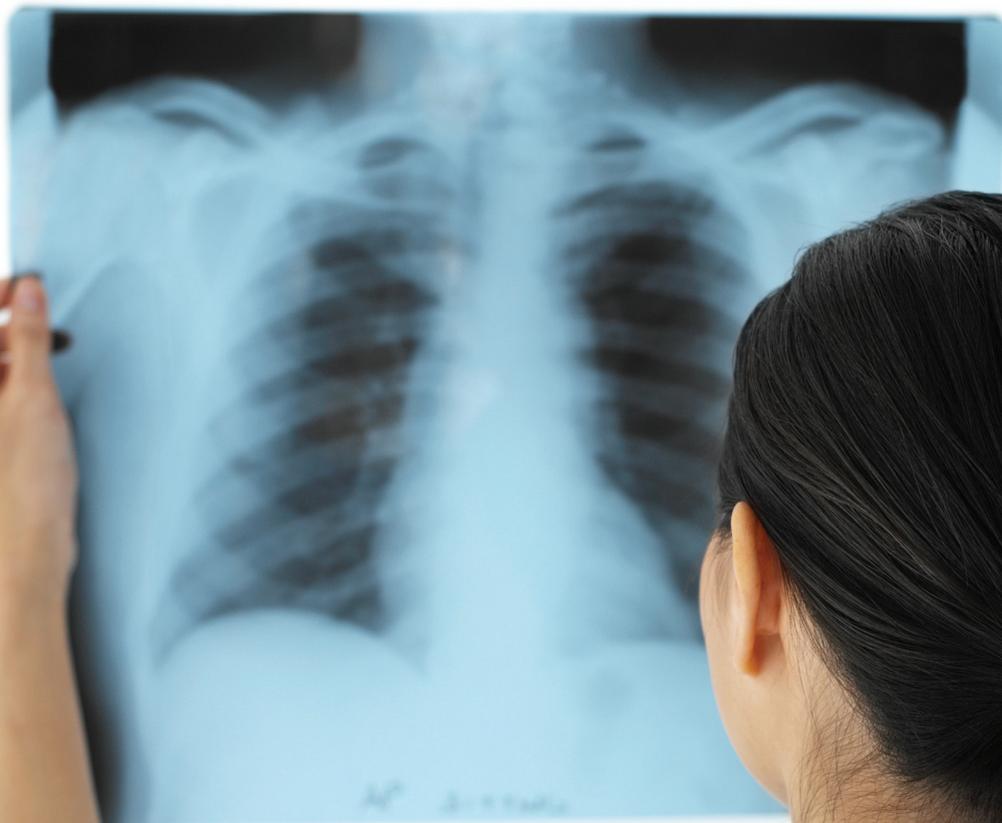


From Symptoms to Liability

The Distinct Roles of Differential Diagnosis and Causation Assessment



by Ronald E. Gots

The patient has tingling in his leg and some weakness. Was your client's aerial spraying of a pesticide responsible? Answering this question requires following an investigative path from symptom to culpability and potential liability, and such seemingly routine questions are an integral part of personal injury trial practice. Eventually, they seem so routine that the medical and scientific complexities of this analytical exercise are overlooked or taken for granted. Yet, the analysis of such matters is not trivial, nor is it a one-step process. It involves two types of expertise and, often, two different experts.

Some attorneys or their consultants in toxic tort or pharmaceutical lawsuits have noticed that physicians are testifying to matters that they know little about, using poorly formulated analytical methods that have no proper place in a courtroom. They do so claiming that they have employed the standard, recognized methodology of clinical medicine—differential diagnosis.

Differential diagnostic methodology is widely assumed, and now supported by some legal precedent, to arrive at causal conclusions in personal injury claims. *It does not.* And until counsel understands why and how to explain this to trial judges, they will be missing important opportunities for pretrial management of their cases and for best serving their clients.

