THE CIRCULATORY SYSTEM

The circulatory system, which comprises the heart and blood vessels, may be directly injured by the blunt effects of the blast wave itself. However, the indirect effects of the blast on the circulatory system can be much more significant: Air emboli, which originate in the lung as a sequela of pulmonary PBI, can occlude coronary blood vessels and cause cardiac arrest.

Air Emboli

Since the early days of blast experimentation, investigators and pathologists have believed that the amount of hemorrhage seen in those animals that died after blast exposure was insufficient to explain their rapid deaths.[9] Neither the amount of lost blood nor the decreased pulmonary surface area that was available for gas exchange appeared to account for such high mortality.

In one key experiment, animals were exposed to underwater blast while their thoraces were submerged and their heads were above water. In spite of the fact that their heads were not subjected to the blast wave, they suffered deficits in neurological control as well as alterations in consciousness.[22] Previously, researchers had believed that these effects were caused by PBI to the brain, but careful pathological studies ultimately revealed the presence of air emboli in cerebral and coronary arteries following blast exposure.[9,10,77] Investigators then demonstrated that an injection of small quantities of air into the carotid artery could reproduce these abnormalities in the central nervous system.

Air emboli originate in the lungs, and are thought to enter the circulation through traumatic alveolovenous fistulae. Mortality is proportionate to the extent of both lung hemorrhage and air emboli at autopsy (Figure 8-15). Thus, the more severe the lung hemorrhage, the greater the likelihood of significant

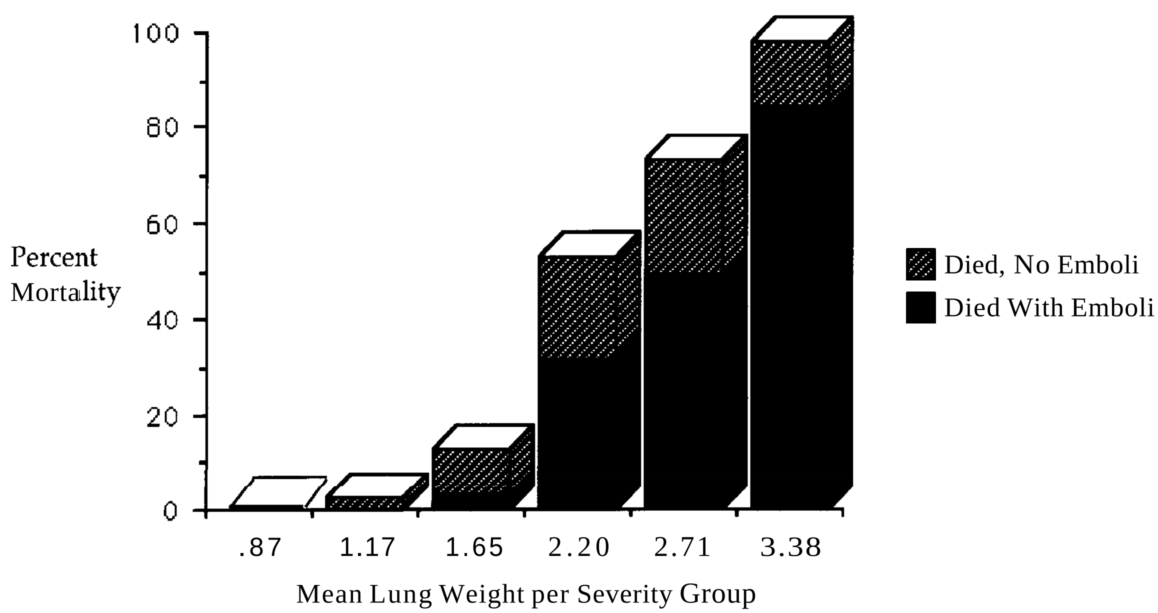

Fig. 8-15. In this graph, each of the six bars represents a group of animals categorized by severity of lung hemorrhage. The mean lung weight below each bar represents the mean lung weight for that particular severity group. Lung weight is proportionate to mortality. In addition, the proportion of dead animals that showed air emboli at autopsy increased in proportion to the amount of hemorrhage.
Source: Redrawn from reference 3

