AMA Guides™ Newsletter

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

May/June 2012

In this issue

Upcoming issues

Maximum Medical Improvement

Apportionment Analysis

Risk Management and Independent Medical Evaluations

Evaluating Causation for the Opposite Upper Extremity

Carpal Tunnel Syndrome and Defense Based Act

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.*

Determining Injury-Relatedness, Work-Relatedness, and Claim-Relatedness

Robert J Barth, PhD

The American Medical Association's *Guides to the Evaluation of Disease and Injury Causation (Causation)*[1] is an important component of the AMA *Guides* library. This text delineates a type of evaluation that is distinctly different from a diagnostic evaluation, a treatment planning evaluation, a prognosis evaluation, or an impairment evaluation. It provides a protocol for determining whether a clinical presentation, in the context of a legal or administrative claim, may be credibly attributed to a claimed cause. In other words, it provides an answer for questions such as:

- How can an evaluator credibly determine if a claimed work-related clinical presentation is really work-related?
- How can an evaluator credibly determine if a claimed injury-related clinical presentation is really caused by the litigated events?

This article:

- presents the evaluation protocol from *Causation*
- provides evaluators with self-assessment questions that can be used to scrutinize whether one's own work complies with that protocol
- highlights that protocol's value as a model for scientifically credible practice in general
- clarifies that the protocol is of relevance to all types of claims that involve forensic causation issues.

The protocol from *Causation* is only needed when referral questions specifically involve a relevant focus on causation. "Causation" in this sense is a forensic issue, rather than being a health care issue. Such causation analysis relating to an external exposure or event is often unnecessary in health care, eg, diagnosing and treating a patient. It is also unnecessary in many forensic circumstances (eg, if the referral issues are limited to diagnosis, treatment planning, prognosis, and/or impairment, then there is no need for the *Causation* protocol to be utilized).

Although impairment evaluation is also a forensic issue, a causation evaluation is distinctly different from an impairment evaluation. Referral issues involving impairment evaluation may be independent from causation (eg, some workers' compensation systems require impairment evaluation for issues that may not be work-related, such as carpal tunnel syndrome, low back pain, complex regional pain syndrome type 1, mental illness, chronic pain, etc).An impairment evaluation can be conducted without a causation analysis, and vice versa.

AMA
AMERICAN MEDICAL ASSOCIATION

As is the case for any forensic work, a causation analysis should be conducted in an independent context.[2] Treating clinicians face considerable financial and social conflicts of interest if they attempt to engage in any forensic activity (such as causation analysis) in regard to their patients. In contrast, independent evaluations minimize the evaluator's conflicts of interest. Therefore, treating clinicians should refrain from engaging in any forensic work that involves their patients (including refraining from causation discussions).

Emphasis on Fact, Rather Than Opinions

Court and administrative systems have an extremely unfortunate emphasis on opinions from experts, rather than on facts. All too often, clinicians who are asked to interact with such systems fall prey to that emphasis, and offer opinions instead of encouraging the court or administrative decision-makers to focus on facts.

The protocol from *Causation* is perhaps one of the best examples of how clinicians can focus on facts and avoid surrendering to the court or administrative system's emphasis on opinions. The protocol provides a mechanism for addressing causation issues in a manner that is standardized, objective, fact-based, and scientifically credible. The protocol can be used in a fashion that is free from opinions. By utilizing this protocol, evaluators can demonstrate that they are men and women of science, rather than sellers of opinions.

One clear example of the importance of this distinction is the popular conception of fact and opinion as opposites of each other.[3] Consistent with this status as the opposite of opinion, fact has also been simply defined as "a thing that has actually happened or that is really true."[4] Science is a systematic and credible method of searching for, testing, and potentially verifying facts (or, alternatively, testing hypotheses so that those that are not factual can be revealed as such). Consequently, from a scientific perspective, *fact* has been defined in terms such as: "concrete observations based on evidence"[5]; truths demonstrated through the principles of science and math.[5]

Science (specifically including health science) values fact. In contrast, and quite astoundingly, court systems and administrative systems place an emphasis on expert opinion, rather than on fact. This emphasis on opinion rather than fact is apparently a consequence of the history of court and administrative systems giving the job of decision maker to people who were thought to be unlikely to understand scientific facts (eg, people such as juries and judges). Because those people presumably cannot understand scientific facts on their own, they are thought to need opinions from experts, rather than needing a presentation of scientific facts.

The following examples are remarkable not only for the manner in which they illustrate the extreme nature of the court/administrative anti-fact bias, but also because they are evident within the tradition of the federal Daubert standards, which is probably the legal tradition that actually provides the greatest hope of scientific credibility in the courts. In other words, even at the highest level of scientific credibility within the courts, an anti-fact bias is still demonstrated.

- There is a high-level legal devaluation of scientific investigations that is designed and initiated specifically because of the case at hand.[6] It would be in a court's best interest for scientific investigations to be designed and conducted for purposes of specifically addressing the issues in the case at hand. But high-level federal court rulings, and other federal efforts, have inexplicably devalued such focused scientific investigations in favor of scientific investigations that were prompted by circumstances other than the case at hand (investigations that will almost certainly be less relevant to the case at hand, because they were not designed or undertaken with a focus on the issues that need to be addressed within that specific case).