Attorneys who believe that differential diagnosis properly leads to a determination of cause are not alone. Many other lawyers and judges do as well, and decisions by widely dispersed state and federal courts have given credence and legal authority to this erroneous notion. Experienced experts have used and contributed to this misperception, averring that their “differential diagnostic methodology is the standard methodology of medicine” and that “it led [them] to conclude that your client’s product caused the claimant’s injury.”

The problems with moving in a one-step process from symptoms to the product or agent that caused them are twofold: 1) two separate and disparate methodologies are rolled into one; and 2) as a corollary, physicians are permit-

ted to jump from symptoms to external cause with neither the expertise, nor the data, to do so.

The Federal Judicial Center’s *Reference Manual on Scientific Evidence* (1994) defined the term “differential diagnosis,” properly, as:

The method by which a physician determines what disease process has caused a patient’s symptoms. The physician considers all relevant potential causes of the symptoms and then eliminates alternative causes based on a physical examination, clinical tests and a thorough case history.

Reference Manual on Scientific Evidence, First Edition (1994) at 214.

The critical component of this definition is that differential diagnosis is a quest for a diagnosis: what is wrong with the patient internally. It is not, inherently, a search for the ultimate cause (critical to liability) of that disease process or disorder. In numerous decisions, however, the courts use *cause of symptoms* and *ultimate cause of the disease process* interchangeably. By doing so, “differential diagnosis” becomes defined, incorrectly, as the methodology by which both a diagnosis and the cause of that diagnosis are assessed.

Distinctions—Diagnosis versus Identifying Causation

Before discussing specific cases, an illustration of the methodological distinctions between diagnosing an illness and identifying its cause may be helpful. Medical students are taught to diagnose and to treat. Most often they are not taught to uncover the causes of an illness. A recognizable analogy is found in the difference between the TV shows “ER” and “CSI.” In the former, diagnosis and treatment are central. Is there abdominal bleeding? Is there head trauma? What do we do for this patient? How do we save his or her life *now*? While

accompanying human interest stories may delve into the reasons for the injuries during the course of a show, they do not affect the care. Rather, the care depends upon a rapid differential diagnosis, followed by a prompt diagnosis that leads to immediate care. The differential diagnosis of an unconscious patient may include head trauma (either requiring surgery or not), diabetic coma, a stroke, a hypoxic brain injury following a heart attack, a blow with a baseball bat or poisoning, among many others. Once the MRI is completed and a skull fracture with a subdural hematoma is found, the differential diagnostic process is over. The diagnosis is made. The treatment begins. How the patient got that injury is neither part of the diagnosis, nor of the treatment.

The careful reader will note an exception to that statement in this example. What if, in the course of the diagnostic work up, the unconscious patient is found to have significantly elevated carboxyhemoglobin (COHgb)—say 50 percent? That finding leads to a diagnosis of carbon monoxide poisoning that also elucidates the outside cause of the unconsciousness: poisoning by carbon monoxide. In that case, the diagnosis necessarily leads to a causal attribution.

For most of the possible diagnoses in unconscious patients, however, the diagnosis explains the internal reason for the unconsciousness, but it does not explain the external cause of that condition. In the patient who is unconscious because of a brain hemorrhage, the neurologist finds and diagnoses that hemorrhage with an MRI. Whether that hemorrhage occurred because the patient was taking a specific medication, or whether it was due to uncontrolled hypertension, or whether it resulted from a congenital aneurysm or a blow to the head is neither part of the diagnosis, nor of the differential diagnostic process.

“CSI,” by contrast to “ER,” is all about cause. Usually the diagnosis is simple—the patient is dead. The question is why and by whom? These are causation questions. In “CSI,” they are not answered by physicians but, rather, by forensic profes-



Ronald E. Gots, M.D., Ph.D., is the CEO of the International Center for Toxicology and Medicine (ICTM) in Rockville, Maryland. For 30 years, Dr. Gots has been an active consultant and expert in major toxic tort and mass tort litigation, working with national law firms and general counsel throughout the U.S. and other countries.

sionals who gather evidence, put the pieces together and induce the causal answers.

