THORACIC TRAUMA

Edited by DeWitt C. Daughtry, M.D.

Little, Brown

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Little, Brown and Company
Boston

First Edition

Library of Congress Catalog Card No. 80-80593

ISBN 0-316-17380-0

Printed in the United States of America

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THORACIC TRAUMA

To my wife Lucille

and to the many patients
who have made this volume possible

PREFACE

This book presents a comprehensive account of the management of thoracic trauma as seen primarily in civilian life but also in certain combat situations. The contributing authors are thoracic surgeons who have had vast experience in their particular areas, and thus each chapter is highly authoritative. *Thoracic Trauma* should serve as a guide for those who are involved in the treatment of thoracic injuries in well-equipped hospitals or in trauma centers.

Trauma, although largely preventable, is the fourth highest cause of death among all ages in the United States and is the leading cause of death under the age of 45 years. Thoracic injuries are becoming increasingly significant because of the large number of automobile accidents and because of the increase in various forms of violence. Twenty-five percent of automobile accident deaths are attributable to the thoracic component of the injury. When associated with other serious injuries, the thoracic component is an even greater contributing factor in disability and death. During wartime, with its attendant rise in bullet and shrapnel wounds, thoracic injuries account for a much higher mortality and morbidity.

In addition to the emphasis placed on management techniques, attention has been directed toward transportation of the injured, the communications response system, and triage at the site of the accident. Fortunately, there is emerging a new specialty devoted to emergency medical care. This should accomplish at least two things: first, better organization of emergency care for the patient in accident care centers; and second, better triage and organization of the consultation team to care for these patients. A review of autopsy material reveals that a large percentage of accident victims could have been salvaged by a better system of care.

In recent years physicians, industry, public officials, law enforcement agencies, and the federal government have become more deeply concerned with accident causation and prevention. Stimulated by the recent fuel shortage, this concern has led to manufacture of lower-horsepower cars and a lowering of speed limits. It is regrettable that the automobile manufacturing industry has been so careless in its attitude regarding safety features. However, measures have been taken to force the adoption of safer engineering principles, and this should further enhance accident prevention and decrease the severity and extent of injuries.

In addition to industry facing its responsibilities, we, as responsible citizens, should become involved and exert pressure upon organizations and individuals to face up to the major problem of accident prevention. Human carelessness causes more accidents and deaths than all other factors combined.

I wish to thank the individual co-authors for their superb contributions to this volume. I wish to express my gratitude to Minor Duggan, M.D., Head of the Department of Publications of the Miami Heart Institute, and also Mrs. Klara Soos of the same department. I would also like to express my appreciation to Mrs. Bronia Barbash, Medical Librarian, and Mr. Rand Johns, Medical Illustrator, of the Miami Heart Institute. Special thanks are due to my wife Lucille for her perseverance and to the staff of Little, Brown and Company for their fine cooperation.

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Chapter 7

THORACIC TRAUMA

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1. THE FORCES PRODUCING CERTAIN TYPES OF THORACIC TRAUMA

Safuh Attar
William H. Kirby, Jr.

The primary purpose of this chapter is to review briefly current information about the biophysics of trauma modalities and to relate this knowledge to clinical observations. It is intended as a contribution toward improved understanding and prevention or minimization of serious injury in the future.

Thoracic injuries may be categorized as either penetrating or nonpenetrating. While those sustained during military activities are usually of the penetrating kind and are due to fragmentation of various types of missiles, the injuries seen in civilian practice are brought about primarily by automobile crashes and comprise a wide variety from each category, both singly and combined. Often these thoracic injuries are complicated when other organs are also involved. In addition to automobile crashes, there are other causative factors such as aircraft crashes, athletic sports, accidental falls, riots (injuries caused by anti-riot weapons), accidental blasts, and criminal and personal conflicts. The common process in all cases is, of course, one of energy transfer. The outcome is dependent on a number of factors, such as the nature of the energy delivery or loading; the age, size, and shape of the recipient; the location and manner in which the energy is dissipated; the environment and accessibility to treatment; the time the accident occurs; and the diagnostic and therapeutic facilities available.

