Chapter *6*

PRIMARY BLAST INJURY AND BASIC RESEARCH: A BRIEF HISTORY

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INTRODUCTION

Wound ballistics—the study of penetrating wounds, which have been and will likely remain the primary mechanism of battlefield casualties—is at the heart of any discussion of the medical consequences of conventional war. However, the use of large conventional munitions during this century's wars, the bombing of civilian population centers in World War II, the threat of nuclear weapons, and the advent of modern diagnostic and therapeutic capabilities have military to a newly recognized type of injury—that which occurs from the blast wave alone.

The section of this volume on blast will first discuss some civilian and military experiences with blast injury. This is followed by a brief history of the scientific advances in the blast research field. The physical basis of blast integrated with the mechanisms of blast injury will then lead into a discussion of pathology of *primary blast injury* (PBI). The final chapter of this section will then concentrate on the medical management of the blast casualty.

An explosion may kill or maim a casualty in several ways. Whether it travels through air or water, the blast wave itself may cause internal damage in air-containing organs without leaving any external sign of injury. Such PBI is the principal subject of this section. A blast may also propel fragments into a casualty, causing *secondary blast injury*, or can bodily displace an individual and cause *tertiary blast injury* upon impact.

PBI is most likely to occur during a conflict between opponents who have sophisticated weapons, Even so, the tragic worldwide increase in small-scale terrorist violence has given the medical community opportunities to supplement both wartime medical commentaries and findings from animal-model blast experimentation.

The scientific study of PBI is a twentieth-century phenomenon. The basis for most of what we know about it came out of British and German investigations during World War II. The German research, in particular, was groundbreaking in both its empirical observations and theoretical constructs. Postwar blast research has centered in
States, with some work reported from the Soviet Union and the People's Republic of China. Initial investigations focused on the physical correlates of PBI and its physiological effects. Experiments in the 1980s looked at the effects of repeated exposure to blast waves and the mechanisms of PBI.

BLAST INJURY IN TERRORIST BOMBING INCIDENTS

In modern times, terrorist bombs have become commonplace elements of some political resistance movements. Although they usually do not have the potential for causing the widespread devastation that military explosive weapons can wreak, the simplicity and small size of terrorist bombs allow them to be easily hidden in areas where unsuspecting civilians are likely to congregate. They can thus capitalize on causing severe local damage, and make up in publicity what they may lack in explosive power.

By its nature, a terrorist bombing is an isolated event, and civilian casualties are usually not vulnerable to the same logistical constraints of medical care that military blast casualties might have to face. Instead, they are most likely to be treated at the site by medical professionals who are not under fire, or in nearby hospital emergency rooms that can be quickly reached by ground transportation rather than by air evacuation or by foot.

Blast-injury data from terrorist bombings are limited by the chaotic nature of an unexpected mass-casualty situation descending upon a civilian medical system. Nevertheless, retrospective reviews of many large and small bombings have yielded much useful information on the mortality and incidence of injury resulting from such attacks. Reports from Israel and Great Britain document their all-too-intimate familiarity with blast injury.

General Murbidity and Mortality Rates

Civilian bombings usually result in relatively low mortality rates unless the casualties have been exposed to *(a)* structural collapse, *(b)* a very large explosive charge, or *(c)* an explosion within a structure or vehicle. About 1%–5% of victims will die on the scene from head injuries, and most casualties who are taken to an emergency room will be slightly injured with lacerations, abrasions, and contusions. About 25%–50% of those transported will need admission for treatment or

observation. A small number of victims (1%–15%of those admitted) will have significant thoracic or abdominal blast injury. Those patients are at high risk of complications and about 15%of them will die in the hospital.

The morbidity and mortality statistics from fourteen reports, involving a total of 3,357 casualties in 220 major terrorist incidents, are summarized in Figure 6-1.[1] These events ranged from the bombing of the U.S. Marine barracks in Beirut, Lebanon[2] to Irish Republican Army (IRA) bombings in Northern Ireland and Great Britain.[3–5]

The on-scene mortality ranged from less than 2% in Northern Ireland[4] to almost 70% for the Beirut barracks attack.[2] Immediate mortality was high in the Beirut bombing, as well as in the train station bombing at Bologna, Italy,[6] because a major structure collapsed in each event and many people died of crush injuries. In general, explosions that occurred indoors caused more severe primary and secondary blast injuries because the blast waves were contained within the structures and more debris was thrown about.

Although these civilian bombings generated many casualties, the overall morbidity and mortality rates were, with the exceptions noted above, relatively low; mortality was generally less than 5%. Terrorist bombs usually weigh only a few kilograms and cause mostly lacerations, abrasions, and contusions! In most instances, fewer than half of the victims who were seen in an emergency room were subsequently admitted to the hospital for treatment or observation. For example, most of the victims of civilian bombings in Israel were slightly injured, according to the Injury Severity Score (ISS), a quantitative trauma rating scale.[7] Physicians at Jerusalem's Shaare Zedek Medical Center classified 87% of bombing victims who were seen in the emergency room as being slightly injured, and only 10%as severely injured? Overall, 28%of patients were admitted to the hospital after initial emergency-room evaluation and stayed an average of 16 days. The in-hospital mortality rate was 2.3%.

Terrorists often hide explosives in public gather-

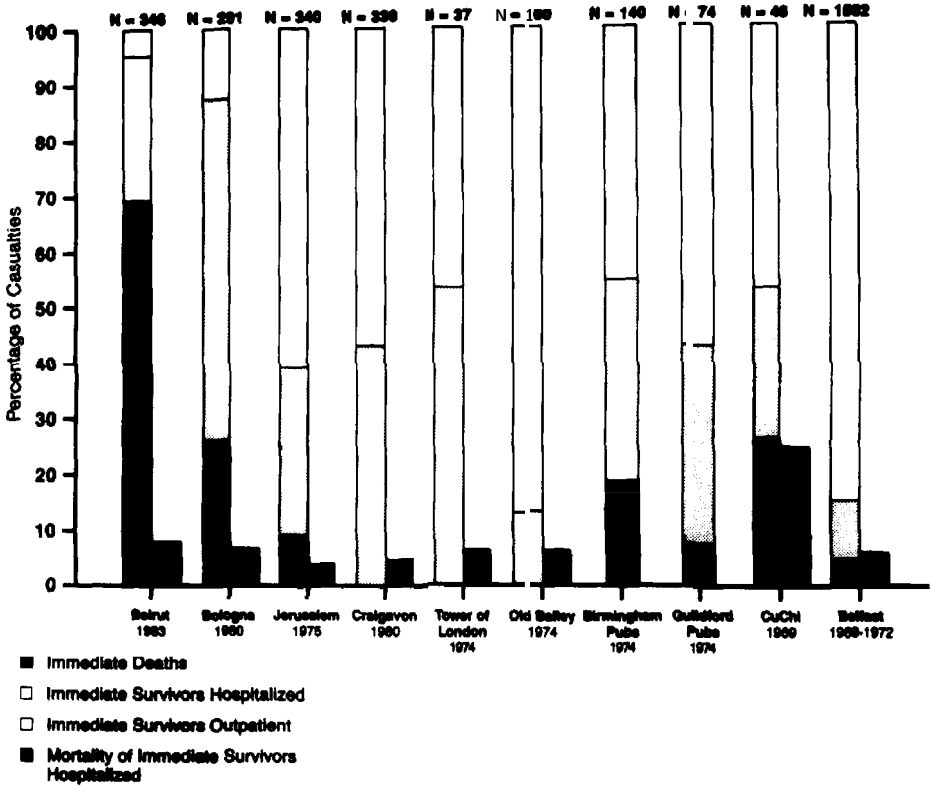

Fig. **6-1.** Morbidity and mortality compiled from reports of terrorist bombings
Source: Reference 1

