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Effects of high explosive detonation upon the human body

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THE EFFECTS OF HIGH EXPLOSIVE DETONATION UPON THE HUMAN BODY

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TABLE OF CONTENTS

INTRODUCTION

Even to a neutral observer this war is truly different from previous struggles, both in its conception of attack and defense, as well as to the danger to civilian communities. As a result of the development of long range artillery operations (aerial warfare) with ~~subsequent developing~~ concomitant danger from bomb explosions, the medical world is presented with a hitherto partially recognized and inadequately studied problem. Pure "blast" injury without visible external wounds was reported during and immediately following the last war, but due to the current medical problems of the time, the subject was not thoroughly investigated and was soon pushed into the background.

However, since the onset of the present crisis, many clinical and experimental studies have been made on the differing types of injuries produced by the detonation of high explosives and the results have given rise to varying conclusions.

It is, therefore, the object of this paper to give a general summary and analysis of the experimental and clinical investigations of the effects of the detonation of high explosives, hoping that it may draw our attention to a serious civilian and military problem of this present war.

Detonation of high explosives or "blast", as it is most frequently cited, produces pathological changes in various regions of the body. The most predominating areas so affected are: (1) the pulmonary system; (2) the abdomen; and (3) the central nervous system. The effects of blast in the air and in the water differ somewhat, so that for all practical purposes each will be discussed separately.

I have, therefore, divided the paper into three separate entities; (1) Air or atmospheric blast; (2) Immersion blast; and (3) The effect of detonation on the central nervous system. These will be discussed in chronological order.

AIR OR ATMOSPHERIC BLAST

I. Nature of the Blast.

Blast is simply an excessively intense sound wave which is longitudinal in propogation; that is to say, the air particles propogate the distance by moving backwards and forwards in the line of disturbance itself. For the sake of simplicity imagine that the origin of the blast is a small sphere of explosive material. When this is fired, it expands rapidly, compressing a spherical shell of air immediately surrounding it. The air cannot avoid this sudden pressure, but once it is released, the elasticity of the compressed air in the shell tends to make it recover its original state. Thus, when a bomb detonates, the solid explosive is converted into gases initially confined in the casing at high pressure. As a result of this pressure, the casing is blown to pieces, the gases escape, and a blast wave in the surrounding air is produced. An outside shell of air immediately surrounding the bomb is, therefore, compressed, leaving a shell of rarefied of air behind. This produces the negative or "suction" pressure wave, which immediately follows the initial pressure wave. Thus, the blast wave is composed of an initial compression wave followed by a suction wave, the two being propagated outwards. The air particles, meanwhile, move outwards from or inwards toward the center, depending upon the particular phase of the disturbance.

This wave moves with extreme rapidity, especially close to the explosion, the point of highest pressure, but falls very rapidly the further the wave moves from the source of explosion. (Fig. 1) The suction wave of the co mponent is of longer duration, but much weaker, than the pressure component and in no case exceeds a pressure greater than fifteen pounds per square inch, since this corresponds to a perfect vacuum. (Fig. 2)

The total duration of a whole wave at any given point within two hundred feet is very brief. Thus at thirty feet from a seventy pound charge, the pressure component lasts about five and the suction component thirty milliseconds. (Zuckerman-78; Sutherland-72; Bernal-4).

Since the shell is moving rapidly, it also exerts in the direction of motion an additional wind pressure. Zuckerman(78) states that close to the explosion this pressure may be as great as the initial hydrostatic pressure, but falls off much more rapidly than the latter, producing, therefore, very little effect. Williams(76), on the other hand, believes this "windage" is responsible for the shattering effects produced in the immediate proximity of the explosion and that, irrespective of any previous damage inflicted, contusional effects can be produced.

The magnitudes of these pressures, the suction components, and the time they last are directly proportional to the

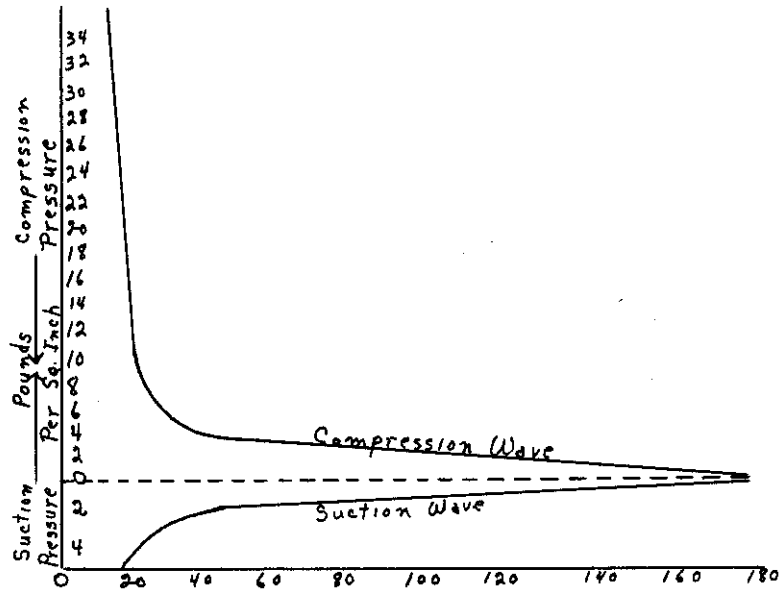


Fig. 1.

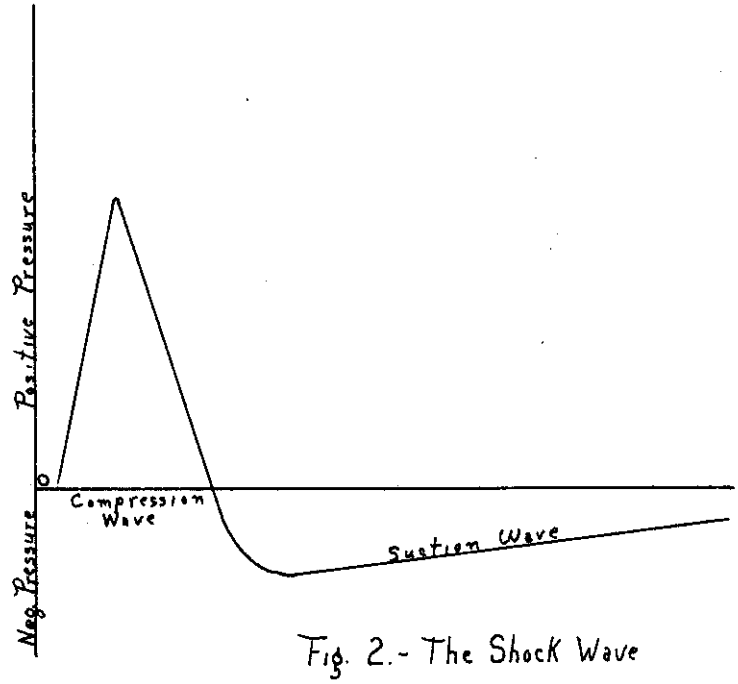


Fig. 2.- The Shock Wave

amount of the explosive, the values being much higher in the case of a larger charge. The pressure recordings from bomb explosions, therefore, vary with the distance, the size, and the weight of the bomb. For example, at a distance of fifteen feet from an exploding one hundred and twenty five pound bomb, the hydrostatic pressure may be as high as two hundred pounds per square inch, whereas at a distance of fifty feet, the maximum pressure recorded will not exceed ten pounds.

Since the majority of bombs explode beneath the ground, the ground wave in the earth or the "ground shock" also deserves some mention. When bombs drop from great heights, they penetrate some distance into the earth. This ground wave has a very high velocity and is characterized by its abrupt rise in pressure. This explains the fact that buildings are often destroyed at considerable distances from the site of explosion.

When a bomb explodes, the case first swells and then breaks into many small fragments. Failure takes place in tension along planes at forty five degrees to the bomb surface, yielding angular fragments which are driven forward by the expanding gases. These fragments have very high velocities, often up to four thousand feet per second, thus producing a high degree of penetration.

In ground blast the most serious effects are produced, as in atmospheric blast, at points nearest the explosion. However, in this case they are due to the bodily movement

of the earth and not to the pressure wave. Following the explosion the earth is pushed aside, forming an initial sphere of expansion. If the explosion is mild, this earth merely fall back into its original position, but if it is severe the displaced earth is moved outwards to a considerable degree, being partly plastic and partly elastic in nature. This is not an instantaneous blow, as in air blast, but more orless of a continued push, thus producing a prolonged effect. This in turn would naturally result in a prolonged pressure effect, so that it can be seen how water and gas mains may be broken.

When the wave from an underground explosion reaches the surface of the earth, it is ordinarily reflected as a tension wave. However, if the force is large enough, the gas passes from a more dense to a less dense medium so that it is transformed into kinetic energy. Then the earth seems to "blow off" forming at first a conical shaped scab which breaks up and leaves the characteristic crater behind. If the bomb explodes too deeply, this process does not occur and the earth merely settles back down to its original position. This is termed a "camouflet". (Bernal-4)

Bomb explosion within a house has been reported by several authors and appears to be more severe than cases subjected to blast in the open. When a bomb penetrates a house, it immediately explodes and results in the formation of a wave

which blows the walls out. Some of these waves, nevertheless, are reflected back in more or less the same direction of the origin of the blast, adding increased and prolonged effects to the original compressive force. This, therefore, tends to be more destructive than the wave in open air blast and explains why more seriously injured patients are oftentimes found within enclosed structures. Osborn(53), however, believes there is little, if any, true blast and is of the opinion that the damage results from compression of the chest by falling masonry and debris at the time of the explosion.

However, it is difficult to accept this latter theory, as consistent reports of authentic men have shown that there is only mild degrees of damage to persons occupying adjoining rooms even though they have been subjected to the falling missiles and debris. If it was due primarily to falling debris, why should not these be affected to as great a degree? Thus, it seems that once these waves strike the walls, they are partially absorbed by their action so that persons in an adjoining room are not subjected to as severe a compressive force.

II. Experimental Evidence.

Conditions which we now know to be the result of high compression waves or blast were recognized during and following the last war, but so many varying signs and symptoms were produced that they were classed under widely variable category of "shell shock". Therefore, due to the misunderstanding of and the lack of interest in the subject, very little early experimentation work was done .

