EIGHTH EDITION

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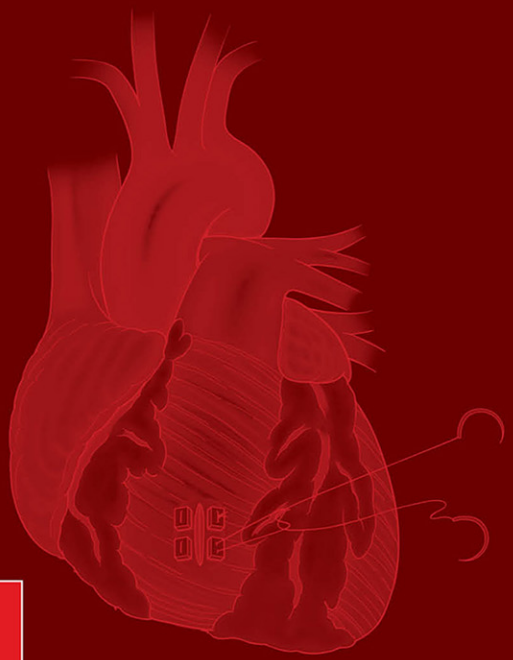

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Eighth Edition

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Mc Graw Hill Education

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Milan New Delhi Singapore Sydney Toronto

ISBN: 978-1-26-012860-4
MHID: 1-26-012860-1

The material in this eBook also appears in the print version of this title: ISBN: 978-0-07-184729-2,
MHID: 0-07-184729-4.

eBook conversion by codeMantra
Version 1.0

To obtain material from the disk that accompanies the printed version of this eBook, please **click here**.

The editors of Trauma, Eighth Edition, gratefully dedicate this edition to our five unique "families":
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Grace S. Rozycki, MD, MBA, David J. Feliciano, Douglas D. Feliciano, JD (DVF);
June Mattox, Kimberly, Dan, Charles, Alex, and Kelsey Toth (KLM);
our trainees, who now dot the globe—our lasting legacy;
our medical schools and academic anchors; our organizations and associations;
our patients, who continue to teach us so much; and our administrative assistants:
Jo Fields (EEM), Karen Lynn and Victoria Dodge (DVF), and Mary Allen (KLM).

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PREFACE

The Eighth Edition of *Trauma* spans more than three decades of development, implementation, and maturation of trauma as an academic discipline. While the term "acute care surgery" has emerged recently, trauma surgeons have always been the go-to surgeon for emergent care, and trauma remains the core. We are very fortunate to have served as editors throughout this period in history, and truly represent the first generation of trauma surgeons in the United States. We experienced the golden age of trauma surgery, during an era in which we did it all: visceral and vascular, torso and extremities. At the outset, virtually all seriously injured patients underwent operative management, primarily based on clinical assessment with the aid of plain x-rays and the venerable diagnostic peritoneal lavage (DPL). Decisions were relatively straightforward since few alternatives existed, and few had the courage to challenge our behavior. By contrast, today the emphasis is on avoiding an operation, and multiple disciplines are involved in the decision making. There is no lack of oversight, monitoring, and data reporting. But the unquestionable benefactor has been the patient, who now survives devastating injuries once considered uniformly lethal. This edition may be the last for us as editors, because we have always believed that to be effective, we must remain active in the trenches to understand the importance of new concepts. While we are all very active in trauma care today, all good things must come to an end.

In the Eighth Edition of *Trauma*, as in the previous editions, we have changed approximately one-third of the authors to ensure the most current knowledge in all topics. In addition, we have expanded our Trauma Atlas, which is designed to provide a quick reference when performing procedures in the ED, OR, or SICU. We are pleased to include a new Trauma Video section, which provides an extensive compilation of technical procedures for the trauma surgeon.

Finally, the editors acknowledge the invaluable assistance of many individuals who have made the Eighth Edition a reality. We are extremely grateful to the authors who have sacrificed their valuable time to share their experience, knowledge, and expertise. The Trauma Video section was generously provided by Demetrios Demetriades and Kenji Inaba, who clearly have seen it all at USC/LA County. Mike de la Flor was persistent and patient in rendering accuracy in the Trauma Atlas. The professional support of McGraw-Hill Education was essential at all levels of publishing; we want to specifically thank Brian Belval, Executive Editor of the Medical Division, and Christie Naglieri, Senior Project Development Editor. And of course, we want to especially recognize the tremendous work of our respective Administrative Assistants: Jo Fields (EEM), Karen Lynn and Victoria Dodge (DVF), and Mary Allen (KLM).

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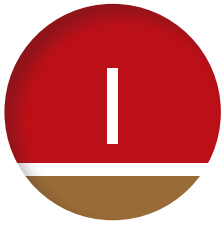

TRAUMA OVERVIEW

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1 Kinematics

Alan B. Marr • Lance E. Stuke • Patrick Greiffenstein

Kinematics (*kn-mtks*) *n*: The science of pure motion, considered without reference to the matter or objects moved or to the force producing or changing the motion. From the Greek κίνημα, κῑνηματ- a motion (κῑνεῖν to move) + -ic suffix.[1]

All injury is related to the interaction of the host and a moving object. That object may be commonplace and tangible, such as a moving vehicle or speeding bullet or more subtle as in the case of the moving particles and molecules involved in injury from heat, blasts, and ionizing radiation. Studying kinematics in relation to trauma uses Newtonian mechanics, the basic laws of physics, and the anatomic and material properties of the human body to explain many of the injuries and injury patterns seen in blunt and penetrating trauma. Injury is related to the energy of the injuring element and the interaction between that element and the victim. Although most patients suffer a unique constellation of injuries with each incident, there are quite definable and understandable energy transfer patterns that result in certain predictable and specific injuries. Knowing the details of a traumatic event may lead the treating physician to further diagnostic efforts to uncover occult but predictable injuries.

This chapter has been organized in a stepwise fashion. First, the basic laws of physics and materials that dictate the interaction between the victim and the injuring element are reviewed. This is followed by a more detailed examination of penetrating and blunt trauma with an effort to dispel some of the common myths about these injury mechanisms. Finally, a synopsis of mechanisms specific to organs and body regions is examined. It is hoped that this will offer the reader a better understanding of specific injury patterns, how they occur, and which injuries may result.

BASIC PRINCIPLES

The goal of studying kinematics in trauma is to help us understand how injuries occur. Understanding the biomechanics of injury may help us prevent and treat these injuries in order to optimize outcomes. It is tempting to believe in the finiteness of the understanding of physics and biomechanics, the sense that all there is to know is already known; however, ever-improving technology is making the experimental study and computer modeling of such phenomena more effective. Therefore, continual reassessment is critical in order to continue to maintain relevance in an ever-changing world. Nevertheless, much of the basis of current understanding has been laid down by the great minds of the past whose insight and understanding, though it might have come from rather humble or mundane observances, has absolute relevance as we examine biomechanics today.

James Prescott Joule, a 19th century English brewer and amateur physicist seeking to optimize the energy needs of his brewing operations, stumbled upon what is now known as the first law of thermodynamics or the law of conservation of energy. It states that, in a closed system, energy can be neither created nor destroyed, only transformed from one state to another.[2] This is in line with *Newton's first law*, which states that an object in motion or at rest will tend to remain in this state unless acted upon by an external force. Thus, kinetic energy, or the energy of motion will be conserved until it is transformed by an external force. When this transformation occurs in the form of transference of energy from one object to another, it can lead to alteration of one or both objects. This is the fundamental principle of traumatic injury.

In order to understand this principle, one must first consider the basic principles of physics. One can divide these principles into two broad groups as follows: principles that describe motion of objects and their interactions, and those that describe the effects of these interactions on the objects themselves. The key principles that describe the former are force, momentum, and impulse. The key elements that describe the latter are stress, strain, and elasticity. First, let us consider momentum (p), which is defined as the product of mass (m) of an object and its speed or velocity (v).

$$p = mv$$

Intuitively, we understand that in order to change an object's momentum, we must typically introduce a force, which will cause the object to either speed up or slow down. When a force causes a change in momentum, it is referred to as impulse. This is a bidirectional exchange, however, where

a force causes a change in momentum and, concomitantly, a change in momentum will generate a force.[3]

Newton's second law builds on the first and further defines a force (**F**) to be equal to the product of the mass (*m*) and acceleration (*a*).

$$\mathbf{F} = ma$$

The application of a force does not occur instantaneously, but over time. If we multiply both sides of the above equation by time, we get:

$$\int \mathbf{F}dt = ma(t)$$

The product of force and time is known as impulse and multiplying acceleration by time yields velocity. This leads us to *Newton's third law*, which states that for every action there is an equal and opposite reaction.[4] For instance, when two objects of equal velocity and mass strike each other, their velocities are reduced to zero at the moment of impact. Each exerts its force on the other and, because these forces are exactly equal and opposite, the net force is zero. Therefore, the net change in momentum is zero. This means that these two objects would change their direction and "bounce" in opposite directions if each was traveling at the exact same velocity, but in the opposite direction. This occurs only if 100% of the energy could be transferred into changing velocity and none into altering mass.

Interactions in which both momentum and energy are conserved are termed elastic. In real trauma scenarios, collisions are inelastic. Inelastic collisions conserve momentum, but not kinetic energy. In these instances the kinetic energy "does work" in the deformation of materials even to the point where objects can conglomerate and form a single object. This is the hallmark of the inelastic collision. This energy transfer to structures that are deformed in response to a change in their momentum, such as organs and bones, is responsible for the injury sustained by the host.

We can understand the simple basics of these complex interactions using the example of two cars colliding. **Figure 1-1A** represents a head-on collision of two vehicles with equal mass and velocity and, thus, equal kinetic energy and momentum in opposite directions. Thus, the total momentum for the system is 0 prior to the crash and, by the law of conservation of momentum, must be 0 after the crash. Because both cars are traveling in exact opposite directions at exactly the same speed, their momentums will cancel each other out. If the cars were made of a perfectly nondeformable material, all kinetic energy would be exchanged and the cars would bounce in opposite directions at the exact same speed. In reality, however, these vehicles will be deformed by this interaction relative to their velocity on impact. Assuming that both cars come to rest as a single mass of entangled metal (referred to as object C), this change in momentum represents a force, which is equally applied to both cars. Because the final velocity is 0, the final kinetic energy is 0, meaning that all the kinetic energy has been converted to work that stops the other car and causes deformation such as breaking glass, bending metal, and causing physical intrusion into the passenger compartment. If the momentum of car A was greater than that of car B by having a greater mass or velocity, the resultant mass C will have momentum the direction of car A prior to impact. As such, the kinetic energy transferred to the occupants of vehicle A will be relatively less than that transferred to car B. This is intuitively accepted as we consider the effects of collisions between a compact car and an SUV with predictable consequences.

