# Advances in the Epidemiology of Injuries as a Basis for Public Policy

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*The selection of injuries as a topic of this landmark session recognizes the fact that accidents are the leading cause of death in our nation in the age group 1–44 years.*

*With a leader in the field such as Dr. William Haddon, Jr., American epidemiology has made enormous strides toward an understanding of the factors leading to injury. Haddon, now president of the Insurance Institute for Highway Safety but formerly with a career in the halls of government, has been an unrelenting exponent of the epidemiologic approach to injury control. His personal contributions in this field have been an outstanding component of these landmark achievements. In his work with the New York State Health Department, he interjected the epidemiologic method into the planning and execution of programs of disease control. His strict interpretation of research data and his persevering integrity have earned him both friends and foes in governmental and industrial circles. His topic, as one of the landmarks of the achievements of these past 50 years, reflects his staunch insistence on the use of epidemiology in the development of a public policy for health and its application to public health practice.*—LEONARD M. SCHUMAN, MD

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MEASURES TO REDUCE INJURIES have been used since ancient times by societies and individuals (*1*). Many have worked so well that they have been used for millennia. We still save entire populations from injury by evacuating them from large-scale environmental hazards—floods, invaders, volcanic eruptions, and so forth—just as in ancient times.

Indeed, societies have largely flourished in direct proportion to their ability to deal with the military and other hazards of their situations (*1*). This fact is one that those concerned with reducing damage from all kinds of environmental hazards would do well to remember and use. Individuals, also, have long reduced their injuries by a variety of measures, of which shoes are the most mundane illustration. Today, as in all past periods of which we have substantial knowledge, societal and individual injury control measures are ubiquitous. Airport control towers, stoplights, fuses, electrical insulation, lead aprons, potholders, sprinkler systems, Band-Aids, emergency medical care, and the treatment of hemophilia, of postmenopausal osteoporosis, and of posttraumatic epilepsy are illustrative.

In parallel, both the substance of injury control and its literature (for example, that part concerned with fire prevention and control) are extensive and largely outside the traditional purview of public health. Nonetheless, in recent years a small number of public health people have contributed important concepts for dealing

with injuries of all types; developed and applied powerful epidemiologic methods; reported many major research results; and had major roles in the implementation of an increasing number of effective injury control programs. In fact, the literature on injuries that is of special interest to public health is already so extensive that to review it even cursorily in a paper of this length is impossible. However, references *2–14* and others cited throughout the paper provide broad access to this literature and reflect the range of points of view represented by the best work in the field. Citation of this work leaves me free to devote the rest of this paper to (*a*) some of the most basic concepts in the epidemiology of injuries—all formulated since 1940—and the relationship of these concepts to classic epidemiology and (*b*) some recent extensions of injury epidemiology of considerable potential use to all those concerned with reducing damage from environmental hazards of all types.

## An Epidemiologic Problem

Injuries have always been at least endemic, and measures for their control have been exceedingly diverse and widespread. It is therefore remarkable that John E. Gordon, only 30 years ago, was apparently the first to understand that broadly considered, they are in many respects a problem in medical ecology. He suggested that injuries, being characterized by point epidemics, seasonal variation, long-term trends, and geographic, socioeconomic, and rural-urban distributions, behave in many respects like the classic infectious diseases and other forms of pathology already well understood (*15*). In fact, widely believed folklore notwithstanding, all known injury distributions are highly nonrandom in time, place, and person, just as one would expect from the nonrandomness of their causes. (The statistically sophisticated may challenge this statement by referring to the well-known Poisson distribution of horse kicks in a Prussian cavalry regiment (*16*). However, neither the periods and places of the use of cavalry, nor Prussian warriors—let alone those of their number exposed to horses—were randomly distributed, as their subsequent extinction demonstrates.) Neither Gordon, nor many of his followers even to this day, correctly recognized that like classic infections, injuries also have "agents" (the term "agent" being used in its most classic epidemiologic sense of an environmental entity whose action is necessary to produce the specific damage of interest and without which it cannot occur). Instead, Gordon erroneously identified as agents such components of the general environment as a glass-paneled door and incorrectly compared the germ in infectious disease with the loose board in a home accident (*15*). But neither the door nor the board is necessary for an injury to occur, and neither is the conceptual or functional equivalent of the organism necessary for a specific infectious pathology to occur.