At first investigators thought the central nervous system was especially sensitive to this form of trauma. This received the full support of Mott(51). However, experimentation on animals has now shown that pulmonary lesions predominate.

Marinesco(46) was among the first to report his animal experimentations. Dogs were exposed to small charges of "fulmicoton". Blood was observed in the nose and mouth of one of the animals; others exposed in shelters later suffered from dyspnea, depression, and difficulty in walking. He reports hemorrhages in the central nervous system, but more obvious ones were found in the lungs.

Carver and Dinsley in 1919(9) report experiments on fish, rats, and mice. They noted hemorrhage from the ear, nose, and mouth with varying degrees of hemmrrhage into the viscera when the animals were sacrificed. The important thing is thatthey observed for the first time rupture of the alveoli of the lungs with mimimal lesions in the central nervous

system.

Mairet and Durante(45) in the same year exposed rabbits to concussion shock and report minute hemorrhages in the lung. They state, however, that the lesions occurred particularly in the spinal and cranial nerve roots at their points of passage through the bony foramina. Blood suffusion, irregularly distributed, occurred on the surface of the spinal cord with small clots adhering to the nerve roots. The brain appeared normal or showed various points of petechial hemorrhage. From these observations they proposed the theory that vibrations in the atmosphere set up a "commotion" within the tissues, especially the central nervous system, and caused the damage. These men continued to observe some of these animals for several months after which they performed autopsies. No residual effects whatsoever were noted. Had there been as severe damage as previously reported, surely some residual scar tissue or abnormalities would have been present.

In 1924 Hooker(33) did some work in determining the physiological effects of air concussion. This was the first time the pulmonary system was noted to be particularly susceptible. He exposed dogs, cats, rabbits, and frogs to small charges of explosive gun blasts at various distances and observed at post mortem that hemorrhagic areas of hepatization was the single gross lesion. He concluded that injury to the lung doubtless contributed to death in the animals, if not the primary cause.

He also observed that the rabbit, with a much weaker bony framework than the dog, was more easily injured by the air concussion. Thus, the resistance of the body tissues seems to be of significance. This may explain why the hemorrhagic lesions found by Mairet and Durante(considered as "commotion") are not present in the human, since the bony framework gives much greater support and protection.

He mentions the fact that the shock produced from concussion differs strikingly from shock produced by any other means and suggests it to have a completely independent etiology. That it was in no way associated with the severity of the pulmonary damage is clearly indicated by the failure to produce any shock when the lung showed the most extreme degree of injury. Veinous pressure was decreased, which is contrary to the general conception of vaso-motor collapse. He concluded that shock from detonation of high explosives results from a complete vaso-motor collapse, affecting not only the arterial bed, but also the veinous and possibly the capillary tone.

His experiments upon frogs did not result in any interference with the peripheral neuro-muscular mechanism or any abnormalities of reflex movement and muscle response to nerve stimulation. This fact points toward no injury to the vital centers of the brain.

Thus, Hooker's magnificent experimentation is the first good evidence toward pulmonary damage in concussion from high explosives and tends to exclude the primary importance of central nervous system injury.

Barcroft(3) in determining the physiological effects of "blast" used goats and rats so arranged that injury by splinters was impossible. The animals were exposed to a severe blast of five hundred pounds both in the open and in shelters. Four deductions were reached: (1) Only animals placed in the open and at fifteen feet from the bomb suffered ill effects; (2) shelters gave protection; (3) animals beyond fifteen feet did not suffer any demonstrable lesions; and (4) the lesions found were essentially in the lungs and were the primary cause of death when it occurred. Lesions occurred only in those animals outside of the shelters, and thus he concluded the injury was due to a distension of the lungs from the compression force acting through the mouth and trachea.

Zuckerman(77) in 1940 makes the first attempt to determine the mechanics by which the injury is produced in the lung. Prior to this, however, various authors had speculated their possibilities. Among these were: (1)that the lesions are formed by the lowering of alveolar pressure from the suction wave acting through the

respiratory passages with consequent rupture of the alveolar capillaries. (Logan-44; Shirlaw-68; Lockwood-43). This was also the theory conformed to by Shirlaw and Haldane(28) in their observations on casualties in Spain.

(2) The second possibility is that the lesions are caused by distension of the lungs with air, the view taken by Barcroft.

(3) The third possibility is that proposed by Hooker(33), namely, that the lesions are due to the impact of the pressure wave against the chest wall. This is also the view taken by Kretcschmar(39) in his recent clinical description of blast casualities in Spain.

Zuckerman(77) concluded that in the case of bomb explosion a man may be wounded by: (1) being hit by fragments or sent flying by the explosion; (2) trauma from the subject's being thrown to the ground or against a solid structure; (3) injury by secondary missiles, such as masonry, etc.; and (4) being affected by the blast wave without being thrown.

He did some very classical experiments on mice, rats, guinea pigs, rabbits, cats, monkeys, and pigeons. At first the animals were exposed at various distances to a blast from a seventy pound high explosive placed in paper bags upon the ground. Later, other animals were subjected to blast from explosions of hydrogen and oxygen in balloons.

In the former experiment, there were no animals killed at distances further than eighteen feet and none were hurt in any observed way at distances greater than fifty feet. Between twenty and fifty feet no animal was killed and very few showed any change in behavior. Animals at twenty feet and those surviving at eighteen feet often breathed irregularly and showed signs of respiratory embarrassment. In summarizing his findings Zuckerman concluded that there are five zones of effect surrounding the explosion. These include (1) a zone immediately adjacent to the explosion in which all animals were blown to pieces; (2) a zone adjacent to the first in which all animals were killed without external injury, but which showed blood stained froth or blood in the nose, mouth, and upper respiratory passages; (3) an area still further away where animals were found alive immediately following the explosion, but died at intervals of between one minute and one day. These also showed blood stained froth, but suffered from dyspnea and tachypnea as well. These animals were apathetic, quiet, and disinclined to feeding. (4) An area still further distally in which the above stated symptoms were present, but in which recovery was complete. (5) A zone where neither internal or external effects were observed.

In all of his specimens there was no evidence of external injury, the damage occurring internally. The most

outstanding lesion was bilateral traumatic pulmonary hemorrhage varying in degree with the distance of the animal from the charge. These hemorrhages tended to follow the line of the ribs and in severe cases spread into the more superficial structures from lacerations. This would suggest that the injury was due to direct compression of the ribs against the lungs.

In all specimens where the degree of lung damage was sufficient to kill the animal, blood was found in the bronchial tubes, trachea, and usually the mouth and nose. This would explain the presence of the bloody discharge evidenced in human cases.

Similar conclusions were reached through his experiments with hydrogen and oxygen in balloons. However, since the animals could be placed much closer to the blast, he found that only the side exposed was affected. This definitely proved that the lesions were produced by the impact of the pressure wave from the outside. For if the blast affects the lungs through the respiratory passages, either by the suction wave or overdistension, why would the animals receive lesions only on one side of the lungs?

In another set of experiments the animals were wrapped in sponge rubber and the injuries were greatly diminished. In some cases only half the body was covered with sponge rubber. When the covered side was placed toward the explosion,

the animal sustained practically no injury, but if this side were away from the explosion the uncovered side received many lesions. He also showed that animals placed in boxes with their heads out had milder lesions. This would definitely exclude the possibility of the blast acting through the respiratory passages as proposed by Barcroft(3).

Microscopically the alveolar walls were often times disrupted and the alveoli were filled with hemorrhage, a result of tearing the capillaries. In severe cases hemorrhage was also present in the larger bronchi.

Thus it can be seen that these experiments have cleared up many controversial points concerning the mechanism of blast injury from high explosive bombs. They have shown that pressure waves seem to directly affect the chest wall and are not a result of the action of the wave thru the trachea and bronchi. However, several authors still conform to this latter theory, especially the suction wave component. In response to these contradicting statements, Latner(42) exposed mice to a rapidly produced lowered pressure of short duration. He reports injuries very similar to those found in blast. As to the mechanism of injury, he, too, agrees that the pressure changes are not directly communicated to the inside of the lungs this being born out by the narrowness of the trachea and the exceedingly

short duration of the blast wave. However, if this holds true, the intra pulmonary pressure would be greater than the outside pressure at the time of the negative pressure change. The prolonged effect of the suction component plus the surface area of the body would tend to make this pressure distance very effective, resulting in over-inflation of the lungs with subsequent tearing of some of the pulmonary capillaries.

Therefore, the effect of the suction wave cannot be entirely excluded and even though the majority of evidence is in favor of the compression w ve against the chest wall, it seems only logical that the suction component may ad to the injurious effects.

No definite factor can be determined regarding the cause of death in these cases. True enough, pre-mortem symptoms indicate that asphyxia might well cause death to those cases which survived the initial blast only to succumb sometime later. This does not explain why death occurs directly at the site of the explosion. Such a sudden death would surely be the result of some fatal injury to the vital centers although Hooker reports that all reflexes, actions, etc., were normal in his animals, suggesting no evidence of injury to these areas. However, in my mind, this possibility is hard to exlude. Could there not be sudden compression of these centers with resulting ischemia

and death without any evidence of damage?

Stewart et al(71) recently reported some experimental work of the effects of high explosives to the central nervous system. They concluded that even though lung injuries are produced, they are not likely to result in sudden death but only contribute. These men suggest that the hydraulic-like pressure on the central nervous system in its firm encasement causes sudden compression of the thoracic cage with a consequent back-pressure on the venous side. This might explain the mechanism by which the vital centers are suddenly compressed.

III. Clinical Investigation

Several things must be observed in considering blast casualities of humans. The effects are not exactly the same as those produced under experimental conditions, as these may be altered by the circumstances under which the explosion occurs. Thus, when a bomb explodes after it has penetrated into the ground, the blast is mainly directed upwards in an expanding zone and people standing or lying close to the edge of the crater will be protected from the wave of increased atmospheric pressure. Furthermore, when a bomb detonates close to walls, the blast is reflected and its energy absorbed in odd ways, so that people situated nearby may escape the impact of the wave. Again, blast waves close to the bomb site are very irregular so that persons situated at the same close distances may be subjected to different pressure waves. This may help to explain some of the odd casualties noted by various authors.