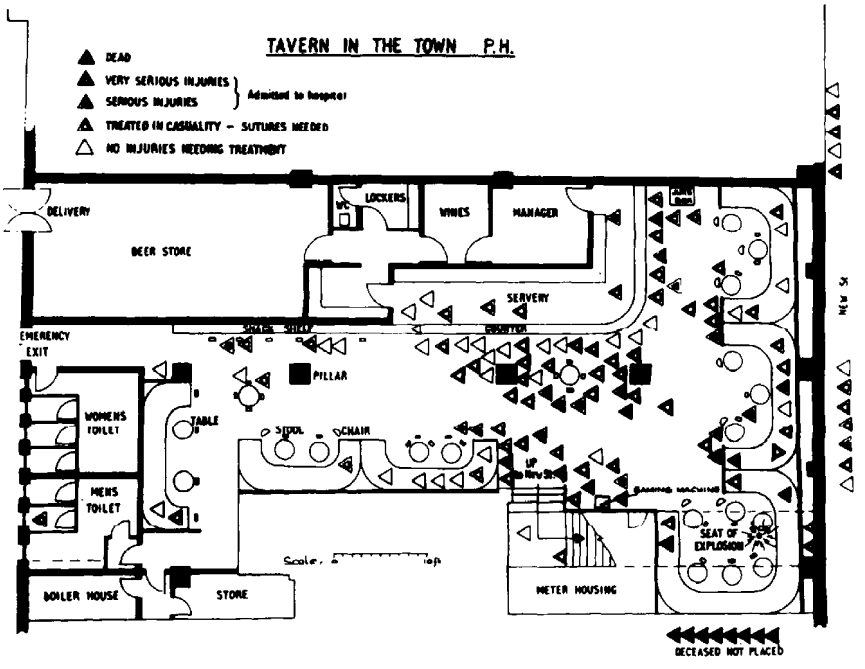

Fig. 6-2. The floor plan of the tavern in the Town Public House in Birmingham, England, depicts the locations of patrons who were killed, injured, or uninjured after a terrorist bombing.
Source: Reference *9*

ing places where detonation can generate great publicity as well as many casualties. Restaurants and bars are especially dangerous because they are likely to be crowded at predictable times of the day. They also contain glassware, other light materials, and furniture that can become mutilating secondary missiles in an explosion. Figure 6-2 illustrates the asymmetrical pattern of casualty generation in the bombing of the crowded Town Public House in Birmingham, England. Although the majority of the severely injured casualties were near the explosion, some victims who were more than 35 feet away were seriously wounded or killed, and people between them and the explosion were relatively unscathed.[9]

In all fourteen terrorism reports reviewed, only one patient died after being evaluated and released from an emergency room, but the cause of death was a heart attack that was not necessarily related to the bombing.[1] Thus, there appeared to have been no overtriage of bombing victims, in which casualties who should have been admitted to the hospital would have been discharged. However, many patients with relatively minor injuries were placed in the hospital for observation (undertriage). This level of caution might have a relatively small effect on a large civilian medical facility, but in a military mass-casualty action it would severely strain the available medical resources and could result in a significant loss of fighting strength.

Mortality Associated with Specific Organ-System Injuries

The mortality and morbidity of blast injuries depend largely on which organs have been affected. Figure 6-3 illustrates *(a)* the incidence of involvement of different anatomical regions in both survivors and on-scene fatalities, *(b)* the contribution of specific injuries to mortality, and *(c)* the mortality associated with organ-system involvement in severely injured survivors who were admitted to the hospital (called specific *mortality).'*

The specific mortality data show that casualties who had truncal injuries were few in number but were more likely to die after being hospitalized. Blast survi-

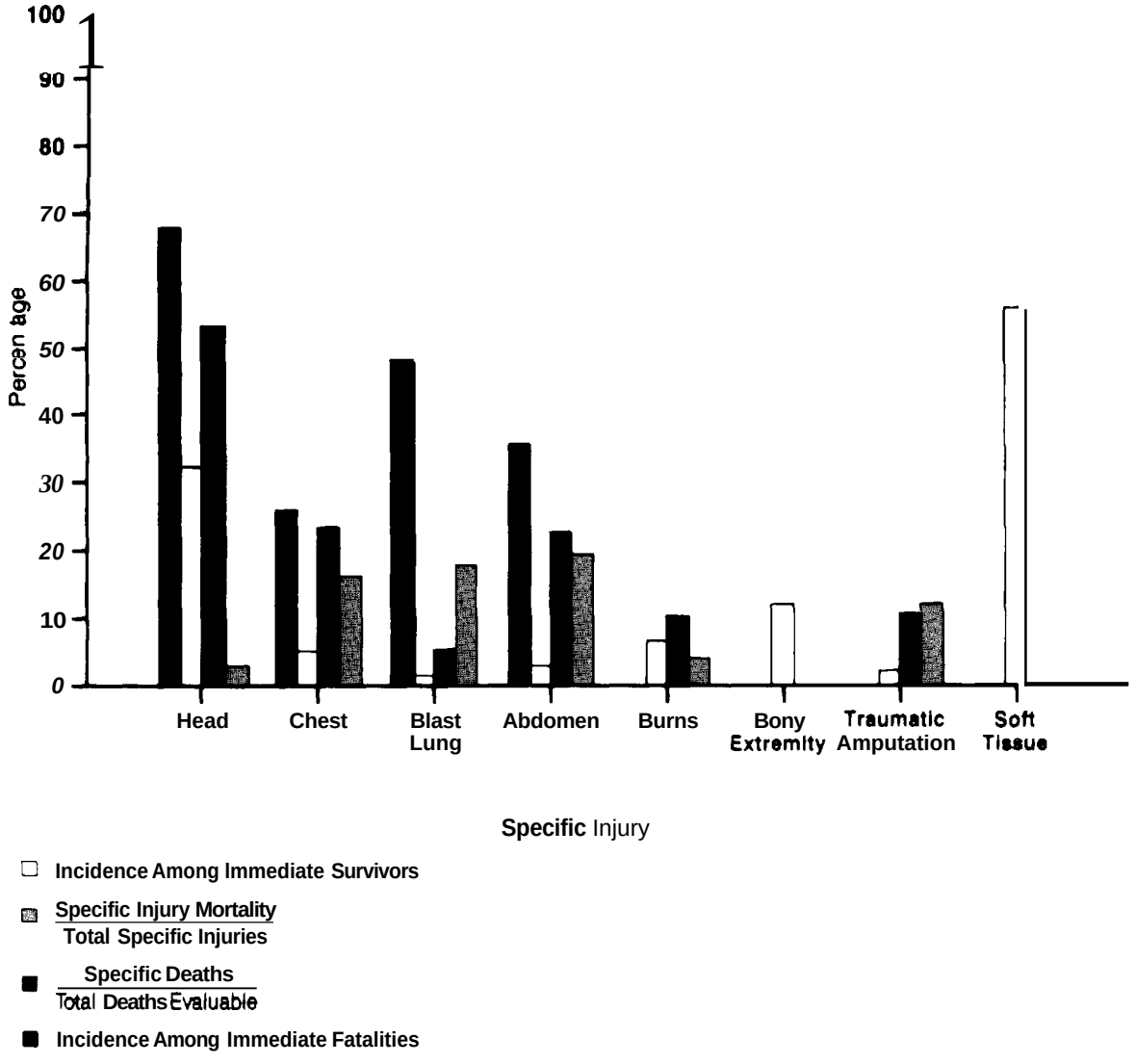

Fig. 6-3. The incidence of involvement of specific body areas in bombing victims is shown for both immediate fatalities and those surviving the attack. Unfortunately, because of incomplete reporting of autopsies, data from only a few sources are included and the figure represents less than a universal survey. The incidence of specific organ damage in immediate fatalities comes entirely from a single review of terrorist bombings in the British Isles.
Source: Reference 1

vors who were admitted to the hospital with chest injuries (7.4% of admissions) had a 15% death rate, and survivors who were admitted with abdominal injuries (3.5%) had a 19% death rate, together accounting for most of the in-hospital mortality from these incidents. Thus, the few casualties who can be expected to present to the triage officer with thoracoabdominal injuries tend to take up a disproportionate amount of medical-care resources but nevertheless represent an important focus for intervention and salvage.

