for the evaluation and treatment of the condition. Such causation issues can also play a role in determining whether any disability compensation or permanent impairment rating is warranted.

When the primary claim involves an extremity, afflicted individuals sometimes report subsequent symptoms in the contralateral, previously "normal" limb, and often attribute this onset to overuse while favoring the initially involved extremity. This overuse hypothesis apparently seems plausible (perhaps even intuitively obvious) to some.

However, health care professionals and scientists cannot credibly rely on superficial considerations of plausibility or intuition. A solid basis in science is required instead. This article provides a review of the medical literature and, in so doing, reveals that there are no credible studies that support such a causative relationship. The concept that favoring one upper limb can result in injury to or illness in the other is not based on scientific evidence; instead, it is an unsupportable myth.

Example—Carpal Tunnel Syndrome

Many laypersons and physicians believe that repetitious use of an extremity will cause illness or injury. While true in some cases, it is often a misconception.

Such findings have relevance for the issue of symptoms developing in an extremity that is opposite to an initially claimed malady. Given the scientific findings that activity and use actually promotes health, it does not appear to be credible to claim that increased use of the opposite limb (prompted by disuse of the limb for which problems were originally claimed) would lead to health problems.

For example, the workers' compensation system has been plagued by scientifically nonsupportable claims that prolonged typing on a computer keyboard could cause carpal tunnel syndrome (CTS). However, multiple attempts to scientifically establish a causative link between typing and CTS have failed to do so.[1-13] In fact, keyboarding may be protective.

An example of purported favoring may be illustrative. After working as a fast food cashier for 4 weeks, a 50-year-old, right-handed woman complained of gradual onset of numbness and tingling in her *left* hand and radial digits. She denied any precipitating injury. The

AMA AMERICAN MEDICAL ASSOCIATION

woman is an insulin-dependent diabetic, who has a congenitally "square wrist" and has rheumatoid arthritis. Family history is remarkable for a mother who had bilateral carpal tunnel releases. Physical findings included a height of 65 inches and weight of 277 pounds, with a calculated (http://www.nhlbisupport.com/bmi/) body mass index (BMI) of 46. According to the National Institutes of Health (NIH), a BMI below 18.5 is underweight, 18.5 to 24.9 normal, 25 to 29.9 overweight, 30 to 39.9 obese, and 40 or higher morbidly obese.

Based on history, clinical examination, and nerve conduction studies, a physician diagnosed left carpal tunnel syndrome, dispensed a left wrist splint, and advised avoidance of "repetitive hands use" at work. Because the employer had no work available that complied with this restriction, the woman was off work. After 2 weeks of wearing the splint on the left wrist, she complained of new onset numbness and tingling of her *right* hand and radial digits. Physical findings were also similar on the right, and a nerve conduction study of right median nerve revealed a distal peak sensory latency of 3.9 milliseconds for a 14-cm distance, interpreted as "possible median nerve entrapment."

The left CTS was accepted as work compensable, but a second claim that "favoring the left" caused overuse of the right, and thus CTS, was denied. Attending physicians are often asked for an opinion on situations such as this one—whether "favoring" the left caused CTS on the right. Without considering the following steps, one physician might say "no," and another might incorrectly say "yes."

Causation Analysis

Determining relationships between risk factors or exposures and medical condition is a complex process. The specific steps are outlined in Table 3-1[14] Steps for Concluding a Causal Association Exists in *Guides to the Evaluation of Disease and Injury Causation (Causation)*:

1. Collect all epidemiologic literature on the disorder.
2. Identify the design of each study.
3. Assess the methods of each study.
4. Ascertain statistical significance and the degree to which change may have produced the results.
5. Assess the studies using the updated Bradford Hill criteria for causation.
6. Conclusion about the degree to which a causal association is or is not present.

This information is then applied to the specifics of the case. This process is reflected in *Causation*, Table 3-2, National Institute for Occupational Safety and Health/American College of Occupational and Environmental Medicine Steps for the Determination of Work-Relatedness of a Disease (which was adapted from Kusnetz and Hutchison, eds. DREW, CDC, NIOSH, Pub. No. PB298-561; 1979 and Occupational Medicine Practice Guidelines, 2nd Ed., ACOEM OEM Press, 2004).[14] The steps are:

1. Identify evidence of disease.
2. Review and assess the available epidemiologic evidence for a causal relationship.
3. Obtain and assess the evidence of exposure.
4. Consider other relevant factors.
5. Judge the validity of testimony.