An Example

An example relevant to defense counsel is the one that opened this article. What was the relationship, if any, between the aerial spraying and the patient's (claimant's) leg symptoms? Here we have two questions: 1) Why does the patient have leg symptoms? 2) Did our spraying cause those? The first is the "ER" question: the second, the "CSI" question. Answering each involves a separate methodology, separate expertise (rarely combined in one person) and more than a differential diagnosis. However, the diagnosis comes first. One cannot evaluate potential causal relationships until one knows what it is to have caused.

The differential diagnostic methodology is, therefore, the first step. Its intent is to answer question number one, but only that question. It is the standard methodology of clinical practice. The steps of the differential diagnostic process include identifying all reasonable potential internal disorders that could account for a patient's clinical presentation. Here, "internal" is the operative word, for diagnosis focuses on what is going on inside the patient; what is wrong with him. In our patient with strange sensations and weakness in his legs the differential diagnosis might include: peripheral neuropathy, a muscle disorder, a vitamin deficiency, potassium deficiency, vascular disease, nerve root compression, commonly related to degenerative lower spine disease, a spinal cord tumor and others. The diagnostic work up—the methodology of clinical practice—is designed to eliminate those possibilities that are incorrect and to elucidate the proper diagnosis. In this case, that would include taking a history from the patient, performing a complete examination—particularly a good neurological examination in this case—and conducting appropriate tests, various blood tests, x-rays of the lower back and electrical testing of nerve and muscle function. It is likely, but not inevitable, that a specialist (most likely a neurologist in this case) would participate in the diagnostic activity. For sake of discus-

sion, assume that the resulting diagnosis is a peripheral neuropathy. This means that something is wrong with one or more nerves in the leg. Moreover, it means that the problem is with the nerve(s) themselves, not with the lower back. This ends the differential diagnostic exercise. The diagnosis of peripheral neuropathy is the end of that methodological approach.

Thus, the differential diagnostic exercise involves six steps:

For most diseases, the diagnosis does not bespeak a cause. It directs care.

- Making a list of possible diagnoses (internal disorders) that could explain the presenting symptoms or observations;
- Taking a thorough medical history;
- Conducting a careful and complete physical examination;
- Ordering and interpreting the indicated tests;
- Ruling out diagnoses that do not fit the history or findings noted above;
- Arriving at the diagnosis that best fits the first five elements.

Before we even get to causation, there is a subpart to the differential diagnosis in this case—the type of peripheral neuropathy that exists. In other types of disorders, such as a malignancy, this determination is part of the differential diagnostic exercise, because generally a biopsy of the tumor is integral to the work up and provides the answer to this question. In the case of a peripheral neuropathy, we need to know what kind it is. The answer to this question gets us closer to potential cause, but is still insufficient. The complexities of this question are beyond the scope of this article—suffice it to say that entire textbooks cover the subject of evaluating peripheral neuropathies. A few of numerous considerations include: is this one nerve or many; axonal or demyelinating, metabolic, vascular, chronic or acute. Complex algorithms describe the diagnostic pathways that lead to the char-

acterization of the type of peripheral neuropathy. A biopsy of the nerve is generally one of those diagnostic elements.

Causation Assessment

At this point, this article will examine an entirely separate exercise—that of causation assessment. Again, we return to the example that introduced this article—whether an exposure to an aerial spraying (crop-dusting) was the cause of the leg symptoms. The elements required to assess this question differ considerably from those required to make the diagnosis and have been discussed extensively in numerous publications. See R.E. Gots and S.W. Pirages, *Applying the Principles of Science to Daubert Motions in Toxic Tort Claims*, Wiley Expert Witness Update, 1–88, Aspen Law and Business (2000). These required elements are also, individually, the subject of numerous *Daubert* decisions. They include, among others:

- Can the agent (pesticide) at issue cause any type of peripheral neuropathy (sometimes called the "general causation" question)?
- If so, does the patient have the type of peripheral neuropathy that can be caused by the exposure, or is this an entirely different type?
- Was there sufficient exposure to account for this condition?
- Are there equally, or more likely alternate, competing causes?
- Did the clinical course follow the sequence known to be associated with this potentially causal agent? (E.g., was there evidence of severe toxicity?)
- Was the temporal relationship the one expected if the agent at issue were causal?