Because pulmonary PRI may be an important component of a blast casualty's trauma, the use of positive-pressure ventilatory support may influence mortality and morbidity rates. Following the Bologna bombing, for example, five out of twenty victims with chest-wall or lung injuries required mechanical ventilation, and three died.6 In three reports from Israel, a respirator was required for seventeen out of forty

victims with lung injury, and four of those patients died.[10–12] Overall in these four reports, twenty-two out of sixty (37%) victims with pulmonary PBI required ventilatory support, and seven died. The specific mortality associated with blast lung was 12% overall, and 22% of those with respiratory failure died. The high mortality may have been partially due to other major injuries that were sustained, but late air embolism and barotrauma caused by mechanical ventilation were likely important (if unrecognized) factors.

The data on head and brain injuries in civilian blast incidents resemble those collected on the various anatomical sites of wounding in military conflicts. Trauma to the central nervous system is highly lethal; injuries to the head and brain were the most significant cause of death at the scene, and most fatalities from head injury occurred within a few hours of the incident.[3,13]

Wounds of the extremities were relatively common in the bombings but had low admission-mortality rates. Traumatic amputations occurred in about 1.3% of victims but carried a relatively high specific-mortality rate of 11%, probably because they occurred in victims who were very close to the detonation site and were thus vulnerable to serious PBI and other extensive tissue disruption.[4,13]

Primary Blast Injury in Terrorist Bombings

PBI is probably quite common in terrorist bombings. However, its clinical significance tends not to be emphasized because individuals so affected were usually mortally wounded by air embolism or other blast effects, particularly head trauma.[3,13] For example, according to a study of casualties who died in 5,600 separate explosions over a 12-year period, 78% of the 495 fatalities died at the scene and another 13% died within 24 hours.[13] Autopsies showed that 66% of all fatalities had brain injuries and 51% had skull fractures. PBI was found in a significant percentage of fatalities as well: 47% had the classic autopsy findings of blast lung and 45% had tympanic-membrane rupture. As a testament to the tremendous force of the explosion and the casualties' proximity to the blast, 34% had liver laceration, an injury that is more characteristic of blunt trauma.

Based upon their experience with civilian casualties, British observers have felt that survivors of a bomb attack have a low incidence of PBI.[9,13,14] For example, only 8 of the 653 IRA-bombing victims who were admitted to a hospital were thought to have had significant PBI to the lungs.[13]

However, survivors of the bombings in Paris during the early 1980s had a higher reported incidence of PBI.[15] Nine of 205 victims admitted to the hospital were diagnosed with blast lung. Of those who had traumatic respiratory insufficiency, four (44%) died.

In the bombing of the Bologna train station, 73 of the 291 casualties were killed, a higher number than might have been expected because the building collapsed.[6] Withe 107 survivors who were admitted to the hospital, nine had pulmonary contusion, pneumothorax, or pneumomediastinum. Eleven others had pulmonary injurics that with rib fractures or other chest-wall trauma. Five patients developed respiratory failure and three ultimately died. Little (if any) PBI of the abdomen was observed.

Explosions within an enclosed space result in more severe PBI (Table 6-1).[11,12] In two bus bombings in Israel, sixteen patients were diagnosed with PBI to the lung, although a few also had chest-wall damage (which is not a characteristic of pure PBI). Direct blast caused some lung damage in 31% of those patients who were admitted to thc hospital. Scvcn victims complained of significant abdominal pain; subsequent laparotomies revealed intestinal perforations in four victims, which is a relatively high rate of PBI of the abdomen for air blast. Not unexpectedly, all who were severely injured also had perforated tympanic membranes,

TABLE 6-1

MORBIDITY AND MORTALITY FROM BOMB ATTACKS ON CIVILIAN BUSES

Circumstance	(N)
Total bus passengers	104
Admitted to hospital	51
Evaluated, not admitted	46
Pulmonary blast injury	16
Killed immediately	7
Abdominal pain	7
Bowel perforations	4

Source: References 11 and 12

MILITARY EXPERIENCE WITH PRIMARY BLAST INJURY

PBI is not commonly reported in casualties of modern military operations, perhaps because the low-intensity conflicts that have occupied Western nations during the second half of this century have employed relatively light ordnance. However, the wars in the Middle East during the same period have involved massed armor and heavy conventional weapons. Israeli reports have detailed some blast injuries, although the reports are limited by a lack of definitions and autopsy data. No compilations of meaningful data from Arab nations or from the Iran-Iraq War are available, perhaps because of the limitations of their medical-care systems and the political costs of detailed casualty reporting.

The principal wounding mechanism of most military ordnance is fragmentation, a form of secondary blast injury. Perhaps because they are easier to recognize and diagnose, or perhaps because they have *so* many characteristics in common with ballistic wounds, secondary blast injuries have received far more attention in military circles than primary blast effects have. Nevertheless, the occurrence of PBI in certain military environments is now recognized as a factor in combat-casualty care.

PBI may be an important injury mechanism for crews of modern armored vehicles in which the threat of fragments and fire has been reduced. Blast may also have serious effects on soldiers who wear body armor, who are exposed to enhanced-blast munitions, or who are caught in underwater detonations. Hearing loss from the effects of blast or repeated loud noises also has potentially damaging effects on a combatant's ability to carry out military operations.

Data Collection

The military medical literature contains remarkably little careful documentation of PBI. Either there have been few such injuries, or whatever blast injuries occurred in a particular incident were unrecognized or unreported. American blast casualties in Vietnam, for example, were probably relatively few in number because the opposing forces there used mostly light, hand-held infantry weapons. They had few large, special-purpose explosive munitions that might be expected to cause blast injury, and little armored warfare was conducted. On the other hand, the opposing forces would have been more likely to suffer blast injuries from American weapons, but they have not reported — and probably were unable to gather — such information.

American Data Collection. The American forces went to great lengths to compile accurate casualty data in Vietnam. The most ambitious data-collection effort was conducted by the Wound Data and Munitions Effectiveness Team (WDMET), which accumulated detailed tactical and medical information on about 8,000 wounded or

In all of the WDMET data, there were only two incidents in which PBI was recognized. In one case, an American patrol was inadvertently hit by a 500-pound conventional bomb dropped by an American aircraft. While fragments from the bomb's detonation caused no wounds, two soldiers presented with hemoptysis and pulmonary contusion without external evidence of injury (Figure **6-4).** Both clearly had blast lung; neither required mechanical ventilation and both survived.

The second incident involved a rocket attack on an armored vehicle, in which two soldiers were killed outright. One was mutilated as he was blown through an open hatch. The other had only a scratch on his chin but was dead at the scene. Autopsy revealed that he had extensive blast injuries to his internal organs and a fatal pulmonary hemorrhage (Figure 6-5). Air embolism was the probable cause of death but was not noted, and evidence of it may not have been sought at autopsy.

Both of these wounding scenarios would have been much more common in a conflict against an enemy who used sophisticated conventional weapons.

Foreign Data Collection. Data from other countries have not always been easy to classify or interpret. According to Chinese experts, blast injuries accounted for only 0.3% of Chinese casualties during the Korean conflict;[16] however, under the prevailing hardships of that time, data collection must have been very difficult. In China's more recent border conflict with Vietnam, 20% of the injuries that were caused by artillery or mines were said to include PBI.[16] Unfortunately, other details of including their morbidity and treatment, are not available.

