



INJURY EPIDEMIOLOGY

RESEARCH AND CONTROL STRATEGIES

Third Edition

Leon S. Robertson

Injury Epidemiology

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Research and Control Strategies

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Leon S. Robertson

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Preface

This third edition of *Injury Epidemiology* includes updated data and discussion regarding the use and misuse of epidemiological methods to research injury and evaluate injury control efforts. Unfortunately, too many articles and books that rely on invalid data and logic continue to appear. Some reviewers of earlier editions considered criticism of certain studies or organizations harsh, while one accused the author of favoring the U.S. auto industry, despite numerous criticisms of some of the actions and products of U.S.-based manufacturers. In fact, the products of many corporations are unnecessarily hazardous. Various government agencies have failed to act or enforce laws and standards. Detailed illustrations and critiques of product evaluations, laws, and standards are included in appendices at the end of chapters 8, 9, 10, 12, 13, and 15.

Reluctance to criticize invites perpetuation of these practices. If injury control is to reach its full potential, we need more, not less, harsh criticism of researchers and organizations that do not do their jobs. Our profession, like many others, suffers from “pronoia,” the aversion to criticism of others’ work, giving them a false sense of accomplishment and self-importance (Goldner, 1982).

A major problem documented in the book is conflicting results in studies that rely on self-reports of injury and behavior. These are similar to conflicting results found in research on the health effects of certain foods, which has become a laughing stock (Shermer, 2007). It is time to stop conducting such shaky “research” before injury research meets a similar fate.

Although I have not reviewed every book, article, or Web site that may be useful to the topics discussed here, new references and links to Web sites have been added to this third edition. Some references are old but just as valid as the day they were written. Too many injury researchers apparently have no knowledge of the older literature, as indicated by their failure to reference it and, in some cases, their repetition of long-known errors.

Some references and other materials in textbooks and articles have always been a year or more out of date by the time they are edited and published. While the principles discussed here are as sound today as when they were first explicated, the data and literature on injuries change daily. To supplement the information in this text, links to data sources and other information available on the Internet will be available at <http://www.nanlee.net>. They will be updated as new information becomes available.

The Internet now allows access to large amounts of information and larger amounts of misinformation. My intention is to maximize the former and minimize the latter. Those who find errors or wish to suggest material to be added may do so by writing me at nanlee252000@yahoo.com. If you send me advertisements, viruses, or other useless or harmful stuff, your e-mail address will become a permanent resident of the junk file, never to be viewed again.

L.S.R.

Green Valley, Ariz.

September 2006

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Shermer M (2007) Eat, drink and be merry. *Scientific American* 296:29.

Preface to the Second Edition

Injury Epidemiology, the first edition of this book, considered implications for injury control. The subtitle of the second edition, *Research and Control Strategies*, reflects this emphasis. Many new sections and references on those subjects have been added.

While the reception of the first edition by students and reviewers was mostly favorable, some readers were apparently confused or misled by the cryptic treatment given certain topics. Where this was made known to me, the sections have been rewritten or augmented in a quest for clarity. Some reviewers considered my criticism of certain studies or organizations harsh, and one accused me of favoring the U.S. auto industry despite numerous criticisms of some of the actions and products of U.S.-based manufacturers. The sad facts are that some researchers produce invalid or misleading research reports, the products of many corporations are unnecessarily hazardous, and government agencies fail to act or enforce laws and standards. Reluctance to criticize invites continuation of these practices and failures. If injury control is to reach its full potential, we need not less, but more, harsh criticism of researchers and organizations that do not do their jobs.

Epidemiologic studies of injuries have increased more rapidly in quantity than in quality in the 1990s. The collection of data with no clear research question or potentially useful results remains all too common. The revised first chapter of this book delineates specific objectives of epidemiologic studies and the data and study designs needed to attain those objectives. Each subsequent chapter has been revised and expanded with these objectives in mind to provide a more progressive step-by-step development of topics.

New analyses and updates include details on potential years of life lost from injuries, cardiovascular diseases, and cancers; effective uses of surveillance systems; clarification of the relevance of causal thinking for prevention; the misuse

of behavioral inference in cases such as the C/K pickup truck fire issue; recent studies of legal intervention; the effects of motor vehicle crashworthiness relative to other factors; questions for research in prevention and rehabilitation; illustrations of cost–benefit, risk–benefit, and cost–savings analyses and evidence of confounding of estimates of effects of alcohol and seat belt use; and the effect of regulatory failure on rollovers of top-heavy vehicles and fuel economy as well as alternatives to regulation.

