Medical and Legal Causation

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It is an interesting and revealing circumstance that one of the first sessions in a workshop entitled "Occupational Health in the 1990s" should concern the problem of medical versus legal causation. This, no doubt, is some measure of the importance that litigation has already assumed as a driving force for much that is both good and bad in medicine in general and occupational health in particular. The directions that disease prevention takes in the 1990s may well be determined in large part by how the question of medical causation evolves. I will attempt to justify this rather rash statement at the end of my brief discussion of the causation problem.

Although differences between medical and legal causation are apparent in several contexts, including regulation, my remarks deal primarily with tort actions, which require plantiffs to demonstrate that a defendant was the "legal" cause of an occupational illness. In contrast to the usual case of occupational injury, which calls for evidence that a specific, concrete event or condition gave rise to the plantiff's harm, in many cases of suspected occupational disease there may be considerable uncertainty about the relation between exposure to a toxic agent and the plaintiff's disease. When this uncertainty exists or is claimed to exist by defendants, scientific evidence about causation becomes an essential part of the case.

I do not have space here to examine the differences between legal and medical causation in detail.[1] Indeed, part of what I would like to claim is that most attempts to set out such differences must shoot at a moving target. Causation is a much abused, but little understood concept, considerably more complex and sophisticated a notion than we generally give it credit for. I do not believe that we can give a good accounting of it yet, the volume of writing on the subject notwithstanding. There are, however, some things we can say about the misunderstandings of causation.

First, causation is a characteristic of a relationship, not an empirical characteristic. Calling a relationship "causal" is a judgment, one based on both theory and empirical evidence. Since much, if not all, empirical evidence is itself theory-laden, causality is ultimately tied intimately to a base in theory. There is no way that causation can be "discovered" without the essential participation of theory. This holds for all kinds of causation, in the laboratory, in the real world, or wherever. The oft-heard pronouncement that epidemiology can reveal only associations, not causation, is fundamentally wrongheaded and mistaken. It is, in some trivial sense, true, but this is a property it shares with every other empirical science as well. Hill's well-known guidelines on causation[2] are just that, guidelines to help make a judgment about causality, not postulates that must be fulfilled for causality to hold.

The confusion over the nature of causation is compounded when two cultures meet, as in the courtroom. That causation means something different in law and in science is well known and will not be belabored here. The point I wish to empha-

size is that not only are the notions different but also there is no fixed meaning in either discipline. In both cases, the notion of causality is both created and modified as the consequences of earlier decisions become evident and are tested in new situations, in the one case by additional experiments or observations of the real world and in the other by testing its appropriateness in new legal cases and situations.

Hence there are many reasons why the topic of this panel, medical and legal causation, is ambiguous and ill-defined. Because causation is a judgment call, we must ask whose judgment will prevail and for what purposes. The judge's judgment is incorporated into his or her decisions on the admissibility of testimony and evidence, and the instructions to the jury, which in turn are based on a body of rules, law, and precedent concerning the nature and requirements for legal causation. The jury's judgment is constrained by the previous decisions of the judge, but it involves in addition a complex web of tacit presumptions and analogies from everyday experience. Rather than discuss the interesting question of legal judgments, however, let us look at scientific judgments.

First, let me address the issue of scientific consensus with respect to causation. When a rough consensus exists in the scientific community, arrived at by whatever method, controversy about legal causation will be lessened, but not necessarily eliminated (e.g., cancer trauma cases.)[3] On many issues such consensus does exist, but on many more there is substantial controversy. One feature that tends to set off those subjects for which there is controversy from those for which there is little or none is the existence of substantial consequences, that is, controversy tends to exist whenever the stakes are high, independent of the degree of "scientific certainty" involved. Stakes can be high because of scientific importance, perhaps because a result calls into question conventional views, or because of practical consequences, for example, it will affect regulation or result in large liability.

The causation issue in an occupational disease suit can be approached in two ways: one from the general or universal point of view, the second from the particular or clinical point of view. In the first approach, an attempt is made to prove that exposures similar to the plaintiff's would cause the observed health effects in the general case, that is, at other worksites, under different conditions, in different populations, via other routes of exposure, and so forth. The plaintiff then becomes merely a special case. This is the type of causation of which Hill speaks in giving us his criteria for causal associations, one of which is consistency with other results in other populations, settings, and the like. The second approach is particularistic: Did exposure cause harm to this worker at this time? This kind of causal judgment is the norm, not the exception. This is the usual clinical situation in which a physician diagnoses the individual person. Clearly the two approaches are related, but they are not identical.