## The Agents

Before identification of their causes, afflictions of the body have always been described in terms of their manifestations. For example, 3-day fever, infantile paralysis, scurvy, diabetes, hypertension, atherosclerosis, Legionnaires' disease, leukemia, and cancer are all descriptive, not etiological, terms. As knowledge of causes has increased, equivalent or reshuffled groupings have typically been substituted, each defined in terms of an etiological factor (*6,7,17*). Thus, for example, *Plasmodium vivax* infection, *Mycobacterium* infection, ascorbic acid deficiency, legionellosis (*18*), and Epstein-Barr virus infection (*19*) are all groups that are defined by the causal agents necessary to produce the specific pathologies so described. Viewed somewhat differently, I believe that a central, if not the central, research question to pose in seeking an understanding of any descriptively defined pathology is whether there are necessary, specific causal factors each of which accounts for a discrete part of the overall group (as proved to be the case with the classic childhood illnesses) or whether only one factor accounts for the entire group.

From this standpoint, the central problem in approaching the control of the pathologies that are grouped descriptively as injury was in determining if there were causal factors each of which was necessary to produce a subset of the overall injury group and without whose action the pathology in that subset could not occur. In other words, were there agents (in the classic epidemiologic sense) whose interactions with people could provide a focus in conceptualization, research, and prevention? Or was there only the obvious global web of causation, which unchanneled through necessary, specific etiological factors was operating damagingly on people?

In a 1949 paper that is sometimes mentioned as having solved this problem, King suggested that accidents increase in frequency with increases in various stresses (*20*). However, although he specifically listed as stresses essentially all the factors that were later recognized as the specific, necessary agents of injuries, King also included such other factors as "aging and disease" and "nutritional deficiency," which are not specific, necessary agents but factors influencing host susceptibilities to them; I discuss these later. Thus, he did not answer the fundamental question.

An experimental psychologist, Cornell University's

James J. Gibson, in 1961, was the first to actually clearly identify, and thus delineate, the necessary, specific agents of injuries (*21*):

> Man . . . responds . . . to the flux of energies which surround him—gravitational and mechanical, radiant, thermal, and chemical. Some limited fields and ranges of energy provide stimuli for his sense organs; others induce physiological adjustments; still others produce injury . . .
>
> Injuries to a living organism can be produced *only* by some energy interchange. Consequently, a most effective way of classifying sources of injury is according to the forms of physical energy involved. The analysis can thus be exhaustive and conceptually clear. Physical energy is either mechanical, thermal, radiant, chemical, or electrical. [Emphasis supplied.]

Students of coincidence in science may be interested that though I had sat in on some of the same conference for which Gibson wrote, I had not heard or read his paper (or King's) when I arrived independently, 3 months later, at essentially the same understanding, namely, that the several kinds of energy are the necessary, specific causes of such injuries as lacerations, burns, electrocutions, acute radiation effects, and corrosive burns, and that other injuries involve agents such as water (as in drowning), carbon monoxide, and cyanide that specifically interfere with normal body energy exchange (*3,22,23*). I also recognized that from this standpoint, frostbite was a type of injury that was etiologically analogous to each of the nutritional deficiency diseases, since it was also specifically caused by the absence of a necessary factor, the ambient heat needed for normal health. (Since the early 1960s, I have found it useful to think of such necessary, specific agents of deficiency states as "negative agents" and to recommend that this notion and usage be generally adopted.)

## Vehicles and Vectors

The recognition of such agents, especially mechanical energy, as necessary and specific causes of various kinds of injuries simultaneously pointed me toward the means of their transmission, again in close analogy to the concepts and substance of classic epidemiology (*22,23*):

> Energy that may reach the body and substances that may interfere with its normal function are usually carried by inanimate objects or living organisms corresponding to the "vehicles" and "vectors" of infectious diseases. Thus, electric lines are vehicles of electricity, hot rivets are vehicles of thermal energy, poison containers are vehicles of their contents, and moving objects are vehicles of mechanical energy. Similarly, poisonous plants and animals are vectors of their toxins, and animals that injure by tearing and crushing are vectors of mechanical energy. This concept is a useful one, since many preventive measures must be directed against the vehicles and vectors rather than against the physical and chemical agents they transmit.