In general, very few cases of direct blast injury have been observed. Circumstances under which people are exposed (flying masonry, etc.) make it difficult to conclude that they could incur injuries directly as has been so remarkably reviewed by Dr. Zuckerman.

Zuckerman(78), himself, concluded that the blast wave

has only a moderate effect beyond thirty feet from the point of detonation. Thus, some of the so called blast casualties so far reported are not true blast injuries, but are associated with other mechanisms.

The literature of this subject is somewhat limited, as little was known concerning it prior to World War I. Since then it has been crowded out by other medical problems.

Hatton(30) reports the earliest cases due to explosion. He received 343 victims from a mine explosion none of whom showed external damage. He states that the majority died from carbon monoxide poisoning whereas immediate death probably resulted from a sudden, compressing force crushing the vital centers.

In the Official History of the Great War it is reported that dead men were sometimes picked up in the field of an explosion without external evidence of injury. Oftentimes blood trickled from the nose and mouth. This is compatible with many of the clinical cases now known to be a result of blast. At that time practically no post mortems were done, and those that were held were observed particularly for lesions of the brain and spinal cord where petechial hemorrhages alone were found. Mitchiner and Cowell(50) and Langdon-Davies(41) reported

that men picked up from the field during the Spanish war often suffered from hemoptysis and considerable respiratory distress and sometimes appeared drowsy or even semi-conscious.

Neverthless, from this meager bit of literature the prevailing ideas as to the production of lesions and death seemed to fall under two categories: (1) That they resulted from gases such as carbon monoxide, (2) That they resulted from a sudden vacuum effect in areas which contain gas (lungs, stomach, or intestine) with subsequent liberation of gas bubbles into the blood stream by the sudden evacuation of air. These bubbles may then block the circulation just as occurs in compressed air workers, divers, etc. (Hill-31; Johannides andTsoulas-35; Thomas-73)

Mott(51) also considered carbon monoxide a factor in many cases of concussion shock, particularly those exposed in trenches and dug outs. However, in the majority of cases he believed the injury was caused by the enormous aerial compression being transmitted to the fluid around the base of the brain causing shock to the vital centers in the floor of the fourth ventricle and resulting in instantaneous arrest of the function of the cardiac and respiratory centers. This, he held, explained many of the instances of sudden death under such conditions. He also noted edema and hemorrhage in both lungs but considered it of minor importance,

stating that the small bulbar hemorrhages, veinous congestion and chromatolysis of the vago-acessory nucleus were the cause of death.

Southard(69) and Stevenson(70) stated that hemorrhage into the corpus callosum and basal ganglia was caused by the transmission of the violent shock to the incompressible fluid within the ventricles of the brain.

Dale and Laidlow(14) were of the opinion that the concussion shock is due to the release of a toxic substance like histamine into the blood stream. The sudden effect of the shock seems to make this point impractical.

Various theories had been proposed, but none of them could be definitely accepted. The majority favored the susceptibility of the central nervous system to the blast, but more recent experiments have shown that pulmonary lesions predominate.

Reports on cases diminished until the onset of the present crisis from which excellent reviews have appeared.

S. T. Falla(16) in 1940 cited the first human case simulating experimental animals previously described. This patient was a man of thirty years who was injured by the explosion of a medium caliber bomb at a distance of forty five feet. He did not recover from the shock incident, but died about twelve hours following. A post

mortem was performed and very striking changes were noted in the lungs. Fresh hemorrhages were evident all over the pleurae. The cut surface of the lung showed innumerable small hemorrhagic areas, both lung fields seeming to be equally affected. Small submucosal hemorrhages were noted in the trachea and bronchi. Microscopic study showed generalized extreme arterial dilatation with intense focal capillary dilatation and exudation of fluid into the alveolar spaces. The other organs showed no change.

Dean, Thomas, and Allison(15) reported a series of twenty seven cases in which only six gave clinical evidence of chest pathology. However, sixteen patients in the group showed physical signs and thus they stressed the relative disproportion of the two stating that chest complications may arise after an explosion blast without definite warning symptoms. This should impress upon our minds the necessity of a routine physical examination upon patients who are apparently unaffected by the blast. They also noted the "blown up" or balloning position of the chest, especially along the costal margins, and the radiological appearances of dimunition of rib expansion together with the slight loss of translucency, suggesting a "bruised pleua".

Heretofore, it had been generally assumed that the symptoms and clinical signs produced from blast were wholly a result of capillary rupture and hemorrhage into

the alveolar spaces.

Hadfield(24) reported a series of post mortem cases in which there was free capillary bleeding over large areas. The bronchioles, atria, and alveoli showed uniform and considerable overdistention. He concluded that the capillary rupture and hemorrhage, though present, was hardly enough to produce the clinical manifestations shown by these patients. It seemed to him that the major clinical manifestations were probably due to capillary dilatation, veinous congestion, and edema in the lung and that the hemorrhage itself was probably more of a mechanism of diapedesis.

In a later report Hadfield and Christie(26) describe a case who lived for fifty one hours following a blast. Post mortem revealed much more extensive pulmonary hemorrhage than was noted in those who died early. They thus concluded that bleeding continued into the lung for twenty four to forty eight hours in cases which have survived the initial blast and that if a patient was transported from one place to another, there is a greater danger of re-establishing the hemorrhages. Osborn(53) disagrees with these men on this point. He cites the following factors as direct evidence against progressive hemorrhage within the lung: (1) that capillary hemorrhage typical of these cases ceases within five minutes and requires only a slight increase in pressure to stop it; and (2) that the most advanced organization of

the hemorrhage was at the upper end of the lesion nearest the unaffected tissues. He still does not explain the fact that more severe grades of hemorrhage are found in cases surviving several hours. At present this point is debatable and requires further study and observation.

Hadfield and Christie also noted greater damage occurring in children after exposure, possibly due to the less rigidity of the thoracic wall. This was the belief of King(37), King and Curtis(38), and Boland(5), but contradictory to that of Payne(60) who states more compressible substances tend to absorb the shock and prevent injury.

In further reports Hadfield(25) remarks that the size of the intrapulmonary hemorrhage reaches its maximum by four hours. Shortly following this a fibrin network forms in the alveoli and a mononuclear cell exudate appears. The histological appearance by thirty six to forty eight hours is strikingly that of the stage of red hepatization in lobar pneumonia and has received the name "pseudo-pneumonia". He demonstrated for the first time infiltration of the intercostal muscles with blood and believed this to be the cause of the extreme tenderness over the thorax.

Roberts(64) pointed out the contraindication to abdominal surgery in these cases, citing two cases in which the abdomen had been opened, neither of which presented any

demonstrable lesion. This, plus the intercostal bleeding, suggests that the pain and rigidity of the abdominal wall arises from irritation of the intercostal nerves innervating the abdominal musculature. He emphasized the fact that no inhalation anesthesia should be given if there is progression of the hemorrhage.

O'Reilly(55) was likewise misled by these abdominal signs. He opened two abdomens and found no pathology whatsoever other than minute submucosal hemorrhage of the intestinal wall. He concluded that the intercostal irritation surely must be the explanation.

Great caution must, therefore, be instituted in interpreting abdominal signs of these cases and surgical procedures should not be considered unless there is absolute indication of intra-abdominal injury with suggestive continuous bleeding.

Heretofore, actual intra-abdominal injury from air blast has not been mentioned. Zuckerman(79) reported small submucosal hemorrhages into the intestinal walls of animals, but observed no perforations. Osborn(54) was the first to make any definite mention of this fact in human cases. He noted perforations in two cases, but states that these resulted from secondary compression by falling debris. He proposed that the submucosal hemorrhage resulted from blast alone, a point also born out by Krohn(40). He likens it

to his "phrenico-costal-sinus pneumonia" and says that injury to this area is good evidence toward rupture of the liver and spleen. This has not been confirmed by other authors. A few other men have mentioned the fact that there is some evidence of intrabdominal injury in air blast, but the only confirmation at autopsy was the previously mentioned submucosal hemorrhage in the intestinal walls. Thus, it is generally conceded that very little abdominal damage results from exposure to blast in the atmosphere.

Osborn(53) mentions the similarity of these lesions to those produced in peace time such as car accidents, etc. He boldly states that "there is no need to assume that blast has any mysterious properties. It is only a more severe diffuse sudden compression of the chest and abdomen than we see in civil cases during peacetime."

Ross(65) in comparing such peacetime and wartime injuries gives the following points of differentiation:

1. In compression asphyxia the lesions are not necessarily symmetrical and bilateral. The hemorrhages are usually subpleural and along the lines of the ribs with a generalized congestion and edema of the lungs.
2. In hemorrhage due to impact of a solid the hemorrhage is in relation to the maximum point of intensity and may be unilateral.

3. In hemorrhagic concussion or blast the lesions are always symmetrical and bilateral with generalized pulmonary congestion. These hemorrhages are much deeper and though subpleural bleeding does occur they are present only as an extension from these deeper areas.

Boland(5) reports several cases of traumatic asphyxia by compression injuries from earth cave ins, etc., simulating this condition. Fallon(17) gives a most interesting account of lung damage with the "intact thorax" due to pulmonary mishaps in every day life.

This theory that the mechanism of injury is produced by the action of the compression wave upon the thorax is generally accepted today by most authors. However, let us consider some of the newer theories which are now beginning to appear. The thoracic cage, as known, gives good protection to the underlying structures, whereas the abdomen is protected only by the thin layers of fascia and muscles. Could not, therefore, the blast by acting upon the abdomen force the diaphragm upward cause further compression of the alveoli? This was first suggested by Hill in 1918 in a letter to Williams(76). Williams gives this as a possible explanation of Osborn's phrenico-costal-sinus pneumonia, suggesting that this pressure from below forces the lungs outward and

causes compression of this area. This may not be the primary factor in the production of lesions, but is important in conjunction with the compression effect of the wave against the chest wall. The diaphragm would not descend to its normal position and would exert a greater pressure upon the air-containing alveolar spaces.