A number of new books that are worthwhile additions to class reading lists have appeared since the first edition was published. They are listed in the references for relevant chapters. To my knowledge, this remains the only textbook setting out general principles for research in injury epidemiology and evaluation of injury control. Good links to Internet sites are found at <http://www.injurycontrol.com>.

L.S.R.

Branford, Conn.

May 1997

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Injury Epidemiology

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Injury and the Role of Epidemiology

For decades, injury deaths from motor vehicles, homicides, suicides, falls, poisonings, and drownings were listed in mortality statistics and largely ignored by researchers in epidemiology and public health. Near the end of the twentieth century, influential and important discussions of the future direction of epidemiological and public health research made no mention of injury (Susser and Susser, 1996; Pearce, 1996). A conference held in 2005 on the future of public health included one brief mention of injury, but more was said about Elvis impersonators than about injury prevention (University of the Sciences in Philadelphia, 2005).

Only occasionally does the issue gain some prominence. Addressing deaths among the young and a significant proportion of health care costs, the executive director of the American Public Health Association wrote “The Solution Is Injury Prevention” (Benjamin, 2004). Nevertheless, injury remains a hugely neglected public health problem.

That is not to say that injury prevention should be left exclusively to public health agencies. Epidemiology and public health bring a perspective on prevention honed from major victories in the control of infectious diseases. As was the case in those triumphs, the implementation of injury prevention requires the involvement of a wide variety of government agencies, businesses, health care providers, private voluntary organizations, and the research community. To implement an injury control agenda effectively and efficiently, the leadership and implementers in these entities must understand the epidemiological and public health perspective (Bonnie and Guyer, 2002).

Lists of numbers of deaths by type can be misleading regarding their relative importance for individual or societal well-being. Neither preventive measures nor medical treatment “saves lives,” as is often claimed. If prevention or treatment is effective, it may delay death for a period of time, but everyone eventually dies. An accurate assessment of deaths and the effects of prevention or treatment on

deaths would indicate potential years of life lost or preserved and years of disability avoided. Such statistics are rarely seen in medical and public health literature.

In 1985, the Committee on Trauma Research appointed by the National Research Council/Institute of Medicine published the report *Injury in America: A Continuing Public Health Problem*. The report summarized the magnitude of the problem:

Each year, more than 140,000 Americans die from injuries, and one person in three suffers a nonfatal injury. ... Injuries kill more Americans aged 1–34 than all diseases combined, and they are the leading cause of deaths up to the age of 44. Injuries cause the loss of more working years of life than all forms of heart disease and cancer combined. One of every eight hospital beds is occupied by an injured patient. Every year, more than 80,000 people in the United States join the ranks of those with unnecessary, but permanently disabling, injury of the brain or spinal cord.

Despite reductions in the rates of fatalities and hospitalizations per population, the absolute numbers increased in the ensuing 15 years in relation to population growth. In 2000, about 149,000 U.S. residents died from injury and more than 1.8 million were hospitalized at an estimated lifetime cost of \$80.2 billion in medical care costs and \$326 billion in lost productivity (Finkelstein et al., 2006).

Although major new concerns about diseases such as HIV/AIDS and diabetes grab attention, injury takes about 10 times the potential years of life as each of these in the United States. The Centers for Disease Control and Prevention (CDC) separates homicide and suicide from unintentional injury, but when they are combined, injury is the leading cause of potential years of life lost through age 70. (For the latest numbers as well as state and regional data, see <http://webapp.cdc.gov/sasweb/ncipc/ypl110.html>, accessed August 2006.) Internationally, injury is a large proportion of the loss of life and disability in low-income countries but is seldom considered in international aid programs (Mock et al., 2004).

The 1985 Committee on Trauma Research report also pointed to the fact that injuries are highly patterned and hence subject to study and targeting of interventions, that many interventions are known to be effective but are unused, and that modest increases in funding would have large payoffs in cost savings. Eight years after the report recommended that a Center for Injury Control be established at the CDC, it was established in 1993. Although the number of professionals doing studies, teaching, and implementing injury prevention projects has grown modestly, the needs both in quantity and quality remain large. A study of 82 medical schools in 31 countries found large gaps in the injury topics addressed (Villaveces et al., 2005).