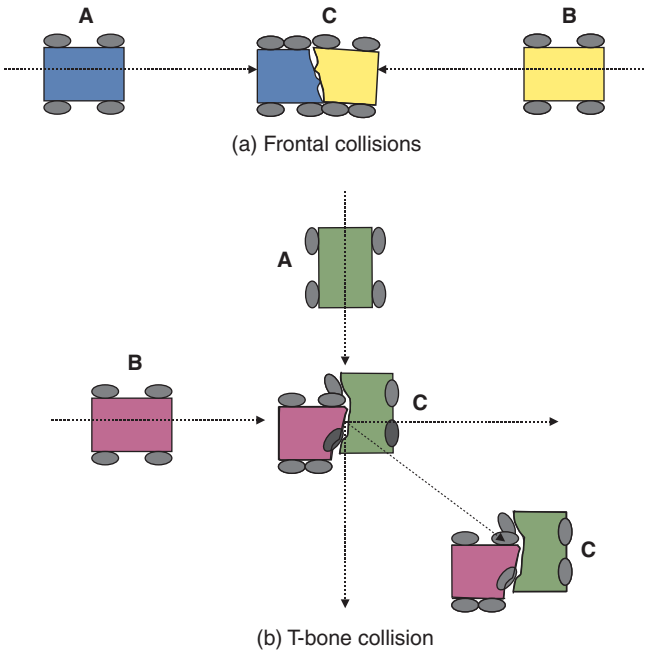


FIGURE 1-1 Energy and momentum available in various motor vehicle crash scenarios. (**A**) Frontal collisions have the greatest change in momentum over the shortest amount of time and hence the highest forces generated. (**B**) T-bone collision. When cars A and B collide their resultant momentum directs them toward their final position C; the individual momentums in the *x* and *y* axis are dissipated over a greater time resulting in smaller forces then head-on collision.

In T-bone type crashes the directions of the momentum of cars A and B are perpendicular and momentum is conserved in a third direction, C, Fig. 1-1B. Because kinetic energy is partly conserved in this new momentum, less energy was transferred to the vehicles (or their occupants) and less deformity occurs. In rear-end collisions, the energy exchange is a function of the net difference in momentum, not absolute momentum. The more momentum the conglomerate of the two vehicles (mass C) can conserve, the less energy is transferred into deforming the vehicles A and B and their occupants. In biomechanics as in life, the key to avoiding destruction when two forces meet is to maintain harmony in motion. If such an ideal is impossible to ensure, the next best thing is to protect the essential core of an object by focusing the deformity on nonessential parts. Modern automobile design, taking the lead from automobile racing engineers whose subjects are exposed to extraordinary speeds, involves building impact zones that deform easily on impact and

absorb more of the energy transfer in the periphery of the vehicle. As the old saying goes, "something has to give."

The deformation of the vehicle (and its occupants, perhaps) can best be described as work done and is an important concept in biomechanics. Work (W) is defined as a force exerted over a distance and is frequently written as

$$W = \int F dx$$

with $\mathbf{F} = ma$ and $a = vdv/dx$

$$W = \int mvdv/dx(dx)$$

which after integration yields the familiar formula for kinetic energy: $1/2mv^2$

$$W = 1/2mv_2^2 - 1/2mv_1^2$$

Therefore, the work being done by one object in motion on another equals the kinetic energy of the object prior to doing work minus the kinetic energy after the interaction. In other words, the work done is equal to the change in kinetic energy of the first object.[5] When this interaction sets the other body in motion, the second body now has kinetic energy of its own equal to the work done, assuming no deformation occurred. The greater the momentum of the first object, the greater the magnitude of the work it can do on the second. A bowling ball in motion is a perfect example of this principle. Although moving at relatively low speed, its mass and nondeformable composition allow it to do more work on objects in its path, such as bowling pins. An object of far less mass, such as a baseball, would have to make up in speed what it lacked in mass, in order to be equally effective.

This illustrates the law of conservation of momentum. The total momentum of a system will remain constant unless acted upon by an external force. The momentum of this two-object system is the same after a collision as it was prior to impact.[6] Likewise, for an object to have the same momentum as another of greater mass, it must be traveling at a commensurately greater velocity.

PENETRATING TRAUMA AND BALLISTICS

Ballistics is the study of objects in flight. *Internal ballistics* is the study of the objects themselves, such as shape, mass and velocity. *External ballistics* is the study of the effects of wind speed, drag, and gravity. What happens when projectiles reach their target is the science of *terminal ballistics*.[7] Although this has been a particular obsession of humankind when the first hunter hurled the first stone at the unsuspecting prey, its application is forever and irrevocably changing. Col. Louis A. Lagarde wrote "…the wounds by firearms of fifty years ago and the results of the treatment then in vogue form no guide for a study of the subject today." This was 100 years ago from the writing of this chapter, when the Western powers were engaged in a war whose savagery had never before been seen.[8] It was the improvement in ballistics and weapons technology in the turn of the century that brought about a completely new and more gruesome way to fight wars. Although weapons technology has not made such a significant leap in the recent decades as it did between 1870 and 1914, the appearance of military-grade weapons in urban America has made treatment of ballistic injuries in civilian trauma centers a common reality.[9]

TABLE 1-1: Velocity and Kinetic Energy Characteristics of Various Guns

Caliber	Velocity (ft/s)	Muzzle energy (ft-lb)
Handguns		
0.25 in	810	73
0.32 in	745	140
0.357 in	1410	540
0.38 in	855	255
0.40 in	985	390
0.44 in	1470	1150
0.45 in	850	370
9 mm	935	345
10 mm	1340	425
Long guns/military weapons		
0.243 Winchester	3500	1725
M-16	3650	1185
7.62 NATO	2830	1535
Uzi	1500	440
AK47	3770	1735

First to consider is that the motion of a projectile has three forces acting upon it as follows: the force of the propellant, such as a discharge or the force of a bowstring being released; the force of gravity pulling it down; and the resistance of the medium—air, water, or tissue—impeding or altering its flight. The performance of a bullet in producing injury is reliant upon velocity, construction of the bullet, and the composition of the target.[10] Thus, if one knows all of the variables in the equation, projectiles have predictable trajectories and effects. The problem, of course, is that one rarely knows *all* of the variables. What follows is an attempt at summarizing current understanding of some of these variables and their clinical relevance in traumatic injury (Table 1-1).

Cavitation

The first concept to consider is the space that a bullet creates in tissue, which is termed cavitation. There are two main forms of cavitation. First, a permanent cavitation caused by the projectile by crushing and displacing the tissue before its leading edge, commonly referred to as the "bullet track" (Fig. 1-2).[11] Then there is the temporary cavity caused by the hydrostatic pressure wave generated by the bullet as it moves through the tissue, which induces stretching of the tissue adjacent to the bullet track. It is this temporary cavity and the injury it may produce that is most often misunderstood. The nature of the temporary cavity and, hence, its potential to cause injury, is a function of several factors.

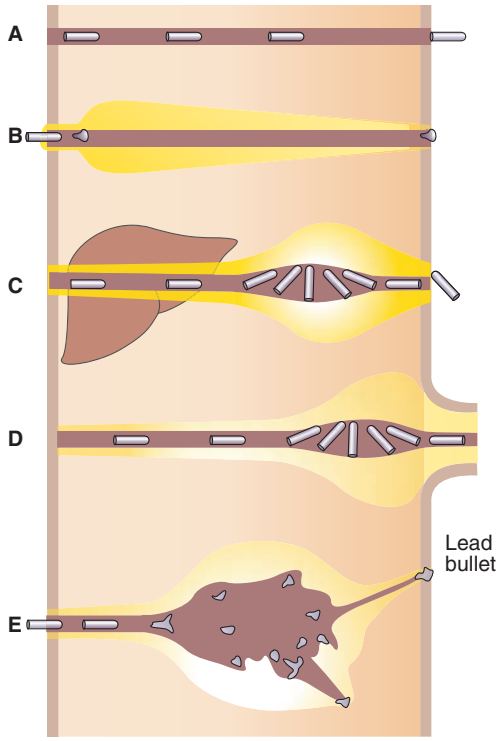


FIGURE 1-2 Wound profile of (**A**) nonfragmenting bullet through tissue with minimal yaw, (**B**) mushrooming bullet, (**C**) bullet tumbling through solid organ, (**D**) bullet tumbling with large exit skin flaps due to hydrostatic pressure wave, (**E**) bullet fragmenting.

Velocity

One of the most widely misconceived notions regarding ballistics is the idea that a projectile's destructive power is directly determined by its velocity. It is true that the kinetic energy carried by a projectile is mainly determined by its velocity, if one considers the formula for kinetic energy (E_k):

$$E_k = \frac{1}{2} MV^2$$

While an increase in velocity will have an exponential effect on the energy of the system and the energy to be released, the determining factor for tissue destruction is not the kinetic energy of the projectile, but how much of that energy can be transferred to the host tissues.[12] This depends on several factors, chief among them is sectional density.

Sectional Density

The delivery of kinetic energy to a medium depends on the interaction of the projectile and the medium through which it travels. This interaction is described in the formula for drag, which is the degree to which a medium affects the motion of a projectile:

$$\frac{\mathrm{CD} \times \mathrm{d} \times v^2}{M/A}$$

where CD is coefficient of drag for the projectile, d is the density of the medium through which it moves, v is the velocity of the projectile, M is the mass, and A is the cross-sectional area of the projectile. The coefficient of drag is difficult to measure, however, and the ballistic coefficient (BC) is more often used in reports.

$$\mathrm{BC} = \mathrm{SD} \times \mathrm{I}$$

where I is a coefficient of form, SD is the sectional density, or the ratio of the surface area of its leading edge (the arrow, bullet, or fragment tip) relative to its overall mass and can be summarized in the following formula[13]:

$$\mathrm{SD} = m/D^2$$

where diameter (D) is the diameter of the frontal surface area presented to the target and mass (m) is the overall mass of the entire projectile. The higher the sectional density, the higher the ballistic coefficient, and the less relative impedance that projectile will undergo through a medium. In other words, projectiles with low sectional density will be subject to relatively greater drag that retards their movement, thus losing their kinetic energy to the environment.