Only after these questions are explored and answered affirmatively can the expert properly offer a causal conclusion. It should be obvious to the reader that the diagnosis (resulting from the differential diagnostic methodology) is only one of many steps required in causation assessment. The others not only require different data, including an appreciation of the dose received by the claimant and a comprehensive knowledge of the toxicology

of the chemical(s) at issue, but they also require specialized and different expertise. Most physicians, even neurologists, who diagnose peripheral neuropathies have limited toxicological backgrounds. The words “organophosphates, pyrethrins, carbamates, piperonyl butoxide, volatile organic chemicals” are rarely spoken in medical schools or in postgraduate medical training programs.

There are a few situations in which the diagnosis does suggest or establish a cause. Clear cut toxicological illnesses come to mind. The patient described earlier, brought comatose to an emergency department, found through blood testing to have a COHgb level of 60 percent, will be diagnosed properly as suffering from carbon monoxide toxicity. Here, the diagnosis explains both the reason for the coma and its cause. A rattlesnake bite or poisoning by aspirin might be similarly diagnosed and causally attributed. Certain allergic responses—anaphylaxis due to peanut allergy or a bee sting—are situations in which the diagnosis may be readily and inextricably tied to the cause. Another notable category of disorders in which diagnosis may suggest the cause is infectious diseases, *e.g.*, Tuberculosis is caused by the TB bacillus. What is it about such disorders that permits this linkage between diagnosis and cause? In the case of poisonings, it is either the laboratory evidence (blood determinations of toxins or their products) or the immediate, specific and profound nature of the response—or both. It is the latter that permits the bee sting/anaphylaxis connection. In these cases, it is not merely the temporal relationship that makes the connection, although that is a factor. The same cannot be applied to vague outcomes with uncertain causes such as non-specific symptoms connected to mysterious or simply speculative indoor environmental agents. Nor can it be applied, absent a thorough causation assessment, to disorders like asthma, RADS or neuropsychological dysfunction, which may have many causes.

For most diseases, the diagnosis does not bespeak a cause. It directs care.

Because physicians are trained to diagnose and treat, they are little involved with causation assessment. How and why the patient developed Hodgkin’s disease or prostate cancer is not important to the practice of oncology. How the lupus or the scleroderma came about is irrelevant to the rheumatologist. And why the patient developed MS is of little importance to the practice of neurology. *Cause is only important to the actual practice of medicine when it affects treatment or future prevention.* Certainly, the allergist may try to figure out what triggers his or her patient’s asthma attacks to treat with immunotherapy (allergy shots), or to suggest avoidance. One wants to know what the poison was to find the right antidote. Culturing the specific bacterium in an infectious disease permits selection of the correct antibiotic. The discussion in this article, however, is not focused on those disorders in which the diagnosis clearly connotes the cause. Rather, it is those disorders, commonly seen in personal injury, particularly toxic tort and pharmaceutical liability claims, in which the diagnosis tells us little about cause and where a separate causal analysis is needed.

Cause is, of course, a central legal concern. It is the cause that links the disorder to the responsible party. The scientific methodology of causation assessment was the focus in the *Daubert* decision. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S.579 (1993).

In *Daubert*, there was no differential diagnosis. The child’s birth defect was visible to a layperson. The only question was what caused it and how the claimants’ experts decided that it was the drug Bendectin taken by the mother during her pregnancy. In concluding that their methodology was flawed, the U.S. Supreme Court enunciated some of the required components for a properly constructed, scientifically based causal conclusion. When it comes to physicians, some courts have determined that since *Daubert* speaks of standard methodology within the relevant community, and since differential diagnosis is the methodology of clinical medicine, that physicians can speak

to cause using that differential diagnostic approach. That, of course, assumes (incorrectly, in the author’s opinion), that physicians are the community “relevant” to determination of cause. Other courts have recognized certain methodological needs beyond differential diagnosis to conduct a causation analysis, but rarely have they enunciated the clear and distinct differences that actually characterize the two.