6. Form conclusions about the work-relatedness of the disease in the person undergoing evaluation.

In this case, both attribution of the initial left-sided symptoms to work and subsequently the right-sided symptoms to those on the left, appear based on *post hoc ergo propter hoc* ("after this, therefore caused by this") reasoning. However, temporal sequence does not prove causation. An example often cited to show the inaccuracy of this kind of reasoning is that a rooster crowing does not make the sun come up, even though the sun rising always occurs after the rooster crows. In causation analysis, one must also consider temporal proximity or disparity, but this is only one of the nine Bradford Hill criteria for causation that should be scientifically established before causation is attributed to an exposure.

Causation analysis must be based on both scientific evidence and the facts of the individual case. To conclude that an occupational exposure and an effect are etiologically associated with a reasonable degree of probability or certainty, all 3 of the following criteria must be met[15]:

1. The patient has an illness (diagnosis is proven) compatible with a disease-producing agent or an injury that could have arisen from a mechanism at work (Bradford Hill criteria for causation have been scientifically established).
2. There probably was sufficient exposure in this worker's occupational environment to potentially cause the disease, or a plausible mechanism of injury of sufficient magnitude to cause the trauma, or a mechanism that could have aggravated a prior condition causing the need for treatment or additional treatment that would not have been necessary without the work-related exposure.
3. The preponderance of evidence supports that the disease or injury is occupational rather than nonoccupational in origin.

This is the principle physicians are asked to consider when assessing causality: that a probable cause and effect likely are etiologically related. If any one of the 3 criteria is *possible* but not *probable*, causation has not been established. Furthermore, 2 or more possible causes do not equal one probable cause. In other words, multiple possible causes are not additive to make a probable cause.

The evaluator needs to be able to determine if a claimed work-related clinical presentation is really work-related and/or caused by litigated events.[16] If an exposure results in symptoms of an underlying condition, it is important to discern whether this reflects a temporary exacerbation or a permanent aggravation (a permanent worsening of the underlying condition altering the natural course of that underlying condition). If science and facts in the case support the conclusion that multiple factors caused or permanently aggravated a condition, it may be necessary to apportion responsibility to the causes. Apportionment must be supportable by science and not merely opinion.

When evaluating causation, the physician must identify the correct diagnosis or diagnoses (the effect) and the possible causes thereof (both occupational and nonoccupational), then assess the likelihood of a causal relationship between them. In other words, causation analysis must be based on an analytical approach. However, as apparently occurred in this case, some physicians opine that an injury or exposure, often at work, caused or aggravated a condition based on temporal sequence alone. Credibility is challenged even more when the source of the claim of a temporal sequence is the patient ("I did not have this before.") in that scientific findings have repeatedly demonstrated that patient-reported histories are extremely unreliable.[17]

The Science

Prospective randomized controlled studies prove or disprove a hypothesis.[15] This study methodology is generally required to prove a test or treatment improves disease outcome. Because in a free society we cannot randomly assign people to potential toxins or to jobs with potential risk factors, the prospective cohort study is our best evidence and is considered the only hypothesis testing methodology for exposure-causation questions. These are expensive and difficult studies to do, so most of the available literature is based on low-quality case control studies.

Therefore, if the hypothesis is that "favoring" an upper limb can result in the same or similar condition in the opposite, previously "normal" limb, the data are going to be limited to indirect evidence. A search of PubMed and MEDLARS using all fields returned 6 articles for the phrase "opposite uninjured arm" and 120 articles for the phrase "uninjured arm." None of these articles address causation or were appropriate for this review. A search for the phrase "asymptomatic shoulder causation" returned 16 articles of which 4 were applicable; "asymptomatic elbow causation" returned 2 articles, neither of which were relevant; "asymptomatic wrist causation" returned 9 articles, 5 of which were applicable; and "asymptomatic hand causation" returned 120 articles, 5 of which were applicable, but 4 of them were duplicates. A general web search engine provided one reference to "symptoms in the opposite or uninjured arm," but it had already been available to the authors. These 6 articles were combined with articles already known to the authors for this review.