Should injuries have a higher priority? Reduction of injuries is justifiable on humane grounds, particularly since they disproportionately affect the health of the young. In a time of concern for health care costs, injury reduction is also an economic necessity. Injury control is one of the most promising ways to reduce health care costs in the immediate future. While attention to diet, increased exercise, and prevention of smoking among the young is worthwhile, the health benefits are usually not realized for decades. An injury prevented or reduced in severity has immediate benefits in reduced costs as well as grief, pain, and suffering.

Despite declines, motor vehicle injuries are the leading cause of injury deaths, while falls are the leading cause of injury hospitalizations. Firearms are the second

leading cause of injury deaths in the United States but not in countries where guns are not prevalent.

This chapter lays out some of the important concepts in injury epidemiology and prevention: the distinction between accident and injury, the application of the epidemiological model for infectious diseases to injuries, and the phases of injury in relation to the factors that contribute to incidence and severity.

ACCIDENTS AND INJURIES

One aspect of injury addressed in this book is the importance of measuring severity when investigating injuries or considering injury control efforts. In the United States, motor vehicles, firearms, falls, poisonings, fire/burns, and drownings account for about 77 percent of deaths but only 36 percent of nonhospitalized injuries severe enough that their sufferers seek medical treatment (Finkelstein et al., 2006). Clearly, emphasis on prevention of all “accidents” will lead to misdirection of effort from the more serious and costly cases.

The evolution in thought about injuries is reflected in how they have been classified. Injuries often are classified as accidental or intentional. Prior to the 1960s, injury control was primarily focused on “accident prevention,” and interpersonal or self-directed violence was largely left to law enforcement, psychiatry, social workers, and the clergy. That is not to say that these professions have not contributed to injury control or that all attempts to prevent “accidents” have been unsuccessful, but the extent of scientific investigation of the effectiveness of these approaches was very limited.

“Accidents” refer to a very large and fuzzy set of events, only a small proportion of which are injurious. Any unintended, incidental event that interferes in one's daily pursuits is an accident. In writing these few paragraphs, the author has had several accidents in typing, but ideally they have been corrected enough so as not to irritate the reader and thus have become irrelevant to exposure of the author to risk of injury.

The word “accident” is also intertwined with the notion that some human error or behavior is responsible for most injuries. This focus of attention on the human actors involved tends to detract from an examination of the full range of factors that contribute to injuries and, particularly, their severity.

Although the word “accident” had various meanings historically (Loimer et al., 1996), it is now primarily a euphemism for lack of intent, as though intent were a primary consideration in injury prevention. If two people have an argument that results in a brief exchange of fisticuffs, the incident usually goes unrecorded as an injury. In a similar situation, if one of the persons has a gun and kills the other with it, the case is classified as homicide as though the person intended to kill, which is often not true. While criminal law has various categories of homicide based on intent, aggregated statistics of the broad category “homicide” are included in “intentional injuries” without any data on the intent of the persons who used the weapon.

Even in the case of suicide, intent can be questioned. Some supposedly suicidal acts are attempts to get attention rather than serious attempts at self-destruction.

But if the attention-getter makes a mistake and dies, he or she will be classified as a suicide (Maris et al., 2000).

Here the term “injury” or reference to specific types of injury—amputations, burns, lacerations—indicates the phenomena of interest. Also, when referring to attempts to reduce injury, the term “injury control” is used. While “injury prevention” has been used by respectable scientists—and on occasion even by the author—and is the name of a reputable journal (<http://ip.bmjournals.com/>, accessed August 2006), the term is less precise than “injury control” when severity of injury can be reduced without reducing incidence.

EPIDEMIOLOGY

The word “epidemiology” is a derivation of the classical Greek word *epidemion*, a verb meaning “to visit,” used in connection with human maladies by Hippocrates circa 2400 B.C. The first known published use of the word in modern languages was in 1598, the Spanish *epidemiologia*, in a study of bubonic plague in Spain (Najera, 1988).

There are all sorts of definitions of epidemiology. The scientific study of the visitations of disease and injury on the population is as good as any. The scientific study of the distributions of diseases and injuries in populations, and their causes and “risk factors,” is somewhat more descriptive of what epidemiologists do.