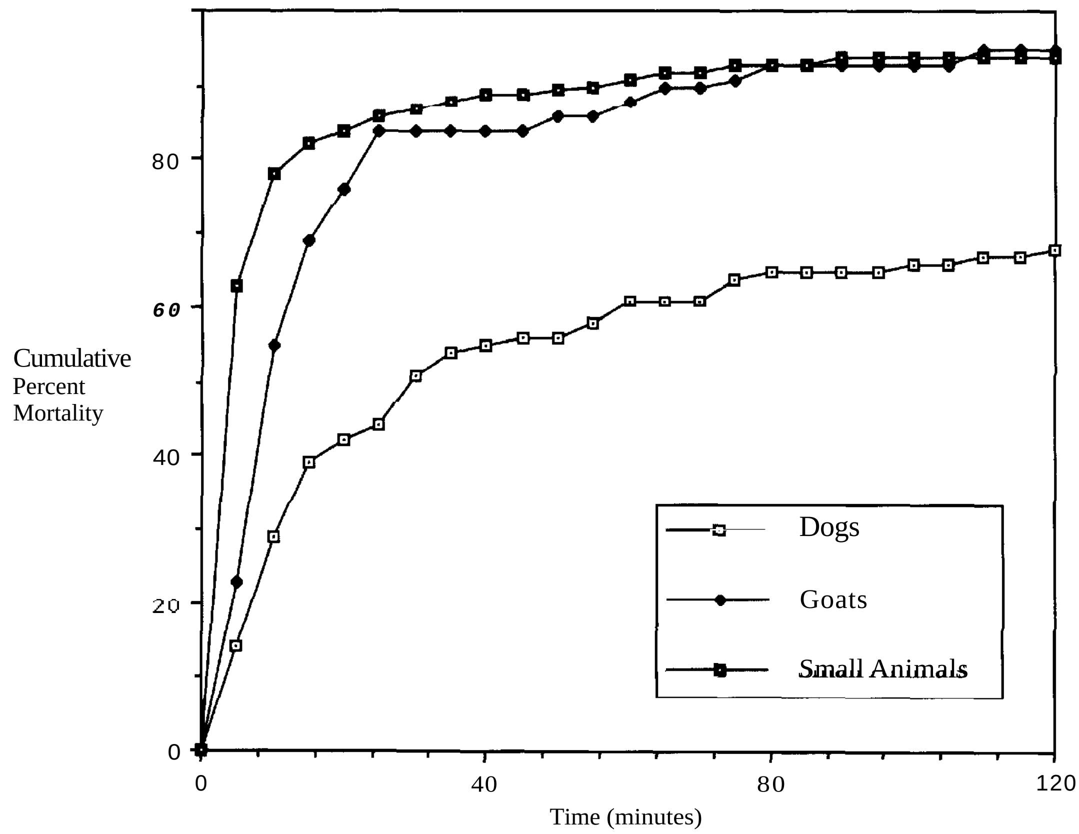

Fig. 8-16. This graph illustrates the cumulative mortality rates for various species of animals exposed to blast overpressures. The initial mortality rates appeared to be lower for the larger species.
Source: Redrawn from reference 9

embolism, which may be the principal cause of deaths that occur within the first hour after blast. In most studies, more than 50% of the deaths from PBI occurred within the first 30 minutes—and 75% during the first **2** hours—after the blast (Figure 8-16)? Autopsies on experimental animals that die soon after blast often reveal many air emboli.

No one knows how long air emboli may continue to be produced after a casualty is exposed to blast, nor how common emboli that are not clinically detected might bc. In a study of only onc animal, researchers used Doppler ultrasound to detect showers of air emboli in the carotid artery for up to 30 minutes after blast exposure.[24] However, most investigators believe that the clinically significant air emboli occur within the first 10 minutes.[9,10,22]

Emboli may be difficult to see at autopsy because the air bubbles may be absorbed after death.[10] Conversely, the prosector may accidentally introduce air into a blood vessel, leading to a false diagnosis of air embolism. In animal studies, the prosector is likely to see **air** bubbles within coronary arteries (Figure 8-17) or in arteries at the base of the brain (Figure 8-18). Air emboli have also been found in renal arteries after blast exposure.

The Heart

The heart may be damaged by blunt trauma from the blast wave, which may result in hemorrhage, or by the effccts of air cmbolism.