Reasonable scientific logic suggests there are 3 key points when considering "favoring":

1. The individual alleges overuse of the "normal" upper extremity because he or she is doing more with that limb ("overuse") and less with the injured side. This scenario is not supported by the literature. In fact, most persons have a significant decrease in total activity because of the first injury or illness. Most persons alleging this mechanism of injury are already on major work restrictions and, thus, either doing a different and easier job or are off work alleging they do very little at home.
2. The incidence rate for favored upper limb onset is not significantly higher than the incidence rate for the dominant side, as would be expected in this situation of assuming the cause is due to "overuse."
3. Much of the literature suggests that the contralateral uninjured or asymptomatic limb at the time of the initial shoulder, elbow, or wrist injury or illness is usually not normal but has already developed some disease. Therefore the development of the condition on the initially uninvolved contralateral side is more probably related to individual risk factors, such as genetics, age, and sex, rather than overuse.

Only one article found specifically addresses the issue of symptoms in the opposite or uninjured arm. It was written in 1999 in response to a request by the Ontario Workplace Safety and Insurance Appeals Tribunal and is entitled "Symptoms in the Opposite or Uninjured Arm." It was written by W. Robert Harris, MD, and Ian Harrington, MD.[18] The authors state their purpose as:

> In recent years the Tribunal has heard an increasing number of appeals in which it is claimed that a painful injury to one upper extremity causes the patient to "favour" it. The patient concludes that this produces "overuse" of and hence pain in the opposite normal one. Usually, but not always, the symptoms in the "normal" limb resemble those of the original side.
>
> There are three conditions that occasion most of such appeals:
>
> 1. Injury to the rotator cuff of the shoulder.
> 2. Injury to the origin of the muscles that move the wrist: lateral epicondylitis (tennis elbow) and medial epicondylitis (golfer's elbow); and
> 3. Carpal Tunnel Syndrome (CTS).

For rotator cuff injury, Drs. Harris and Harrington concluded:

> If the symptoms in the opposite side are similar to those of the injured one, then it must be proven that a) there is a rotator cuff injury and b) that favouring the injured side obliges the patient to strain the "normal" cuff by repetitive overhead use of the shoulder. If the symptoms in the opposite limb are different than those of the injured one, then it must be clearly shown how "favouring" the injured side could have caused them.
>
> Furthermore, other causes of arm pain, such as aging changes in the neck with referred pain in the arm, must be ruled out.
>
> Shoulder and elbow symptoms are in most cases the result of aging changes which can occur simultaneously, in [both] . . . sides, so that symptoms are commonly bilateral. Symptoms usually begin in the dominant side. There must be strong evidence that the compensable injury did cause similar overuse of the "normal" side.

For lateral and medial epicondylitis the authors concluded:

> If the symptoms are similar to those on the injured side, then it must be proven that "favouring" that side obliged the patient to undertake activity that involved excessive wrist movement against resistance. In this respect, activities of daily living such as dressing, washing, writing or eating cannot be classified as involving excess wrist movement against resistance.

For carpal tunnel syndrome they concluded:

> If the symptoms do not resemble those of the injured side, then the examiner must identify their cause, and try to show how this was the result of "favouring" the injured side. Again, other causes of pain, such as aging changes in the neck with pain referred down the arm, must be ruled out.
>
> Nearly always when this is claimed, the symptoms on the "normal" side are similar to those on the injured one. The examiner must be certain that the diagnosis is proven. One must be satisfied that the "favouring" obliged the patient to overuse the finger flexor muscles on the "normal" side. In this respect, it is important to differentiate between wrist movement (which does not require use of the finger flexor muscles) and finger movement.
>
> Idiopathic CTS is commonly bilateral (87% of cases in Padua's series),[19] and it is difficult to prove that symptoms that began on one side caused similar ones in the other. And it should be remembered that diabetics with diabetic neuropathy may have symptoms that mimic CTS.

In summary, Drs. Harris and Harrington suggest:

> Claims of serious or persisting painful syndromes in the arm or hand opposite to the injured one are seldom supported by adequate clinical scientific evidence. If it (the favoring hypothesis) were true, one would expect that nearly everyone with pain in one upper extremity would develop pain in the opposite one, and that simply does not occur.

Shoulder

Direct Evidence for the Shoulder

No additional studies were found. (No one has published a study with a prospectively acquired data set to assess this question).