These points are still especially important because of the continuing confusion about them, even in recently published work, for example, that of Gratz (*24*). Typically, this confusion results from, and produces, a muddled conceptual framework, just as poorly suited for dealing precisely and effectively with the theoretical and practical problems in this field as if in malaria epidemiology and control, no etiological distinctions were made between a mosquito vector and the plasmoidal agent it carries.

## Susceptibility to Injury Agents

I also recognized that epidemiologically, variations in resistance to the agents of injury often greatly influence the occurrence and nature of injuries in populations, just as variations in resistance to microbial agents influence the occurrence and nature of infections (*3,22,23*). As with infections, some of these differences in susceptibility to injury agents are genetic (for example, in hemophilia and osteogenesis imperfecta), and some are acquired (for example, in scurvy and probably in postmenopausal osteoporosis). Consequently, as with infections, raising body resistance that is abnormally low (as in hemophilia treatment) or preventing its decline (as in maintaining adequate ascorbic acid intake to prevent scurvy) is a sometimes useful choice from among the 10 fundamental injury control strategies (page 418). Moreover, as with vaccination, raising normal resistance is one objective, for example, of military basic training, various athletic practices, and suntanning (*4,6,22*).

Gibson mentioned the role of "environmental dangers" in natural selection (*21*), and in commenting on such factors many years ago, I wrote the following, which still largely applies today (*3*):

> . . . genetic factors associated with the initiation of accidents, with susceptibility to injury, and with the ability to recover from injury are probably being differentially selected even in present-day populations, just as they have undoubtedly been in the past. In fact, since accidents are currently the leading cause of death in many societies throughout most of childhood and the childbearing years, it is quite possible that they are one of the principal contemporary means by which the composition of population gene pools is changing, a point almost universally overlooked in research and evolutionary speculation by geneticists and others. Nonetheless, many evidences of genetic factors of possible relevance may be cited. These include the apparent role of genetic factors in myopia; the demonstration of strain differences in experimental animals in preference for alcohol; genetically mediated variations in susceptibility to injury, as in hemophilia, albinism, porphyria, and osteogenesis imperfecta; and the probability among more normal individuals of similar genetically mediated variations in ability to survive injuries of various types once they are sustained. [References in original omitted.]

However, not only abnormal and induced variations in injury susceptibilities (often referred to by experts as "injury thresholds") are critically important. So, too, are their absolute magnitudes, since these, together

with various relationships—for example, between rates of change of velocity (accelerations) of bodies of given mass and the forces required to produce them, as described by elementary physics—determine the magnitudes and the rates of the energy exchanges that can be tolerated without injury under given environmental conditions. In illustration, huge amounts of mechanical energy are imparted to astronauts' bodies in accelerating them and are removed in their deceleration; yet because the rates of energy exchange, and hence the maximum forces involved, are kept well below the well-known, very high (absolute) injury thresholds of healthy adults in good physical condition, no injury is sustained.

The late Hugh De Haven, from whose work much of the modern field of injury control derives, was the first to realize the central importance of injury thresholds in body mechanical energy exchanges and that it was these thresholds, together with impact conditions (not velocity of impact per se) that solely determine the injury outcome when specific amounts of energy are dissipated. Epidemiologists will recognize that this interaction provides a straightforward example of the epidemiologic triad: the host—the person susceptible to injury—interacts with the necessary, specific agent—mechanical energy—and with the environment—the impacted structure, and the interacting characteristics of all three determine whether the specific pathology, that is, mechanical energy exchange injury, occurs and with what timing, characteristics, and severity.

De Haven's classic papers were the first to focus competently on understanding—and thereby reducing—the actual problem, the occurrence of injury per se, rather than concentrating on finding the initiating shortcomings of the people involved. These papers began with "Mechanical Analysis of Survival in Falls From Heights of Fifty to One Hundred and Fifty Feet" (*25*), which was published in 1942, after De Haven had persuaded the department of physiology of Cornell Medical College to take him under its wing as a research associate. In most previous work, a search for the initiating shortcomings of the people involved in injuries had been the virtually exclusive preoccupation, and unfortunately that emphasis has also often continued in subsequent work. A number of De Haven's more accessible papers (*25–37*), including the one just mentioned, as well as several papers by others about his work (*38,39*), are must reading for all those seriously interested in injury research and control. They exemplify well the crucial distinction between the ability to observe and interpret, on the one hand, and the ability only to count and analyze, on the other. (Stapp, whose work began after De Haven's, and many others also have contributed great amounts of information concerning the thresholds of injury in mechanical energy exchanges (*3,40*).)