McKibben(48), as early as 1919, suggested that this exposure to blast might result in the production of fat emboli. He experimented on rabbits, cats, and dogs and did find intravascular fat in the cerebral vessels. He further states, however, that he also found an equal amount in the same vessels of dogs killed by ether inhalation or by bleeding.

Robb-Smith(63) relates one patient who had died from bomb explosion without gross injury and that considerable fat emboli were found at autopsy. He acknowledges the fact that his findings have not been confirmed by other authors, but remarks that the pathologists have failed to look for fat emboli in pulmonary concussion. He suggests that symptoms may be the result of a combination of pulmonary concussion and fat emboli. Regardless of the true cause, these should be sought for in post mortem examinations on air raid casualties, the only method by which this can be clarified.

Rowlands and Wakeley(66) submit a method of differenting the two. In blast injury pulmonary symptoms are present immediately, whereas in fat emboli the manifestations

usually do not appear before the third day following injury. Likewise physical signs of the former are different, there being diminished diaphragmatic movements, emphysematous appearance of the chest, and impairment of the percussion note at one or both bases with or without crepitation; facts not noted in cases with fat emboli. A careful history is also of significance.

Zuckerman(78) did not find any evidence of fat embolism in his animals which had been exposed strictly to blast, but states that this point cannot be completely excluded. He proposes that this might be a means of differentiating cases that are exposed to blast alone and those which are exposed to blast as well as an impact with a hard surface.

Connor(11) designates fat embolism more as an accompanying condition than a primary result. We can agree with him in the fact that there has not been, as yet, enough factual evidence to establish or exclude the idea that such a condition does exist.

Based upon reports of several competent observers (O'Reilly, Gloyne, & Roodhouse-56; Palma & Enright-58; Travers-75; Palma-57.) the common complaints and physical signs in blast injury are: (1) Shock, (2) Pain in the chest and abdomen, (3) dyspnea, (4) restlessness, (5) hemoptysis, (6) cough, (7) ruptured tympanic membrane, and (8) bulging of the chest wall.

As previously mentioned, the impressive feature in shock is the degree of this condition out of proportion to the apparent injury. Pallor, cyanosis, and a weak rapid pulse volume are all striking. It is undoubtedly due to some form of peripheral vascular collapse from (1) direct concussion of the head (Connor-11), (2) from transmission of the wave up the spinal cord (Mott-52; Mairet & Durante-45), or forceful retropulsion of the blood from the lungs into the head (Williams-76). Whatever the cause, it is a severe form of shock and varies directly with the severity of the compressing force.

The chest pain is of two types--a central deep constant pain and a less severe lateral pain augmented by breathing (this remains for a longer period of time than the deep pain). The deep pain is probably the result of mediastinal hemorrhage, whereas the lateral pain undoubtedly due to contusion of the intercostal muscles since hemorrhage into the extrapleural spaces is a constant finding at autopsy. These hemorrhages might well explain the bulging of the chest wall, as contusion of the intercostal muscles with subsequent hemorrhage could easily result in spasm and contraction of the intercostal muscles causing the chest to assume the inspiratory position.

Pain in the abdomen may be severe or mild. In some cases there is great pain and tenderness with muscular

rigidity. This has led many observers to do exploratory laparotomies only to find a few submucosal hemorrhages in the gut wall. It would seem that this is probably a result of irritation to the intercostal nerves. Zuckerman(79) reports this finding to be true in some experimental animals with the hemorrhages continuous with those in the sheaths of the intercostal nerves. O'Reilly, Gloyne, and Roodhouse(56) suggest that this may be in part the result of contusion of the abdominal walls themselves, but no investigation concerning this point has appeared in the literature. The important fact, therefore, is the contraindication to surgery in these cases even though the abdominal tenderness and rigidity is suggestive of a widely disseminated peritonitis.

Dyspnea is a constant feature, resulting in part from the pain and in part from the extreme bulging of the chest wall (always nearly complete inspiration). The respiratory rate is rapid, the excursions shallow, and inspiration, particularly, labored.

The cough is also a prominent feature and, though rarely appearing before twenty four hours, is spasmodic, frequently intractable, and often productive of a frothy, blood-tinged sputum. Free hemoptysis is rare and is never a prominent symptom although blood-tinged mucous in the respiratory passages is a constant finding at autopsy.

Restlessness is constantly present and bears a direct

relationship to the severity of the blast. The presence of subarachnoid hemorrhages in the central nervous system would suggest an irritative lesion rather than merely psychic trauma. This fact is supported by other cases of similar incidents where the principle injuries were caused by violence (falling debris, etc.). The restlessness in severe cases is extreme and out of proportion to their injuries. Morphine is helpful, but does not completely alleviate the symptom as it does in most cases of non-violent origin.

A good lead to the diagnosis of severe blast is furnished by otoscopic examination of the ears. A recently ruptured tympanic membrane usually indicates exposure and necessitates an X-Ray examination to rule out injury to the lungs. (Perlstein-61).

Craig(12) describes the following auditory lesions from exposure to detonation:

1. A rupture of the drumhead (the commonest finding).
2. Hemorrhage into the middle ear.
3. Hemorrhage into the fundus of the internal meatus.
4. Minute hemorrhages among the fibers of the Facial nerve and vestibular division of the Auditory nerve as well as into the canal of the tensor tympany muscle.

Most of the cases develop otitis media and perforations were common and often quite extensive. Hearing loss varies and in most cases is of the middle ear variety.

He states that the noteworth thing is the insidious development of infection seldom associated with any pain. He reports only one patient who complained of pain and that was at the time of the explosion.

Physical signs appear for the most part in the chest and vary with the severity. Respiratory excursions are reduced, usually bilaterally, and air intake is diminished. The percussion note may be resonant in the early stages with localized areas of dullness later if complications ensue. Breath sounds are faint, especially at the bases, and coarse sibilant rales are usually audible over the whole of the lung fields. There is often extreme tenderness in the intercostal spaces with muscle guard over the abdomen. Less severely blasted patients may show no definite physical signs other than a moderate bulging of the chest wall with reduced excursions and movements.

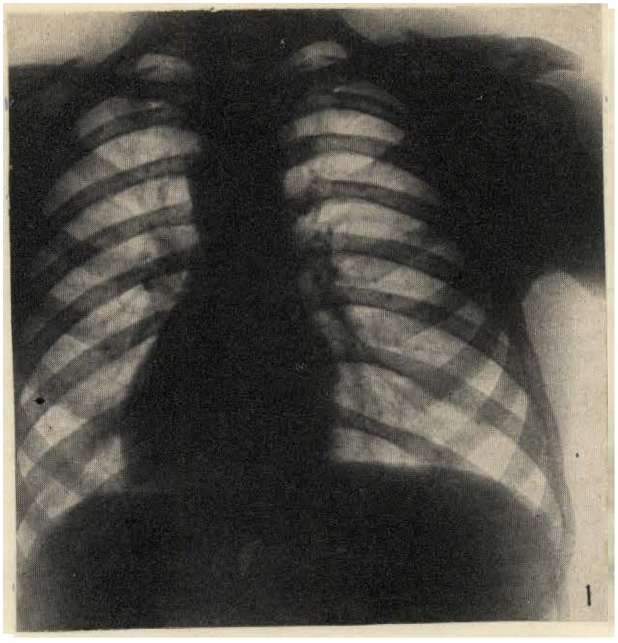
Travers(75) reports cases with surgical emphysema in the neck and thorax even in the absence of fracture anywhere in the thoracic cage. This always appeared first at the root of the neck and spread to the surface through the mediastinum.

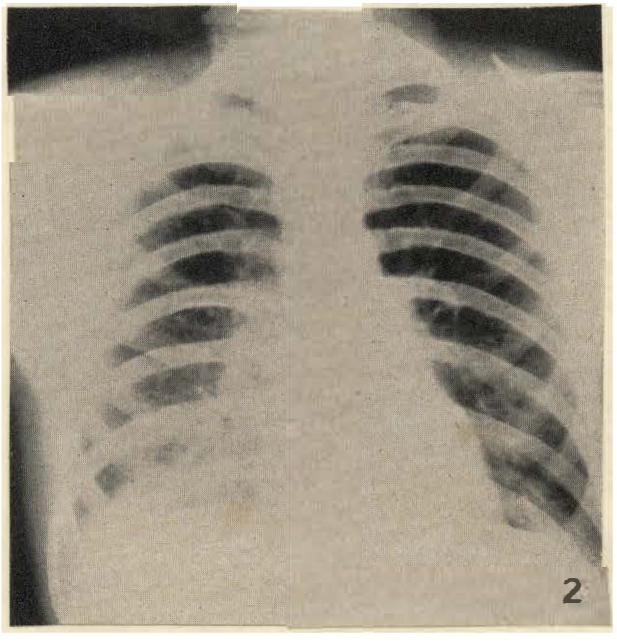
Blast may also produce pathological changes within the eyeball. Holmes(32) states that the cornea is never ruptured, but that in severe cases derrangements within the bulb are produced. Disturbances of accomodation, intra-ocular

hemorrhages, cataracts, dislocation of the lens, and choroidal or retinal tears do occur.

Roentgenograms furnish the most satisfactory method of diagnosis and evaluation of the severity of damage from blast. These reveal characteristic poorly demarcated areas of increased density (mottling) scattered throughout both lung fields, resembling somewhat a patchy pneumonic consolidation. This density is most marked just above the diaphragm laterally and in the costo-phrenic angle.(Gates-20; Osborn-53). The linear course of the densities is in most cases parallel to the course of the postero-lateral portions of the lower ribs (fig. 1 & 2). Gates(20) gives an excellent report upon roentgenographic findings in under water blast. The similarity of this condition to air blast findings leads me to cite him in these cases. The margins of the zones of density are poorly demarcated both at their proximal and distal ends. These zones vary in size from about three by one half centimeters to about five by one centimeter. About thirty per cent of the cases observed by him showed associated pleural changes, appearing as a line of pleural thickening or a film of fluid in the costo-phrenic angle.