Examples of Case Law on the Issue

The examples to follow are selected from nearly a hundred such decisions, but are illustrative of the confusion that pervades this issue.

In *Moore v. Ashland Chemical*, one of the better discussions of the role of differential diagnosis took place. The Fifth Circuit Court of Appeals overturned the trial court’s decision to exclude a medical expert who had reached a causation conclusion. *Moore v. Ashland Chemical Co., Inc.*, 126 F.3d 679 (5th Cir. 1997). There, the appellate court had concluded that *Daubert* factors were inappropriate for determining the reliability of testimony from a clinical medical expert. It suggested that physicians who use “differential diagnosis” methodologies to reach causal conclusions are not subject to the scientific exclusions enunciated in *Daubert*.

The Fifth Circuit granted rehearing to consider this case *en banc* and to clarify the standards district courts should apply in determining whether to admit expert testimony. On rehearing, the court held that expert testimony must demonstrate that findings and conclusions are based on scientific methodology. 151 F.3d 269 (5th Cir. 1998). They concluded that the testifying physician had not satisfied the elements of proper causation methodology. He had no support for the general causation issue (that the industrial solvent, toluene, could cause reactive airways dysfunction syndrome (RADS)). He had no exposure data. The fact that symptoms developed shortly after exposure was not a sufficient basis to assert causation.

Thus, we have two competing decisions, both flawed in their reasoning, but one closer to the mark than the other. In

the first instance, the court determined that a differential diagnostic methodology led physicians to causal conclusions and, since that methodology was specific to medicine, it satisfied *Daubert*. In other words, merely claiming that “I performed a differential diagnosis” was sufficient to insulate a physician from fulfilling any of the other essential causation methodological elements.

The *en banc* decision added a causation element—general causation—to the physician’s methodological requirement saying that, at least, he had to have some basis for claiming that the agent was an established potential cause of the disorder. And, they noted further, that a specific causation element—dose—was also important and not part of differential diagnosis.

In *Heller v. Shaw Industries*, following a *Daubert* hearing, the trial court excluded the testimony of the claimant’s medical expert who supported her claim that off-gassing from carpeting was a cause of the claimant’s respiratory difficulties saying: “I concluded that the carpeting in her house was the major factor in her illness.” *Heller v. Shaw Industries, Inc.*, 167 F.3d 146 (3rd Cir. 1999). He based this testimony on a “differential diagnosis.”

The Third Circuit Court of Appeals agreed with the trial court’s exclusion of plaintiff expert’s testimony. However, and distressingly, the court’s reasoning was not that it was the lack of scientific support for the general causation conclusion that rendered the physician’s opinion inadmissible; rather, it was his flawed differential diagnosis.

Apparently, the appeals court in this case believed that a correctly performed differential diagnosis was the proper way to arrive at a causal conclusion.

In *Cavallo v. Star Enterprise*, 892 F.Supp. 756 (E.D. Va. 1995), residents of a community near a fuel tank farm alleged a variety of illnesses from fugitive emissions and several small releases into their community. In discussing the claimant’s medical expert’s differential diagnostic assessment, the court arrived at a conclusion with reasoning that reflects both confusion about the meaning of differ-

ential diagnosis and some understanding that causal assessment involves more. The court’s comments are revealing.

...he primarily applied a methodology of differential diagnosis. Thus, he determined from Ms. Cavallo’s medical history, from her description of the spill incident, from his initial examination of her, and from the timing of the spill in relation to her development of symptoms, that her exposure to the

Diagnoses do not involve products; they involve disorders or diseases.

petroleum hydrocarbons could have caused her chronic illness. 892 F.Supp. at 771.

In accepting this testimony, the court accepted an admixture of differential diagnostic methodologies—medical history and examination—with causation methodologies—description of the spill, and timing of the spill. In the same ruling, the court distinguished between specific and general causation saying:

The process of differential diagnosis is undoubtedly important to the question of “specific causation.”... But it is also important to recognize that a fundamental assumption underlying this method is that the final, suspected “cause” remaining after this process of elimination must actually be *capable* of causing the injury. That is the expert must “rule in” other possible causes. And, of course, expert opinion on this issue of “general causation” must be derived from a scientifically valid methodology.