Hemorrhage. As is the case in other organs, hemorrhage is the most common blast-related lesion found within the heart. Cardiac hemorrhage is rarely seen without accompanying pulmonary hemorrhage, although the production of cardiac lesions may require higher blast doses than the production of pulmonary lesions does.

Epicardial hemorrhage usually occurs along the posterior surface where the heart directly apposes the diaphragm (Figure 8-19). Thus, the mechanism of

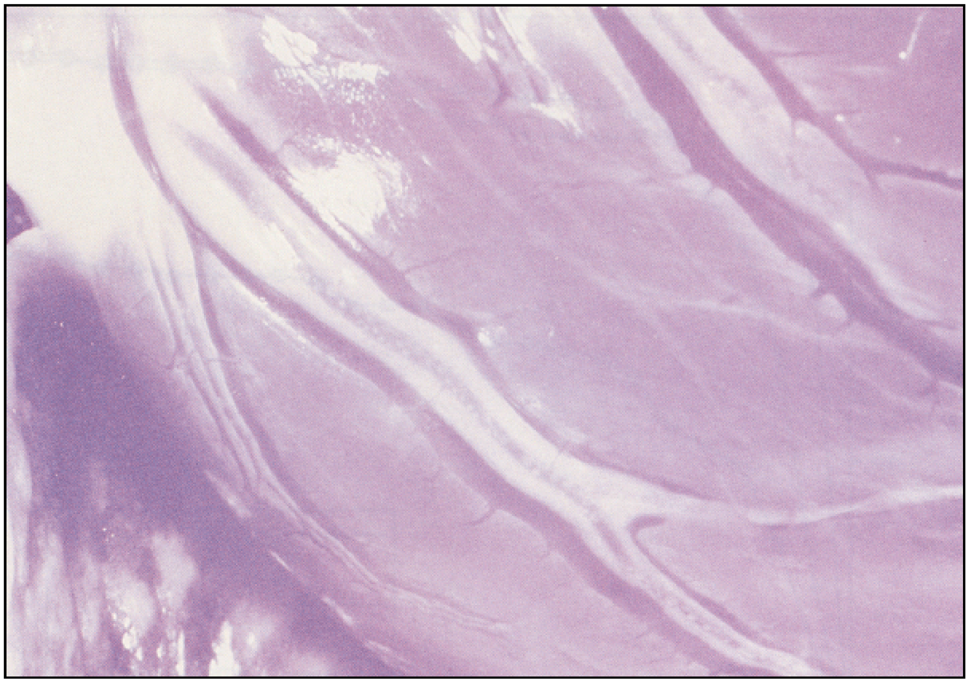

Fig. 8-17. This autopsy specimen from a dog exposed to a lethal blast dose shows air emboli within branches of the left coronary artery.
Source: D. R. Richmond

injury is probably direct contusion, which results from the diaphragm's rapid upward displacement. Myocardial hemorrhage is less frequent,'" and an occasional cardiac tear or laceration has been documented.[9]

Endocardia1 hemorrhage can commonly be seen at the base of the papillary muscles, in the surrounding ventricular endocardium, and occasionally on the valve leaflets (Figure 8-20). Histologically, erythrocytes infiltrate the endocardia1 connective tissue and may be seen surrounding the Purkinje fibers.

Cardiac Effects of Air Embolism. Changes within the myocardium from the presence of air emboli in coronary vessels are thought to be a major cause of death in blast casualties. The emboli may produce ischemia, which leads to subsequent degeneration and necrosis of myocytes (infarction). In the living subject, electrocardiography is a useful method for detecting these changes. Myocyte degeneration has rarely been described histologically in postmortem examinations of blast subjects.[25,26]