The Israeli experience in the 1980s is also unclear. Israeli Defense Force physicians reported that blast was responsible for 2.3% of the casualties in Lebanon in 1982.[17] Whether this percentage refers only to PBI or also to the mutilating effects of mines and other explosives is unknown, although artillery explosions (which

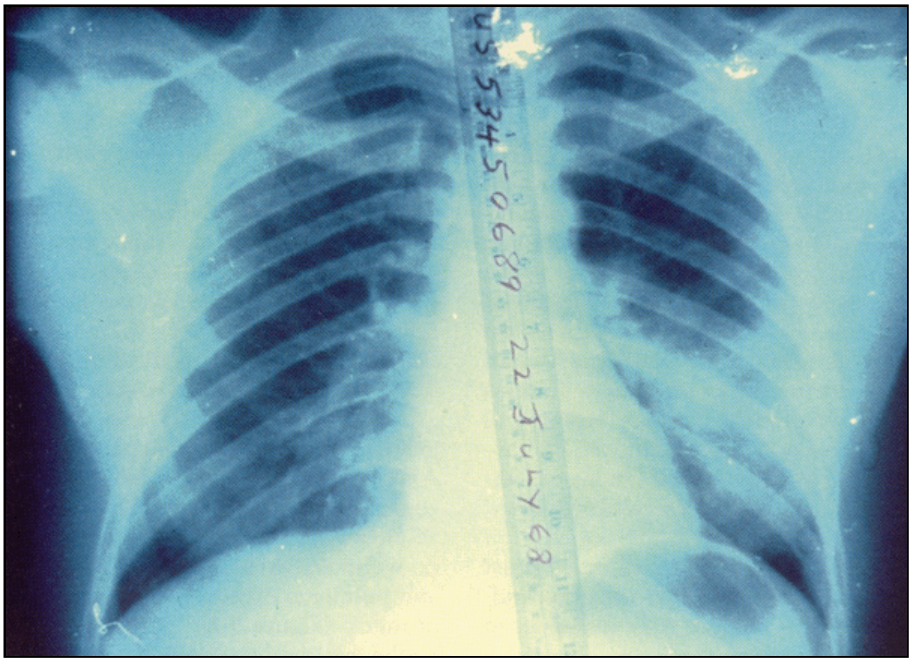

Fig. 6-4. This chest roentgenogram of a soldier who was injured by blast shows bilateral infiltrates from pulmonary contusion. The patient survived without sequelae.
Source: Wound Data and Munitions Effectiveness Team

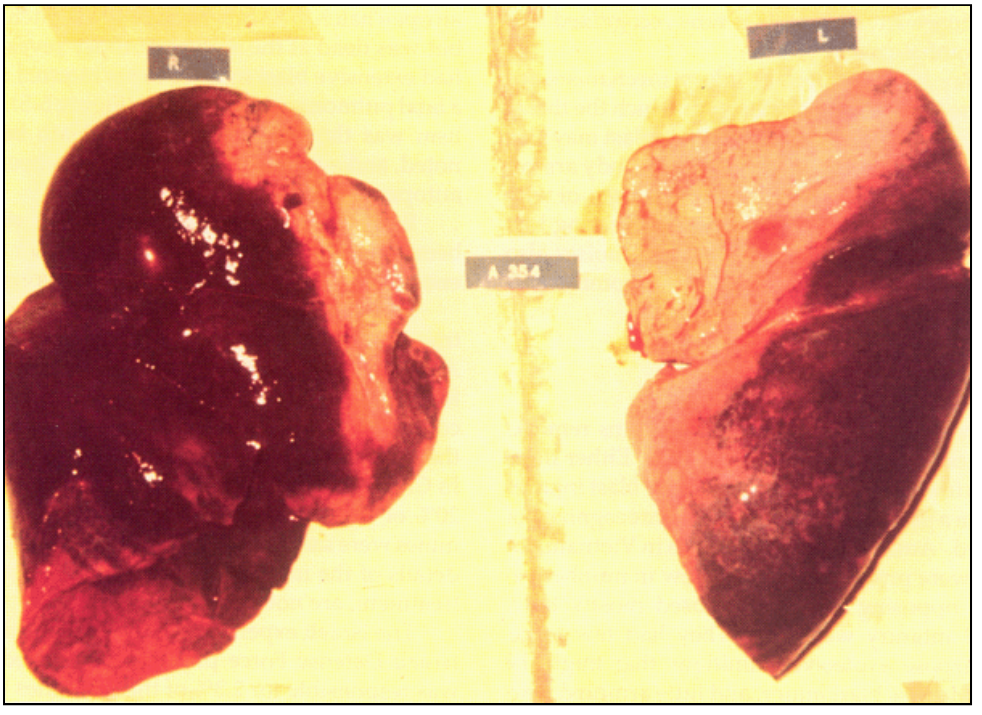

Fig. 6-5. This autopsy specimen from a soldier who was killed inside an armored vehicle by a penetrating antitank munition shows extensive pulmonary hemorrhage. The soldier exhibited no significant external injury.
Source: Wound Data and Munitions Effectiveness Team

caused 53% of battle injuries) were separated out as sources of blast injury, suggesting that the 2.3% blast casualty rate may refer to PBI only. Armored-vehicle personnel accounted for 14% of all casualties, but the data do not indicate whether PBI was a significant cause of injuries in tank crews. The collection of Israeli mortality data is complicated by religious objections to autopsy, and a definitive cause of death cannot always be determined.

A long-term British project to collect military-casualty data in Northern Ireland—called the *Hostile Action* Casualty System (HACS)—includes data from terrorist bombings as well as data from wounding by modern small arms.[18] The project reported on 828 armed-forces personnel who were injured or killed from explosions, and excluded those who were struck by bullets. Not surprisingly, the fatality statistics are similar to the civilian bombing data. There were 216 fatalities, 174 (80%) of whom were dead on the scene. Autopsies revealed a high incidence of both head injuries (33%) and pulmonary PBI *(32%).* Twenty-four soldiers, most of whom were wearing body armor, were killed by pulmonary PBI alone and had few external injuries. The ballistic vests they were wearing may have protected them from truncal fragment injuries but did not ameliorate the blast effects. Despite the high incidence of lethal pulmonary blast injury, only two soldiers who were admitted to the hospital required mechanical ventilatory support for blast lung as their only significant injury. Nine other soldiers died of respiratory failure in the hospital (two during surgery); blast lung was thought to play a role in most of these deaths, although sepsis and massive trauma certainly contributed to them. Abdominal blast injury was rarely noted. Tympanic-membrane rupture was seen in 86% of fatalities and in 47% of survivors. Traumatic amputations were associated with a very high lethality; only nine of fifty-two soldiers who were so afflicted survived.

Armored Vehicles

Historically, fragments and fire have caused the most injuries in armored fighting vehicles that were penetrated by antitank munitions. However, design improvements in these vehicles have significantly reduced the vulnerability of the crew to these injuries.[19] Such improvements include *(a)* spall-suppression linings, *(b)* the compartmentalization of fuel and munitions, *(c)* the extensive use of low-flammability materials, and *(d)* rapid automatic-fire suppression systems. Paradoxically, as the threat of fragment and burn injuries lessens, of blast and other ancillary effects of armor penetration becomes more significant.

Many Israeli armor casualties in the 1967 War were inside armored vehicles that were penetrated by antitank guided missiles (ATGMs) equipped with shaped-charge warheads (see Chapter One). These soldiers suffered from respiratory failure and extensive (but superficial) burns, a combination of symptoms that became known as the *ATGM Syndrome.*[20] The pulmonary component was attributed to a combination of PBI and toxic-fume inhalation.