Id.

The court goes further in a related footnote discussion.

...Ms. Cavallo contends that Dr. Bellanti applied both 1) the toxicological methodology previously described to determine whether the AvJet spill could have caused her injuries and 2) a differential diagnosis to rule out other possible causes.

Id.

Here, the court reached a conclusion that general causation is a separate methodology, falling under “toxicological methodology.” While that is true, they incorrectly concluded that all of specific causation is satisfied through a differential diagnostic, medical methodology. Differential diagnosis is, in fact, one part of specific causation (which this court earlier said), but the other parts—dose, timing, ruling out alternate causes—are not. These are part of causation analysis and might better be placed, using this court’s terminology, under “toxicological methodology.”

The medical expert’s testimony was excluded in this case because he had failed to assess “general causation.” The remainder of his reasoning, a combination of differential diagnosis and elements of specific causation, was accepted.

Another recent case involving orthopedic spinal screws was dismissed by the trial court for a number of reasons. *Lawrence v. Synthes, et al.*, (No. 1623 EDA 2003 Pa.Super.). Among them was the lack of reliability of plaintiff’s expert physician’s testimony. The motion *in limine* and summary judgment were reviewed and the decision was upheld. The appellate court properly noted:

Differential diagnosis is the determination of which of two or more diseases with similar symptoms is the one from which the patient is suffering, by a systematic comparison and a contrasting of the clinical findings.... In other words, differential diagnosis is used to determine what the condition is and how to treat it. Causal assessment, on the other hand, is the methodology used to determine how the condition arose and to determine responsibility or liability.... The differential diagnosis methodology does not lead to a causal determination.

The claimant’s experts in *Mattis v. Carlon Electrical Products*, 114 F.Supp.2d 888 (D.S.D. 2000), incorporated the cause of the RADS within their diagnosis, asserting not only that the patient had RADS, but providing a causal attribution. “RADS due to toluene exposure,” was the offered diagnosis. The court found, incorrectly,

that the determination of cause was part of that “differential diagnostic” activity.

The opinions of Dr. Hansen and Mr. Wabeke, and indeed the theory of RADS itself, are based upon a technique called differential diagnosis. “Differential diagnosis, or differential etiology, is a standard scientific technique of identifying the cause of a medical problem by eliminating the likely causes until the most probable one is isolated.”

Id. at 892. (*Aff’d*, 295 F.3d 856 (8th Cir. 2002).)

“Differential diagnosis” is not equivalent to “differential etiology.” “Etiology” speaks more to the ultimate cause, that is, the cause of the disease process. Diagnosis speaks to the disease process: what is wrong with the patient. Thus, in this case, the differential diagnosis would establish the existence of RADS (Reactive Airways Dysfunction Syndrome). This diagnosis is the disorder that the patient has, not what caused it, and is the end of the differential diagnostic analysis.

Etiology, or cause of the RADS, is the *next* question. This involves a new process necessitating answers to other types of questions: whether the agent at issue is capable of producing RADS, *i.e.*, whether it has irritant properties; approximately what level of exposure occurred (dose); what was the timing between symptom onset and exposure (clinical course); were there alternate potential causes of the RADS considered and ruled out? The evaluation of these critical elements is not part of diagnosis, but is central to causation analysis methodology.

In *Glastetter v. Novartis Pharmaceuticals*, the trial court dismissed the claimant’s expert testimony that the drug Parlodel caused the claimant’s intracranial hemorrhage (ICH). 252 F.3d 986 (8th Cir. 2001). Affirming that decision, the appellate court discussed, properly, certain elements of causation assessment, but they incorporated, improperly, cause into the concept of a differential diagnosis. They said:

Each of Glastetter’s experts conducted a “differential diagnosis,” which concluded that Parlodel caused her ICH. In performing a differential diagnosis,

a physician begins by “ruling in” all scientifically plausible causes of the plaintiff’s injury. The physician then “rules out” the least plausible causes of injury until the most likely cause remains. *The final result of a differential diagnosis is the expert’s conclusion that a defendant’s product caused* (or did not cause) the plaintiff’s injury. [Emphasis added.]