Recalling that the energy of a system will be conserved, we must accept that some or all of the kinetic energy of a projectile will be transferred to the medium in which it is moving as it loses momentum. It is the transfer of this kinetic energy that results in disruption of the medium through which it passes, its potential destructive force. The destructive force is influenced by a corollary to this equation, namely that the shape (and thus the sectional density) of a bullet is subject to change within the tissue through which it passes in three ways including *tumbling*, *mushrooming*, and *fragmentation*.

The degree to which a projectile's tip deviates from its flight path is its yaw (Fig. 1-2).[1] An arrow is an extreme example of a projectile with a very high sectional density that remains stable as it traverses the host tissue, whereas the relatively short bullet is much more prone to extreme yaw and flip on its axis as it slows, which is termed "tumbling." This will produce an immediate decrease in the sectional density, increasing drag and thus releasing more kinetic energy into the surrounding tissue. Given that the displacement of tissue occurs not gradually but instantaneously and that force is a function of both mass and the change in speed over time (acceleration), the destruction of tissue occurs not just because it is displaced, but because it is displaced extremely rapidly. The point of both maximal velocity and lowest sectional density corresponds with the site of greatest temporary cavitation (Fig. 1-2).

The most extreme form of change in sectional density is fragmentation, where the bullet breaks up into smaller pieces that will each produce separate injury tracks and patterns. In their experimental model, Fackler et al. noted that fragmentation of a bullet caused far greater tissue injury than did temporary cavitation in striated muscle.[14] The greater injury was due to the greater number of "projectiles" lacerating and crushing tissue that might otherwise be only slightly affected by being subjected to temporary cavitation.

Tissue Characteristics: Density and Elasticity

Sir William MacCormack's experiments with ballistics showed that, when fired through a metal can filled with water, a bullet will leave an exit hole of approximate size to its own; however, the hydrostatic pressure wave it carried resulted in a final exit hole that was larger and more irregular.[7,14] When reapproximating the pieces of the metal can together, it was obvious that the bullet exited first, creating a small hole, and then the hydrostatic pressure wave impacted the wall of the can tearing it asunder. The transfer of energy is far more perfect in an incompressible liquid like water and the hydrostatic wave more destructive. In the human body, differences in density, elasticity, compliance, and water content of various tissues along the path of a bullet make the precise nature of the hydrostatic wave less predictable. Nevertheless, this experiment served to illustrate the overall nature of the kinetic energy transfer of a projectile through tissue.

Some of the characteristics of wounds inflicted by bullets have been studied in real-time by the use of high-speed photography and so-called ordinance gelatin. Dr Martin Fackler, as head of the Wound Ballistics Laboratory at the Letterman Army Institute of Research in the 1980s, showed that 10% gelatin was an ideal medium for the purposes of studying ballistics in tissue.[11] He has also been its most vocal critic, stating that "with the advent of the high-speed movie camera … emphasis in wound ballistics has shifted from sound scientific method to spectacular cinematography—a triumph of high technology over common sense."[11] Nevertheless, several key observations have been made using this method of ballistic testing and analysis of what he coined the *wound profile*, or the visual composite of trajectory characteristics in the medium (Fig. 1-1).

In this medium he recognized that, although the gelatin's high elasticity allowed it to be displaced by the projectile and return to its original shape, radial lacerations occurred that correlated directly with the size of the temporary cavity. Unlike ordinance gelatin, the variance in tissue characteristics through which a projectile can pass makes accurate predictions of cavitation injury far more challenging.

Tissue with low density such as fat and lung may not impart much resistance to a projectile and not cause it to fragment or yaw significantly. Indeed, Kocher's visit to Prussian field hospitals in the latter years of World War I confirmed many of his predictions regarding ballistic injuries of the newer forms introduced at the turn of the century. For example, he noted that a fine-tipped, smaller, harder projectile produced such minimal cavitation that through-and-through injuries of the thorax through lung resulted in mere weeks of convalescence prior to soldiers returning to the front in stark contrast to what he had seen in the prior decades when broad-tipped, soft lead bullets resulted in far more destructive injury patterns.[15] Furthermore, each tissue has unique thresholds of stress and strain and a recoverable limit beyond which permanent destruction occurs.[11] In the case of hepatic tissue, which is very inelastic, the temporary cavity will result in tissue destruction comparable to the size of the temporary cavity, as anyone who has treated these injuries can attest.

Stopping Power

The image of gunshot wound victims being hurled bodily by the impact of bullets is a factual misrepresentation. If momentum of an object is mass × velocity, the maximum momentum transferred from any small arms projectile including an assault rifle or shotgun results in a backward motion of an 80 kg target body of under 0.2 m/s, which is negligible compared to the momentum of an adult even at a slow walk.[16] The effect of the momentum transferred from the missile is virtually zero and there is no backward motion. The only injury that causes instant "stoppage" is a direct one to the brain or spinal cord inducing instantaneous paralysis. The remainder of injuries relies on exsanguination to produce the same effect by reducing blood flow to the central nervous system to below critical levels. In theory, greater cavitation effects are more likely to produce an exsanguinating injury, and a larger vascular defect will produce this effect more quickly; however, the stopping power of the weapon is mainly a function of its ability to produce rapid hemorrhagic shock and not any sort of physical counteraction in the target's momentum.[16,17] As LaGarde concluded from his experiments, "We are not acquainted with any bullet fired from a hand weapon that will stop a determined enemy when the projectile traverses soft parts alone."[8]

As we have discussed, there are many factors that influence the destructive force of a bullet, not the least of which are the intrinsic characteristics of the bullet itself. British soldiers noted that their new hard-jacketed, sharp bullets were not as effective at stopping the advancing enemy. They discovered that, by cutting off the tip, they exposed the soft lead core, and the bullets were predictably more effective at halting the enemy. The arsenal where these first bullets were produced was in Dumdum, India, so the term for an expanding or mushrooming bullet is *dum-dum*. These were effective enough that the 1899 International Convention at the Hague banned their use in warfare, although this ban did little to curb their use then or thereafter.[7,11]

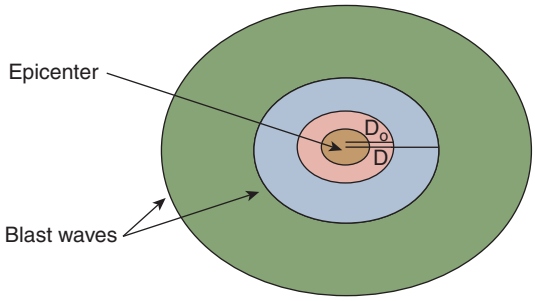
Contact Shots

Gunshot wounds where the muzzle is in contact with the skin are termed "contact shots" and have a very different wound profile. The muzzle blast releases expanding gases contained by the skin and subcutaneous tissue and directed inward with devastating effects.[18] The result is a contained explosion that is more destructive than the temporary cavity of a passing projectile, given that the blast velocity of gunpowder is upward of 20,000 ft/s, manyfold greater than the velocity of even the highest-velocity small arms projectile (<4000 ft/s). In 1984, American television actor Jon-Erik Hexum killed himself on set when he fired a revolver loaded with blanks into his temple. Despite the absence of an actual projectile, the muzzle blast was contained by the skin and focused into

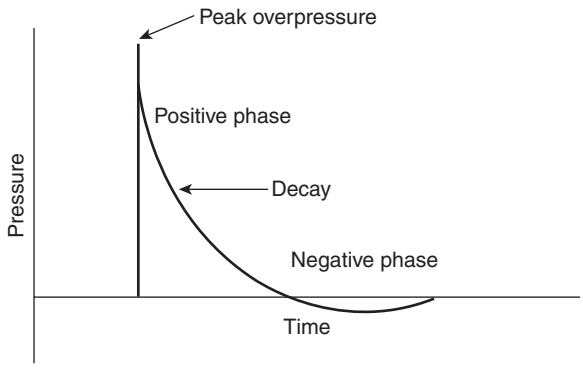
his temple with sufficient force to cause a devastating brain injury that killed him instantly. The surgeon must take into account the circumstances of the wounding and the possibility that the tissue injury beneath the skin surface may be significantly greater than expected in the case of contact or near-contact shots.

Ballistic Myths Dispelled

- Bullets do not ricochet within the body.[17]
- Velocity is the main predictor of tissue destruction. Destructive force is a function of several factors pertaining to both the projectile and the target.[7,11–13,17]
- The largest portion of the temporary cavity is not closest to the surface, where the velocity is the greatest. The degree of cavitation depends on both the velocity of the projectile and its surface density along that portion of the trajectory (Fig. 1-3).[7,11,12]
- Bullets are not superheated when fired and thus not sterile on impact. Bullets can and do carry bacteria from the surface (clothes, skin) to the deeper tissues.[13,15]



A The Scaling Laws



B The Pressure–Time relationship at any given distance from the epicenter

FIGURE 1-3 Physical characteristics of an explosive blast. (**A**) The scaling laws relate the overpressure at specific distances to the ratio of distances from the epicenter of a blast and the cube root of the ratios of corresponding weights of the charges. (**B**) The pressure–time relationship at any given distance from the epicenter—the peak overpressure represents the passing wave front with a subsequent decrease in pressure until ambient pressure is reached. This is known as the positive phase. The passing wave will then cause a decrease in pressure below baseline resulting in a relative vacuum, or negative pressure phase.