- There is a court and administrative emphasis on the individual expertise of the person who is offering information, instead of on the credibility of the information itself. In science, it does not matter who first introduces information. If the information is independently verifiable as credible, then the expertise of the individual who first introduces the information is irrelevant. But instead of recognizing the potential irrelevance of individual expertise, federal standards actually place an emphasis on the expertise of the individual who presents the information.[6]

The *AMA Guides* library has made many valuable contributions toward the goal of encouraging doctors to avoid cooperating with the anti-fact bias of court and administrative systems. This is inherent in the "evidence-based" focus of the *AMA Guides to the Evaluation of Permanent Impairment*, Sixth Edition.[7] One especially illustrative example is the discussion of mild traumatic brain injury (MTBI) in the *Guides, Sixth*. Specifically, while court and administrative systems regularly entertain and endorse claims of permanent impairment caused by such injuries, the *Guides, Sixth* simply explains "in contrast to previously held belief, the symptoms of mild traumatic brain injury generally resolves in days to weeks, and leave the patient with no impairment" (6th ed, 330).

Of all the *AMA Guides* library publications, *Causation* makes the strongest contribution toward combating the anti-fact bias of court and administrative systems. For example, the book provides a protocol that allows evaluators to make causation determinations in a manner that is fact-based, rather than opinion-based. Additionally, *Causation* provides several examples (similar to the *Guides, Sixth* example of MTBI) of issues for which scientific findings or principles have provided relatively clear facts that can be used to efficiently and credibly resolve almost every relevant claim (eg, mental illness cannot be credibly attributed to adult life events, complex regional pain syndrome type 1 is an inherently non-injury-related concept, the dominant risk factors for chronic benign pain are of a social or psychological nature, etc). Further, *Causation* uniquely provides explanatory discussions of the distinction between science's emphasis on fact versus the court and administrative systems' anti-fact bias.

For example, page 15 of *Causation* provides a historical perspective that helps to explain the emergence of that distinction. That passage explains that the courts have a historical focus on the way that legal authorities believe reality *should* be. In contrast, science is focused on developing an understanding of the *actual* nature of reality (even if reality fails to comply with the expectations of legal authorities). In the example of MTBI, this aspect of the distinction is exemplified by court and administrative systems' apparent premise that an individual claim of permanent impairment from such an injury *should* be considered as potentially feasible, as compared with the scientific knowledge base indicating that there *actually* is not a credible foundation for any such claim. Another example is the court and administrative systems' apparent premise that the historical reports from a claimant or plaintiff *should* be considered in the process of resolving the claim, versus the fact that scientific findings have repeatedly revealed that such reports are actually unreliable.[8]

Page 17 of *Causation* further highlights the difference between science and court and administrative systems. Specifically, while science places an almost exclusive emphasis on facts, court and administrative systems can (and do) opt for convenience over fact, truth, or justice. Examples provided in the text include:

- A legal standard has arisen in many systems that calls for hypertension to automatically be considered work-related when it is diagnosed for a police officer or firefighter, because of an assumption that the hypertension is caused by job stress. This legal standard has been widely adopted, in spite of scientific findings that indicate that job stress might actually help to prevent the development of hypertension.[9] The administrative systems have apparently decided that it is more convenient to automatically grant benefits than to consider facts and make determinations that are credible and fair.
- Many court and administrative systems have developed a presumption that an opinion from a treating clinician has greater credence than any facts that might be presented by any other expert. This presumption is severely misdirected, in that treating clinicians are plagued by the most extensive and extreme financial and social conflicts of interest (eg, independent clinicians are free from some of the conflicts of interest that plague treating clinicians).[2] The administrative systems have apparently decided that it is more convenient to allow a severely conflicted treating clinician to offer opinions than it would be to arrange for an independent consultant to apply factual information to the claim.

Further discussion of the distinctions between science and the court and administrative systems is provided on page 115 in *Causation;* that passage explains all of the following:

- Science seeks understanding through credible means, such as population-based prospective studies of large groups. Page 18 in *Causation* elaborates further on this point, by explaining: "Most epidemiologists are comfortable only when studies of causation are large, carefully controlled for all possible risk factors, and, ideally, repeated with similar findings." Such credible research facilitates the analysis of any individual case, because it allows for the generation of reliable scientific findings that can be applied to that individual case (eg, almost 40,000 available scientific citations have failed to reveal a relationship between MTBI and permanent impairment; therefore, there is no credible justification for claiming that the MTBI in the case at hand has caused permanent impairment).[10] In contrast, court and administrative systems act as if the anecdotal details of an individual case are a more important source of understanding that large-scale and replicated independent studies would be. Specifically, the idiosyncratic details of the individual case at hand (eg, plaintiff-reported history of plaintiff-reported complaints) will almost always be considered in every court or administrative process, but the court system lacks any such automatic consideration of relevant scientific findings. Relevant scientific findings will only be considered if the involved attorneys and experts do their jobs in an honest and credible fashion by bringing such scientific findings to the attention of the administrative decision makers and if administrators (eg, judges) allow for the presentation of such scientific findings. Even when attorneys and experts actually bring relevant science into the discussion, there is no guarantee that the decision makers will use that information. Page 17 in *Causation* explains that court and administrative systems are not under any obligation to make scientifically credible decisions.