THE LOADING PROCESS

Two major difficulties prevent a comprehensive understanding of the effects of the various forms of impacting energy on the thorax and its component subsystems. In the first place, we know little about the basic mechanics of thoracic tissue, individually or as a whole. The body has often been described as a great nonlinear damping system. Clemedson and associates [7] have pointed out the extreme complexity of the human body, particularly from a mechanical standpoint, and have described it as a heterogeneous viscoelastic mass. The same authors have also noted that an impacting source imparts mechanical and thermal disturbances to the body, with the former the more important. This is followed by a deformation and displacement of tissues at and near the points of contact, with the development of a pressure wave or pulse that then propagates through the tissue until damped out or further transmitted out of the body.

The role of biomechanics, by applying physical engineering and mechanical principles to the study of bodily trauma, is to provide a better understanding of the cause and effects of the mechanisms and processes involved in thoracic trauma. Goldsmith [12] speaks of these principles and their application to head injuries, but they apply equally well to those of the thorax. Specifically, the principles of continuum mechanics or of the dynamics of discrete systems can be applied to mathematical models of the response of the thorax and its attachments to a given external stimulus, which in turn leads to a prediction of the history of the field parameters, such as stress, strain, displacement, acceleration, pressure for the entire domain, and the determination of the motion of specified elements. The tools of experimental mechanics can also be employed to measure physical changes produced by impact or impulsive loading in replicas of the thorax, which could be animal specimens, cadavers, or inanimate models. Tests of this type on living animals are accompanied most frequently by concurrent physiological measurement and subsequent pathological examination for the purpose of correlation. Data from such experiments serve the following purposes: to substantiate or disprove theoretical hypotheses concerning trauma mechanisms and their patterns; to establish tolerance levels and failure limits for various types of loading; to assess the efficacy of protective devices and

Supported by the United States Public Health Service, Research Grant Nos. HE-09341-07 and HE-09341-08.

environments; to provide statistical data; and to make available information concerning the basic mechanical properties of the components involved.

From the standpoint of mechanics, the causes of thoracic injuries may also be divided into categories on the basis of the manner of the loading application. An impact or blow upon the thorax may be due to a solid object traveling at an appreciable velocity, e.g., missiles or bullets; automobile components under crash conditions; slower moving objects such as knives; or even the intensive pressure waves caused by a blast. Each of these impacts may be further subdivided. In contrast we might consider the static or quasi-static loading produced by a relatively long-term crushing action. These situations encompass different physical phenomena, and their mathematical representations would emphasize different loading and response factors. Some of the injuries discussed later in this chapter demonstrate that clinical effects can often be distinguished according to the type of load that caused the injury.

PENETRATING THORACIC TRAUMA

Thoracic penetration by fast-moving particles such as bomb fragments or bullets is of concern to both military and civilian surgeons. Medical interest in the battle casualty includes the type and anatomical location of wounds as well as the correlated visceral damage and the causative agents, and has existed since the earliest days of organized combat. Much speculation and some observation has been devoted to the magnitude of a missile wound and its correlation with momentum, kinetic energy of the missile, or the rate with which the energy was dissipated. It has become apparent that all physical phenomena connected with wound formation are direct results of kinetic energy, meaning that the mass of a missile is not as important as the velocity with which it strikes.

Amato and associates [2] studied the events that occur within various tissues after high-velocity bullet-wounding. As tissues are struck by a missile, multiple disruptive forces combine to produce tissue destruction. The major forces consist of the velocity, the mass, and the change in shape of the bullet when it reaches the tissue. The most important force is the velocity of the missile within the tissues. The initial velocity on impact is called *striking velocity*. If the bullet leaves the tissue, the remaining velocity is the *residual velocity*. The effective velocity of the injury is the difference between the two velocities: $(V_1 - V_2)$. The kinetic energy (KE) imparted to the tissues expressed in foot pounds can be written:

$$KE = M(V_1^2 - V_2^2)/2g$$

where M equals mass, and g equals gravity. If small amounts of energy are released within the tissue, as in the low-velocity wound, destruction will be confined to the pathway of the bullet. The energy released by high-velocity missiles forms initial shock waves with a pressure up to 100 to 200 atmospheres, imparting momentum to the tissues both forward and laterally. These tissues accelerate in an outward direction, creating a large space known as the *temporary cavity*. The tissues undulate and undergo stretching and compression. Because of the heterogeneity and varied density of the tissues, there is added mechanical damage due to the shearing effect between these tissues. The retentive forces that combat the disruptive forces vary with individual tissues. The characteristic pattern of injury is determined by the density of tissues combined with the degree of elasticity and cohesion within these tissues. Using high-speed photography and roentgenography, Amato and his colleagues [2] demonstrated experimentally the formation of temporary cavities in muscle, liver, and lung. Although previously never visualized and thought possibly nonexistent, a temporary cavity was formed within the lung tissue by a missile striking at 3000 feet per second. In angiograms a small cavity within the lung parenchyma was demonstrated by showing disruption of the blood vessels. The temporary cavity was smaller and less impressive than that in muscle or liver tissue. The elastic fibers within the spongework of lung parenchyma absorb the energy and recoil so that the missile tract is hardly perceptible.

Stab wounds and wounds caused by large objects are in a separate category. The loading process is of a much lower order and the outcome somewhat more predictable in that the damage is more closely related to the structural and functional deficits of the components directly traumatized.

NONPENETRATING THORACIC TRAUMA

These injuries may also be subcategorized according to the loading process. Essentially we are

concerned with the effects of pressure waves due to blast and impulses generated by blunt objects striking the body.

Blast Injury

When an explosion occurs, a blast or shock wave develops that is similar in all fundamental respects to those produced by supersonic aircraft or by missiles. The parameters of a blast wave have been carefully studied from a biological point of view and comprise: (1) the peak over pressure and (2) the positive duration of this peak over pressure. According to DeCandole [9], one might visualize a shock wave by considering the consequences of a very brief but violent blow impinging uniformly on the side of the body. A significant part of the energy is transmitted through the tissues.

Clemedson [6] points out that an absorbed blast wave is propagated through the body as a pressure wave. The latter may be higher than in the primary wave due to so-called damping up of pressure against the body surface. The degree of injury to a tissue or an organ from a shock wave depends on their physical properties in relation to the characteristics of the wave. If the shock wave derives from a large explosive charge at a considerable distance, the destruction is related chiefly to the peak pressure. However, if the shock wave is caused by a small charge at a short distance, then the damage will depend primarily on the impulse.

Not all tissues are equally susceptible to blast injury. Most internal organs are considered virtually incompressible and subject to minimal displacement. The most vulnerable parts of the body are those containing air or gas, namely, the ear drums, lungs, and intestines. This is thought to be due essentially to a bursting or shredding effect that occurs at the interface and strikes the internal organs one by one during the succeeding millisecond or less. Thoracic structures, especially the lungs, cause a marked distortion of the pressure wave because of large differences in tissue density. The pathophysiological events following exposure of an animal to a high-compression, high-velocity shock wave are characterized by lesions appearing in various internal organs, particularly the air-containing structures, without signs of external injury. The clinical picture may be compounded by additional trauma due to flying debris and/or bodily displacement and impact. Furthermore, there may be other significant environmental influences, depending on the nature of the explosion, temperature conditions, and so on.

The question of the immediate cause of death in blast injury has been reviewed extensively. While a number of theories are proposed, the final impression is that the immediate cause of death may differ under different conditions. The various possibilities may be classified as respiratory, circulatory, cerebrospinal, and others. In severe blast injury, massive pulmonary hemorrhage with obstruction of the respiratory passages by blood and froth leads to suffocation. One view, however, is that the respiratory symptoms apparently are not the cause of death but the consequence of circulatory failure. This suggests that attention should be focused on the mechanisms associated with the onset of circulatory shock as a sequel to the various forms of blast injury. Much more work is needed to help us clarify our understanding of the pathophysiological and biophysical aspects of this form of injury.

Blunt Injury

Information on the effects of blunt trauma to the body is very scanty, especially as it applies to the thorax. Clemedson and associates [7] believe that the pathoanatomical damage to the lungs and heart may be essentially the same in the three different types of loading (penetrating, blast, or blunt), although the thresholds and injury-producing values of impact velocity and impact energy in blast exposure are not valid for the other two types. Trinkle and colleagues [35] studied the anatomical and physiological lesions of the lung produced when a .38-caliber blank cartridge was fired against the chest of anesthetized dogs. Disruption of the alveolar-capillary integrity caused a bruise of the underlying lung with hemorrhage and edema of both the alveolar and interstitial spaces. The resulting pulmonary contusion has been described in blast injuries, direct chest trauma, and recently in penetrating wounds of the chest produced by high-velocity missiles.