Indirect Evidence for the Shoulder

A longitudinal study of 45 patients (22 men, 23 women) from a cohort of 58 potential subjects with unilateral shoulder symptoms who had contralateral asymptomatic rotator cuff tears confirmed by ultrasound found that 23 (51%) of the previously asymptomatic shoulders became symptomatic with a mean of 2.8 years. The average Activities of Daily Living (ADLs) score for those remaining asymptomatic was 28.5 of 30 and for those becoming newly symptomatic, 22.9 of 30 ($P < .5$). The mean visual analog pain score (1 = no pain) for those remaining asymptomatic was 1.1 and for the newly symptomatic patients, 4.0. Of the 23 patients who returned for ultrasound, 9 were asymptomatic and 14 symptomatic. Only 2 of the 9 patients remaining asymptomatic had progression of their tears. Overall, 9 of 23 patients had tear progression. No patient had a decrease in the size of the tear. This study demonstrates that previously asymptomatic rotator cuff tears can develop symptoms in the context of a contralateral symptomatic tear. There appears to be a risk for tear size progression over time.[20]

A longitudinal study published in 2004 using a random sample of 826 individuals with 48% retention, first assessed as high school students 15 to 18 years old and later as 22- to 25-year-olds, found the prevalence of weekly neck and shoulder pain to increase from 17% to 28%. Of those who were asymptomatic at baseline, 59% had weekly neck and shoulder pain 7 years later with psychosomatic stress being a high risk factor (predictor) while p*articipation* in sports that dynamically loaded the upper extremities resulted in decreased risk of symptoms.[21] This study supports the conclusion that activity is beneficial.

A prospective cohort study followed 501 active workers for an average of 5.4 years. Incident cases were defined as workers who were asymptomatic at baseline testing and had no prior history of upper extremity tendinitis (shoulder) and went on to be diagnosed with an upper extremity tendinitis (UET) during the follow-up period or at follow-up evaluation. The incident cases were compared to the subset of the cohort who also had no history of an UET and did not develop tendinitis during the study. The cumulative incidence in this cohort was 24.3%, or 4.5% annually. The factors found to have the highest predictive value for identifying a person likely to develop an UET in the near future included age over 40, a BMI over 30, a complaint at baseline of neck or shoulder discomfort, a history of CTS, and a job with a higher shoulder posture rating. The risk profile identifies both ergonomic and personal health factors as risks, and both categories of factors may be amenable to prevention strategies.[22]

A cohort of 195 subjects with an asymptomatic rotator cuff tear was prospectively monitored for pain development and examined annually for changes in various parameters such as tear size, fatty degeneration of the rotator cuff muscle, glenohumeral kinematics, and shoulder function. Forty-four subjects were found to have developed new pain, and the parameters before and after pain development were compared. The 44 subjects were then compared with a group of 55 subjects who remained asymptomatic over a 2-year period. The study found with pain development, the size of a full-thickness rotator cuff tear increased significantly, with 18% of the full-thickness tears showing an increase of >5 mm; while 40% of partial-thickness tears had progressed to a full-thickness tear. All measures of shoulder range of motion were decreased except for external rotation at 90 degrees of abduction.[23] The above studies were prospective cohorts. The following studies are lower methodologic–quality case control series.

A case series found that the individual's anthropometrics affected his or her perception of musculoskeletal mechanical loads on the shoulder.[24]

A case series compared 37 shoulder MRI scans from paraplegic subjects (26 symptomatic, 11 asymptomatic) and 27 MRI studies from able-bodied subjects (17 symptomatic, 10 asymptomatic) to evaluate the possible effect of "overuse" on the risk of shoulder impingement. Of the symptomatic paraplegic subjects, 73% of shoulders imaged showed a rotator cuff tear compared to 59% of shoulders in able-bodied symptomatic subjects. Of all subjects with paraplegia, 57% of shoulders imaged showed rotator cuff tears, and the prevalence and severity of tears correlated positively with age and duration of spinal cord injury. Tears involving the posterior rotator cuff were found in 74% of the paraplegic subjects compared with 50% of the able-bodied subjects.[25]

In a randomized controlled trial, 67 symptomatic male construction workers (mean age 49) were assigned to a treatment intervention group (N = 34) or a control group (n = 33). Twenty-five symptomatic subjects served as an additional control group. Subjects in the intervention group were instructed in a standardized 8-week home exercise program of 5 shoulder stretching and strengthening exercises. Subjects in the control groups received no intervention. Testing after 8 to 12 weeks revealed the intervention subjects reported significantly greater reductions in pain and disability than controls.[26] This study also supports the conclusion that appropriate activity is beneficial.