- While science dismisses findings that are not reproducible across different observers, court and administrative systems act as if a claim can be credible in spite of unreliability across different observers. This is exemplified by the manner in which court and administrative systems appear to think it is normal for scientific testimony to NOT be reproducible from one expert to another, and to allow such non-reliable expert testimony to be used in attempts to support a legal claim. In the example of MTBI claims, courts regularly allow an individual expert witness to testify (or imply) that his or her idiosyncratic, unreliable, and un-reproduced observations indicate that the scientific knowledge base (that factually indicates against permanent impairment) is somehow not relevant to the specific case at hand (and such experts are often not required to present any scientific support for that aspect, or any other aspect, of their testimony). Such anti-science testimony is entrenched in court and administrative systems, due to those systems encouraging clinicians to base testimony on their training and experience, which is always an idiosyncratic and nonreproducible set of circumstances, rather than on the scientific knowledge base.
- This section reiterates the court and administrative systems' emphasis on expert opinion (which, as was discussed above, has actually been defined as the opposite of fact). For the example of MTBI, the scientific facts include a lack of credible scientific support for claims of permanent impairment and repeatedly established indications that eligibility for compensation/litigation is actually the best predictor of persistent complaints (while the injury itself is not predictive of persistent complaints).[7, 10] Court and administrative systems regularly fail to emphasize such facts (which are readily available through means that are not dependent on expert testimony) in favor of expert opinions (including regularly allowing for the introduction of expert opinion that is dramatically contradicted by scientific facts).

Page 436 in *Causation* adds a discussion of the additional anti-fact tendencies that are inherent in many workers' compensation systems, such as:

- Many systems ask clinicians to make causation determinations at the beginning of the claim. This elevates the risk of cases being incorrectly classified as work-related. This risk stems from the tendency for an early classification of work-relatedness to be applied long term, even when the course of the clinical presentation takes a non-work-related turn (eg, an inherently non-work-related presentation of complex regional pain syndrome type 1 is covered under workers' compensation because a work-related fracture preceded it). The text explains that it can be almost impossible for workers' compensation systems to prevent such unjustifiable generalizations.
- Many states mandate "lifetime medical treatment." This set of circumstances is based on an inherent, but unjustifiable, assumption that "the original work injury is responsible for all subsequent disease or illness."
- Many systems demand "yes" or "no" responses from doctors for questions of work-relatedness. The text from *Causation* advises doctors to resist such pressure toward unjustifiably dichotomous thinking.
- The text advises that if the above examples of misdirection had not been adopted by the involved systems, determina*tions of work-relatedness* would be more accurate and more just, claimants would receive better health care, health outcomes would be better, and the societal effects would be less harmful. The text further advises: "The cost to society of misclassification of conditions as occupational greatly exceeds that of establishing a formalized investigative period before appropriate determination."

The above discussion can be summarized, for the sake of simplicity and emphasis, in the following manner. The court and administrative systems have placed an unfortunate and misdirected emphasis on opinions from doctors (and on convenience) rather than on fact. Far too often, clinicians who were educated in a tradition of science abandon that tradition and fall prey to the opinion-focused misdirection from the court system. Such clinicians regularly offer opinion-based testimony, based on nonscientific foundations, such as their own idiosyncratic experience and training, rather than focusing on scientific facts.

Causation is especially notable for the powerful manner in which it directs doctors away from the anti-fact bias that is inherent in court and administrative systems. In addition to providing the above explanatory descriptions of the differences between court and administrative systems and science, *Causation* provides a protocol that is standardized, fact-based, and rooted in science. Through this protocol, the AMA has given clinicians a tool that can be used to effectively and emphatically focus on fact, rather than on opinion. By using this protocol, evaluators can demonstrate their allegiance to, and adherence to, the scientific tradition of professional health care and can demonstrate and justify their resistance to the anti-fact bias of court and administrative systems. Details of that protocol are provided in the final section of this article.

Editorial Note: Unrelated to the focus on the court system of causation analysis, the common workers' compensation scenario should be mentioned. An employee has a symptom that he/she thinks might have been caused by work (no significant injury incident). The employee tells the employer about the symptom and the possibility of work relatedness. The employer does not know the diagnosis and thus schedules an appointment for the employee with a doctor, expecting the doctor to determine both the diagnosis and the potential work-relatedness of the condition. The doctor sees the employee, and because the appointment was scheduled by the employer, the doctor assumes the employer and the workers' compensation insurer have already accepted the claim as work-related. Thus, the doctor in medical records states the diagnosis and that it is work-related. Later, the employer and/or insurer write the doctor with facts asking for a work-relatedness determination. Because doctors rarely change opinions once they are in print, the doctor continues to state the condition is work-related.

Avoiding Common Trends Toward Reliance on Unreliable Information and Logical Fallacies

All too often, perhaps even in most cases, a health science expert offers an opinion of the following nature regarding issues of work-relatedness or injury-relatedness: "My opinion is that this clinical presentation is injury-related, and that opinion is based on the patient telling me that he/she did not have this problem prior to the litigated accident." There are at least two problems with this common set of circumstances, and both are addressed in *Causation*.

First, page 324 in *Causation* explains, in a brief fashion, that this common basis for causation claims is not credible because such reports from examinees are not reliable. Relevant scientific findings have been more extensively reviewed in a more recent AMA *Guides* library publication.[8] Such findings include: when an examinee blames someone else for his or her injury or accident, and claims that he or she never experienced relevant health problems before the injury or accident; such denials of pre-existing health problems are almost always found to be false (when a relatively comprehensive set of pre-existing records are available for the purpose of scrutinizing the denial of pre-existing problems). Given the extreme unreliability of such denials of relevant pre-existing problems, the common reliance on reports from claimants or plaintiffs for purposes of making causation determinations is not justifiable.

Even if such denials of relevant pre-existing problems were reliable, they still could not serve as a credible basis for causation determinations. This additional issue is addressed on page 16 in *Causation*. That discussion actually identifies such reasoning as being a logical fallacy ("false reasoning"), rather than being a credible basis for causation determinations. The text explains: "It is a fallacy to conclude that one event followed by a second necessarily demonstrates a causal relationship between the events. A collision that occurs minutes after a black cat crosses a person's path does not establish a causal relationship between the encounter and the collision because the cat did not cause the accident." Given this identification of the relevant thought process as "false reasoning," the common reliance on claimed pre-versus-post differences for purposes of making causation determinations is not justifiable.