- Bullets traveling at the speed of sound (>1.5 km/s) will not result in increased tissue disruption compared to parts of the trajectory, where the bullet travels subsonically.[19]
- Bullets do not tumble in the air. Yaw actually decreases the farther it is from the barrel. A bullet's yaw is negligible in flight and bullets will strike the target tip-first unless altered in their flight path by another force or object.[13,17]
- The amount of tissue destroyed does not usually significantly exceed the size of the bullet. The ability of tissue to absorb the energy of temporary cavitation will largely determine its ability to remain uninjured. As a result of both clinical and experimental studies, the recommendation is that surgeons limit debridement to what is grossly nonviable when indicated.[11,12,17]

STRESS, STRAIN, AND BIOMECHANICS

When a force is applied to a particular material, it is typically referred to as a stress, which is a load or force per unit area. This stress will cause deformation of a given material. Strain is the distance of the deformation caused by the stress, divided by the length of the material to which the stress was applied.[20]

Strain can be tensile, shear, compressive, or overpressure (a relative of compressive strain) (Fig. 1-4). Tensile strain of

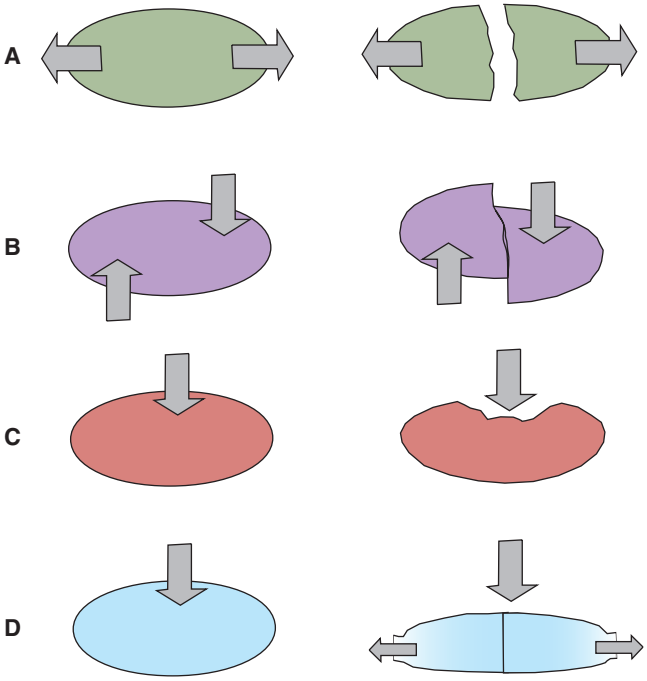


FIGURE 1-4 Biomechanical mechanisms of injury. (**A**) Tensile strain—opposite forces stretching along the same axis. (**B**) Shear strain—opposite forces compress or stretch in opposite direction but not along the same axis. (**C**) Compressive strain—stress applied to a structure usually causing simple deformation. (**D**) Overpressure—a compressive force increases the pressure within the viscus passing the "breaking point" of the wall.

a particular structure or organ occurs as opposing forces are applied to the same region. The forces are opposite and concentrated upon a particular point. This essentially interrupts the integrity of the structure by pulling it apart. Shear strain occurs as opposing forces are applied to a particular structure, but at different points within that structure. This can be caused by an application of opposing external forces or can arise from a relative differential in the change of momentum within a single structure or between structures that are attached to one another.[21]

Compressive strain is the direct deformation that occurs as a result of impact. The energy involved with a particular force does work on the structure causing a crushing-type injury resulting in deformation and interruption of the structural integrity of the injured organ. Overpressure is a type of compressive strain that is applied to a gas- or fluid-filled cavity. The energy applied to a gas- or fluid-filled viscus can deform that structure and cause a decrease in the volume of the structure. Following Boyle's law:

$$P_1V_1 = P_2V_2$$

The product of the pressure and the volume prior to an applied force must be equal to the product afterward.[6] Therefore, a decrease in its original volume will increase the pressure inside that viscus. If the rise in pressure, which is a force, overcomes the tensile strength of the viscus, it will rupture.[22]

When stress is plotted on the same graph as strain, there are several clear and distinct aspects to the curve. The elastic modulus is that part of the curve in which the force does not cause permanent deformation, and a material is said to be more elastic if it restores itself more precisely to its original configuration.[23] The portion of the curve beyond this is called the plastic modulus and denotes when an applied stress will cause permanent deformation.[24] The tensile, compressive, or shear strength is the level of stress at which a fracture or tearing occurs.[25] This is also known as the *failure point*. The area under the curve is the amount of energy that was applied to achieve the given stress and strain (Fig. 1-5).[26]

How well tissue tolerates a specific insult varies with the type of force applied and the tissue in question. In blunt and penetrating trauma, the higher the density of a particular tissue, the less elastic it is and the more energy is transferred to it in a collision. Lung is air-filled and extremely elastic. In lower velocity blunt trauma, energy tends to be dissipated across the lung easily, while in penetrating trauma, the actual destruction of the permanent cavity and stretch caused by the temporary cavity are better tolerated because of the elasticity of the lung. In contrast, solid organs such as spleen, liver, or bone tend to absorb energy and will have greater tissue destruction as a consequence.[27] In blast injury it is the air-filled structures of the lung and bowel that tend to be injured because of their ability to transmit the blast wave and cause localized pressure increases that overcome the structural failure point of the organ.[28]

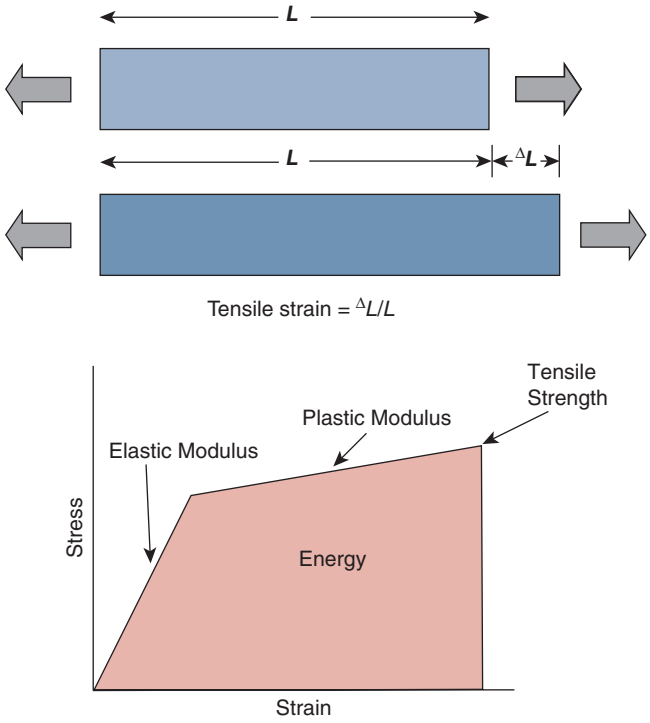


FIGURE 1-5 The concept of stress, strain, elastic modulus, plastic modulus, tensile strength and energy as demonstrated by a tensile stress applied to a given structure. The tensile strain is the change in length under a stress divided by the original length. This concept is applicable to compressive and shear strain. In the stress/strain relationship the elastic modulus is the portion of the curve where permanent deformation does not occur as opposed to the plastic modulus where it does fracture or tearing occurs at the tensile strength. The energy applied is the area under the curve.

BLUNT TRAUMA MECHANISMS AND PATTERNS OF INJURY

The transfer of energy and application of forces in blunt trauma is often much more complex than that of penetrating trauma. The most frequent mechanisms of blunt trauma include motor vehicle crashes, motor vehicles striking pedestrians, and falls from a significant height. In these instances there are typically varying energies and forces in both the victim and the striking object. Other variables that complicate care include the larger surface area over which the energy is dispersed as compared to penetrating trauma and the multiple areas of contact that can disperse energy to different regions of the victim's body. The interactions and directions of these lines of force and energy dispersion are often instrumental in causing specific kinds of injury.

Motor Vehicle Crashes

Although there are frequently confusing vectors for energy transfer and force in a victim of a motor vehicle crash, mortality is directly related to the total amount of energy and force available. Mortality from motor vehicle crashes is accounted

for largely by head-on collisions with mortality rates up to 60%. Side impact collisions (20–35%) and rollovers (8–15%) have progressively lower mortality rates with rear-end collisions (3–5%) having the lowest.[29,30] Rollover crashes have a lower than expected mortality because the momentum is dissipated, and forces generated and projected to the passenger compartment are in a random pattern that frequently involves many different parts of the car. Although there are certain forces and patterns of energy exchange that occur in a motor vehicle crash, the vehicle itself does offer some degree of protection from the direct force generated by a collision. Patients who are ejected from their vehicle have the velocity of the vehicle as they are ejected and a significant momentum. They typically strike a relatively immobile object or the ground and undergo serious loads. Trauma victims who were ejected from the vehicle were four times more likely to require admission to an intensive care unit, had a fivefold increase in the average Injury Severity Score, were three times more likely to sustain a significant injury to the brain, and were five times more likely to expire secondary to their injuries.[31]

Understanding the changes in momentum, forces generated, and patterns of energy transfer between colliding vehicles is important. Yet, the behavior of the occupants of the passenger compartment in response to these is what helps identify specific patterns of injury. In frontal collisions the front of the vehicle decelerates as unrestrained front-seat passengers continue to move forward in keeping with Newton's first law. Lower extremity loads, particularly those to the feet and knees, occur early in the crash sequence and are caused by the floorboard and dashboard that are still moving forward. Therefore, relative contact velocity and change in momentum are still low. Contact of the chest and head with the steering column and windshield occurs later in the crash sequence; therefore, contact velocities and deceleration, change in momentum, and contact force are higher.[29,32]

Types of injuries are dependent on the path the patient takes. The patient may slide down and under the steering wheel and dashboard. This may result in the knee first impacting the dashboard causing a posterior dislocation and subsequent injury to the popliteal artery. The next point of impact is the upper abdomen or chest. Compression and continued movement of solid organs results in lacerations to the liver or spleen. Compression of the chest can result in rib fractures, cardiac contusion, or a pneumothorax from the lung being popped like a paper bag. Finally, the sudden stop can cause shear forces on the descending thoracic aorta resulting in a partial or full-thickness tear. The other common path is for the occupant to launch up and over the steering wheel. The head then becomes the lead point and strikes the windshield with a starburst pattern resulting on the windshield. The brain can sustain direct contusion or can bounce within the skull causing brain shearing and a contrecoup injury. Once the head stops, forces are transferred to the neck which may undergo hyperflexion, hyperextension, or compression injuries, depending on the angle of impact. Once the head and neck stop, the chest and abdomen strike the steering wheel with similar injuries to the down and under path.