252 F.3d at 989.

While the subsequent discussion delineated the elements of causation assessment, it is clear from that discussion and from the above statement that the court failed to understand the definitional distinction between differential diagnosis and causation assessment. The suggestion that differential diagnosis has anything to do with a product is illustrative. Diagnoses do not involve products; they involve disorders or diseases. Here the differential diagnoses and the ultimate diagnosis which arose from that differential diagnostic process were clear: intracranial hemorrhage. That diagnosis was the end of the differential diagnostic exercise. The next question “What caused the ICH?” begins the new analytical exercise: the causation analysis.

The Fourth Circuit case, *Westberry v. Gislaved Gummi AB*, 178 F.3d 257 (4th Cir. 1999), is frequently cited to support the use of differential diagnosis as the methodologically proper way of assessing causation. In the underlying case, the claim was that the inhalation of talc from workplace exposure caused a sinus disorder in the claimant. A verdict was delivered for the claimant. The appeal argued, among other things, that the claimant’s physician’s methodology in arriving at a causal conclusion was defective. The appeals court disagreed with this argument saying:

GGAB contends that Dr. Isenhower’s testimony was inadmissible because it was not based on reliable scientific methodology. This is so, it argues, because Dr. Isenhower had no epidemiological studies, no peer-reviewed published studies, no animal studies and laboratory data to support a conclusion that the inhalation of talc caused Westberry’s sinus disease. Further, GGAB continues, Dr. Isenhower did not have any tissue sam-

ples indicating that talc was found in Westberry’s sinuses, nor did he have studies showing that talc, at any threshold level, causes sinus disease. Instead, Dr. Isenhower merely relied on differential diagnosis—supported in part by the temporal relationship between Westberry’s exposure to talc and the problems he experienced with his sinuses. . . . GGAB maintains that neither a differential diagnosis nor a temporal relationship between exposure and onset or worsening of symptoms is sufficient to establish the reliability of Dr. Isenhower’s opinion. We disagree.

178 F.3d at 262.

Here, the court noted some of the necessary elements of causation when it repeated the defense’s arguments. While rejecting those, it accepted the identification of a temporal relationship as a component of “differential diagnosis.” This should rarely be the case, except in acute poisoning matters. Rather, temporal relationships are a regular component of “causation assessment.” Finally, the court ruled that differential diagnosis is the proper methodology for assessing causation. In fact, assessing causation requires knowledge of those elements enumerated in the defense’s arguments and, sometimes, others as well.

In *Cutlip v. Norfolk Southern*, 2003 WL 1861015, 2003 Ohio App LEXIS 1785 (Ohio App. April 11, 2003), the claimant’s medical experts testified that the claimant’s respiratory disorders arose from exposure to diesel fumes in his worksite. In doing so, they used “differential diagnosis” as their methodological argument, which was accepted by the trial court. The appellate court agreed, stating the following:

In this case both physicians testified that they personally examined appellee, they reviewed his medical records, they took a history, they ordered tests and they reviewed the results of those tests. They both also considered other possible causes (such as the chest wound and smoking) and ruled those out as possible causes of appellee’s asthma. Based on the law discussed above, we conclude that Drs. Khan and Kelly arrived at their conclusions following

a thorough differential diagnosis, and their testimony is therefore reliable under *Daubert* and Evid R. 702.

Cutlip at *7.

The first sentence of the above statement correctly delineates the process of differential diagnosis. The second sentence, including ruling out other causes of the disease, does not. Once again, the court intermingled “differential diagnosis” with “causation assessment.”