Because of the above considerations, such claims of pre-versus-post differences are not considered within *Causation's* protocol.

Relevance to All Types of Medical-Legal Claims

The text in *Causation* explains that its protocol is relevant to tort cases, as well as workers' compensation claims (for example, see page 15). Given this relevance to multiple types of claims, and given the strong focus on scientific credibility, the author of this article has often informed various stakeholders within court and administrative systems that *Causation* is perhaps the most important text for medical-legal claims of all kinds.

Some such stakeholders, from systems other than workers' compensation, have responded with disappointment after reading *Causation*. They have explained that their disappointment was caused by the text's frequent use of words such as "work-related" and "occupational." The use of such words has apparently precipitated malicious attempts, from some unscrupulous attorneys and doctors, to mislead decision makers into thinking that the protocol from *Causation* is not relevant to claims that are not in the workers' compensation system.

Such false allegations are a major reason for the creation of the current article. The detailed description, provided below, of *Causation's* protocol has been crafted in a manner that highlights and emphasizes that the protocol is relevant to any medical-legal claim that involves forensic causation issues (eg, injury-relatedness in tort claims).

The Protocol

Causation specifies that the protocol is based on previous work conducted by other groups. For example, the original work toward the creation of this protocol is credited to the National Institute of Occupational Safety and Health, and prior adaptation of the protocol is credited to the American College of Occupational and Environmental Medicine (see pages 40 and 43 in *Causation* for more detailed referencing).

The text in *Causation* points out that this protocol, in its various forms, may be the most common structured method used. The text also points out that the protocol does not seem to have major weaknesses.

Causation's protocol is presented repeatedly in the text of the book. The following represents an attempt to incorporate all those passages into a single discussion.

The protocol involves a six-step process, as described below. Page 40 in *Causation* explains that the six steps must be performed in sequence, and the failure to complete any individual step halts the entire process. Any such set of circumstances (failure to complete any step in a manner that credibly supports a causative relationship) eliminates credibility for claims of injury-relatedness, work-relatedness, etc.

Step 1: Definitively establishing a diagnosis

This step is simply a diagnostic evaluation. An unusually high level of rigor and scrutiny might be warranted, due to the elevated jeopardy that an evaluator faces in a forensic process, but this step is entirely consistent with the best diagnostic evaluations that take place outside of forensic contexts.

This step involves:

- For the aspects of the examinee's clinical presentation (complaints, symptoms, signs, etc) that are the focus of the medical-legal claim, the examiner applies health science expertise in an attempt to identify any objective and credible evidence of a diagnosis that might explain the presentation.
- If such objective and scientifically credible evidence is discovered, then a diagnosis is established in a credible and definitive fashion.

If an evaluator intends to make a diagnostic claim, then the following self-assessment questions can be used in order to scrutinize that claim:

- What diagnostic method or protocol have I used?
- Have I clearly documented my utilization of that method in this case, and the results of that utilization?
- If asked, can I reference health care literature that verifies that my method is actually an example of professional clinical work (eg, in some way consistent with professional standards or with scientific credibility)?
- Has my method been scientifically validated?
- If asked, can I reference scientific validation of my diagnostic method?
- Do I know the error rate that has been scientifically established for this method? If asked (for example in a Federal Court Daubert Challenge as an expert witness), can I reference the scientific findings regarding that error rate?
- Is my diagnosis based on objective and scientifically credible evidence, instead of being based on the examinee's subjective complaints?
  - Did I find evidence of the diagnosis that is completely independent of what the examinee told me (in contrast to allowing the examinee to self-diagnose, eg, "the accident caused me to have migraine headaches," "the accident injured my back," etc)?
  - Would my findings justify this diagnosis, even if the patient did not have the ability to communicate his or her complaints? For example, if I am diagnosing an injury, is that diagnosis based on something more objective and more credible than a complaint of pain?
  - If my diagnosis is completely based on subjective complaints, have I documented the nature of that set of circumstances and the associated vulnerability of the diagnosis to manipulation by the examinee?
- Have I engaged in, and documented, a comprehensive and credible differential diagnostic process?
  - Have I conclusively ruled out all other potential explanations for this clinical presentation, especially those that are more likely than my diagnosis? For example, for a complaint of back or neck pain, have I ruled out the social and psychological risk factors that have been scientifically established as being more important than general medical factors (for example, see Linton[11])?
  - Have I clearly documented that differential diagnostic process?
  - Do I even know where my diagnosis lies on the continuum from statistically most likely explanations for this type of presentation to least likely?
- Is the history, as documented in a relatively extensive set of records that I was able to obtain and review, especially including preclaim records, adequately supportive of my diagnosis?
  - Did I ask for a full set of records, especially preclaim records?
  - Was I provided with such a set of records?
  - Was I authorized to dedicate time to reviewing those records?

- Does the diagnosis really explain the clinical presentation?
  - For example, if the presentation involves back pain, most types of spine abnormalities would be a poor diagnostic choice, given the lack of association between most types of spine abnormalities and back pain (see reference Carragee[12] as an example of scientific findings that demonstrate the lack of association between spine abnormalities and back pain).
  - Has the claimed course of the clinical presentation been consistent with the scientifically established course of the diagnosis? For example, a diagnosis of mild traumatic brain injury is inconsistent with a clinical presentation that involves a claimed course of persistent impairment or worsening impairment,[7, 10] even if the occurrence of a mild traumatic brain injury has been confirmed, and even if the early symptoms are consistent with the short-lived effects of such an injury.