In a case series, 118 shoulder outlet X rays and ultrasounds were performed on 59 asymptomatic patients in various age groups. Acromial morphology and age were then correlated

with the ultrasound findings. Older patients were noted to have a high incidence of type II and III acromions (93% of those over 70). Full- and partial-thickness tears were more common in patients with type II or type III acromions compared with type I. Patients over 50 years of age had a high incidence of full-thickness tears (40%), but the incidence did not increase with advancing age past 50. These findings lend credence to the multifactorial etiology of rotator cuff tears. The patient's age (degeneration) and acromial morphology (impingement) are two causative factors in rotator cuff tears. The incidence of cuff tears and type III acromions was high in this group of asymptomatic subjects. The authors conclude that both of these findings should be regarded as part of the natural aging process.[27] Findings on imaging studies do not necessarily reflect a clinical diagnosis.

A case series prospectively followed 51 patients 60 years old or younger who had a full-thickness rotator cuff tear equal to or larger than 5 mm treated nonoperatively. At a follow-up of 25 to 39 months (mean, 29), 49% of the 61 tears (30 tears) had increased in size, 43% (26 tears) had not changed, and 8% (5 tears) decreased in size. Of the 41 initially intact shoulders, 25% (10 shoulders) developed a new full-thickness rotator cuff tear. No correlation was found between the change in tear size and age of the patient ($P = .85$), sex ($P = .93$), existence of a prior trauma ($P = .63$), size of tear at index ultrasound ($P = .62$), and bilateral tears ($P = 1.00$). There was a correlation between the existence of considerable pain at the time of the follow-up ultrasound and a clinically significant increase in tear size ($P = .002$). The authors concluded that full-thickness rotator cuff tears tend to increase in size in about half of patients ages 60 years or younger.[28]

A case series of 588 consecutive patients in whom a standardized ultrasound had been performed found 212 had an intact rotator cuff bilaterally, 199 had a unilateral rotator cuff tear (partial or full thickness), and 177 had bilateral tears (partial or full thickness). The presence of rotator cuff disease was highly correlated with age. The average age was 48.7 years for patients with no rotator cuff tear, 58.7 years for those with a unilateral tear, and 67.8 years for those with a bilateral tear. Logistic regression analysis indicated a 50% likelihood of a bilateral tear after the age of 66 years ($P < 0.01$). In patients with a bilateral rotator cuff tear in whom one tear was symptomatic and the other asymptomatic, the symptomatic tear was significantly larger ($P < 0.01$). The average size of a symptomatic tear was 30% greater than that of an asymptomatic tear. Overall, patients who presented with a full-thickness symptomatic tear had a 35.5% prevalence of a full-thickness tear on the contralateral side.[29]

A case series of 96 individuals with asymptomatic shoulders were evaluated to determine the prevalence of a rotator cuff tear. The overall prevalence of a cuff tear in all age-groups was 34%. There were 14 full-thickness tears (15%) and 19 partial-thickness tears (20%). The frequency of full-thickness and partial-thickness tears increased significantly with age ($P < .001$ and .05, respectively). Twenty-five (54%) of the 46 individuals who were more than 60 years old had a tear of the rotator cuff: 13 (28%) had a full-thickness tear, and 12 (26%) had a partial-thickness tear. Of the 25 individuals who were 40 to 60 years old, one (4%) had a full-thickness tear, and 6 (24%) had a partial-thickness tear. Of the 25 individuals who were 19 to 39 years of age, none had a full-thickness tear, and 1 (4%) had a partial-thickness tear.[30]

A case series of 90 asymptomatic adults between the ages of 30 and 99 years evaluated the rotator cuff using ultrasound. The study found no statistically significant difference in the incidence of impingement findings between dominant and nondominant arms or between the sexes. The prevalence of partial- or full-thickness tears increased markedly after 50 years of age: these were present in more than 50% of dominant shoulders in the seventh decade and in 80% of subjects over 80 years of age. The authors concluded that rotator-cuff lesions are a natural correlate of aging and are often asymptomatic.[31]

In summary, the articles reviewed do not support "favoring" as a reasonable cause for development of symptoms in the contralateral shoulder. Rotator cuff lesions are seen with aging and are often asymptomatic. Typically, activity is beneficial, not detrimental. Although workplace risk factors may contribute to shoulder symptoms, as outlined in *Causation*, it appears most shoulder conditions develop at a similar rate. In most cases "favoring" is not a probable cause of shoulder pathology. Thus, even if symptoms in the second limb develop after symptoms are present in the first limb (a temporal relationship, or one of time), there is no scientific support for the concept that having symptoms in the first limb causes an increased rate of disease in the second limb.