Lateral collisions, specifically those that occur on the side of a seated passenger, can be devastating because of the small space between the striking car and the passenger. Therefore, resistance to slow momentum of the striking car prior to contact with the passenger is limited. If the side of the car provides minimal resistance the passenger can be exposed to the entire momentum change of the striking car. These loads are usually applied to the lateral chest, abdomen, and pelvis and, as such, injuries to the abdomen and thorax are more frequent in lateral collisions than in frontal collisions.[33] Injuries to the chest include rib fractures, flail chest, and pulmonary contusion. Lateral compression often causes injuries to the liver, spleen, and kidneys, as well. Finally, the femoral head can be driven through the acetabulum.

Rear-end collisions are classically associated with cervical whiplash-type injury and are a good example of Newton's first law at work. When the victim's car is struck from behind, the body, buttressed by the seat, undergoes a forward acceleration and change in momentum that the head does not. The inertia of the head tends to hold it in a resting position. The forward pull of the trunk causes a backward movement on the head causing hyperextension of the neck. Similarly, this injury pattern can also be seen in head-on collisions, where a sudden deceleration of the trunk with a continued forward movement of the head is followed by a backward rotation resulting from recoil.[34,35]

Pedestrian Injuries

Pedestrian injuries frequently follow a well-described pattern of injury depending on the size of the vehicle and the victim. Nearly 80% of adults struck by a car will have injuries to the lower extremities. This is intuitively obvious in that the level of a car's bumper is at the height of the patient's knee. This is the first contact point in this collision sequence, with the largest force being applied to the lower extremity. Those struck by a truck or other vehicle with a higher center of mass more frequently have serious injury to the chest and abdomen, since the initial force is applied to those regions. In the car-pedestrian interaction, the force applied to the knee region causes an acceleration of the lower portion of the body that is not shared by the trunk and head, which, by Newton's first law, tend to stay at rest. As the lower extremities are pushed forward they will act as a fulcrum bringing the trunk and head forcefully down on the hood of the car applying a secondary force to those regions, respectively. The typical injury pattern in this scenario is a tibia and fibula fracture, injury to the trunk such as rib fractures or rupture of the spleen, and injury to the brain.[36,37]

Falls

Falls from height can result in a large amount of force transmitted to the victim. The energy absorbed by the victim at impact will be the kinetic energy at landing. This is related to the height from which the victim fell. The basic physics formula describing the conservation of energy in a falling body states that the product of mass, gravitational acceleration, and

height, the potential energy prior to the fall, equals the kinetic energy as the object strikes the ground. With mass and gravitational acceleration being a constant for the falling body, velocity, and, therefore, momentum and kinetic energy are directly related to height.[6] The greater the change in momentum upon impact the larger the load or force applied to the victim. Injury patterns will vary depending upon which portion of the victim strikes the ground first and, hence, how the load is distributed.

The typical patient with injuries sustained in a free fall has a mean fall height of just under 20 ft. One prospective study of injury patterns summarized the effects of falls from heights ranging between 5 and 70 ft. Fractures accounted for 76.2% of all injuries, with 19–22% sustaining spinal fractures and 3.7% showing a neurologic deficit.[38] Nearly 6% of patients had intra-abdominal injuries, with the majority requiring operative management for injury to a solid organ. Bowel and bladder perforation were observed in less than 1% of injuries.[39]

Anatomic Considerations

INJURY TO THE HEAD (BRAIN AND MAXILLOFACIAL INJURY)

The majority of closed-head injuries are caused by motor vehicle collisions, with an incidence of approximately 1.14 million cases each year in the United States.[40,41] The severity of traumatic brain injury represents the single most important factor contributing to death and disability after trauma and may contribute independently to mortality when coexistent with extracranial injury.[35,42,43] Our knowledge of the biomechanics of injury to the brain comes from a combination of experiments conducted with porcine head models, biplaner high-speed x-ray systems, and computer-driven finite element models.[44] There are a multitude of mechanisms that occur under the broad heading of a traumatic brain injury. They are all a consequence of loads applied to the head resulting in differing deceleration forces between components of the brain. Brain contusion can result from impact and the direct compressive strain associated with it. The indirect component of injury to the brain on the side opposite to that of impact is known as the *contrecoup* injury. This occurs because the brain is only loosely connected to the surrounding cranium. As a result, after a load is applied to the head causing a compressive strain at the point of impact and setting the skull in motion along the line of force, the motion of the brain lags behind the skull. As the skull comes to rest, or even recoils, the brain, still moving along the line of the initial load, will strike the calvarium on the opposite side and another compressive strain is generated. The existence of the coup-contrecoup injury mechanism is supported by clinical observation and has been confirmed by a three-dimensional finite element head model and pressure-testing data in cadavers.[45] It is even suspected that this forward acceleration of the brain relative to the skull may set up a tensile strain in the bridging veins causing their laceration and formation of a subdural hematoma.[46]

Injury to the superficial regions of the brain is explained by these linear principles; however, injury to the deep structures of the brain, such as diffuse axonal injury (DAI), is more complicated. Several authors have tried to explain DAI as a result of shear strain between different parts of the brain, but there is also another model known as the stereotactic phenomena. This model relies more on wave propagation and utilizes the concavity of the skull as a "collector," which focuses multiple wave fronts to a focal point deep within the brain, causing disruption of tissue even in the face of minimal injury at the surface of the brain.[47] This "wave propagation" through deeper structures within the brain, such as the reticular-activating system, with subsequent disruption of their structural integrity is thought to account for a loss of consciousness, the most frequent serious sign after blunt trauma to the brain.[48] An injury caused by shear strain is the laceration or contusion of the brainstem. This is explained by opposing forces applied to the brain and the spinal cord perpendicular to their line of orientation, with the spinal cord and brainstem being relatively fixed in relation to the mobile brain.

Maxillofacial injuries are associated with injuries to the head and brain in terms of mechanism and are a common presentation in motor vehicle trauma. The classic force vector that results in mid-face fractures is similar to that of a traumatic brain injury and occurs when the occupant of a motor vehicle impacts the steering wheel, dashboard, or windshield. Nearly all of these subtypes of injury are secondary to compressive strain. This mechanism is associated with the greatest morbidity for the driver and front-seat passenger, while the forces are attenuated for the back-seat passenger impacting the more compliant front seat.

THORACIC INJURY

The primary mechanism of blunt thoracic trauma involves inward displacement of the body wall with impact. Musculoskeletal injury in the chest is dependent upon both the magnitude and rate of the deformation of the chest wall and is usually secondary to compressive strain from the applied load. Patterns of injury for the internal organs of the thorax frequently reflect the interactions between organs that are fixed and those that are relatively mobile and compressible. This arrangement allows for differentials in momentum between adjacent structures that lead to compressive, tensile, and shear stress.

The sternum is deformed and rib cage compressed with a blunt force to the chest. Depending on the force and rate of impact in a collision, ribs may fracture from compressive strain applied to their outer surface and consequent tensile strain on the inner aspects of the rib. Indirect fractures may occur due to stress concentration at the lateral and posterolateral angles of the rib. Furthermore, stress waves may propagate deeper into the chest resulting in small, rapid distortions or shear forces in an organ with significant pressure differential across its parenchymal surface (ie, the air and tissue interface of the lung). This is thought to be the mechanism causing a pulmonary contusion.

Blunt intrusion into the hemithorax and pliable lung could also result in overpressure and cause a pneumothorax. A direct load applied to the chest compresses the lung and increases the pressure within this air-filled structure beyond the failure point of the alveoli and visceral pleura. This overpressure mechanism may also be seen with fluid (blood) instead of air in a blunt cardiac rupture. High-speed cineradiography in a model of anterior blunt chest trauma using a pig has demonstrated that the heart can be compressed to half of its precrash diameter with a doubling of the pressure within the cardiac chambers.[49] If the failure point is reached, rupture occurs with disastrous results.

There are several examples of indirect injury secondary to asynchronous motion of adjacent, connected structures and development of shear stress at sites of attachment.[50] Mediastinal vascular injury and bronchial injury are examples of this mechanism. Transaction of the thoracic aorta is a classic deceleration injury mediated by shear forces. This injury can occur in frontal or lateral impacts[51] and occurs because of the continued motion of the mobile and compressible heart in relation to an aorta that is tethered to more fixed structures. In frontal and lateral impacts the heart moves in a horizontal motion, relative to an aorta that is fixed to the spinal column by ligamentous attachments. This causes a shear force applied at the level of the ligamentum arteriosum. When the stress is applied in a vertical direction, such as a fall from a height in which the victim lands on the lower extremities, the relative discrepancy in momentum is in that plane and a tensile strain is generated at the root of the thoracic aorta (Fig. 1-6). Injury to a major bronchus is another example of this mechanism. The relatively pliable and mobile lung generates a differential in momentum in a horizontal or vertical plane depending on the applied load as compared to the tethered trachea and carina. This creates a shear force at the level of the main stem bronchus (Fig. 1-7) and explains why the majority of blunt bronchial injuries occur within 2 cm of the carina.

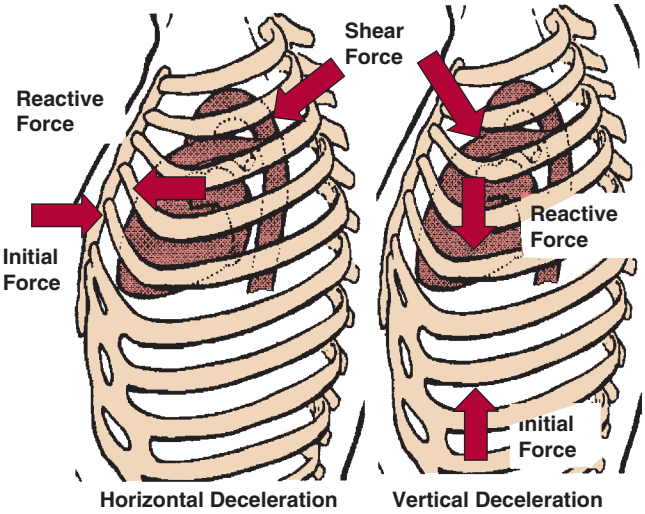


FIGURE 1-6 Various mechanisms of injury to the thoracic aorta. In a horizontal deceleration the heart and arch move horizontally away from the descending aorta causing shear strain and tearing at the ligamentum arteriosum. A vertical deceleration causes caudad movement of the heart, causing a strain at the root of the ascending aorta.