Step 2: Applying relevant findings from epidemiologic science to the individual case

This step involves applying scientific findings, which are of relevance to causation, to the case at hand. The obvious place to look for relevant scientific findings is in scientific publications, although it is feasible for unpublished scientific findings to be of value. (Although, as was reported above, research that was prompted by the case at hand is inexplicably devalued by court and administrative systems, and such research would probably not be published during the course of the case that prompted it. The same court standards devalue all other unpublished research, without regard to the inherent credibility of that research. Evaluators do not have to adopt or endorse such examples of court and administrative anti-fact bias, but it is helpful to be aware of such examples and of the bias in general.)

The key issue for this step is: In order for a causative relationship between a definitively established diagnosis and a suspected cause to be claimed in a credible fashion, the claim must be based on credible and reliable scientific findings that have convincingly established that such a specific causative link actually exists. In other words, such claims are only credible if they are grounded in credible and reliable scientific findings that indicate that the specific claimed cause is a significant risk factor for the specific claimed clinical presentation. Additionally, any such claim must also account for any contradictory scientific findings, by involving clear justification for concluding that the contradictory findings are not relevant to the case being evaluated.

For an evaluator, preliminary efforts in this regard can include reading scientific reviews of the etiology of the definitively established diagnosis. Such reviews can provide direction (at least preliminary direction) regarding at least two primary considerations:

- Has the claimed cause (in this individual case) been scientifically identified as a significant risk factor for the diagnosis that was definitively established in step 1?
- What are the most well-established risk factors? Scientifically established findings in this regard can then be applied to the case at hand, in order to determine which scientifically established risk factors are relevant for this particular case (whether the scientifically established risk factors include the claimed cause or not).

This process can go beyond reviews, by involving literature searches of the nature that is described in Chapters 4 and 9 in *Causation*.

If scientific findings that appear to support the causative relationship that is being claimed in the case at hand are discovered, then those scientific findings must be scrutinized in an attempt to determine if they are sufficiently credible and reliable. Many passages in *Causation* provide direction on how to determine whether scientific findings that seem to establish causative relationships are credible and reliable, but pages 34-39 focus on that issue most intensively.

If an evaluator is considering endorsing a claimed causative relationship, then the following self-assessment questions can be utilized for this step:

- Am I aware of scientific findings that provide credible and reliable support for the premise that this specific claimed cause is capable of producing this specifically claimed clinical presentation?
- If asked, can I reference and explain the relevant scientific findings?

Step 3: Obtain and Assess the Evidence of Exposure

If credible and reliable scientific support for a claimed causative relationship is actually found in step 2, then the causation analysis continues with consideration of whether the case at hand involved sufficient exposure to the claimed cause.

The essential questions that need to be addressed in this step are:

- Magnitude of exposure: what evidence, predominantly objective, is available that clearly verifies that the exposure to the claimed cause was of sufficient magnitude to account for the development of the claimed clinical presentation?
- Relationship in time between the exposure and the clinical presentation: Did the clinical presentation develop and evolve in a manner that has been scientifically established as being consistent with the claimed or documented temporal exposure to the claimed cause?

Causation explains that actual measurements of exposure are the most reliable information for this step, while the examinee's report of exposure is among the least reliable information.

An example of the need for this step is provided by water poisoning. Drinking water is a risk factor for seizures, coma, respiratory arrest, brainstem herniation, and death.[13] However, the relationship between drinking water and these consequences is only manifested when the exposure to drinking water is extremely high and when the health problems develop shortly after the extreme water drinking.

This example illustrates that it is not credible to conclude that a scientifically established risk factor caused the definitively established diagnosis in the case at hand, simply because the risk factor is relevant to the case (eg, it is not credible to conclude that drinking water caused a brainstem herniation, when the only reasons for that conclusion is that there has been verification that the patient drank water and it has been scientifically established that drinking water is a risk factor for brain stem herniation). Before such a conclusion can be credible, evidence has to be collected that verifies that the exposure to the risk factor was of a sufficient magnitude to cause the clinical presentation (eg, evidence verifies that water was consumed in sufficient quantities, within a sufficiently limited time period, to create the level of exposure to drinking water that has been scientifically established as a risk factor for brainstem herniation). Additionally, in order for such a conclusion to be credible, the clinical presentation must develop and evolve in a manner that is temporally consistent with the scientific findings regarding the timing of the relationship (eg, a herniation due to water drinking occurs within a few hours of the extreme water drinking[14] rather than days later).

In general terms, the history of the clinical presentation should correlate in time with the exposure to the risk factor. Exposure to more extreme levels of the risk factor should be associated with more severe clinical reactions (an allergic reaction to rubber is more severe when the level of exposure to rubber is more extreme). A closer relationship in time should be associated with more severe clinical reactions (symptoms are more intense in the short-term aftermath of an injury and less intense as the person moves away from the injury in time).

If an evaluator is considering endorsing a claimed causative relationship, then the following self-assessment questions can be utilized for this step:

- Have I collected evidence of the details of the claimed exposure?
- Is my method for collecting such evidence credible? If asked, can I reference and explain scientific validation of my method?
- Have I documented my utilization of a method for collecting evidence of exposure in this case and the results of that utilization?
- Was the level of exposure sufficient to cause the claimed clinical condition? Have I explained this in my documentation? If asked, can I reference and explain scientific findings that verify that this level of exposure is sufficient to cause this presentation?
- Is the timing of the claimed exposure and claimed clinical presentation consistent with relevant scientific findings? Have I explained this in my documentation? If asked, can I reference and explain scientific findings that verify that the timing details from this case are consistent with the claimed causative relationship?