De Haven's work was of immense practical importance because it showed that the ability of the normal body to sustain very brief mechanical energy exchanges without fatal injuries is so unexpectedly great that a substantial majority of the vehicle occupants fatally injured in crashes would have survived, often without injury, if their vehicles had been designed to provide appropriate occupant "crashpackaging" (*13*). That even the newest vehicles do not provide sufficient, practical crash protection is one of the greatest and most tragic health scandals of our century.

## Injury Versus Disease

These cursory illustrations indicate the ease with which a straightforward extension of the host-agent-environment model of classic epidemiology accommodates injury epidemiology. Growing substantive knowledge, the increasing numbers of examples of successful injury control measures, and the remarkable discrepancy between injury totals and the amount of truly professional attention devoted to their reduction have all helped erode the resistance to considering injury epidemiology and control as a legitimate and useful field. However, I believe there is a far more basic, ancient reason for such resistance, one which only recently has begun to be discussed. This reason, which I believe underlies the fact that virtually everyone makes a distinction between injury and disease, is not based on differences in agents. As Susan Baker and I recently pointed out (*13*):

> . . . in some cases, the etiologic agents are identical: for example, the result of brief exposure to high concentrations of a toxic gas is called "injury," whereas the eventual pulmonary effect of chronic exposure to low concentrations of the same agent is called "disease." Similarly, mechanical forces produce "injury" to the spine when applied in large doses; in smaller doses over long periods they produce lumbar disc "disease." . . .

Altogether, although the point deserves far more professional and reliable scrutiny than mine, I am informed that the injury-disease difference is clear cut at least in the Indo-European languages (including Danish, Dutch, French, German, Lithuanian, Norwegian, Portuguese, Sanskrit, Spanish, and Swedish); in Arabic and Hebrew; in Chinese; in Japanese; and in Bengali, Gujarati, Hindi, Finnish, Hungarian, Malayalam, Marathai, Punjabi, Tamil, Vietnamese, and Yoruba. However, R. J. Smith III of the Indian Health Service informs me that the distinction does not appear to be present in the Navajo language. If this absence is confirmed, this and any other such examples would

tend to point to an acquired, rather than a biologically innate, basis for this ancient injury-disease distinction.

Possibly, the relevant fact is that when languages and their associated cultures were incorporating this distinction, diseases were not understood, whereas injuries were probably then as now regarded as being the clear result of whatever or whoever "caused" the damaging incidents, for example, what we now consider the vectors and vehicles of injury (such as wild animals, assassins, and spears) or situations (such as war). Important cause-and-effect relationships (but not all of the intervening steps, such as the necessary energy transfers) must have been obvious then, as they are today, whereas diseases usually must have seemed to lack clear antecedents.

If we remember that both disease and injury are, in essence, descriptive, less than precise lay terms, which are used also by medical and public health professionals, we find a related, but much simpler explanation. With rare exception, the concept of injury has been used since antiquity for pathologies that become apparent very shortly—even, it often seems, immediately—after their agents (or vehicles and vectors, for example, falling trees and wild animals) first begin to interact with the body. In contrast, the term disease has long been used not only for pathologies such as smallpox and black lung that first become manifest only after much longer periods following the first exposure to their causes, but also for other conditions whose causes are less well understood, if understood at all—in other words, for pathologies of more obscure origin.

When the pathology is known to be due to an infectious agent, the time for it to become manifest is called, of course, the incubation period ("*Incubation period* —the time interval between exposure to an infectious agent and appearance of the first sign or symptom of the disease in question" (*41*)). However, because of the many established infectious disease overtones of this term, I suggest it not be used with respect to injury. The term "time to be manifested," though cumbersome, will have to do until someone suggests a better one.