The difference is not as stark in the case of a single worker as it is when groups of workers are involved. Consider, for example, a large group of workers exposed to an organic solvent. These workers complain of a wide variety of problems, from heart disease to depression to frequent respiratory problems. One way to establish causation is to perform an epidemiologic study to demonstrate that this group of workers, taken together, had an illness experience different from that of a suitable comparison group. Although this is possible, and in fact forms the bread and butter of the occupational epidemiologist, the constraints and difficulties of this approach are well known. Success even in the face of a real and substantial effect is often meager, and the result is still difficult to apply because it pertains to the group, not necessarily to all individuals within the group. However, we could

also imagine putting each worker through a battery of tests, from neuropsychologic tests to complete cardiovascular workups to immune panels. The results of this battery in each case would be examined to determine if they were consistent with exposure to the solvent in question.

I can already hear the objections to this way of proceeding. It will be called anecdotal, unscientific, too uncertain, and unrigorous a judgment on which to rest weighty matters of dollars and sense. But let me emphasize again that it differs little from the usual instance of clinical diagnosis that occurs thousands of times daily. Drugs are prescribed or withdrawn, tests are ordered, and surgery is performed, each time on the basis of the same kind of causal judgment but with far less evidence than is present in these cases. Moreover, this happens even when we know that there are parties that stand to gain or lose in the process, and that there are significant practical consequences to these decisions for the patient. We recognize that we cannot wait indefinitely for some unstated threshold of proof to be crossed before a patient is treated, and furthermore that professional judgment is itself a powerful intellectual tool that can divine connections in particular instances without a standard algorithm that can be routinely applied to every case. This situation is no less true in court than in a doctor's office. And because of the adversary process, there is always a "second opinion." Ultimately the jury, like the patient, must decide.

I make these remarks in defense of clinical causation, not to disparage the attempt to establish universal or general propositions, but only to suggest that this is not the only practically significant standard of causation that we use. Most of my professional life is taken up with trying to establish such general propositions about the effects of chemical exposure on populations, and hopefully the results will be useful to those who must apply them clinically to individuals as well as to the public health community. But to use the criteria for this purpose for causal decisions about individuals places a heavy burden on a clinician or a community.

The population approach, however, must show that the particular case represents a general phenomenon. This is an appropriate objective for science, but a heavy burden for a community, plaintiff group, or single patient to bear. In some cases, such a demonstration may be beyond the limits of our tools. A rare disease or idiosyncratic reaction following a rare exposure, such as aplastic anemia after chlordane exposure, may be obvious from case reports but may not be demonstrable by epidemiologic study.

To return briefly to the significance of this element to occupational disease prevention in the 1990s, I believe that conceiving the causation problem from the clinical rather than the epidemiologic point of view will allow many more plaintiffs to obtain some compensation for harm. Whether this is good or bad depends, I suppose, on whose ox is being gored. More importantly, however, it will also result in the same kind of defensive behavior on the part of employers that is now so typical of medical practitioners faced with the specter of a malpractice claim. Conversely, requiring an epidemiologic standard of proof in these cases will essentially foreclose on the ability of many truly injured workers to recover any damages. The result will be to remove what apparently is one of the more potent driving forces behind workplace preventive measures.

The fear of liability is certainly not the only lever that can move the heavy weight of prevention. But it is an important ingredient in employer decision-making, and factors that affect it may in turn advance or retard the introduction of preventive measures in the workplace. The appearance of this panel's subject matter in this workshop was a recognition of these complex connections.

NOTES AND REFERENCES

1. There is a large literature on this subject. For a guide to some of this research and a recent statement about it, cf. BODEN, L. I., R. MIYARES & D. OZONOFF. 1988. Science and persuasion: Environmental disease in US Courts. Soc. Sci. Med. **27:** 1019–1029.
2. HILL, A. B. 1971. Principles of Medical Statistics, 9th ed. Oxford University Press. New York.
3. TILEVITZ, O. E. 1977. Judicial attitudes towards legal and scientific proof of cancer causation. Columbia J. Environ. Law. **3:** 353.