In general the zones of increased density are less sharply demarcated and somewhat fainter than in true cases of bronchopneumonia or atelectasis. This, plus the constitutional symptoms accompanying the latter conditions





1. NOTE THE FUZZY LINEAR ZONE OF INCREASED DENSITY CLOSELY PARALLELING THE COURSE OF THE POSTERIOR PORTION OF THE LEFT NINTH RIB. (HEART TRANSPOSED.)—2. BILATERAL BASAL CHANGES. NOTE THE FUZZY LINEAR ZONES OF INCREASED DENSITY CLOSELY PARALLELING THE POSTERIOR COURSES OF THE TENTH RIBS. THE UPPER LUNG FIELDS ARE CLEAR.

would simplify the diagnosis of blast injury.

These areas of increased density are presumably due to interstitial and alveolar hemorrhage with accompanying edema and vary in extent with the clinical and pathological findings. The striking thing in these cases is the rapid resolution. The lungs are clear or almost clear by seven to ten days after injury and usually parallel the clinical course. Mild cases may show no roentgenographic signs, a fact which must constantly be kept in mind.

Pathologically, the outstanding lesions are found in the lungs, the abdomen, and the central nervous system. In mild cases the lungs alone seem to be affected and the first evidence of injury is rupture of the capillaries causing hemorrhage into the intra-alveolar septum with separation of the two layers of the alveolar epithelium. This hemorrhage may spread along the septal walls to collect around the bronchioles and larger vessels or may dislodge the alveolar epithelium and find its way into the alveolar spaces.

In the more severe lesions the interalveolar septa, including capillaries and epithelium, are disrupted and destroyed. This allows the blood to pour directly into the alveolar sacs producing a traumatic emphysema. Palma and Enright (58) demonstrated rupture of the elastic tissue of the alveolar walls in the most affected parts.

Permanent damage may thus result from such an injury to the lung tissue, as the ruptured elastic fibers and capillaries are not likely to undergo repair even though the blood corpuscles could presumably be removed from the air spaces.

No pathology is found in the abdomen other than the small mucosal and submucosal hemorrhages in the intestinal walls previously mentioned. The muscular rigidity apparently arises from hemorrhage into the intercostal spaces causing irritation of the intercostal nerves.

Pathological findings in the central nervous system will be dealt with more thoroughly in the section on this subject.

Although chest complications are relatively few, we must constantly keep in mind the fact that superimposed infections might easily gain a hold on such patients. Physical exhaustion and undernutrition of soldiers in active service in conjunction with the lesions would favor the spread of latent or mild infections of the upper respiratory into the depths of the lungs. Bronchopneumonia is undoubtedly the most frequent complication and should be considered if the physical signs persist more than six to seven days and are accompanied by a septic response. Zuckerman(79) also reports pneumonias in his animals with a preponderance of monocytic infiltration.

Diagnosis has been, more or less, considered in connection with various topics of the paper, but it is well

to mention two other important conditions, namely, pneumothorax and hemothorax. The principle difficulties arise in cases where there are other gross injuries such as a fracture of the sternum, ribs, etc. Hemothorax gives a similar picture, but produces dullness on percussion. Likewise, pneumothorax simulates it, but the marked hyper resonant percussion note points to this diagnosis.

Pathological factors affecting recovery from blast concussion are similar to nearly every other form of disease or disability. These include extreme exhaustion; anemia and subsequent anoxemia; frustration with resulting anxiety, and the instinctive impulse toward aggression, flight, or an appeal for protection. Age, arterial degeneration, emotional instability, and an abnormal sexual life may be influencing factors. Crichton-Miller(13) considers many of these factors of major importance in the production of some of the symptoms, but in my mind the presence of the previously mentioned pathology would greatly overshadow the factors.

Endocrine factors are still obscure, but there is evidence that desoxycorticosterone has a direct action in increasing aggressiveness. Toxic absorption from a septic focus also influences recovery.

At the present time Physicians are prone to dismiss many cases of blast injury too readily only to find that

serious organic lesions do persist. Therefore, since bombing of the civilian population has come to occupy such an important place in the present crisis, medical practitioners, even in civilian life, should receive more education concerning this form of injury, since ready recognition and treatment are the best adjuvants to early and complete recovery.

IV. Treatment.

The best treatment of this condition would be to devise some method of protection for both civilian and military personal and thus eliminate, or at least diminish, the severity of the injuries. At present the occupation of air raid shelters by the civilian population is giving excellent protection from the effects of the blast. People are being educated that in emergencies, where shelters are not accessible, they should lie flat in the prone position upon the ground or in ditches, holes, and gutters. The heavy musculature of the back affords much better protection than the chest wall anteriorly. However, this still does not adequately protect the men in combat. Most of the work along this phase is still in the experimental stage. King and Curtis(38), Latner(42), and others suggest covering the chest with a light bandage or jacket such as sponge rubber to protect the chest from the direct force of the compression wave. Zuckerman(77) had previously demonstrated that by wrapping animals in sponge rubber, the evidence of shock could be prevented or at least greatly diminished. It would probably be advisable to include a protection for the abdomen as well as the chest since Williams(76) showed that animals immersed in water with the chest protection alone received as severe an injury as those with no protection;

whereas, animals having abdominal protection showed minimal lesions, suggestive of upward compression of the diaphragm. As yet such a protective coat or jacket has not been devised, so that we are still confronted with patients who have been exposed to the full force of the wave.

Osborn(53) suggests that since in inspiration the lungs move down into the phrenico-costal sinuses, the blast effects would be minimized if people would withstand the tendency to take a deep breath and hold it in emergencies. He explains that at the end of the inspiratory movement the glottis is closed and the diaphragm and other respiratory muscles are relaxed ready for expiration. This portion of the lung is then left more or less unprotected, since it is covered only by relaxed muscles, making it more liable to injury.

Once the injury has been received, early recognition and prompt application of bed rest are of prime importance. Morphine in doses not less than one half grain should be given immediately to relieve the restlessness and pain. This dose, however, should not be great enough to depress the cough reflex especially in cases with hemoptysis. Some men do not agree to the use of morphine(Robb-Smith-63; Williams-76) as it depresses the respiratory center. However, Whitby(80) says that the proper use of oxygen and carbon dioxide with respiratory stimulants will easily counteract

this peripheral and central respiratory depressive effect. Williams substitutes heroin in doses of one-eighth grain. Thompson gives allopan believing it to be more effective and producing less unpleasant after effects. He stresses the fact that if inadequate sedation is given, the continued restlessness will cause further hemorrhage while being transported to a base, decreasing the chance of recovery.

Shock should be combatted immediately by bed rest and elevation of the foot of the bed. Hot water bottles and blankets should be applied freely. Fluids should be administered by vein either in the form of whole blood or plasma, preferably the latter. Matthews(47) suggests the use of powdered citrated plasma dissolved in distilled water as it is easier to administer and cheaper to produce than the popular dried-serum-protein. This has become the standard intraveinous solution for severely shocked air raid casualties in England and the Allied armies are using it nearly exclusively on the field of battle today.

Earlier investigators suggested venesection in these cases since there was a hemoconcentration of the blood with a subsequent strain on the right heart with veinous congestion. It is now generally accepted, however, that this hemoconcentration is a result of a perfusion of plasma into the extracellular tissues, so that administration of plasma will readily dilute the blood. The benefit of whole blood transfusions is more limited as it is difficult to see how a

lung containing large amounts of effused blood with rupture of the elastic tissue and capillaries can benefit to a great extent.

Kekwick, Marriott, et al(36) state that a patient with a systolic blood pressure below one hundred millimeters of mercury should immediately receive two pints of plasma after which there should be a prompt blood pressure rise of thirty to forty millimeters. If this does not occur, more fluids should not be forced, but rather the patient should be kept warm and given morphine. Such cases will often respond readily to such management. If the pressure continues to drop or is transient, bleeding from some focus should immediately be sought out.

The amount of intraveinous fluids given to any one patient cannot be listed in round figures, but must depend upon the response and needs of the patient. The judgment of the physician in charge is the prime factor in this phase of management.

Oxygen should be administered continuously for at least the first seven or eight days in association with the above procedures. This may be given by catheter, tent, or mask as all are quite satisfactory. The rate of flow should be at least eight to twelve liters per minute.

Codeine, grains one half, may be necessary to relieve the spasmodic cough, but dosages beyond this should not be

employed as it is essential that the patient expectorate the excessive accumulation of mucous within the throat.

The use of sulfonamides, particularly sulfapyridine, gives very excellent results in the prevention of secondary complications. A four gram initial dose followed by one gram every four hours is the most satisfactory. Daily study of the blood picture and urine should eliminate the possibility of complications from these drugs.(Booth-6; Bailey-2: Travers-75; O'Reilly, Gloyne, & Roodhouse-56.)

If an additional hemothorax or spontaneous pneumothorax ensues, various procedures are recommended. Lockwood(43) advises immediate aspiration of the fluid with continuous drainage by a small rubber catheter. This should be accompanied by oxygen administration to expand the lungs. Sellors(67) also favors this method as hemothorax is an excellent media for the growth of bacteria. Fallon(17), however, believes the presence of hemothorax or pneumothorax is no indication for operation providing the clinical picture remains good. If the hemothorax is unchanged over a period of ten days or a septic course intervenes, however, aspiration with continued drainage should be instituted, augmented by the oral administration of sulfonamides.

Kretschmar(39) suggest supplementing these conditions by an artificial pneumothorax to prevent traumatic

pneumonia and stop the hemorrhage. It seems logical that this procedure would also immobilize the lung, thus assisting it to heal after which re-expansion could be accomplished. It would further prevent the formation of adhesions between the visceral and parietal pleurae, a condition which might otherwise easily develop.

The value of immobilization of the lung in enhancing the healing process is debatable , but external application of a binder surely deserves a trial. In fact this would be more practical than producing an artificial pneumothorax in uncomplicated cases of hemothorax, the danger of secondary infection by the latter procedure is always present. Hadfield (24) believes inadequate immobilization may be the exciting cause of relapses in some patients who have apparently completely recovered.

General anesthesia is absolutely contraindicated in these cases. An early roentgenographic plate of the chest should always be done before any operative procedures are instituted. Falla(16) reports one case in which pulmonary blast had been overlooked and a general anesthesia given to repair a hip laceration. This patient very shortly went into shock and died. Autopsy showed the typical minute hemorrhages over the lung fields. Intravenous evipan or pentathol sodium augmented by small amounts of cyclopropane with oxygen will usually suffice in cases where operations are absolutely necessary

indicated. (Cohen & Schulenbergh-10)

A sterile needle puncture will usually give immediate relief to pressure symptoms upon the trachea and mediastinal structures from traumatic emphysema.