More specifically, injuries in many cases become manifest in the literally split second that their first perception requires. For example, motor vehicle injuries (which are typically produced even in low-speed crashes within considerably less than one-tenth of a second from the instant that the damaging forces of the energy exchange first begin to operate on the body) are manifested in about the same amount of time, or somewhat longer, that is, in the time physiologically needed for the host, or an observer, to perceive them. In other cases—the whiplash neck injury produced in many vehicle crashes and many minor musculoskeletal injuries are examples—a day or longer may be needed. Therefore, I suggest that we clean up the definition of our subject matter by defining injuries as those kinds of damage to the body produced by energy exchanges that are manifested within 48 hours, or usually within considerably shorter periods. This definition does not preclude the use of such parallel terms as bee sting and acute food poisoning, but the time it takes injuries to be manifested separates them from most infections, since with the exception of cholera and some other pathological conditions, the incubation periods for infections tend to be more than 2 days (*41*). This definition also separates injuries from conditions such as lead poisoning whose nonliving agents also require periods longer than 2 days to produce manifest pathological change. In fact, one could argue, as I have elsewhere (*42*), that it would be useful to describe as injury any damage to the body produced by any kind of a necessary and specific agent whatever its speed of action.

The techniques used by epidemiologists and others in studying the body damages produced by the several forms of energy exchange vary somewhat, as do the techniques used, for example, in studying infectious agents, heavy metals, and pathogenic fibers. Moreover, because of the rapidity of damaging energy exchanges, often devices are required that record very transient events; ionizing radiation detectors, accelerometers, and high-speed motion picture cameras are illustrative. With the high-speed motion camera, incidentally, the viewing of rapid events can be stretched out over longer periods. It is closely analogous to the microscope, which stretches the apparent size of very small objects such as bacteria. Despite differences in some of the methods used, there is no logical reason why the speed with which pathogenic agents act on the body should determine either their suitability for professional study or the priority to be given to measures for reducing the damage they produce.

Incidentally, it has long seemed to me that if the damaging interactions between injury agents and the body occurred at much slower rates—at rates as slow, for example, as those of smallpox, for which the period between infection with the virus and the end of disease communicability extends over weeks—the nature of injury processes and the opportunities for mitigating them would long since have been starkly obvious both to professionals and the general public. The high-speed motion picture camera and the slow-motion films it makes possible provide the means for such a perceptual translation as far as mechanical energy exchange injuries are concerned. In several films produced in the past decade by the Insurance Institute for Highway

Safety, this device has been used to help people understand motor vehicle injury events. First-time viewers commonly exclaim that they had never previously understood what went on in motor vehicle crashes (*43–49*).

## Strategy of Injury Control

Beginning with the work of De Haven, professional thinking about the strategy of injury control has been transformed, a process that continues. McGavran, and Barry have stressed that the successes of modern public health measures are substantially the result of a shift from an individual- to a community-centered emphasis (*7,50*). Illustrations of the latter include purifying milk and water, cleaning up smokestack emissions, and insulating lamp cords (as opposed to trying to change individual human behavior by emphasizing personal avoidance of the hazard and personal actions such as boiling water and wearing gas masks and protective gloves). Yet, although the control of infectious agents, lead, and other traditional hazards has long illustrated this shift in emphasis, the picture is mixed with respect to control of the several kinds of energy exchange injuries. At one extreme, the reduction of injuries from electricity is accomplished almost totally by community-centered, rather than individually centered, means—insulation, shielding, fuses, circuit breakers, placing high-tension wires out of reach, and, in some situations, using less hazardous voltages. At the other extreme, many people still automatically assume that the reduction of mechanical energy injuries, whether the vehicle of injury is a bullet or an automobile, calls primarily for measures directed at individuals.

With injuries, as with various other pathological conditions long since brought under control, this preference for emphasizing the individual typically results in blame being placed on the victim. As a result, it is argued that measures directed elsewhere and the idea that a responsibility exists to employ them deserve no consideration. Motor vehicle manufacturers, unwilling to provide better occupant crash protection, have used this tactic successfully for many unnecessarily bloody years (*51*). Emphasis has been on the responsibility of individuals in the general public to take steps to reduce injuries, not on the responsibility of the small number of key individuals in public and private power structures. This damagingly lopsided balance is now, however, ponderously shifting as more legal responsibility is being placed on policy-level executives to do what they can (*51*).