Deceleration Injury

Perhaps an even more difficult correlation problem occurs when the body is the moving entity and comes to a sudden halt, with or without the impaction of external objects. A great deal of work has been done, including voluntary human experiments, by Stapp [34] and other investigators regarding the effects on body structure and function

of sudden stops (deceleration trauma). This kind of investigation obviously permits the gathering and assessment of data under reasonably controlled conditions.

The form of body deceleration trauma in which there is also impact with external objects presents a complex picture due to the combination of effects from tissue displacement by both blunt and penetrating forces. This kind of injury is often seen as a result of automobile accidents. Unfortunately, while we can speak of forces, velocity, acceleration, kinetic energy, and other physical factors, we are not yet in the position of relating them to types and degrees of injury. Each serious automobile accident is so different that predictions of outcome, except in a very broad sense, are impossible.

SELECTED TYPES OF INJURY

This discussion will be concerned mainly with the biomechanics of blunt trauma, which causes the majority of thoracic injuries encountered in civilian and military practice. Of 585 deaths from traffic accidents reported by Kemmerer and associates [16], the percentage of thoracic injuries was as follows: rib fractures, 39 percent; hemothorax, 28 percent; lung laceration, 10 percent; ruptured great vessel, 10 percent; lung contusion, 6 percent; lacerated diaphragm, 5 percent; sternal fractures, 5 percent; myocardial injury, 6 percent; and lacerated trachea, 1 percent. In almost 25 percent of those deaths, death could be attributed to the thoracic trauma. In 80 percent of those with rib fractures, there was an associated serious intrathoracic injury.

The thoracic skeletal system is frequently involved in direct injury. This results in rib fractures, with or without an accompanying fractured sternum, depending on the mode of injury. Children and adolescents, whose chests are more flexible, may experience greater deformations of the chest cage without rib fractures. The upper ribs are protected to some extent anteriorly by the clavicles, posteriorly by the scapulae, and laterally by the heavy musculature of the upper thorax and shoulder girdle. Rutherford [33] points out that for this reason fractures above the fifth rib imply considerable trauma, not uncommonly associated with serious intrathoracic injuries such as rupture of the tracheobronchial tree. The fifth through the ninth ribs are the ones most frequently involved in blunt thoracic trauma. A direct blow may cause breaks at the point of impact. With crushing injuries, however, the same is seldom the case. More commonly the ribs give way at the point of maximum convexity in the region of the costal angle.

Experimental work using human cadavers has been carried out in which thoracic response and tolerance levels were reported by Patrick, Mertz, and Kroell [30]. In this research, embalmed cadavers were seated on the sled of a horizontal accelerator and driven to a specific velocity. Having reached the desired speed, the sled was stopped abruptly. During deceleration the cadavers slid forward relative to the sled and impacted their chests. A flat and padded surface 6 inches in diameter was used as the chest impactor. The corresponding displacement of the sternum relative to the thoracic vertebrae was obtained by photographically monitoring the movement of a probe attached to the sternum and passed through the thoracic cavity. Four rib fractures were observed at a sled velocity of 16.8 miles per hour (mph) and a maximum chest load of 1340 pounds, while extensive fractures resulted at 19.5 mph with a load of 1859 pounds. The maximum chest deflection was 2.25 inches at 1545 pounds. Nahum and colleagues [27] studied the interrelationships between force of chest impact, chest wall deflections, and resulting trauma in blunt sternal impact to the human cadaver with high-speed photography. The preliminary studies showed great variations in response to trauma associated with age. The younger cadaver specimens suffered virtually no skeletal damage, whereas the older specimens sustained extensive damage, in the form of rib fractures (7 to 20 rib fractures), sternal fractures, and lacerations of heart and lungs. In a similar experiment Kroell, Gadd, and Schneider [18] concluded that force per se cannot be correlated with thoracic injury. The magnitude of skeletal deflection and the time required to develop such deflections would appear to be much more directly associable with thoracic injury, both skeletal and visceral. Significant skeletal damage was found at both the 11 and 16 mph levels. The authors suggested that a skeletal deflection not exceeding 51 mm (2 inches) for midsternal blunt impact should be a reasonable criterion for the preclusion of serious thoracic trauma.