Historically, early epidemiologists concentrated their attention to what were later called “infectious diseases.” Although the specific organisms that often infected populations were microscopic and unknown, quantification of the numbers of illnesses and deaths by location or exposure to certain environmental conditions led to actions that reduced disease and death in some instances. In nineteenth-century London, John Snow found that cholera occurred largely among people who used one water supply and not among those who used another. He did not know what was in the water that caused the disease, but he knew enough to stop the flow from that supply (Evans, 1993).

Similarly, when there are known effective means of reducing injury or severity, relatively simple studies of when, where, and how people are injured can lead to large reductions by targeting the relevant injury control measures to the circumstances to which they apply (chapter 7). This approach avoids the question of causation that is a point of both fascination and contention among epidemiologists as well as other scientists.

Some epidemiologists avoid the word “cause” and use euphemisms such as “risk factor” and “etiology.” Often the incidence and severity of disease and injury are coincident with several factors, some of which may contribute to the incidence or severity and some of which may be correlates of the real causes but play no meaningful role in incidence or severity. For example, if a disease or injury is seasonal, as many are, increase in incidence may be correlated with other seasonal happenings. In northern areas of the United States, the vast majority of injuries to people while riding motorcycles occur during the season that robins are in the environment, but the correlation is known to be spurious. Few, if any, motorcyclists

crash when impacting robins. The correlation occurs because both robins and motorcyclists prefer warm weather for their activities.

Since correlations thought to represent causal connections occasionally turn out to be false inferences, the embarrassment to the scientists proved wrong leads other scientists to great caution in causal inference. When dealing with maladies that kill or maim, however, one must sometimes risk embarrassment to contribute to the welfare of fellow human beings. At some point, as evidence accumulates, an attempt to change conditions coincident with the disease that would likely reduce it is appropriate. The amount, type, and quality of the evidence necessary to reach that point are matters of controversy addressed in this book, particularly in chapter 8.

A few epidemiologists believe that objective scientific investigation is incompatible with recommendations for changes in policies or practices that epidemiological research suggests would reduce harm. Some journal editors do not even allow discussions of policy implications of research results in articles reporting the results, much less recommendations for policy changes. Two important points are ignored by those stances: (1) The work of the scientist who proposes change based on research is likely to be scrutinized more carefully for bias and error. To the extent that such scrutiny weeds out invalid results, the science is improved. (2) Self-imposed or institutional bars from the policy debate of the scientists closest to the data increase the probability of misinterpretation or misuse of the data. Therefore, this book is devoted not just to the application of the theory and methods of epidemiology in injury research, but also to the uses and misuses of epidemiological data relevant to injury control.

Epidemiological methods are also applied to the study of the effectiveness of policies and practices aimed at the reduction of injury incidence and severity. Although this is called “applied” research and somehow has less prestige than “basic” research, the distinction is false. The study of human activities and other phenomena that increase the risk of injury is no more “basic” than the study of the human activities that seek to intervene in the causal process to reduce injury. Indeed, reduction of injury (or disease) by deliberately changing a factor inferred as a condition necessary for, or contributing to, specific injuries is additional evidence of causation, particularly if the change is introduced in such a way as to rule out the contribution of other factors to the reduction. Chapters 11–14 discuss the use of scientific methods to evaluate the effects of programs, laws, environmental modifications, medical care, and rehabilitation to reduce injuries or their severity.

Of course, disciplines other than epidemiology also study injury. Clinicians often describe a case or series of cases involving injury, and sciences ranging from physics to biomechanics contribute to our understanding of the mechanisms of injury. The research methods used by epidemiologists are shared by many disciplines. Statisticians, sociologists, psychologists, and physical and biological scientists use more or less the same methods. Many epidemiologists were originally trained in those disciplines as well as medicine. Nevertheless, the concepts of epidemiology provide a valuable perspective, particularly with respect to reduction of disease and injury in the population as a whole or in subsets of the population.

THE EPIDEMIOLOGICAL MODEL

Based on the experience of their predecessors in the scientific investigation of infectious diseases, many injury epidemiologists conduct their investigations mindful of a theoretical model developed by infectious disease epidemiologists. The core concepts of this model include the host (the person injured), the agent that injures, and the vector or vehicle that may convey the agent, as well as other environmental factors.

Early epidemiological investigations of what came to be known as infectious diseases showed correlations of the diseases to seasons, water sources, economic status of the populations primarily affected, and the like (Buck et al., 1988). We now know that in some cases these correlates of the diseases were carriers (vehicles or vectors) of infectious agents, and in others they were factors that increased or decreased host exposure or susceptibility to the agents—and some were spurious.