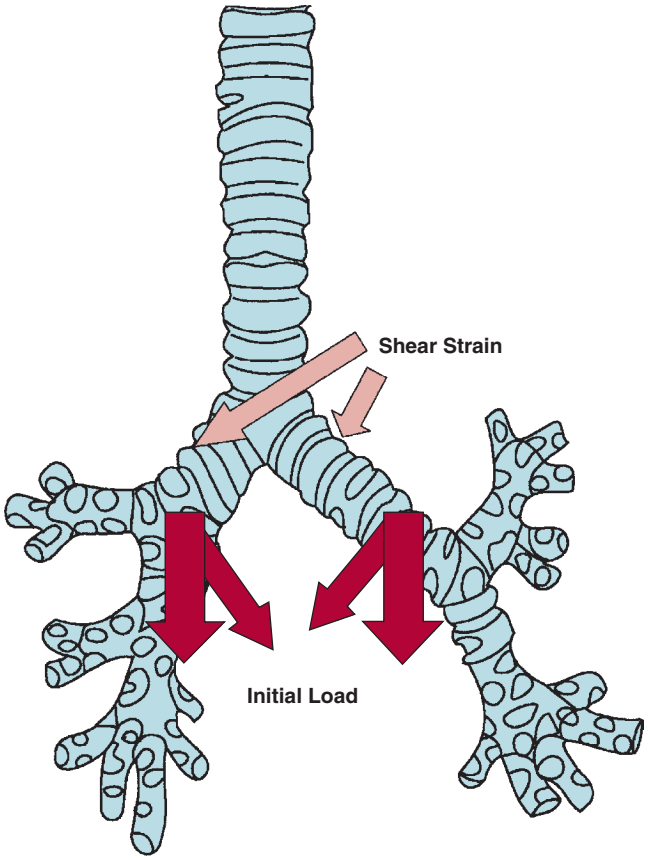


FIGURE 1-7 Mechanisms of injury for bronchial injury. The carina is tethered to the mediastinum and spinal complex while the lungs are extremely mobile, setting up shear strain in the main stem bronchus upon horizontal or vertical deceleration.

ABDOMINAL INJURY

Abdominal organs are more vulnerable than those of the thorax because of the lack of protection by the sternum and ribs. A number of different mechanisms account for the spectrum of injury observed in blunt trauma to the abdomen. With regard to the solid abdominal organs, a direct compressive force, with parenchymal destruction, probably accounts for most observed injuries to the liver, spleen, and kidney. Yet, shear strain can also contribute to laceration of these organs. As with the previous description of strain forces, a point of attachment is required to exacerbate a differential in movement. This can occur at the splenic hilum resulting in vascular disruption at the pedicle or at the ligamentous attachments to the kidney and diaphragm. Shear forces in the liver revolve upon the attachments of the falciform ligament anteriorly and the hepatic veins posteriorly, explaining injuries to the parenchyma in these areas. Another significant injury related to this mechanism is that of injury to the renal artery. The renal artery is attached proximally to the abdominal aorta, which is fairly immobile secondary to its

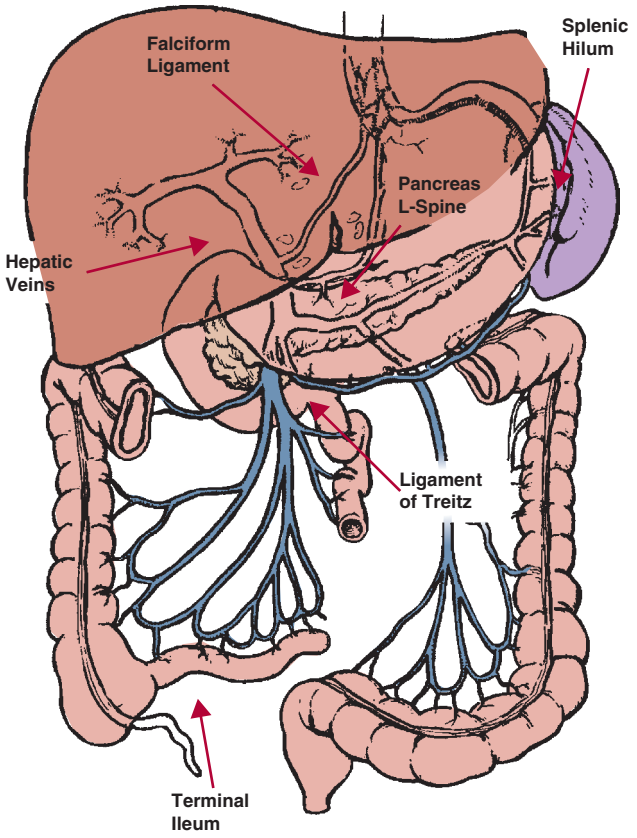


FIGURE 1-8 Points of shear strain in blunt abdominal trauma. All of these points occur where a relatively fixed structure is adjacent to a mobile structure.

attachments to the spinal column, and distally to the kidney, which has more mobility. A discrepancy in momentum between the two will exact a shear stain on the renal artery resulting in disruption.[52] This same relation to the spinal column occurs with the pancreas (Fig. 1-8). The relatively immobile spine and freely mobile pancreatic tail predispose to a differential in momentum between the two in a deceleration situation leading to fracture in the neck or body of the pancreas. The biomechanics of such injuries suggest that the body's tolerance to such forces decreases with a higher speed of impact, resulting in an injury of greater magnitude from a higher velocity collision.[26]

Perforation of a hollow viscus in blunt trauma occurs in approximately 3% of victims.[53] The exact cause is a matter of debate. Some believe that it is related to compressive forces, which cause an effective "blowout" through generation of significant overpressure, while others believe that it is secondary to shear strains.[54] Both explanations are plausible, and clinical observations have supported the respective conclusions. Most injuries to the small bowel occur within 30 cm of the ligament of Treitz or the ileocecal valve, which supports the shear force theory (Fig. 1-8).[55] Yet, injuries do occur away from these points of fixation. Also, experiments have documented that a "pseudo-obstruction" or temporarily closed loop under a load can develop bursting pressures as described by the overpressure theory.[56] Most likely, both proposed mechanisms are applicable in individual instances. The most common example of the pseudo-obstruction type is blunt rupture of the duodenum, where the pylorus and its retroperitoneal location can prevent adequate escape of gas and resultant high pressures that overcome wall strength.

Another important example of overpressure is rupture of the diaphragm. The peritoneal cavity is also subject to Boyle's law. A large blunt force, such as that related to impact with the steering wheel applied to the anterior abdominal wall will cause a temporary deformation and decrease in the volume of the peritoneal cavity. This will subsequently raise intra-abdominal pressure. The weakest point of the cavity is the diaphragm with the left side being the preferred route of pressure release as the liver absorbs pressure and protects the right hemidiaphragm. The relative deformability of the lung on the other side of the diaphragm facilitates this.

MUSCULOSKELETAL INJURY

By far, the most common type of blunt injury in industrialized nations is to the musculoskeletal system. The ratio of orthopedic operations to general surgical, thoracic, and neurosurgical operations is nearly 5:1. As stated earlier, seatbelts and air bags have significantly decreased the incidence of major intracranial and abdominal injuries; however, they have not decreased the incidence of musculoskeletal trauma. Although these are not usually fatal injuries, they often require operative repair and rehabilitation and can leave a significant proportion of patients with permanent disability.[57] With the advent of seatbelt laws, improved restraint systems, and air bags in motor vehicles, the incidence of lower extremity trauma, in particular, has increased. It is thought that these patients in the past may have suffered fatal injuries to the brain or torso and, therefore, their associated fractures of the femur, tibia, and fibula were not included in the overall list of injuries.

The type and extent of injury is determined by the momentum and kinetic energy associated with impact, underlying tissue characteristics, and angle of stress of the extremity. High-energy injuries can involve extensive loss of soft tissue, associated neurovascular compromise, and highly comminuted fracture patterns. Low-energy injuries are often associated with crush or avulsion of soft tissue in association with simple fractures. Injuries to soft tissue are usually secondary to compressive strain with crush injury as an example. Tensile and shear strain mechanisms, however, are present with degloving and avulsion injuries, respectively.

Most of that written about musculoskeletal injury involves fractures of long bones. Although each fracture is probably a consequence of multiple stresses and strains, there are four basic biomechanisms (Fig. 1-9). In a lateral load applied to the mid shaft of a long bone, bowing will occur and compressive strain occurs in the cortex of the bone where the load is applied. The cortex on the opposite side of the bone will undergo tensile strain as the bone bows away from the load. Initially, small fractures will occur in the cortex undergoing tensile strain because bone is weaker under tension than it is

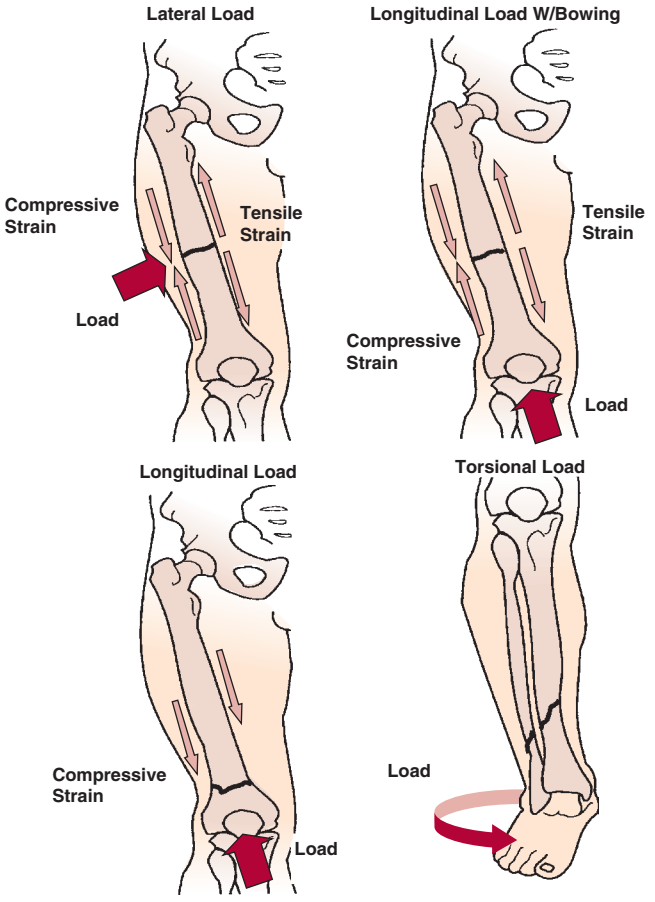


FIGURE 1-9 Fracture mechanics. A lateral load causing "bowing" will create tensile strain in the cortex opposite the force and compressive strain in the adjacent cortex. If a longitudinal stress caused "bowing" a similar strain pattern occurs. If no bowing occurs the strain is all compressive. A torsion load will cause a spiral fracture.

under compression.[58] Once the failure point is reached on the far side from the load, the compressive strain increases markedly and the failure point for the side near the applied load is reached, also, resulting in a complete fracture. This mechanism can be seen in passengers in lateral collisions, pedestrians struck by a passenger car in the tibia and fibula region, or in the upper extremities from direct applied force in victims of assault with a blunt instrument.