Elbow

Direct Evidence for the Elbow

No additional studies were found. (No one has published a study with a prospectively acquired data set to assess this question).

The British Systematic Review of upper limb (and elbow) tendinitis and tendinopathy did not find evidence to attribute the development of elbow tendinitis to ergonomic exposures, even in cases in which first one side became symptomatic and then, later, the other side.[32]

Thus, if there is not sound scientific evidence to attribute the development of symptomatic elbow tendinopathy on the first side to workplace exposures, there is even less evidence to attribute the development of symptomatic elbow tendinopathy on the second side to favoring the original malady.

Indirect Evidence for the Elbow

As discussed by Harris and Harrington and *Causation*, it is important to confirm a specific diagnosis for the elbow, not simply elbow pain. Specific risk factors for the elbow can be reviewed in Chapter 9, Upper Limb, in *Causation*. Currently there is insufficient evidence to support "favoring" as a probable cause for symptom onset in the originally uninvolved elbow.

Carpal Tunnel Syndrome

Direct Evidence for the Hand or Wrist (Carpal Tunnel Syndrome)

No additional studies were found. (No one has published a study with a prospectively acquired data set to assess this question).

Indirect Evidence for the Hand or Wrist (Carpal Tunnel Syndrome)

A prospective cohort studied 266 hands in 133 patients with carpal tunnel syndrome (CTS) and found the incidence of bilateral clinical CTS in the study population was 87%. Neurophysiological impairment of median nerve was observed in about half of the asymptomatic hands. Follow-up of patients with unilateral CTS showed that contralateral symptoms developed in most cases. The authors concluded that bilateral impairment of median nerve is the rule in patients with CTS and probably it has been underestimated in previous studies. According to neurophysiological classification, most patients had a similar grade of CTS in both hands with a maximum of one grade of difference. This observation suggests that further investigation may be required when there is atypical involvement with a marked difference in impairment between the hands. The authors postulated that CTS is bilateral in almost all cases, with similar nerve impairment, and that most cases of unilateral CTS will probably become bilateral.[33]

A prospective cohort of 77 asymptomatic workers with electrodiagnostic findings of median mononeuropathy were compared to an age and sex–matched control group. Follow-up was completed an average of 70 months later, and subjects who reported pain, numbness, tingling, or burning in the distribution of the median nerve, based upon a hand diagram, were classified as having CTS symptoms. The follow-up participation rate was 70%. Among subjects with abnormal median sensory latencies, 23% went on to develop symptoms consistent with CTS within the follow-up period, compared with 6% in the control group (P = .010).[34]

A case series compared three cohorts: control subjects without occupational exposure to highly forceful or repetitive hand exertions (N = 105), industrial workers with hand/wrist symptoms (N = 103), and asymptomatic industrial workers (N = 137). The study found mean sensory amplitudes were significantly smaller (P < 0.05) and motor and sensory distal latencies were significantly longer ($P < 0.001$) in the industrial "asymptomatic hand" population compared to the control population. Prolongation of median relative to ulnar latency was significantly longer in the asymptomatic industrial population ($P < 0.05$).[35]

A retrospective case series of 131 patients who had open carpal tunnel release for CTS in 229 hands found the symptoms were present bilaterally in 59% of patients when first seen. Neurophysiological impairment of the median nerve was observed in 66% of the asymptomatic hands, and 73% of patients in this group developed symptoms of CTS after the opposite side had been operated on. Follow-up of patients with unilateral CTS showed that subsequent development of disease in the unaffected hand is very common. The authors concluded CTS is a bilateral disorder and becomes more evident as time passes. There is a correlation between the duration of symptoms and bilateral occurrence. A recent publication revealed the incidence of bilateral symptoms is between 60% and 87% and was 59% at the first visit in their study. In those with unilateral symptoms, more than half had positive electrodiagnostic test results in the asymptomatic, contralateral hand.[36]

In summary, the articles reviewed above suggest CTS is, or will be, a bilateral condition. Most individuals have some decreased median nerve function in the asymptomatic wrist at the time of onset of their contralateral CTS symptoms. Most studies have shown CTS is a result of complex interaction between individual and workplace and/or avocational risk factors.[37-40] Causation analysis for workplace factors is hence very challenging, as discussed in Chapter 9, Upper Limb, in *Causation* and requires a systematic approach as outlined above. Although workplace risk factors may contribute to CTS symptoms, it appears, as delineated in *Causation*, in the majority of cases occupation and occupational exposure are not major factors in the development of carpal tunnel syndrome on the first side. It is common for symptoms to manifest in the contralateral hand, regardless of whether the first hand diagnosis results in modification of work and recreational activity or no modification in work or recreational activity.