Although visceral thoracic injuries are often associated with injury to the chest wall and sternum,

they are sometimes sustained without chest wall involvement and include lung contusions, ruptured bronchus, ruptured diaphragm, ruptured aorta, and cardiac injuries.

Lung contusions may result either from blast injury or blunt trauma. In military practice they are caused by nonpenetrating wounds from high-velocity missiles, high-explosive shells, and detonations. In civilian life, they are most frequently secondary to automobile accidents, although falls and industrial mishaps may also be causative. The pulmonary injury can be traced to the direct effect of blast-induced variations of the environmental pressure. The extent of the injury depends on the magnitude of the loading force, the physical characteristics of the shock wave, the medium that transmits the shock wave, and the dissipation of energy through the chest wall and pulmonary tissue. Following a blast or violent blow to the chest, much of the energy is reflected, but at least part is transmitted to the thoracic viscera. Compressibility permits displacement, and whenever tissues of different density exist side by side, the amount of displacement and its time course vary from point to point with subsequent distortion and tearing. Within the lungs, the alveolar septa are most affected and are torn so that the parenchyma shears away from the vascular structures and the alveolar epithelium is shredded. The outcome is failure of the blood-air barrier, with collection of blood in the pulmonary tissue and escape of air into the pulmonary veins.

Although extensive data have been accumulated on blast injuries, relatively scant information is available on blunt or deceleration trauma to the thorax. Since, for obvious reasons, destructive impact tests cannot be made on living human subjects, most of the facts have therefore been obtained from animal experimentation. However, the thorax of quadruped mammals is quite different in shape from that of man. This difference influences the behavior of the intact thoracic cage under impact loading, and caution should be exercised in the interpretation of findings when applied to man.

Beckman and Palmer [4] have studied the thoracic force-deflation characteristics of an impactor in the living subhuman primate as a step toward the analysis of cardiothoracic injuries. Mechanisms of injury were investigated by subjecting Rhesus monkeys seated on a stationary, freely movable sled to static and dynamic chest wall displacement. This was achieved by means of a cartridge-fired projectile equipped with a diameter impact plate that is 3 inches in diameter and an accelerometer compensated-force transducer. Forces up to only 30 pounds were developed in static pressure tests for 2-inch deflections but reached 700 pounds for the same deflection using dynamic tests. These authors postulate that the force generated on dynamic deflection of the anterior chest wall, aside from the static reaction, may have components due to: (1) the spring mass effect of the anterior chest wall, (2) the compression of air in the lungs, and (3) the viscous or viscoelastic behavior of the thoracic tissue.

Tracheal and bronchial injuries are seen with increasing frequency following blunt trauma, especially when this has occurred in an automobile accident. Rupture separation of the cervical portion of the trachea usually succeeds a direct force, tending to avulse the larynx and cricoid cartilage from the trachea. Intrathoracic rupture of the trachea seems to result from a sharply localized force applied to a relatively elastic chest wall anteriorly, leading to compression of the trachea against the rigid vertebrae posteriorly. This in turn causes the membranous part of the trachea to be torn from its cartilaginous attachment, or, by being sharply overstretched, the trachea is fractured just above the carina. This mechanism is apparently similar to that of traumatic bronchial rupture. The following mechanism is suggested by Lloyd and colleagues [21] for bronchial ruptures: when blunt trauma is applied to the anteroposterior diameter of the chest, this diameter decreases while the transverse dimension increases. The lungs, owing to their negative intrathoracic pressure, remain coapted to the chest wall and are subjected to tension at the carina. If the lateral excursion of either lung is carried beyond the point of elasticity of the bronchi, a tear results at or near the carina. These factors are augmented when the blow is administered to the chest in the anteroposterior diameter when the glottis is closed.