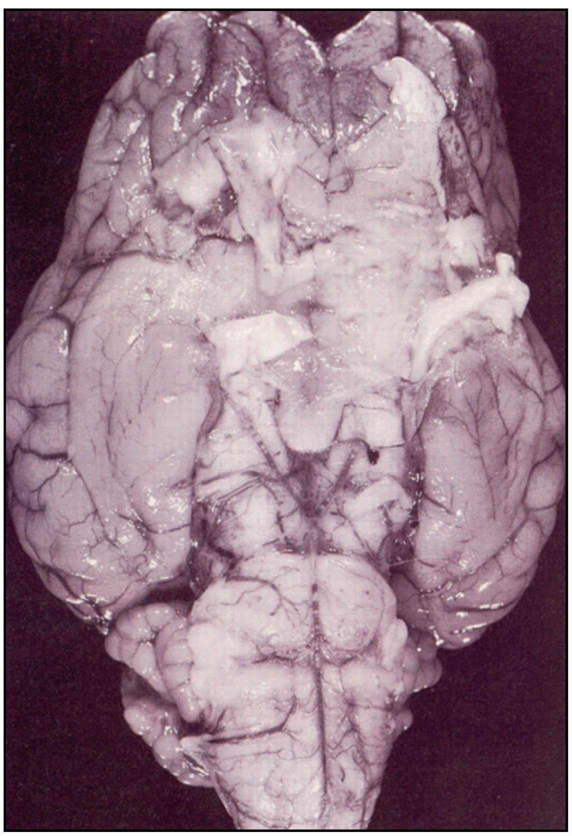

Fig. 8-18. This autopsy specimen from a sheep exposed to blast overpressure shows **air** emboli within the basilar artery and the posterior portion of the arterial circle of the brain.
Source: D. R. Richmond

The Blood Vessels

The blast wave affects the blood vessels in vulnerable organs, inasmuch a3 every hemorrhagc involvcs some blood-vessel damage. Although this damage may be widespread, it does not result from an injuring event that affects the circulatory system as a whole; rather, it is localized and is intrinsically related to the unique structures of the organs in which the vessels are found.

Autopsies of animals that had PBI have shown fibrin thrombi in the small blood vessels of the kidneys, adrenal glands, and heart.[23] These microthrombi **have** appeared **as** early as ***5* minutes** after the blast, and may be related to ischemic changes caused by the presence of air emboli. They may also be a manifestation of *disseminated intravascular coagulation* (DIC), rather than the result of direct ischemic injury to a blood vessel. For example, of five casualties of a civilian bus bombing who had lung injuries and severe respiratory failure, three also had DIC.[27]

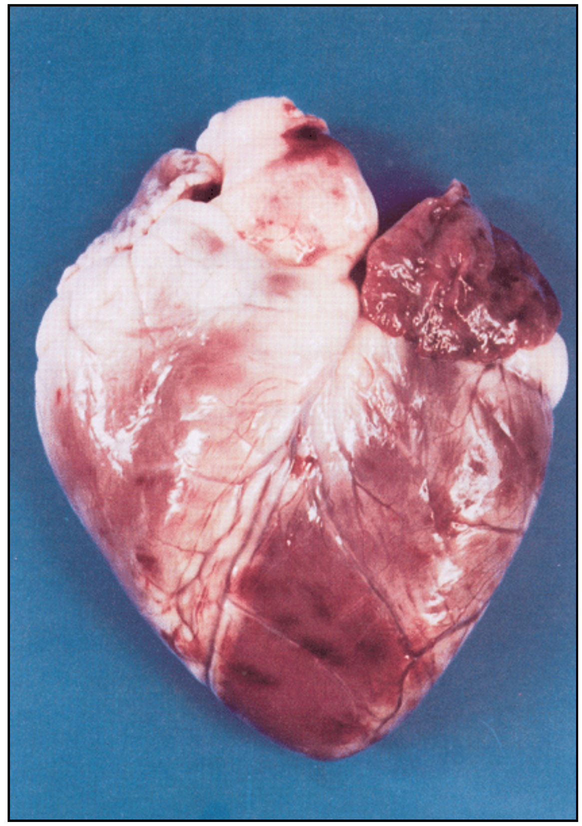

Fig. 8-19. This autopsy specimen from a sheep exposed to blast overpressure shows focally extensive epicardial and subepicardial hemorrhage. The hemorrhage is most severe on the left ventricular wall at the apex. In addition, ecchymoses can be seen on the right ventricle.
Source: Walter Reed Army Institute of Research