When a longitudinal load is placed on a long bone, bowing can also occur, and the compressive and tensile strain patterns will be similar to that previously described. If bowing does not occur, then only a compressive strain is seen and a compression fracture can occur. In the case of the femur this usually occurs distally with the shaft being driven into the condyles. These mechanisms can be seen in falls from a height, but are more frequently seen in head-on collisions resulting in fractures of the femur or tibia. In these cases deceleration occurs and the driver's or passenger's feet receive a load from the floorboard or the knee receives a load from the dashboard upon deceleration. This causes a longitudinal force to be applied to the tibia or femur, respectively. A torsional load will cause the bone to fracture in a spiral pattern.

INJURY TO THE SPINE AND WHIPLASH

Injury to the vertebral column and spinal cord can be devastating and is frequently the result of a complex combination of specific anatomic features and transmitted forces. These can cause a wide variety of injury patterns distributed through the different portions of the vertebral column. Deceleration forces in motor vehicle crashes, such as impact with the windshield, steering assembly, and instrument panel, inertial differences in the head and torso, or ejection are responsible for both flexion and hyperextension injuries. Although the biomechanics of transmission of force can be readily demonstrated for the vertebral column's individual components (disks, vertebrae, etc), a model demonstrating injury patterns in the intact spinal unit is lacking.[34] The cervical spine is most frequently injured in motor vehicle crashes, due to its relatively unprotected position compared to the thoracic and lumbar regions. Injuries are related to flexion, extension, or lateral rotation, along with tension or compression forces generated during impact of the head. The direction and degree of loading with impact account for the different injury patterns in trauma to the cervical spine.[26] Approximately 65% of injury is related to flexion–compression, about 30% to extension–compression, and 10% to extension–tension injuries.[59] Fracture dislocations of the vertebrae are related to flexion and extension mechanisms, whereas fractures of the facets are related to lateral-bending mechanisms. In contrast to trauma to the cervical spine, injury to the thoracic or lumbar spine is more likely related to compressive mechanisms. The rib cage and sternum likely provide stabilizing forces in motor vehicle crashes and lessen the risk of injury in these regions.

Whiplash refers to a pattern of injury seen often in motor vehicle collisions with a rear-end impact. The injury is usually a musculoligamentous sprain, but may be combined with injury to cervical nerve roots or the spinal cord. Patients typically experience neck pain and muscle spasm, although an additional spectrum of symptoms has been described.[60] The etiology of whiplash probably relates to acceleration and extension injury, with some rotational component in non–rear-impact crashes. Factors related to poor recovery following whiplash injury are a combination of sociodemographic, physical, and psychological, and include female gender, low level of education, high initial neck pain, more severe disability, increased levels of somatization, and sleep difficulties.[61]

Kinematics in Prevention

The ideas of William Haddon have become the cornerstone of injury prevention, and approximately a third of his strategies involve altering the interaction of the host and the environment.[62] Understanding forces and patterns of energy transfer have allowed the development of devices to reduce injury. Most of this understanding has been applied to the field of automotive safety.

The first set of design features revolve around the concept of decreasing the force transmitted to the passenger compartment. This includes the "crumple zone," which allows the front and rear ends of a car to collapse upon impact. The change in momentum the passenger compartment undergoes in a collision will, therefore, occur over a longer period. Going back to the impulse and momentum relation, this means less force will be transmitted to the passenger compartment. In terms of energy, work is done in the crumple zone and energy is expended before reaching the passenger compartment.[63] The second design feature directs the engine and transmission downward and not into the passenger compartment decreasing intrusion into the passenger compartment.

Passenger restraint systems, which include safety harnesses and child car seats, keep the passengers' velocity equal to that of the car and prevent the passengers from generating a differential in momentum and striking the interior of the car. Also, they more evenly distribute loads applied to the victim across a greater surface area thus decreasing stress.

Even with restraint systems the occupants of a car can develop relative momentum and kinetic energy during a crash. This energy and momentum can be dissipated by air bags, which convert it into the work of compressing the gas within the device. The helmets used by cyclists and bicyclists work on a similar principle in that a compliant helmet absorbs some of the energy of impact, which is, therefore, not transmitted to the brain. Many studies have demonstrated the benefits of using seatbelts and air bags with mortality reductions ranging from 41 to 72% for seatbelts, 63% for air bags, 80% for both, and 69% for child safety seats.[58] Seatbelts and air bags have also significantly reduced the incidence of injuries to the cervical spine, brain, and maxillofacial region by keeping the forward momentum of the passenger to a minimum and preventing the head from striking the windshield.[44] Also worth mentioning is the headrest that has decreased whiplash-type injury by 70% by preventing a difference in momentum between the head and body and hyperextension of the neck in rear-end collisions.[64]

Despite their effectiveness, air bags can be responsible for injury in motor vehicle crashes. Approximately 100 air bag–related deaths were confirmed by National Highway Traffic Safety Administration (NHTSA) over a 5-year period, many associated with improper restraint of small adults or children in front-seat locations. Additionally, a spectrum of minor injuries such as corneal abrasions and facial lacerations have been seen in low-speed impacts. Injuries can occur from the use of safety belts, as well. Lap seatbelts can cause compressive injuries such as rupture of the bowel, pelvic fractures, and mesenteric tears and avulsions. They can also act as a fulcrum for the upper portion of the trunk and be associated with hyperflexion injuries such as compression fractures of the lumbar spine. As a consequence, newer automobiles are required to have the more extensive and protective lap and shoulder harness style belts. Even still, shoulder harnesses can cause intimal tears or thrombosis of the great vessels of the neck and thorax and fracture and dislocation of the cervical spine in instances of submarining, where the victim slides down under the restraint system.[65] Even when a shoulder harness system works as intended, clavicular and rib fractures or perforations of hollow viscera in the abdomen secondary to a compressive-type mechanism can occur.[66,67]

SPECIAL CONSIDERATIONS

Pediatrics

Differences have been noted between adults and children in both patterns of injury and physiological responses to injury. In one analysis of adults and children sustaining comparable degrees of injury from blunt trauma, significant differences were noted in the incidence of thoracic, spinal, and pelvic injuries in children. Although the overall incidence of injury to the brain is higher in blunt pediatric trauma, thoracic and pelvic injuries occur less frequently.[68]

Overall mortality is generally higher for adults than it is for children sustaining comparable degrees of injury. When assessed by mechanism, however, mortality is slightly higher for children in motor vehicle crashes.[69]

The most significant difference between adults and children is in the compliance of the bony structures. This difference is seen commonly in the resilience of the chest wall. The incidence of rib fractures, flail chest, hemo-pneumothorax, and injury to the thoracic aorta in children is significantly less than that in adults, though the incidence of pulmonary contusion is higher. Because of this resilience, the chest wall can absorb a greater impact in children while demonstrating less external sign of injury. In children the index of suspicion for a pulmonary contusion, in the absence of rib fractures, must be higher than in an adult.

Injury to the spinal cord is rare in children, representing only 1–2% of all pediatric trauma. The cervical spine is injured in the majority of cases (60–80%), compared to 30–40% in adult injury. The immature spinal column has incomplete ossification, a unique vertebral configuration, and ligamentous laxity, which accounts for this difference in pattern of injury. The proportionally larger head and less developed neck musculature of younger children (10 years old) account for more torque and acceleration stress in the higher cervical spine during injury, as well. Young children have high rates of dislocations and spinal cord injury without radiographic abnormality (SCIWORA), and these are more likely to be seen at the upper cervical levels. As older children have a low fulcrum of cervical motion (C5–C6) and more ossification and maturity of the vertebral bodies and interspinous ligaments, they have a high incidence of fractures in the lower cervical spine.[70] SCIWORA is associated with 15–25% of all injuries to the cervical spine in pediatrics and represents a transient vertebral displacement and realignment during injury, resulting in damage to the spinal cord without injury to the vertebral column.