Another injury that is seen with increasing frequency following blunt trauma is rupture of the diaphragm. It occurs after a steering wheel injury to the lower part of the chest and upper abdomen. In mining and industrial areas, diaphragmatic hernia is more likely to result from falls of coal and rock, or from crushing by trucks and pit cages. It is seen less frequently on the right (5 to 20 percent), probably because of the protective effect of the liver on the right leaflet of the diaphragm. Al-

though the heart, pericardium, and lungs may act as cushions to the left leaflet, they are less effective. Most ruptures of the diaphragm seem to occur in association with severe injuries to the musculoskeletal system, especially trauma to the pelvis, lumbosacral spine, and lower extremities. The patients most likely to suffer diaphragmatic rupture from increased intra-abdominal pressure are infants, young children, and pregnant women whose abdomens have been run over. In the Evans' series [11] five out of seven hernias due to nonpenetrating injury followed such accidents; four of the patients had fractures of the pelvis, none had fractured ribs, and all escaped visceral injury.

The site of rupture of the left dome of the diaphragm correlates well with the embryological point of weakness in its posterior and lateral walls. It would appear that the relatively weak diaphragmatic structure yields between the solid viscera of the abdomen and the pliant thoracic structures when a sudden force is applied to either the thoracic or abdominal wall. According to Desforges and associates [10] the direction of trauma per se is unimportant in the disruptive mechanism. Forces applied to the anterior abdomen and the flanks are transmitted equally in all directions, and since the diaphragm offers the least resistance, it fails by disruption. Another possible mechanism suggested by Desforges is that forces compressing the chest from opposite directions could rupture the diaphragm like the membrane of a drum. Under these conditions the tear would be expected to occur in the line of compression and to be centrally located. Once a rent occurs, the abdominal organs tend to herniate into the chest due to the greater intra-abdominal pressure. In Marchand's study [23], the intraperitoneal pressure after a rent varied from +2 cm to +10 cm of water during quiet breathing in the supine position, while the intrapleural pressure varied from −5 cm during expiration to −10 cm during inspiration. This gradient of 7 to 20 cm could exceed 100 cm with maximum respiratory effort. A search of the literature for an estimate of the magnitude and distribution of the forces that lead to rupture of the diaphragm was not fruitful. One can only infer that they are of the same magnitude as forces leading to fracture of the pelvis or lumbar spine.

With the advent of the automobile, high-speed deceleration impact injuries have markedly increased. Ventricular impact, abrupt transfers of kinetic forces to the victim, and sudden decelerations of bodily viscera and blood column momentum all operate sequentially to produce cardiovascular injuries [19]. The extent and severity of these injuries is determined by several biomechanical interactions, including (1) the magnitude of deceleration, (2) the total duration of exposure to the forces of deceleration, and (3) the rate of time change of deceleration, according to Newtonian laws of dynamics: $F = MA$, or in the case of deceleration, $F = MD$, where F is the resultant of forces acting on the viscera, M is the scalar mass of the viscera involved, D is the magnitude of deceleration, V_2 and V_1 are terminal and initial velocities, respectively, and t is the time duration of velocity change.

Traumatic injuries to the cardiovascular system following blunt trauma have been well documented. Parmley and colleagues [29] reviewed 546 cases of fatal nonpenetrating cardiac trauma. They recorded a predominance of myocardial rupture—353 cases; there was also myocardial contusion and/or laceration in 129, pericardial laceration in 36, hemopericardium in 25, and single cases of valvular injury, papillary muscle rupture, and coronary artery laceration. The incidence of cardiac contusion in 507 patients with nonpenetrating chest trauma was reported by Jones, Hewitt, and Drapanas [15] to be 9.41 percent (48 patients). They were all involved in automobile accidents and had evidence of moderate to severe trauma to the thorax. Twenty-two patients had associated rib fractures and 13 had sternal fractures. The physical forces involved in the production of such injuries were summarized by Parmley and associates [29] as follows: (1) unidirectional force against the chest; (2) bidirectional or compressive force against the thorax; (3) indirect forces, i.e., compression of the abdomen and lower extremities resulting in a marked increase in intravascular pressure; (4) decelerative forces, particularly when imparting differential deceleration to the heart and great vessels; (5) blast; and (6) concussive force, usually produced by rapid motion without bringing about demonstrable pathological changes.