Summary

Causation is a complex issue that involves both a medical determination and legal threshold.[41] Although epidemiological studies can provide general information regarding risk, this must be filtered by taking specific steps for assessing causal association for a disorder and determining if the injury is work related and then applying this to a specific individual. It is hoped this review will provide insight into the appropriate methods for providing causal opinions and result in improved consistency thereof based on scientific evidence.

The authors wish to invite individuals with opposing views, additional articles, or research materials to contact Dr. J. Mark Melhorn at Melhorn@onemain.com.

References

1. Melhorn JM, Martin D, Brooks CN, Seaman S. Upper limb. In: Melhorn JM, Ackerman WE, eds. *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL: AMA Press; 2008:141-202.
2. Ring D. Carpal tunnel syndrome causation. In: *74th Annual Meeting of the American Academy of Orthopaedic Surgeons at San Diego, CA Feb. 16, 2007*. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2007:1-2.
3. Fisher B, Gorsche R. *Diagnosis, Causation and Treatment of Carpal Tunnel Syndrome: An Evidence-Based Assessment*. Alberta, Canada: Workers' Compensation Board; 2004:1-153.
4. Lo SL, Raskin K, Lester H, Lester B. Carpal tunnel syndrome: a historical perspective. *Hand Clin*. 2002;18:211-217.
5. Egilman D, Punnett L, Hjelm EW, Welch L. Evidence for work-related musculoskeletal disorders. *J Occup Environ Med*. 1996;38:1079-1080.
6. Brenner LH, Bal BS, Brenner JA. Beyond medical negligence–judgement immunity. *Ortho Med Leg Advisor*. 2007;1:1-4.
7. Nordstrom DL, Vierkant RA, DeStefano F, Layde PM. Risk factors for carpal tunnel syndrome in a general population. *Occup Environ Med*. 1997;54:734-740.
8. Andersen JH, Thomsen JF, Overgaard E, et al. Computer use and carpal tunnel syndrome: A 1-year follow-up study. *JAMA*. 2003;289:2963-2969.
9. Nathan PA, Meadows KD, Istvan JA. Predictors of carpal tunnel syndrome: an 11-year study of industrial workers. *J Hand Surg*. 2002;27A:644-651.
10. Clarke Stevens J, Witt JC, Smith BE, Weaver AL. The frequency of carpal tunnel syndrome in computer users at a medical facility. *Neurology*. 2001;56:1568-1570.
11. Hadler NM. *Occupational Musculoskeletal Disorders*. Philadelphia, PA: Lippincott Williams & Wilkins; 1999:1-300.
12. Garland FC, Garland CF, Doyle EJ, et al. Carpal tunnel syndrome and occupation in U.S. Navy enlisted personnel. *Arch Environ Health*. 1996;51:395-407.
13. Garg A, Kapellusch J, Hegmann K, et al. The Strain Index (SI) and Threshold Limit Value (TLV) for Hand Activity Level (HAL): risk of carpal tunnel syndrome (CTS) in a prospective cohort. *Ergonomics*. 2012;55:396-414.
14. Hegmann KT, Oostema SJ. Causal associations and determination of work-relatedness. In: Melhorn JM, Ackerman WE, eds. *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL, AMA Press; 2008:33-46.
15. Melhorn JM, Hegmann KT. Methodology. In: Melhorn JM, Ackerman WE, eds. *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL, AMA Press; 2008:47-60.
16. Barth RJ. Determining injury-relatedness, work-relatedness, and claim-relatedness. *Guides Newsletter*. May/June 2012;95:1.
17. Barth RJ. Examinee-reported history is not a credible basis for clinical or administrative decision making. *Guides Newsletter*. September/October 2009;79:1.
18. Harris WR, Harrington IJ. *Symptoms in the Opposite or Uninjured Arm*. Ontario, Canada: The Workplace Safety and Insurance Appeals Tribunal; 1999:1-10. Copyright Queen's Printer for Ontario, 1999. Reproduced with Permission.
19. Padua L, Padua R, Nazzaro M, Tonali P. Incidence of Bilateral Symptoms in Carpal Tunnel Syndrome. *J Hand Surgery Br*. 1998;23b(5):603-606.