General considerations which must be attended in these patients include such things as maintenance of fluid and electrolyte balance with glucose and saline, nourishment by mouth, enemas or Wangensteen suction for intestinal distress, and caloric and vitamin requirements.

Nurses, relatives, etc. should be required to wear masks, thus minimizing the danger of droplet or air born infections to which they are so susceptible. Strict attention to nursing hygiene is essential. Nursing care under strict isolation might even be of value providing such facilities are available.

In case of ear injury Craig(12) recommends mopping away of the discharge with a cotton wad and either blowing in iodine or boric acid powder (.75%) or dressing with one half inch ribbon gauze soaked in acriflavine (1:1,000). Insufflation of sulfonamides or ear plugs of cotton wool impregnated with petrolatum soap or candle grease will suffice if infection is present. It has been suggested that the patient bite on a cork to keep the mouth open and thus equalize the pressure within the middle ear. However, the relative differences in the diameters and length of the exit of the

meatus and the eustachian tube along with the fact that the latter is opened normally only by the act of swallowing would tend to make this procedure seem a bit irrational.

Eyes which have been injured by the blast should be immediately placed at rest by dilatation of the pupil and a binocular bandage immediately applied. The patient should be confined strictly to bed for at least one week.

IMMERSION BLAST INJURY

Blast injury to the body from the detonation of high explosives under water has been given the name of immersion blast and although similar in many respects to atmospheric blast, deserves special mention.

The mechanism and biophysics of immersion blast injury results principally in damage to the lungs and to the walls of certain loops of intestines. This damage may be slight or extreme depending upon the intensity and proximity of the blast wave. Effects in the lung are homologous to those found in atmospheric blast, but intra-abdominal injury is more pronounced consisting of severe submucosal and mucosal hemorrhage or complete perforation.

Several theories have been propounded as to the nature of the blast in under water explosions, none of which have been generally accepted. It is agreed that sound waves are much more readily transmitted in liquid than in air, the mechanism of which is beyond the scope of this paper. Likewise, all observations on immersion concussion have shown that the critical distance from the center of detonation of a given charge of high explosives is approximately four times greater than in atmospheric or air blast. Therefore, rather than twenty feet, as shown by Zuckerman on his laboratory animals, it is increased to at least eighty feet in water. This is propounded on the fact that the blast wave is an

excessively intense longitudinal sound wave and that since sound waves travel four times as fast as in air, it is only reasonable to assume that the critical distance is four times as great in the water.

Friedell and Burke(18) give an excellent account explaining the mechanism of the immersion blast. There are two types of effects, the instantaneous percussive effect and the continued pressure effect. The first maximum is the result of brisance of the explosion with very little, if any, gas pressure. The continued pressure effect on the other hand is due to this gas pressure which is present in water, but not in air. If this latter effect continues to develop, the entire column of water is then propelled in the direction of the propelling force. As water is a dense and practically incompressible media, the gas pressure can be readily transmitted, whereas in the air this is immediately dissipated and plays only a minor role.

The initial percussive force alone is produced in air blast. Since only hemorrhagic lesions of the abdomen are are found with no perforations, it seems only practical to assume that these result from the effect of the percussive force alone, representing a force greatly mitigated by the effects of distance. In water, however, the pressure continues to be exerted, expressing itself in the motion of the entire column of water. This is much greater than the

primary percussive blow alone and explains why the effect of immersion blast is great at an increased distance. This greater force against the abdominal wall was their explanation of the production of perforations in immersion injuries.

The mechanism of injury once the compressive wave has struck the body wall has not, as yet, been considered. The injury to the lungs beneath water is altogether different from that of air blast. It will be recalled that in air blast the injury is due to the compression of the chest wall against the air filled alveolar spaces. In immersion blast the mechanism is not one of compression, but rather the result of shredding and re-expansion of a compressed air bubble enclosed within the body tissues. Ordinarily a compression wave tends to reflect as a tension wave when it strikes another medium such as the surface. However, if this force is approximately five hundred pounds per square inch, it breaks through into the air with a shredding effect and literally "blows off" the surface. An object in the water will thus be projected into the air as a result of this force.

When the air exists as a bubble within an enclosed space, such as the body cavities, it undergoes collapse and compression for that fraction of a second during which the compression wave acts with immediate re-expansion upon passage of the wave. Upon striking the body such a comp-

ression wave of five hundred pounds per square inch or greater is transmitted through the tissues and upon reaching the alveolar air breaks through into this gaseous medium with a shredding effect, tending to "blow off" the surface of the tissues. This is sufficient to damage the alveolar walls with its vulnerable enclosed capillaries.

Perforations and hematomas of the intestinal walls are also produced by this mechanism, discrediting the early belief that they occurred from overdistention of the intestines by the inrush of water into the anus. More evidence in favor of the shredding theory is the fact that most lesions are found in the air containing portions of the intestine. Also in all cases thus far reported only one has complained of the sensation of water rushing into the anus and penis.

McMullen(49) suggests another mechanism by which perforations might occur. He states that rupture of the intestinal vessels results in ecchymoses and hematomas in the intestinal walls, which in turn produce ileus. This gradually increases the intra-intestinal pressure until the intestines subsequently perforate. This pressure then continues to act by keeping the perforation from sealing off so that a generalized peritonitis or an abscess may ensue.

Hamlin(29) likewise suggests that the blast interrupts the reflex mechanism of the bowel wall resulting in ileus

thus decreasing the motility and blood supply to the area so that contused areas of the bowel become susceptible to gas pressure, necrosis, and possibly perforation.

However, Williams(76), Greaves(23), and Friedell & Ecklund(19) report in both clinical and experimental observations that the perforations are definitely related to the presence of gas within the lumen and that no lesions were found where the gastro-intestinal tract was empty and collapsed. The lesions themselves showed little interstitial hemorrhage and no bleeding into the lumen was noted, giving the appearance of a sudden disruption of the wall from within outwards. There was no evidence whatsoever of the necrosis of the bowel wall as mentioned by McMullen and Hamlin.

Greaves(23) conducted two additional experiments to further prove this point. He first injected twenty cubic centimeters of air into the peritoneal cavity of a rat which was then subjected to a lethal underwater blast. Laceration of the liver and spleen with extensive hemorrhage into the abdominal cavity resulted, conditions not noted in the normal blast exposure. The second experiment consisted of exposing four normal segments of rabbit intestine, tightly ligatured at both ends, to a blast. The first was completely collapsed, the second filled with air, the third with normal saline solution, and the fourth with saline and air. The first and third were unaffected, whereas the two containing the air

were ruptured. This is strong evidence that air is undoubtedly a contributing factor.

The gastro-intestinal tissue itself is resistant to the effect of the wave. However, if air or gas is present within the lumen, the wave will be shredded as it passes through. If this gas can be displaced without compression, a hemorrhagic lesion of the wall, marking the site of the shredding, will be the only result. Sometimes, however, these bubbles become entrapped within the lumen. These are then compressed by the pressure of the wave only to re-expand once the wave has passed. If the wall has been sufficiently weakened by the shredding effect, it will break down before the responding bubble and a "blow out" perforation will occur.

When the pressure approaches five hundred pounds per square inch, pulmonary damage can be expected, but a considerably larger force is required before severe intestinal damage occurs. This tends to displace the earlier belief that intra-abdominal injury is the outstanding pathology in immersion blast, a theory supported by Palma(57), Palma and Uldall(59), and Atkins(1). That pulmonary damage occurs first, as in air blast, is further stressed by Friedell and Ecklund(19) who exposed guinea pigs to immersion concussion. They found that animals with chests protected did not die as soon as those without protection. Pulmonary lesions were produced with relatively small charges whereas much larger

forces were required to produce evidence of intra-abdominal injury. All unprotected animals who showed intestinal perforation were immediately killed by the blast. They demonstrated, however, that by protecting the chest of these animals with sponge rubber intestinal perforations could be produced without being fatal. These animals could also withstand more severe degrees of exposure. From these remarkable experiments they concluded that injury to the lung was the immediate cause of the lethal outcome. This theory is supported by Cameron, Short, & Wakeley(8), Williams(76), and Greaves(23).

With the relatively meager amount of literature so far reported it is definitely impossible to conclude which area of the body is more effected. We do know that intra-abdominal injury is more outstanding in immersion than in atmospheric blast, though not to such an extent to exceed the pulmonary effects to any marked degree.

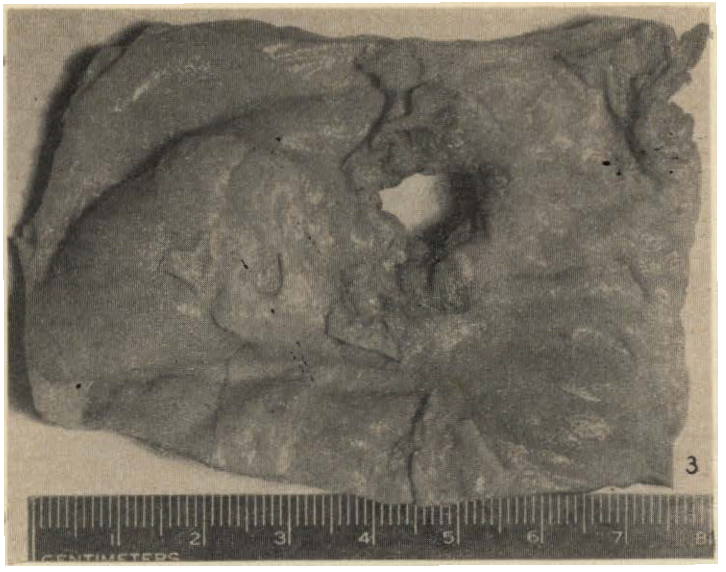
There is comparatively little literature concerning immersion blast in humans. Atkins(1) was the first to report any cases. He reviews three patients injured by depth charges while swimming. Two of these had perforations of the ascending colon and the other showed multiple petechial hemorrhages and edema of the intestinal walls. He also describes lesions and lacerations of the lungs and brain.