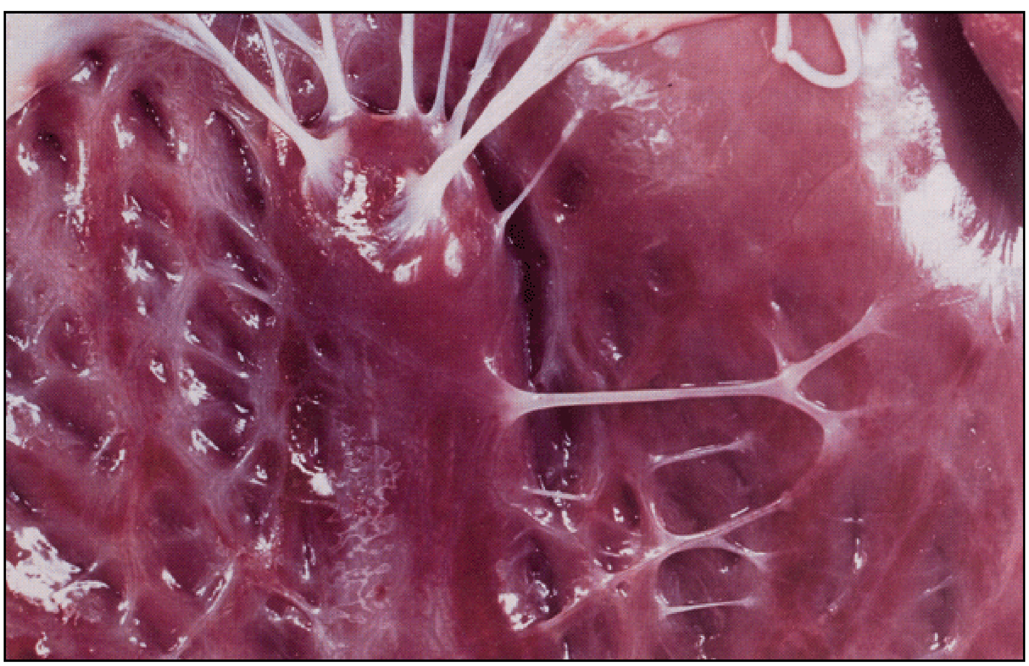

Fig. 8-20. This autopsy specimen from a sheep exposed to blast overpressure shows endocardia1 hemorrhage within a papillary muscle and the surrounding septal wall of the left ventricle.
Source: Walter Reed Army Institute of Research

THE DIGESTIVE SYSTEM

Like the organs of the chest cavity, the organs within the abdominal cavity that contain gas pockets—such as the gastrointestinal tract—are the most susceptible to blast injury. Solid abdominal organs may also be damaged at higher blast doses.

The Gastrointestinal Tract

Next to the damage they do to the respiratory system, pressure waves from air blasts most often injure the gastrointestinal tract, causing hemorrhages and tearing some organs within the abdominal cavity, particularly at high blast doses. For example, of twenty-nine casualties of a civilian bus bombing, four had injuries to the intestinal tract and eleven suffered lung injury.[28] Most studies of single air-blast exposure, however, indicate a much lower incidence of gastrointestinal injury.[2]

Studies performed on sheep have shown that gastrointestinal injuries from air-propagated blast waves may be more significant if the animals have been exposed to repeated blasts.[18,20] Sheep exposed to fifty blasts showed gastrointestinal lesions at overpressure levels that were actually lower than those that produced lung lesions. This information may be most important for those casualties who receive occupational-blast exposures, such as artillery personnel. Blast waves that are propagated in water, however, are much more likely to cause gastrointestinal lesions than those that are propagated in air.

Although they have been less thoroughly studied, the characteristics of blast injury in the abdominal cavity resemble those of blast injury in the thoracic cavity: *(a)* the blast wave strikes and displaces the body wall, causing distortion of the tissues within that results in their stress and failure; *(b)* no external injury is visible on the body wall; *(c)* tissues that contain gas are more vulnerable to injury; and *(d)* the most common lesions are hemorrhage and tearing of tissue.

Hemorrhages. Hemorrhages may appear along the entire gastrointestinal tract, but are most often found in the lower small intestine and the cecum, where gas commonly accumulates. They also seem to have a predilection for the antimesenteric surface.[29]

Hemorrhages that involve the intestinal tract range from small petechiae to large hematomas that may be found within the intestinal or gastric walls. Higher doses of blast pressure may cause a hemorrhagic ring, or annular band, which involves the entire circumference of a segment of gut wall (Figure 8-21). This damage may be the result of vascular compromise and is a likely site for perforation several days after the blast has occurred.