20. Yamaguchi K, Tetro AM, Blam O, et al. Natural history of asymptomatic rotator cuff tears: a longitudinal analysis of asymptomatic tears detected sonographically. *J Shoulder Elbow Surg*. 2001;10:199-203.
21. Siivola SM, Levoska S, Latvala K, et al. Predictive factors for neck and shoulder pain: a longitudinal study in young adults. *Spine*. (Phila Pa 1976) 2004;29:1662-1669.
22. Werner RA, Franzblau A, Gell N, Ulin SS, Armstrong TJ. A longitudinal study of industrial and clerical workers: predictors of upper extremity tendonitis. *J Occup Rehabil*. 2005;15:37-46.
23. Mall NA, Kim HM, Keener JD, et al. Symptomatic progression of asymptomatic rotator cuff tears: a prospective study of clinical and sonographic variables. *J Bone Joint Surg Am*. 2010;92:2623-2633.
24. Carnide F, Veloso A, Gamboa H, Caldeira S, Fragoso I. Interaction of biomechanical and morphological factors on shoulder workload in industrial paint work. *Clin Biomech*. 2006;21 Suppl 1:S33-S38.
25. Escobedo EM, Hunter JC, Hollister MC, Patten RM, Goldstein B. MR imaging of rotator cuff tears in individuals with paraplegia. *Am J Roentgenol*. 1997;168:919-923.
26. Ludewig PM, Borstad JD. Effects of a home exercise programme on shoulder pain and functional status in construction workers. *Occup Environ Med*. 2003;60:841-849.
27. Worland RL, Lee D, Orozco CG, SozaRex F, Keenan J. Correlation of age, acromial morphology, and rotator cuff tear pathology diagnosed by ultrasound in asymptomatic patients. *J South Orthop Assoc*. 2003;12:23-26.
28. Safran O, Schroeder J, Bloom R, Weil Y, Milgrom C. Natural history of nonoperatively treated symptomatic rotator cuff tears in patients 60 years old or younger. *Am J Sports Med*. 2011;39:710-714.
29. Yamaguchi K, Ditsios K, Middleton WD, et al. The demographic and morphological features of rotator cuff disease. A comparison of asymptomatic and symptomatic shoulders. *J Bone Joint Surg Am*. 2006;88:1699-1704.
30. Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB. Abnormal findings on magnetic resonance images of asymptomic shoulders. *J Bone Joint Surg Am*. 1995;77A:10-15.
31. Milgrom C, Schaffler M, Gilbert S, van HM. Rotator-cuff changes in asymptomatic adults. The effect of age, hand dominance and gender. *J Bone Joint Surg Br*. 1995;77:296-298.
32. Palmer KT, Harris EC, Coggon D. Compensating occupationally related tenosynovitis and epicondylitis: a literature review. *Occup Med*. 2007;57:67-74.
33. Padua L, Padua R, Nazarro M, Tonali P. Incidence of bilateral symptoms in carpal tunnel syndrome. *J Hand Surg Br*. 1998;23B:603-606.
34. Werner RA, Gell N, Franzblau A, Armstrong TJ. Prolonged median sensory latency as a predictor of future carpal tunnel syndrome. *Muscle and Nerve*. 2001;24:1462-1467.
35. Stetson DS, Silverstein BA, Keyserling WM, Wolfe RA, Albers JW. Median sensory distal amplitude and latency: comparisons between nonexposed managerial/professional employees and industrial workers. *Am J Ind Med*. 1993;24:175-189.
36. Bagatur AE, Zorer G. The carpal tunnel syndrome is a bilateral disorder. *J Bone Joint Surg Br*. 2001;83B:655-658.
37. Melhorn JM, Wilkinson LK, O'Malley MD. Successful management of musculoskeletal disorders. *J Hum Ecolog Risk Assess*. 2001;7:1801-1810.
38. Melhorn JM, Wilkinson LK, Riggs JD. Management of musculoskeletal pain in the workplace. *J Occup Environ Med*. 2001;43:83-93.
39. Lozano Calderon SA, Anthony S, Ring D. The quality and strength of evidence for etiology: example of carpal tunnel syndrome. *J Hand Surg Am*. 2008;33:525-538.
40. Uejo C. Carpal tunnel syndrome–occupationally related or not? *Guides Newsletter*, May/June 2009;77:1.
41. Melhorn JM, Ackerman WE, Glass LS, Deitz DC. Understanding work-relatedness. In: Melhorn JM, Ackerman WE, eds. *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL, AMA Press; 2008:13-32.