Breden, d'Abreau, and King(7) review the literature and

add histories of cases subject to detonation while in the water. They noted submucous hemorrhage and subperitoneal hemorrhage with perforation, but stated that very little bleeding occured in the area adjacent to the perforations. They also described two cases with rectal and subphrenic abscesses which recovered after drainage. Gordon-Taylor(22) attributes this abscess formation to organisms which have the ability to permeate the contused intestinal walls, especially the colon.

Reports since this time have agreed on several striking features; namely, the severe intra-abdominal injury noted in each instance without visible external injury; the tendency for the ileo-cecal region to sustain the severest damage; the "blown out" appearance of the perforations; the extent of peritonitis and the attempts at walling off exhibited; the absence of rupture of other abdominal viscera; and the fact that in no autopsy was mesenteric thrombosis with subsequent devitalization of the bowel demonstrated.

The perforations are usually oval in shape and are often situated in linear tears through the mucosa and submucosa in the bases of encircling folds (Fig. 3 & 4). The edges may be slightly necrotic, but the adjacent gut wall is in comparatively good condition. They seem to be located in portions of the intestine containing gas, but may appear in areas containing fecal material in scybalous form. This



ig. 3. Perforation in duodenum 8 cm. from pylorus.

Fig. 4. One of two perforations in in posterior wall of ecu .

helps to explain the common location, i.e. the terminal ileum, cecum, posterior wall of the ascending colon, and the first part of the jejunum.

Microscopic examination of cut sections through the perforations show that the mucosa, submucosa, muscularis, and serosa are fragmented and perforated. The opposing edges are heavily infiltrated with leukocytes, lymphocytes, and plasma cells, with considerable superficial necrosis. The adjacent serosa has a heavy deposit of exudate. Comparatively little regenerative cell growth, fibroblastic activity, capillary extension, or evidence of repair is hardly ever noted around the perforations. Smooth muscle fragmentation is also striking.

If perforation has occured, the peritoneal cavity contains purulent fluid which often drains into the pelvis. This usually produces a very fecal odor. There is evidence of a generalized, diffuse, exudative peritonitis with massive extravasions of blood into the subserosal tissues from contusion of the anterior abdominal wall.

In cases where perforation does not occur, massive hemorrhage into the serosal surfaces of the terminal ileum, cecum, and ascending colon is a constant finding at autopsy. This appears to be the result of innumerable small torn vessels rather than from a single source. The peritoneal cavity usually contains varying amounts of fluid or clotted

blood.

With or without perforation the lumen of the intestines always contains varying amounts of gross blood.

The lung findings are also outstanding and conform very closely with those previously described in air blast, so that further discussion of that subject need be necessary.

The cause of immediate death in such cases is similar to that of atmospheric blast, but differs if the patient has survived the initial compression and has developed intestinal perforations. He then undoubtedly succumbs to the extreme toxemia from the diffuse generalized peritonitis. The mortality rate in cases thus far reported is relatively higher in immersion than in air blast. Undoubtedly some cases were not seen until one to several days had elapsed from the time of injury, so that a non-operable peritonitis had already ensued. This, plus the intense lung injury, had subjected the body to a severe toxemia. Had they been seen earlier, their condition could have been treated with far better results. Therefore, with the increasing amount of literature physicians today should acquaint themselves with this form of injury and adopt an early treatment which would help decrease the mortality rate.

Symptoms and case histories of immersion blast casualties fall into three main groups: (1) mild pulmonary damage with obvious abdominal injuries that recover completely without

operation; (2) severe pulmonary damage with severe abdominal injuries, such as lacerations disclosed at operation; and (3) late complications, such as pneumonia and abscess formation.

In the first group the clinical features are almost identically similar. Vomiting within an hour, hematemesis, abdominal pain with tenderness and rigidity, melena with diarrhea of about two to three days duration, and pain in the testicles.

The symptomsin (2) are similar, but more marked with extreme muscular rigidity, tenderness and distention; hyperpyrexia(102-103°); extreme air hunger with cough and expectoration of frothy blood stained sputum; and tachycardia.

Complications should be closely guarded against. Abscesses may develop quite insidiously often appearing as firm, tender, palpable masses in the sub-phrenic or peri-rectal areas in patients who otherwise seem well on the road to recovery. The salient features are the gradual development without alarming signs, a moderate pulse and temperature rise, no abdominal rigidity, and a leukocytosis rarely exceeding twelve thousand.

There are several factors which enter into each individual case and explain the variation in the symptoms. The distance from the center of detonation is of extreme importance, severity of symptoms decreasing proportionately with an increase in distance. This applies, likewise, to the

size of the bomb. Relative position in the water also modifies or increases the symptoms depending on whether they are horizontal (prone or supine), perpendicular, facing the blast, or with their back turned. Palma and Uldall(59) report one case who was swimming on his right side at the time of the explosion with the left side of the chest practically out of the water. Upon admission his findings were nearly completely limited to the right side.

Those facing the blast or swimming in the prone position develop symptoms as soon as ten minutes following the explosion. These symptoms vary from mild air hunger and fleeting abdominal pains to severe hemoptysis and dyspnea with extreme abdominal pain, tenderness, and cramps. The groups having their backs toward the blast or swimming in the supine position develop similar but milder symptoms and these tend to be mostly in the chest.

All cases develop some degree of shock. Hemoptysis always appears within the first twenty four hours, if at all. Abdominal distention usually does not make its appearance before the second or third day following exposure.

Jacobs(34) reports that a common experience was the sensation of the feet and legs being torn from the body or shortened out of proportion to the rest of the body. Others thought they had been struck in the base of the spine while others had the urge to urinate and defecate. None complained

of the sensation of water rushing into the rectum and penis. These symptoms are similar to those found in animal experimentation since many of the animals were unable to walk immediately after the exposure, but dragged their hind legs for several minutes, apparently completely recovering after this time. (Friedell & Burke-18; Greaves et al-23) This is probably the result of compression of the spinal nerves against the vertebrae at their point of ~~emission~~ exposure from the vertebral foraminae.

The testicular pain is an inconstant complaint and consists of a dull aching sensation which usually persists for several hours. Direct compression of the wave against the testicle is the probable etiological factor.

Physical findings of chest are homologous to those in air blast and the reader is referred to that portion of the discussion. Abdominal signs are striking and vary from moderate generalized soreness and rigidity to abdominal distention and board-like rigidity. Percussion usually yields hyper-resonance with the presence of shifting fluid level in cases of perforation. The severe cases have limited diaphragmatic movement, abdominal tympani, and a silent abdomen on auscultation. There is often ecchymosis of the abdominal wall with bluish discoloration around the recti muscles, suggestive of laceration and contusion of the intestinal walls.

Roentgenographic findings are of great significance and give much information as to the extent of injury. These findings include: (1) Gaseous distention of the small intestine and colon indicating ileus and not obstruction. These are essentially the same as peritonitis of any origin. (2) Free gas in the peritoneal cavity indicating perforation of some viscus (Fig. 5). (3) Abnormal soft tissue densities, presumably fluid, hemorrhage, or exudate (Fig. 6). (4) Numerous gas bubbles within the areas of soft tissue densities in some cases, thought to be due to perforations which were dissecting into the abdominal wall or retroperitoneal structures. (5) In some cases gastro-intestinal studies may reveal slight dilatation of some of the loops of the jejunum and ileum with slight "puddling" as well as some segmentation. These abnormalities are apparently due to hemorrhage and edema of the intestinal walls, a condition which is nearly always found at post mortem examination. Gates(20) reports a case which developed a fistulous tract which was detected by this method.

These are very characteristic findings, but it must be remembered that they do not all occur in any one patient and are merely suggestive signs to the underlying pathology. They are of extreme value to the surgeon who must weigh all evidence before deciding upon any surgical procedure.

This discussion has presented a more or less "textbook"

picture of cases of immersion blast injury. It must be kept in mind that such a complete picture seldom, if ever, presents itself to the medical man, but with a knowledge of the above facts he may be led immediately to suspect such a condition in patients who have been exposed to blast, making him realize the seriousness of it.

Translating the above observations and reports into the field of practical application we can see there are two things which must be considered. The first is the prevention or minimizing of the injuries and the second is the intelligent treatment of injuries based upon the type that may occur.

Zuckerman(77) reported that when his animals were protected by a covering of foam rubber about the trunks injuries were minimized. Similar studies were undertaken by Greaves et al(23)

Four materials were investigated--foam rubber, kapok, adhesive plaster, and thin metal obtained from the metal packing material in a blood plasma container. Coverings resembling life jackets were made of these substances and the animals exposed to under water blast. Foam rubber and kapok both proved to be very satisfactory if kept from getting saturated with water. It is believed that the protective action of these substances is due to their ability to disperse the compression wave sufficiently to prevent or minimize its destructive effect. Metal was slightly less protective,

but there were multiple fractures where the body came in contact with the metal edges (especially the extremeties).

These writers - authors fellows state that, no matter what protective device is used, that it must have the following properties before it can be given serious consideration: (1) it must possess protective qualities; (2) it must not interfere with the actions of the men aboard ship; (3) adjustment must be as simple and rapid as possible; and (4) it must contain materials with a low priority rating.

They suggest a sleeveless type coat of kapok enclosed in muslin and this in turn enveloped in a pliable, waterproof material. This kapok should be placed in compartments of the outer covering of the jacket. It should have an attached, padded collar which fits snugly about the neck when tied in front. The coat should be slightly double breasted and the skirt should reach to the level of the greater trochanters of the femur. There should be a padded tail to be drawn up snugly between the legs forming a crotch piece to protect the scrotum and testicles. This should then have three ties across the front. The thickness of the pads should be the same as the standard Navy life jacket. This should give adequate protection and greatly minimize the dangers of blast exposure as well as add to the buoyant qualities of the now standard jacket.