Neither British nor American casualty data from armored conflicts during World War II are particularly useful in determining the role, if any, of blast injury in that era.

In the mid-1980s, public and congressional concern focused on the supposed vulnerability of the U.S. Army's Bradley Fighting Vehicle. Some critics feared that if the vehicle's aluminum armor were penetrated, the blast inside the crew area would be tremendously increased because of the *vaporific* effect. The vaporific effect is the result of a process in which aluminum (or any material) in the armor is vaporized and fragmented, and then undergoes an explosive exothermic reaction with atmospheric oxygen in the crew area.

The U.S. Army Medical Research and Development Command (USAMRDC) conducted experiments using anesthetized sheep and pigs inside armored vehicles. These experiments demonstrated that—outside of the fragment-spall cone (see Chapter One)—significant injury to the lungs and intestine did not occur when either the Bradley or the M113 armored personnel carriers were penetrated by either small antitank munitions (such as the American-made LAW, the Soviet-made RPG6, or the Swedish-made Karl Gustav) or larger-caliber warheads (such as the American-made TOW1, the Soviet-made Sagger, or the European-made Milan).[21]

Under congressional mandate, all American military weapons systems must be tested against threat munitions under realistic operating conditions. The U.S. Army has conducted an extensive series of tests with the Bradley Fighting Vehicle, the M60A3 tank, and the M1A1 Abrams tank.[22] Casualty evaluations were made for *(a)* fragments, *(b)* fire, *(c)* acceleration/displacement, *(d)* blast, and *(e)* inhalation of toxic fumes. Although the results of these tests are classified, PBI can be expected in a few cases. The number of casualties in a modern armor conflict who will have PBI is unknowable, but it is nevertheless likely to be much lower than the number of casualties who will suffer fragment wounds. In an armored vehicle that has been defeated by a large warhead, 1%–20% of the survivors would have some degree of PBI in addition to their other wounds. Whether the blast effects would pre-

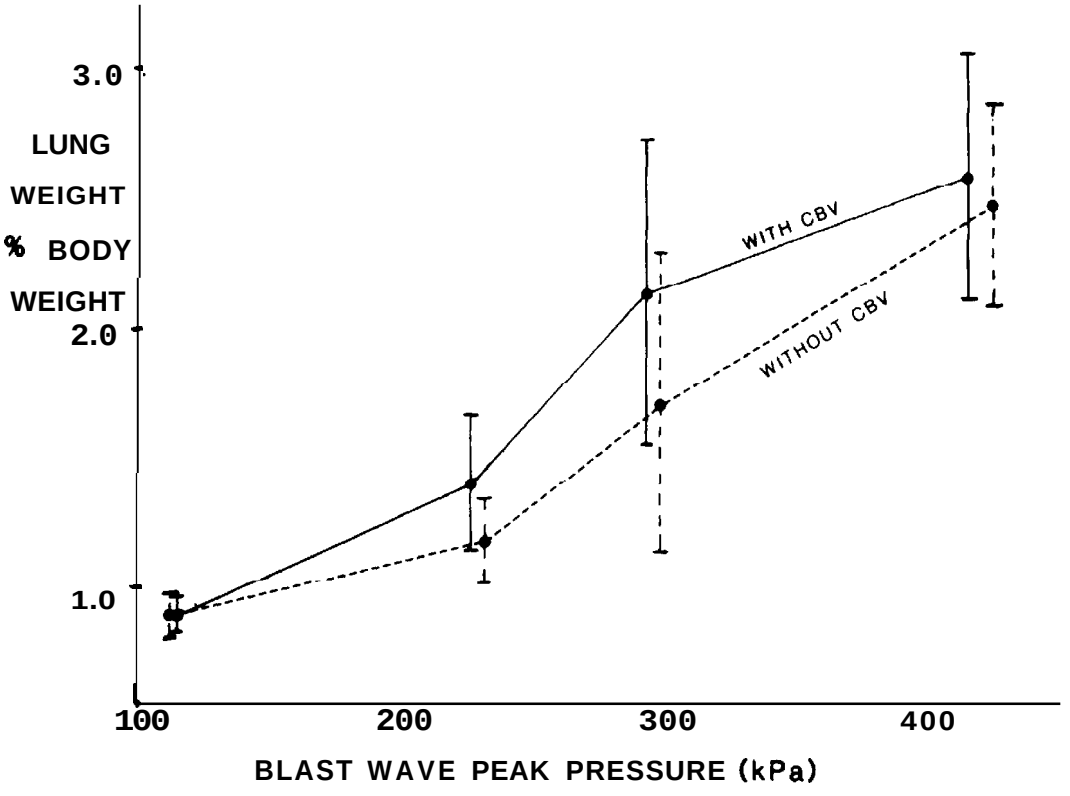

Fig. 6-6. The effect of wearing a cloth ballistic vest (CBV) on lung injury as measured by increased lung weight is shown for sheep over a range of blast-injury severity. At the highest level of blast, five out of **six** animals that were wearing a CBV were killed, whereas only two out of the six animals that were not wearing a CBV died of PBI.
Source: Reference 25

dominate or complicate the clinical course of these casualties is not known.

Body Armor

Just as ballistics researchers' concerns for protecting personnel from fragments led to the development of light, effective body armor, blast researchers have also sought to find a practical way to protect the individual soldier from PBI. A Swedish study demonstrated that covering a rabbit with foam rubber markedly increased a blast's lethality, whereas shielding the animal's chest with steel pipe was effective in reducing injury.[23] **A** study with demonstrated that, even at the low overpressure levels that would be found in the crew areas of heavy weapons, the U.S. Army standard-issue cloth ballistic vest caused an increase in intrathoracic pressure over the intrathoracic pressure that was observed when the vest was not worn.[24] Although the minimal effects seen at **such** low overpressure levels were not **injurious,** a study using large animals and much higher levels of blast showed that the ballistic vest caused a significant increase in both lung hemorrhage and mortality.[25] Over a range of blast exposures, animals covered with the vest suffered significantly more injury than those that were uncovered (Figure 6-6). At the highest overpressure level, five of the six animals wearing the vest were killed by the blast, compared with only two of the six uncovered animals.

Although combat records do not confirm this effect, the British HACS study of IRA bombings did find lethal pulmonary blast injuries in soldiers who wore vests.[18] These soldiers had no external injuries. The vests may have saved them from potentially lethal fragment injuries, but may have done so at the cost of compounding the PBI.

Israeli soldiers commonly wear body armor, but there are no data on the incidence of PBI in soldiers who were wounded in recent Middle-East conflicts.

The mechanism for this enhancement of the primary blast effect is unknown. The vest may serve to

increase the surface area that is exposed to the blast, thereby increasing the total energy delivered to the chest.[25] Theoretically, the vest may also improve the efficiency of energy transmission from the blast wave to the thorax.[25,26] A study that used small animals has suggested that effective blast protection may be afforded by putting a rigid covering over a foam material.[26] Such a protective garment might not be practical for all troops, but could be important for special clothing used by ordnance-disposal personnel.

This review of the evidence that personal body armor may enhance primary blast effects must not be construed as a recommendation for abandoning its use. Soldiers already know that body armor is hot and heavy; they do not need another excuse to shed it. The ballistic vest is a lifesaver, and the threat of injury by bullets or fragments is far greater than the threat of pure PBI on any imaginable battlefield.

Instead, these data should be used by physicians when they make triage and treatment decisions. Soldiers who were wearing vests when they were exposed to an explosion may owe their lives to the vests, but physicians should be aware that these troops may have sustained PBI to the lungs and abdomen even in the absence of significant truncal ballistic wounds.