Microbiologists subsequently identified tiny biological structures (bacteria, parasites, viruses) that secreted toxins in an invaded host, or removed elements from the host, or caused other changes at the cellular or organ levels, that resulted in sickness and death. Then epidemiologists knew to look for these agents in the seasons, water, living conditions, and so on, associated with a given disease (Lilienfeld and Lilienfeld, 1980).

In some cases, the microorganism was conveyed to the host by inanimate media, such as water and milk, which came to be called vehicles. Others were carried to human hosts by insects or by animals or were directly transmitted from human being to human being. These animate carriers came to be called vectors. Living conditions, often related to economic status, increased or decreased exposure to the carriers of the agents or increased susceptibility to infection due to nutritional or other factors.

All of these discoveries had implications for control of infectious diseases. The agents could be eliminated from the media in which they reached hosts such as water and milk. In some cases, susceptibility could be reduced by modification of the immune mechanisms of the potential hosts. In others, antibiotic agents could be introduced into the infected hosts to reduce the severity of the illness. Elimination or control of certain carriers, such as rodents and insects, could be tried and, in some cases, accomplished. While diseases sometimes declined as people improved their living conditions, removing harmful agents or carriers in the process, epidemiological evidence on vehicles or vectors, as well as times, places, and populations involved, was crucial in the deliberate attempts to reduce many infectious diseases.

Although injuries can be characterized using the concepts of infectious disease epidemiology, injury epidemiology lagged in development by decades. The twentieth century was almost two-thirds past before the agents of injury were accurately identified as the various forms of energy: mechanical, thermal, chemical, electrical, ionizing radiation—or too little energy in the case of asphyxiation (Gibson, 1961). And that identification came from a psychologist, not an epidemiologist. Before and since that date, certain authors referred to motor vehicles, guns, and alcohol as agents of injury, but that is inaccurate in the epidemiological

use of the concept of agent. Motor vehicles and guns are vehicles of mechanical energy in epidemiological parlance, and alcohol contributes to injury by sometimes affecting behavior that places people at greater risk of injurious energy exposure as well as perhaps increasing vulnerability of tissues to energy insults.

Prior to these insights, the research on injury was primarily focused on human characteristics and human behavior correlated with injury incidence and, more rarely, severity—with occasional studies of seasonal and geographical variations and the like. A few isolated researchers looked at human tolerance of mechanical energy as important (DeHaven, 1941; Haddon et al., 1964; Stapp, 1957).

It is not that the characteristics of the energy were unknown. The leading source of injury by far is mechanical energy, the characteristics of which have been known since Sir Isaac Newton's work on the laws of motion in the seventeenth century. Although Newton's laws of motion do not apply near the speed of light, they are applicable to moving motor vehicles, bullets, and falling human beings.

Why did it take so long to recognize energy as the agent of injury? It is more difficult to prove why something does not happen than why it does. After the great decline in death due to infectious diseases in the first half of the twentieth century, many epidemiologists turned their attention to cardiovascular diseases and cancers, at least partly due to the increases in government support of research on those diseases. Popular folklore focused on human behavior or human error as the cause of "accidents," and the small amounts of research funds available were devoted to reinforcement of that view.

Manufacturers of motor vehicles—by the 1920s the leading source of mechanical energy leading to death—deliberately supported the behavioral approach to divert attention from their vehicles (Eastman, 1984), a tradition that continues (Robertson, 1997a, 1997b; see also <http://www.nanlee.net/evans>, accessed August 2006). The National Rifle Association, the leading opponent of regulation of guns, coined the slogan, "Guns don't kill people. People kill people." Mosquitoes don't have lobbyists, but motor vehicles and guns do.

FACTORS AND PHASES OF INJURY

The transfer of energy to human beings at rates and in amounts above or below the tolerance of human tissue is the necessary and specific cause of injury. The amount of the energy concentration outside the bands of tolerance of tissue determines the severity of the injury.

Injury usually refers to the damage to cells and organs from energy exposures that have relatively sudden, discernible effects, although some researchers have included damage from chronic low-energy exposures, such as back strain or carpal tunnel syndrome, as injury (Waller, 1985). Chemical and radiation exposures that produce cellular changes resulting in neoplasm are usually called cancerous rather than injurious. A debate regarding the appropriateness of inclusion or exclusion of any harmful condition as injury or disease would be pointless, but at the fuzzy edges of a set of harmful consequences from energy exchanges, a given researcher should make clear the cases that are considered injuries.