In order to investigate the possible mechanism of myocardial injury secondary to blunt trauma the following methods were pursued:

1. An experimental model was developed by Anderson and Doty [3] to study myocardial contusion. Isolated myocardial injury was produced

in 20 anesthetized dogs, using the impact of a captive-bolt handgun with semielliptical steel disc positioned over a point of maximum cardiac impulse. When the weapon was discharged, the bolt was propelled forward, transferring kinetic energy through the intact chest wall to the underlying heart. Nine animals died within minutes of the contusion; the other 11 recovered until they were sacrificed for pathological study. The animals that died immediately after injury showed similar severe, isolated injury to the heart. Circular areas of transmural contusion with subepicardial hematoma and subendocardial ecchymosis were present over the left ventricle, anterior septal area, and cardiac apex. Disruption of surface coronary vessels was not observed. Animals sacrificed at intervals up to 72 hours showed injuries of identical extent and distribution, with progressive resorption of hemorrhage. By three weeks, when the last animal was sacrificed, the heart appeared to have patchy areas of scarring but its appearance was otherwise normal. The outstanding physiological consequences of isolated contusing injury to the myocardium was ventricular tachyarrhythmia, which was observed in every dog at least transiently following trauma, and which often progressed to uniformly fatal ventricular fibrillation. The severity and duration of tachyarrhythmia and consequently the survival of the animal, correlated with the size of the powder charge used in relation to the size of the animal. There is a suggestion that the location of the myocardial injury may determine the type of cardiac arrhythmia. Mosley, Vernick, and Doty [26] reported conduction defects and reduction of heart rate in sinus rhythm following blunt chest injury when the injury force was applied to the right side of the chest, presumably affecting the right atrium and conduction system. This is in contrast to predominantly ventricular rhythm disturbances when the injury force was to the left side of the chest over the ventricular mass.

2. High speed radiographs were obtained at selected time intervals during controlled deceleration of beagle dogs subjected to ± Gz impact on a decelerator [14]. Preliminary data indicate that the heart undergoes considerable inertial movement during deceleration. The average displacement suggests a sine wave function with superimposed damping after one cycle. There is first an upward displacement of the heart followed by an inertial rebound in a downward direction.

Rupture of the heart may occur immediately or within the first two weeks after trauma when softening of the contused myocardial segment occurs. The immediate rupture of a cardiac chamber usually occurs in one of the following ways: the heart may be lacerated by a rib or other bony fragments when the anterior chest is driven in by an external force, or it may be ruptured by forceful compression against the vertebral column, either when it is empty and relaxed or when it is filled. Important factors in determining whether or not heart rupture will occur are the direction of chest compression, and the phase of the cardiac cycle in which the compression occurs. Life and Prince [20] impacted dogs during either ventricular diastole or systole. Of the animals struck during systole, 85 percent had ruptures of one or both ventricles, while of the animals impacted during ventricular diastole none had ventricular rupture. The velocity with which the pressure is applied and the rise of intracardiac pressure produced are also important factors in cardiac rupture.

Rupture of the aorta is among the most frequently encountered severe injuries following blunt trauma to the thorax. Automobile accidents account for 61 to 95 percent of these cases. Greendyke [13] reported 42 cases of traumatic rupture of the aorta in 1259 medicolegal autopsies. One of every six victims of fatal automobile accidents sustains aortic rupture. In about 20 percent of the cases, rupture is caused by a variety of mechanisms such as falling from a height and landing on feet or buttocks, burial under masses of earth or snow, a blow to the chest by a stone, or a blow from a piece of wood flying from a circular saw. The site of rupture is related to the mode of injury. The most common site is the descending thoracic aorta at the isthmus just beyond the origin of the left subclavian artery and is associated with crushing and horizontal injuries. Vertical deceleration trauma occurring with jumps or falls from great heights produces a rupture of the ascending aorta just above the aortic valve. Much speculation has been generated about the mechanisms and forces leading to aortic rupture; this has recently led to experimentation that might shed light on this complex problem. The vast literature on this subject contains theories that are summarized below.

1. *Deceleration mechanism.* The most widely accepted theory to explain the frequency of rupture at the aortic isthmus is based on the fact that