Enhanced-Rlast Munitions

Enhanced-blast munitions are designed to injure by means of their blast effects rather than by fragmentation. Most modern military powers have either fielded or evaluated enhanced-blastmunitions, usually in the form of some type of fuel-air explosive (FAE). The FAE concept is described in some detail in Chapter One of this textbook.

The role of enhanced-blast munitions as antipersonnel weapons is not established. U.S. Army doctrine calls for the use of FAE weapons to clear minefields or possibly to be used against "soft" military materiel, such as vans and light structures.[27] In an example of the effectiveness of such a tactic, the Egyptians reportedly used FAE munitions to flatten and thereby disable Israeli radars during the Yom Kippur War.[28]

Although Soviet military doctrine for enhanced-blast weapons is unknown, they have been used in armored-assault training maneuvers against fortifications and dug-in troops.[29] Whether they were used for their direct casualty effects or as psychological weapons is unclear.

Large numbers of air-delivered weapons—the names of which were literally translated as "volume bombs"—were reportedly used by the Soviet forces in Afghanistan.[29] The detonation of these weapons allegedly resulted in extensive fires. Whether the munitions were primarily blast or incendiary weapons is unknown.

Unlike a fragment or a projectile, the blast effect is not limited to a linear trajectory. Not only can the blast wave go around an object, but its effect will be magnified in an enclosed area. Thus, although its potential role as an antipersonnel weapon has not been established, an enhanced-blast munition might be particularly effective against personnel in caves, rocky terrain, open foxholes, or trenches.

Underwater Blast Injury

Blast injuries were common in those World War II combatants who had been forced to abandon their ships and were in the water near a subsurface detonation of a torpedo, a depth charge, or an aerial bomb. Both American and British authors wrote of thousands of such casualties and noted that the number of dead from blast, although unknowable, was probably quite large.[30–32] Fifty casualties with abdominal blast injuries were evacuated to Pearl Harbor following the Battle of Midway in 1942.[30] One British ship's physician noted that only 90 of 125 sailors who had safely abandoned ship wearing life preservers survived a nearby depth-charge explosion.[31] Twenty-four sailors in a North Sea action were rescued after surviving an underwater explosion, but within a few days eleven of them died, seven of whom had intestinal perforations.[32]

During the 1967 War, an Egyptian missile struck the Israeli destroyer *Eilat*.[10] The crew abandoned ship and, while they were in the water, another missile detonated nearby. The number of deaths was unreported, but thirty-two survivors were rescued within a few hours. All casualties had PBI except one, who fractured his tibia while on board ship. Twenty-seven survivors had blast lung; five of them required ventilatory support. Twenty-four had abdominal signs and symptoms and underwent laparotomy; of these, twenty-two had bowel perforations. Nineteen sailors had both thoracic and gastrointestinal injuries. Four victims died, three of whom expired during or shortly after general anesthesia.

In another incident, an underwater charge was detonated near thirteen soldiers who were swimming for recreation.[33] All quickly got out of the water. However, within a minute, two sailors suffered cardiac arrest; within 10 minutes, two more died, and by 30 minutes after the blast, two more had succumbed. The remaining seven soldiers were evacuated by helicopter and, despite heroic efforts, only three ultimately survived. These casualties were almost certainly killed by air embolism to the heart and brain, which causes most of that occur immediatcly

TABLE 6-2

HEARING LOSS IN BRITISH COMBATANTS
DURING THE FALKLAND ISLANDS CONFLICT

Test Group	Hearing Category	Before Blast Exposure (N)	After Blast Exposure (N)	Percentage of Combatants Whose Hearing Deteriorated
All Infantry	1+2	316	271	13.3
	3	21	52	
	4	1	15	
Infantry using small arms	1+2	105	97	7.3
	3	4	9	
	4	0	3	
Tnfantry using 81-mm mortars	1+2	73	55	23 7
	3	2	17	
	4	1	4	
105-mm light gun crew	1+2	78	68	11.9
	3	6	14	
	4	0	2	

*Hearing categories are by United Kingdom definitions, with 4 being the most severe.

after blast exposure.

The position of the swimmer in the water is an important factor in the severity of underwater blast injuries.[34] Sir Zachary Cope, a prominent British surgeon during World War II, observed that

> if the person were **floating on the bock** so **that** neither the abdomen nor the chest were directly opposed to the blast wave, no serious injury was sustained.[31]

Because the shock wave reflects as a tension (negative) wave from the water-air interface, the effective force loading of the blast will be greater the deeper one is submerged. Thus, the abdomen will be injured out of proportion to the chest in most underwater exposures Floating on the water's surface is the safest possible body position when exposure to underwater blast is a possibility.

Hearing Loss Data

In general, the most sensitive organ to the primary effects of a blast wave is the ear. Military personnel are at particular risk for hearing loss, not only from blast exposure but also from exposure to the loud noises that are inherent in the firing of many weapons and the operation of aircraft and tracked vehicles. Not surprisingly, hearing loss is the single largest category of medical disability for the U.S. military, resulting in the payment of more than $165 million in benefits in 1985.

Documentation of combat-related hearing loss is scanty. In a study of 338 soldiers with hearing loss sustained during the Falkland Islands conflict, patients were divided into three groups based on their type of exposure: *(a)* operators of support weapons, such as mortars, *(b)* infantrymen who used small arms, and *(c)* Welsh guardsmen who were survivors of blast injuries sustained in an attack on a transport ship.[35] Those using heavier weapons were at greater risk; **the** mean hearing loss from preconflict hearing levels was 5.1 dB in the right ear and 5.5 dB in the left ear. Compliance with hearing protection was so poor that no conclusions could be reached regarding its effectiveness. In a second study, 114 soldiers in the Royal Artillery were exposed to very high levels of impulse noise from the sustained firing of 105-mm artillery.[36]

Table 6-2 summarizes the British data on hearing loss during the Falkland Islands conflict.

The high incidence of tympanic-membrane rupture following blast exposure in civilian bombings and recent military conflicts attests to the frequency of aural injury to be expected in combat.[37] The complex reverberant blast environment inside a penetrated armored vehicle is likely to be very injurious to ears, should personnel escape other casualty-generating effects. In one study, large animals were placed inside armored vehicles that were penetrated by shaped-charge warheads. When a 5-inch warhead was used, tympanic-membrane rupture occurred in 71% of ears, compared to 36% when a 3-inch round was used.[21] Based on experiments with anthropomorphic mannequins, the use of conventional hearing protection (either insertional or circumaural) should greatly reduce or eliminate the risk of tympanic-membrane rupture.[38]

EXPERIMENTAL BLAST RESEARCH

Like wound ballistics, blast-injury research was spawned on the battlefield. Controlled experiments have been intertwined with military developments, not only preceding the introduction of new blast weapons or protective equipment, but also resulting from the use of innovative blast munitions by opposing combat forces or unusual blast environments that combatants may have faced in battle.

Blast Research Before World War II

Anecdotes of what can now be recognized as death from PBI were reported earlier than World War I, but this war's heavy use of high explosives exposed the potentially lethal nature of the blast wave itself.

Blast injuries to experimental animals were first studied systematically in 1914, after the Swiss researcher Franchino Rusca observed that three soldiers who had been killed by a bursting grenade during the Balkan Wars had no external injuries. To study this phenomenon, he placed rabbits inside a sand pit that had a and exposed them to blasts from 100 g of dynamite detonated within the pit. Rusca could not account for the animals' sudden deaths, which later research has proven to be the result of air embolism. However, the gross blast injuries that he described (pulmonary parenchymal hemorrhage, gastrointestinal contusions, and gastrointestinal ruptures) are now recognized as PBI.[39]

Medical personnel in World War I knew that a blast could blow a casualty to pieces, cause bodily displacement, and result in ear injury, but no systematic accounts of internal injuries or deaths from PBI were reported in the military medical literature. At that time, the blast wave was believed to affect primarily the nervous system, possibly because the many "shell-shock" casualties of World War I exhibited a variety of psychophysiological symptoms after prolonged exposure to heavy artillery barrages.