Emphasis on the individual's responsibility per se confuses the objective of effectively reducing society's injury problem—huge amounts of damage to people and property and all the associated costs (what a businessman would call the bottom line)—with emphasis on people as the frequent initiators of many sequences that result in injury. The same approach to poliomyelitis would still have us emphasizing the need to change children's behavior (for example, by closing swimming pools) to keep them away from the viral hazard, rather than using the more effective means, vaccination, which operates later in the causal sequence and does not require continuing behavioral change. This does not mean, however, that injury countermeasures directed at individuals have no place, but only that the same criterion—the extent of favorable influence exerted thereby on the societal problem—should govern, just as when any other emphasis is chosen.

**Active-passive distinction.** In the early 1960s, I initiated the use of the term active to categorize injury control and other public health measures that require much action on the part of individuals and the term passive to categorize those measures at the other extreme that require no individual action (*52–55*). I pointed out that "It has been the consistent experience of public health agencies concerned with the reduction of other causes of morbidity and mortality that measures which do not require the continued, active cooperation of the public are much more efficacious than those which do. Consequently, a much higher value and, hence, priority should be placed on proven measures in the 'passive' than in the 'active' area (*53*)." This concept, which has been further elaborated by Robertson (*56*) and Baker (*57*), provides one meaningful dimension for scaling all public health measures for the control of disease and injury. Its use also forces an examination of who should do what in control programs. This point is illustrated by the now more than 12 years of intense conflict in the executive, legislative, and judicial branches of the U.S. Government over whether motor vehicle manufacturers should be required to provide increased passive (more recently often labeled "automatic") crash protection (*12,51, 58–78*). The companies opposing such requirements have argued that providing such protection is no responsibility of theirs; they have emphasized instead the responsibility of the private individual, rather than of their own executives or the community as a whole (*51*).

**Haddon matrix.** I was also responsible for another aid to resource allocation analysis, strategy identification, and planning, the so-called Haddon matrix—actually a family of matrices varied to suit their uses (*12,17,79,80*). In their simplest form, these matrices have two dimensions. The first is based on the fact that all the undesirable societal end results of damaging

interactions with environmental hazards are preceded by processes that naturally divide into three stages. For the general case, I label these three stages the "pre-event," "event," and "postevent" phases; for motor vehicle collisions, my labels for the three phases are the now widely used terms "precrash," "crash," and "post-crash."

The second dimension of these matrices, in its least detailed form, is divided into the three factors "human," "vehicle" (or "vector"), and "environment," with "environment" often subdivided into "physical" and "sociocultural." A considerable literature illustrates the practical application of such matrices to the study of motor vehicle crashes and their results (*12,17,79,80*). Here, therefore, I am merely illustrating their utility when the possibilities for reducing mechanical energy exchange injuries produced by an industrial machine are under consideration.

Following is the basic Haddon matrix along with a list of examples of control measures and results of their application. The cells in the matrix are numbered to show to which phase and to which factor each of the tactical examples in the list would be assigned. Interactions between cells in the matrix, for example, the interaction in the event phase between the body and the vehicle (which in the example given is the machine) can be diagramed with arrows between cells.

| | *Factors* | | | |
|---|---|---|---|---|
| *Phases* | *Human* | *Vehicle* | *Environment* | |
| Pre-event ..... | 1 | 2 | 3 | |
| Event ..... | 4 | 5 | 6 | |
| Postevent ..... | 7 | 8 | 9 | |
| Results ..... | 10 | 11 | 12 | 13 |

In the example illustrating use of the matrix, possibilities for the reduction of injuries produced by an industrial machine are under consideration. Following is a list of possible control tactics; each number corresponds with one in the matrix.

1. Increase worker injury avoidance.
2. Shield moving parts.
3. Provide better supervision.
4. Do not hire workers with low injury thresholds.
5. Automatic machine shutoff and alarm if worker is enmeshed.
6. Alarms for others to initiate.
7. Teach workers how to respond when injured.
8. Emergency machine release (for example, to disengage hand still enmeshed after machine is turned off).
9. Provide emergency and subsequent care and rehabilitation.
10, 11, and 12. The end results after the process has ended (for example, given amounts, respectively, of damaged or disrupted people, equipment, and environmental and societal systems and in each case, the associated costs).
13. Grand total of all of the results of the damaging interaction.