Most of the concentrations of energy involved in severe and fatal injuries are the result of human inventions that alter the energy inherent in matter (Robertson, 1983). Some falls occur from heights unmodified by human construction. Lightning, tornadoes, and hurricanes kill a few hundred people annually. But motor vehicles and guns, as well as cigarettes, which cause more house fires than any other ignition source, and home swimming pools, which drown more children than other bodies of water, are examples of human inventions that are some of the major sources of serious injuries.

To alert researchers to the factors contributing to injury incidence and severity, and the timing of involvement of those factors, Haddon (1972) devised a matrix of broad categories of factors and phases of injury. This matrix, along with some examples of factors known to be important in each category, is shown in table 1-1.

Before injury, human, vehicle, and environmental factors contribute to the increase or decrease in exposure to potentially damaging energy. For example, alcohol impairs human perceptions, reactions, and judgments and may increase aggressive behavior. The weights of heavier vehicles extend stopping distances. Hedges at intersections reduce visibility of oncoming traffic.

During the energy exchange that injures, the susceptibility of the host's tissue to damage may vary. For example, a person with osteoporosis may be disabled by a fall that would hardly bruise a teenager. Sharp points and edges concentrate energy on the host. Flammable building materials increase intensity of heat and smoke in fires.

After the initial energy exchange, the condition of the host, the potential for more energy exposure, and the responses from the environment substantially affect survival and the time and extent of return to preinjury functioning of those who survive. For example, if hemorrhaging is not stopped, the host may die from loss of blood. If a gas tank is ruptured by the initial energy exchange, a spark can result in thermal energy beyond host tolerance. If emergency response is delayed, the remainder of a life that could be preserved by surgery or other intensive treatment may be lost.

Each epidemiological investigation does not have to measure all of the human, vehicle, and environmental factors at various phases to be useful, but in those cases where there is a synergistic effect of particular factors on the outcome being studied, failure to consider them can be misleading. One crucial consideration in the relevance of epidemiology for injury control is the modifiability of the factors measured.

Table 1-1. The Haddon Matrix, With Examples

Phases	Factors		
	Human	Vehicle	Environment
Preinjury	1. Alcohol intoxication	4. Braking capacity of motor vehicles	7. Visibility of hazards
Injury	2. Resistance to energy insults	5. Sharp or pointed edges and surfaces	8. Flammable building materials
Postinjury	3. Hemorrhage	6. Rapidity of energy reduction response	9. Emergency medical response

For example, a large number of injury investigations are limited to the distributions of rates of a particular type of injury by age and gender. It is important to know how many potential years of life are affected by injury and whether subsets of the population are disproportionately involved, but other than those considerations, age and gender distributions are uninteresting. We are not going to change the age and gender of individuals in the population.

The major modifiable factors that contribute to injury are energy and the characteristics of vehicles of energy. Human behavior that increases exposure or concentrates energy can be modified under certain conditions, but not by changing immutable factors such as age and gender (Robertson, 1983). Human tolerance to energy exposures varies among individuals by age, gender, and other factors that affect the condition of tissue, such as decalcification of bones and hemophilia. To the extent that diseases that increase susceptibility can be treated, the severity of injury can be reduced by that method, but there are substantial limits to increasing tolerance to energy.

The importance of focus on degree and type of energy exposures was most dramatically illustrated to the author while conducting a study of worker injuries. In two adjacent buildings of the same company, the injuries to the workers were remarkably different. In building A, there were virtually no injuries to the workers that were severe enough to be reported on the log required by the Occupational Safety and Health Administration (OSHA) during the eight-year period studied. In building B, about one in five workers had an OSHA-reportable injury each year. There were some age, gender, and other differences between the work forces in the two buildings, but these accounted for only a small amount of the variation in injuries among the workers in building B (Robertson and Keeve, 1983), and there was essentially no variation in injuries while at work among people in building A to investigate.