Within the gut wall, most of the extravasated erythrocytes are found within the submucosal or subserosal spaces.[9,10,18,30] The mildest hemorrhages are almost always located in the submucosa, although they have rarely been seen in the mucosa.[29] If the hemorrhages are the result of very high blast pressures, they may extend beyond the submucosal or serosal layers, and, as transmural hemorrhages, may involve the entire intestinal wall. Blood clots are commonly found within the lumen of the intestinal tract at the site of severe mural hemorrhage.

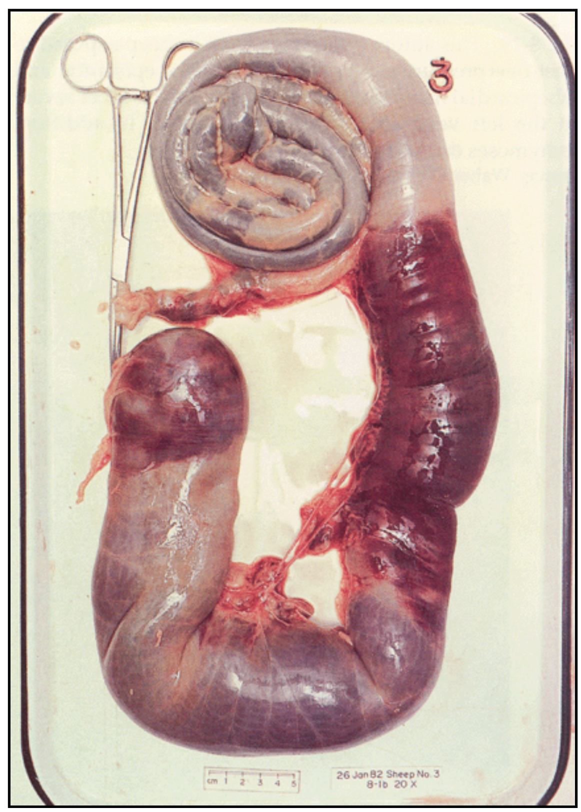

Fig. **8-21.** This autopsy specimen from a sheep that had been exposed to blast overpressure shows segmental hemorrhage of the intestinal wall.
Source: D. R. Richmond

Perforations. In the most severe blast exposures, the intestinal wall may actually rupture, resulting in blood and ingesta spilling into the peritoneal cavity.[9,18] The rupture may occur either immediately or up to many days after injury.

Like hemorrhage, gastrointestinal perforation tends to occur in the tissues surrounding gas pockets. In humans, for example, the ileocecal junction is a common site of intestinal perforation.[28,29,30] In sheep, the large gas-filled rumen is the most frequent site of both hemorrhage and perforation.[18]

Other Abdominal Organs

Other regions within the abdominal cavity also suffer the effects of the blast wave. *Paint-brush ecchymoses,* which are produced by approximately the same blast dose that causes intestinal hemorrhage, will commonly be seen multifocally throughout the mesentery, and occasionally will be associated with mesenteric tears.

Hemorrhage may also occur within the retroperitoneal space. In one pathological study of twelve casualties who had been exposed to immersion blast, ten of them had retroperitoneal hemorrhage into the loose areolar tissue behind the right colic flexure.[30]

The spleen and liver may be damaged by the violent displacement of the abdominal organs after the blast wave strikes. Relatively small, multifocal, subcapsular hemorrhages may occur in the parenchyma of these two organs. The capsules surrounding them may be ruptured, or the organs themselves may be fractured. The rupture of either organ will cause hernoperitoneum.

Subcapsular hemorrhage occurs in other organs (such as the pancreas, adrenal glands, and kidneys) but seems to be of minor clinical significance.[9,29] In one case, a laceration was found in the head of the pancreas.[10] A common lesion in male immersion-blast casualties is hemorrhage into the tunica albuginea of the testicle.[29] The urinary bladder and gall bladder are rarely affected by blast.[10]

THE EYE AND ORBIT

Because the globe of the eye has relatively equal density throughout, it is quite resistant to blast overpressure waves. Blast casualties may rarely experience transient blindness after exposure, as well as hyphema and conjunctival hemorrhage. Fundoscopic studies have revealed air emboli within retinal vessels, which may be the cause of transient blindness.[6]

One unusual blast effect in the orbital region, called involves portions of the frontal, sphenoid, and lacrimal bones on the medial surface of the orbital wall. In studies using dogs, these bones fractured from the force of the blast, and projected medially into the nasal fossa.[9] However, such orbital bone fractures have not been reported in humans, and may be unique to the shape of these dogs' skulls. In addition, the fractures occurred at such high overpressure levels that only a supralethal blast dose, such as might be caused by a nuclear bomb, would be likely to cause them. Such fractures cannot be easily detected, and may be missed on clinical or postmortem examination unless a retrobulbar hemorrhage has resulted in proptosis.