Such matrices provide a means for identifying and considering, cell by cell, (*a*) prior and possible future resources allocations and activities, as well as the efficacies of each; (*b*) the relevant research and other knowledge—both that already available and that needed for the future; and (*c*) the priorities for countermeasures, judged in terms of their costs and their effects on undesirable injury results, that is, on the problems to be reduced.

**The 10 strategies.** Beginning in 1962 (*6,22,23,81*), I developed a different, and in many respects more satisfactory, analysis of strategy options, which reached its mature form as far as injuries are concerned in a paper with the title "On the Escape of Tigers" (*4*). First published by the Massachusetts Institute of Technology's Technology Review in 1970, this short piece in one form or another has been extensively republished by other journals, including the American Journal of Public Health (*82*), which presented it in an edited form as an editorial the same year.

I began the "Tigers" paper by pointing out (*4,82*):

> A major class of ecologic phenomena involves the transfer of energy in such ways and amounts, and at such rapid rates, that inanimate or animate structures are damaged. The harmful interactions with people and property of hurricanes, earthquakes, projectiles, moving vehicles, ionizing radiation, lightning, conflagrations, and the cuts and bruises of daily life illustrate this class.
>
> Several strategies, in one mix or another, are available for reducing the human and economic losses that make this class of phenomena of social concern. . . .

I then formulated 10 logically based strategies that are available to counter such damage and illustrated them with examples from the innumerable tactics used by present and past societies and individuals. In illustration, consider my comments on 1 of the 10 (*4,82*):

> The *fifth* strategy is to separate, in space or time, the energy being released from the susceptible structure, whether living or inanimate: the evacuation of the Bikini Islanders and test personnel, the use of sidewalks and the phasing of pedestrian and vehicular traffic, the elimination of vehicles and their pathways from community areas commonly used by children and adults, the use of lightning rods, and the placing of electric power lines out of reach. This strategy . . . has as its hallmark the elimination of *intersections* of energy and susceptible structure—a common and important approach.

It seemed likely that an analysis identifying the strategies generically available for reducing damage from one major group of agents—the various forms of energy—could be extended to encompass all other environmental hazards. However, I only recently got around to extending my analysis to cover them.

The result of this extension subsumes and tightens the prior "Tigers" analysis (*1*). It demonstrates, in considerable detail and with a variety of tactical illustrations from public health and a wide range of other fields, that 10 basic, generalized strategies for reducing damage (strategies derived from, but not identical to, the 10 original "Tigers" ones) encompass not only all injury reduction countermeasures, but also, literally, all measures actually or theoretically available to reduce damage to all animate and inanimate structures and systems from any and all environmental hazards (not just from necessary and specific agents) including weapons, pathogenic micro-organisms, toxins, fibers, oil spills, the various forms of energy, and even whalers and regimes that may, or do, wage war. Therefore, the resultant strategy analysis should become a basic tool both in the public health profession and in many others.

In brief, these 10 general strategies, each illustrated by 3 of its many tactical examples, are:

1. To prevent the creation of the hazard in the first place. *Examples:* prevent production of plutonium, thalidomide, LSD.
2. To reduce the amount of hazard brought into being. *Examples:* reduce speeds of vehicles, lead content of paint, mining of asbestos.
3. To prevent the release of the hazard that already exists. *Examples:* pasteuring milk, bolting or timbering mine roofs, impounding nuclear wastes.
4. To modify the rate or spatial distribution of release of the hazard from its source. *Examples:* brakes, shutoff valves, reactor control rods.
5. To separate, in time or space, the hazard and that which is to be protected. *Examples:* isolation of persons with communicable diseases, walkways over or around hazards, evacuation.
6. To separate the hazard and that which is to be protected by interposition of a material barrier. *Examples:* surgeon's gloves, containment structures, childproof poison-container closures.
7. To modify relevant basic qualities of the hazard. *Examples:* altering pharmacological agents to reduce side effects, using breakaway roadside poles, making crib slat spacings too narrow to strangle a child.
8. To make what is to be protected more resistant to damage from the hazard. *Examples:* immunization, making structures more fire- and earthquake-resistant, giving salt to workers under thermal stress.
9. To begin to counter the damage already done by the environmental hazard. *Examples:* rescuing the shipwrecked, reattaching severed limbs, extricating trapped miners.
10. To stabilize, repair, and rehabilitate the object of the damage. *Examples:* posttraumatic cosmetic surgery, physical rehabilitation, rebuilding after fires and earthquakes.