The major difference between the two buildings was the exposure to energy. In building B, some of the workers poured molten metal, heated to thousands of degrees, from large vats. Others worked adjacent to machines that rolled, shaped, and stamped the metal—forming it into wire, rods, and keys. A fall or a movement of any sort in the wrong direction would place one or another part of a worker's anatomy in contact with thermal or mechanical energy beyond the tolerance of human tissue. The people in building A also worked with machines—typewriters, photocopying machines, and computers—that contained potentially injurious electrical energy. But the manufacturers of those machines had the foresight to shield the energy from the workers so that the potential for contact with the energy was minimal.

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Energy Characteristics and Control Strategies

Injury epidemiologists should have at least a rudimentary understanding of the forms of energy involved in injury and the tolerance of human beings to exposure to energy. Since incidence and severity of injury are usually classified by vehicles or circumstances rather than by agent, the precise numbers attributable to a given agent are not available in many data sets. At the turn of the century in the United States, about 58 percent of fatal injuries and 62 percent of hospitalized injuries were attributed to motor vehicles, falls, and firearms (Finkelstein et al., 2006). These injuries, and a proportion of those injuries associated with factors and activities grouped into the “other” category (industrial machines, farm machines, knives, sports), indicate that mechanical energy accounts for the substantial majority of severe injury. As of 2002, each year about 1 in 110 U.S. residents was discharged from a hospital after an injury and about one in seven people in the U.S. population visited a hospital emergency room, mostly with injury from mechanical energy (see http://www.cdc.gov/nchs/about/otheract/injury/injury_hospital.htm, accessed August 2006).

Some energy releases, such as a nuclear explosion, include multiple forms of energy—mechanical, heat, chemical, and ionizing radiation. The acute effects of ionizing radiation have been rare and are not discussed in the following brief descriptions of common types of energy that cause the bulk of acute injuries.

MECHANICAL ENERGY

Any object, animate or inanimate, in motion at velocity substantially less than the speed of light has energy in relation to its mass and speed that is described by a simple formula:

$$K = mv^2/2 \tag{2.1}$$

where

K = energy in foot-pounds

m = mass

v = velocity in feet per second

A foot-pound is the energy needed to raise a pound of material one foot from the ground at the earth's surface. Mass can be calculated at the earth's surface by dividing weight in pounds by 32. Velocity in miles per hour can be converted to feet per second by multiplying miles per hour by 1.467. Therefore, a 150-pound motor vehicle occupant traveling at 30 miles per hour has about 4,539 foot-pounds of energy, that is,

$$[(150/32) \times 44^2]/2 = 4539 \text{ foot-pounds}$$

Increase in mass or speed increases the energy, speed obviously more so than mass because it is squared in the calculation. At 60 miles per hour, the 150-pound vehicle occupant has about 18,158 foot-pounds of energy, twice the speed but about four times the energy compared with 30 miles per hour.

If the person must stop suddenly, as in a crash of the vehicle, that energy must be dissipated in the vehicle, in the environment, or in the tissues of the individual. When the vehicle stops, the occupant will continue to move at the precrash speed and collide with the interior structures or, if ejected, with materials in the exterior environment—the so-called second collision. The load on the tissue is measured in pounds per square inch. The shape and elasticity of the materials struck will determine the damage to the tissue. Inflexible, protruding or pointed objects on dashboards, for example, will penetrate the heads or other parts of the anatomy of people who move into them in crashes at common traveling speeds.

Devices such as child restraints, lap/shoulder belts, and air bags reduce the severity of injury by reducing contact with less flexible surfaces. They also increase the uniformity of deceleration of occupant and vehicle and spread the load over dozens of square inches. Energy-absorbing materials, both in the vehicle and the environment, can also dissipate energy so that the individual's deceleration is less rapid (Nahum and Melvin, 2002). Helmets used by motorcyclists, bicyclists, and in certain sports, as well as padding used in certain sports and on the hips of residents in certain nursing homes, also absorb energy if designed to do so.

The extent of damage to tissue is a function of the structure of the part of the body affected. Contact with an energy source generates forces counter to the load, called stresses. These constitute the resistance to deformation of the bonds among tissue molecules. The same tissue may have different capacity for tension stress (pulling molecules apart), compression stress (pushing molecules together), or shear stress (tearing from a tangential force).

Strain refers to the extent of deformation and may be classified as resulting from tension, compression, or shear stress. Tissues vary in elasticity—the extent to which strain is eliminated when the load is removed—and in the regenerative capability of the organism (Stephenson, 1952). Nerves do not regenerate when severed, so injuries to the brain and spinal cord are especially disabling