Childhood obesity is recognized as a leading public health issue in the United States. Childhood obesity is defined as an age and sex-specific body mass index (BMI) at the 95th percentile or higher, while overweight is defined as a BMI

between the 85th and 95th percentiles. Based on this definition, 32% of all children were overweight or obese from 2011 to 2012.[66] When compared to their nonobese counterparts, obese children between the ages of 2 and 5 who are injured in a motor vehicle collision are at an increased risk for major injuries to the brain and chest. Obese children above the age of 5 involved in an MVC are at an increased risk for major thoracic and lower extremity injuries in comparison to nonobese children of the same age and sex.[71]

Nothing has reduced the incidence of injury to children and infants more than the mandatory use of safety belts and restraints. The problem still to be faced is the different contours and shapes with infant restraints. Also, there has been increased interest in the issue of pediatric restraint systems because of a number of injuries related to air bags. It is recommended that restrained infants and children not be placed in a front seat and that all children under age 12 ride in the rear seat. Injuries related to air bags have ranged from minor orthopedic trauma to fatal injury to the brain.[72]

Unfortunately, child abuse is a reality in the pediatric population and must be considered when evaluating a child who has been injured in less than clear circumstances or has multiple injuries of varying ages. Although injury to soft tissue is the most common presentation, fractures follow as a close second. There is a high rate of spiral fractures of the humerus and femur secondary to a torsional force, applied by an adult grabbing the child's extremity in a twisting motion. Injury to the brain is the third most common injury, with skull fractures thought to be secondary to direct blows to the child's head or the dropping and throwing of the child. Intracranial hemorrhage has been noted in the "shaken impact syndrome" and is thought to result from significant acceleration and deceleration forces followed by direct force transfer with impact. Subdural and subarachnoid hemorrhages can often result, as blood vessels between the brain and skull are ruptured. Retinal hemorrhage may also be identified in this pattern of injury and occurs in approximately 3% of cases. Impact injury to the abdomen is common in child abuse and can result in injury to solid organs (liver, spleen, or kidney), duodenal hematoma (sometimes with delayed symptoms of intestinal obstruction), pancreatitis, injury to the colon or rectum, or mesenteric bleeding. In addition, falls from even very small heights may cause severe intracranial hemorrhage in the infant or child.[73]

Pregnancy

Injury to pregnant women in motor vehicle crashes is estimated to account for 1500–5000 fetal deaths each year. There has been little investigation into specific forces and the kinematics of injury in pregnancy. Several studies have demonstrated that the most common cause of fetal demise in motor vehicle crashes with a viable mother is placental abruption. The biomechanics of this injury involves generation of tensile and shear forces, with the circumferential forces in the uterine wall inducing a shear strain across the placental surface, resulting in placental strain and subsequent abruption. Shorter women have a higher incidence of fetal demise with automotive crashes because of their close proximity to the steering wheel. As in other populations, restraints have been demonstrated to increase survival in both mother and fetus.[74]

Geriatrics

Trauma remains a disease of the young, though there is a significant incidence of morbidity and mortality in the elderly population. Approximately 1 million Americans over the age of 65 are affected by trauma annually and it is the ninth leading cause of death in this age group.[75] The most common mechanisms of injury in the elderly are falls, fires, and vehicular trauma.

When patients with similar injury levels are compared with respect to age and mortality, the incidence of fatality in older persons is 5- to 10-fold higher than it is in the younger population. It is not the severity of injury that is crucial, but rather the incidence of comorbid factors in this population, especially cardiac and vascular disease. Most likely it is the patient's inability to demonstrate a cardiovascular reserve that is a contributing factor to their subsequent increased morbidity and mortality. The Injury Severity Score and other predictors of outcome do not hold up in the geriatric or pediatric populations. Another significant finding in this population is that most (as many as 88%) of these patients never return to their previous level of independence.[76]

The incidence of falls in the geriatric population is high, with an annual incidence of approximately 30% in those over 65, and approximately 50% in those over 80 years of age. Falls account for approximately half the cases of geriatric trauma. Most falls in the elderly occur from standing with mortality secondary to the comorbid factors mentioned earlier.[77] The propensity for fracture is also increased secondary to a loss of bone density with aging, with hip fractures being one of the most common injuries.

BLAST INJURIES

Blast injuries are among the most dramatic and devastating wounds encountered by the trauma community. The National Counterterrorism Center documented approximately 11,800 terrorist attacks in 2008, resulting in over 54,000 deaths and injuries.[78] Although the number of terrorist incidents decreased from the previous year, overall fatalities had increased.[78,79] The vast majority of these attacks occurred in the Middle and Far East, but the United States was not immune from blast incidents. The Bureau of Alcohol, Tobacco, and Firearms noted an average of 182 annual injuries and 23 annual deaths from explosive incidents in the United States from the period of 2004 to 2006.[80]

Blast injuries are broadly categorized as primary, secondary, tertiary, quaternary, and quinary, based on a taxonomy of explosive injuries published by the Department of Defense in 2006.[81] The trauma practitioner should be familiar with each of these patterns of injury and be able to predict associated injuries from each category (**Table 1-2**).

TABLE 1-2: Department of Defense Classification of Blast Injuries from Explosive Devices

Classification	Definition	Common injuries
Primary	Blast overpressure injury (blast wave) Direct tissue damage from the shock wave Air-filled organs at highest risk (ears, lungs, gastrointestinal tract)	Tympanic membrane rupture Blast lung Gastrointestinal tract perforation/hemorrhage Ocular Concussion
Secondary	Primary fragments—from the exploding device (either from pieces of the device itself or from projectiles placed intentionally into the device to increase the lethality of the device) Secondary fragments—from the environment (glass, small rocks, etc)	Lacerations Penetrating injury Significant soft tissue injury (including traumatic amputations) Ocular
Tertiary	Acceleration/deceleration of the body onto nearby objects or displacement of large nearby objects onto an individual	Blunt trauma Traumatic amputation Crush injury
Quaternary	Injuries due to other "explosive products" effects—heat, toxidromes from fuel and metals, and so on	Burns Inhalation injury
Quinary	Clinical consequences from postdetonation environmental contaminants including bacteria, radiation, and tissue reaction to fuels and metals	Radiation Sepsis

The damage imparted by the initial shock wave after an explosion is called the primary blast injury and is determined by several factors. These include the type and size of the explosive charge, the distance from the charge to the target, the medium through which the wave will pass, and the composition of the target.[82] The strength of the charge is typically expressed by a standardized methodology known as the detonation velocity. TNT, having a detonation velocity of approximately 6900 m/s, is the frequent benchmark to which all explosives are compared. Some modern plastic explosives have detonation velocities in excess of 10,000 m/s.[83]

The medium through which the shock wave travels is an important determinant of the energy that will eventually reach the target. Air is extremely compressible and, as such, absorbs much of the energy of the initial blast. Water, being relatively incompressible, transmits much more of the energy farther.[84] In water, the formula derived by Arons yields the pressures generated in underwater explosions in terms of the size of the charge (w) and the distance from the charge to the victim (R): The constant 2.16×10^4 is specific for TNT.[85]

$$p_w = 2.16 \times 10^4 \left(\frac{w^{\frac{1}{3}}}{R} \right)^{1.13}$$

Using a derivation of this formula, Hirsch demonstrated the relationship of pressures within differing mediums at the same distance from the blast as functions of their density and speed of sound in each medium.[86]

$$P_a = P_w \frac{D_a C_a}{D_w C_w}$$

where P_a and P_w are relative shock wave pressures occurring after blasts in air and water medium, respectively, D is the density of the medium and C is the speed of sound in the medium.

Thus,

$$\frac{D_a}{D_w} = \frac{1.16}{1000} \text{ and } \frac{C_a}{C_w} = \frac{1}{5}$$

$$P_w = P_a \frac{5000}{1.16}$$

Demonstrating that the pressure wave of a similar charge, at a similar distance will be 4310 times greater when occurring underwater as compared to in air.

The characteristics of the target are also important for several reasons. First, when the shock wave travels through a medium less dense than the target, such as air to a human body, much of the wave is reflected and not absorbed by the victim.[84] When the target and medium have similar densities, such as a human body and water, the energy of the sound wave is almost entirely transmitted to the victim. On land or under water, when the wave goes from a denser to less dense medium, rarefaction waves cause cavitation.[87] This mini explosion causes shear stress and explains why most damage occurs in the air-filled organs of the lung, bowel, and tympanic membrane and, especially in underwater explosions, where the rarefaction wave goes from dense skull to brain.[86,87] Underwater explosions, therefore, are several-fold more dangerous than those occurring on land. Conversely, primary blast injuries are less common in open-space explosions, but this changes when the explosion occurs within a confined space, which allows the blast wave to reflect off of fixed structures.[88]

Rupture of the tympanic membrane is the most common manifestation of primary blast injury, occurring in up to one half of patients injured in an explosion.[89] An intact

tympanic membrane, however, is not a strong negative predictor of severe blast injury.[90,91] The most common fatal injury among blast victims is to the lung, often referred to as "blast lung injury." The blast wave causes tissue disruption at the capillary–alveolar interface, resulting in pulmonary edema, pneumothorax, parenchymal hemorrhage, and, occasionally, air embolus from alveolovenous fistulas.[89] Clinical diagnosis of blast lung injury is dependent on the presence of the triad of hypoxia, respiratory distress, and bilateral or central infiltrates on a chest radiograph.[92] The infiltrates are usually present on admission and can worsen with aggressive fluid resuscitation. These central infiltrates are also referred to as "butterfly" or "batwing" infiltrates and are pathognomonic for blast lung injury, in contrast to the peripheral infiltrates commonly seen with pulmonary contusions from blunt injury. Management of the ventilated patient with blast lung injury includes avoidance of positive pressure ventilation, minimization of positive-end expiratory pressure (PEEP), and judicious fluid resuscitation.[89] Fluid management in these patients will often be challenging due to associated injuries from secondary and tertiary blast effects, which often require greater amounts of intravenous fluid for adequate resuscitation.

Secondary blast injuries are created by debris from the explosive device itself or from surrounding environmental particles. Many devices contain additional munitions consisting of nails, pellets, ball bearings, and scrap metal designed to increase the lethality of the explosion. Fragments from the surrounding environment, including glass and small rocks, can become secondary missiles, as well. Secondary blast injuries are more common than primary blast injuries as the debris and added fragments travel over a much greater distance than does the shock wave from the primary blast.[93] Lacerations, penetrating injury, and significant soft tissue defects are the most common injuries seen from secondary blast injuries.

Tertiary blast injuries are caused by the body being physically thrown a distance or from a solid object falling onto a person as a result of the explosion. Most tertiary injuries are from a blunt mechanism, and crush injuries or traumatic amputations are not uncommon. Quarternary and quinary blast injuries have only recently been defined. They are miscellaneous blast injuries caused directly by the explosion but often due to other mechanisms, such as burns, inhalation injuries, and radiation effects.

Children injured by explosions suffer a different injury pattern as compared to adults.[94] Children are more likely to sustain life-threatening injuries and a traumatic brain injury. They are less likely to have an extremity injury or significant open wounds. The adolescent injury pattern resembles that of the adult, although they are more likely to have fewer internal injuries, more contusions, and have a higher risk of requiring surgical intervention for mild or moderate wounds when compared to adults.

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