In 1918–1919, the American scientist David Hooker conducted blast-injury studies at Sandy Hook Proving Grounds in New Jersey.[40] He exposed dogs, cats, rabbits, and frogs to muzzle blasts from 10-inch naval cannons and 12-inch mortars. Hooker measured the very sudden increase in air pressure (the overpressure) that the blast produced. The blasts from the 10-inch cannons repeatedly produced shock in dogs (that is, they caused the animals' blood pressure to drop to one-half the normal level), whereas the blasts from the 12-inch mortars did not, even though they had a higher peak pressure. Hooker correctly attributed the pathophysiological abnormalities produced by the cannon's blast to the longer duration of its positive pressure phase. He took comprehensive pathological and physiological measurements, but did not find any gross or microscopic evidence of lesions in the brain or nerve tissues.

Blast Research During World War II

Some soldiers who fought during the Spanish Civil War (1936–1939) were found dead with few, if any, external injuries. However, such detonation deaths did not attract much attention until World War II, when the number of blast casualties increased considerably because of aerial bombing attacks on German and British cities.

British Studies. In Great Britain, researchers who conducted interspecies studies related the blast overpressure levels required to kill 50% of exposed animals (P_{50}) with the animals' body weight.[41] Researchers exposed mice, rabbits, guinea pigs, goats, and monkeys to blasts from 1-, 8-, and 66-pound charges, and observed a striking rise in P_{50} for increasing body size. They extrapolated these data to predict a P_{50} of 370 psi for a 60-kg human and a P_{50} of 470 psi for an 80-kg human. Based on this work with small rodents, the researchers thought that the P_{50}

would be constant for any amount of explosive. Although the explosive charges in the experiments were relatively small by human body-weight standards, they were huge by murine standards, and produced blasts that had relatively long overpressure durations for animals of such small size. Hooker's experiments over 20 years earlier had demonstrated the importance of the blast wave's duration in determining lethality.[40] It was not until after the war that British scientists acknowledged the interactive effect of duration and pressure level.

The pathological nature of the blast injuries, along with the physiological effects, were carefully described by the British also showed that the blast wave must hit the thorax directly to produce lung hemorrhage, and suggested that sponge rubber might shield the body from some direct blast effects.

German Studies. Military medical studies of blast waves were also underway in Germany throughout World War II, but the results were not made known until after the end of the war. German civil law prohibited autopsies on most bombing victims, which delayed pathological descriptions of PBI. In particular, four researchers (Hubert Schardin, Theodor Benzinger, Robert Rossle, and Hans Desaga) conducted extensive experiments on the effects of blast,[43–46] which Benzinger described as follows:

> The blast wave is a shot without a bullet, a slash without a sword. It is present everywhere within its range. Blast would be as dreaded... as chemical [weapons], if its range, when explosives are used, were not limited to small areas. However, it would be premature to believe that this situation will always remain the same."

The Germans were the first to discover that arterial air embolism is the cause nf immediate death frnm blast injury. They reasoned that air entered the pulmonary venous circulation from the disrupted alveoli and was then distributed to the coronary vessels, the brain, and the vascular beds in other organs of the body. By injecting very small volumes of air into the carotid artery, researchers could produce the central nervous system signs that had yreviuusly been observed in blast-injured dogs and humans. Electrocardiographic changes observed in blast-exposed animals were reproduced by 1cc nf air intn the pulmonary veins of dogs. These experimental results supported autopsy findings of internal air emboli as the most probable mechanism of early death from PBI.[45]

The *duration effect* was another significant German finding.[46] The researchers found that the P_{50} for dogs decreased by a factor of three when the duration of the blast wave's positive phase was increased from 1.8 to 12.0 msec. In order to disassociate pressure and duration, the investigators used a variety of different explosive weights and placed the experimental animals at various distances from the point of detonation.

The German investigators noted that the nature of the internal pathoanatomical and pathophysiological changes caused by air and underwater blast were the same.[44] One experiment demonstrated that *(a)* the animal's head was not vulnerable to underwater or air blast, and *(b)* the blast must strike the thorax to inflict lung injury and to cause air embolism (Figure 6-7).

In another experiment, the animals' tracheas were opened widely to the air blast so that any lung injury would bc intcnsificd if the blast wave indeed entered the body through the upper respiratory passages (Figure 6-7).[44] This had been suggested by some researchers who believed that the glottis ought to protect the lungs from blast effects, because they had seen injuries to the epiglottis and hemorrhages in the laryngeal mucous membrane in exposed animals whose lungs were not injured in the blast. Five tracheotomized dogs were placed at different distances within and beyond the lethal limit for untreated blast-exposed dogs. Not only did all of the tracheotomized dogs survive, but the tracheotomies actually seemed to have some protective effect. Lung injury did not occur when the upper airway alone was open to the blast and the trunk was protected.

Unlike the British scientists, German researchers found that placing foam rubber materials around the thorax not only provided no protection from air-blast injury, but in fact intensified lung hemorrhage.[46] The injury-enhancement characteristics of this type of material have been affirmed in more recent investigations.[23,25]

Schardin took an engineering approach to the and proposed three damage mechanisms for blast injury: *(a) spallation, (b) implosion,* and *(c) inertia.*[43] In the spallation effect, a blast wave that passes from a more dense medium into a less dense medium reflects into the dense material as a tension wave that throws off (or spalls) material at the interface. In an experiment, a small lead azide charge was detonated in the center of a glass disk. Before the breakage cracks reached the outer edges of the glass disk, the shock front had already reflected from the periphery and spalled material frnm it The same effect can also be seen when the water surface breaks up when the blast wave from an underwater explosion (for example, from a depth charge) reaches the water-air interface. Schardin speculated that a similar effect might happen at the air-tissue interface in organs that were exposed to an intense blast, but such an injuring mechanism has never been pioved to exist.

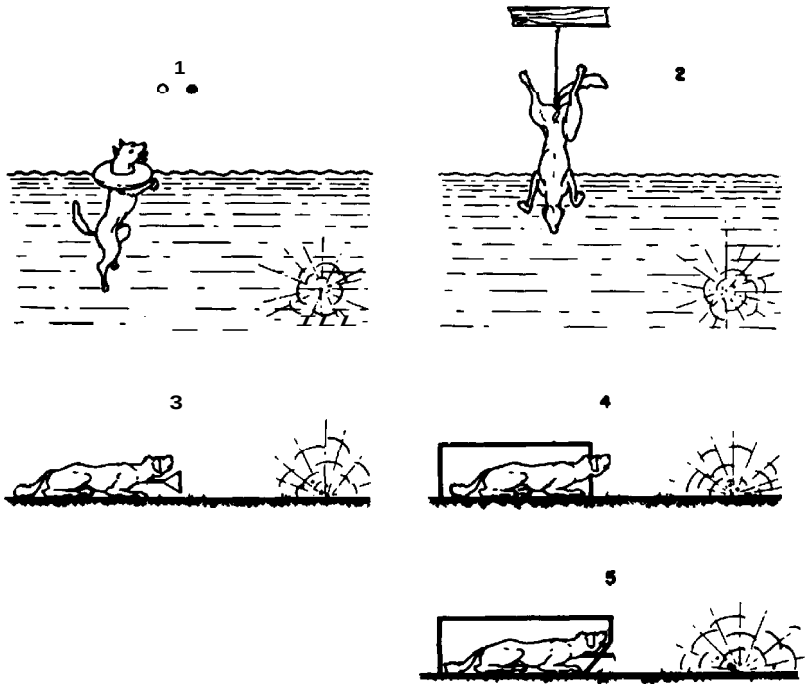

Fig. 6-7. The experiments in this pictoral summary were conducted by German scientists during World War II. The experiments resulted in several findings: The effects of immersion blast were found to be pathologically identical to those of air blast **(1).** The exposure of the dog's head to immersion blast or air blast resulted in no injury to internal organs (2 and 4). By using a funnel to channel air into the animal's lungs (3), researchers found that injury from air blast was not caused by air being forced down the trachea, and no injury was detected when an animal's tracheotomy was exposed to the blast while its body was protected (5).
Source: Reference 44.