The *Hardyman* case involved a claimant with carpal tunnel syndrome (CTS), the alleged cause of which was a specific set of occupational activities. In a Sixth Circuit decision overturning the trial court’s exclusion of the claimant’s expert, there was extensive discussion of both differential diagnosis and of cause. At the trial level, the claimant’s expert himself clearly separated the elements of each, but the appellate court merged them. Dr. Linz, the claimant’s medical expert, said:

[O]nce we go beyond the issue of general causation and get away from evaluating and criticizing the literature whose only purpose really is to establish how well-established this work is [*sic*] causation in general, then we get to specific causation. And the approach to specific causation is really quite difficult. We are looking at: 1) whether or not the person had carpal tunnel syndrome. It is absolutely clear in this case that he did; and 2) does he have documented medical conditions which might be significant contributors to his condition. That was evaluated by me and I felt that he did not. The third is whether his work activities are significantly more hand intensive than his nonwork activities, and in addition are his work activities significantly more hand intensive than other jobs [*sic*]. . . .

Hardyman v. Norfolk & Western Railway, 243 F.3d 255, 262 (6th Cir. 2000).

The appellate court misconstrued this correct analytical approach as a “differential diagnosis” when it commented upon that testimony:

Although the district court acknowledged that differential diagnosis is an acceptable method of determining

causation, it not only failed to recognize that Dr. Linz applied a method of differential diagnosis in reaching his conclusion, it seemed actually to reject this method. . . .

Id. at 261.

The statement by Dr. Linz noted above and cited by the appellate court actually incorporated both methodologies: differential diagnosis and causation assessment. The end of the differential diagnostic

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exercise was the diagnosis (carpal tunnel syndrome). Whether or not that diagnosis was accurate could be a matter of dispute between medical experts, but the method of arriving at that diagnosis, history, physical examination, laboratory testing, is not. In contrast to the misunderstanding of the appellate court, however, the diagnosis—and only the diagnosis—was the outcome of the differential diagnosis. All of the other components (which the claimant’s expert acknowledged as difficult) were elements of causation analysis. This included the general causation question: Can such activities cause this disorder, CTS? The causation analysis also included the specific causation questions: Have other causes been considered and ruled out? Was the work activity sufficient? Was it more significantly intensive than non-work-related activities?

Finally, in a recent decision, the U.S. District Court for the Southern District of New York, enunciated the difference between the customary medical use of the term “differential diagnosis” and the interpretation of that by the courts (*In re Rezulín Products Liability Litigation*, 2005 WL 583751, MDL No. 1348, Master file 00 Civ. 2843 (LAK) (S.D.N.Y. March 14, 2005). In this multidistrict decision, the court said:

Courts have come to use the term “differential diagnosis” differently from prac-

ticing physicians. In clinical medicine, “differential diagnosis” describes “the process of determining which of several diseases is causing the patient’s symptoms.” *Reference Guide on Medical Testimony, supra*, at 443. Expert witnesses and courts, however, frequently use the term “to describe the process by which causes of the patient’s condition are identified, particularly causes external to the patient.” *Id.* At 443–44. On the whole, the plaintiff’s counsel and their experts have used the term the latter way.

Id. at *24 n.224.

The court went on to reject this latter use of the term in which differential diagnosis and causation assessment were employed interchangeably.

Conclusion

Courts’ failures to understand the important differences between differential diagnosis and causation assessment are largely based upon confusion about, and misconstruction of, the meaning of the word “cause.” In case after case, the courts allow physicians to testify to “cause” using a differential diagnostic approach, noting that this approach is the standard methodology of clinical medicine. The error is in failing to ask: cause of what? If, by “cause,” the courts were referring to the cause of the patient’s symptoms or internal disorder, *i.e.*, the diagnosis, they would be correct. Uncovering that through a differential diagnostic approach is the standard methodology of clinical medicine. But in the context of a tort claim, there are at least two causes: the internal cause or diagnosis, and the external cause, the subject of the liability claim. It is the failure to make this critical distinction that has characterized often flawed decisions. In some cases there may even be a third causal question—whose chemical, *e.g.*, which asbestos or silica or lead.

Differential diagnosis asks: what is wrong with the patient? Causation asks: how and why did the condition arise? For counsel to challenge appropriately, physicians’ ill-founded causal attributions, they must understand the difference and be able to explain that to the court. **FD**