Two points, however, should be kept in mind in connection with the use of this delineation of the basic, and at least theoretically available, strategies (and also in connection with the use of the matrices of the general type described previously). First, the suggested analysis provides an aid to cognition, judgment, consideration of actual and possible control programs, and teaching; it does not provide a formula or guide for action in specific cases, each of which must necessarily be dealt with on its own at least partially unique merits. The analysis is not per se a means for choosing policy (such as whether to reduce workplace injuries), but is rather an aid for identifying, considering, and choosing the various means by which policy might be implemented.

Second, the analysis does not center on causation per se—despite the frequent usefulness of relevant knowledge of causes—but rather on the means available for reducing the specific kinds of undesirable morbidity and mortality that are of concern, which is a substantially different matter indeed. In fact, with this orientation and focus, reductions in undesirable end results can often be achieved without exhaustive knowledge of their exact causes. In illustration: pasteurization and water purification can control milk and waterborne diseases even in the absence of specific knowledge as to which pathogens would otherwise reach the publics to be protected; evacuation of residents from the vicinity of a chemical plant fire can protect them from toxic materials even though no one may have specific information as to which agents are present; and the use of guards and electrical insulation can prevent injuries regardless of the state of knowledge of the reasons people come into contact with the machines and wires that are so shielded.

## Conclusion

I have emphasized the conceptual basis of injury control because I believe the principal reasons it has been largely ignored have been the widespread lack of familiarity with the advances of recent decades and misapprehensions concerning the field. Progress in the field is, however, accelerating, and I wish that I could have reviewed some of the many important advances of recent years in methodology, research, and control, including the large decreases both in fatality rates per motor vehicle and per mile driven, before and after the fuel crisis of late 1973 and early 1974 (*12,83–86*), and the significant reduction in child poisonings (*7*). Moreover, modern ideas and findings related to injury epidemiology and control have now already been taught for several years on a few major campuses and when faculty has been available, also in a summer course in epidemiology at the University of Minnesota. These developments are both symptomatic of changes and an indication that as a public health problem, injuries may eventually command the attention from public health people that is more nearly proportionate to their prominence as the leading cause of death in the United States from the first year of life to middle age (*13,87*).

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## SYNOPSIS

HADDON, WILLIAM, Jr. (Insurance Institute for Highway Safety): *Advances in the epidemiology of injuries as a basis for public policy. Public Health Reports, Vol. 95, September–October 1980, pp. 411–421.*

Successful injury control measures (stoplights, sprinkler systems, electrical insulation, evacuation) have long been commonplace. However, progress in injury control has been hampered by the failure to recognize that injuries cannot occur without the action of specific agents analogous to those of the infectious diseases and likewise transmitted by vehicles and vectors. These agents are the several forms of injury. Varying and interacting with the characteristics of the host and the environment, they constitute the classic epidemiologic triads that determine injury distributions, none of which are random.

The injury-disease dichotomy, a universal in most of the world's major languages, may have resulted from the fact that at least some of the causes of injuries (for example, wild animals or falling trees) are more identifiable and proximate than the causes of diseases. The etiology of injuries suggests that for epidemiologic and public health purposes, the term injury should probably be defined so as to encompass those kinds of damage to the body that are produced by energy exchanges and that are manifested within 48 hours, or usually within considerably shorter periods.

Strategies for injury control can be extended to the control of other pathological conditions. The active-passive distinction (the dimension expressing the extent to which control measures require people to do something) has a direct bearing on the success of public health programs, because passive approaches have historically had a far better record of success than active ones. Ten basic strategies have been identified that provide options for reducing the damage to people (and property) caused by all kinds of environmental hazards.