Step 4: Consider other relevant factors

This step can be conceived of as an expansion and double-check of step #2. The purpose is to consider the relevance of scientific findings for causation of the definitively established diagnosis, with an emphasis on risk factors other than the cause that is being claimed in the case at hand. In other words, this step is dedicated to determining if other risk factors provide a better explanation for the clinical presentation than the explanation that is provided by the claimed cause.

Causation specifies that questions that need to be addressed in this step include:

- Are there risk factors, other than the cause that is being claimed in this specific case, that could contribute to the development of the claimed clinical presentation?
- Are any such risk factors relevant to this case?

In order for a causation conclusion to be credible, the process of creating that conclusion must have included comprehensive consideration of the epidemiological scientific findings for the definitively established diagnosis, determination of risk factors for the diagnosis (based on scientific findings), determination of which of the risk factors apply to the case at hand (which ones are relevant), and determination of which relevant risk factors are of greatest significance for the diagnosis in general and for this case in particular.

Causation offers this example: if the claim involves carpal tunnel syndrome being attributed to work, have the non-work-related factors that have actually been scientifically established as risk factors for that diagnosis been considered in this case (risk factors such as pregnancy, obesity, diabetes, etc)? This example can be considered further by a review of Chapter 9 in *Causation*, which provides a discussion of the manner in which non-work-related factors are the dominant risk factors for carpal tunnel syndrome (and a discussion of the insignificance of most work-related factors).

The direction for this step that is provided in *Causation* provides a basis for the following self-assessment questions. These questions can be used, for evaluation of one's work, by any examiner who is considering the endorsement of a causation claim:

- Have I thoroughly reviewed scientific findings in regard to risk factors for this diagnosis? Can I reference and explain relevant scientific findings if asked to do so?
- Have I evaluated this specific case for the relevance, or lack thereof, of every scientifically established risk factor for the diagnosis, and especially for all risk factors that are not being claimed as a cause in this individual case? Have I documented my work in this regard?
- Have I considered scientifically identified covariates and confounders (eg, issues that might have an indirect effect, an interaction effect, that might create a false impression of a causative effect, etc)? Have I documented my work in this regard?
- Have I had an opportunity to review a significant portion of preclaim records, so that I can consider issues such as preclaim injuries and other preclaim health issues? Have I documented my work in this regard?
- Have I taken steps to ensure that my considerations have *not* been artificially limited to risk factors that are within the typical boundaries of my specialty? For example, has an orthopaedic surgeon gone beyond the typical limits of orthopaedics to consider the dominant role of psychological and social issues in the genesis of chronic pain complaints?[11]
- Have I considered the effects of treatments? For example, in cases of chronic pain, have I considered the effects of prescription narcotics, which scientific findings have indicated as a reliable cause of worsening of chronic pain?[15]
- For all of the relevant risk factors in the case at hand, have I provided an objective and credible basis for determining which are of primary importance in the creation of the claimed clinical presentation? This question is especially important when the claimed cause is credibly established as a significant factor and there are other significant factors as well.

Step 5: Scrutinizing the Validity of the Evidence

This step calls for the evaluator to intensely scrutinize the evidence from the individual case. *Causation* explains that the primary goal of this step is to determine if there are any data that contradict or confound the information that has otherwise been presented in the case.

Chapter 3 in *Causation* states that this step involves two main issues:

- Details of the claim: Is there conflicting information regarding date of injury or timing of exposure, mechanism of injury or exposure, prior injuries or prior health problems, the examinee's activity level, the examinee's ability to work, etc?
- Adequacy of professional services: Have other clinicians offered opinions that lack scientific credibility? Have clinical services been relied upon that actually lack scientific credibility or that lack relevance to the specifics of this case?

Causation advises evaluators to "qualify all statements appropriately by noting a lack of knowledge and/or certainty" when the scrutiny that is called for in this step is not feasible (eg, the evaluator does not have an opportunity to compare and contrast different sources of information regarding the claimed exposure).

Relevant self-assessment questions can include:

- Have I carefully scrutinized the details of this case, such as scrutinizing the claimed details of exposure and injury?
- Have I carefully scrutinized the examinee's reported history? In this regard, evaluators should note the scientific findings, which indicated an approximate rate of 100% of examinee-reported histories being false when the examinee was blaming someone else for his or her health complaints.[8]
- Do I have enough information to engage in such scrutiny in a meaningful way?
- If I am relying on any previous clinical conclusions, have I studied the documentation of those clinicians in an effort to make sure that their work was scientifically credible?
- Have I documented discoveries of conflicting information and noncredible clinical work?
- Have I documented the uncertainty that is associated with any aspects of the evidence that cannot be intensely scrutinized?

Step 6: Evaluation of the Results from All of the Above Steps, and Generation of Conclusions

This step involves a reexamination of the findings from all of the previous steps and determining whether they coherently justify the claimed causative relationship.

The essential self-assessment questions for this step are:

- Have I completed the first five steps in a credible fashion?
- Do the findings from those steps provide credible and convincing support for the claimed causative relationship?

Causation is an essential reference for all stakeholders involved with medical-legal claims of any kind. It provides hope that the resolution of such claims can be based on science and other sources of facts, rather than on unsubstantiated opinions.