In experiments designed to illustrate the implosion effect, Schardin passed a stream of air bubbles through water and sent a blast wave through them. The inwardly rushing water surrounding the bubbles accumulates considerable kinetic energy, and the bubble is compressed to a much smaller volume than would be expected from the pressure in the blast wave. **As** the strongly compressed bubbles expand, they become the centers of new pressure waves. Schardin speculated that a similar phenomenon might occur in the lung tissues or in the gas-containing portions of the gastrointestinal tracts of animals that are **struck** by a strong shock wave.[43] This hypothesis, however, has never been supported by experimental data.

In an **example** nf the **inertia effect,** an intense air blast that hits a structure like a leaf blows away the delicate portions between the sturdier veins but leaves the veins themselves. Because the veins have more inertia (density) than the light tissue between them, the two materials could be expected to accelerate at different rates when struck by the blast, thereby creating great stress at their boundaries.[43] Schardin speculated that the delicate alveolar tissue between bronchi and blood vessels might be vulnerable to similar stress. Pathological studies did indeed show hemorrhage around the bronchovascular structures, which are analogous to the veins in the leaf.[45]

In keeping with a mechanistic interpretation, Schardin also suggested that, if the duration of the blast wave is less than the natural *period* of the system (that is, the frequency at which it resonates), then the *impulse* (or pressure-time integral) becomes the determining damage component of the blast wave. Thus, consideration of both peak pressure and duration are important in determining injury. If, on the other hand, the duration of the blast wave is greater than the natural period, the peak pressure alone will account for the damage observed. Such long-duration blast waves may be seen with very large bombs or nuclear detonations. Schardin's view was consistent with the British observations that blast damage in mice (which have a small mass and therefore a short natural period, and for which almost all blast waves are effectively of long duration) **was** dependent on peak pressure alone

and independent of charge size, the main determinant of duration.

American Studies. During 1942–1945, American researchers studied the effects of air and underwater blasts using experimental animals. They concluded that PBI from a blast in air occurred only when the animals were within the radius of the blast's fireball and was not an important casualty generator.[30] These researchers studied only blasts that occurred in an open field, however, and the results may not be the same for humans who are within enclosures when they are exposed to air blasts.

During the World War II era, American researchers realized that underwater blast was an important casualty producer because of the many accounts of death and injury among sailors who had been in the water during explosions of depth charges and torpedoes.[30,47] Some experimental work was performed by the U.S. Navy on the pathology of what was then called immersion blast.

Modern **Blast Experimentation**

Since World War II, blast research has been driven by the increasing lethality of conventional munitions and the tremendous blast effects caused by nuclear detonations. The greatest volume of work has been done in Sweden and the United States, and much of the data is available in unclassified reports.

Swedish Studies. Research on blast injury in Sweden began about 1940 and has continued actively to the present time. Researchers have studied the relationships between the physical qualities of **blast** waves and changes in physiological and biochemical parameters, particularly as they apply to primary blast injury. The researchers have conducted investigations on the respiratory and circulatory changes following blast injury and on air embolism as the cause of death in blast-exposed animals.[48,49]

In studies of potential protective materials, they found that rigid materials afforded protection from blast waves, but that soft materials (such as foam plastic) did not.[23]

Swedish researchers also measured the deformation of the body wall and the overpressure at several locations inside the animal during blast exposure. They developed physical and mathematical models simulating their empirical findings.[50,51]

French-German Studies. Following World War II, German scientists continued their blast research at the Franco-German Research Institute in Saint-Louis, France. There, researchers studied the relationship between blast-wave components and mortality, and showed that animals can tolerate very high overpressures if there is no shock front—that is, if the peak pressure is reached relatively slowly. In the 1980s, they studied the effects of multiple-blast **exposure** in both rats and swine.[52,53]

British Studies. At the Chemical Defense Research Establishment in Porton Down, Salisbury, Wiltshire, blast research has focused on behind-armor effects, blunt body trauma, protective garments in a blast environment, and a variety of research concerns (many of which are classified) that are related to terrorist bombings and civil disturbances.[26,54]

Chinese Studies. Since the 1964 detonation of its first nuclear weapon, the People's Republic of China has conducted uninterrupted blast research.[16] Wang Zheng-Guo has led investigations to determine the blast levels required for threshold injuries, severe injuries, and mortality from nuclear and high-explosive blasts. Chinese researchers have assessed the injury potential of repeated lower-level blasts that approximate heavy-artillery muzzle blasts, and have evaluated the practicality of blast-protective garments.

Soviet Studies. Blast research has been conducted in Union continuously since World Wnr 11, but only a few reports have appeared in the open literature. Published accounts have accurately described the pathology of PBI and have discussed the interaction of blast waves with the body.[55,56] Soviet research has drawn heavily from American and Western European reports; their medical writings often intermingle PBI with other mechanisms of blast injury and seem to stress the neuropsychiatric response to explosions.[57]

American Studies. In 1953, the Atomic Energy Commission contracted with the Lovelace Foundation to study the biological effects of nuclear blast waves, and established a blast-research facility at Kirtland Air Force Base in Albuquerque. Under a series of umbrella organizations and sponsored by many government agencies, the Kirtland facility has been the source of the most comprehensive work on blast biology in the world.

Nuclear-weapons blast research 1958–1964 aimed to develop casualty and risk criteria for both PDI and blast displacement.[58,59] Initial tests by the Lovelace group involved exposing animals to nuclear blasts while they were inside open blast Nevada Test Site. Injury criteria were developed for personnel who were in the open field as well as inside fortifications.

For several years in the mid-1960s, researchers at Kirtland conducted tests to determine the effectiveness of FAE munitions against a variety of military targets. In 1968, an underwater test facility was con-

structed at Kirtland to study immersion-blast effects. In 1978, research was directed toward determining damage-risk criteria for personnel who were exposed to repeated muzzle blasts from **that** ranged from mortars to large field guns.[60] Since 1980, government researchers and contractors have been studying the physiological consequences and biomechanics of blast injury.[61,62]

SUMMARY

Although fragments cause most of the injuries that result from small terrorist bombings, PBI is often noted at autopsy and may be a contributor to early mortality in many cases. **A** small number of survivors will have pulmonary PBI that results in respiratory failure and high in-hospital mortality. Positive-pressure ventilation or general anesthesia will put such casualties at an even higher risk.

The determination of the incidence of PBI in military casualties may be clouded by the low-intensity nature of most American combat operations since the Vietnam War and a lack of blast-casualty data from Middle-East wars. Future military conflicts will result in casualties with PBI, usually as a component of combined injuries. PBI will continue to be an important factor in armored warfare, in underwater explosions that affect submerged or swimming soldiers, in explosions from enhanced-blast munitions, and in explosions that occur near soldiers who are wearing ballistic body armor.

Even in peacetime, hearing loss is a common injury in soldiers. The limited available data and common sense suggest that intense combat operations—even those that are as brief as the Falkland Islands conflict—will result in significant hearing loss for many combatants. Tympanic-membrane rupture and associated hearing loss will be common, especially for casualties in armored vehicles. Wearing standard hearing protection should ameliorate this form of acoustic injury.

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