THE AUDITORY SYSTEM

The ear is the organ that is most frequently damaged by blast waves, and, at low blast doses (those below the level at which minimal respiratory lesions appear), may be the only anatomical site of detectable PBI. **As** is the case in other body systems that are vulnerable to PBI, the ear is damaged when the blast wave strikes and causes tissue distortion, tissue stress, and ultimately tissue failure. The ear is unique, however, in that its primary function is to transmit pressure waves from the environment to the inner ear, where they are converted into nerve impulses that are sent to the brain. Its structure amplifies any pressure waves—including damaging blast waves—along this existing conductive pathway, increasing the ear's sensitivity to levels of blast overpressure that might not be sufficient to cause PBI in other organs.

The auricle collects the sound waves, which are then focused within the external ear (auditory) canal. These waves cause the tympanic membrane at the end of the canal *to* vibrate. The air waves are transduced to mechanical vibrations and are transmitted through the ossicles to the perilymph of the inner ear (through the apposition of the stapes with the membranous oval window of the inner ear). These mechanical vibrations are transmitted through the fluid-filled chambers of the membranous labyrinth to the organ of Corti in the

Fig. 8-22. This osmium-stained example of a triangular rupture (arrow) in the tympanic membrane of a chinchilla is viewed from the external ear canal. The *a* in the lower right corner refers to artifact.
Source: R. P. Hamernik

cochlea, where transduction again occurs and mechanical vibrations become nerve impulses.

Waves with sufficient peak pressures may overload the auditory system and cause damage to any of its component parts, including *(a)* ruptures of the tympanic membrane, *(b)* dislocations or fractures of the ossicular chain, and *(c)* damage to the organ of Corti within the cochlea.

In addition to the damaging effect of the transmission of the blast wave along the auditory conduction pathway, the rapid overpressure that develops within the air-filled tympanic cavity of the middle ear as the tympanic membrane is pushed inward by the blast wave may play a role in auditory blast damage. Normally, pressure equilibration within the tympanic cavity occurs via the auditory (or eustachian) tube, which connects the middle ear with the pharynx. This conduit is usually closed, but will open to equilibrate pressure during chewing, swallowing, or yawning. A blast wave, however, increases the air pressure within the tympanic cavity so suddenly that the overpressure cannot be relieved through the auditory tube. The resulting distortion of the surrounding tissues damages them, especially the delicate tympanic membrane that separates the cavity from the external ear canal.

The ear facing the blast will usually be more severely damaged, although injury is frequently bilateral, especially when the casualty is within an enclosed structure and vulnerable to complex blast waves that can damage the contralateral ear.

The Tympanic Membrane

Most of the tympanic membrane comprises the *pars tensa*, a thin, bilayered, collagenous sheet that is sandwiched between an external layer of skin and an inner layer of simple squamous epithelium. A small portion of the membrane in the anterosuperior quadrant is devoid of collagen fibers, and is called the *pars flaccida*.

Tympanic-membrane rupture frequently occurs with blast exposure (Figure 8-22). In humans, the pars

Fig. 8-23. This scanning electron micrograph shows a noise-induced dislocation of the organ of Corti. Note the relatively intact line of inner hair cells (I) and outer pillar-cell processes (P). Some Claudius cells (C) also have been dislodged.
Source: R. P. **Hamernik**

tensa is the portion of the tympanic membrane that is perforated by blast waves.[31] Perforations of the pars flaccida have been documented in experimental animals that were exposed to blast, but even these ruptures were less frequent than and always occurred in conjunction with ruptures of the pars tensa.[32] At doses below those that cause perforation, abnormal tympanograms indicate the probable rupture of radial collagen **fibers,** producing **a** flaccid **but** not perforated membrane? In humans and animals, perforations usually involve less than one-third of the surface area of the tympanic membrane.