References

1. Melhorn JM, Ackerman WE. *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL: American Medical Association; 2008.
2. Barth RJ, Brigham CR. Who is in the better position to evaluate, the treating physician or an independent evaluator. *Guides Newsletter*. September/October 2005:8-11.
3. Answers Corporation. http://wiki.answers.com/Q/What_is_the_opposite_of_the_word_opinion. Accessed April 27, 2012.
4. Agnes M, Guralnik DB, eds. *Webster's New World College Dictionary*. 4th ed. Foster City, CA: IDG Books Worldwide; 2001.
5. Huppke RW. Facts is dead; long live whatever rules now. *Chicago Tribune*. April 19, 2012.
6. Sinclair DC. Epidemiology in the courtroom: an evidence-based paradigm for the determination of Causation in Compensation Environments. *J Occup Environ Med*. 2010;52(4):456-461.
7. Rondinelli RD, Genovese E, Katz RT, et al, eds. *Guides to the Evaluation of Permanent Impairment*. 6th ed. Chicago, IL: American Medical Association; 2008.
8. Barth RJ. Claimant-reported history is not a credible basis or clinical for administrative decision making. *Guides Newsletter*. September/October 2009: 1-7.
9. Barth RJ, Roth VS. Claims of hypertension caused by job stress. *Occup Environ Med Rep*. 2002;16(9):70-72.
10. Carroll LJ, Cassidy JD, Peloso PM, et al. WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on mild traumatic brain injury. *J Rehabil Med*. 2004;(suppl 43):84-105.
11. Linton SJ. Psychological risk factors for neck and back pain. In: Nachemson AL, Jonsson E. *Neck and Back Pain*. Philadelphia, PA: Lippincott, Williams & Wilkins; 2000.
12. Carragee E, Alamin T, Cheng I, Franklin T, van den Haak E, Hurwitz E. Are first-time episodes of serious LBP associated with new MRI findings? *Spine J*. 2006;6(6):624-635.
13. Ballantyne C. Strange but true: drinking too much water can kill. *Scientific American*. June 21, 2007. http://www.scientificamerican.com/article.cfm?id=strange-but-true-drinking-too-much-water-can-kill.
14. Petzold A, et al. Marathon related death due to brainstem herniation in rehydration-related hyponatraemia: a case report. *J Med Case Rep*. 2007;1:186.
15. Barth RJ. Prescription narcotics: an obstacle to maximum medical improvement. *Guides Newsletter*. March/April 2011: 1-7.

Evaluating Causation for the Opposite Lower Limb

By Christopher R. Brigham, MD, Charles N. Brooks, MD, and James B. Talmage, MD

Causation analysis should always be based on current scientific evidence and the facts of a specific case. However, certain beliefs have evolved that lack scientific basis. One unsupportable myth is that "favoring" one lower extremity will often result in injury or illness of the opposite lower limb.

This is exemplified by the case of a 40-year-old male longshoreman who reportedly sustained an injury to left knee at work on September 1, 2009. His past history is remarkable for prior knee problems bilaterally, including high school football injuries that resulted in "arthroscopy" (details unavailable) and subsequent documented osteoarthritis. He weighs 225 pounds and is 5 feet 6 inches tall. The calculated body mass index of 36.3 meets the criterion for obesity. In December 2009 the longshoreman underwent an arthroscopic partial lateral meniscectomy of left knee. Two years following this left knee surgery he developed increased right knee pain attributed to osteoarthritis. His treating physician opined the right knee pain was a "compensatory consequence of limping and full weight bearing pivoting as a body mechanics change in limping and unloading his left knee following his left knee injury." Review of the longshoreman's medical records fails to demonstrate any notation of limping or an antalgic gait.

This is a common scenario, where an opinion on causation appears based on *post hoc ergo propter hoc* ("after this, therefore caused by this") reasoning, but is unsupported by science. Temporal sequence does not prove causation. An example often cited to point out the false logic in such causal opinions is that even though one follows the other, the rooster crowing doesn't make the sun come up. In causation analysis, one must also consider temporal proximity, or in this case disparity: whether there was an injury or exposure likely to cause the condition in question, and if there is another, more probable, cause for it.

As implied, causation analysis must be based on both scientific evidence (in this case, the medical literature) and facts of the individual case. To conclude that a given cause and effect are etiologically associated with a reasonable degree of medical probability or certainty (ie, more than 50% probability), all 3 of the following criteria must be met:

1. The cause is medically probable: more likely than not, the patient had a trauma or exposure.
2. The effect is medically probable: more likely than not, the patient has the injury or illness.
3. The cause and effect probably are etiologically related: more likely than not, the trauma caused the injury or the exposure the illness.

This is the premise physicians are asked to analyze and to support or refute when assessing causality: that a probable cause and effect likely are etiologically related. If any 1 of the 3 criteria is possible but not probable, causation has not been established. Further, 2 or more possible causes do not equal a probable one. (They are not additive.)

When evaluating causation, the physician must identify possible causes (occupational and nonoccupational) and the correct diagnosis or diagnoses (the effect), and then assess the likelihood of a causal relationship between them. In other words, causation analysis must be based on an analytical approach.[1] 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. Sometimes a causal opinion is based on patient history. However, the premise that history reported by claimants and plaintiffs is accurate has repeatedly failed scientific testing, as detailed in the September/October 2009 issue of the *Guides Newsletter*.[2]

In this case, the longshoreman had three known risk factors for osteoarthritis: age, obesity, and prior injury in high school.[3] He had documented osteoarthritis in both knees before the claimed occupational injury. However, the question remains, was the arthritis in the right knee aggravated (permanently worsened) due to "favoring" the left lower extremity after the knee injury on that side?