As a further means of prevention the personnel should

be thoroughly instructed how to cope with underwater blasts, the following standards being stressed: (1) wear the life jacket properly adjusted and tied at all times; (2) swim away from an expected explosion as rapidly as possible; (3) Make use of any floating object which will draw the body out of the water as far as possible; (4) keep the head above the water, as Greaves has shown skull fracture with meningeal laceration does occur with the head immersed; (5) Do not lie on partly submerged rafts when explosions are expected, but stand or sit so that the shock won't be transmitted to the chest or abdomen; and (6) turn the back toward the point of an expected explosion.

b Adherence to these above principles should greatly minimize the dangers in immersion blast injuries. These statements are, as yet, only theoretical, but with the increasing incidence and knowledge of this condition and the severe damage thus far reported, surely application of such principles demands a fair trial.

Adequate treatment is based on alleviating the effects produced by the pathology. The shock should be treated by immediate bed rest, warmth and the administration of plasma as described in air blast. Severe lung injury likewise occurs in this condition, so that application of the principles of air blast should all be carried out in these patients.

The two types of lesions that occur in the gastro-intestinal tract make it mandatory to differentiate between them before treatment can be undertaken. Due to the fact that the majority of the patients do not sustain perforations, even though physical signs may be suggestive, conservative management should be carried out. Surgical intervention is indicated only in cases with definite signs and symptoms of intestinal perforation. The abdominal distention can usually be relieved by enemas and decompression with a Wangensteen suction or a Miller-Abbott tube. Some sulfonamide, preferrably sulfadiazene, should be given in adquate dosage. An initial dose of four grams followed every four hours by one gram is preferable. The general practice in the armed forces in the past year has been to give it in large doses until the blood level is approximately forty milligram per cent. This is contradictory to statements in nearly all modern medical books, but it is proving very much more effective than the above described dosage; secondary manifestations are no more frequent. Nevertheless, since adequate reports have not been submitted, this treatment has not been and will not be completely accepted for some time.

If an abscess has developed, excellent results are obtained from surgical drainage.

Cases which are obtained within twelve hours after injury and give definite indications of intestinal perfor-

ation, should be subjected to immediate laparotomy with rapid transverse closing of the perforations. This should be accompanied by washing out the free intestinal contents and possibly the introduction of sulfonamide powder directly into the peritoneal cavity, depending upon the experience and belief of the surgeon. Patients received beyond the twelve hour limit, however are not likely to benefit by an abdominal exploration, but rather should be treated conservatively with morphine, oxygen, decompression, saline, glucose, and absolute bed rest with sulfonamides.

Jacobs(34) advises placing a short piece of a rectal tube through a small incision in the abdominal wall of patients received several hours after the injury. This, he states, would prevent the abdominal wall from stretching. It is a minor procedure and can be done under local anesthesia without moving the patient. He reports excellent results. It is hard to accept this, however, as being more beneficial than the conservative treatment alone for it might increase the danger of secondary infection of the peritoneum by the introduction of organisms from the outside.

Pugh(62) reports a case of volvulus of the small bowel which became gangrenous. He attributed it to an adhesion apparently received from a previous immersion blast injury. The gangrenous segment was exteriorized and drainage instituted. One week later, the patient's abdomen was again

opened under spinal anesthesia and the two bowel segments reunited by a Murphy button. His recovery was satisfactory.

It must be remembered that in cases of severe abdominal injury requiring surgical intervention there is also severe damage to the pulmonary tissues, so that general anesthesias are contraindicated. Most men report satisfactory results with eith spinal anesthesia, intraveinous pentathol sodium, and a combination of both.

All in all treatment is dependent upon several factors and the choice of procedure is dependent once again upon the good judgment of the physician in charge.

THE EFFECT OF DETONATION
UPON
THE CENTRAL NERVOUS SYSTEM

Judging from the literature on blast injury in general, little, other than theoretical considerations, can be said regarding the exact physical mechanism and the neuropathology of the effect upon the central nervous system. It is generally concluded that the central nervous system is not as vulnerable as the thoracic and abdominal organs when exposed to under water detonation. The exact mechanism of death in air and immersion blast, as previously cited, is still undecided, but the quick death of well preserved specimens of fish in experimental studies suggests a neurogenic cause, although there are undoubtedly other factors. (Mairet & Durante-45)

Hamlin, who gives an excellent report on immersion blast casualities, favors the early belief that the so called "shell shock" from windage was due to the transmission of the compression wave to the spinal cord and thence to the cranial cavity with resulting capillary rupture and damage to the vital centers in the base of the brain, a theory even yet not disproved. The fact that the bony casement of the skull and spinal canal would afford excellent protection for its fluid-cushioned contents in contrast to that provided for the abdomen and thorax would favor such a theory. (Mott-51;

Githens & Metzler-21) This force could conceivably damage the delicately supported vessels of the leptomeninges, giving rise to subarachnoid hemorrhage and neurological symptoms, such as headache and backache. Injury to the cortex and deeper brain centers is also possible.

Many men report mild subarachnoid hemorrhage found at autopsy, but few relate any neurological symptoms other than restlessness. Williams reports that many of his animals were unable to walk immediately following exposure, but soon recovered; the major effect appearing mostly in the lower extremities. It was previously reported that a few authors reported such sensations in patients. This is presumably the result of compression of the nerve against the vertebra or from a stretching effect.

Stewart, Russel, and Cone(71) report the case of a pheasant suffering from the blast effects of a high explosive bomb. This bird was in a dazed, non-responsive condition resembling catatonia. They found capillary congestion throughout the cerebrum with gross hemorrhage into the fore-brain. Such widespread hemorrhage has not been presented upon clinical cases, but from such a report it may be assumed that a similar condition could occur providing the compression force was severe enough.

Several cases have been knocked unconscious by the initial blast. & according to Williams(76), is due to a con-

cussion in no way different from that caused by any other form of violence. Hamlin(29), however, theorizes that this unconsciousness is probably more often caused by a rapid shift in the vascular reservoirs of the great vessels, producing transient ischemia of the brain rather than concussion.

Since the head is nearly always out of the water in immersion blast, it seems it would be less affected than if it was immersed at the time of the explosion. Most men report, however, that injury to the head itself rarely occurs whether above or below the surface. Greaves(23), by immersing the heads of animals and subjecting them to blast, showed that they all presented extensive epidural and subdural hemorrhage, lesions not found in animals with their heads above the water. He also noted fracture of the base of the skull in several animals. Thus, the direct compressive force would seem to have some effect. This merely adds to the confused picture so far presented and stresses the point that this subject needs more observation before any conclusions can be drawn.

Hamlin(29) gives the only detailed report to date on the neurological observations from blast injury. His patients (ten in all) complained of mild to moderately severe headache, but due to their cloudy sensorium, were unable to give a reliable history. Their memory for sequence of events was poor and the history obtained was pieced together from

many interviews and from corroboration obtained from other victims. The only persistent neurological finding in all cases was a dimunition in reflexes. Persistent abnormal neurological findings were recorded in only two cases which included a bursting type of headache, sensitivity to noise, nightmares, loss of perception of high tones, spontaneous involuntary movements of the upper and lower extremities, and an occasional contracture in the musculature of the neck and trunk. Free intervals were present and movements were absent during sleep. One case had some personality change and speech defect, but as a general rule everything seemed to be on an organic basis, suggesting focal extra-pyramidal damage as well as diffuse cortical insult.

Crichton-Miller(13) thinks the emotional factor of fear is also of significance and that the reaction patterns are definitely related to the psychological state of the patient when the blast knocks him out.

Of the ten cases reported by Hamlin, three showed sub-arachnoid red cells and slight elevation of the total protein of the spinal fluid. Some degree of concussion of the leptomeninges, therefore, did presumably occur in these cases.

Thus, it can be seen that the central nervous system is liable to a relative degree of injury in blast which should be sought for by clinical examination and lumbar

puncture. Complete confirmation of neurological notions about this condition must await histological study of the nervous system of fatal cases and similar material derived from animal experimentation.

Treatment of this condition cannot be delineated as in other conditions since a definite pathological pattern has yet to be described. Therefore, the general measures of bed rest, good nursing care, and the administration of morphine, as prescribed in the previous conditions will suffice.

May I conclude this discussion by saying that an excellent article entitled "An Experimental Study of Under-Water Concussion", has been submitted for publication by a group of men working at Bethesda, Maryland, but owing to understandable restrictions it was not considered feasible to release. We hope that this can soon be published, as perhaps it may clarify some of the mystification on this subject.

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SUMMARY AND CONCLUSIONS

1. A summary of the early and recent knowledge concerning "blast" injury is given. This includes historical development, nature of the blast, experimental evidence, clinical evidence, and treatment of atmospheric and immersion "blast" with a short resume as to the effects of the detonation upon the central nervous system.

2. The blast wave is composed of an initial compression wave followed by a suction, the two being propigated outwards.

3. The pathology is primarily produced by the impact of compression wave against the chest wall. This may be supplemented to a minor degree by the "windage" and the suction component of the wave.

4. Pulmonary damage with marked hemorrhage into the alveolar spaces of the lungs appears to be the immediate cause of death in both atmospheric and immersion "blast".

5. Abdominal injury is more severe when the body is subjected to underwater detonation. These injuries are largely confined to the gas containing portions of the hollow organs, with hemorrhage in the walls and lumina and occasional perforation.

6. Fat embolism and liberation of nitrogen bubbles into the blood stream play a minor role in this condition.

7. Roentgenograms furnish the most satisfactory method of diagnosis and evaluation of the severity of the damage.

8. The striking feature in these cases is the rapid resolution of the hemorrhage, paralleling the clinical course.

9. General anesthesia is absolutely contraindicated.

10. Hemorrhage into the intercostal spaces with subsequent irritation of the intercostal nerves may result in rigidity of the abdominal wall, simulating a severe peritonitis.

11. A sleeveless type coat of kapok enclosed in muslin and enveloped in a pliable, waterproof material similar in thickness to the standard Navy life jacket is suggested as a prophylactic measure in immersion "blast".

12. The following factors modify the effects: (1) distance from the explosion; (2) size of the detonating object; (3) relative position of the subject to the bomb; (4) state of inflation of the lungs; (5) presence of a protective covering; and (6) the presence of air in the viscera or tissues.

13. Cerebral concussion is not a common result from the impact of the compression wave. Death is apparently due to some other factor although the possibility of compression and ischemia of the vital centers of the medullat cannot be definitely excluded. Subarachnoid petechial hemorrhages have been found in a few autopsies.

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