Experimental animals with blast-induced auditory trauma may suffer hemorrhage within the lamina propria of the tympanic membrane, which may progress to form a hematoma. This hemorrhage is often found around the periphery of the pars tensa or immediately below the anterior and posterior malleal folds.[32]

Although most perforations of the tympanic membrane heal spontaneously, cholesteatoma formation may be a sequela in rare cases.[31] Cholesteatoma is formed when the epidermis from the external tympanic membrane grows through the site of perforation into the middle ear, and forms a cystic structure into which layers of keratin admixed with cholesterol crystals are secreted. These cysts can grow, eventually impairing hearing and eroding surrounding structures.[34]

The Ossicular Chain

The tiny ossicular bones lie within the middle ear. The *malleus* is attached to the tympanic membrane laterally, the *stupes* is attached to the inner ear at the oval window, and the *incus* is suspended between the two. These bones normally transmit and amplify mechanical vibrations from the tympanic membrane to the inner ear.

During blast exposure, the ossicular chain may be damaged by direct displacement of the bones, or—more likely—by a severe distortion of the tympanic membrane at the attachment of the malleus. Ossicular damage is rare, however, even if the tympanic membrane has been injured. In fact, even blast doses that are strong enough to cause inner-ear injury may not damage the ossicular chain.[35]

When blast-induced ossicular damage does occur, pathological changes may include *(a)* medial displacement of the malleus handle with disruption of the incudomalleal joint, or *(b)* less commonly, incudostapedial joint separation with and without stapes fracture.[36] In studies with experimental animals, researchers noted that 29% of animals that had rupture of the tympanic membrane also had fractures of the malleus handle.[32]

The Cochlea

The transduction of mechanical vibrations into neural impulses occurs within the cochlea at the organ of Corti, a highly specialized epithelial layer that lies upon the basilar membrane and is comprised of sensory hair cells, sustentacular cells, and a tectorial membrane. The apical surface of this epithelial layer is bathed in the endolymph of the cochlear duct and is called the Here, vibrations in the basilar membrane cause distortions of the hairs and a subsequent neural impulse within the auditory branch of the eighth cranial nerve.

In blast injury to the cochlea, abnormally intense mechanical vibrations reach the oval window of the inner ear through the ossicles, where they are transferred to the perilymph of the labyrinth and loaded onto the basilar membrane. The delicate organ of Corti cannot withstand the resulting stress.

Rcscarchcrs who study thc cffccts of blast wavcs on experimental animals have noted severe damage to the organ of Corti.[32] Scanning electron microscopy most commonly revealed fracture of the reticular lamina and dislocation of portions of the organ of Corti from the basilar membrane (Figure 8-23). Less severe changes include loss or fracture of inner and outer hair cells.

SUMMARY

Casualties who have been exposed to blast waves suffer a variety of lesions, the extent and severity of which depend upon the dose. Despite the multisystemic nature of these lesions, it is important to remember that it is the lung injuries—the occurrence of alveolar hemorrhage and the production of air emboli—that are usually involved in the casualty's death.

Much of the knowledge of blast pathology presented in this chapter has been attained through animal experimentation that has been conducted over the past half-century. These lesions qualitatively correspond well with those that have been described in reports of autopsies of human blast casualties. Difficulty arises, however, in predicting the extent of injury for a given dose of blast exposure. Researchers have expended extensive time and resources to model and predict injuries in humans based upon experimental-animal data. This chapter has cmphnsizcd thc qualitative aspects of PBI; evaluation of the dose-response data is beyond its scope.

Current studies of the pathology of PBI are being conducted on several fronts. Using some of these data, researchers are developing computer programs that can predict injury and lethality thresholds in humans based on blast dose. Further studies are also needed to understand the factors that may complicate the progression and resolution of PBI, such as exercise and infectious disease. Research is being initiated to study the role of inflammatory mediators and cytokines in the progression of PBI, as well as the development of more specific diagnostic tests and therapeutic regimens.

Medical officers who may have to treat blast casualties need a thorough understanding of this trauma, which is often hidden from visual inspection. Familiarity with the pathology of PBI can help medical and triage officers to recognize its signs and initiate proper clinical intervention.

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