Review of the medical literature reveals no generally accepted studies that support such a causal relationship, nor is there any reasonable scientific logic therefor. In fact, the literature available, most notably an editorial entitled "Can favoring one leg damage the other?" by Ian Harrington, MD, and W. Robert Harris, MD,[4] refutes the reported cause and effect relationship. They explain:

> Lay people, and many doctors as well, believe that pain or disability in one leg can stress the other one and produce symptoms in it.
>
> We believe that there is no scientific basis for such reasoning. The mechanics of limping are poorly documented in the orthopaedic literature and we have found few references to the effect of a limp on the other leg. To clarify the position for lay adjudicators and the physicians who advise them we reviewed the mechanics of the two basic limps: paralytic and antalgic. In the former, the muscles of the weak leg are not strong enough to balance body-weight and the patient walks with a characteristic lurching gait. The trunk, head, and arm are displaced towards the affected side, moving the body's centre of gravity directly over the weak leg and thereby reducing the muscle force required to balance the body weight. In the antalgic gait, the patient shortens the stance phase by adopting a similar Trendelenburg lurch.
>
> It may seem logical that manoeuvres designed to lessen the load on one leg must increase that on the other, but there is no evidence to support this.[4]

Harrington and Harris reference gait studies using force plates on patients with longstanding poliomyelitis who had a paralytic and short-leg limp that confirmed the force transmitted in the affected lower extremity was reduced, but the force in the opposite limb was the same as in normal individuals.[5] Similar findings were seen with an antalgic gait resulting from arthritis.[6,7] They also noted studies revealing the magnitude of hip force in normal individuals varies with body weight, stride length, and walking speed.[8] Because someone with lower limb pain typically walks more slowly than an asymptomatic person, shortens his/her stride length, and reports the injury or illness of the originally involved lower limb resulted in a marked decrease in weight bearing activity (steps taken each day), both the forces and number of loading cycles on the unaffected limb are likely to be *less*, not more, than before the original injury or illness.

It is improbable that crutch or cane use would stress the uninvolved lower extremity because there is little change in the rhythm of gait, and the force transmitted thereby is increased only by the weight of the walking aid. In fact, using a cane may reduce the force in the normal lower limb because cane users walk more slowly.

Harrington and Harris note that in the days of poliomyelitis, when limping was common, symptoms in the normal leg were seldom attributed to the limp, and that amputees rarely develop arthritis in the joints of the uninvolved limb, despite the fact that no artificial leg or brace can restore a normal gait.

In 2005 Harrington provided a discussion paper entitled "Symptoms in the Opposite or Uninjured Leg" prepared for the Workplace Safety and Insurance Appeals Tribunal in the Province of Ontario[9] that concluded:

> There is no clear evidence to suggest that an injury to one lower extremity would have any significant impact on the opposite uninjured limb unless the injury resulted in major muscle or nerve damage causing partial or complete paralysis of the damaged leg, and/or shortening of the injured lower extremity resulting in a limb length discrepancy of more than four or five centimetres so that the individual's gait pattern has been

AMERICAN MEDICAL ASSOCIATION

515 N. State St.
Chicago. IL 60654

The AMA *Guides Newsletter* is published 6 times a year by the American Medical Association (AMA). Subscription rates are $150 for AMA members, $200 for nonmembers. To order by telephone, call 800 621-8335. Fax orders should be sent to 312 464-5600. Mail orders should be sent to Order Department, American Medical Association, PO Box 930876, Atlanta, GA 31193-0876. Specify product number NG034096 in ordering.

Evaluating Causation (continued)

altered to the extent that clinically there is an obvious lurching type gait (a significant limp). In order for this type of gait to have impact on the opposite or uninjured leg, it is likely that the abnormal gait or limp would need to be present over an extended period–years. A temporary abnormality in gait, eg, a limp over a relatively short period of weeks or months is unlikely to have any effect on the opposite leg. The use of a cast, cane, and crutches is also unlikely to have any major impact on the stress borne by the uninjured limb. Increased body weight (obesity) does, however, have a detrimental effect on both lower extremities and magnifies all of the previously described risk factors.[9]

In assessing causation it is imperative to base conclusions on scientific evidence and facts of the case at hand rather than relying solely on patient history or false logic such as *post hoc ergo propter hoc* reasoning.

References

1. Melhorn JM, Ackerman WE. *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL: AMA Press; 2008.
2. Barth RJ. Examinee-reported history is not a credible basis for clinical or administrative decision making. *Guides Newsletter*. September/October 2009. 1-7.
3. Subin KP, Brigham CR. Lower limb. In: *Guides to the Evaluation of Disease and Injury Causation*. Chicago, IL: AMA Press; 2008: chap 10.
4. Harrington IJ, Harris WR. Can 'favouring' one leg damage the other? *J Bone Joint Surg Br*. 1994;76-B:519-20. http://www.bjj.boneandjoint.org.uk/content/76-B/4/519.full.pdf.
5. Harrington IJ. A bioengineering analysis of force actions at the knee in normal and pathological gain. *Biomed Eng*. 1976;11:167-172.
6. Harrington IJ. Static and dynamic loading patterns in knee joints with deformities. *J Bone Joint Surg Am*. 1983;65-A:247-259.
7. Harrington IJ. Knee joint forces in normal and pathological gait. In: Niwa S, Perren SM, Hattori T, eds. *Biomechanics in Orthopedics*. Tokyo: Springer-Verlag. 1992:121-146.
8. Paul JP. The effect of walking speed on the force actions transmitted at the hip and knee joints. *Proc R Soc Med*. 1970; 63:200-202.
9. Harrington IJ. Symptoms in the Opposite or Uninjured Leg. Discussion paper prepared for the Workplace Safety and Insurance Appeals Tribunal, Province of Ontario, August 2005. http://www.ontla.on.ca/library/repository/mon/23004/291538.pdf. Accessed June 11, 2012.